AN INFORMATION PROCESSING OR COGNITIVE NEUROPSYCHOLOGICAL APPROACH TO UNDERSTANDING APHASIA: DIAGRAM MAKING

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Introduction (Gall and Broca)

The information processing or cognitive neuropsychological approach to understanding the speech/language disorders associated with brain dysfunction has as one of its major premises that the brain is organized in a modular fashion. The concept that the brain is organized in a modular fashion dates back to the early part of the nineteenth century when Franz Joseph Gall proposed that specific areas of the brain are important for mediating different cognitive functions. Unfortunately, based on this localizing hypothesis, together with the postulate that bigger is better, led to the pseudoscience of phrenology where practitioners believed that by palpating and measuring the skull one could determine a person's mental abilities. Although phrenology had been discredited, the postulate of localized function was widely discussed in nineteenth century Europe.

Paul Broca, a French physician-surgeon and anthropologist, was strongly influenced by Auburtin. During one of many discussions Auburtin stated that he, along with his father-in-law Bouillard, who was a student of Gall, believed that speech is mediated by the frontal lobes. According to Head (1926), so strong was his belief that he offered to recant his faith in the localization doctrine of Gall if anyone could show him a patient with a loss of speech who did not have a lesion of the frontal lobes. After hearing Auburtin make this comment Paul Broca invited Auburtin to see the patient Leborgne. This patient was recently admitted to the hospital because of cellulitis of his leg. He had a history of a loss of speech and a right hemiplegia. At the hospital the patient was known as "Tan" because that was the only word he could say. However, using this word he was able to express many of his emotions by superimposing prosodic intonations on the word tan. After Auburtin saw this case he agreed that the patient had a loss of speech and should have a lesion of the anterior lobes of the brain. Paul Broca noted that while Tan was unable to speak or write he was able to understand spoken language. About six days later the patient died and a post mortem examination revealed that the patient had a discrete lesion of the left hemisphere that was about the size of an egg. This lesion included inferior portions of the frontal lobe, portions of the insula, corpus striatum and the anterior superior temporal lobe. Broca presented this patient's brain to members of the Anthropology Society, thereby providing support to the localizationist postulates of Gall, Bouillard and Auburtin. The inability of this patient to speak according to Broca was caused by a disorder of the special faculty of articulated language. Paul Broca called this disorder of speech "aphemia." Subsequently, Paul Broca saw another patient with a fractured femur who also had aphemia. This patient also died and on post mortem
examination was also found to have a lesion of the second and third frontal convolutions providing further support for the localizationist approach. Paul Broca's contribution had a dramatic influence on thinking in all of Europe including England where he lectured and presented his findings. In 1864 Trouseau learned from a Greek physician that Broca's term "aphemia" meant infamous and therefore suggested the term aphasia. The aphasia named after Paul Broca is now a well recognized syndrome where patients are non fluent and have phonemic disintegration. However, there comprehension of speech is relative spared. Patients with this form of aphasia have syntactic disorders both in expression and comprehension. The may also have problems with working verbal memory. The may make phonological errors and also have problems with naming and repetition. Jay Mohr demonstrate that when a patients lesion is confined to the inferior frontal operculum including par triangularis and opercularis the impairament of speech may only be temporary and that a larger lesion that involves motor and insular cortex and subcortical structures such as the basal ganglia may be important for persistent defects. The observation that lesions restricted to the frontal operculum may not cause a persistent speech disorder does not contradict Broca's localization of this disorder but rather suggests that other areas may be important for compensation.

Paul Broca also observed several other patients, who had a right hand preference, and had aphasia associated with a right hemiplegia. Based on these observations Broca provided strong evidence that in humans speech is mediated primarily by the left hemisphere. This report provided strong evidence that in humans that Gall's localizationist or modular postulate was correct and that certain areas of the brain are important for mediating specific functions.

Wernicke's Model

It is not clear if Gall's hypothesis was the impetus, but during the later half of the nineteenth century there was an increase interested in neuroanatomy. Investigators such as Meynert demonstrated that sensory input to the cerebral cortex is predominately to the posterior portions of the cortex and motor output is mainly anterior. Accordingly, Broca's aphasia could be considered primarily an efferent disorder. Karl Wernicke in 1874 wrote what may be the most influential paper on aphasia which is called "Der aphasische symptomencomplex." In this article Wernicke contrasted Broca's (motor) aphasia with a sensory form of aphasia that is currently called jargon or Wernicke's aphasia. Unlike patients with Broca's aphasia who were non fluent, patients with sensory aphasia were fluent. Some patients may be so fluent that they have logorrhea. However, these patients spontaneous speech not only contains phonological and semantic errors but many pseudo or non words called neologisms. Some times there entire speech output is entirely comprised of neologisms such that it may sound like the patient is speaking a foreign language. Unlike patients with Broca's aphasics, patients with Wernicke's aphasia have a profound comprehension disorder, but like the patients with Broca's aphasia they may be impaired at naming and repetition. Whereas patients with Broca's aphasia have anterior perisylvian lesions patients with Wernicke's aphasia have posterior perisylvian lesions. Wernicke suggested that the critical area may be the posterior portion of the superior
temporal lobe, a portion of auditory association cortex. Wernicke posited that patients with sensory aphasia have lost the memory of how words sound. In the absence of this store of information, now called the phonological lexicon, words spoken to these patients would sound like a foreign language that they never learned. When attempting to name an object, they would be unable to recall the set of speech sounds or phonemes that represented this object. They would have this same problem when attempting to speak spontaneously. Because, in these patients with Wernicke's aphasia, Broca's area is intact and can program the production of phoneme sequences patients with this disorder can speak but the words used by these patients would not be constrained by lexical knowledge and therefore the patients would have jargon aphasia.

According to this formulation Wernicke not only suggested a second speech-language module but also was the first to posit an information processing network when he suggested that the area that contains the memories of how words sound (now called Wernicke's area) provides information to the area where the sounds of these words are programmed (now called Broca's area). Therefore, he suggested that the posterior portion of the superior temporal gyrus must be anatomically connected to Broca's area in the frontal operculum.

Based on his information processing system (see Figure 1), Wernicke further posited that if this connection between Broca's area and the posterior portion of the superior temporal gyrus were disconnected, by a lesion, the patient would also be aphasic. Because the phonological lexicon can not entirely inform Broca's area about the sounds (phonemes) by which words are constructed the production of words would be impaired with patients making phonological errors. However, unlike the patients with sensory (Wernicke's) aphasia who have destroyed their phonological lexicon and therefore, can not monitor their errors, patients with this disconnection disorder would be able to monitor their errors and may attempt to correct these speech errors. In this hypothetical form of aphasia the phonological lexicon has been disconnected from Broca's area, therefore, according to this information processing model these patients should also have problems with repetition and naming. However, because these patients maintain an intact phonological lexicon, in the posterior portion of the superior temporal gyrus, these patient should be able to comprehend speech. After Wernicke posited such an aphasia cases that followed his predictions were reported. The term used for this aphasic syndrome is conduction aphasia.

Although one of Wernicke's most important contributions was the development of information processing models, Henry Head (1926) dismissed this information processing approach as "diagram makers." He also thought that the diagram makers reports were biased. For example, when discussing one of Wernicke's reports Head wrote, "No better example could be chosen of the manner in which the writers of this period were compelled to lop and twist their cases to fit the Procrustean bed of their hypotheses." However, subsequent reports have replicated the model makers clinical observations and the information processing model approach has been demonstrated to be important for educational purposes, and have heuristic value. Just as it helped Wernicke predict
conduction aphasia, information processing models may allow one to develop hypotheses about how the brain works and what type of dysfunction may be seen with a variety of brain lesions. In spite of Henry Head's admonitions in the past three decades there has been a renewed interest in using information processing models to help explain cognitive deficits and to develop new a priori hypotheses.

**Kussmaul's and Lichtheim's Model**

About three years after Wernicke's influential paper Bastian (1869) and Kussmaul (1877) described a syndrome where the patient was unable to comprehend speech or repeat but could comprehend written language, name, and speak normally. As we mentioned above, Meynert demonstrated that auditory input from the thalamus projects to the primary auditory cortex which is on the dorsal surface of the superior temporal gyrus and that Wernicke posited that sensory or Wernicke's aphasia was caused by a lesion of auditory association cortex that is posterior to this primary auditory cortex. Based on the information processing model of Wernicke, the disorder described by Kussmaul, called *pure word deafness*, is thought to be related to an inability of auditory information to access an intact Wernicke's area.

Eleven years after Wernicke's influential report there was the third landmark paper written by Lichtheim and published in *Brain* (1885). In this paper, he first reviewed the seminal contributions of Paul Broca (1861) who described patients who had non-fluent speech with impaired repetition and naming, but with relatively preserved comprehension. He also reviewed the works of Karl Wernicke (1874) who described patients who were fluent, but made frequent paraphasic errors, used neologisms when speaking and had impaired comprehension, naming, and repetition. In this article Lichtheim proposed modifications of Wernicke's schema (see Figure 2). This new information processing model or schema contained "Wernicke's arc," that included; the primary auditory cortex, that performs an auditory analysis of spoken words; the posterior superior temporal gyrus (Wernicke's area), that contains the memories of how words sounded or what today is called the phonological lexicon; connections from Wernicke's area to the anterior perisylvian region where words sounds are programed (Broca's area) and then projections to the primary motor cortex. According to Lichtheim, in addition to Wernicke's arc, the speech cortex contains a region "where concepts are elaborated..." the conceptual or semantic field.

Before describing the cases that Lichtheim thought supported this model, he first explained how this system worked and also discussed an alternative schema, that of Kussmaul. According to Lichtheim, when one hears another speak, after auditory analysis, information goes to the phonological lexicon. After phonological lexical analysis, information is then transmitted to the area where concepts are elaborated (conceptual-semantic field). Repetition would take place much as Wernicke would have posited, namely, auditory analysis, phonological-lexical activation, transmission of this lexical information through the arcuate fasiculus with activation the motor-phonetic representations. These motor speech representations would then activate motor cortex.
According to Lichtheim, spontaneous speech starts with activating concepts that activate the motor representations of speech sounds and then the motor cortex.

According to Lichtheim's model, one could explain most of the aphasic syndromes that had been reported prior Lichtheim's paper. A lesion which interrupted auditory information from reaching the phonological lexicon would impair comprehension and repetition because auditory information could not reach the phonological lexicon and the area of concepts. However, spontaneous speech would be normal. This disorder as we discussed has been termed pure word deafness. Damage to the phonological lexicon would also impair comprehension, spontaneous speech, and repetition (Wernicke's aphasia). However, a disconnection between the lexical representations and motor representations would impair spontaneous speech and repetition but should not impair comprehension because the lexical representations could still get access to the region where concepts are elaborated (conduction aphasia). A destruction of the motor representation of speech would also not impair comprehension but would impair both spontaneous speech and repetition (Broca's aphasia).

Based on Lichtheim's schema, two additional types of aphasic syndromes may be posited. If there was a functional disconnection between the phonological lexicon and the area of concepts patients should not be able to comprehend; however, unlike Wernicke's aphasics, these patients should be able to repeat. To support his postulate, Lichtheim presents the case of J. U. Schwarz, a 60-year-old man who had an impaired ability to understand with a preserved ability to repeat.

Lichtheim's schema also suggests that interruption between the area of concepts and motor representations should also produce an aphasia. Unlike patients with Broca's aphasia, who are non-fluent and are impaired when repeating, these patients should have spared repetition. In support of this postulate, Lichtheim presents the case of Dr. C. K., who following a carriage accident, became non-fluent. Initially, he could only say yes or no. Although this patient was non-fluent, he was able to comprehend well. Unlike patients with Broca's aphasia, Dr. C. K., even early in the course of his disease, was able to repeat flawlessly.

Although Wernicke's schema could not account for these patients behavioral profiles, Kussmaul (1877) did present an alternative schema that could account for the patient who could not comprehend, but who could repeat. Kussmaul's schema is presented in Figure 3. Kussmaul's model differs from Lichtheim's in that the area of concepts can only gain access to the motor representations via the phonological lexicon. As in Lichtheim's schema, inability of the phonological lexicon to activate concepts would also induce a defect in speech comprehension. However, because Wernicke's arc would be spared, repetition would also be intact. Lichtheim, however, rejected Kussmaul's model. His rejection of this model was based upon an experiment he performed with a Broca's aphasic. After showing this patient objects he asked the subject to indicate by squeezing his hand how many syllables were in the word that denoted this object. He found that Broca's aphasics could not adequately perform this task. According
to Lichtheim's model, (Figure 2), conceptual representations can not directly access the phonological lexicon. Therefore after seeing the object and activating the area of concepts, motor representations are activated in concert with activation of the phonological lexical representations. Since the motor representations in Broca's aphasia are destroyed, the patient is unable to detect how many syllables a word may have, because they can not activate the phonological lexicon. In Kussmaul's model, the area of concepts directly accesses the phonological lexicon. Therefore, according to Kussmaul's model patients with destruction of the motor representations should have no difficulty in accessing the information stored in the phonological lexicon. Since the patients with Broca's aphasia that Lichtheim assessed appear to be impaired at accessing the lexicon, he rejected Kussmaul's model. Although knowledge of how a word sounds may be stored in the phonological lexicon deriving information about the number of sounds or syllables may require additional processing. With each new speech sound or phoneme there is a new movement of the articulatory apparatus. Therefore, knowledge of the number of sounds (phonemes) or syllables in a word may depend upon the successful articulation of the word (Heilman et al., 1996). Patients with Broca's aphasia may be impaired at this articulatory process and because they can not articulate, they may not be able to parse words into its phonological or syllabic components.

According to Lichtheim's model, when patients with Broca's or conduction aphasia are presented with pairs of pictures of objects and required to determine if the names of the two objects are the same (homophones), they should be unable to do so. They should be impaired at this homophone test because lesions in Broca's area or its connections to Wernicke's area should prevent the conceptual field from accessing the phonological lexicon, where word sounds are stored. Feinberg et al., (1986) demonstrated that patients with conduction aphasia could successfully perform homophone judgements on words that they could not vocalize. The evidence that these patients could access their phonological lexicons provides support against Lichtheim's model and for Kussmaul's model.

Lichtheim's model has two other possible flaws. According to this model (Figure 2) patients with a disconnection between the lexicon and the area of concepts would be impaired at comprehending speech but able to repeat just as he demonstrated in his case report of J. U. Schwarz. However, a review of this case revealed that this patient used incorrect words and occasionally mutilated words. He also had problems with naming. According to Lichtheim's model, the area of concepts has direct access to motor representations and the motor representations can access the phonological lexicon. Since these three areas are intact and interconnected, this patient should have had normal spontaneous speech and should have been able to name, but this patient was impaired.

Lichtheim's model, cannot also account for the patient with anomic aphasia who has an isolated impairment in naming both in spontaneous speech and in response to the presentation of stimuli in all modalities. Lichtheim was aware that his model could not account for anomic aphasia. He considered the interruption of the pathway between the area of concepts and motor speech representations could theoretically produce a defect in
naming. However, this is the same defect that he posited would cause decreased fluency with intact repetition and comprehension. This syndrome, currently called **transcortical motor or adynamic aphasia**, is usually not associated with a naming deficit. In addition, unlike patients with anomic aphasia, who are fluent, patients with this transcortical motor aphasia are non-fluent. Because he realized his model could not account for anomic aphasia, he tried to dispense with anomia as a specific aphasic subtype, "it seems to me questionable, however to place amnesia (anomic aphasia) on a par with the other phenomena of aphasic disturbance." He went on to state that "...it is not a sign of a focal lesion ..." Finally, he concluded, "the word amnesic aphasia had better be abandoned." Although Lichtheim is partly correct, when he noted that anomia is often a residual of many different forms of aphasia, it has also been clearly demonstrated that anomia, in isolation, can occur after a discreet lesion. Whereas patients with deficits in the semantic or conceptual field and in the phonological lexicon may be impaired at naming, such patients have deficits in either comprehension or repetition. The true isolated anomic aphasia should have normal comprehension and normal repetition. Unlike Lichtheim's model, Kussmaul's model has pathways that not only go from the lexicon to the area of concepts or semantics, but also another pathway that goes from the area of concepts to the lexicon. Impairment of the ability of semantics to access the phonological lexicon in Kussmaul's model may be the best explanation of pure anomic aphasia. This explanation of anomic aphasia presumes that a patient can have a one way disconnection or dissociation between the conceptual-semantic field and the phonological lexicon. If anomic aphasia is caused by the inability of the semantic field to access the lexicon with a preserved ability of the lexicon to access the semantic field one may also expect that the opposite one way dissociation to occur. Heilman, et al (1981) reported a patient who had impaired comprehension, but intact repetition, naming, and spontaneous speech. This patient's clinical profile (**transcortical sensory aphasia with intact naming and speech**) suggests a one way dissociation such that the phonological lexicon can not access semantics and the conceptual field but the conceptual field can access the lexicon.

**Updated Models**

Kussmaul's model can not only explain all the traditional aphasic syndromes including **transcortical sensory aphasia** (impaired comprehension, naming, spontaneous speech with intact repetition) from a complete functional disconnection between the conceptual-semantic field and the phonological lexicon or a destruction of the conceptual-semantic field. Kussmaul's model can also explain pure anomic aphasia and transcortical sensory aphasia with intact naming and speech. However, this model cannot explain transcortical motor aphasia, first described by Lichtheim, where patients are non-fluent but comprehend and repeat normally. The dorsolateral and medial frontal lobe together with the anterior cingulate gyrus and basal ganglia form what we have termed an intentional system. It is damage to these areas that are often associated with transcortical motor aphasia. There may be two forms of transcortical motor aphasia. In one there is an inability to activate the semantic-conceptual network (**adynamic aphasia**) and in the other there is a deficit of motor activation (**speech akinesia or extrasylvian motor aphasia Type II of Benson**). Nonfluency is the major sign in both of these disorders, but unlike patients
with Broca's aphasia these patients are able to normally repeat. These patients also have good comprehension. Whereas patients with adynamic aphasia may be mildly impaired at naming those with speech akinesia can name normally. Benson and Ardila (1996) have suggested there whereas adynamic aphasia is association with dorsolateral frontal lesions (superior to Broca's area) speech akinesia is more often associated with medial frontal lesions. In model illustrated in Figure 4 damage to the intentional system may induce both of these intentional disorders. According to this model a functional disconnection between the intentional system and the conceptual-semantic field would induce adynamic aphasia and a functional disconnection between these intentional systems and motor speech areas would produce speech akinesia.

There is another problem with Kussmaul's model. Several investigators have described a rare aphasic disorder called deep dysphasia (Katz and Goodglass, 1990). These patients are like conduction aphasics except that when asked to repeat, unlike the patients with classic conduction aphasic, who make phonological errors, these patients make semantic errors (e.g., good English words, but the incorrect words.) These patients also can not repeat non words. Kussmaul's model can not account for this syndrome. However, a further modification of the Wernicke-Lichtheim-Kussmaul's model can account for deep dysphasia. Cognitive neuropsychologists have suggested that the phonological lexicon may be composed of two separate but connected modules, a phonological input lexicon and a phonological output lexicon (Figure 4). According to this model there are two possible forms of conduction aphasia. One form could be caused by a functional dissociation between the phonological input and output lexicons and the second form could be caused by the functional dissociation between the phonological output lexicon and Broca's area. According to our modification of the Wernicke-Kussmaul-Lichtheim model the functional disconnection between the phonological output lexicon and Broca's area would cause the traditional conduction aphasia. However, with a functional disconnection between the phonological input lexicon and the phonological output lexicon one could access the phonological information in the output lexicon, indirectly by way of the semantic field. The semantic field codes meanings and concepts not phonology. Therefore, using this indirect route may lead to the semantic paraphasic errors associated with deep dysphasia. For example, when a patient with deep dysphasia is asked to repeat the word "sea" the phonological representation, activated in the input lexicon, may activate the nodes that represent the concept of a large body of water. Subsequently, when the nodes in the semantic-conceptual field, that represent a large body of water, accesses the output lexicon they may activate the word "ocean" and the patient may say "ocean" rather than "sea."

Optic aphasia is another aphasic syndrome for which the Wernicke-Kussmaul-Lichtheim model can not account. This disorder was first described by Freund (1889) who described a patient with a right hemianopsia who was unable to name objects presented in the visual modality. Although patients with optic aphasia can not name objects in the visual modality they can describe and pantomime the use of these objects. Their naming in other modalities is normal. Freund thought that this disorder was caused by a disconnection between the visual areas in the occipital lobe and the speech areas in
the left hemisphere, important in naming. According to the model we have presented in Figure 4, patient with optic aphasia would have a functional dissociation between the portion of the brain that contain object recognition units and the phonological lexicon. However, they can describe the use of objects because these object recognition units could gain access to semantic-conceptual field which can then access the phonological output lexicon. Based on this model the normal naming of objects may require that object recognition units gain direct access to the phonological lexicon. This same system may allow people to even name non-objects. Several years ago we saw several patients with degenerative dementia in our clinic who a clinical picture that was the reverse of optic aphasia. Unlike the patients with optic aphasia who can name to definition but not with visual confrontation these patients could name well with visual confrontation, but could not name to definition. We, therefore, called this disorder non-optic aphasia. We think these patients could name because their object recognition units could access the phonological lexicon and this intact lexicon could access Broca's area. Because their repetition was flawless we think that Wernicke's arc (primary auditory cortex-phonological input lexicon-phonological output lexicon-Broca's area-motor cortex) was intact. When these patients spoke spontaneously they had semantic jargon and these patients also had poor comprehension. Naming to definition, comprehension and normal speech all requires an intact semantic-conceptual field and we think these patient's non-optic aphasia was caused by degradation of the semantic field.

Conclusions

The modified Wernicke-Lichtheim-Kussmaul model illustrated in Figure 4 can explain most of the aphasic disorders we see in the clinic. However, this model primarily addresses the phonological, lexical and semantic deficits associated with aphasia and not other aspects, such as the disorders of syntax seen with certain aphasias (e.g. Broca's aphasia). It is also important to recognize that whereas Wernicke's model was developed a priori the modified model illustrated in Figure 4 is post hoc, in that it was developed to help explain the aphasic syndromes. Although techniques such as functional imaging has provided support for the postulate that speech and language is mediated by an anatomically distributed network, further research is need to validate, reject or alter the model that we have presented.
Figure #1
Wernicke's Schema

Legend:
a= auditory input and analysis
A= Wernicke’s area
M= Broca’s area
m= motor system and speech out
Figure # 2
Lichtheim's Schema

Legend:
a= auditory input and analysis
A= Wernicke's area
M= Broca's area
m= motor system and speech out
B = conceptual-semantic field
Figure # 3
Kussmaul's Schema

Legend:
a= auditory input and analysis
A= Wernicke's area
M= Broca's area
m= motor system and speech out
B = conceptual-semantic field
LEGEND

Pure Word Deafness = A
Wernicke's Aphasia = B
Deep Dysphasia = C
Conduction Aphasia = D and E
Broca's Aphasia = F
Aphemia = G
Transcortical Sensory Aphasia = I or H and J
Transcortical Aphasia with Intact Naming or Spontaneous Speech = H
Anomic Aphasia = J
Adynamic Aphasia = M
Akinetic Aphasia = O
Optic Aphasia = N
Non-Optic Aphasia = K and I
Global Aphasia = B and F
Mixed Transcortical Aphasia = H or I and P
Selected References

Bastian, HC. Aphasia and other speech defects. London: H. K. Lewis (1898).


Wernicke, C. Das Aphasische Symptomenkomplex. Breslau: Cohn and Weigart, 1874.