Advanced Cognitive Processes
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Lecture – 21
Aphasia

Hello and welcome [noise] to the course introduction to advanced cognitive processes. I am Ark Verma from IIT, Kanpur. And we have been about language and various aspects related to language comprehension, production. Last few lectures we were talking about various aspects of reading. In the last lecture [vocalized-noise] if I remember correctly, we were also talking about disordered aspect of reading which was referred to as dyslexia and we kind of saw that what kind of patterns lead to various kinds of reading difficulties and then they were types of dyslexia we talked about. Today's lecture I thought I talked about the disorder of speech production,

The disorder to speech production is commonly referred to as aphasia. However, aphasia is not really a singular disorder, there are various kinds of aphasia and there are various aspects of the process of reproduction that when damaged lead to different profiles of aphasia.

In today's lecture we will talk about some of the foundational issues of aphasia and some of the basic aspects of how the models of speech production and perception evolves together to lead to an explanation of different kinds of aphasias that were possible. Again I am not really going into a lot of detail of these processes, but just to give you a flavor of how speech disorders or language disorders kind of play out I will talk about different categories of aphasia.

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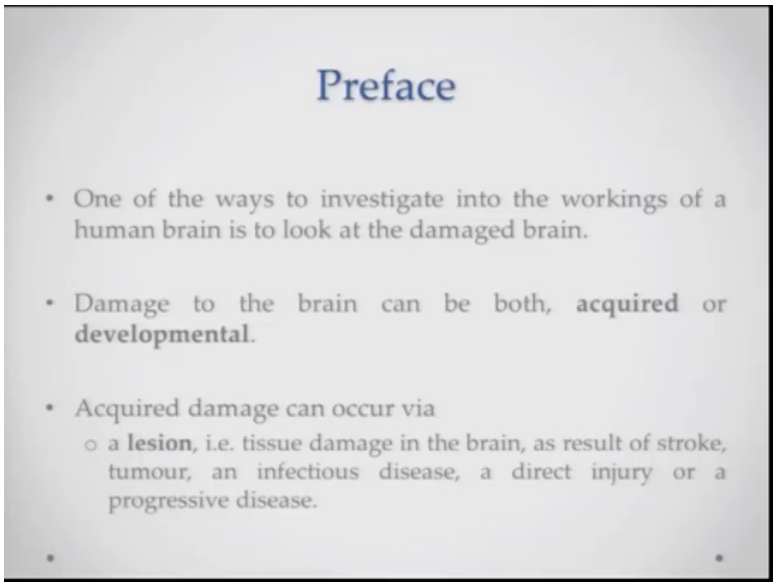

Now, I have told his earlier as well and let me repeat this today as well. One of the very nice ways to basically look into the workings of the brain is when you can actually look into the disorder brain, when you can look into the damage brain because this is where it tells you that you know [vocalized-noise] which aspects of the brain, which parts of the brain related what kind of cognitive processes. And you can reduce more clearly the link between the specific brain areas and particular cognitive functions.

Now, this damage or these disorders of the brain can be a both acquired or developmental. Acquired damage to the brain basically could occur because of tumor stroke or say for example, in infectious disease or sometimes it direct injury to the brain.

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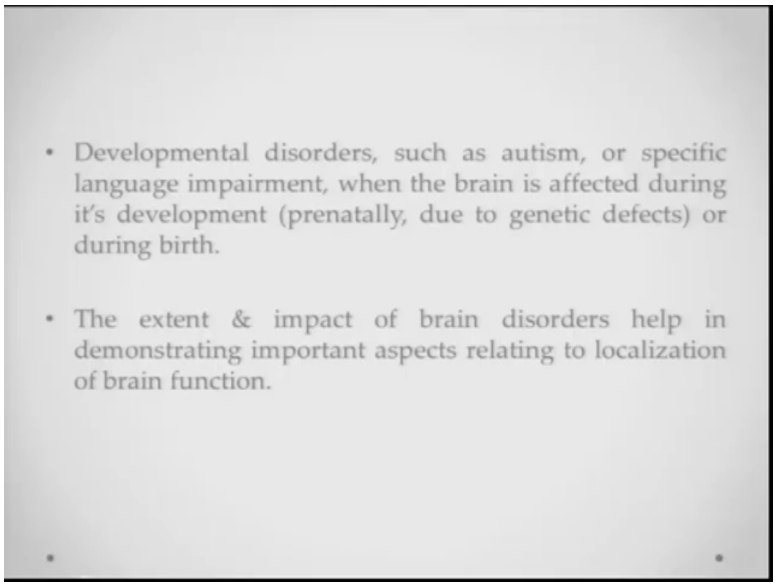

On the other hand developmental disorder such as autism or specific language impairments are basically coming to the, for when the brain is affected during its developmental phases; for example, [vocalized-noise] prenatally or due to genetic defects or due to exposure to certain kind of harmful chemicals. Now the extent and impact of these brain disorders is very helpful in demonstrating the important aspects related to localization of brain function.

In today's lecture we will talk about aspects which leads to particular profiles of difficulty in production of speech.

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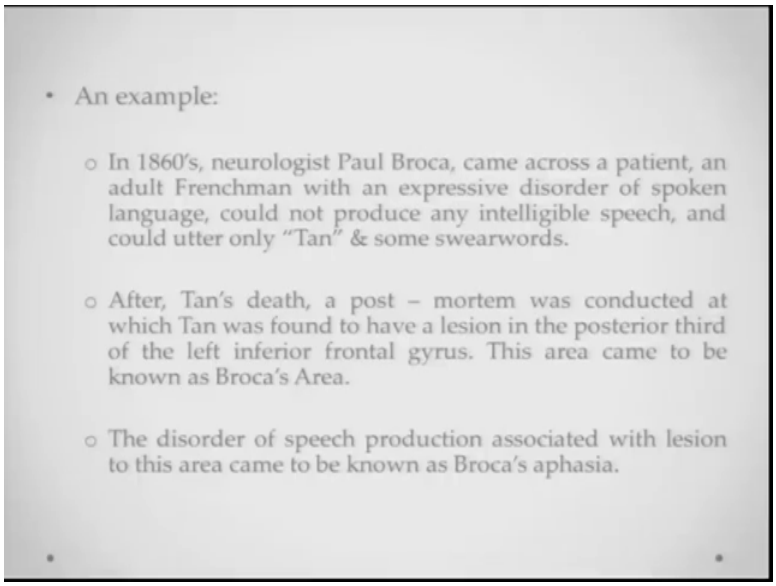

Let us take an example which kind of made this link possible and is probably one of the most cited example in whole of neuropsychology. In 1860s, neurologist Paul Broca, comes across a patient an adult [noise] French man who has a disorder of expressive disorder speech production and he could not really produce any intelligible speech, he could not say anything rather than he could just produce words like tan, tan, tan, and he could not say anything else or some swearwords etcetera.

After tan died a post mortem was conducted and it was found that he has a very specific lesion in the posterior third of the inferior frontal Gyrus. This area was linked with his speech production difficulties [vocalized-noise] and came to be known as Brocas Area. The disorder of speech production basically associated with lesion to this area later came to be known as Brocas Aphasia.

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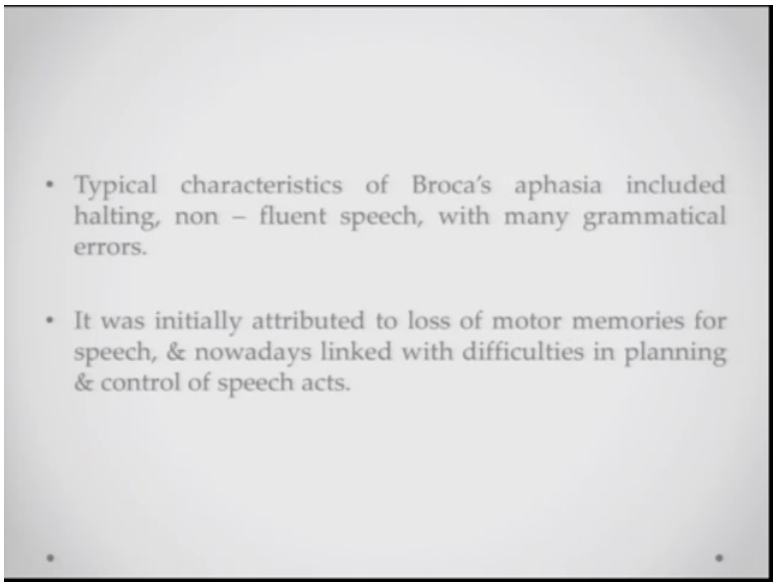

Brocas Aphasia is probably the most cited aphasia profile and it is something that is seen more often than other profiles of aphasia. [noise] Typical characteristics of Brocas Aphasia included halting, non fluent speech, and with the speech would have many grammatical errors. [vocalized–noise] It was initially attributed to the loss of motor memories for speech and nowadays it is linked with difficulties in planning and control of speech acts as well.

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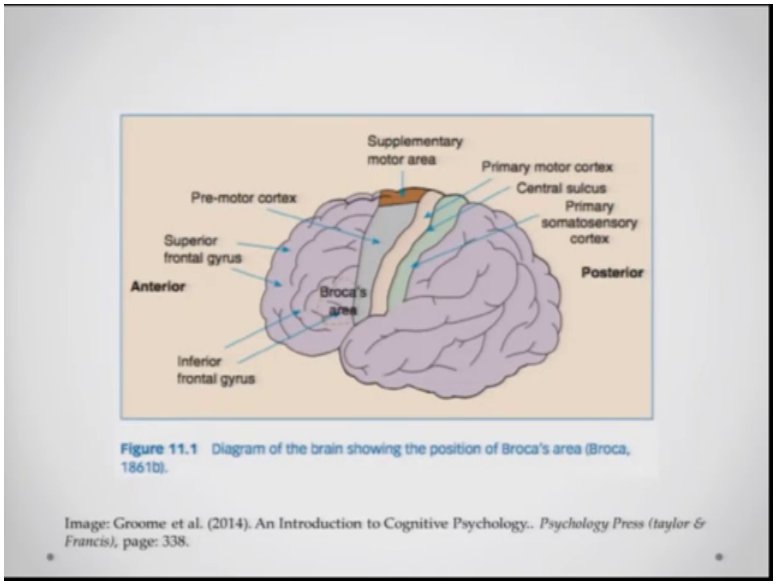

So, this is one of the examples, how damage to the particular aspect of the brain can leads to particular times of disorders, This is an example diagram borrowed from David Groomes book on Introduction to Cognitive Psychology and here you can see that Brocas Area is basically just to the front of the brain and [vocalized–noise] it is very close to the motor cortex, the primary motor cortexes and it is inferior to the supplementary motor area.

This area is supposed to be involved in planning and execution of speech acts and hence it is one of the most important areas which are responsible for production speech.

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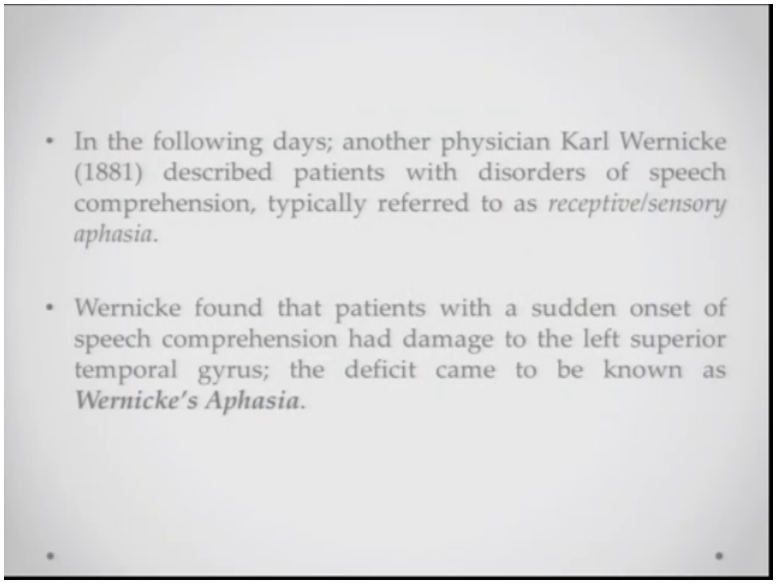

In the following days; another physician Karl Wernicke in 1881 started describing patients who are came with disorders of speech comprehension. These patients were basically referred to as having receptive or sensory aphasia. Now Wernicke found out that these patients who are coming within sudden onset of speech comprehension difficulties had damage to the left superior temporal gyrus. And this deficit then came to be known as Wernickes Aphasia. This area at the left superior temporal gyrus came to be known broadly as the Wernickes Area. Here is the diagram again borrowed from Groomes book just for your illustration. You can see that Wernickes Area is on the superior part of the superior temporal gyrus and it is on the left hemisphere.

So, superior temporal gyrus in that sense is connected to comprehension of language and it kind of further experiments of and later research has kind of built this link even stronger.

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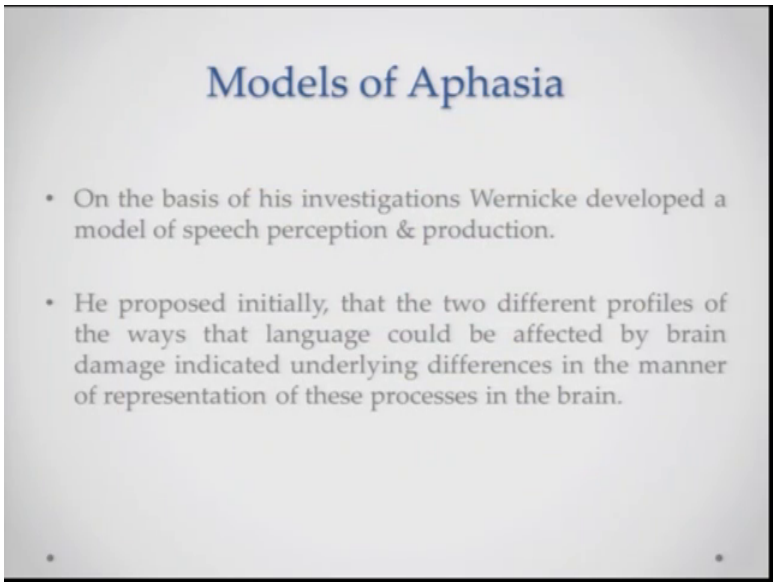

Now, on the basis of whatever experiments in the different kinds of profiles that Wernicke and Brocas and other Broca and other people were coming across. Wernicke developed a model of speech perception and production, which was another first unified models of production interception of speech. He proposed initially, that because they were two different profiles of the way language could be affected by brain disorders or this split could basically indicate that there are underlying differences in which the perception and production of brain is a perception and production of language is organized in the brain.

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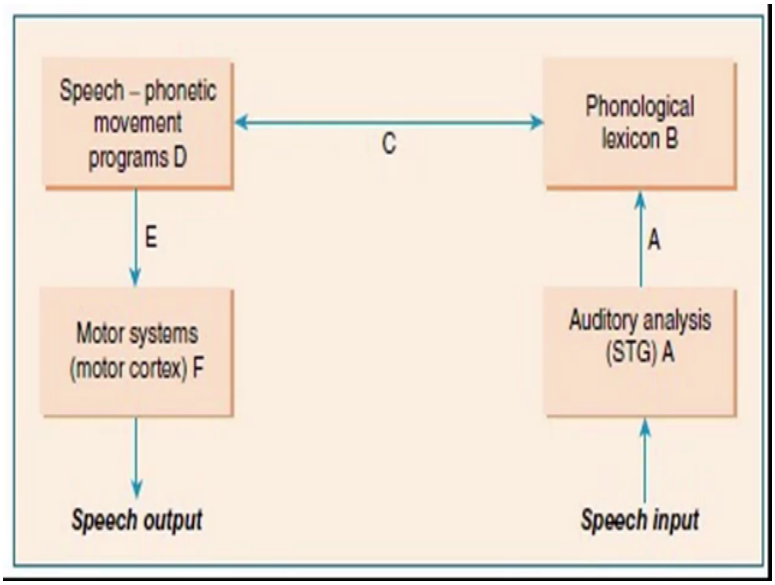

Here is the diagram which is the simplest model which was initially proposed by Wernicke in 1881. You can see that on the left hand side there is a speech phonetic movement programs. They are connected to the phonological lexicon and then there are speech phonetic movement programs are connected [vocalized–noise] to the motor systems and that is the motor cortex and that leads to speech output. While the phonological lexicon basically is receiving input from auditory analysis and that is what is basically analyzing that and probably he is helping a comprehension of speech.

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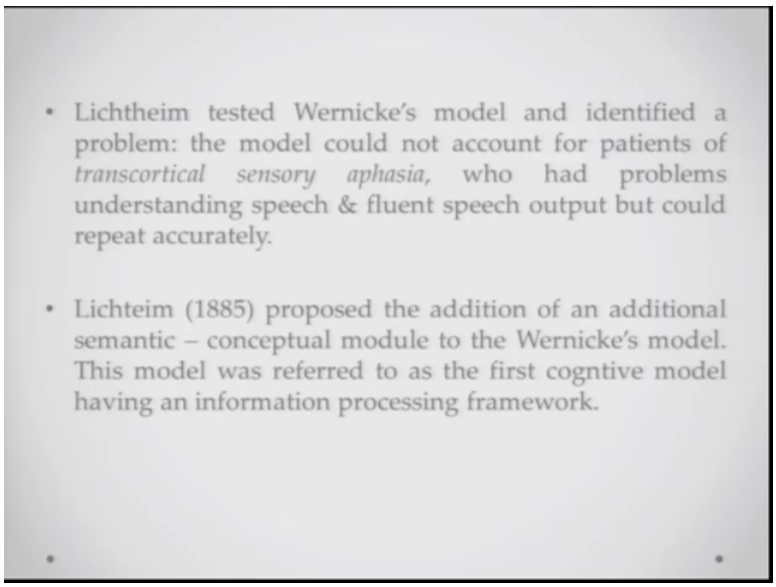

So, this is again one of the very simplest models of perception production of speech; obviously, it had to be developed further. So, Lichteim started testing Wernickes model and he identified a [noise] very important problem. He said that the model could not really account for patients who are coming with this disorder then turned transcortical sensory aphasia. So, the problem with this patience was that they had problems in understanding speech and they had problems in a producing speech, but they could repeat whatever was said to them rather accurately.

Now Lichteim propose that you know if we add an additional semantic and conceptual module to the Wernickes Model this would be able to account for this kind of patients.

So, this particular model which was given by Lichteim was referred to as one of the first cognitive models which basically [vocalized–noise] was based on an information processing framework.

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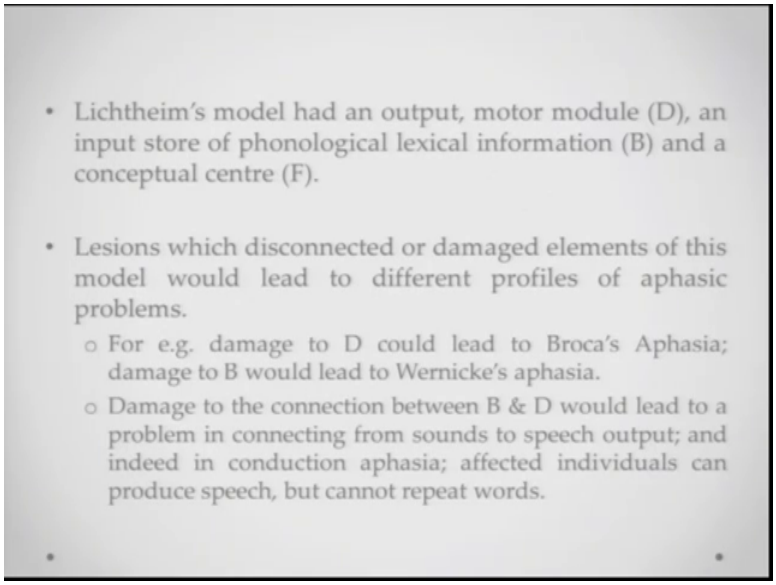

So, Lichteims model basically it has an output motor module denoted by D, it has an input store of lexical phonological and lexical information which is B and it also has a conceptual centre.

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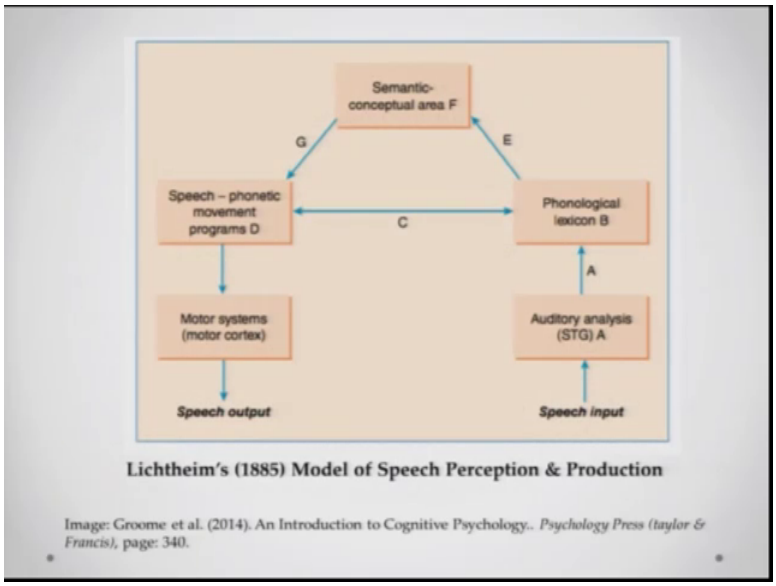

Here you can see the model that was proposed by Lichteim. We will probably refer to it later as a Wernicke Lichteim module because this is in a sense further development of the model that was proposed by Wernicke.

So, you can see in addition to the speech phonetic movement program which in D and the phonological lexicon which is in B, there is another area call the semantic conceptual area which is denoted by F. Now you can see that there are connections, there are lines connecting the semantic conceptual area with the speech phonetic movement programs and the semantic conceptual area with [vocalized–noise] the phonological lexical. And then you have the motor systems at the left side and they have the auditory analysis on the right side. Now this model is a specified such as a lesions to different aspects of the module, different parts of the module or lesions which could affect the connections between these different aspects of the model could lead to different profiles of aphasia.

It started explaining the kind of patients that were coming in at that point in time. For example, if somebody came with a complainer brocas aphasia you could assume, what you could presume that the damage was to the section D, which is the speech phonetic module. And then if somebody [noise] would come with damage to the, if somebody would come with Wernickes Aphasia, then you could assume that there is damage to the phonological lexical.

Also if damage was there between the connection between B and D which is basically your line C then, people would come up with complaints of they will have damage of Cs production and comprehension, but repetition will be all right. So, they will have basically what is known as[vocalized–noise] conduction aphasia. In conduction aphasia damage to the connection between B and D would lead to a problem in connecting sounds to speech output and basically the affected individuals would be able to produce speech, but they will not be able to repeat words; this is the model.

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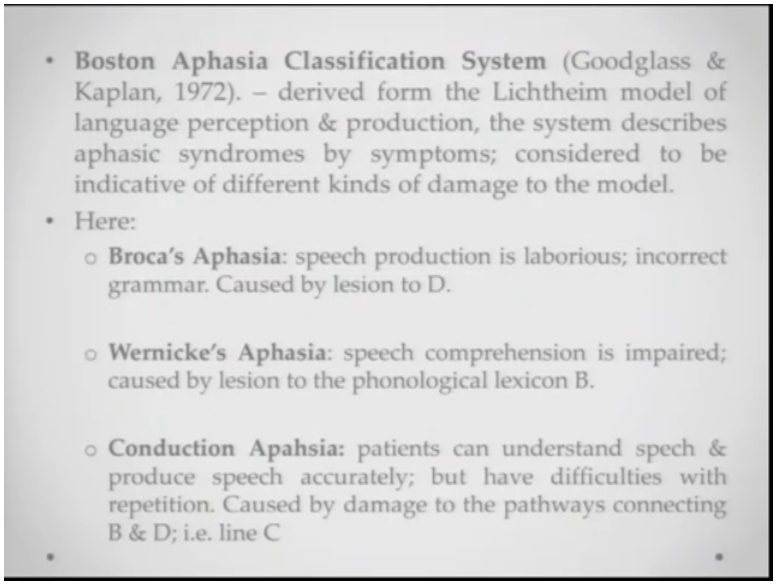

Now, the development of both of these models, actually lead to an classification system for aphasia.[noise] It is also referred to as one of the classical aphasia systems. This was basically system that would describe different kinds of aphasia profiles and would also specify the damage to the aspect of the model which would have led to such a profile. So, I will just briefly go over this. [vocalized–noise]So, Brocas Aphasia you will find the speech is very effortful, it is laborious, the grammar is incorrect and this is basically caused by lesion to D which is the speech phonetic movement programs. Wernickes Aphasia basically happens due to damage to aspect of the module B and which is basically the phonological input lexicon and here you will see that the patients have impaired speech comprehension.

In conduction aphasia patient will be able to understand speech and produce which accurately, but we have difficulties with repetition. This will have been caused by damage to line C connecting the modules B and D.

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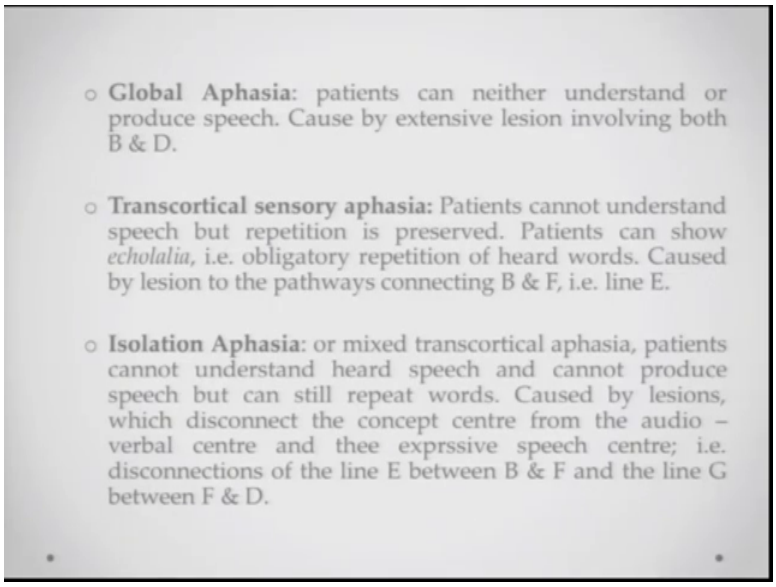

Similarly, global aphasia patient can neither understand or produce speech correctly and this should have been caused by an extensive damage to both modules B and D of the model. Then it is transcortical sensory aphasia; transcortical sensory aphasic patients cannot understand speech, but they can repeat back whatever you said to them. These patients often show the symptom called [vocalized–noise] equal area; that is obligatory repetition of heard words. So, if you tell them something they will repeat it again and again. This would have been caused by the damage between the pathways connecting B and F which is the phonological input lexicon and the conceptual centre and which is basically denoted by line C in the model you can see it here.

Similarly, there is isolation aphasia or transcortical motor aphasia. Transcortical motor aphasia basically a patients would not be able to understand heard speech and they can now produce speech, but they can still repeat words. This profile of aphasia will be caused by when they will be a, [vocalized–noise] disconnect from the concept centre to the audio verbal centre and the expressive centre. The disconnections of line E between B and F and line G between F and D; you can see it here.

So, we are talking about disconnection of line G, which is semantic conceptual and speech phonetic movement program and line E, which is semantic conceptual and phonological etcetera.

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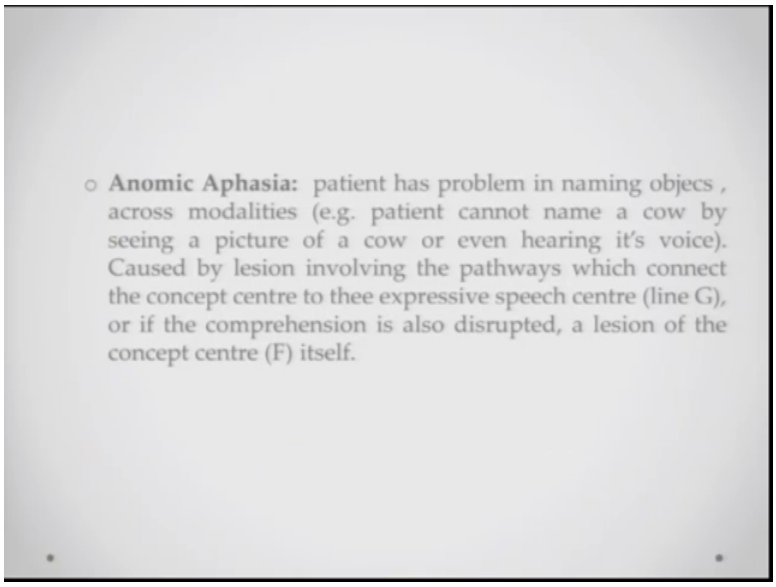

A different kind of aphasia would be anomic aphasia. Patient suffering from anomic aphasia would have a problem in naming objects across modalities. Say for example, even if you show them a picture of a cow, [vocalized–noise] they will not be able to name the cow. Even if make them here to the sound of cows voice, they will not be able to name the cow.

Now, this would basically be caused by lesion involving the pathways which connects the concept centre to the expressive speech centre. So, basically we are talking about the line G, which is basically connecting the sematic conceptual area to the speech phonetic movement programs area. So, this is basically some of the aspects of what the Boston Aphasia Classification System [vocalized–noise] gives us and it is a good link. Obviously, it is something that was developed to diagnose the different kinds of aphasic profiles that were coming in.

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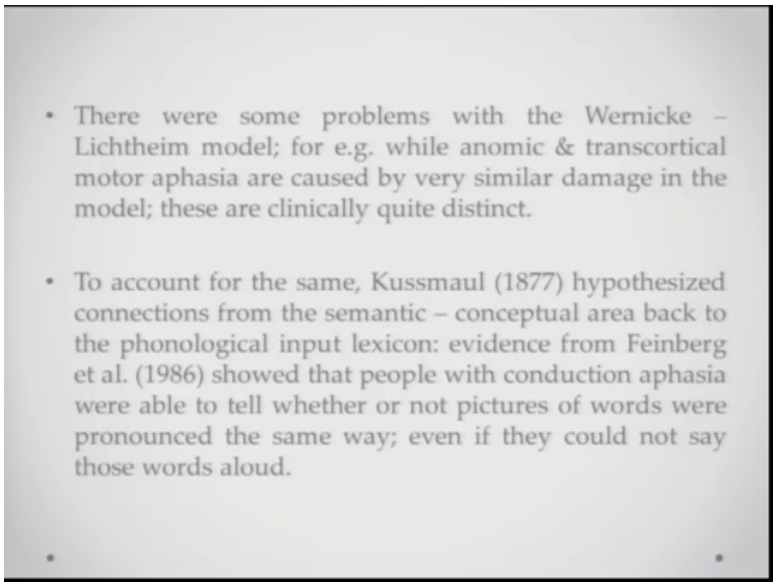

Now, even these models basically the one proposed by Wernicke and then improve later by Lichtheim known as the Wernicke Lichtheim Model, it has some problem. In this model if you notice the anomic and transcortical motor aphasics or the isolation aphasics are basically being caused by very similar damage in the model.

But when you look at the patients and when the clinicians were looking at these patients, they found that these two profiles are clinically very distinct; [noise] patients were showing very different symptoms.

So, to actually account for the same, to make the model explain these kind of profiles Kussmaul in 1877, hypothesize that they should be a feedback connection from the semantic conceptual area back to the phonological input lexical. And evidence for the fact that disconnections are there came from[vocalized–noise] Feinberg and colleagues study where they showed that people with conduction aphasia were able to tell whether or not pictures of words were pronounce in the same way even though they were not being able to produce the words correctly. So, they would know how these words are pronounced, but they will not be able to execute them correctly.

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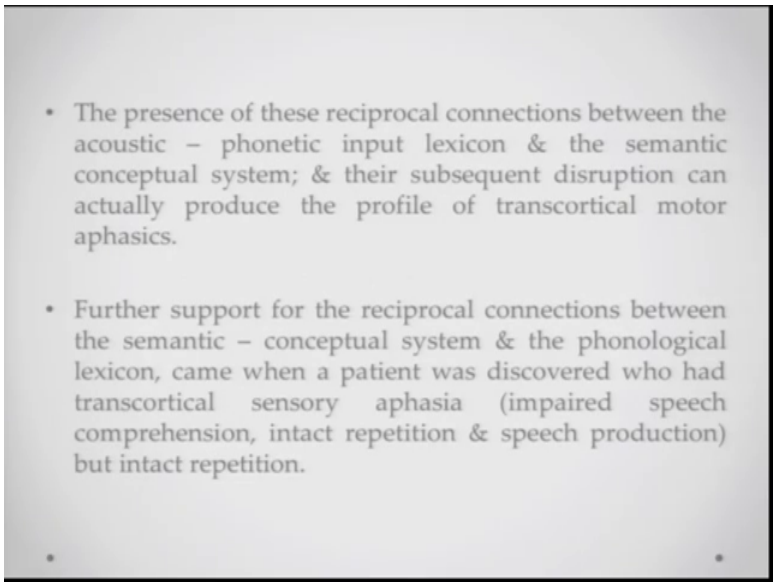

And he presence of these reciprocal connections between acoustic phonetics input and the semantic conceptual store and then their subsequent disruption could actually account for such a profile of transcortical [vocalized–noise] motor aphasics. Further, the support for these reciprocal connections between semantic conceptual area and the phonological lexicon also came when a patient was discovered who had transcortical sensory aphasia. That is these people had impaired speech comprehension, but intact repetition and production of speech.

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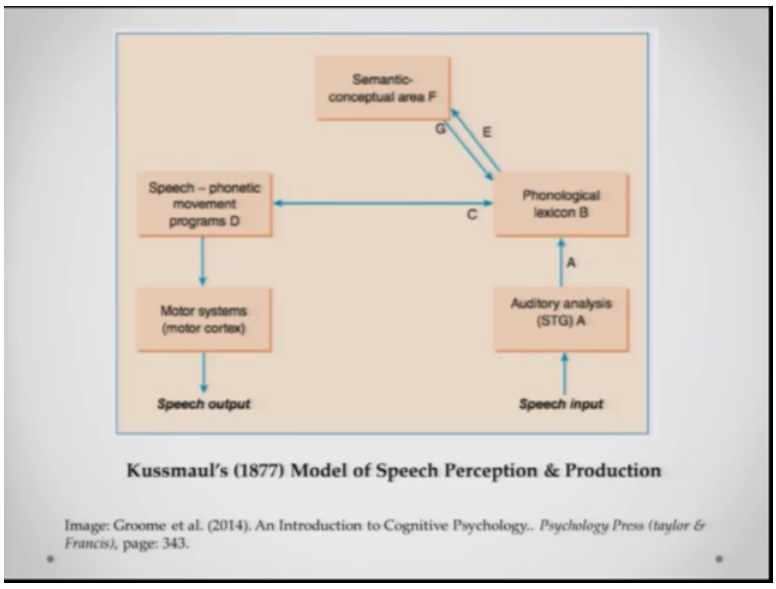

So, you can see that the model basically now comes from being called as the Wernicke Lichtheim model to the Wernicke Lichtheim Kussmaul model. And you will see that there are these feedback [vocalized–noise] connections from the semantic conceptual area to the phonological lexicon, while they have omitted the connection between the speech phonetic movement program area to the semantic conceptual area. We will see that this kind of lead to bit of a problem later.

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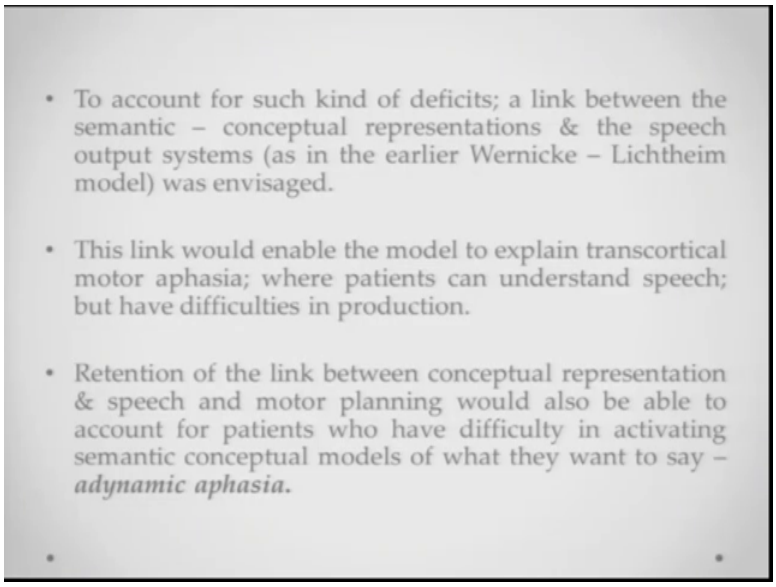

[vocalized–noise] Now, basically the account for a different kind of deficit there has to be this link was envisaged between the semantic conceptual representations and the speech output systems as was there in the Wernicke Lichtheim Model. Now if you have this link because this the absence of this link was leading to problems. Now with the presence of this link the model could now explain [noise] the profiles of transcortical motor aphasia were patients could understand speech, but they would have difficulties in production. Retention of the link between this conceptual representation and the speech phonetic motor planning would also be able to account with a patients who had difficulty in activating the semantic conceptual models of what they wanted to say.

This basically, this is very specific profile of aphasia was referred to as adynamic aphasia at some point.

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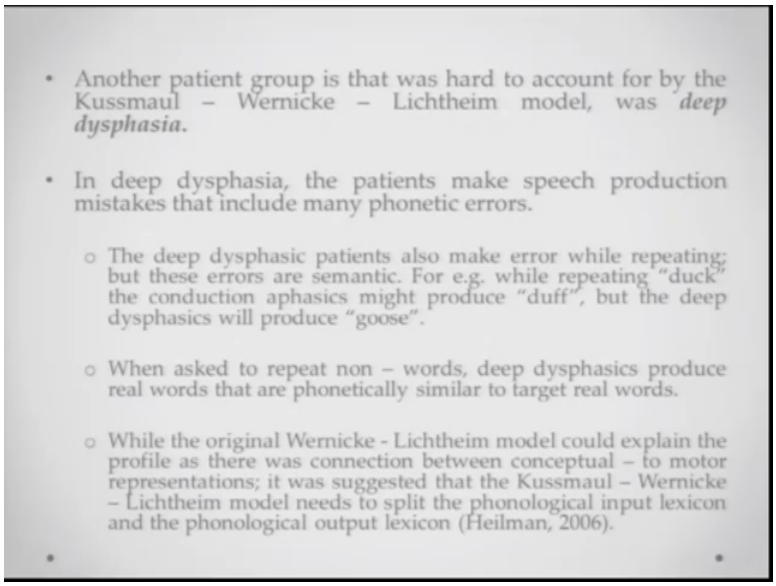

Now, you see also there was this another profile that this particular Kussmaul Wernicke Lichtheim Model could not explain was this aspect with deep dysphasics. Now deep dysphasics basically our patients that can make a speech production mistakes, but the speech production mistakes basically have a lot of phonetic error. Also deep dysphasic patients when they make errors [vocalized–noise] while repeating, but these errors are semantic in nature, these errors are basically. Suppose for example, if you are talking about, even if you ask these people to repeat a word duck again and again the conduction aphasics might actually commit errors of the kind that they will produce duff instead of duck.

But the deep dysphasics would actually produce a semantic relative of duck rather than duck itself. So, they would basically produce goose when you ask them to repeat duck. Also when asked repeat non words these deep dysphasic patients would produce real words that are phonetically similar to target real words. So, they have this impairment of repeating non words.[noise]

While the original Wernicke Lictheim Model could actually explain these kind of profiles as there was the connection between the semantic conceptual area and the speech movement program area. The Kussmaul Wernicke Lictheim Model basically could not do it as it was proposed. So, Heilman, 2006 basically suggested that the Kussmaul Wernicke Lictheim Model needs to split the phonological output lexicon and

phonological input lexicon. [vocalized–noise] So, if you remember there is this module B here, which is the phonological lexicon. Heiman in 2006 suggested that, in order to be able to explain the profile of deep dysphasics and conduction aphasics, they need to split this phonological lexicon into two parts into the phonological input lexicon and the phonological output lexicon.

Let us see how this model looks once this is there.

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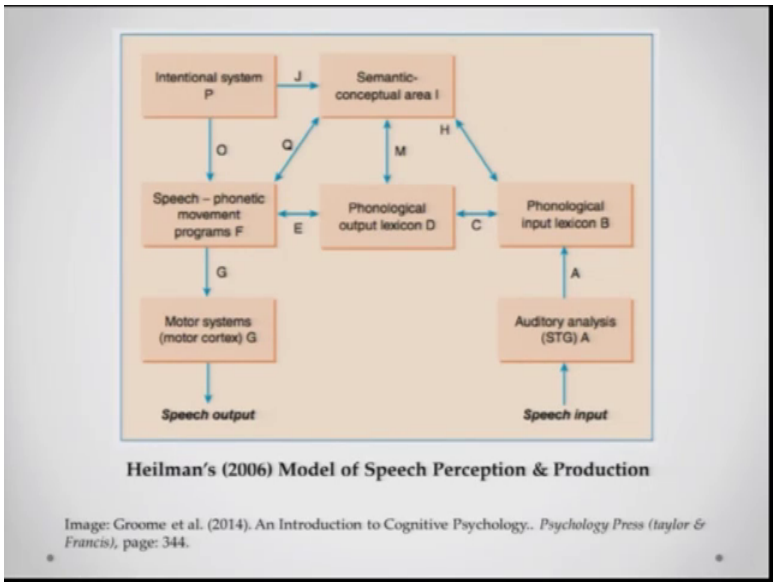

Heilman's (2006) Model of Speech Perception & Production

Image: Groome et al. (2014). An Introduction to Cognitive Psychology.. *Psychology Press (taylor & Francis)*, page: 344.

So, this is how the model started looking now. So, you have the semantic conceptual area, the link between the semantic conceptual area and speech phonetic area is also retained and the phonological lexicon is now split into the phonological input lexicon and the phonological output lexicon. [vocalized–noise]

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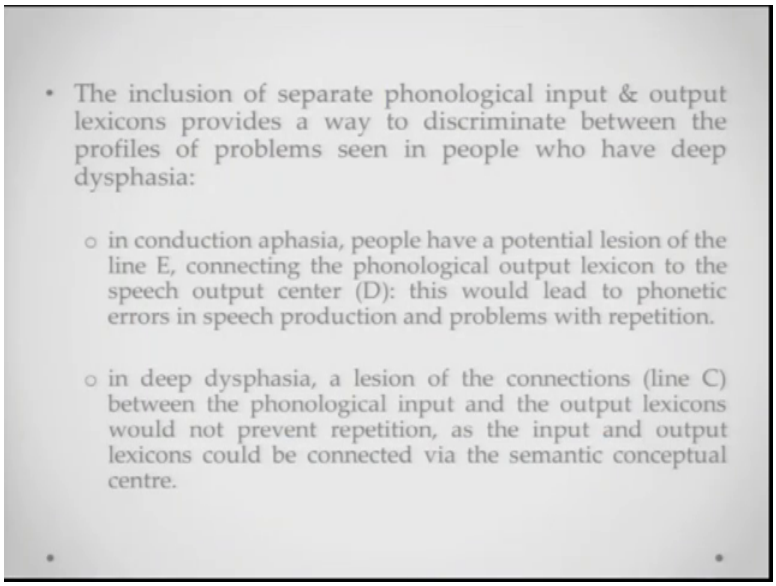

Now, the inclusion of these separate phonological input and output lexicon provided a way to discriminate between the profiles seen in the problems of people who had deep dysphasia. In conduction aphasia people have, [vocalized–noise] so this is how the deference was made. In conduction aphasia, people would have a potential lesion of the line W which connects the phonological output lexicon to the speech output center.

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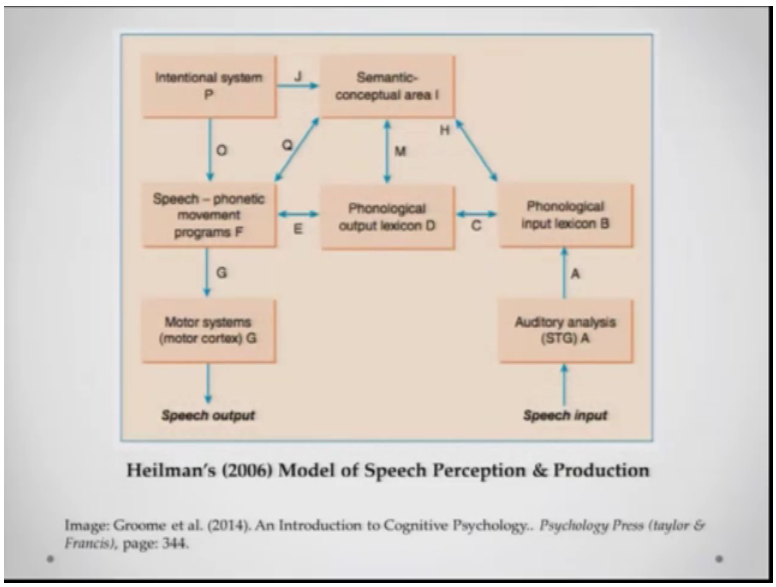

You can see here, I am talking about the line E, which is connecting phonological output lexicon to the speech [noise] phonetic movement programs. And this would basically

lead to errors in speech production and having problems with reputation. When you talk about the deep dysphasic patients, a lesion of the connections between the phonological input and output lexicons can be envisaged. [vocalized–noise]

So, you see there is this line C, which is connecting the input and output lexicon. Now damage to this line C basically would not really prevent repetition as the input and the output lexicons are connected via the semantic store, but it would lead to a different kind of a profile.

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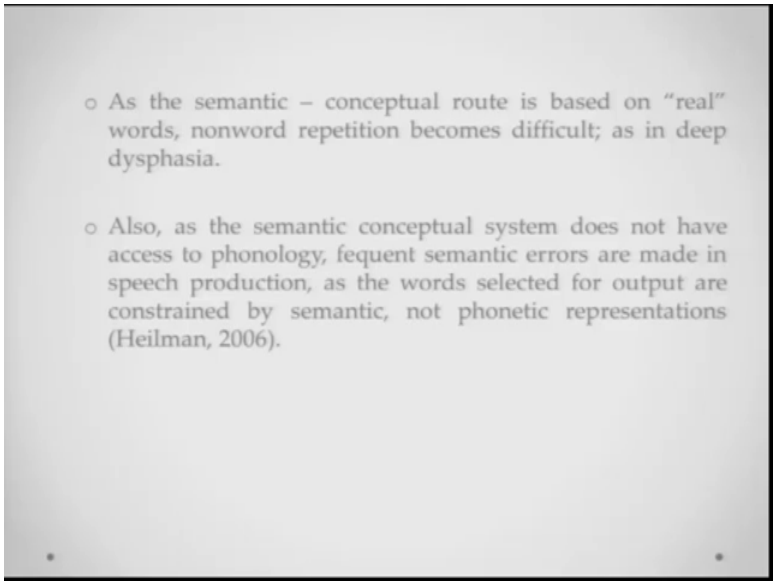

As [noise] the semantic conceptual route is based on real worlds, non word repetition will become difficult as actually happens in deep dysphasics. Also has the semantic conceptual system does not really have access [vocalized–noise] to phonology the fequent semantic errors will be made as an speech production, but because the words selected are constrained by semantics and not by phonetic representations.

So, they will be less phonetic errors when repetition is ask for, but they will be more semantic errors when repetition ask for. So, this is basically, this was how these models were developed.

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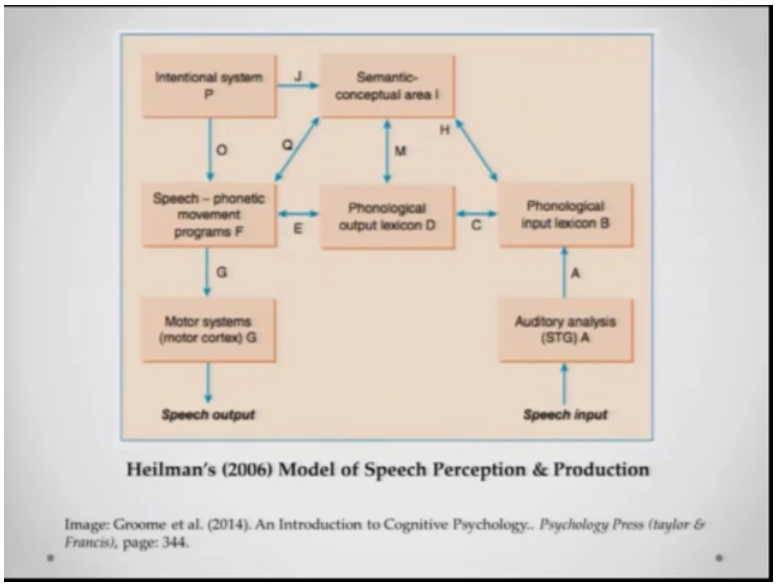

And this was the most recent model given by Heilman and this is basically one of the models which kind of [vocalized–noise] explains most of the profiles of aphasia that can be observed.

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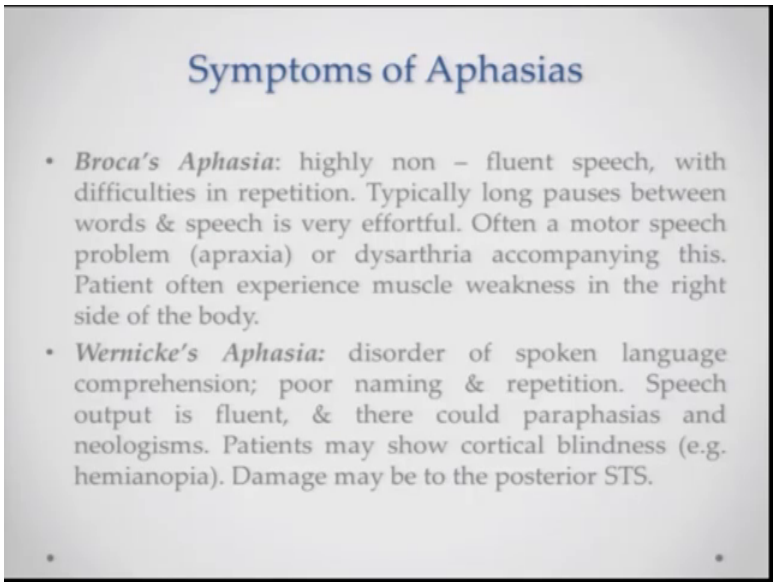

Now, [vocalized–noise] I just wanted to give you a brief review into the different kinds of aphasias and their symptoms also just hint about what kind of lesions they might be arising out of. So, the first and the most important Brocas Aphasia, [vocalized–noise] it is characterized by highly non fluent speech with difficulties in repetition. Speech is

characterized by a typically long pauses between words and it is very effortful and often you will see that these patients will basically experienced motor speech problem planning with speech acts or dysarthria accompanying this.

Patients also sometimes can experience muscle weakness in the right side of the body because see the left Brocas Area is in the inferior frontal cortex which is very adjusting to the motor area and because the left side of the brain controls right side of the body, that is one of the reasons why sometimes Brocas Aphasics could feel muscle weakness in the right part of the body or even paralysis of the right part of the body.

The next profile is of Wernickes Aphasia. Wernickes Aphasia is a disorder of language comprehension. It is characterized by poor naming and repetition speech output, on the other hand is fluent and they could also be sometimes you know paraphasias when people are you know speaking in jumbled words everything is getting mixed up in everything and also neologisms that, they are forming new words which are not there previously. Also some of these patients suffering from Wernickes Aphasia could show aspects of cortical blindness things like hemianopia, you know when there is visual neglect of one side of the visual feed.

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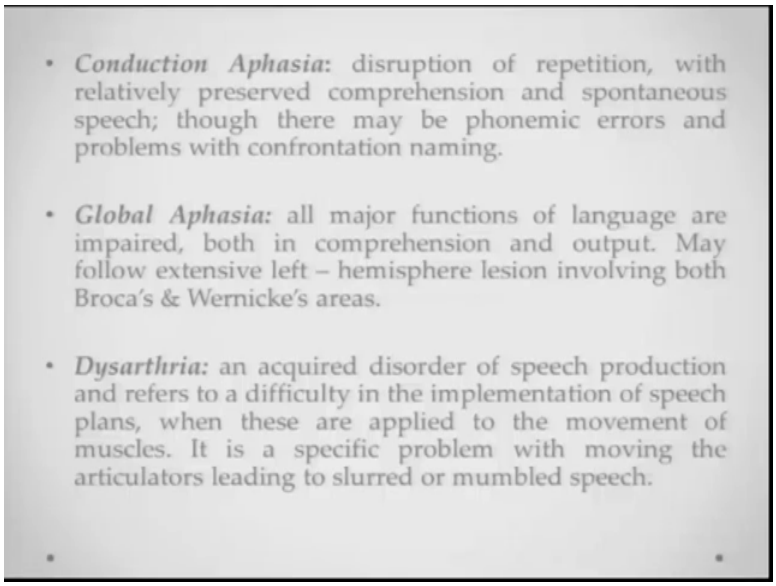

Wernickes Aphasia has been links to the posterior superior temporal sulcus. Then there is conduction aphasia. Conduction aphasia is basically a problem with repetition. We will relatively preserved comprehension in spontaneous speech, although the repetition might

be fraught with phonemic errors and also the errors problem with confrontation naming. When you show them pictures and you ask them to name these pictures then, you will see that these patients will have certain problems. Then there is global aphasia. In global aphasia, all the major functions of [vocalized–noise] language comprehension and production are effected and it is basically coming as a result of extensive left hemisphere lesions involving both the Brocas and the Wernickes Areas. Then there is dysarthria. Dysarthria basically is an acquired disorder of speech production and it basically refers to the difficulty in implementation of speech plans, as they are applied to the movement of speech muscles.

It is a very specific problem with moving the articulators that is, I was talking about the vocal track when we were talking about speech production. So, in dysarthria basically there is a problem between moving a manipulating these articulators which leads to slurred or mumbled speech.

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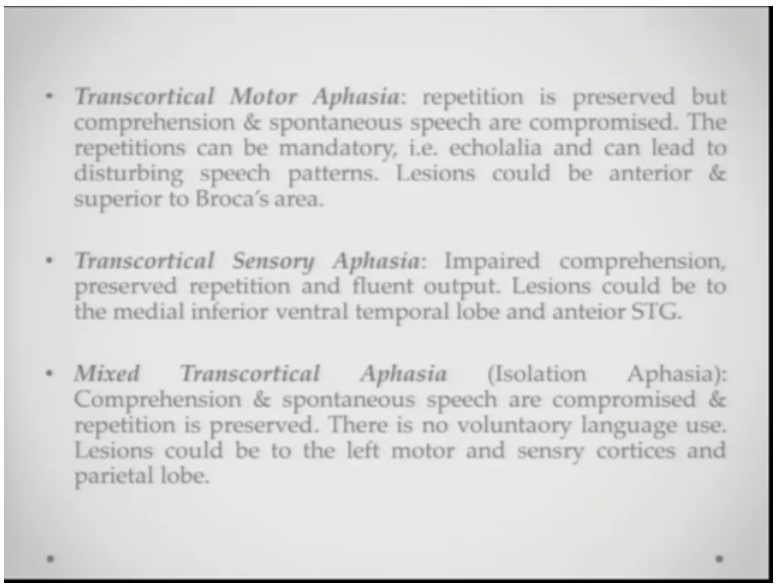

We talking about transcortical motor aphasia. Transcortical motor aphasia patients basically can repeat properly, but comprehension in spontaneous speech production or compromised. The repetitions are often mandatory in this case. So, patients display what is called echolalia, that the kind of you know keep repeating whatever is set to them or whatever they over here. It has been linked to lesions with on the interior and superior of Brocas Area. Then there is transcortical sensory aphasia which is basically link to

impaired comprehension, but the preservation of speech repetition and fluent output is there.[noise]

This could be because of lesions to the medial inferior ventral temporal lobe and the anterior superior lobe gyrus. Then there is the mixed transcortical aphasia or also referred to as the isolation aphasia. Comprehension and spontaneous speech are compromised, but repetition is preserved and there is no voluntary language used in patients who are suffering from mixed transcortical aphasia.

Mixed transcortical aphasia has been linked to lesions to the left motor and sensory cortices and also lesions to the parietal lobe.

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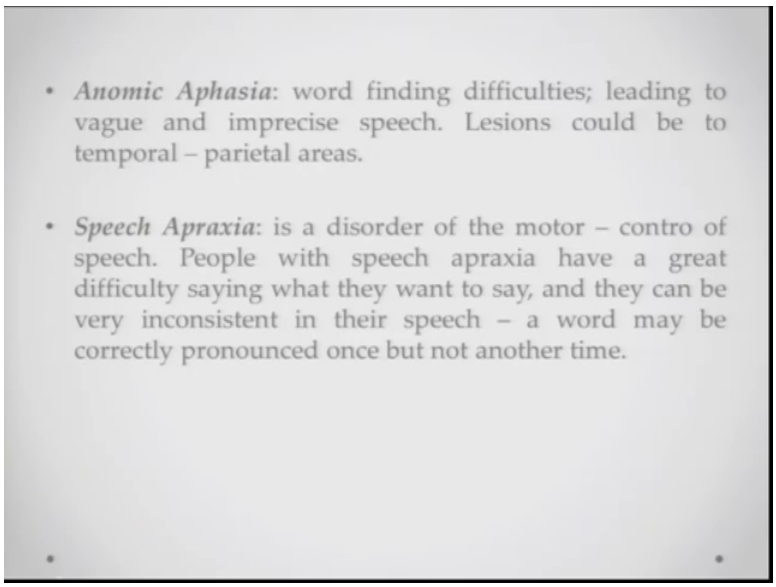

Then you have the anomic aphasia anomic aphasia is link to word finding difficulties, naming problems and it often leads vague and imprecise speech. This has been link to damage to temporal parietal areas. Finally, we will talk about speech apraxia. Speech apraxia is basically again disorder of the motor control of speech. People with speech apraxia have a great difficulty [noise] in saying what they want to say because they cannot plan the speech and they cannot execute the speech and this is one of the reasons they are very inconsistent in speech. A word may be correctly pronounced sometimes, but the next time they cannot really execute the movement associated with production of that kind of effort.

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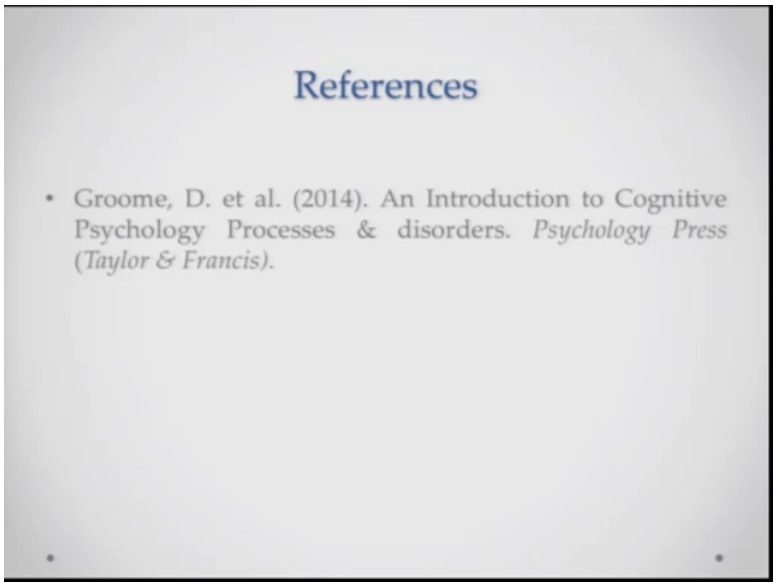

So, this is all from me on aphasias, I have not really gone into a lot of detail of it, but I have just try to give you a flavor of problems associated with language production.

Thank you. [noise]