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Comparison Between Jakobson's and Grodzinsky's Models of Aphasia

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Aphasiology is the scientific study of aphasias. Many researchers tend to explore the symptoms and causes of different kinds of aphasias. Luria and Jakobson proposed their theories in *Ciba Foundation Symposium* in 1964. Then Jakobson classified the impairments of aphasias from a linguistic and structuralist point of view based on Luria's study. By contrast, Grodzinsky proposed trace deletion theory in terms of a generative point of view. Jakobson's model laid emphasis on the significance of linguistics methodology and knowledge in aphasia analysis and included all types of impairments of aphasias and provided a "clear-cut and symmetrical linguistic classification" with clear damaged areas in the brain. However, this classification which is based on the one-to-one relationship is not rigorous and cannot use some advanced technologies such as CT, MRI, MEG, and ERP method to explore patients' brains, leading to limited insight into possible brain regions affecting language abilities. By contrast, although Grodzinsky's trace deletion hypothesis in generative model could explain the comprehension problems of non-fluent aphasics to some degree, its assumptions that they could perform well in understanding active and other canonical sentences and that they will have a distinct pattern of comprehension performance and perform in a relatively neat pattern in different tasks are proved to be wrong. This analysis could show that Jakobson's model is more convincing.

Keywords: aphasia, model of aphasia, trace deletion hypothesis

Introduction

The symptoms of aphasias and what causes them have been explored by scientists for centuries. To solve this problem, some of them are dedicated to classifying and modelling different aphasias. Based on Luria's (1964) preliminary classification, Jakobson's (1964) study tried to add new classifications to the impairments of aphasias in terms of linguistic and structuralist points of view. By contrast, Grodzinsky's (1986) study modelled the impairments from a generative point of view. His famous hypothesis is the trace deletion hypothesis. This essay will compare these two models critically and argue that Jakobson's model is more convincing by analysing the advantages and limitations of the two models.

Advantages and Limitations of Jakobson's Model

A critical advantage of Jakobson's (1964) study is the emphasis on the significance of linguistics methodology and knowledge in aphasia analysis. When human beings have trouble in language production or

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comprehension, some functions in the brain must be damaged. These damages should be observed by the lesions in the brain. According to this logic, by analysing dysfunctional speeches of aphasiacs, the damaged functions and actual lesions in the brain and their correlation could be figured out. As stated by Jakobson (1964), because the verbal expressions and their defects are clearly in the field of linguistics, without which "the key to the most striking symptoms of aphasia" cannot be discovered (p. 21). Further, the experimenter of the aphasiacs must know some linguistic knowledge to conduct relevant experiments. If the experimenter is not adequately acquainted with the science of language, he will interpret the findings incorrectly, especially if his categorization criteria are derived from outmoded school grammars and have never been subjected to a rigorous linguistic examination (Jakobson, 1964). As a result, the study of aphasias should have a collaboration of scientists from different fields, especially linguistics.

The second advantage of his study is that he tried to include all types of impairments of aphasias and provided a "clear-cut and symmetrical linguistic classification" with clear damaged areas in the brain. The most important classification is the classification between selection (decoding) and combination (encoding). They are two basic operations in humans' verbal behaviour (Jakobson, 1964), so any impairments of aphasias could be included into this classification. It also highlighted the mutual influence relationship between impairments in selection and combination process. Impairments in any coding process will also impact the other process (Jakobson, 1964). This point is meaningful because extensive research has found that the Broca's area traditionally known as the cause of language production problems also has particular problems in language comprehension (Caplan, 2006; Caramazza & Zurif, 1976). This classification also differentiates selection and combination aphasias in terms of the levels of phonemes, meaningful units, and morphology. This can explain some speech disturbances in aphasia patients from linguistic point of view. For example, Fry (1959) noted that a patent read a sequence of words: *wood*, *kick*, *wear*, *feet*, he would substitute the consonant of the first word for those of the following words. This phenomenon could be explained by losing some abilities of combination in terms of phonemic level, so that encoding aphasiacs are hard to transit one phoneme to another.

However, Jakobson's (1964) study also has some limitations. Firstly, this classification is based on Luria's (1964) study which claims that different types of aphasias should be linked with specific lesions in the brain; for example, the sensory and afferent motor aphasias are linked with left temporal lobe's lesions and "post-central part of the cortex" respectively (p. 152). Luria (1964) claims that speech is supported by multitude zones in the cerebral cortex; every zone should contribute specifically to speech development. Nevertheless, the lesions and certain aphasic symptoms are not necessary to form a one-to-one relationship. A large examination of individuals with diverse patterns of language impairments and lesions of varying sizes in various brain regions reveals that there is no apparent association between symptoms and lesion location (Traxler, 2012). Caplan and Hildebrandt (1988) also argued that it is hard to figure out the clear correlation between lesions and symptoms after observing a large number of patients with brain damage and difficulty in language comprehension. Moreover, in Murdoch's (1988) neurological study, two patients were classified as Wernicke aphasics; however, their Wernicke's areas were not damaged. These studies show that it is not rigorous to link aphasic symptoms with specific lesions in the brain. Due to the complexity of the brain and the fact that every aphasic is relatively unique, there should be more scientific studies with enough samples and data or more advanced technology to figure out the correlation between the lesions and corresponding aphasiac symptoms. Jakobson's (1964) classification which is based on the one-to-one relationship is therefore not rigorous.

Secondly, Jakobson (1964) and Luria (1964) cannot use some advanced technologies such as CT, MRI, MEG, and ERP method to explore patients' brains, leading to limited insight into possible brain regions affecting language abilities. Their classifications may therefore be not accurate enough to describe the corresponding damaged parts of the brain. For example, Brunner et al. (1982) claim that the basal ganglia play an important role in determining the symptoms of Broca's aphasics; the patients can have the normal speech disturbance only when their basal ganglia are damaged. Further, by using CT and MRI methods to locate lesions of patients with speech apraxia, Dronkers (1996) maintains that the insula, as an important part in language production, could determine whether the patients suffering from strokes or tumors will have the apraxia of speech. These two studies prove that with the development of technology, the insight into human brains will subsequently change. Any theory trying to explain or classify aphasias should always optimize the most advanced technology and latest data. Basal ganglia and insula, two significant parts of brains in aphasiology, are not mentioned or located rather vaguely in Jakobson's (1964) classification, which will affect its applicability.

Advantages and Limitations of Grodzinsky's Model

As an important part of Grodzinsky' generative model of aphasia, the trace deletion hypothesis could explain why some kinds of sentences, for example, sentences involving fillers and gaps, cannot be understood by non-fluent aphasics without simply considering them lose the grammatic knowledge. The Broca's aphasia patients are usually considered to have production problem whereas they also have problems in comprehension, which is different from Wernicke's aphasias. It is argued by Caramazza and Berbdt (1978) that patients with agrammatical speeches have the fundamental problem with syntactic processing. This could easily attribute the comprehension and production problems of non-fluent aphasia patients to syntactic system; however, it seems not the case. Although the patients' performances are poor in the task of comprehension of sentences which are passive or have displaced elements, they perform well in judging their grammaticality (Traxler, 2012). Linebarger (1995) tested whether non-fluent aphasics could judge the different types of sentences and results show that they could correctly judge the grammaticality of sentences, far above chance. As a consequence, it can be argued that non-fluent aphasics preserve all (at least some) abilities in syntactic processing. If it is not the problem of syntactic processing function, what is wrong in the patients' minds?

The trace deletion hypothesis proposes that in patients with agrammatical speech, their Braca's areas are damaged and components of a sentence's syntactic representation that lack an obvious phonological form are deleted and "any task that recruits traces is bound to fail" (Grodzinsky, 1986, p. 243). Under this circumstance, it would be hard for the aphasics to assign semantic roles to the filler-gap sentences, thereby leading to inability to comprehend. This could explain why the filler-gap sentences cannot be understood by the aphasics. Further, the passive sentences can be regarded as the filler-gap sentences to some degree because the filler and the gap can be connected by a co-indexation operation in them. In this way, the non-fluent aphasics will have the same problem when processing the passive sentences as those with fillers and gaps (Traxler, 2012). In conclusion, the trace deletion hypothesis can fairly explain the comprehension problems with at least filler-gap and passive sentences in non-fluent aphasics.

However, this hypothesis also faces a lot of criticism. One potential problem is that the hypothesis' premise that agrammatic aphasics have no difficulty in understanding active and other canonical sentences appears to be unjustified (Traxler, 2012). For example, Hickok, Zurif, and Canseco-Gonzalez (1993) tested

comprehension of a patient with the non-fluent aphasia by using the sentences which are similar to *The tiger that chases the lion is big*. Considering that sentences of this kind cannot use the trace deletion hypothesis because they do not have fillers and gaps, non-fluent aphasics are expected to comprehend them accurately; however, a patient performed as poorly in comprehending those sentences as sentences with long-distance dependencies (Traxler, 2012). Another example could be found in Schwartz, Saffran, and Marin's (1980) study, in which they tested the understanding of sentences similar to *The box is in the cage* in five agrammatic aphasiacs and found that they have trouble in interpreting those canonical sentences. These two studies show that the trace deletion hypothesis fails to explain non-fluent aphasics' understanding problems in canonical sentences.

Furthermore, according to the trace deletion hypothesis, non-fluent aphasics will have a distinct pattern of comprehension performance. Comprehending passive and object relative sentences will be harmed as a result of the fillers and gaps while sentences in the active voice will remain comprehensible since they do not contain fillers or gaps (Traxler, 2012). However, empirical evidence from non-fluent aphasia demonstrates that this clean dichotomy does not correctly explain how non-fluent aphasiacs respond to various types of sentences (Caplan, DeDe, & Michaud, 2006). Some non-fluent aphasiacs can comprehend filler-gap sentences with high accuracy while some patients perform rather poorly in comprehension of canonical sentences (Caramazza, Capitani, Rey, & Berndt, 2001).

Another problem with this hypothesis is that it predicts the non-fluent aphasics will perform in a relatively neat pattern in different tasks; for example, non-fluent aphasics cannot perform well in any task containing long-distance dependencies because they are not able to process them. However, this prediction is wrong. The task performance of non-fluent aphasias could be tested separately through comprehension and grammaticality judgement tasks (Traxler, 2012). Non-fluent aphasiacs perform poorly in lots of comprehension tasks whereas performing well in the grammaticality judgement tasks of the sentences of the same types (Caplan & Hildebrandt, 1988; Grodzinsky, 1995; Linebarger, 1995). Furthermore, comprehension ability can even be tested through different types of tasks. Based on trace deletion hypothesis, the non-fluent aphasics should have trouble in all types of comprehension tasks of the same type of sentence (e.g., passive voice sentences). However, this could be rebutted by Caplan's (2006) study. Caplan (2006) tested 42 patients who can be classified as non-fluent aphasiacs; however, only one patient has comprehension problems of the passive and object relative sentences in various tasks while having no problem in comprehending other sentence type. As a result, the trace deletion hypothesis fails to explain this phenomenon; therefore more research needs to be done to provide more details and explanations.

Conclusion

Jakobson's structuralist model emphasizes the significance of linguistic methodology in analysis of aphasia and could provide a relatively comprehensive classification of aphasias in terms of linguistic point of view, despite ignoring the complex correlation between lesions in the brain and aphasic symptoms and some important parts of brain related to the symptoms due to the lack of advanced technology. By comparison, although Grodzinsky's trace deletion hypothesis in generative model could explain the comprehension problems of non-fluent aphasics to some degree, its assumptions that they could perform well in understand active and other canonical sentences and that they will have a distinct pattern of comprehension performance and perform in a relatively neat pattern in different tasks are proved to be wrong. Therefore, Jakobson's model is more convincing than Grodzinsky's model.

References

Brunner, R. J., Kornhuber, H. H., Seemüller, E., Suger, G., & Wallesch, C. W. (1982). Basal ganglia participation in language pathology. *Brain & Language, 16*, 281-299.

Caplan, D. (2006). Aphasic deficits in syntactic processing. *Cortex, 42*, 797-804.

Caplan, D., & Hildebrandt, N. (1988). *Disorders of syntactic comprehension*. Cambridge, MA: MIT Press.

Caplan, D., DeDe, G., & Michaud, J. (2006). Task-independent and task-specific syntactic deficits in aphasic comprehension. *Aphasiology, 20*, 893-920.

Caramazza, A., & Berndt, R. S. (1978). Semantic and syntactic processes in aphasia: A review of the literature. *Psychological Bulletin, 85*, 898-918.

Caramazza, A., & Zurif, E. B. (1976). Dissociation of algorithmic and heuristic processes in language comprehension: Evidence from aphasia. *Brain & Language, 3*, 572-582.

Caramazza, A., Capitani, E., Rey, A., & Berndt, R. S. (2001). Agrammatic Broca's aphasia is not associated with a single pattern of comprehension performance. *Brain and Language, 76*, 158-184.

Dronkers, N. F. (1996). A new brain region for coordinating speech articulation. *Nature, 384*, 159-161.

Fry, D. B. (1959). Automatic speech recognition: A review. *Language and Speech, 2*, 52.

Grodzinsky, Y. (1986). Language deficits and the theory of syntax. *Brain & Language, 27*, 135-159.

Grodzinsky, Y. (1995). A restrictive theory of agrammatic comprehension. *Brain & Language, 50*, 27-51.

Hickok, G., Zurif, E., & Canseco-Gonzalez, E. (1993). Structural description of agrammatic comprehension. *Brain & Language, 45*, 371-395.

Jakobson, R. (1964). Towards a linguistic typology of aphasic impairments. *Disorders of Language, 21*, 42.

Linebarger, M. C. (1995). Agrammatism as evidence about grammar. *Brain & Language, 50*, 52-91.

Luria, A. R. (1964). Factors and forms of aphasia. *Disorders of Language, 143*, 161.

Murdoch, B. E. (1988). Computerized tomographic scanning: Its contributions to the understanding of the neuroanatomical basis of aphasia. *Aphasiology, 2*, 437-462.

Schwartz, M., Saffran, E., & Marin, O. (1980). The word order problem in agrammatism: I. Comprehension. *Brain & Language, 10*, 249-262.

Traxler, M. J. (2012). *Introduction to psycholinguistics: Understanding language science*. New York: Wiley-Blackwell.