LINC403

CLINICAL LINGUISTICS

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SYLLABUS

Objectives:

Clinical linguistics is an integrated field drawing from the field of speech and language pathology and linguistics besides basic input from medical and paramedical sciences like anatomy, neurology, pediatrics, genetics, physiotherapy, occupational therapy, behavior sciences like clinical psychology, education etc. Clinical linguistics thus involves collaborative work with interdisciplinary interactions studying language development and disorders in a clinical setting. Keeping this in view, this course is intended for improving the clinical skills and linguistic insights into speech and language disorders.

UNIT- I: Introduction to Clinical linguistics

Multidisciplinary in nature – integration of disciplines such as psychology, phonetics, linguistics, Medicine and pedagogy – communicative difficulties due to speech and language pathology - causes - anatomy of the brain: cerebral cortex - hemispheres - lobes – cerebellum - mid brain - thalamus - corpus callosum - cranial nerves - physiology of speech process.

Exercises

UNIT – II: Language disorders

Communication disorders: Speech disorders and language disorders – Dyslexia - Dysgraphia - language in ADHD - language in Autism Spectrum disorders - language in schizophrenia – Alzheimer's - language in mental retardation - language in cerebral palsy - Hearing Impairment - language in hearing impairment - Language delay/SLI.

UNIT – III

Aphasia

Aphasia – etiology – characteristics – the contributions of Paul Broca and Carl Wernicke – Classification of Aphasia – Anomia - linguistic interpretations

of aphasia - phonological –morphological- syntactical - semantical -fluent as non fluent- Agrammatism and paragrammatism.

UNIT - IV

Speech disorders

Articulation disorders: cleft palate, cerebral palsy - Fluency disorders: Stuttering, cluttering - voice disorders: dysarthria, dysphonia – Assessment of speech disorders using PRAAT.

UNIT – V

Assessment and Remediation

Need for assessment – Test batteries – BDAE and WAB and other important tools – Assessment of linguistic parameters – Phonological, morphological, syntactic and semantic assessments – clinical solutions – remediation and suggestion for linguistic development – evolution after therapy – problem solving test programmes – Training for the data collection from the subjects.

UNIT – I

Clinical Linguistics: Multidisciplinary in Nature

Linguistics being a science, which studies the structure of languages, has its full potentiality over each and every human being's life. It is like; life without language for a human is life less. In recent years, some remarkable growth has taken place in our knowledge and management of language and speech disorders in children and adults.

Medical disciplines have been working a lot for past two decades to identify, assess and to remediate these problems. Also theoretical developments in linguistics and its applications have been utilized for the study of speech and language disorders, by clinicians during past decade. This applied study of linguistics with medical discipline is very much useful for the diagnosis and treatment of language and speech disorders. This study is termed as clinical linguistics.

According to Crystal (1986) 'Clinical Linguistics is the application of the theories, methods, and findings of linguistics (including Phonetics) to the study of those situations where all language handicaps are diagnosed and treated'.

This is otherwise also called as "Remedial Linguistics" as it is used in non- medical settings like educational and psychological context to diagnose and to remediate a problem of a student in educational and a client in psychological settings. So, clinical linguistics attempts to use the linguistics techniques to solve the problems in the domains like assessment of language and speech disorders, language teaching and speech therapy. It also applies linguistic theories to study the language disabilities in all its forms.

CLINICAL LINGUISTICS: ROLE OF LINGUISTICS ALONG WITH OTHER FIELDS

Clinical Linguistics deals only with communication disorders (i.e. speech and language disorders) which has only linguistic symptoms. So, along with Clinicians, speech- language pathologist, psychologist and educationist, there is a major role for a linguist to be played in the Classifying, Describing, Diagnosing, Assessing and providing Intervention to the disorders along with other professionals.

Concerning classification; the linguist has to clarify the areas of confusion found in the traditional metalanguage and classification of the disabilities. The terminologies given for each disorder are often confusing, overlapping and also misinterpreted. For example, 'Learning Disability' is now widely used as an umbrella term for the listening, reading, writing and mathematical disorders. But, Louise Cummings (2008) in his book 'Clinical Linguistics' uses this term for Mental Retardation or handicap, where the cognitive ability will be subnormal. But this subnormal cognitive ability is not seen in the children who have listening, reading, writing and mathematical disorders. Consecutively, according to OnitaNakra (1996) and PrathibaKaranth (2003), children with learning disability have normal/above normal intellectual capability. Thus these confusions can be resolved by involving the linguists for providing systematic linguistic descriptions.

On the subject of the descriptions of disorders, there is a great need for descriptive case studies of the language of disordered people. Also normative models of language development are must to describe the delay found in child language acquisition, which can be provided only by a linguist.

Next, in the part of diagnosis and assessment there is a need to classify the linguistic behavior and to list out the deviant linguistic features of disordered population. Widely the disorders are classified in terms of medical terminologies. When medical cause is found it is easy to put them under such

terms. But if a person has a language delay who does not have any medical explanation, then clinicians try to transfer their burden to speech language pathologist without any explanation. But if these disorders are classified under linguistic levels, such as phonetic, phonological, grammatical, semantic, pragmatic, etc. then it is better to list out the deviant linguistic features and understand their problems and then to go for appropriate intervention strategies.

Regarding intervention, the linguist's role is to help the clinician in planning the linguistic interventions if needed and to monitor the outcome of intervention over a period of time. It is the role a linguist to investigate the language behavior of the intervention provider, teaching materials used, and the environment of intervention provided, as it also can modify the out come.

So, when a patient comes to a clinic with a complaint of speech or language disorder, usual thing that happen is finding the medical/clinical cause. Usually physicians whether the cause is found or not, will divert the patients to speech-clinicians for further assessment and remediation. Here Speech-clinician/speech-language pathologist can only identify the language problem, but may not know whether psychological and sociological background persists or not for that problem. Without this knowledge the intervention provided may lower down the problem but will not eradicate it. So speech-language pathologist has to coordinately work with a psychologist and linguist for the assessment and to provide Remedial measures. If the problem is found in a school going kid then the intervention provider may be an educationist who must also be coordinated in the above said team. This holistic approach should be followed in this discipline. Otherwise the problem may be uncovered. So, as Clinical linguistics includes people from multidiscipline, it can be represented as multidisciplinary study.

COMMUNICATIVE DIFFICULTIES

Development of communication:

Communication:

The word 'communication' is used to talk about how people share information (including their thoughts and feelings). Often when people think about communication, they think about talking and listening. However, people also send information by:

- the tone of their voice
- the look on their face (facial expression)
- the way they use their hands (gestures)
- the way they move and hold their body (body language).

When children are very young, parents understand their needs from the way they behave. If babies cry, they could be in pain, hungry, thirsty, lonely, frightened or in need of a nappy change. As they get a little older, children learn to express their needs through facial expressions, gestures (such as nodding and pointing) and sounds. Between 12-18 months of age, they begin to use words. From a very early age, children also learn to understand other people. They learn that words, voice tone, facial expressions and gestures are all part of the messages other people give them.

Learning to understand and talk occurs gradually. Most children have learned basic talking and understanding skills by the ages of 3 to 3½ years. By the time they start school (around age 5 years), their speech will also have more formal structure, including full sentences and descriptive language. Opportunities to practice talking and listening with adults and other children help children to develop their communication skills.

Communicative Difficulties:

Some children do not develop speech as easily as others due to physiological/psychological/genetic complications. Sometimes, they do not find it easy to understand the meaning of words or gestures. These problems are examples of communication difficulties. When a child has a communication difficulty, it may be necessary to get help from teachers, speech pathologists and other professionals.

Communication problems may affect a child's ability to **speak** (speech disorders/impairments) and/or the ability to **understand** and **use** spoken language (language disorders/impairments). These are the few symptoms to identify communication difficulties. Professionals talk about these as **expressive** and **receptive communication difficulties**.

Symptoms

Children have difficulty with:

- speech sounds (saying the words clearly or correctly)
- speaking fluently (without hesitating too much or stuttering)
- using words and grammar (rules about word order and usage of appropriate)
- putting words together to let others know what they think or want
- understanding what others say.

Expressive communication difficulties

Children with an **expressive** communication or language problem have trouble giving (or expressing) information to other people in a way that other people can easily understand. They may have trouble learning and remembering:

- words, such as the names of things
- the rules of grammar and how to connect words to make sentences

· how to use language to meet their needs (such as to ask questions, describe events, give instructions or tell a story).

Receptive Communication Difficulties

A **receptive** communication problem means that children have trouble understanding (or receiving) the meaning of information being given to them. There may be difficulty with understanding:

· individual words, including words that describe things (such as big/little, all, different)

· phrases

· information when it is put into sentences

· questions

· longer instructions

· descriptions.

Receptive communication problems can also be due to hearing problems. Children with communication problems need a hearing test.

They may also gain understanding from watching other people and from the use of routines. There are many **types of communication difficulties** and vary in degree.

Communication problems can be mild, moderate or severe. Sometimes, a communication difficulty will happen as part of a syndrome or disability.

Types of Communication Difficulties

There are many **types of communication difficulties**. These include:

- speech delay/disorder/impairment

- language delay/disorder/impairment
- expressive language disorder
- receptive language disorder
- stuttering or dysfluency
- verbal dyspraxia (difficulty in making muscle movements that are needed for speaking clearly and quickly)
- semantic/pragmatic disorder (this affects a child's use of language for social purposes)
- Central Auditory Processing (CAP) disorder (this affects a child's listening and understanding of language)
- dyslexia.

Communication processes and disorders

Communication processes	**Communication disorders**
Communicative intentions	Problems formulating and establishing communicative intentions. Associated with psychotic disorders (e.g.schizophrenia), mental retardation (e.g. Down's syndrome), autism and dementia (e.g. Alzheimer's disease)
Language encoding and decoding Problems	formulating and understanding various levels of language. Disorders include acquired aphasia and phonological disorder, specific language impairment and pragmatic language impairment in children. Also includes a rare disorder in children, Landau-Kleffner syndrome
Motor programming	Apraxia (developmental and acquired)
Motor execution	Disorders of speech production. Includes dysarthria

	(developmental and acquired), disorders related to articulation (e.g. cleft lip and palate), phonation (e.g. vocal nodules), resonation (e.g. velopharyngeal incompetence) and fluency (e.g. stuttering). Also includes related disorders of swallowing (dysphagia) and drooling
Sensory processing	Hearing disorders (e.g. conductive and sensorineural deafness) and oral sensory dysfunction
Perception	Agnosia. Affects different sensory modalities, resulting in auditory agnosia, visual agnosia, etc.

CAUSES FOR COMMUNICATION DISORDERS

There are many potential causes of communication impairment;

- the most common is mental retardation.

Other causes include:

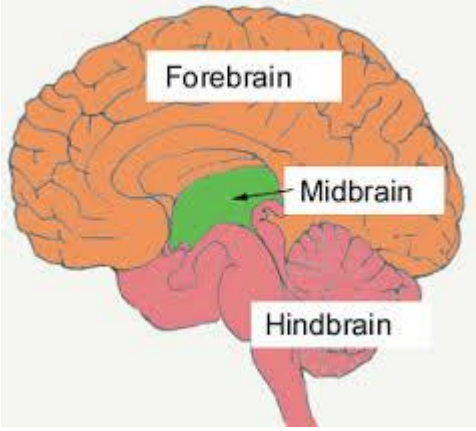
- Hearing impairment
- ADD
- Learning disabilities
- Autism
- Schizophrenia
- Cerebral palsy
- Physical disability: cleft lip and palate, or malformations of the mouth or nose
- Significant behavior or emotional problems, including deficits in social skills
- Vocal cord injury
- Cri-du-chat syndrome
- Gilles de la Tourette syndrome
- Ill-fitting dentures

- Alcohol intoxication
- Neurological disorders and diseases
- Head trauma
- Alzheimer's disease
- Stroke
- Transient ischemic attack (TIA)

Introduction of the Anatomy of the brain

The anatomy of the brain is complex due its intricate structure and function. This amazing organ acts as a control center by receiving, interpreting, and directing sensory information throughout the body. There are three major divisions of the brain. They are the forebrain, the midbrain, and the hindbrain.

Anatomy of the Brain: Brain Divisions

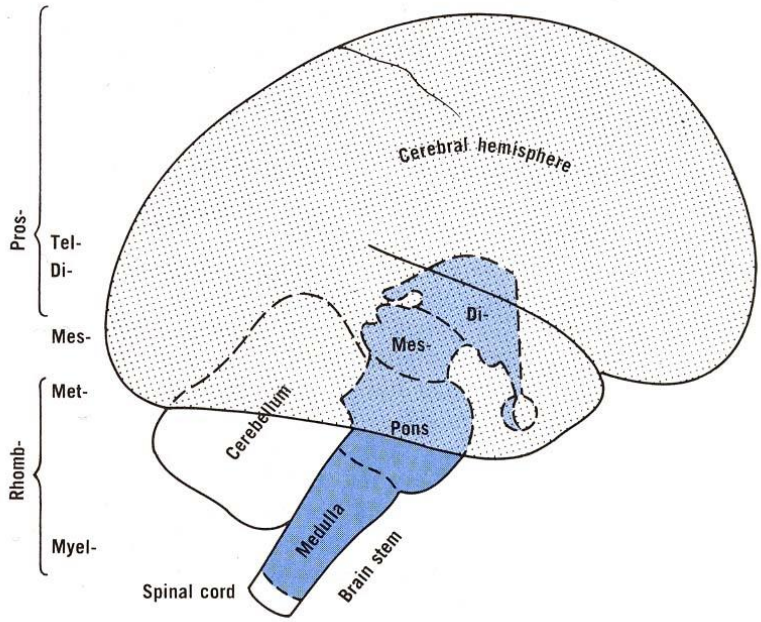


The **forebrain** is responsible for a variety of functions including receiving and processing sensory information, thinking, perceiving, producing and understanding language, and controlling motor function. There are two major divisions of forebrain: the diencephalon and the telencephalon. The diencephalon contains structures such as the thalamus and hypothalamus

which are responsible for such functions as motor control, relaying sensory information, and controlling autonomic functions. The telencephalon contains the largest part of the brain, the cerebrum. Most of the actual information processing in the brain takes place in the cerebral cortex.

The **midbrain** and the hindbrain together make up the brainstem. The midbrain is the portion of the brainstem that connects the hindbrain and the forebrain. This region of the brain is involved in auditory and visual responses as well as motor function.

The **hindbrain** extends from the spinal cord and is composed of the metencephalon and myelencephalon. The metencephalon contains structures such as the pons and cerebellum. These regions assists in maintaining balance and equilibrium, movement coordination, and the conduction of sensory information. The myelencephalon is composed of the medulla oblongata which is responsible for controlling such autonomic functions as breathing, heart rate, and digestion.

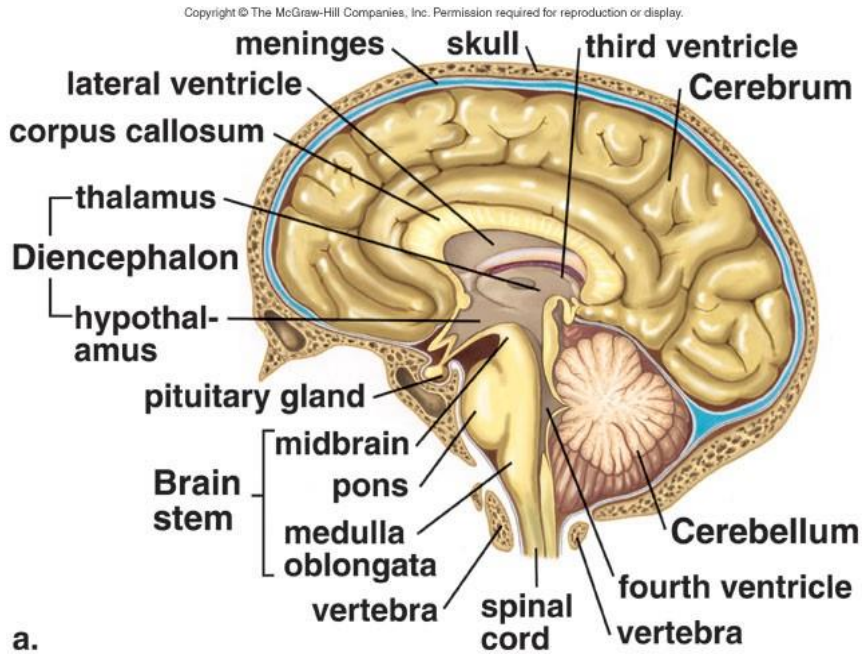


- Prosencephalon - Forebrain
  - Diencephalon
  - Telencephalon

  Mesencephalon - Midbrain

  Rhombencephalon – Hindbrain
  - Metencephalon
  - Myelencephalon

- **Anatomy of the Brain: Structures**



The brain contains various structures that have a multitude of functions. Below is a list of major structures of the brain and some of their functions.

Basal Ganglia

- Involved in cognition and voluntary movement
- Diseases related to damages of this area are Parkinson's and Huntington's

Brainstem

- Relays information between the peripheral nerves and spinal cord to the upper parts of the brain
- Consists of the midbrain, medulla oblongata, and the pons

Broca's Area

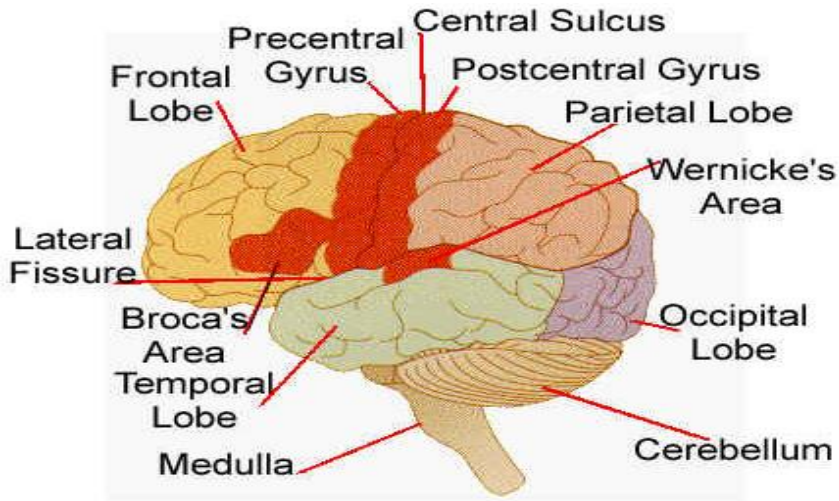
- Speech production
- Understanding language

Central Sulcus (Fissure of Rolando)

- Deep grove that separates the parietal and frontal lobes

Cerebral Cortex Lobes

- Frontal Lobes -involved with decision-making, problem solving, and planning
- Occipital Lobes-involved with vision and color recognition
- Parietal Lobes - receives and processes sensory information
- Temporal Lobes - involved with emotional responses, memory, and speech



Cerebellum

- Controls movement coordination
- Maintains balance and equilibrium

Cerebral Cortex

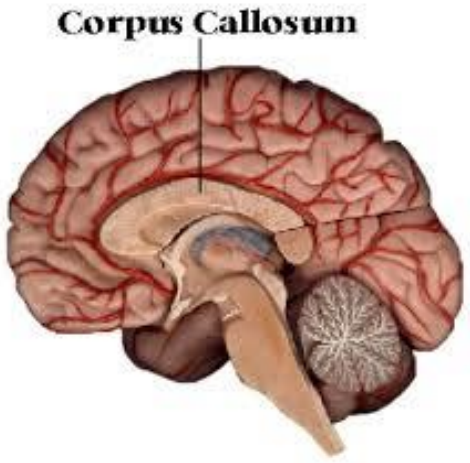
- Outer portion (1.5mm to 5mm) of the cerebrum
- Receives and processes sensory information
- Divided into cerebral cortex lobes

Cerebrum

- Largest portion of the brain
- Consists of folded bulges called gyri that create deep furrows

Corpus Callosum

- Thick band of fibers that connects the left and right brain hemispheres



The corpus callosum is a thick band of nerve fibers that divides the cerebrum into left and right hemispheres. It connects the left and right sides of the brain allowing for communication between both hemispheres. The

corpus callosum transfers motor, sensory, and cognitive information between the brain hemispheres.

Function:

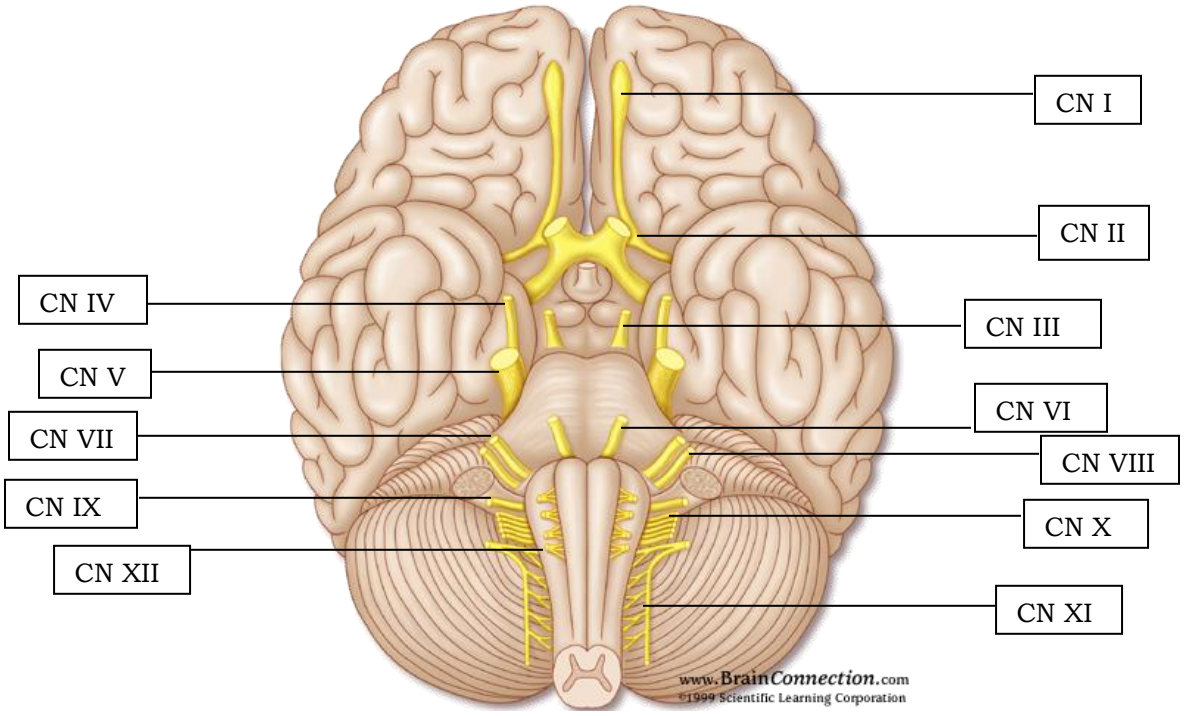
The corpus callosum is involved in several functions of the body including:

- Communication Between Brain Hemispheres
- Eye Movement
- Maintaining the Balance of Arousal and Attention
- Tactile Localization

Cranial Nerves

- Twelve pairs of nerves that originate in the brain, exit the skull, and lead to the head, neck and torso

There are 12 cranial nerves. The most important point for the study of communication is the cranial nerves. Their role is to link the brain with the head and neck.They are;



CN I - Olfactory nerve (sensory)

Functionof sensory component –smell (from nose to brain)

Functionofmotor component – no motor nerve

CN II – Optic nerve (sensory)

Function of sensory component – vision (from eye to brain)

Functionofmotor component –no motor nerve

CN III - Oculomotor nerve (motor)

Function of sensory component - Sensations from eye muscles

Functionofmotor component – Eye movements, pupil constriction

CN IV - Trochlear nerve (motor)

Function of sensory component - Sensations from eye muscles

Functionofmotor component - Eye movements

CN V - Trigeminalnerve (mixed, (i.e) sensory & motor)

Function of sensory component - Sensations from skin of face,nose andmouth

Functionofmotor component – Chewing, swallowing

CN VI - Abducens nerve (motor)

Function of sensory component - Sensations from eye muscles.

Functionofmotor component - Eye movements

CN VII - Facial nerve (mixed, (i.e) sensory & motor)

Function of sensory component – Taste from the anterior two-thirds of the tongue, visceral sensations from the head

Functionofmotor component – Facial expressions, crying, salivation, and dilation of blood vessels in the head

CN VIII - Acoustic, Auditory, or Vestibulocochlear nerve (sensory)

Function of sensory component – Hearing, equilibrium

Functionofmotor component – no motor nerve

CN IX - Glossopharyngeal nerve (mixed, (i.e) sensory & motor)

Function of sensory component – Taste and other sensations from throat and posterior third of tongue

Functionofmotor component – Swallowing, salivation, dilation of blood vessels

CN X - Vagus nerve (mixed, (i.e) sensory & motor)

Function of sensory component – Taste and sensations from neck, throrax, and abdomen

Functionofmotor component – Swallowing, control of larynx, parasympathetic nerves to heart and viscera

CN XI - Spinal nerve (motor)

Function of sensory component –no sensory nerve

Functionofmotor component – Movements of shoulders and head; parasympathetic nerves to viscera

CN XII - Hypoglossal nerve(motor)

Function of sensory component – Sensations from tongue muscles

Functionofmotor component – Movement of tongue

Fissure of Sylvius (Lateral Sulcus)

- Deep grove that separates the parietal and temporal lobes

Limbic System Structures

- Amygdala - involved in emotional responses, hormonal secretions, and memory
- Cingulate Gyrus - a fold in the brain involved with sensory input concerning emotions and the regulation of aggressive behavior
- Hippocampus - sends memories out to the appropriate part of the cerebral hemisphere for long-term storage and retrieves them when necessary
- Hypothalamus - directs a multitude of important functions such as body temperature, hunger, and homeostasis
- Olfactory Cortex - receives sensory information from the olfactory bulb and is involved in the identification of odors
- Thalamus - mass of grey matter cells that relay sensory signals to and from the spinal cord and the cerebrum

Medulla Oblongata

- Lower part of the brainstem that helps to control autonomic functions

Meninges

- Membranes that cover and protect the brain and spinal cord

Pituitary Gland

- Endocrine gland involved in homeostasis
- Regulates other endocrine glands

Pons

- Relays sensory information between the cerebrum and cerebellum

CEREBRAL CORTEX

The cerebral cortex is the layer of the brain often referred to as gray matter. The cortex (thin layer of tissue) is gray because nerves in this area lack the insulation that makes most other parts of the brain appear to be white. The cortex covers the outer portion (1.5mm to 5mm) of the cerebrum and cerebellum. The portion of the cortex that covers the cerebrum is called the cerebral cortex.

The cerebral cortex consists of folded bulges called gyri that create deep furrows or fissures called sulci. The folds in the brain add to its surface area and therefore increase the amount of gray matter and the quantity of information that can be processed.

The cerebral cortex is divided into right and left hemispheres. It encompasses about two-thirds of the brain mass and lies over and around most of the structures of the brain. It is the most highly developed part of the human brain and is responsible for thinking, perceiving, producing and understanding language. It is also the most recent structure in the history of brain evolution.

Most of the actual information processing in the brain takes place in the cerebral cortex. The cerebral cortex is divided into lobes that each have a specific function. For example, there are specific areas involved in vision, hearing, touch, movement, and smell. Other areas are critical for thinking and

reasoning. Although many functions, such as touch, are found in both the right and left cerebral hemispheres, some functions are found in only one cerebral hemisphere. For example, in most people, language abilities are found in the left hemisphere.

Cerebral Cortex Lobes

Parietal Lobe - involved in the reception and processing of sensory information from the body.

Frontal Lobe - involved with decision-making, problem solving, and planning.

Occipital Lobe - involved with vision.

Temporal Lobe - involved with memory, emotion, hearing, and language.

HEMISPHERES

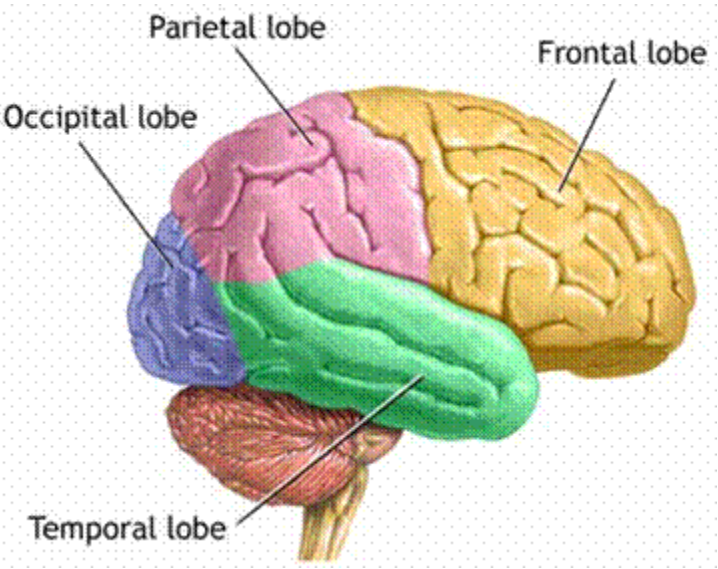
The brain is divided into left and right hemispheres. Each hemisphere controls its own unique set of activities or tasks. The right side of the brain tends to be more dominant in creative activities, while the left side of the brain tends to be more dominant in logical or analytical activities. These hemispheres communicate with each other through a large bundle of nerve fibers called the corpus callosum, and through several smaller nerve pathways.

The right side of the brain is more visual oriented, involved in activities such as visual imagery and face recognition. The right side of the brain tends to view information as a whole, rather than as individual details. It also tends to process information more intuitively or randomly. The right side of the brain is involved in spacial abilities, such as judging the position of things in space, and knowing your body position.

The left side of the brain processes information more logically or sequentially. The left side of the brain is dominant in understanding and using language, including listening, reading, speaking and writing. It is involved in the memory for spoken and written messages, and plays a major role in the analysis of information.

The right side of the brain controls muscles on the left side of the body. It also receives sensory information from the left side of the body. The left side of the brain controls muscles on the right side of the body, and receives sensory information from the right side of the body.

LOBES OF THE BRAIN



FRONTAL LOBE

Front part of the brain; involved in planning, organizing, problem solving, selective attention, personality and a variety of "higher cognitive functions" including behavior and emotions.

- The frontal lobe (which contains the motor cortex and the prefrontal cortex) extends from the central sulcus to the anterior limit of

the brain. The posterior portionof the frontal lobe is the **precentralgyrus**, which is specialized for the control of the fine movements, such as moving one finger at the time. It has separated areas responsible for different parts of the body, mostly on the contralateral side of the body, but with slight control of the ipsilateral side, too.

- The anterior (front) portion of the frontal lobe is called the prefrontal cortex, is a fairely large structure, especially in species with the large brain overall, such as humans. It is not primary target for any single sensory system, but it receives information from all the sensory systems, including sensations from the interior of the body. This is only the cortical area known to receive input from all sensory modalities. It is very important for the "higher cognitive functions" and the determination of the personality.
- The posterior (back) of the frontal lobe consists of the pre-motor and motor areas. Nerve cells that produce movement are located in the motor areas. The pre-motor areas serve to modify movements.
- The frontal lobe is divided from the parietal lobe by the central sulcus.

PARIETAL LOBE

- Located between the occipital lobe the central sulcus, one of the deepest grooves in the surface of the cortex.
- Specialized primarily for dealing with body information, including touch, muscle-stretch receptors, and joint receptors. It is concerned with perception of stimuli related to touch, pressure, temperature and pain.
- One of the two parietal lobes of the brain located behind the frontal lobe at the top of the brain. The area just posterior to the central sulcus, called the postcentralgyrus or the primary somatosensory cortex is the primary target for touch sensations and other skin and muscle information.

- Direct electrical stimulation of the postcentral gyrus evokes sensations on the opposite side of the body, often described as tingling or unnatural sensations.
- The postcentral gyrus includes four bands of cells running parallel to the central sulcus. Along each band are separate areas that receive information from different parts of the body. Two of the bands receive mostly light-touch information, one receives deep-Pressure information, and one receives a combination of both. That is,thepostcentralgyrus contains four separate representations of the body.
- Damage to this lobe results in partial lose of the sense of touch, or the muscle and joint senses. They suffer a variety of symptoms that suggest difficulty in interpreting such information and in using it to control movement. The people with such damage may neglect the opposite side of the body, especially neglect of the left side after right-hemisphere parietal lobe damage. The People with such damage may fail to dress the left side of body, read only the right side of the page, and describe from memory only the right side of a familiar scene. Inability to draw and follow maps, describe how to get somewhere, or say what something might look like when viewed from a different angle.
- Parietal Lobe, Right - Damage to this area can cause visuo-spatial deficits (e.g., the patient may have difficulty finding their way around new, or even familiar, places).
- Parietal Lobe, Left - Damage to this area may disrupt a patient's ability to understand spoken and/or written language.
- The parietal lobes contain the primary sensory cortex which controls sensation (touch, pressure).
- Behind the primary sensory cortex is a large association area that controls fine sensation (judgment of texture, weight, size, shape).

TEMPORAL LOBE

- There are two temporal lobes, one on each side of the brain that is located laterally in each hemisphere near the temples; at about the level of the ears.
- It is it is the primary cortical target for auditory information. In humans the temporal lobe-especially the left temporal lobe in most cases-is essential for understanding spoken language.
- The temporal lobe also, contributes to some of the more complex aspects of vision, including perception of complex patterns such as faces. A tumor in the temporallobe may give rise to elaborate visual hallucinations, whereas a tumor in the occipital lobe ordinarily evokes only simple sensations, such as flashes of light. In humans, the left temporal lobe is also important for the comprehension of language.
- These lobes allow a person to tell one smell from another and one sound from another. They also help in sorting new information and are believed to be responsible for short-term memory.
- Right Lobe - Mainly involved in visual memory (i.e., memory for pictures and faces).
- Left Lobe - Mainly involved in verbal memory (i.e., memory for words and names).
- Located below the lateral fissure.
- Concerned with perception and recognition of auditory stimuli (hearing) and memory (hippocampus).
- The temporal lobes also play a part in emotional and motivational behaviors. Temporal lobe damage can lead to a set of behaviors known as the **KlüverBucy syndrome.** Monkeys with damaged temporal lobes fail to display normal fears and anxieties.The

OCCIPITAL LOBE

- Located at the back of the brain, behind the parietal lobe and temporal lobe which processes visual information. That is, located at the posterior end of the cortex which is the main target for axons from the thalamic nuclei that receive input from the visual pathways.
- The very posterior pole of the occipital lobe is known as the *primary visual cortex* or as the *striate cortex* because of its striped appearance in cross section.
- Not only is the occipital lobe mainly responsible for visual reception, it also contains association areas that help in the visual recognition of shapes and colors.
- Concerned with many aspects of vision.
- Damage to this lobe can cause visual deficits. Destruction of any part of *striate cortex* causes loss of vision in part of the visual field. The location of the damagedetermines which part of the visual field will become blind.
- Extensive damage to the *striate cortex* of the right hemisphere causes blindness in the left visual field. Blindness from occipital lobe damage is called cortical blindness. A person with cortical blindness has normal eyes, normal pupillary reflexes, and some eye movements, but no pattern perception and no awareness of visual information.

PHYSIOLOGY OF SPEECH PROCESS

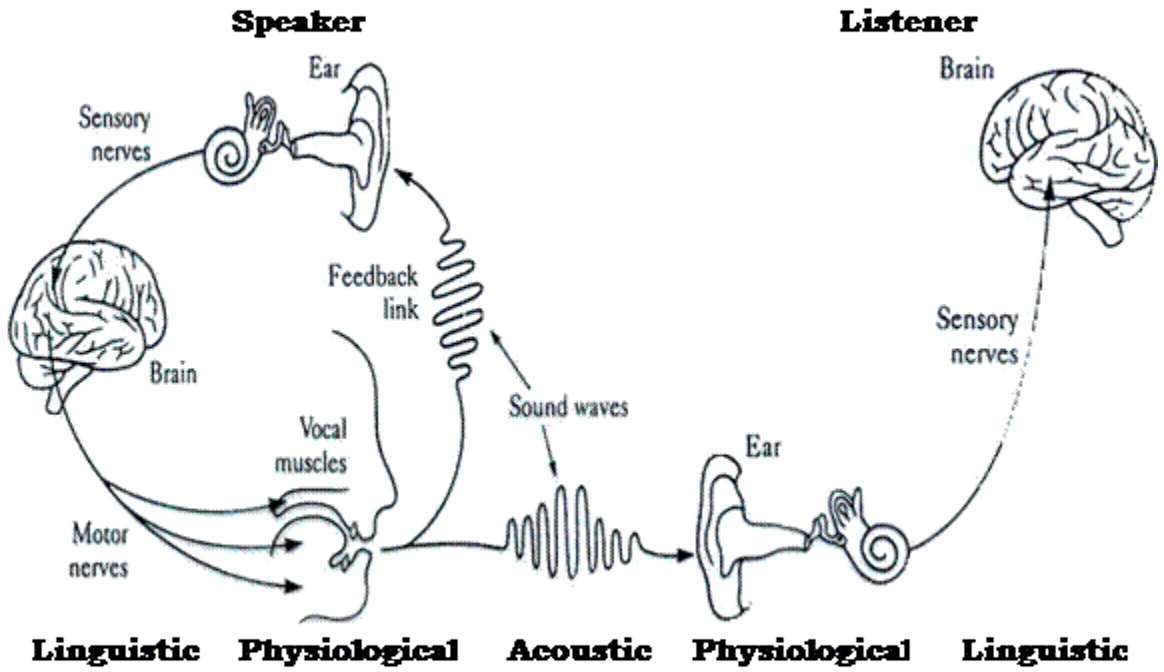
The information process is in terms of the sequence, a chain of events - a chain which can be used as a basis for analyzing what happens in any communicative activity. Basically seven steps are involved.

First, there must be an **information** source. This source has internal properties which enable it to construct a signal, or message; this process of construction is referred to as **encoding** and this constitutes the second step in the chain. The third step is **production**; the encoded signal is made public accessible to direct observation. This signal is then sent along a medium or

channel and this constitutes the fourth step; **transmission**. In the fifth step the signal is **received** by some other device. The internal property of the device which receives the signal enables it to be **decoded**, and this constitutes sixth step in the chain. The remaining step is the decoded signal arriving at its destination, where the significance of the message will be registered in some way. We can summarize this process in a single line as follows:

Information source **Encoding** **Production** **Transmission** **Reception** **Decoding** **Destination**

The Speech Chain



Human Communication: Processes

Before we can utter a single word, we have to be capable of forming thoughts that are appropriate for communication. The qualification 'appropriate' is important in this context, as we all entertain thoughts that violate social and moral codes and are rarely, if ever, communicated. Moreover, many other thoughts are below the level of our conscious awareness and, as such, are not communicated. In short, we each entertain many more thoughts than are ever

actually communicated. Those thoughts that are to be communicated give rise to a communicative intention. Pragmatists argue that it is only when a hearer has established a speaker's communicative intention in speaking that a speaker can be said to have communicated anything at all.5 In order that the hearer may identify the speaker's intention, the speaker must first encode it using a conventional symbol system (one that is recognised and understood by other communicators) that can be readily transmitted. Language is such a symbol system.

Language encoding is a complex process that involves many interrelated stages. These stages involve a combination of lexical, semantic, syntactic and phonological processes. Their combined effect is to transform an abstract, nonlinguistic intention into a still abstract, but now linguistic representation. This representation is still not of a form where it can be uttered by a speaker. Various neuromuscular selections must be made during motor programming, before the speaker is finally able to translate these selections into movements of the articulators during speech production (a stage called motor execution).

Thus far, we have outlined the four main processes of communication that are essential to the production (expression) of an utterance.

To recap, these processes are: (1) thought genesis, (2) language encoding, (3) motor programming and (4) motor execution. However, even if a communicator is able to fulfil the requirements of these processes, he or she still has not communicated anything at all. As we discussed above, communication can only be said to occur when a hearer is able to retrieve the intention that motivated the speaker's utterance. Our four productive processes must now be matched to four receptive processes, the combined function of which will be to determine this intention on the basis of an input linguistic utterance. In the first of these receptive processes – sensory processing – sound waves are converted into mechanical vibrations via the actions of the tympanic membrane (ear drum) and ossicles. These vibrations trigger a series of neurochemical reactions within the cochlea of the inner ear. From here, nerve impulses make their way along auditory nerves to the auditory cortices of the brain. These cortices, both of which are located in the temporal lobes, are integral to our

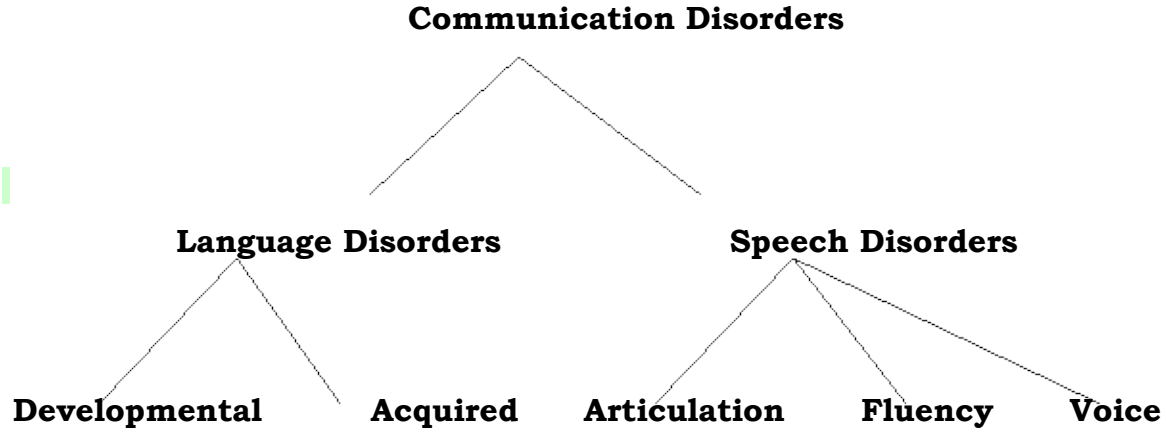
second main receptive process, speech perception. Although the exact mechanism by means of which hearers perceive speech sounds is still uncertain, it seems clear that top-down processes and contextual influences play a significant role (Massaro 2001). Speech perception is vital to the eventual recovery of a speaker's communicative intention. However, it is by no means the only form of perception that plays a role in this process. One need only consider how often visual information serves to disambiguate a speaker's utterance to appreciate the significance of visual perception in this process too. Before complete disambiguation can occur, the product of perception must undergo a third receptive process, language decoding. In decoding, structural (syntactic) relations within sentences are determined alongside the semantic features of constituent lexemes. Decoding arrives at a propositional meaning of the sentence which is not yet the full intended meaning of the speaker's utterance. This latter meaning can only be obtained by establishing the speaker's communicative intention in producing the utterance, a process that leads us back to the domain of thoughts. It can be seen that in an effort to describe communication between a speaker and a hearer, we have effectively come full circle, a fact that is aptly demonstrated by Figure above.

UNIT – II

COMMUNICATION DISORDERS

Communication disorders can be classified into speech and language disorders. According to etiology of each disorder it is classified into

1. Language Disorders
2. Speech Disorders



Clinical Linguistics deals with all types of speech and language disorders. According to etiology of each disorder it is classified into language and speech disorders.

Language Disorders

Language disorder is a disorder that is found in the development or use of the knowledge of language. It shows the breakdown in the development of language abilities on the usual developmental schedule.

Language disorders can be further classified into **Developmental** and **Acquired** disorders. Developmental language disorders are the disorders that show the inheritance that is, that persists during birth time itself. Language disorders exist because of the hindrance in the language development due to several factors such as brain damage or neurological dysfunction caused during prenatal or natal or post natal period.

Acquired disorders are the disorders which are not inherited, but may have lost the ability to use language due to environmental factors such as trauma after birth, some accident or trauma after birth, some accident or trauma on the head, epilepsy etc.

The disorders that come under language disorders are,

1. Autism, 2.Learning Disability, 3.Mental Retardation, 4.Specific Language Impairment, 5. Aphasia, 6.Schizophrenia, 7.Dyspraxia, 8. Dysphagia etc.

Speech Disorders

Speech disorders are the disorders, in which the speech mechanisms like soft palate, tongue, lips, etc are the locus of delay. They can be further classified into;

Articulation disorders:

It is a disorder due to the problem that occurs in movement of various structures of speech mechanism such as soft palate, tongue, lips, etc. This can be classified into 1. Functional – which have no specific cause, 2. Organic – which occurs due to structural abnormalities like **cleft palate** and **cerebral palsy**, and 3.others – which occurs due to any other speech and language disorders such as hearing loss, mental retardation, etc.

e.g. Dysarthria, cleft palate and cerebral palsy

***Fluency disorders*:**

Communication needs smooth and easy flow of the utterances. Thus the effortless and continuous speech with the rapids speed is called fluency. So if problem persists in the above said effort, continuity and speed then it is said to be a fluency disorders. For example, **stuttering** and **cluttering**.

Voice disorders:

If the pitch, loudness or quality of the voice differs from that of the normal / standard voice due to abnormalities in the vocal mechanisms is said to be a disordered voice. The two types of voice disorders are phonation and resonance.

e.g. dysphonia or those caused by cleft lip or palate

These language and speech disorders can exist together or by themselves.

Learning disability (LD)

Children with special learning disabilities exhibit a disorder in one or more of the basic psychological process involved in understanding or in using their ability for speaking and writing. These may be manifested in disorders of listening, thinking, talking, reading, writing, spelling, or arithmetic.

Wecanalsosaythat

1. The LD child shows a discrepancy between achievement and intelligence.

2. Handicaps such as mental retardation, visual and hearing impairment and emotional behavioural disorders must be ruled out.
3. A LD is psychological processing disorder and presumes a central nervous dysfunction.
   70% of LDs are right brain dominants.

Types of LD:

Dyslexia:

Dyslexia is the presence of a significant discrepancy between intellectual ability and reading and/or writing performance.

Different types of dyslexia:

There are two terms sometimes used to describe typical symptoms of dyslexia - 'dysphonetic' and 'dyseidetic'. Someone who is dysphonetic finds it hard to connect sounds to symbols. They would make spelling mistakes regularly and have many difficulties in sounding out words. It is also sometimes referred to as auditory dyslexia since it is related to the way in which the child processes sound.

Someone who is dyseidetic has difficulties with word recognition and spelling. It is also referred to as surface dyslexia or visual dyslexia since it is related to the way in which the child processes visual information

Dyscalculia: Dyscalculia is a specific learning disability in mathematics; in particular, a difficulty in performing arithmetic operations.

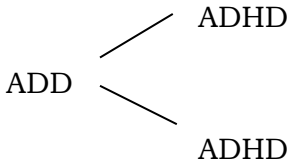
Dysgraphia: Dysgraphia is essentially a difficulty in handwriting.

There are three main types of dysgraphia and they are as follows:

Dyslexic dysgraphia: Illegible written text, poor oral spelling, normal drawing and copying of original text, normal finger tapping speed (a measure of fine-motor speed).

Motor dysgraphia: Illegible written and copied text, normal oral spelling abilities, difficulties in drawing and abnormal finger tapping speed.

Spatial dysgraphia: Illegible writing, whether spontaneously produced or copied, normal oral spelling and fingers tapping speed, but great difficulties drawing.



Some children have more trouble paying attention in class and completing academic assignments than others. It is estimated that from 3 to 10 percent of the population has a condition known as Attention Deficit Disorder (ADD)

The degree of dyslexia ranges from mild to severe.

Difficult to find pure dyslexic.

Dyslexics differ with their problems.

Cause for learningdisability

1. Hereditary
2. Birth trauma
3. Post - natal problem
4. Pre - natal problem
5. If motheris an alcoholic

Dyslexia

Dyslexia is a complex reading, writing and learning troubles in which letters and numbers are reversed or even seen and written upside down. Children are affected more and more with Dyslexia which in turn extends in their later life and we have Dyslexic adults also. It may contribute to functional illiteracy in an estimated 25 % of Indians. It also causes low self-esteem and frustration on a massive scale! This article aims to show

that dyslexia is *not* due to brain damage or stupidity. Many dyslexics are far brighter than average. Treating them as though they are 'dumb' or 'slow' is absolutely incorrect.

Actually, dyslexics suffer not so much from a learning disability, but from a teaching disability: few teachers know how to teach these children.

Symptoms

Indications of dyslexia usually begin to show up around age five or six. Symptoms many include:

- Shapes or sequences of letters or numbers appear changed or reversed.
- Spelling is incorrect or inconsistent.
- Words or lines are skipped when reading or writing.
- Letters and numbers appear to move, disappear, grow or shrink.
- Punctuation marks or capital letters are omitted, ignored or not seen.
- Words and letters are omitted, altered or substituted while reading or writing.
- Some speech sounds are difficult to make or mispronounced.
- False sounds are perceived.
- The person appears to not listen or hear what is said.
- One can experience dizziness or nausea while reading.
- There is a poor sense of direction.
- Inability to sit still.
- Problems with balance and coordination.
- Hyperactivity or hypoactivity can occur due to frustration.
- Excessive daydreaming, and trouble being on time.

Most people think that **dyslexia** causes a person to see words or sentences backwards, or that it causes a person to confuse the letter 'b' with the letter 'd.' This is just one form of dyslexia, known as **strephosymbolia**. Dyslexia, also known as **developmental reading disorder**, affects a person's ability to comprehend either oral or written language or sometimes both. In other words, it is a general, language-related learning disorder. Tasks and activities many of us take for granted, such as writing out a grocery list, reading the newspaper or listening to a book on tape, could be problematic for someone with dyslexia.

While reading the following deviations in language production are found:

1. Addition, 2. Deletion, 3.Substitution, 4.Repetetion, 5.Reversal.

For example
1.Addtion:-
Vowel :- In Initial position

1. avalai> a:valai
2. taram > ta:ram
3.palakkam > pa:lakkam
4.natakka > na:takka

In Medial position: No addition is found

In Final position

1.a:nvar > a:nva:r
2.ku:ruvar > ku:ruva:r

Consonant:- Doubling

1.veku > vekku
2.palakum > palakkum

Mute consonant > Mute consonant +cv
$c_1 > c_1 + c_1 u$

2.Deletion:-

Vowel:- In initial position

1.pa:rtta:rkal > parttarkal (also medial)
2.a:kaya:l > akaya:l
3.va:ykka:l > vaykka:l

In final position

1.valankuva:r > valankuvar

Consonant:- In medial position

1.ko:likkunca:lum > ko:likuncum

2.arivutamaya:l > arivutama:l

3.Subtitutions:-

Words:-

When story is known or if able comprehend to the last part of the sentence, with out reading last word / last part, substitutes with synonyms.

1.vi:cina:l > vi:cuva:rkal
2.tappittukollala:m > tappikkala:m
3.tappittatu > pilaittatu
4.virittanar > vi:cinar
5.perumaipe:ciyatu > perumaiatittatu

Also words from spoken language are substituted.

Vowel substitution:-

Often o, o:, e, e: are confused when a combined with consonants and occurs as syllables. This is because of the grapheme problem of Tamil, which is to be properly taught to children.

Eg,

1.perumai > porumai
2.therivikka > thorivikka
3.ke:TTa:r > ko:TTa:r
4.e:te:num > e:tenum

Consonant substitution:-

R > r

1.paRRi > parri
2.na:yirrukilamai > na:yirukilamai

4.Repetitions:-

For every sentence, atleast one word is repeated once or twice mostly, they are case markers

Eg: pa:tiliruntu. Here iruntu is reapted.

5.Reversals:-

Vowel:-

1.ku:riyava:ru > ku:riya:varu

Consonant

1.kavilntum > kalvintum
2.amarntanar > armantanar
3.irunta:r > inruta:r

Dysgraphia

Dysgraphia is a learning disability resulting from the difficulty in expressing thoughts in writing and graphing. It generally refers to extremely poor handwriting. It is a deficiency in the ability to write, primarily in terms of handwriting, but perhaps also in terms of coherence.People with dysgraphia usually can write on some level, and often lack other fine motor skills and may be cross dominant, finding tasks such as tying shoes difficult. It often does not affect all fine motor skills. They can also lack basic grammar and spelling skills (for example, having difficulties with the letters p, q, b, and d), and often will write the wrong word when trying to formulate thoughts (on paper). In childhood, the disorder generally emerges when the child is first introduced to writing. The child may make inappropriately sized and spaced letters, or write wrong or misspelled words despite thorough instruction. Children with the disorder may have other learning disabilities, but they usually have no social or other academic problems.

Types of dysgraphia

Three principal subtypes of dysgraphia are recognized. Some children may have a combination of two or all three of these, and individual symptoms may vary in presentation from what is described here.

1. **Dyslexic dysgraphia**

With dyslexic dysgraphia, spontaneously written work is illegible, copied work is fairly good, and spelling is bad. Finger tapping speed (a method for identifying fine motor problems) is normal, indicating the deficit does not likely stem from cerebellar damage. A dyslexic dysgraphic does not necessarily have dyslexia. (Dyslexia and dysgraphia appear to be unrelated but are often found together.)

2. **Motor dysgraphia**

Motor dysgraphia is due to deficient fine motor skills, poor dexterity, poor muscle tone, or unspecified motor clumsiness. Motor dysgraphia may be part of the larger

problem of motor apraxia. Generally, written work is poor to illegible, even if copied by sight from another document. Letter formation may be acceptable in very short samples of writing, but this requires extreme effort and an unreasonable amount of time to accomplish, and cannot be sustained for a significant length of time. Writing long passages is extremely painful and cannot be sustained. Letter shape and size becomes increasingly inconsistent and illegible. Writing is often slanted due to holding a pen or pencil incorrectly. Spelling skills are not impaired. Finger tapping speed results are below normal.

3. **Spatial dysgraphia**

A person with dysgraphia due to a defect in the understanding of space has illegible spontaneously written work, illegible copied work, but normal spelling and normal tapping speed.

Symptoms

A mixture of upper/lower case letters, irregular letter sizes and shapes, unfinished letters, struggle to use writing as a communications tool, odd writing grip, many spelling mistakes (sometimes), pain when writing, decreased or increased speed of writing and copying, talks to self while writing, muscle spasms in the arm and shoulder (sometimes in the rest of the body), inability to flex (sometimes move) the arm (creating an L-like shape), and general illegibility.

Many people who are dysgraphic experience pain while writing. The pain usually starts in the center of the forearm and then spreads along the nervous system to the entire body. This pain can get worse or even appear when a dysgraphic is stressed. Few people who do not have dysgraphia know about this, because many with dysgraphia will not mention it to anyone. There are a few reasons why pain while writing is rarely mentioned:

- Sufferers do not know that it is unusual to experience this type of pain with writing.

- If they know that it is different from how others experience writing, they feel that few will believe them.
- Those who do not believe that the pain while writing is real will often not understand it. It will usually be attributed to muscle ache or cramping, and it will often be considered only a minor inconvenience.
- For some people with dysgraphia, they no longer write, and just type everything, so they no longer feel this pain.

Dysgraphics who experience this pain may exhibit reluctance or refusal to complete writing tasks.

Problems of Dyslexics and Dysgraphics:

In Reading and Writing are found in the following,

- Spelling
- Morphology
- Syntax
- Comprehension

The addition, deletion, substitution and reversals are the main problems found in their reading and writing.

Reading

Spelling:

	[deviant--form]	[Correct--form]
Addition:		
Initial:	black -	back
Medial:	oven -	own
Final:	battli -	battle
	boys -	boy
Substitution:		
Initial:	dabl -	trouble
	harer -	However

Medial:	trow	-	through
Deletion:			
Initial:	tips	-	troops
	ha:ndli-		handedly
Medial:	refd	-	refused
	indias	-	Indians
Final:	no	-	known
	luk	-	lucky
Reversal:	ben	-	pen

Morphology:

Addition:			
Initial:	dis	-	un
Final	ing	-	ed
	ed	-	ing
	Negative marker		
Deletion:	negation		
Substitution:	pronouns, lexical items.		

Syntax:

1. Substitution in 'wh' words
2. Reversal of word order.

Writing

Spelling:

Addition:			
Initial:	tricket	-	ticket
Medial:	reaypai	-	repay
	quine	-	queen

	Final	thife -	thief
		theafe	
Deletion:			
	Initial:	rigule -	wriggle
		cinties-	Scientist
		sintest	
	Medial:	usful -	useful
		knoldge-	Knowledge
	Final:	killdele -	killed

Reversal:

	buring -	during
	foriegn -	Foreign
Substitution:		
Initial:	phasin -	faster
Medial:	facine -	fasten
Final:	plased -	placed

Morphology:

Addition:			
	Initial:	dis -	un
	Final	ing -	ed
		ed -	ing
		Negative marker	
Deletion:		negation	
Substitution:		pronouns, lexical items.	

Syntax:

1. Substitution in 'wh' words
2. Reversal of word order.

These problems increase in these children is also due to the complexity of the language. Teachers must have this in their mind while teaching a language. Let us see some of them in the following.

Spelling:

English have,

Silent letters

Blends

Digraphs

Complexities in pronouncing vowel that occur in different environment confusion in 'c' and 'k', and in 'g' and 'j'.

In Tamil the orthographic complexities leads to increase the problem of children.

Morphology:

Problems found in affixes

Syntax:

Word order problem is found

Substitution of lexical items and 'wh' words are found.

Mostly lexical expansion is found.

Comprehension:

Interpreting the meaning of content is problem for these children.

Autism

Autism is one of the most prominent disorders encountered by clinical linguists. It is a complex neurological disorder that involves the functioning of the brain. Autism is classified by the World Health Organization and American Psychological Association as a developmental disability caused due to the disorder of central nervous system. According to the World Health Organization's International Classification of Diseases (ICD-10), Autism is estimated to affect 5 in every 10,000 children and manifests before the age of three years. As per *Diagnostic and Statistical Manual of Mental Disorders* (DSM IV), Autistic children are marked by delays in their "social interaction, language as used in social communication or symbolic or imaginative play".

Public awareness of autism was raised by the film *Rain Man*, in which actor Dustin Hoffman portrayed an autistic man. Autism refers to a spectrum of developmental disorders characterized by social interaction deficits, language/communication impairments, and abnormal, stereotyped behavior patterns. Autistic children do not seek and develop a nurturing relationship with their parents, nor do they develop friendships. Instead, these children prefer to play alone inside their homes, often excessively interested in the parts or movement of objects. Autistic children develop routines and rituals, typically with no functional value, and are uncomfortable with change. New foods, toys, or clothing are disagreeable. Changes in routine are stressful. Autistic children may become distressed for no apparent reason and may even throw tantrums.

According to the research of Wing and Gould (1979), Autism is considered to be neuro-developmental disorder and it is diagnosed on the basis of a triad of behavioral impairments or dysfunctions: 1. impaired social interaction, 2. impaired communication and 3. restricted and repetitive interests and activities.

Even though typical characteristics like problems with social relationships and emotional communication, as well as stereotyped patterns of interests, activities and behaviors are found, it cannot be said as simple psychiatric disorder. It impacts the normal development of the brain in the areas of social interaction and language. Sensory integration is one of their main problems. Typically, it appears during the first three years of life. Early diagnosis and appropriate educational programs are very important to children with autism.

Causes

1. So far no definite causes have been authentically proved.
2. Dr. Eric Courchesne in late 1980's in his study found out that 'two areas of the cerebellum, lobules VI and VII were significantly smaller in some autistic children when compared to normal children'.
3. Genetic influence comprises a significant aspect of research in the causes of autism. The genetic alterations and deletions will simply bring about a changed

structure or process which affects many other needed structures and processes of human development. The American Journal of Psychiatry says that about 10 different genes are identified to be involved in the development of autism. Moreover, some research says that, it is located in a suspect area of chromosome 7 that has been previously linked to autism spectrum disorders. The research also advances basic understanding in the genetic architecture of the genome of autistic individuals and helps in focusing future research.

4. Though not authentically proved, some of the reports are linking the measles, mumps, rubella (MMR) vaccine with the cause for autism.
5. Environmental factors such as mercury and radiation have been proposed as possible causes of Autism.

Symptoms of Autism:

The following symptoms of autism are seen in the language, social behavior, and behaviors concerning objects and routines:

- Lack of Communication in both verbal (spoken) and non-verbal (such as pointing, eye contact, or smiling).
- Lack of Social interactions like sharing emotions, understanding others thoughts and feelings (sometimes called empathy), and conversing with others, as well as the amount of time spent for interacting with others.
- Lack of spontaneous pretend play (i.e) impairment in imagination.
- Prevalence is seen in 'routines or repetitive behaviors', which is often called as stereotyped behaviors, such as repeating words or actions, fixatedly following routines or schedules, inappropriate ways of playing with toys or objects in repetitive manner, or having very specific and rigid ways of arranging items.

The language and communication component of autism is characterized by delayed development of spoken language with lack of alternative communication strategies in the absence of spoken language (such as gesturing). Autistic children make little or no eye contact and do not respond well to verbal communication, sometimes appearing

deaf by their lack of response. Echolalia, also a symptom of TS, may be used instead of appropriate conversational responses. Autistic children may laugh inappropriately. They show limited ability to hold a conversation and do not appear to consider what other people think or understand. They are unreceptive to nonverbal communication such as tone of voice or body language. Autistic children do not engage in the make-believe or socially imitative play that other children exhibit. The extent of language development by age 7 is indicative of the severity of a child's autism, the symptoms of which will persist throughout life.

Autism may be accompanied by other features, including general intelligence deficits and epilepsy. Autistic children's IQs are often 70 or lower (average IQ is 100). Typically, scores on verbal tests are poorer than those based on motor or spatial skills. Some autistic children show unusual ability in selective skills, such as complex mathematics or music. Because of their other deficits, however, autistic children are typically unable to use these skills in a productive manner.

Schizophrenia

Schizophrenia is a chronic, severe, and disabling mental illness. It affects men and women with equal frequency. People suffering from schizophrenia may have the following symptoms:

- Delusions, false personal beliefs held with conviction in spite of reason or evidence to the contrary, not explained by that person's cultural context
- Hallucinations, perceptions (can be sound, sight, touch, smell, or taste) that occur in the absence of an actual external stimulus (Auditory hallucinations, those of voice or other sounds, are the most common type of hallucinations in schizophrenia.)
- Disorganizedthoughts and behaviors
- Disorganized speech
- Catatonic behavior, in which the affected person's body may be rigid and the person may be unresponsive

Schizophrenia and other mental health disorders have fairly strict criteria for diagnosis. Time of onset as well as length and characteristics of symptoms are all

factors. The active symptoms of schizophrenia must be present at least 6 months, or only 1 month if treated.

> Schizophrenia is usually diagnosed in people aged 17-35 years. The illness appears earlier in men (in the late teens or early twenties) than in women (who are affected in the twenties to early thirties). Many of them are disabled. They may not be able to hold down jobs or even perform tasks as simple as conversations. Some may be so incapacitated that they are unable to do activities most people take for granted, such as showering or preparing meal. Many are homeless. Some recover enough to live a life relatively free from assistance.

Schizophrenia Causes

The causes of schizophrenia are not known. However, interplay of genetic, biological, environmental, and psychological factors are thought to be involved. We do not yet understand all the causes and other issues involved, but current research is making steady progress towards elucidating and defining causes of schizophrenia.

In biological models of schizophrenia, genetic (familial) predisposition, infectious agents, allergies, and disturbances in metabolism have all been investigated.

The current concept is that multiple genes are involved in the development of schizophrenia and that factors such as prenatal (intrauterine), perinatal, and nonspecific stressors are involved in creating a disposition or vulnerability to develop the illness. Neurotransmitters (chemicals allowing the communication between nerve cells) have also been implicated in the development of schizophrenia. The list of neurotransmitters under scrutiny is long, but special attention has been given to dopamine, serotonin, and glutamate.

Also, recent studies have identified subtle changes in brain structure and function, indicating that, at least in part, schizophrenia could be a disorder of the development of the brain.

It is important for doctors to investigate all reasonable medical causes for any acute change in someone's mental health or behavior. Sometimes a medical condition that might be treated easily, if diagnosed, is responsible for symptoms that resemble those of schizophrenia.

Schizophrenia Symptoms

Usually with schizophrenia, the person's inner world and behavior change notably. Behavior changes mightinclude the following:

- Social withdrawal
- Depersonalization (intense anxiety and a feeling of being unreal)
- Loss of appetite
- Loss of hygiene
- Delusions
- Hallucinations (e.g., hearing things not actually present)
- The sense of being controlled by outside forces

A person with schizophrenia may not have any outward appearance of being ill. In other cases, the illness may be more apparent, causing bizarre behaviors. For example, a person with schizophrenia may wear aluminum foil in the belief that it will stop one's thoughts from being broadcasted and protect against malicious waves entering the brain.

People with schizophrenia vary widely in their behavior as they struggle with an illness beyond their control. In active stages, those affected may ramble in illogical sentences or react with uncontrolled anger or violence to a perceived threat. People with schizophrenia may also experience relatively passive phases of the illness in which they seem to lack personality, movement, and emotion (also called a flat affect). People with schizophrenia may alternate in these extremes. Their behavior may or may not be predictable.

In order to better understand schizophrenia, the concept of clusters of symptoms is often used. Thus, people with schizophrenia can experience symptoms that may be grouped under the following categories:

Positive symptoms- Hearing voices, suspiciousness, feeling under constant surveillance, delusions, or making up words without a meaning (neologisms).

Negative (or deficit) symptoms - Social withdrawal, difficulty in expressing emotions (in extreme cases called blunted affect), difficulty in taking care of themselves, inability to feel pleasure (These symptoms cause severe impairment and are often mistaken for laziness.)

Cognitive symptoms - Difficulties attending to and processing of information, in understanding the environment, and in remembering simple tasks

Affective (or mood) symptoms - Most notably depression, accounting for a very high rate of attempted suicide in people suffering from schizophrenia

Helpful definitions in understanding schizophrenia include the following:

Psychosis: Psychosis is defined as being out of touch with reality. During this phase, one can experience delusions or prominent hallucinations. People with psychoses are not aware that what they are experiencing or some of the things that they believe are not real. Psychosis is a prominent feature of schizophrenia but is not unique to this illness.

Schizoid: This term is often used to describe a personality disorder characterized by almost complete lack of interest in social relationships and a restricted range of expression of emotions in interpersonal settings, making a person with this disorder appear cold and aloof.

Schizotypal: This term defines a more severe personality disorder characterized by acute discomfort with close relationships as well as disturbances of perception and bizarre behaviors, making people with schizophrenia seem odd and eccentric because of unusual mannerisms.

Hallucinations: A person with schizophrenia may have strong sensations of objects or events that are real only to him or her. These may be in the form of things that they believe strongly that they see, hear, smell, taste, or touch. Hallucinations have

no outside source, and are sometimes described as "the person's mind playing tricks" on him or her.

Illusion: An illusion is a mistaken perception for which there is an actual external stimulus. For example, a visual illusion might be seeing a shadow and misinterpreting it as a person. The words "illusion" and "hallucination" are sometimes confused with each other.

Delusion: A person with a delusion has a strong belief about something despite evidence that the belief is false. For instance, a person may listen to a radio and believe the radio is giving a coded message about an impending extraterrestrial invasion. All of the other people who listen to the same radio program would hear, for example a feature story about road repair work taking place in the area.

FormalThoughtDisorder

The language deficits of schizophrenia are mainly seen in individuals fitting the disorganized classification. These prominent language deficits are typically described as “formal thought disorder” (indicating a problem with the content of speech, rather than the speech itself), though some abnormalities in voice quality, articulation, grammar, and fluency may occur. Word approximation, neologisms, and association chaining are all language deficits observed in schizophrenics.

Word approximation is the use of words that only approximate the intended meaning. For instance, a schizophrenic may use the word “reflector” when he means to say “mirror,” or “handshoe” when he means to say “glove.” The use of word approximation suggests that schizophrenics may not have appropriate recall of all of the words in their vocabulary, as their vocabularies are often extensive and they know and understand the words that they sometimes approximate.

Neologisms are made-up words. Schizophrenics may insert made-up words into their conversation, typically using them in forms that follow standard syntax and grammar, such as using conventional endings for verb tense. For example, “I gotangrywhenhetwiggled me.”

Association chaining occurs when schizophrenics get sidetracked in their language by words that may have alternate meanings. For example, “The pigs escaped from the pen. This pen has ink.” Interestingly, when a word has two possible

meanings, schizophrenics may interpret the word to mean the more common use, rather than determining its meaning by context. In the sentence, “Every Friday night they play bridge,” a schizophrenic may interpret the word “bridge” to mean a structure that spans over water, in spite of the fact that the context of the sentence (the word “play”) would indicate that “bridge” is a card game. Similarly, schizophrenics may also take idioms literally when it is possible to visualize them in a literal manner: “He kicked the bucket.” When there is no clear literal meaning, however, schizophrenics are more likely to understand that these are merely figures of speech, as in the phrase, “She paid through the nose for that dress.”

Dementia

Dementia is a loss of brain function that occurs with certain diseases. Alzheimer's disease (AD), is one form of dementia that gradually gets worse over time. It affects memory, thinking, and behavior.Memory impairment, as well as problems with language, decision-making ability, judgment, and personality, are necessary features for the diagnosis. It refers to the results of a number of different diseases all of which lead to a loss of intellectual abilities. It is caused by the deterioration of brain tissue. Different dementia affects different parts of the brain.

Causes, incidence, and risk factors

Age and family history are risk factors for AD.

- As you get older, your risk of developing AD goes up. However, developing Alzheimer's disease is not a part of normal aging.

- Having a close blood relative, such as a brother, sister, or parent who developed AD increases your risk.
- Having certain combination of genes for proteins that appear to be abnormal in Alzheimer's disease also increases your risk.

Other risk factors that are not as well proven include:

- Longstanding high blood pressure
- History of head trauma
- Female gender

There are two types of AD -- early onset and late onset.

- In early onset AD, symptoms first appear before age 60. Early onset AD is much less common than late onset. However, it tends to progress rapidly. Early onset disease can run in families. Several genes have been identified.
- Late onset AD, the most common form of the disease, develops in people age 60 and older. Late onset AD may run in some families, but the role of genes is less clear.

The cause of AD is not entirely known, but is thought to include both genetic and environmental factors. A diagnosis of AD is made when certain symptoms are present, and by making sure other causes of dementia are not present.

The only way to know for certain that someone has AD is to examine a sample of their brain tissue after death. The following changes are more common in the brain tissue of people with AD:

- "Neurofibrillary tangles" (twisted fragments of protein within nerve cells that clog up the cell)
- "Neuritic plaques" (abnormal clusters of dead and dying nerve cells, other brain cells, and protein)
- "Senile plaques" (areas where products of dying nerve cells have accumulated around protein).

When nerve cells (neurons) are destroyed, there is a decrease in the chemicals that help nerve cells send messages to one another (called neurotransmitters). As a result, areas of the brain that normally work together become disconnected.

The buildup of aluminum, lead, mercury, and other substances in the brain is no longer believed to be a cause of AD.

Symptoms

Dementia symptoms include difficulty with many areas of mental function, including:

- Language
- Memory
- Perception
- Emotional behavior or personality
- Cognitive skills (such as calculation, abstract thinking, or judgment)

Dementia usually first appears as forgetfulness.

Mild cognitive impairment is the stage between normal forgetfulness due to aging, and the development of AD. People with MCI have mild problems with thinking and memory that do not interfere with everyday activities. They are often aware of the forgetfulness. Not everyone with MCI develops AD.

Symptoms of MCI include:

- Forgetting recent events or conversations
- Difficulty performing more than one task at a time
- Difficulty solving problems
- Taking longer to perform more difficult activities

The early symptoms of AD can include:

- Language problems, such as trouble finding the name of familiar objects
- Misplacing items
- Getting lost on familiar routes
- Personality changes and loss of social skills
- Losing interest in things previously enjoyed, flat mood
- Difficulty performing tasks that take some thought, but used to come easily, such as balancing a checkbook, playing complex games (such as bridge), and learning new information or routines

As the AD becomes worse, symptoms are more obvious and interfere with your ability to take care of yourself. Symptoms can include:

- Forgetting details about current events

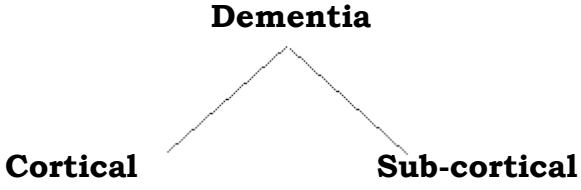
- Forgetting events in your own life history, losing awareness of who you are
- Change in sleep patterns, often waking up at night
- Difficulty reading or writing
- Poor judgment and loss of ability to recognize danger
- Using the wrong word, mispronouncing words, speaking in confusing sentences
- Withdrawing from social contact
- Having hallucinations, arguments, striking out, and violent behavior
- Having delusions, depression, agitation
- Difficulty doing basic tasks, such as preparing meals, choosing proper clothing, and driving

People with severe AD can no longer:

- Understand language
- Recognize family members
- Perform basic activities of daily living, such as eating, dressing, and bathing

Other symptoms that may occur with AD:

- Incontinence
- Swallowing problems



Sub cortical - Parkinson's disease

- This is a kind of brain tissue deterioration
- Sig impact ability to produce speech
- Slow in all motor activities, as well as to control vocal apparatus.
- Dysarthria – different in articulating speech and dysgraphia is found.
- Written form
- The boy on the stolltripp and the girl laugh at the boy and then she spill water on the floor.
- Error on inflectional endings
- Attention to one tack
- New verb – forgotten/ semantic use only in memory

Cortical – Alzheimer's disease

Cellular damage in the brain temporal and frontal lobe

Samples

"No, for goodness sake. What is you doing? Coming home from a story, or playing? My parents is a has a present for you…….Ah, your parents has the house cleaning. Timmy. We, we, no. running out at three then, the car wash, they, uh, fill, four, happy, everyone. Then can come back again"

- Cohesion is lacking
- Over- frequent topic shifts
- Repetitive and faily empty
- Morphological and syntactical errors

- A lexical selection error (story and is)
- Problem in number agreement. What is you doing/your parents has
- Subject lacking, running out/ can come back again.
- Only word order is presented.

Lexicon

Word finding difficulties

Mental Retardation

Mental Retardation is the most common cause of delayed language development among children. It is identified at a very early age. Many people have misperceptions on the characteristics and abilities of mentally retarded children. It is important to remember that a mentally retarded child's language will not be much different from other children's language, it will just be slower.

What is mental retardation?

Mental retardation, also known as intellectual disability, is a term used when there are limits to a person's ability to learn at an expected level and function in daily life. Levels of mental retardation vary greatly in children – from a very slight problem to a very severe problem. Children with mental retardation might have a hard time letting others know their wants and needs,

and taking care of themselves. Mental retardation could cause a child to learn and develop more slowly than other children of the same age. It could take longer for a child with mental retardation to learn to speak, walk, dress, or eat without help, and they could have trouble learning in school. Mental retardation can be caused by a problem that starts any time – even before birth / before a child turns 18 years old . It can be caused by injury, disease, or a problem in the brain. For many children, the cause of their mental retardation is not known. Some of the most common known causes of mental retardation – like Down syndrome, fetal alcohol syndrome, fragile X syndrome, genetic conditions, birth defects, and infections – happen before birth. Others happen while a baby is being born or soon after birth. Still other causes of mental retardation do not occur until a child is older; these might include serious head injury, stroke, or certain infections.

There are many signs of mental retardation. For example, children with mental retardation may:

■ sit up, crawl, or walk later than other children

■ learn to talk later, or have trouble speaking

■ find it hard to remember things

■ have trouble understanding social rules

■ have trouble seeing the results of their actions

■ have trouble solving problems

Language in Mental Retardation

Most children with Mental Retardation acquire a language system and functional speech. The rate and extent at which a child learns language is in proportion to the degree of the retardation. The more extensive the retardation is, the harder it is for the child to learn language. Mentally retarded children begin producing single words about 2 years later than normal children. Their vocabularies are limited, their sentences are shorter and structurally less complex than the average child's. Unlike normal learning children, mentally retarded children tend to use their speech more for demand than for conveying ideas. These children don't just have delayed learning in language, they are delayed in almost all parts of development.

Phonology and Morphology

Difficulties with speech production (articulation) are more common among children with mental retardation than among children without (Long & Long, 1994). However, according to Shriberg and Widder (1990), estimates of the incidence of these speech-production deficits have been reported as low as 5 percent and as high as94 percent. Most studies have found that although there is an increased incidence of speech production problems among children with mental retardation, these children, appears to follow the same course of development as children without retardation and make similar phonological errors (Shriberg&Widder, 1990). The most common phonological errors are

reduction of consonant clusters (saying bake for break) and final consonant deletion (saying call for cat). It appears that children with more severe mental retardation have a greater incidence of speech-production problems. However, some studies that have directly investigated the relationship between 1Q and articulation have failed to find that children with lower 1Q scores have more articulation difficulties. It may be that because children with more severe disabilities often have many related physical problems (such as cleft palate, protruding tongue. and the like), it is these problems, rather than IQ score, that relate to the higher incidence of speech-production problems. Factors other than physical characteristics have also been suggested to cause speech production problems. For example, Shriberg and Widder (1990) suggested that children with mental retardation appear to have difficulty with phonological encoding (similar to the problems experienced by many children with learning disabilities). Pruess, V'adasy, and Fewell (1987) noted that there is a higher incidence of otitis media (middle-ear infections) in children with Down syndrome. Otitis media has been found as a cause for fluctuating hearing loss, which can cause impairments in articulation. Therefore, hearing problems are another possible cause of the articulation problems frequently found among children with mentalretardation. The research on speech production in children with mental retardation suggests that education professionals should be prepared to help these children to enhance their speech skills. Many children with mental retardation have articulation difficulties that interfere with their ability to be successful in school and in social interactions. However,

as Shriberg and Widder (1990) point out, speech training for children with mental retardation is being deemphasized in schools. There are concerns about the stow rate of progress of such instruction and about the amount of time that speech training takes away from the teaching of what maybe more functional skills. They suggest that microcomputer training programs might be useful in delivering speech training to children with mental retardation. In addition, it may be possible for teachers to incorporate some articulation training into regular classroom routines. Studies of the development of morphological skills in children with mental retardation have generally found that these skills develop in a manner similar to that of children without retardation but at a significantly slower rate (Newfield &Schlanger, 1968). In other words, although children with mental retardation appear to be delayed in their ability to form words, they follow the same sequence of development as nondisabled children.

Syntax

Research on syntactic skills development in children with mental retardation has also generally found that while there are delays in development of these skills, the pattern of development is the same as that found in nondisabled children. Lackner (1968) examined the syntax production of five children with mental retardation, ages 6 and 16. He found that their sentence length increased with mental age and was similar to that of nondisabled children of similar mental age. Lackner also found that the order of development of syntactic rules was similar. One difference that Lackner found in his sample of

individuals with mental retardation was that they less frequently used the more advanced syntactic structures. Kamhi and Johnston (1982) found similar results in their study of the language development of children with mild mental retardation. When compared to that of nondisabled children of similar mental age, the syntactic development of the children with mental retardation appeared to be quite similar. Interestingly, the researchers also compared the children with mental retardation to children with specific language impairments but who had IQ scores in the normal range. They concluded that the language produced by the children with language impairments was less complex and contained more errors than that produced by the children with mental retardation. Both the Kamhi and Johnston (1982) study and other studies (e.g., Naremore&Dever. 1975.) found that children with mental retardation had more difficulty with more advanced language constructs. For example, Kamhi and Johnston (1982) found that the nondisabled children produced more sentences with questions and with conjunctions. These findings suggest that there may be limits to the syntactic development of children with mental retardation-that is, although their early development may be similar to that of nondisabled children (although with delays), there may be a plateau of development. After this plateau, further syntactic development may be difficult. We cannot be sure there are limits to the syntactic development of children with mental retardation. One reason is that there may be methodological problems with the research, as Kamhi and Johnston (1982) themselves pointed out. Another reason is that there is a great deal of variability within the

population called mentally retarded. There are undoubtedly some individuals with mental retardation who are able to acquire more advanced syntactic skills. Thus, research results can be used as a guide for intervention but should never be used to justify the denial of services to any individual. The research on syntactic skills of children with mental retardation suggests that education professionals may generally expect slow development of these abilities along a normal developmental course, with the possibility of students' having particular difficulty in the acquisition of more advanced syntactic skills. Teachers may need to simplify their own language, as well as written text, to point out some of the more advanced syntactic structures (such as the passive voice)when they occur, and to encourage the use of more complex syntactic skills in older children with mental retardation.

Semantics

There has been relatively little research on the semantic abilities of children with mental retardation. The research that has been done indicates that children with mental retardation tend to be more concrete in their understanding of words, having more difficulty, for example, interpreting idiomatic expressions (e.g., he broke her heart) (Ezell & Goldstein, 1991). This tendency to be more concrete may be the result of delays in development of semantic abilities (Rosenberg, 1982). Some studies have found that an area of strength for children with mental retardation is that of vocabulary skills. In a study of the comprehension of syntax and vocabulary conducted by Chapman,

Schwartz, and Kay Raining-Bird (1991), the authors found that their subjects with mental retardation performed significantly better on the vocabulary comprehension task than on tests of syntactic skills, in fact, outscoring a mental-age matched control group on their vocabulary comprehension. Other studies have found that examination of language produced in natural settings shows children with Down syndrome have a more diverse vocabulary than do non-disabled children matched for mental age (Miller, 1988). To understand these results, one should keep in mind that in these studies the children with mental retardation were older than the control group and, therefore, may have had more of an opportunity to learn vocabulary skills. Even so, their vocabulary skills are not equivalent to those of nondisabled children of the same chronological age. Another aspect of semantics involves the organization of language information. If children are given groups of pictures and asked to remember them, they tend to organize the pictures in their minds and recall them in groups. These groups may be based on physical characteristics or function of the items or on the conceptual category to which the items belong (e.g., toys, animals). Children with mental retardation have been found to lag behind in their development of organizing strategies (Stephens, 1972) and to use more concrete concepts (MacMillan. 1982), suggesting that children with mental retardation have some difficulty in developing and using semantic concepts.

Hearing Impairment

A generic term indicating a hearing disability which may range in severity from mild to profound: it includes the subsets of deaf and hard of hearing.

A deaf person is one whose hearing disability precludes successful processing of linguistic information through audition, with or without a hearing aid.

A hard of hearing is one who, generally with the use of a hearing aid, has residual hearing sufficient to enable successful processing of linguistic information through audition.

Prelingual deafness

Prelingual deafness is deafness present at birth, or occurring early in life at an age prior to the development of speech or language.

Postlingual deafness

Postlingual deafness is deafness occurring at any age of following the development of speech or language.

The following hearing threshold classifications are;

- Mild:
    - for adults: between 25 and 40 dB
    - for children: between 20 and 40 dB
- Moderate: between 41 and 55 dB
- Moderatelysevere: between 56 and 70 dB
- Severe: between 71 and 90 dB
- Profound: 90 dB or greater

Classifications

There are three major classifications:

Conductive Hearing loss:

A conductive hearing loss refers to impairment that interfere with the transfer of sound along the conductive pathway of the ear.

Sensorineural hearing loss:

Sensorineural impairments involve problems confined to the inner ear.

Mixed hearing loss:

A combination of the conductive loss and Sensorineural hearing loss.

Range of human Hearing

The human ear responds to a wide range of frequencies and intensities. We hear very low pitch sound of 20Hz as well as very high pitch sound of 20,000 Hz. We hear sounds of very low intensities as well as very high intensities. The softest signal or the lowest intensity an average adult can hear is 0 dB Hl. Sounds of intensities above 120 dB Hl cause discomfort and may be painful to individuals with normal hearing. That means the range of hearing is dB HL to 120 dB HL and 20 Hz to 20,000 Hz.

The range of human hearing is sufficient for the hearing and understanding of all the speech sounds. The lowest frequency which occur in speech is about 200Hz while the h8ighest frequency which occur in speech is about 8,000 Hz. Whispered speech has an intensity level of about 30 dB SPL. Conversational speech has an average intensity of between 55 and 65 dB SPL.

Ear and Hearing

Parts of the ear

You know that we have two ears, one on either side of the face. The ear or the auditory system can be divided into three main parts, the outer or external ear, the middle ear and the inner ear. The auditory nerve which is connected to the inner ear links the ear with the auditory nervous system.

The outer ear

The outer ear or the external ear consists of pinna or auricle, the part of the ear that is visible outside and the ear canal. The ear canal is S- shaped and is approximately 2.5 cm in length. The outer portion of the canal, about one-third of its length is cartilaginous and remaining two thirds is bony. At the end of the ear canal is the eardrum or the tympanic membrane which separates outer ear and middle ear.

The middle ear

The middle ear is a small cavity with six walls. Ear drum is of its walls. It has three small bones, hammer or malleus, anvil or incus and stirrup or stapes. These three bones or ossicles are connected to each other and form a chain called ossicular chain. The handle of the malleus is connected to the ear drum and the footplate of stapes is connected to the bony wall of the inner ear. The middle ear cavity if filled with air and it is ventilated by the Eustachian tube through which it is connected to the throat.

The inner ear

The inner ear is housed in the temporal bone, which is a part of the skull and consists of fluid filled tubes. The bony tubes, called as bony labyrinth, are filled with a fluid called perilymph. Within this bony labyrinth, a membranous labyrinth consisting of delicate cellular tubes are present. The membranous labyrinth filled with a fluid called endolymph. This membranous labyrinth contains the actual hearing cells, the hair cells of the organ of corti. The bony labyrinth consists of the following three main parts:

- The front portion is the snail-shaped cochlea, which is the organ of hearing.
- The rear part, the semicircular canals, which helps maintaining balance.
- Labyrinthestibule, which interconnects the cochlea and the semicircular canals. It contains the sense organs responsible for balance, the utricle and saccule.

The inner ear has two membrane covered windows into the air filled middle ear – the oval window and the round window. The oval window is immediately behind the steps, the third middle ear bone.

Auditory nervous system:

The eighth cranial nerve, which is called as the auditory nerve or the vestilocochlear nerve connects the inner ear to the brain. There are two hearing centers in the brain, one in each half of the brain. Nerve fibers from each reach both the hearing centers.

How do we hear?

Each part of the ear has a specific function or role in hearing. You have learnt earlier that when a sound is produced, the sound wave causes movement of particles in the ear. The pinna or the auricle collects these sound waves which are funneled by the external auditory canal to the tympanic membrane. When the sound waves hit the tympanic membrane, in start vibrating. These vibrations are transferred to the handle of the malleus and the entire ossicular chain starts vibrating. Remember that the footplate of the stapes is connected to the oval window. So this vibration moves the footplate of the cochlea is set into motion. The hair cells of the cochlea convert this vibration or the mechanical energy or impulses. These electrical impulses are then carried by auditory nerve centre of the brain, where they are heard as sound.

You must have experienced that if you do not want to see something, you can close your eyes. But you cannot close your ears and stop hearing. This is because there are two modes or routs for hearing. They are air conduction and bone conduction. In air conduction hearing, signals travel through outer ear, middle ear and then reach inner ear whereas in bone conduction hearing, inner ear is directly stimulated through vibration of the skill. In everyday situation, we mainly hear through the air conduction mode. The bone conduction path is activated when the intensity of the signal is high (more than 70 Db HL)or when the head is in contact with the source of sound.

Causes of hearing loss

A number of causes can lead to hearing loss. Some of the causes of hearing loss are observable but of the causes cannot be seen. Any problem in the external, the middle or the inner ear or the auditory nerve can lead to hearing loss. The causes of hearing have been divided into causes of external ear problems, middle ear problems and inner ear problems.

Causes of external ear problems

Congenital malformations- these are abnormalities present since birth.

The common abnormalities seen are

Deformity of the pinna

Closure of the ear canal

Blockage of the ear canal due to

Impacted wax

Foreign bodies

Tumor/growths

Inflections of the external ear

Causes of middle ear problems

1. Rupture or perforation of the eardrum
2. Infections of the middle ear
3. Ossiculararabnormalities
    - Congenital absence of one or more ossicles
    - Accidental fracture of one of the three ossicles
    - Improper connection among the ossicles themselves
    - Fixation of one of the ossicles
    - Fluid or any growth in the middle ear which disrupts the movement of the ossicles.

Inner ear and the auditory nerve

a) Congenital malformation of the inner ear and/ or the auditory nerve
b) Destruction of the hair cells of the cochlea due to
    - Aging
    - Trauma/injury
    - Noise
    - Drugs
    - Viral and bacterial infections
c) Changes in the pressure of inner ear fluids

d) Abnormalities in the blood supply to the cochlea

e) Destruction of the nerve cells

The cause for these abnormalities in an individual could be genetic or nongenetic. If the cause of hearing loss is genetic, i.e., related to abnormalities in the genes or chromosomes, they can be passed on from one generation to the other. Such individuals will generally report of a family history of hearing loss. A genetic counselor can predict the chances that an individual with a family history of hearing loss will have hearing loss. Non genetic factors that lead to hearing loss may occur before the birth of a child is born (postnatal causes)

You all must have listened to grandmothers telling pregnant women that they need to be very careful about their health and take nutritious food as it can affect the baby to be born. Listening to the advice of grandmothers can avoid hearing loss in the baby to be born by preventing the prenatal causes of hearing loss. Especially during the first three months of pregnancy, an expecting mother should avoid getting infections. One of the most common prenatal causes of hearing loss is maternal rubella or German measles in the expectant mother. But infections agents such as toxoplasmosis, syphilis, herpes and cytomegalovirus can also lead to hearing loss. Other prenatal causes of hearing loss include exposure to X- rays or radiations, side effects of certain medicines.

A common cause that can affect hearing during the birth of a baby is lack of oxygen to brain or the ear. Lack of oxygen may occur if the child does not cry immediately after birth. Damage to the ears can occur if proper medical care is not taken at the time of delivery of the baby. Low birth weight also indicates that the baby is at risk for hearing loss. Other risk factors include the baby spending more than 10 days on a mechanical ventilator after birth, neonatal jaundice (jaundice especially during the first 10 days after birth). Causes of hearing loss in older in order children and adults include measles, mumps, meningitis (brain infection), ear infection, adverse side effects of certain medications, head injury, exposure to loud sounds.

Language and speech development

By far the most severely affected areas of development in the hearing impaired person are the comprehension and production of the language. The distinction is important because hearing impaired people can be expert in their own form of language. The current opinion is that hearing impaired individuals who use manual sign language are taking part in the production and comprehension of true language. It is much more difficult for Prelingual deaf children to learn to speak than those who have acquired their deafness; mainly they do not receive auditory feed back from the sounds they make. In addition, they have not heard an adult language model. It is thought that these differences occur because hearing infants are reinforced by hearing either themselves or others, are not reinforced. The lack of feedback has also been named as a primary cause of deaf children's poor speech production. Hearing children learn to associate the sensations they receive when they move their jaws, mouth, and language with the auditory sounds these movements produce. Hearing children are obviously handicapped in this process. In addition, they have a difficult time hearing the sounds of adult speech, which other children hear and imitate, so they are deprived of an adequate adult model.

Errors seen in children with hearing disorders

- **<u>Syntacticfactorsseen</u>**
  - ❖ aNNanvaainRu*keek*paricutaa.

    Brother come today cake gift give.
  - ✓ inRu*piRan]tan]aLukku*paricu tara aNNanvarukiRaan.
- Errorsfound:
  - Absence of **case marker** - *ukku*
  - Absence of **tense marker** - kiR
  - Absence of **Infinitive marker** – a
  - Absence of **PNG marker** - aan

- **Sentence structure** – NP VP interchange

Comparative Analysis

ChildrenwithHearing	**ChildrenwithHearingImpairment**
Absence of case marker - 46%	Absence of case marker - 100%
Absence of tense marker - 33%	Absence of tense marker - 91%
Absence of Infinitive marker - 37%	Absence of Infinitive marker - 96%
Absence of PNG marker - 24%	Absence of PNG marker - 78%
Sentence structure problem - 21%	Sentence structure problem - 95%
Reached Positional and sound level	**Only in the Message or Functional level**

- **<u>Semanticfactorsseen</u>**

- Errorsfound:
  - **Lexical substitutions –**

    Today, tomorrow, yesterday confusions are found. (Problem in time concept)

    A. Synonyms

    accam – payam

    paacam – anpu

    tunpam – cookam

    aanan⌉tam – maki<u>l</u>cci

    tuniccal - viiram

    B. Hypernyms

    caikil, kaar, pas, paik, aaTToo – vanTi

    rooja, malli, alli – pu:

    baniyan, caTTai, kaalcaTTai – caTTai

    appil, aaranju, koyyaa, maatuLai – pa<u>l</u>am

    C. Substitutions with semantic field related words.

    piRan⌉tan⌉aaL - keek
  - **Lexical Expansions –**

    kaTikaaram – maNipaarkkumporuL

coompeeri – veelaionRumceyaathavan

Comparative Analysis

ChildrenwithHearing	**ChildrenwithHearingImpairment**
Lexical substitution - 32%	Lexical substitution - 99%
Lexical Expansions – 21%	Lexical Expansions – 47%

Developmental language disorders/Language Delay

Developmental language disorder (called as language delay) is a condition wherein a child does not learn language as quickly as his/her peers. For example, a 5-year-old child may speak and understand language like a 3 year old. These children may have normal intelligence, or they may have a condition involving mental retardation, which cause a language delay. Developmental language disorders in the absence of mental retardation or any other impairment may be hereditary, or genetic. Recent neuroimaging (brain imaging—i.e. MRI) studies have identified differences in the shapes of brains of persons with such developmental language disorders. Further, developmental language disorders could be a result of hearing loss, which may be permanent (i.e. congenital—present at birth, genetic) or transient (i.e. due to middle ear infection—see section on otitis media).

Children with developmental language disorders learn language in the same sequence as their normally developing peers, but the pace is delayed. For example, typically developing children produce first words between the ages of 10-12 months, increasing the number of single words produced by 12-18 months, speaking in two-word phrases between the ages of 18-24 months, and

using 2-3 word phrases in the second year of life. They use 3-4 word "adult-like" sentences in their third year of life. By the age of 5, normally developing children have learned 90% of the grammar they will need in life. Children with more moderate or severe language disorders show delays in these early milestones as well as delays in learning language in school (i.e. learning vocabulary words, spelling, reading, writing). These children may recover with or without treatment to "catch up" with their peers; however, depending on the severity, speech/language therapy is usually advisable. Early intervention is frequently a key to successful remediation before the school-age years. If difficulties persist into the elementary school years, this disorder is called a language learning disability.

Developmental language disorders (vs. a delay which resolves with or without treatment) never "go away", and they will always be a part of a person's life. These disorders can best be diagnosed and treated by a speech-language pathologist. In most cases, the continued efforts of a speech-language pathologist in working with a child's family and classroom teachers are necessary. However, a great deal of research has been conducted with persons with these disabilities, and speech-language pathologists have found effective ways to help these students develop strategies that are effective in facilitating learning and independence in learning. Parents and classroom teachers are in key positions to help the speech-language pathologist evaluate and treat the child.

UNIT – III

Aphasia

Aphasia is a disturbance of one or more aspects of the complex process of comprehending and formulating verbal messages that result from newly acquired disease of the central nervous system (CNS). We shall begin by considering this operational definition of aphasia, analyzing each of its components.

Newly Acquired Disease:

It is important to note that the disease that produces aphasia is both acquired and recent (e.g., cerebral infarction, tumor, or contusion) rather than congenital and long standing (e.g., genetic or environment induced prenatal cerebral defect). The former (acquired disease) deals with individuals previously capable of using language appropriately. The latter may produce developmental language defects in young individuals whose ability to use language will never attain a normal level.

Most clinicians will agree that although aphasic disability is complex, many patients are clinically similar and will fall into recurring identifiable groups. This is a basic thesis of this book. There are many classifications, indicating that none is altogether satisfactory, but also that this effort is useful and even necessary to diagnose and treat aphasics or to understand the phenomena. The bewildering proliferation of the nomenclature deters most but the truly devoted to master classification. With clinical experience comes the realization that:

1. Indeed there is a need for classification.
2. The patients and their symptomatology are complex yet similar enough to the experience of others.
3. Many of the classifiers describe the same phenomena from a different angle and in fact, complement rather than contradict each other.

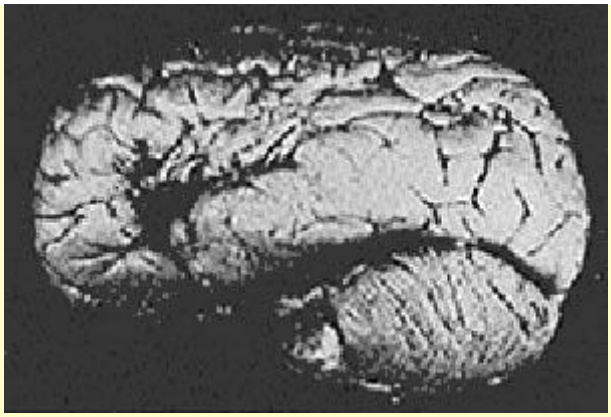
The opponents of classification point out the numerous disagreements among observers, the many exceptions that cannot be fitted into categories, and the frequent evolution of certain types into others. They also object to the over simplification involved in any practical, descriptive system.

The controversy can be reduced to a few issues. Is aphasia a unitary disturbance or are there several kinds of aphasias? The answer, of course, is yes to both. There is something qualitatively different about aphasic language disturbance which sets it apart from dysarthria, mutism, confusion psychotic speech, just to mention the main problem areas in differential diagnosis. What makes aphasic disturbance so palpably different is difficult to define to everyone's satisfaction, but a multidisciplinary approach may be acceptable to most: *aphasia is a neurologically central disturbance of language characterized by paraphasias, word finding difficulty, and variably impaired comprehension,* associated with disturbance of reading and writing, at times with dysarthria, nonverbal constructural, and problem-solving difficulty and impairment of gesture.

BROCA'S AREA , WERNICKE'S AREA, AND OTHER LANGUAGE-PROCESSING AREAS IN THE BRAIN

The process of identifying the parts of the brain that are involved in language began in 1861, when Paul Broca, a French neurosurgeon, examined the brain of a recently deceased patient who had had an unusual disorder. Though he had been able to understand spoken language and did not have any motor impairments of the mouth or tongue that might have affected his ability to speak, he could neither speak a complete sentence nor express his thoughts in writing. The only articulate sound he could make was the syllable "tan", which had come to be used as his name.

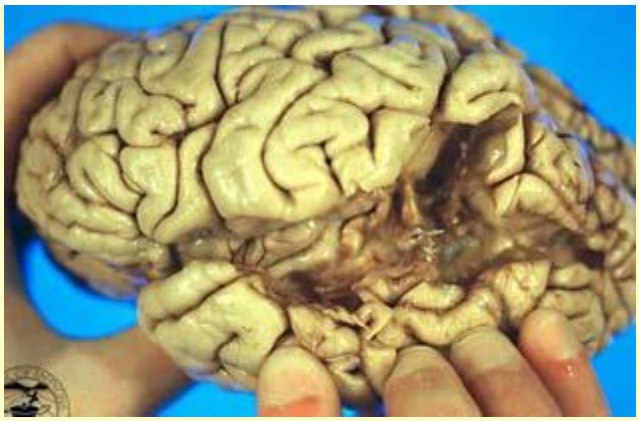
Paul Broca



Tan's brain

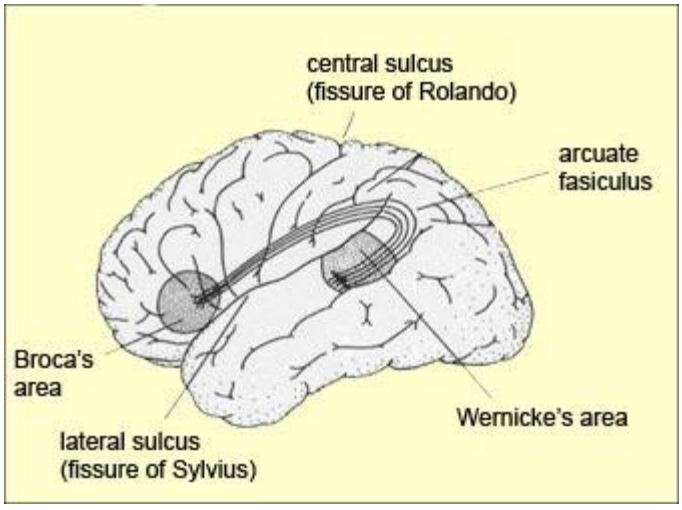
When Broca autopsied Tan's brain, he found a sizable lesion in the left inferior frontal cortex. Subsequently, Broca studied eight other patients, all of whom had similar language deficits along with lesions in their left frontal hemisphere. This led him to make his famous statement that "we speak with the left hemisphere" and to identify, for the first time, the existence of a "language centre" in the posterior portion of the frontal lobe of this hemisphere. Now known as Broca's area, this was in fact the first area of the brain to be associated with a specific function—in this case, language.

Ten years later, Carl Wernicke, a German neurologist, discovered another part of the brain, this one involved in understanding language, in the posterior portion of the left temporal lobe. People who had a lesion at this location could speak, but their speech was often incoherent and made no sense.



Carl Wernicke **Brain with a lesion causing Wernicke's aphasia**

Wernicke's observations have been confirmed many times since. Neuroscientists now agree that running around the **lateral sulcus** (also known as the fissure of Sylvius) in the left hemisphere of the brain, there is a sort of neural loop that is involved both in understanding and in producing spoken language. At the frontal end of this loop lies **Broca's area**, which is usually associated with the production of language, or language outputs . At the other end (more specifically, in the superior posterior temporal lobe), lies **Wernicke's area**, which is associated with the processing of words that we hear being spoken, or language inputs. Broca's area and Wernicke's area are connected by a large bundle of nerve fibres called the **arcuate fasciculus**.



This language loop is found in the left hemisphere in about 90% of right-handed persons and 70% of left-handed persons, language being one of the functions that is performed asymmetrically in the brain. Surprisingly, this loop is also found at the same location in deaf persons who use sign language. This loop would therefore not appear to be specific to heard or spoken language, but rather to be more broadly associated with whatever the individual's primary language modality happens to be.

In addition to Broca's and Wernicke's areas, a third area of importance for language, located in the parietal cortex, has been described more recently.

BROCA'S APHASIA

What Broca (1861) described as aphemia. Wernicke (1874) called motor aphasia. Marie (1906) did not consider Broca'saphemia true aphasia. Pick (1913) labelled it expressive aphasia with agrammatism, and Weisenburg and McBride (1935) popularized expressive aphasia, which still enjoys favor among many. The problem with the term expressive is, of course that all aphasias have some "expressive" difficulties. Then came the "innovators" such as Henry Head (1926), whose distaste for his predecessors, diagrams resulted in a unique psycholinguistic classification which is difficult to apply to clinical cases. Broca's aphasia thus became verbal aphasia. After Head, only Wepman (1951) used similar terminology extensively in the literature. Luria's (1964) physiological concepts led to "efferent motor" aphasia. Jacobson's (1964) linguistic approach used "contiguity" or combination disorders for this phenomenon, and Osgood (1963) called it "encoding" disturbance. Schuell's (1964) classification is highly individualistic and difficult to correlate with others. Her Group 3 severe reduction of language with sensorimotor disturbance corresponds best to Broca's aphasia. Bay (1964) like Marie (1906) considered "aphemia" different from aphasia and gave it the term "cortical dysarthria," a theoretical deviation from the consensus which considers these patients aphasic. "Anarthria", the term used by Marie for the same phenomenon is used by modern neurologists to describe the most severe dysarthria, due to bulbar or "pseudobulbar" paralysis (Critchley, 1970). Recently interest in aphemia or cortical dysarthria has been renewed by Darley (1964) and other speech pathologists, who have popularized the term "apraxia of speech" or "verbal apraxia" to identify a purely motor speech disorder, distinct from dysarthria and often seen in association with aphasia. Darely (1964) and Johns and

lapointe(1976) emphasize the necessity to separate this entity from the rest of the aphasias, from the point of view of therapy. The relationship of Broca's aphasia to apraxia of speech remains controversial. Geschwind (1965) DeRenzietal (1966) and Heilman (1973) suggest that Broca's aphasia and facial appraxia may vary independently. Many cases of marked "verbal apraxia" have comprehension deficit, and the articulatory disorder is similar to the phonemic paraphasias seen in other aphasics. Yet there are some patients in whom the articulatory disorder is distinctive enough to justify nosologic separation.

More recent clinically and linguistically oriented classifications place an emphasis on the fluency-nonfluency dichotomy in aphasia. Goodglass and Kaplan (1972) and many others have recognized the clinical relevance of measuring fluency. They also advocate the retention of the classic eponym "Broca's aphasia," rather than using "motor" or "expressive" aphasia, in order to avoid suggesting that speech output is normal in other forms of aphasia. The agrammatism of Broca's aphasia is characterized by the relative preservation of substantive words in contrast to the syntactical modifiers. Short nouns are more likely to remain than verbs (telegraphic speech). Only short propositional phrases or automatic sentences appear. The linguistic analysis of comprehension in Broca's aphasia suggested that this parallels the expressive difficulties. These patients have more difficulty comprehending grammatical modifiers than substantive words; a similarly specific deficit for reading exists also.

Much of the controversy about Broca's aphasia centers on the existence of comprehension deficit. These patients are characterized by relatively well preserved comprehension and their major disability is in language output. However, if comprehension is examined extensively, it is found to be impaired to a certain extent, almost without exception. This prompted many investigators to emphasize that

comprehension deficit is an all-pervasive feature of aphasia, and that the variable amount of motor difficulty, at times labeled "cortical dysarthria," superimposed on aphasia results in the variation of the clinical picture called "Broca's aphasia," Mohr (1978) makes the point that persisting Broca's aphasia with severely reduced speech output and agrammatisms, is the result of a large infarct extending beyond Broca's area and Broca's area involvement results in mild usually transient, motor speech disturbance. He usefully splits the traditional entity of Broca's aphasia on the basis of persistence, and lesion size and location.

Most clinicians agree, however, that motor aphasia primarily expressive aphasia or Broca's aphasia, is an identifiable, common, aphasic syndrome with scant, hesitant, effortful and paraphasic, spontaneous speech, at times, slightly better repetition, and relatively good comprehension. Besides the restriction of vocabulary and grammar, there is, often, impaired articulation. They read aloud poorly, but reading comprehension is often good. Writing is affected similarly to speech. The variability of features and severity led to splitting and redefinition of the syndrome by some authors, although it continues as a real entity in the clinical in research.

WERNICKE'S APHASIA

Sensory aphasia, as described by Wernicke (1874) in his famous paper "Der aphasischesymptomenkomplex" is recognized by everyone, with the notable exception of Hughlings Jackson. Jackson's (1879) hierarchial view of language dissolution included "jargon" as a disturbance of expression, but these recurrent utterances were more stereotypic than the profuse phenomic or semantic jargon of sensory aphasia. Marie (1906) claimed that sensory aphasia was the only true aphasia and this is still championed by Bay (1964). Schuell (1964) was also impressed by the auditory disturbance as the *sine qua non* of aphasia. Curiously, her classification does not have

a single group which could be identified unequivocally with sensory or Wernicke's aphasia. Head (1926), like Wepman and Schuell after him, in order to avoid the input - output dichotomy and the notion of pure language defects, created novel classifications, deviating from the clinically obvious, and confusing generations of readers for many years. His syntactic aphasia is not the same as Wepman's who called sensory aphasia "pragmatic," and the motor, "syntactic," Jacobson's(1964) "similarity" or "selection" disorder encompasses a range of clinical disturbances, such as "sensory," "semantic" and "acoustic amnestic" aphasia, as he used Luria's (1964) terminology. According to Jacobson, sensory aphasia is characterized linguistically by preserved syntactic units and phonemic combinations, althoug certain phonemioc distinctions are lost. Osgood's (1963) decoding disturbance is in this category also.

Jargon aphasia is at times identified as a separate entity, although most writers will classify it with Wernicke's or sensory aphasia. The fluent, profusely paraphasic speech may be usefully subdivided into semantic and neologistic Jargon (Kertesz and Benson, 1970), depending on the degree of phonemic distortions or neologisms (the paraphasic and asemantic jargon or Alajouanine (1956) These patients are often curiously unaware of their faulty communication and this is described as "anosognosia" for speech. Their speech is often under pressure, "logorrheic." The variability of language production in Wernicke's aphasia induced some to split the symptom complex further. Huber et al. (1975), for instance, differentiate four varieties, such as with (1) predominantly semantic paraphasias (2) semantic jargon (3) phonemic paraphasias and (4) phonemic jargon although a qualitative basis for the discrimination is not provided.

Wernicke's aphasia as described by Goodglass and Kaplan (1972) features impaired comprehension and fluently articulated, but paraphasic speech. Nouns are most often substituted by paraphasias and other substantive, informative elements of

speech are missing, in spite of the fluent use of grammatical connecting words, complex verb tenses and embedded subordinate clauses. The syntax and prosody of langauge is retained to a greater extent. Their speech is paragrammatic rather than agrammatic as in Broca's aphasia. Augmentation and pressure of speech is, at times, associated with phonemic or semantic jargon. Impaired recognition naming or word-finding difficulty and impaired reading and writing are always present. At times, the fluent paraphasic writing with repetitious phrases resembles spoken jargon (see a sample of this "graphorrhea"). Since various degrees of impairment are seen and the fringes of the entity are often ill-defined and controversial, retention of the eponym seems useful to describe this clinically valid and common aphasic impairment.

The Etiology of Aphasia:

The etiology of aphasia is one of the few areas in which general agreement can be found. Aphasic symptoms are caused by brain damage that may result from such diverse factors as cerebral vascular accidents, tumor, penetrating wounds, and other diseases that produce cerebral lesions.

Cerebral vascular accident (CVA), what the layman now knows as strokes and formerly knew as apoplexy - is the major etiology of aphasia. Over two million Americans have suffered cerebral vascular accidents. With the improved methods of medical treatment now available, more of the CVA patients survive. Many of these survivors experience aphasia. [10]

Brain damage results from a CVA, most authorities believe, because of the damage that occurs primarily from changes in the brain cells after their oxygen supply has been depleted. [11] The oxygen supply to the brain can be disrupted in three ways by CVAs: (1) a blood clot in a cerebral artery; (2) a ruptured artery; and (3) the compression of an artery.

There are four forms of CVAs that produce these disruptions of the oxygen supply to the brain: (1) thrombosis; (2) embolism; (3) hemorrhage; and (4) the

compression of a cerebral artery (Figure 1). Each of these types of CVAs reduces the blood supply to the brain, producing irreparable damage to the cerebrum's neural cells.

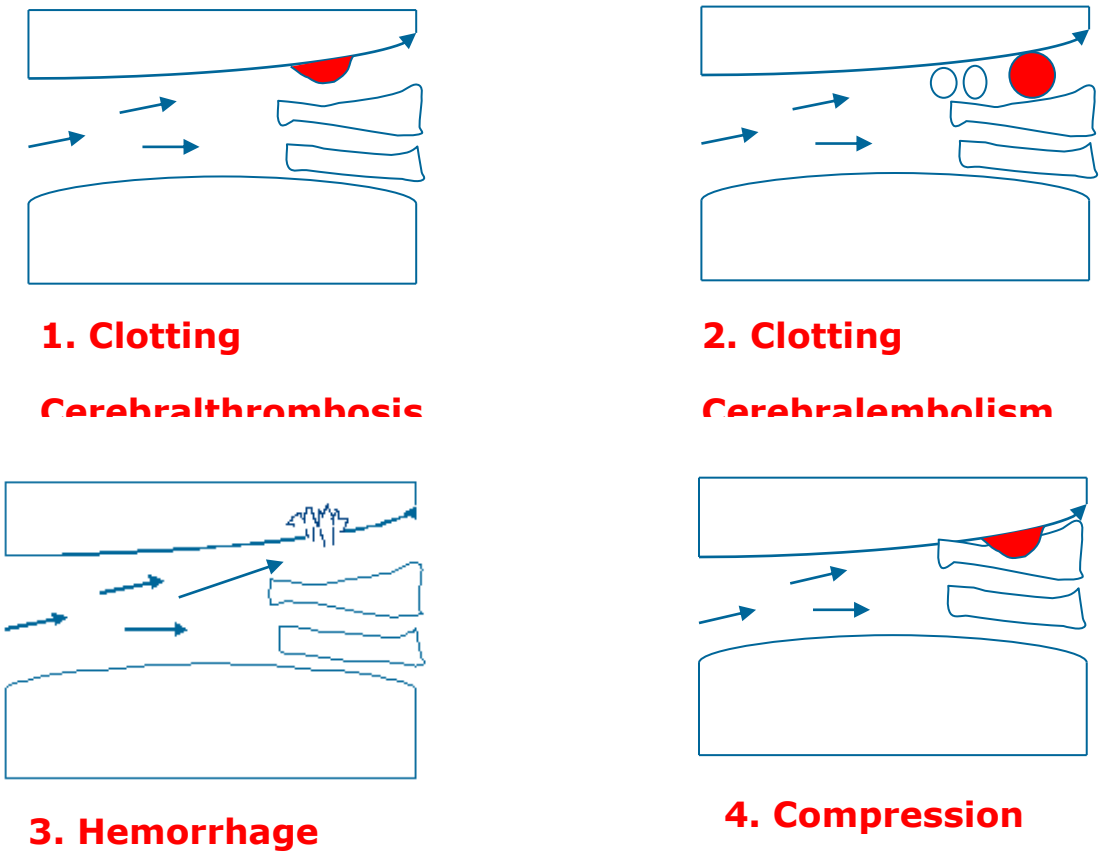


Figure 1

Thrombosis and embolism are types of blood clots that form inside a cerebral artery, thereby interfering with the blood supply to the brain. Cerebral thrombosis is the condition in which a stationary blood clot, called a thrombus, forms inside an artery obstructing the cerebral supply. Cerebral thrombosis sometimes occurs when the arterial walls, damaged by arteriosclerosis, are covered with a thick deposit that slows the blood flow by narrowing the passageway. Clots are sometimes then produced by the blood as it flows past the rough deposits that project from the arterial walls, restricting the blood flow. Cerebral embolism occurs when a blood clot, instead of remaining stationary as does the thrombus, is carried free in the blood stream. When this travelling clot, called an embolus, becomes wedged in one of the cerebral arteries,

interference with the blood supply results. The consequence, as with cerebral thrombosis, is permanent brain damage.

Cerebral hemorrhage results when a diseased artery bursts, flooding the surrounding brain tissues with blood. Hemorrhage produces two adverse effects that result in brain damage. First, neural cells dependent upon the escaped blood for nutrients and oxygen, of course, suffer irreparable damage. Second, the residual blood that flows out into the surrounding cerebral tissues may further disrupt brain function. Hemorrhage may result from various etiologies. It may result from the combination of arteriosclerosis and high blood pressure. [12] Also, aneurysms, abnormal pouch-like structures filled with blood that balloon out from the arterial walls, may burst, producing hemorrhage. Head injury, caused by a blow to the head or accident, also may produce hemorrhage.

Compression of a cerebral artery is a type of CVA produced by pressure exerted on the brain tissue or cerebral arteries. Hemorrhage, for example, may produce spilled blood that eventually forms a solid mass that exerts pressure against an artery, impairing the flow of blood. Any type of brain tumor may also press against surrounding brain tissue or artery. In each of these cases, compression serves to reduce the flow of blood to the cerebral cells, producing permanent brain injury.

Cerebral vascular accidents, as stated previously, are the chief cause of aphasia, although head wounds and injuries resulting from combat in war or highway accidents also deserve mention. Other causes of aphasic symptoms are neoplastic growths such as tumors (either benign or malignant), infectious processes such as meningitis, and degenerative diseases such as multiple sclerosis.

Classification of Aphasia :

Classifications are a necessary evil, but reviewing the classification systems of aphasia can be a discouraging task. The variety of criteria used over the past 100 years may disorient the reader at first. The diversity of the nomenclature will cause exasperation. The seeming conflict between systems that include as many as eight different varieties of aphasia and those that limit themselves to two or three will be a

source of puzzlement. Yet, the student of aphasia should realize that the diversity and conflict reflect a historical evolution of the science of the aphasias and are more apparent than real. From the practical standpoint, few of the many available classification systems have survived. Current researchers and clinicians in leading aphasiological centers use but one or two of the more recent systems. Furthermore, some of the apparently discrepant systems are not really so, since they derive from different points of view in relation to the phenomena of aphasia. For instance, Weisenburg and McBride's (1935) classic designations of EXPRESSIVE, RECEPTIVE, and MIXED aphasia reflect a clinical vantage point. Luria's (1966) nomenclature - for example, EFFERENT and AFFERENT MOTOR, or DYNAMIC - reflects a physiological approach. On the other hand, Jakobson's (1964) description of CONTIGUITY (or combination) and SIMILARITY (or selection) defects is the product of a psycholinguistic point of view. It should be clear that the systems do not conflict but rather complement each other. Be that as it may, a modern researcher or clinician should have a working knowledge of the different classification systems, from Wernicke's (1874) to Geschwind's (1965). This should be complemented with a fully conversant use of one modern classification system : the proper definition of each of its categories, their anatomical and physiological significance, and their prognostic implications.

In our opinion, the system generally associated with the Boston school of aphasia is currently the most useful one. It can be used in conjunction with most forms of laboratory and bedside assessment and does not necessarily require the use of the Boston Diagnostic Aphasia Examination (BDAE). The Boston classification comprises all of the frequently encountered syndromes for which there is an established and accepted anatomical correlation. The nomenclature utilizes a combination of eponyms, clinically descriptive terms, and physiologically based terms and is quite evocative (see Goodglass& Kaplan, 1972). The following paragraphs contain a standard description of the major syndromes, in their acute phase. But the reader should be advised that some cases will only approximate the description and will fail to manifest all the characteristics detailed here.

The Syndromes of Aphasia :

Wernicke's Aphasia

Wernicke's aphasia is the most fundamental and least controversial of aphasic syndromes. Speech is fluent and well articulated, with frequent paraphasias but preserved syntactic structure. Aural comprehension is defective. Repetition of words and sentences is defective also. In general, both reading and writing are disturbed.

Most patients present with language difficulties and may have no other evidence of neurological disease. (Right hemiparesis is infrequent and can be transient; right visual field defects are not the rule). Thus, the diagnosis rests almost solely on the signs of aphasia, and the accuracy of the diagnosis is mandatory: For the unskilled examiner, a patient with acute Wernicke's aphasia may sound "confused", with the consequence that a psychiatric rather than neurological diagnostic approach may be taken. Even assuming that the mistake is corrected eventually, the delay can be disastrous.

In our experience, patients with Wernicke's aphasia are less easily frustrated than those with Broca's aphasia. Yet, the suspicious tendency of the Wernicke patients is more evident than in Broca's aphasia, and it should be recalled than these are among the few neurological patients who can develop a major paranoid syndrome and become homicidal.

This complex syndrome, which combines both output and input disturbances, is also known as RECEPTIVE aphasia, from Weisenburg and McBride's classification (1935), and as SENSORY aphasia, as Wernicke himself called it (1874), with appreciable modesty but little physiological sense. Kleist (1934) aptly called it WORD DEAFNESS, but the term is rarely used, while Brain (1961) named it PURE WORD DEAFNESS, an inaccurate designation, since patients with Wernicke's aphasia are indeed word deaf but clearly not in pure form. (Patients with pure word deafness do exist, however; they are unable to understand speech and to repeat words but speak fluently and WITHOUT paraphasias.) Head (1926) called it SYNTACTIC aphasia, which is an ambiguous designation.

Broca'sAphasia :

The existence of Broca's syndrome is currently well established. Yet some of the major controversies in the history of aphasia have revolved around the nature and pathological correlation of Broca's aphasia. The first patient described by Broca in

1861 did not have what came to be known as Broca's aphasia, and it appears that the degree of involvement of Broca's area and of the surrounding frontal operculum produce considerably different degrees of aphasia (Mohr et al., 1978). What currently is called Broca's aphasia can be defined as the opposition of Wernicke's aphasia. The speech is nonfluent. There are few words, short sentences, and many intervening pauses, and what words there appear are produced with labor and often with distorted sounds. The melodic contour is flat. The general appearance of speech is telegraphic, due to the rather selective deletion of many connective words. On the other hand, aural comprehension is relatively intact in colloquial conversation, although formal testing often discloses a defective performance. Repetition of words and sentences is impaired.

Unlike patients with Wernicke's aphasia, the patient with Broca's aphasia invariably presents with a right-sided motor defect (often a complete hemiparesis more marked in the upper extremity and face). As a consequence, patients with Broca's aphasia are less vulnerable to mis-diagnosis. Their presentation is clearly neurological. On the other hand, they are often depressed and respond to testing failures with ''catastrophic'' reactions (sudden weeping and refusal to proceed with examination) more frequently than do Wernicke's aphasics.

Broca's aphasia has also been known as EXPRESSIVE (Weisenburg& McBride, 1935) and MOTOR (Goldstein, 1948; Wernicke, 1874). For a time it was refused the status of aphasis and called ANARTHRIA (Marie, 1906), and, later, DYSARTHRIA (Bay, 1962). Head (1926) called it VERBAL aphasia.

Conduction Aphasia:

The speech of conduction aphasics is fluent although usually less abundant than that of Wernicke's. There are commonly minor defects in aural comprehension, though comprehension of colloquial conversation is intact. But it is the impairment in repetition of words and sentences that dominates this syndrome. The defect takes many forms. Most commonly, patients repeat words with phonemic paraphasias, but often they will omit or substitute words, and they may fail to repeat anything at all if function words rather than nouns are requested. Comprehension of the defectively repeated sentences is good. Similarly, patients comprehend the sentences that they

read aloud with numerous paraphasias. (This TRANSCODING performance from reading to oral expression is a form of repetition.)

Conduction aphasics often have some accompanying motor signs (paresis of the right side of the face and of the right upper extremity), but recovery is good. The syndrome has been known as CENTRAL aphasia, Goldstein's (1948) curious designation, and as AFFERENT MOTOR, Luria's term. Luria attempted to break down the syndrome, giving it a motor component (AFFERENT MOTOR) and an auditory one (ACOUSTIC AMNESIC). Kertesz (1979) proposed a comparable distinction (EFFERENT CONDUCTION and AFFERENT CONDUCTION).

Transcortical Sensory Aphasia (TSA):

Patients with TSA have fluent and paraphasic speech (global paraphasias predominate over phonemic ones) and a severe impairment in aural comprehension. Yet their repetition is intact (occasionally echolalic), setting them clearly apart from Wernicke's aphasics. The distinction of the syndromes is important since the localization of the lesion is different (see Chapter 2 on localization). This underscores the need to test repetition is every aphasic patient.

TRANSCORTICAL was the original designation of Goldstein, and it has held well through the years, both for TSA and for transcortical motor aphasia, some cases of which Luria preferred to call DYNAMIC aphasia (Luria &Tsevtkova, 1968).

Transcortical Motor Aphasia (TMA)

Patients with TMA have intact repetition, just as patients with TSA, and can have echolalia too. But the speech is nonfluent and troubled by phonemic and global paraphasias, perseveration, and loss of connective words. In our experience, auditory comprehension is impaired too when tested formally, although patients can often carry on a simple conversation at bedside.

Patients with TMA should be distinguished from those with mutism on several counts. Firstly, patients with TMA are inclined to communicate and do so, within their verbal limitations. Patients with mutism do not and are as impoverished in nonverbal as in verbal communication. Secondly, the speech of TMA is clearly aphasic; for example, there are unquestionable phoentic, lexical, and syntactical errors, whereas

patients with mutism either produce no speech at all or utter a few short but linguistically correct sentences. Again, the distinction is important because the localization of the lesion is different.

Global Aphasia:

As the name implies, global aphasics present with an almost complete loss of ability to comprehend or formulate verbal communication. Propositional speech may be reduced to a few words, the remainder of verbal communication consisting of emotional exclamations and serial utterances. Auditory comprehension is often reduced to a variable number of nouns and verbs, while the comprehension of functor words or of syntactically organized sentences is virtually negligible.

Anomic Aphasia:

Nominal aphasia (also known as *anomic aphasia*) is a form of aphasia (loss of language capability caused by brain damage) in which the subject has difficulty remembering or recognizing names which the subject should know well. The subject speaks fluently, grammatically, has normal comprehension, and the only deficit is trouble with "word finding," that is, finding appropriate words for what they mean to say.

Subjects often use circumlocutions (speaking in a roundabout way) in order to express a certain word for which they cannot remember the name. Sometimes the subject can recall the name when given clues. Sufferers are often frustrated when they know they know the name, but cannot produce it.

"Hold on, I should know the name of that thing... Give me a minute ..."

Sometimes subjects may know what to do with an object, but still not be able to give a name to the object. For example, show a subject an orange, and ask what it's called. The same subject may be well aware that the object can be peeled and eaten, and may be able to demonstrate this by actions or even

verbal responses. Whether such a subject could name the *color* of the orange is unknown.

Anomia is caused by damage to various parts of the parietal lobe or the temporal lobe of the brain. This type of phenomenon can be quite complex, and usually involves a breakdown in one or more pathways between regions in the brain. The responses may also differ depending on whether objects are shown in the right or left hand side of the visual field.

"Averbia" is a specific type of anomia in which the subject has trouble remembering only verbs. This is caused by damage to the frontal cortex, in or near Broca's area.

Another type of anomia is "color anomia", where the patient can distinguish between colors but cannot identify them by name.

Alexia with Agraphia:

The pure syndrome of alexia with graphia is rare if at all existent. More often than not, patients have signs of Wernicke's aphasia or of transcortical sensory aphasia. In the absence of aphasia, they generally have notable parietal lobe signs. But it is reasonable to make the diagnosis of alexia with agraphia when the disturbances of reading and writing predominate over the aphasic or parietal symptomatology. The fact that this syndrome can be associated with impaired as well as intact repetition, and with a greater or smaller extent of accompanying signs, suggest that a large segment of parietal and temporal lobe structures, cortical and subcortical, is engaged in the complex processes of reading and writing. Therefore, the anatomical significance of this entity is considerably smaller than that of most aphasic syndromes or of the syndrome of alexia WITHOUT agraphia (pure alexia).

Alexia without Agraphia (Pure Alexia):

As the designation implies, patients presenting alexia without agraphia become unable to read while they continue to be able to write, spontaneously or to dictation. (Many such patients can also copy writing, although they do so with difficulty.)

Speech, auditory comprehension, and repetition are intact. Oral spelling of words (or its converse, the construction of words spelled orally) is normal. Reading in the tactile mode is normal, too. Whatever visual reading they can do is of single letters. (This often allows the patient to read aloud the letters of a word, one by one, and then reconstitute the word from this operation of spelling.)

Most patients have some form of accompanying visual function impairment. It can be a right homonymous hemianopia (the field of vision to the right of the vertical median is blind) or else a right hemiachromatopsia (loss of color perception without true blindness in the right hemifield). Most patients also have color anomia (a disturbance of naming colors with otherwise normal color perception). Some present with optic ataxia (a disturbance in the visual guidance of hand movements).

First described by Dejerine (1892), the syndrome was long forgotten and even denied but was revived by Geschwind (1965), who used it as a cornerstone for his theory of disconnection syndromes.

Agrammatism

- UsuallyassociatedwithBroca'saphasia
- Generallypresent in Broca'saphasia
- But other aphasics also have grammatical dysfunctions
    - Paragrammatism – common in Wernicke aphasia
- A lot of variation among different patients

Agrammatism vs. Paragrammatism

- Paragrammatism – toomuch speech
    - Normal or excessive fluency
    - Use of inappropriatewords
    - Neologisms
    - No lack of function words and inflections
        - But not alwaysusedappropriately
    - Common in Wernicke'saphasia

- Agrammatism – not enough speech
    - Lack of fluency
    - Omission (NOT deletion!) of function words and inflections
    - Common in Broca'saphasia

Omission vs. Deletion

- Goodglass:
    - Sentences with a deleted main verb ("Joan and I . . . Coffee") may continue to appear.
    - . . . misuse or deletions of morphology . . .
- Is he talking about deletion or omission?
- Deletion implies that it was first there, and then removed
- Omission – it wasn't put in at all
- Goodglass is following a practice that was common among linguists at the time he wrote the book

Agrammatism

- Damage to frontal lobe
    - Mainly, inferior frontal gyrus
- Largely intact comprehension
- Nonfluent, agrammatic speech
- "Telegraphic speech" –
    - Abundance of content words (e.g., nouns)
    - Lack of function words (e.g. prepositions)
- Impairedverbprocessing
    - Bates, Chen, Tzeng, Li & Opie, 1991; Damasio&Tranel, 1993; Daniele, Giustolisi, Silveri, Colosimo&Gainotti, 1994; Lamb & Zhang, 2010; Shapiro &Caramazza, 2003
- Patients can readily point to individual objects or body parts named by the examiner

- But when asked to point to the same items in a specific sequence they often fail at the level of only two or three items

Agrammatism: an early observation (1819)

- Deleuze (1819), referring to a French-speaking patient: The patient "used exclusively the infinitive of verbs and used no pronouns. … Sheproducedabsolutely no conjugatedverb."

Example of agrammatic speech

Examiner: Can you tell me about why you came back to the hospital?

Patient: yes … eh … Monday … eh … dad … Peter Hogan and dad … hospital.

Er … two … er … doctors … and … er … thirty minutes … and … er … yes … hospital. And … er … Wednesday … Wednesday. Nine o'clock. And … er … Thursday, ten o'clock … doctors … two … two … doctors… and … er … teeth … fine.

E: Not exactly your teeth … your g-

P: Gum … gum …

E: What did they do to them?

P: And er … doctor and girl … and er … and er gum …

Somefeatures of agrammatism

- Telegraphic speech
    - Short utterances
    - Omission of grammatical functors
- Relative abundance of substantives
- Verbs are uncommon, rare in some patients
    - When present, uninflected or *–ing* form
        - For French aphasics, infinitive form
- Use of word order is generally spared
- Comprehension is impaired for complex sentences

Problems in the study of agrammatism

- Must bedistinguishedfromparagrammatism
- Grammatical aberrations – even among Broca aphasics – vary from patient to patient
- Linguistics has not (yet) provided clear answers to important basic questions:
    - What normal grammatical functions are
    - How theyoperate

Syntax

- First, we need to dispel the notion that syntax is one capacity, that can be lost (or spared) as a unit
- Syntax can be understood as a set of constructions
    - Learned by children (and others) one by one
        - Likevocabulary
    - Some can be lost, others spared, in aphasia
- It is a label of the grammarian for multiple things
- Word order is often spared in Broca's aphasia while a lot of syntax is lost

Stability of word order in agrammatism

- Agrammatic patients can usually handle word order in both production and comprehension
- Evidence (comprehension)
    - passive sentences misconstrued
    - *The horse was kicked by the dog*
        - Broca'saphasic: horse as kicker
        - Passive marker not apprehended
    - Canonical word order guides the interpretation
        - Possiblyaided by conceptualknowledge

Reading and writing in agrammatism

- Agrammatic difficulties are also seen in
    - Oral reading
    - Writing to dictation

    - Repetition
- But:
    - *Some* patients are agrammatic in speech but not in writing (Goodglass 1993: 110)
    - *Some*canrepeatcorrectly
    - How to explain?
- Menn&Obler (1990) describe some patients who are less agrammatic in oral reading than in spontaneous speech (Goodglass 1993:111)
- Syntax and morphology (study of agrammatic French aphasics)
    - Some patients have fairly good syntax but defective morphology
    - Some patients have fairly good morphology but defective syntax
    - Both types of patients fail to use inflected verb forms
- Gleason et al. observations (1975)
    - Some patients use *–s* plural marker but not articles
    - Other patients use articles but not *–s* plural marker

Loss of the use of relational markers in receptive agrammatism

- E.g. *father'ssister*
    - Ex: Is "my father's sister" a man or a woman?
    - Patient answersrandomly
        - Unable to grasp the relational function of *–'s*
- Command given in testing:
    - Ex: Touch the comb with the pencil
    - Patient may touch the pencil with the comb
        - Perhaps picks up comb because the word *comb* comes first in the instruction
- Locative relations somewhatless fragile
    - *in back of/in front of, over/under, before/after*

UNIT – IV

Speech Disorders

Speech disorders are the disorders, in which the speech mechanisms like soft palate, tongue, lips, etc are the locus of delay. They can be further classified into;

1. **Articulation disorders:**

It is a disorder due to the problem that occurs in movement of various structures of speech mechanism such as soft palate, tongue, lips, etc. This can be classified into 1. Functional – which have no specific cause, 2. Organic – which occurs due to structural abnormalities like **cleft palate** and **cerebral palsy**, and 3. others – which occurs due to any other speech and language disorders such as hearing loss, mental retardation, etc.

2. Fluency disorders:

Communication needs smooth and easy flow of the utterances. Thus the effortless and continuous speech with the rapids speed is called fluency. So if problem persists in the above said effort, continuity and speed then it is said to be a fluency disorders. For example, **stuttering and cluttering**.

3. Voice disorders:

If the pitch, loudness or quality of the voice differs from that of the normal / standard pitch, loudness and quality then that voice is said to be a disordered voice. For example, **dysphonia.**

Cleft lip and Cleft palate

Cleft lip and cleft palate are facial and oral malformations that occur very early in pregnancy, while the baby is developing inside the mother. Clefting results when there is not enough tissue in the mouth or lip area, and the tissue that is available does not join together properly.

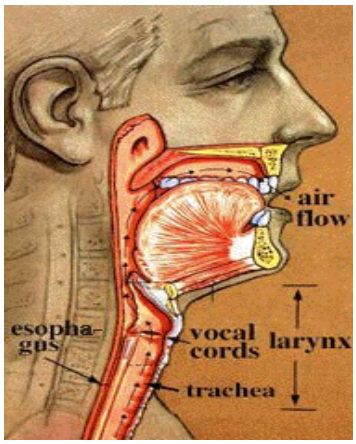
A cleft lip is a physical split or separation of the two sides of the upper lip and appears as a narrow opening or gap in the skin of the upper lip. This

separation often extends beyond the base of the nose and includes the bones of the upper jaw and/or upper gum.

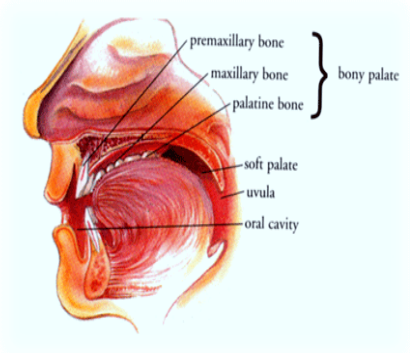
A cleft palate is a split or opening in the roof of the mouth. A cleft palate can involve the hard palate (the bony front portion of the roof of the mouth), and/or the soft palate (the soft back portion of the roof of the mouth).

Cleft lip and cleft palate can occur on one or both sides of the mouth. Because the lip and the palate develop separately, it is possible to have a cleft lip without a cleft palate, a cleft palate without a cleft lip, or both together.

Disorders involving cleft lip and palate involve the lips, hard palate, and the velum



PALATE



Anterior 2/3 is the hard palate

- Stationary
- Purpose is to separate the oral and nasal cavities

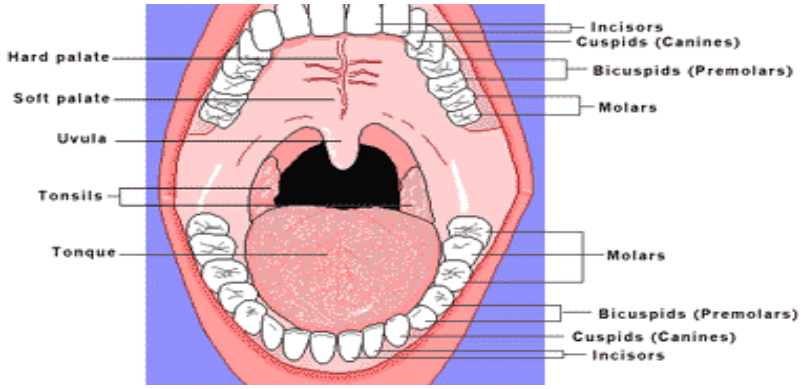
Posterior 1/3 is the soft palate

- Mucosal tissue and muscles

Velopharyngeal Mechanism

Components

1. Velum
2. Muscles in the back of the throat



The Purpose of the Velum

The velum needs to be closed and the oral and nasal cavities separated when we swallow and during the production of most English speech sounds

Cleft Lip

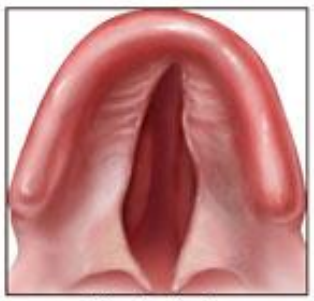
The lip forms between the fourth and seventh weeks of pregnancy. A cleft lip happens if the tissue that makes up the lip does not join completely before birth. This results in an opening in the upper lip. The opening in the lip can be a small slit or it can be a large opening that goes through the lip into the nose. A cleft lip can be on one or both sides of the lip or in the middle of the lip, which occurs very rarely. Children with a cleft lip also can have a cleft palate.

Baby with cleft lip

Cleft Palate

The roof of the mouth (palate) is formed between the sixth and ninth weeks of pregnancy. A cleft palate happens if the tissue that makes up the roof of the mouth does not join together completely during pregnancy. For some babies, both the front and back parts of the palate are open. For other babies, only part of the palate is open.

Baby with cleft palate



Cleft palate

The causes of orofacial clefts among most infants are unknown. Some children have a cleft lip or cleft palate because of changes in their genes. Cleft lip and cleft palate are thought to be caused by a combination of genes and other factors, such as things the mother comes in contact with in her environment, or what the mother eats or drinks, or certain medications she uses during pregnancy.

Recently, CDC reported on important findings from research studies about some factors that increase the chance of having a baby with an orofacial cleft:

- Smoking—Women who smoke during pregnancy are more likely to have a baby with an orofacial cleft than women who do not smoke.[2-3]
- Diabetes—Women with diabetes diagnosed before pregnancy have an increased risk of having a child with a cleft lip with or without cleft palate, compared to women who did not have diabetes.[5]
- Use of certain medicines—Women who used certain medicines to treat epilepsy, such as topiramate or valproic acid, during the first trimester (the first 3 months) of pregnancy have an increased risk of having a baby with cleft lip with or without cleft palate, compared to women who didn't take these medicines

Do children with cleft palate have speech problems?

It is common for children who are born with a cleft palate to have speech problems at some time in their lives. Over half of them will require speech therapy at some point during childhood. However, many children who are born with a cleft palate develop normal speech by the age of 5.

Regular evaluations by the speech-language pathologist on your child's cleft team will help you decide if speech therapy services or other types of interventions are needed. The speech-language pathologist will be able to assess your child's speech production and language development and make appropriate therapy recommendations.

It is also extremely important for your child's ears and hearing to be examined on a regular basis, whether you suspect a problem or not. Children with cleft palate are more susceptible to ear infections (otitis media) than children without clefts. Children with many ear infections are at risk for hearing loss,

language delays, and speech problems because they are not able to hear language normally when fluid collects in the middle ear.

What speech problems might children with clefts have?

Before the palate is repaired, there is no separation between the nasal cavity and the mouth. This means that a) the child cannot build up air pressure in the mouth because air escapes out of the nose, and b) there is less tissue on the roof of the mouth for the tongue to touch. Both of these problems can make it difficult for the child to learn how to make some sounds.

It is not unusual for a child who is born with a cleft palate to show a delay in both the onset of speech and the development of speech sounds during the first 9-24 months of age. Therefore, it is important to talk to your child and to encourage your child to talk to you. Once the palate has been repaired, your child may be able to learn more consonant sounds and say more words, but speech may still be delayed during the early years. Articulation problems (difficulties in making certain sounds) may persist in some children throughout early childhood for a variety of reasons. If your child's teeth do not "line up" correctly, speech may be understandable, but some sounds (like "s" or "sh") may sound distorted or "mushy." It is also important to remember that some children, with or without a cleft palate, may simply develop speech more slowly than others.

When speech is produced correctly, the soft palate lifts and moves toward the back of the throat, separating the nasal cavity from the mouth so that air and sound can be directed out of the mouth. The inability to close off the nasal cavity from the mouth is called velopharyngeal inadequacy. Children who have velopharyngeal inadequacy may sound like they are "talking through their noses." This problem occurs because when the soft palate cannot close off the nose from the mouth, air and sound can escape through the nose during speech, possibly resulting in hypernasality and nasal emission of air. (It is normal for air and sound to come out of the nose when saying the "m," "n," or

“ing” sounds.) Approximately 25% of children with repaired cleft palates still show signs of velopharyngeal inadequacy.

You may notice that your child produces “grunt” or “growl” sounds. These sounds represent a behavior that some children learn in an attempt to compensate for velopharyngeal inadequacy. This behavior usually begins before the palate is repaired, but it may continue even after the palate is closed. Although it can be corrected by speech therapy, you should not encourage your child to make these types of sounds, because it can be difficult for him or her to “unlearn” this habit.

Children with velopharyngeal inadequacy may also have a voice disorder. In this case, your child’s voice may sound hoarse or “breathy” and may fatigue easily. This problem is usually caused by the strain that he or she puts on the vocal cords while trying to build the pressure necessary for normal speech.

Communicative problems associated with clefts:

About 80% of people born with clefts that aren’t associated with a syndrome and receive palatal repair by 18 mo can expect reasonably good speech w/out intervention. The other 20% can have extensive communication problems. Therapy is often long-term and prognosis is variable.

Resonance problems:

Because of the VPI, they have “hypernasal” voice quality. This is a resonance disorder, not a voice disorder and comes about because the nasal cavity isn’t separated from the oral cavity during speech. They talk thru their nose.

Nasality in speech really falls along a continuum from hyponasality (lack of nasal resonance) thru hypernasality (too much nasal resonance and not enough oral resonanace).

Play the tape example of samples of speech representing a continuum ranging from hyponasality thru very severe hypernasality.

People with VPI exhibit hypernasality of varying degrees. It's associated mainly with vowel sounds, especially in conversational (connected) speech.

Articulation problems:

The result of VPI, structural deviations in the oral cavity, dental problems and anamolies, faulty learning. Phonemes that tend to be misarticulated the most include /s/, /z/, /th/, /ch/, and /ts/.

Some phonemes that require a considerable build-up of air pressure in the oral cavity (stop consonants) are especially problematic because air will escape thru the nose(nasal emission). This isn't the same thing as hypernasality. Nasal emission is audible release of air thru the nose.

Compensatory articulation errors—this is a gross sound substitution error that is an attempt to compensate for the physical inability to produce a given sound correctly. A common one is a glottal stop

Voice problems:

Most common vocal pathology is vocal nodules, which produce a hoarse, breathy vocal quality. The nodules are thought to arise from "vocal hyperfunction" due to screaming, yelling, etc in non-cleft kids. The larynx is moved high in the neck.. This is a compensatory strategy in cleft-kids to help increase their volume.

examples of some voice disorders

Some illustrate mild and severe degrees of hoarseness

Some illustrate extreme vocal tension and the use of inhalation tension.

What can be done about speech problems?

Speech therapy alone may be able to correct your child's speech disorder. Therapy can be extremely effective for children with mild hypernasality, an articulation disorder, or speech delay. The goal of speech therapy will be to develop good speech habits as well as to learn how to produce sounds correctly. Speech therapy alone will generally NOT correct hypernasality that is caused by moderate to severe velopharyngeal inadequacy.

The type of therapy your child receives will be determined by the type of problem your child has. Furthermore, the amount of therapy your child needs will depend on the severity of the speech problem. If your child's articulation difficulties are related to a dental abnormality, the combination of articulation therapy and dental treatment can help to minimize the problem.

If your cleft palate team decides that speech therapy alone will not correct your child's speech problem, there are some other options. Your child may require another palate surgery to help with speech. The two most common speech surgeries are 1) pharyngeal flap and 2) sphincter pharyngoplasty. (The surgeon may also choose to redo the original repair.) Your child's speech-language pathologist and surgeon will work together to determine the most appropriate type of surgery for your child. Talk to your surgeon about which procedure he or she intends to perform. It is important to remember that surgery is not a "quick fix." It is almost always necessary for a child to participate in speech therapy after surgery to practice correct articulation and good speech habits.

Although surgery is the most frequently-chosen approach for improving velopharyngeal function, a prosthetic device may be an option for some patients. These speech aids are placed in the mouth, much like an orthodontic retainer. The two most common types are 1) the speech bulb and 2) the palatal lift. The speech bulb is designed to partially close off the space between the soft palate and the throat. The palatal lift appliance serves to lift the soft palate to a position that makes closure possible. Many professionals feel that prosthetic appliances work best in children who are at least five years of age. However,

each patient should be evaluated on an individual basis to determine if one of these devices is appropriate for him or her.

Fluency disorders:

Fluency disorders are one of the speech disorders. Communication needs smooth and easy flow of the utterances. Thus the effortless and continuous speech with the rapids speed is called fluency. So if problem persists in the above said effort, continuity and speed then it is said to be a fluency disorders. So, fluency needs 3 basic parameters they are, 1. effort, 2. continuity and 3. rate of speech.

Effort – refers to both mental and physical effort. Mental effort is coding process and physical effort is muscular efforts of the phonatory, respiratory and laryngeal systems

Continuity – refers to a smooth movement from phoneme to phoneme, between syllables, across words, and from phrase to phrase.

Rate of speech – refers to number syllables uttered per second or number of words uttered per minute.

The persons with fluency disorders used to have problem at least in any one of the above. For example, **stuttering and cluttering**.

Stuttering

Stuttering is a speech disorder in which sounds, syllables, or words are repeated or prolonged, disrupting the normal flow of speech. These speech disruptions may be accompanied by struggling behaviors, such as rapid eye blinks or tremors of the lips. Stuttering can make it difficult to communicate with other people, which often affects a person's quality of life.

Symptoms

Symptoms of stuttering can vary significantly throughout a person's day. In general, speaking before a group or talking on the telephone may make a person's stuttering

more severe, while singing, reading, or speaking in unison may temporarily reduce stuttering. Stuttering is sometimes referred to as *stammering* and by a broader term, *disfluent* speech.

Stuttering affects people of all ages. It occurs most often in children between the ages of 2 and 5 as they are developing their language skills. Approximately 5 percent of all children will stutter for some period in their life, lasting from a few weeks to several years. Boys are twice as likely to stutter as girls; as they get older, however, the number of boys who continue to stutter is three to four times larger than the number of girls. Most children outgrow stuttering. About 1 percent or less of adults stutter.

Causes for stuttering

Although the precise mechanisms are not understood, there are two types of stuttering that are more common. (A third type of stuttering, called psychogenic stuttering, can be caused by emotional trauma or problems with thought or reasoning. At one time, all stuttering was believed to be psychogenic, but today we know that psychogenic stuttering is rare.)

1. **Developmental stuttering**

Developmental stuttering occurs in young children while they are still learning speech and language skills. It is the most common form of stuttering. Some scientists and clinicians believe that developmental stuttering occurs when children's speech and language abilities are unable to meet the child's verbal demands. Developmental stuttering also runs in families. In 2010, for the first time, NIDCD researchers isolated three genes that cause stuttering. More information on the genetics of stuttering can be found in the research section of this fact sheet.

2. **Neurogenic stuttering**

Neurogenic stuttering may occur after a stroke, head trauma, or other type of brain injury. With neurogenic stuttering, the brain has difficulty coordinating the different components involved in speaking because of signaling problems between the brain and nerves or muscles.

Diagnosis

Stuttering is usually diagnosed by a speech-language pathologist (SLP), a health professional who is trained to test and treat individuals with voice, speech, and language disorders. The speech-language pathologist will consider a variety of factors, including the child's case history (such as when the stuttering was first noticed and under what circumstances), an analysis of the child's stuttering behaviors, and an evaluation of the child's speech and language abilities and the impact of stuttering on his or her life.

When evaluating a young child for stuttering, a speech-language pathologist will try to predict if the child is likely to continue his or her stuttering behavior or outgrow it. To determine this difference, the speech-language pathologist will consider such factors as the family's history of stuttering, whether the child's stuttering has lasted six months or longer, and whether the child exhibits other speech or language problems.

Speech or Language Problems

1. An abnormal amount of segment, syllable, word or phrase repetition, as in p- p- p- please, the pol-pol- policeman, I think he's got a - got a - got a -.

2. Obstructions to the air-flow, due to the inability to release the tension that has built up in preparing to articulate a sound: on a tape, the result is a long pause; but face to face there may be many signs of the struggle that is going on within for control-facial spasms and grimaces, sudden movements of the head, or of the whole body, and awkward gestures. This phenomenon is referred to as blocking, and the individual sounds affected as blocks or hard contacts.

3. Abnormal prolongations of sound segments, as in *f-f-f-feels*, where the initial *f* is being produced as a single lengthened sound, and not with brief pauses between (cf. (1) above).

4. The introduction of extra words or sounds with a solely emotional force at points of difficulty, e.g. gash, oh, tut.

5. Erratic stress patterns in words, and abnormal intonation and tempo patterns on sentences, mainly because of the very hesitant speech, and the accompanying irregular breathing.

6. Words being left unfinished.

7. Awkward circumlocutions; the stutterer may know very well which types of sound cause him particular problems, and will therefore go out of his way (linguistically) to avoid saying them - sometimes to quite surprising lengths; one stutterer, in the middle of a story, said '..... and lying in the road was one of those - animals that meow.....'

Treatment for stuttering

Although there is currently no cure for stuttering, there are a variety of treatments available. The nature of the treatment will differ, based upon a person's age, communication goals, and other factors. If you or your child stutters, it is important to work with a speech-language pathologist to determine the best treatment options.

For very young children, early treatment may prevent developmental stuttering from becoming a lifelong problem. Certain strategies can help children learn to improve their speech fluency while developing positive attitudes toward communication. Health professionals generally recommend that a child be evaluated if he or she has stuttered for three to six months, exhibits struggle behaviors associated with stuttering, or has a family history of stuttering or related communication disorders. Some researchers recommend that a child be evaluated every three months to determine if the stuttering is increasing or decreasing. Treatment often involves teaching parents about ways to support their child's production of fluent speech.

Parents may be encouraged to:

Provide a relaxed home environment that allows many opportunities for the child to speak. This includes setting aside time to talk to one another, especially when the child is excited and has a lot to say.

Refrain from reacting negatively when the child stutters. Instead, parents should react to the stuttering as they would any other difficulty the child may experience in life. This may involve gentle corrections of the child's stuttering and praise for the child's fluent speech.

Be less demanding on the child to speak in a certain way or to perform verbally for people, particularly if the child experiences difficulty during periods of high pressure.

Speak in a slightly slowed and relaxed manner. This can help reduce time pressures the child may be experiencing.

Listen attentively when the child speaks and wait for him or her to say the intended word. Don't try to complete the child's sentences. Also, help the child learn that a person can communicate successfully even when stuttering occurs.

Talk openly and honestly to the child about stuttering if he or she brings up the subject. Let the child know that it is okay for some disruptions to occur.

Cluttering

A speech defect where the speech is rapid, disordered and lacks fluency. It is a common occurrence in children during their initial speech development when they find difficulty finding the right words to express themselves. Speech of the clutterers is difficult for listeners to understand due to rapid speaking rate, erratic rhythm, poor syntax or grammar, and words or groups of words unrelated to the sentence.

Symptoms

- Repeating part of sentence
- Repeating words
- Disordered speech
- Difficulty finding words
- Fast talking
- Talking in spurts

Like stuttering, cluttering is a fluency disorder, but the two disorders are not the same. Cluttering involves excessive breaks in the normal flow of speech that seem to result from disorganized speech planning, talking too fast or in spurts, or simply being unsure of what one wants to say. By contrast, the person who stutters typically knows exactly what he or she wants to say but is temporarily unable to say it. To make matters even more confusing, since cluttering is not well known, many who clutter are described by themselves or others as "stuttering." Also, and equally confusing, cluttering often occurs along with stuttering.

The definition of cluttering adopted by the fluency disorders division of the American Speech-Language-Hearing Association is: Cluttering is a fluency disorder characterized by a rapid and/or irregular speaking rate, excessive disfluencies, and often other symptoms such as language or phonological errors and attention deficits. To identify cluttering, you must listen to nonstuttered speech of the speaker. Evidence for a fluency disorder (one that is not stuttering) and excessive disfluencies, would be present in a speaker who meets all of the following:

- Does not sound "fluent," that is, does not seem to be clear about what he or she wants to say or how to say it.
- Has excessive levels of "normal disfluencies," such as interjections and revisions.
- Has little or no apparent physical struggle in speaking.
- Has few if any accessory (secondary) behaviors.

A rapid and/or irregular speaking rate would be present in a speaker who has any or all of the following:

- Talks "too fast" based on an overall impression or actual syllable per minute counts.
- Sounds "jerky."
- Has pauses that are too short, too long, or improperly placed.

These fluency and rate deviations are the essential symptoms of cluttering. In addition, however, there are a number of symptoms suggested in the latter part of the above definition that may or may not be present but add support to the impression

that a person is cluttering. Accordingly, the clinical picture of a typical cluttering problem would be enhanced if the person in question had any of the following:

- Confusing, disorganized language or conversational skills.
- Limited awareness of his or her fluency and rate problems.
- Temporary improvement when asked to "slow down" or "pay attention" to speech (or when being tape recorded).
- Mispronunciation or slurrring of speech sounds or deleting non-stressed syllables in longer words (e.g., "ferchly" for "fortunately").
- Speech that is difficult to understand.
- Several blood relatives who stutter or clutter.
- Social or vocational problems resulting from cluttering symptoms.
- Learning disability not related to reduced intelligence.
- Sloppy handwriting.
- Distractibility, hyperactivity, or a limited attention span.
- Auditory perceptual difficulties.

Cluttering versus stuttering

Cluttering and stuttering sound very similar to the lay ear, especially when they are at their worst. However, they are markedly different disorders and clutterers and stutterers are very different.

Stutterers:

- Are very aware of their disorder
- Perform worse when speaking under stress
- Have a hard time fluently giving short answers
- Often have inhibited, neat handwriting
- Therapy focuses on relaxation techniques, calling attention away from speech
- Typically were fluent, but then started stuttering
- Know exactly what they want to say but cannot say it
- Have organized speech

- Have good listening skills

Clutterers:

- Are very unaware of their disorder
- Perform better when speaking under stress
- Have a hard time fluently giving long answers
- Have hasty, repetitious, uninhibited, messy handwriting
- Have little to no fear of their speech and are careless in speech
- Therapy focuses on calling attention to speech details
- Are typically outgoing or extroverted
- Typically were never very fluent
- Do know exactly what they want to say, but it becomes disorganized while actually speaking
- Have disorganized, tangential, grammatically incorrect speech with word substitutions
- Are impatient listeners, frequently interrupt, and have poor turn-taking skills in conversation

Dysarthria

Dysarthria is defined as a difficulty in articulating speech due to weakening or dysfunction of the muscles of the mouth, face, and/or respiratory system, specifically due to brain injury. It is a speech disorder resulting from neurological injury. Any of the speech subsystems (respiration, phonation, resonance, prosody, articulation and movements of jaw and tongue) can be affected.

Symptoms of dysarthria may include slurred speech; speaking softly or barely in a whisper; slowed speech or rapid, mumbled speech; restricted movement of the tongue, lips, and jaw; abnormal rhythm of speech; or altered vocal quality (the speaker may sound stuffy, nasal, hoarse, or breathless). Dysarthria may also include chewing or swallowing difficulty or drooling and poor control of saliva. Here the speech is affected due to some disorder in the nervous system, which in turn hinders control over for example tongue, throat, lips or lungs. Swallowing problems, dysphagia, are often present.

Cranial nerves that control these muscles include the facial nerve (VII), the glossopharyngeal nerve (IX), the vagus nerve (X), and the hypoglossal nerve (XII). Problems may present in one or more nerves specified here.

Dysarthria often accompanies other disorders, such as cerebral palsy, Parkinson's disease, amyotrophic lateral sclerosis (also called Lou Gehrig's disease), Huntington's disease, and multiple sclerosis.

Treatment of Dysarthria

Treatment depends on its cause and the precise symptoms that develop. Therapy is used to help patients articulate more clearly and loudly through exercises in breath support and muscle strengthening or alterations to the speed of speech. Therapy may focus on increasing movement of the mouth, tongue, and lips. Dysarthria does not impair an individual's ability to comprehend language or to form coherent expressions. The problem is strictly due to an inability to control the muscles needed to form speech. Therefore, if these strategies do not sufficiently improve communication, people may use sign language, communicate by writing their ideas, or use electronic or computer-based devices to aid them in expressing themselves. These devices write and produce language.

Dysphonia

Voice disorders characterized by hoarseness, weakness or even loss of voice are called dysphonia. In every human voice box or larynx is a framework of cartilage containing vocal cords. These structures vibrate to produce the sound of one's voice.

Spasmodic dysphonia is a voice disorder characterized by intermittent, involuntary tightening or constriction of the larynx (voice box) during phonation. The interruption of air flow results in staccato, jerking, labored speech. Occasionally, vocal spasms can abduct or separate the vocal folds resulting in breathy voice breaks (abductor spasmodic dysphonia).

Causes

Overuse, emotional stress, trauma and illness can cause chronic spasm, weakness, or scarring of the vocal cords. Sometimes trouble stems from paralysis that damages nerves that move the vocal cords. The injury interferes with the smooth passage of air over the cords, making voice production less efficient and altering the sound produced.

Chronic dysphonia can occur in people such as teachers who use their voices a lot, and in people who have experienced trauma or surgery that affects the larynx. It can also start out as a virus that causes chronic laryngitis. In some cases, gastro esophageal reflux can cause chronic hoarseness.

Diagnosis

Some voice changes can signal disorders such as vocal cord polyps or the onset of cancer or other diseases, so that it is important to pinpoint the source of the problem promptly. To do so, the physician can refer you to an ear, nose and throat specialist who can perform a diagnostic procedure called indirect or direct laryngoscopy, or video-laryngoscopy.

Treatment

Conservative treatment includes avoiding vigorous use of the voice (singing, shouting) and throat lozenges. Acute laryngitis from a virus should resolve on its own. Acute laryngitis from bacteria may be improved with an antibiotic. In gastroesophageal reflux disease, treatment with an acid blocker may be effective. Some cases of dysphonia can require surgical intervention.

UNIT-V

Assessment

Identifying the existence of a language problem is the first step in the assessment process. When professionals use the term 'assessment' they are generally concerned with a more detailed and systematic set of investigations that attempt to specify the nature of the child's language difficulties and possibly identify factors that may have caused the problems. These more formal assessments usually involve the use of some set of tests. Often assessments have the aim of classifying a child's difficulties to allow for a more precise description and analysis. Researchers will then want to carry out further assessments to investigate their hypotheses about the nature and cause of the problem. Practitioners, in contrast, will want to carry out further assessments to investigate the child's difficulties and plan interventions. Broad ranges of information-gathering activities are available to meet these goals, including observation and testing. A wide variety of assessment devices can be used for the evaluation and diagnosis of school-age children. The situation with younger children is different with fewer valid instruments available from birth to three years. There are certain features of the language system that make it complicated for any assessment process. It is important to bear these in mind when considering what measures to use. Firstly, language is multidimensional and as such does not easily lend itself to single unitary measures. It is important to consider comprehension and production as well as the more subtle aspects of the language system, such as pragmatics. Secondly, there is

much variation in normal patterns of development and this makes it difficult to draw precise boundaries between typical and atypical development.

Assessment involves evaluating a child's performance and comparing it either with their own performance on different types of tasks or with the performance of other children. Frequently these comparisons can involve standard tools. Four types of comparisons can be made; these may be used either alone or in combination:

- standardized norm-referenced tests;
- criterion-referenced tests, developmental profiles or checklists;
- assessments of the processes of learning - dynamic assessments;
- experimental tasks.

Aims of Assessment

Assessment aims to verify the presence or absence of external factors (to use the term in Harris and Cottam, 1985) contributing to the presenting speech disorder. It should explain its nature; determine a hierarchy of contributing of factors, enable planning of remediation and predict the outcome of intervention.

Components in Assessment

Assessment must look to several sources of information including;

1. The speech data, a corpus of recorded speech
2. Non-speech data gained from clinical observations, data about the speaker which cannot be found from speech data alone, e.g. from language assessment, behavioral observation, etc.
3. Reports from other professionals, e.g. of ENT procedures, psychological assessment, etc,
4. The case history.

Whilst the purpose of assessment is to explore in some depth the first two of these four components, which rely on one another to provide any explanation of disorder, assessment is incomplete if it fails to include four elements. It is taken for granted in this context that the reader will understand the need include the other two. Neither analysis of the corpus of speech data nor other clinical observations are mutually exclusive. One is seldom complete without the other; analysis alone, however thorough, cannot explain the data, cannot account for the process of production of which the speech data are the end product. Analysis of the speech data provides the answer to 'what is produced?', but assessment is incomplete without an answer to 'why is speech produced in this way?'

Only clinical examination, including observation of the speaker's behaviour during speech production, can answer this. The resources available to the clinician for collecting speech data and speaker information may be:

1. Predetermined /published procedures, for example articulation tests, which involve 'fixed' and often quantitative assessment and are frequently termed 'formal assessments'
2. Procedures not dependent on any published, recommended or standardized protocol, sometimes termed 'informal assessment'.

Selectivity in assessment

In some cases it is necessary, at least in the initial stages of management, to assess for all levels of breakdown. In others, the investigator starts with a clear 'signpost' into assessment, which then serves less to inform diagnosis than to establish the details of involvement, and to form the basis for remediation. It is in cases where the point of origin of the disorder is less clearly 'signposted'. And several aspects of speech assessment require attention, that skill is needed in selecting appropriate assessment.

Listed below are many items of potential interest in investigating production disorders, but this does not imply that the assessor is bound to include all of them. Attention is given only to aspects judged to be of greatest informative value, but this is not to say that other aspects are ignored. Assessment of the same individual at a later stage may reveal other facets which require closer scrutiny. Clinicians should be aware of all aspects of potential breakdown, including the neurological, neuroanatomical, neurophysiological, neuromotor, psychological and linguistic bases. Through education and experience, skill is acquired in selecting those procedures most capable of granting insight.

The order presented here places speech data before the clinical observations; in reality both co-occur. Speech data are collected early, setting the requirement for further clinical observation. Sometimes non-speech observations are made first, in which case it is important not to prejudice judgment of the nature of speech production in the individual concerned. The two parts of assessment overlap considerably because, whilst data collection is made, the clinician is simultaneously observing the speaker's behavior.

WAB - Western Aphasia Battery

The *Western Aphasia Battery* (WAB) is the highly respected and widely used instrument for assessing adult patients with aphasia. It is an individually administered assessment for adults with acquired neurological disorders (e.g., as a result of stroke, head injury, dementia). WAB assesses the linguistic skills most frequently affected by aphasia, in addition to key nonlinguistic skills, and provides differential diagnosis information. It is composed in large part of a selection of subtests from the BDAE. The WAB has four oral language subtests (spontaneous speech, comprehension, repetition and naming) upon which an aphasia quotient (AQ) is based. Additional tests of reading, writing, praxis,

drawing, block designing, calculation and portions of raven's colored progressive performance quotient (PQ) are also included.

AQ and PQ combined provide (CQ) cortical quotient as a summary of cognitive function of aphasia language performance scale (ALPS). The four subsets have ten-subtests which cover the modalities of listening, talking, reading and writing etc. each subtest permits scaling of performance within the modality from one to term different levels of performance being defined in terms of message length and complexity.

WAB

Spontaneous speech

- Information content
- Fluency

Comprehension

- Yes – No Questions
- Auditory word Recognition
- Sequential commands.

Repetition

Naming

- Object naming
- Word fluency
- Sentence completion
- Responsive speech

Aphasia Quotient

- Reading and writing
- Reading

- Writing

Construction (non-linguistic skills)

- Drawing
- Block design
- Calculation
- Praxis
- Raven's score

BDAE- The Boston Diagnostic Aphasia Examination

Comprehensive exploration of a wide range of communication skills is accomplished with a battery of thirty one subtests and an evaluation of conversational speech that yields seven additional scores plus supplementary groups of thirteen language and fourteen non language tests.

I. Conversational and Expository speech

II. Auditory comprehension

- Word discrimination
- Body part identification
- Comments
- Complex ideational material

III. Oral expression

- Oral agility
- Automatized sequence
- Recitation, singing and rhythm
- Repetition of words.
- Repeating phrases
- Word reading

- Responsive naming
- Visual confrontation naming
- Animal naming
- Oral sentence reading

IV. Understanding written language

- Symbol and word discrimination
- Phonetic association
- Word recognition
- Comprehension of oral spelling
- Word – phrase matching
- Reading sentences and paragraph

V. Writing

- Mechanics of writing
- Recall of written symbols
  Serial writing

  Primary level writing

- Written word finding
  Spelling to dictation

  Written confrontation naming

- Written formulation

A sample study of Aphasia

I. Phonology

- Phonetic Discrimination

- Phonetic Expression

II. Syntax

- Morphophonemic structures
- Phrasal forms
- Tenses
- PNG markers
- Case markers
- Transitive, Intransitive and causative
- Sentence types
- Predicates
- Conjunctives, comparatives and Quotatives
- Conditional clauses
- Participle constructions

III. Semantics

- Semantic discrimination
  - ✓ Colors
  - ✓ Furniture's
  - ✓ Body parts
- **Semantic Expression**
  - ✓ Naming
  - ✓ Lexical category
  - ✓ Synonymy
  - ✓ Antonymy
  - ✓ Homonymy
  - ✓ Polar questions
  - ✓ Semantic anomaly
  - ✓ Paradigmatic relations
  - ✓ Syntagmatic relations
  - ✓ Semantic contiguity

- ✓ Semantic similarity

- **Discourse**

  Discourse analysis

Phonological/Articulation Disorders: Assessment

Causes of articulation disorders:

Functional/Organic Correlates:

- Hearing Loss
- History of Otitis media during the first few years of life
- Speech sound perception and discrimination ability
- Tooth alignment and missing teeth
- Impaired oral-motor skills
- Eating problems
- Tongue thrust swallow after 6 years of age
- Neuromotor disabilities
- Mental retardation
- Language problems
- Reading disorders
- Family history

Identify the articulation problem if disorder is found:

Compare child's production to normal speech development.

- Age 2
  - /p, b, n, h, k/
- Age 3
  - /m, w, g, f, d, j/
- Age 4

- /t, s, v/ and “sh”
- Age 5
  - /r, l, z/ and “ng,” “ch,” and “j”.
- Age 6
  - “th”, “zh”

Assessment:

Aims of Assessment

- Describe current phonological status.
- Identify differences between client’s performance and typical speaker.
- Determine communicative implications of phonological pattern.
- Identify factors related to cause and maintenance.
- Develop recommendations/treatment plan.
- State prognosis (with/without treatment).
- Monitor change over time.

These goals are met by testing:

- Speech sound inventory
  - list of sounds clients can produce
  - organize by manner of articulation, position within syllables and words
  - syllable position
  - word position
- Syllable and word shape inventory.
  - V, CV, VC, CVC, CCV, VCC, CVCC, CCVC, etc.
- Listing of errors.
- Phonological processes (if necessary).
- Overall intelligibility.

Procedures involved in assessment of articulation/phonological disorders:

Informal Screening

Age appropriate tasks

Name and address

Count 1 to 10

Talk about a TV show

Grandfather passage, Rainbow passage

Formal Screening

Single-word articulation tests

Often part of a more detailed test

Formal Testing

Articulation tests

Test items

- Words with all vowels and consonants in initial, medial and final.
- Consonant clusters in initial, medial and final.

3 components:

- Elicitation of speech sample
- Recording of client response
- Scoring and interpretation of recorded responses

Identification of Articulation errors:

- **S**ubstitution

- **O**mission
- **D**istortion
- **A**ddition

Identification of Phonological processes:

Phonological processes are systematic changes that affect a syllable or an entire category of phonemes

- Processes that affect syllable structure
  - Final consonant deletion
  - Weak syllable deletion
  - Reduplication
  - Cluster reduction
- Processes that affect sound classes
  - Substitution processes
    - For example:
      - Fronting
      - Stopping
      - Gliding
      - Vocalization
      - Deaffrication
  - Assimilation processes (e.g., velar assimilation)

Test "strength" of phonological processes:

$$\frac{\text{Number of times the phonological process was applied}}{\text{Number of opportunities to apply the process}} \times 100$$

Intelligibility:

Subjective description

- Readily understandable when context not known.

- Intelligible with careful listening when context not known.
- Intelligible with careful listening when context known.
- Unintelligible with careful listening when context known.

Objective description

$$\frac{\text{Number of words that can be understood (are intelligible)}}{\text{Total number of words produced}} \times 100$$

Phonological skill Test for Reading Assessment

1. Oral Reading: Fifteen words and fifteen non words were selected and arranged based on complexity.
2. Writing: Writing to dictation task was carried out using twenty five words. It was done as a group task.
3. Metaphonologial Tests: It includes nine subtests.
   a) Rhyme Recognition: 15 pairs of rhyming and non- rhyming words (nine rhymes and non-rhymes) were used. The pairs of words was presented to the children and they were asked to identify whether the paired words are rhyming or not (eg. /paTTu – laTTu/)
   b) Syllable Stripping: 15 words were taken and on presentation children had to delete a syllable indicated and say the rest of the word. as a meaningful word. (eg. /pakaTTu/ – /paTu/)
   c) Syllable Reversal: children were presented with 15 words and were asked to reverse the word syllabically. (eg. talai - laita)
   d) Syllable Oddity: 15 sets of four words each with CVCV or more configurations were presented to the children. They were asked to read to

the words and choose the one that did not belong to the set (eg. /paTam/, /paNam/, /n□e:cam/,/pan□ti)

e) Syllable Blending: 15 words segmented into two were presented and were instructed to say the word by blending the syllable segments into word. (eg. /cam+paLam/ = /campaLam/

f) Phoneme Oddity: 15 sets of four non words each with CVCV or more configurations were presented orally to the children. They were asked to read the non words and choose the one that did not belong to the set (eg. /pacan/, /pilikam/, /ta:nu/, /pu:nuvar/)

g) Phoneme Stripping: 15 words were presented and were asked to read the words and delete a small part of the word and say the rest. as meaningful word. (eg. /pa<u>n</u>tam/ Stripping /patam/)

h) Phoneme Reversal: 15 words were presented and were instructed to say the word by reversing the word phonemically (eg. /ma<u>l</u>ai/-/ai<u>l</u>am/)

i) Phoneme Blending: 15 words segmented into two were presented and were instructed to say the word by blending the phonemes into word. (eg. /varuv+a:n/ =/varuva:n/

4. SHWA Test: Test item consisted of two phonemes which are not present in tamil language and three vowels /i/, /u/and/e/ - their both long and short form. Here the child requires combining the given vowel with a phoneme represented by a visual symbol (grapheme) both of which do not exit in the particular orthography. For eg: o + , =op and œ + , = œp where o has to be pronounced as Z and where œ has to be pronounced as g^h.

LARSP

The approach to grammatical disability known as LARSP (Language Assessment, Remediation and Screening Procedure) is summarized in the form of a single-page profile chart, on which the various patterns of grammatical strength and weakness in a clinical sample can be plotted. The profile chart contains several kinds of information, organized in terms of three main dimensions:

(a) the main types of organization in sentence structure and function are represented under various headings laid out horizontally on the chart;
(b) the main stages of grammatical acquisition are laid out vertically on the chart, beneath the thick black line;
(c) the main patterns of grammatical interaction between T and P are summarized above the thick black line, in Sections B, C and D (*'P' refers to patient or pupil, 'T' to teacher or therapist.*).
In addition, the bottom line of the chart contains certain kinds of summarizing information, and Section A is included primarily as a time-saving device in using the procedure.

For a full assessment, it is recommended that a sample of approximately 30 minutes' duration be obtained of an unstructured *T and P* interaction, using whatever stimuli are felt likely to facilitate a free conversation (e.g. toys, pictures, magazines, questions about pastimes). In practice, it is recognized that it is often impossible to obtain such a sample, and that various kinds of structuring may need to be introduced into the situation. All relevant variables (e.g. type of materials used, number of participants. character of any formal stimuli) should be noted on the top of the chart. Likewise, if a shorter (or longer) sample has been taken. this too should be indicated at the top of the chart.

Whatever the size and type of sample. T should try to obtain P responses relating to two types of stimuli: *(i)* stimuli relating to the immediate environment of their interaction (e.g. questions about the room they are in, or about the toys or pictures being used); *(ii)* stimuli relating to objects, events, etc. *not* visible to T or P (e.g. recent events in P's life, what is about to happen to P). This is to ensure that a reasonable opportunity is given for P to use a wide range of structures, relating to both types of situation—different tense forms, for example. A similar distinction should be borne in mind if written or signed samples are being used as input to a LARSP analysis.

PROPH

The segmental phonological profile known as PROPH *(‚Profile* of Phonology') is essentially a presentation of the English sound system on a 2-page chart. To facilitate the compilation of the

profile. a transcriptional page is added. To facilitate the interpretation of the profile. a separate 3-page section provides various suggestions about ways of summarizing the main patterns in the data.

The data base of the procedure is a sample of P's connected speech of up to 100 word-types (see further below). But there is no magic in the figure of 100. As with all profiles, one transcribes as much as is necessary to demonstrate a pattern in P's disability. Routinely, one might transcribe only a few dozen items to begin with and profile these; the chart might then be sufficiently full to indicate the nature of the phonological problem. But if no clear pattern was emerging at this stage, a further sample would need to be taken. lOO- word samples are usually enough to establish a pattern; but if necessary, the sample could be larger, using supplementary transcriptional pages. Sometimes, one has to be satisfied with what one can get!

Similarly, there is no obligation to use connected conversational speech, though this is what we aim for in the first instance. *Any* sample can be profiled (e.g. confrontation naming. the results of an articulation test. or the imitation of T speech), Information about sample type is indicated at the top of the page. Samples of different types should *not* be profiled together, unless some kind of typographical distinction is made on the chart.

Each word-type in the sample is assigned a line and subsequent word-tokens of the same type are placed on the same line. For example, if p's first sentence were *daddy go* ['dred\ 'goul. the transcription would appear as follows:

1 *daddy* 'dædı’

2 *go* 'gou

If, later in the sample. P used these words again. they would be placed along the same lines as their first occurrence.

PROP

The nonsegmental phonological profile known as PROP (Prosody Profile) is a single-page chart, on which can be placed information about the main prosodic patterns encountered in a sample of clinical data. The term 'prosody' here refers to the *linguistic* use of pitch, loudness. speed of speech, pause, and rhythm-in other words, to the way in which these variables can alter the meaning of what we say. PROP is not intended for the description of

phonetic abnormalities in the use of these variables. as will be found under the heading of 'voice disorders' (in relation to the term *dysprosody,* for example).

PROP is an aspect of *phonological* analysis, complementing the analysis of the segmental aspects of pronunciation. While in principle any of the prosodic variables of speech could be disturbed, and contribute to P's communication problems, in practice it is *pitch* which causes most linguistic difficulties, and the bulk of the profile chart is therefore given over to an analysis of this variable. The linguistic use of pitch is usually referred to as *intonation,* and indeed it is intonation with which we are most regularly concerned, in clinical settings. But other prosodic factors need to be related to intonation, if a full understanding of p's difficulties is to emerge-and in any case, we need to be able to note the occurrence of other categories of prosodic problem, if the principle of profile comprehensiveness is to be maintained (d. 1.9). There is therefore space on the chart for a reference to other things than intonation.

The intonational theory which the chart reflects recognizes 3 main distinctions in the way pitch patterns are used in a language: the organization of connected speech into *tone units;* the use of specific *tones* within these units; and the phenomenon of *tonicity,* within these units. Distinct patterns of disorder are associated with each of these notions, but first their normal use in relation to English needs to be explained.

The primary organization of speech is into *lone units* (sometimes called *tone groups).* A tone unit is a finite set of pitch movements. grouped into a distinctive contour and uttered with a distinctive rhythm. Tone units are often bounded by pauses, and these boundaries generally coincide with points of grammatical junction. A tone-unit boundary is marked with a slant line, in this system of transcription, as follows:

if you see him/ ask him to cal/!

J'd like 10 buy some eggs/ a pinl of milkl and some bUllerl

the dog bit the cat/ and the cat bit the dog!

PRISM

Any attempt at a semantic profile chart. given our limited theoretical and empirical knowledge of the way linguistic meaning is structured and acquired, is full of major pitfalls. It is certainly not possible to construct a chart which could claim to be as principled as those discussed elsewhere in this book. On the other hand. semantic problems do playa significant role

in the assessment and remediation of many child and adult Ps, and some systematic way of focusing T's attention on the nature of these problems is urgently needed. The procedure known as PRISM (‘Profile *in* Semantics') is a first attempt towards this goal. It has been a useful tool in organizing ideas about semantic disability and remediation, even though it raises several problems for which arbitrary solutions have had to be devised, in order to promote consistency of use.

There are two main features to note about the PRISM procedure. Firstly, it is presented as a combination of two subprocedures-one dealing with the relationship between semantics and grammar (PRISM-G), the other with the relationship between semantics and lexicon (PRISM-L). It is important tostress the importance of having both a grammatical and a lexical dimension for semantic analysis. While most people identify semantic analysis with
vocabulary (or lexicon), it must not be forgotten that the meaning of a word is largely dependent on the context in which it is used, and that therefore theword's role in a sentence needs to be taken into account. Notions such as 'actor', 'action' and 'location' provide an essential alternative dimension of analysis to the grammatical concepts of Subject, Verb, Adverbial etc. PRISM-G is essentially a descriptive framework for the analysis of the meanings conveyed by the different grammatical elements of a sentence. It is a 3-page chart, constructed along similar lines to LARSP, but with the patterns defined according to semantic (as opposed to syntactic or morphological) criteria. Secondly, the most noticeable characteristic of the procedure is the size of the PRISM-L component-16 pages, instead of the 1 or 2 typical of other profiles. The reason for the chart's greater *size* is the extent of the vocabulary which it has to incorporate. Phonological and grammatical procedures are relatively straightforward, in that they have to deal with only 100 or so variables; it is therefore reasonable to expect that short samples of connected speech will be fairly representative (apart from in certain well-recognized situations). and that it will be possible to summarize the use of these variables in a page or two. But the commonly-occurring 'domestic' vocabulary of daily life runs into several thousand lexical items. It is obvious that a sense of P's lexical range will not be established from a short sample, nor can it be usefully organized into a couple of pages. The primary characteristic of PRISM-L, accordingly, is its extensive inventory of lexical fields, into which (in principle) the whole of P's vocabulary can be incorporated. The detailedclassification is needed in order to provide a discriminating assessment of P's lexical range, and to identify lexical areas for remediation. To meet these

criteria, a chart of several pages is unavoidable.

Before outlining the descriptive frameworks, certain theoretical considerations need to be briefly reviewed:

*(a)** Lexical terminology needs to be clearly distinguished from grammatical, if the discussion of disability is to be unambiguous. The term *word* is a recognized part of grammatical metalanguage, and hence a different term isneeded to discuss the minimal units of vocabulary. Here, the terms *lexical item* and *lexeme* are used (interchangeably) to refer to these minimal units--in other words, the items which would be listed as head-words in a dictionary. It should be noted that grammatical variants are ignored inspecifying lexemes: for example, *walk, walks, walking, walked* are all variants of the same lexeme WALK (conventionally printed in small capitals): *is, are,am, be, been, was, were* are all variants of the lexeme BE: and so on. Also. It should be noted that some lexical items may consist of more than one word, e.g. *spick and span, kick the bucket* ('die') (and all idioms), *switch on* (and all phrasal verbs), *too many cooks spoil the broth* (and all proverbs).

(b) While the PRISM-L procedure is presented as an inventory of lexical fields, it must be remembered that this is only the first step in theinvestigation of P's semantic system. The lexicon of a language is *not* an inventory, but a system of contrasts. The business of lexical analysis is to identify the types of contrast which interrelate the lexical items in a language-1:ontrasts such as sameness of meaning, oppositeness of meaning, and so on. These contrasts can not usually be established on the basis of a profile analysis of a sample of spontaneous speech, however; they need to be elicited in structured situations. On the other hand, unless one has some units of vocabulary with which to operate, it is impossible to carry out the more advanced, structured work. The aim of PRISM-L, therefore, is to provide an initial classification of lexical items, which can provide the motivation for a more principled investigation of P's semantic system than would otherwise be possible.

*(c)** In its emphasis on vocabulary (in the present case, on English), the business of semantic analysis must be rigorously distinguished from the concerns of cognitive studies. While cognitive problems are often the reasons for the failure of P to develop an adequate lexical system, any statement of lexical difficulty can and should be made, in the first instance, independently of cognitive considerations. It is possible to have semantic disability within an otherwise intact cognitive ability, which illustrates the need to keep the distinction clear.

(d)· Similarly, the various units and categories presented on the PRISM charts are 'neutral' in respect to the question of production or comprehension.

One may use PRISM, as with any profile, either as a guide to production or as a guide to comprehension of the lexical items and sentence patterns it contains. Unless one introduces a specific convention, marking a specific lexical item or sentence pattern onto the charts says nothing about whether P has understood it: it simply indicates that he has used the item/pattern (if the study is of his expressive language), or had the item/pattern used to him (if the study is of his receptive language). It is a separate analytical decision to say how efficiently these items/patterns have been processed by P, whether some kind of 'comprehension problem' is involved.