Dementia-induced language deficit

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ABSTRACT

This study attempts a comparison between language disorders induced by dementia as manifested in Alzheimer’s and Parkinson’s disease. It allows for a comparison of the cognitive and linguistic decline in each of the two forms of brain damage and argues that both AD and PD population suffer from impaired speech with uncommon content. Patients with dementia show greater weakening of the ability to encode meanings or to connect cognition with language. The study concludes that, contrary to past convictions, language deficits are important indications of cortical and subcortical dementias as manifested in AD and PD.

Key words: Alzheimer’s, Parkinson’s, cognition, dementia, aphasia, synapse.

INTRODUCTION

As a generic term, dementia is the result of deterioration in mental, linguistic and cognitive abilities (Bier, 2019). Although the precise cause of dementia is yet to be determined, it seems to be largely connected to atrophy in different parts of brain tissue. Dementia-related illnesses may be the direct result of the loss of brain chemicals and the diminishing of cerebral gray matter which happens when neurons become affected and die without replacement (Sugunendran, 2020). However, unlike Wernicke’s aphasias, dementias do not trigger obvious brain damage in distinctly localized areas; instead, they relate to general brain atrophy. Recent studies have shown that poor cross-neuronal communication can be responsible for dementias where electric neuronal impulses, unable to cross the synaptic cleft to the postsynaptic cell, are constrained in the presynaptic cell.

A synapse in the nervous system is a structure that permits a neuron (or nerve cell) to pass an electrical or chemical signal to another neuron or to the target effect or cell. Synapses are essential to neuronal function: neurons are cells that are specialized to pass signals to independent target cells, and synapses are the means by which they do so. At a synapse, the plasma membrane of the impulse-generating neuron (the presynaptic neuron) comes into close approximation with the membrane of the target (postsynaptic) cell. Both the presynaptic and postsynaptic sites contain extensive arrays of molecular machinery that link the two membranes together and carry out the signaling process. In many synapses, the presynaptic part is located on an axon and the postsynaptic part is located on a dendrite or soma. Astrocytes also exchange information with the synaptic neurons, responding to synaptic activity and, in turn, regulating neurotransmission. Synapses (at least chemical synapses) are stabilized in position by synaptic adhesion molecules (SAMs) projecting from both the pre- and post-synaptic neuron and sticking together where they overlap. SAMs may also assist in the generation and functioning of synapses as shown in Figure 1.

METHODOLOGY

To establish the correlation between language deficits and dementias as manifested in Alzheimer’s and Parkinson’s diseases, I will adopt the analytic, qualitative method throughout this study. This is crucial to prove that language impairments are a clear indication of dementias.

RESULTS

Based on previous studies, the present study presents compelling evidence that in both cortical and subcortical dementias speech deficits may arise. Demented individuals seem to experience subtle linguistic deficits including mild anomia, alexia, agrammatism, in addition to problems in list
generation, morphological affixes and sentence encoding and comprehension. The study shows that in cortical dementias, language problems are more noticeable. Although the patient's phonology, surface-level syntax and reading aloud remain relatively intact, his lexicon and semantics are severely impaired. Further, the patient's simple pragmatic abilities are spared until a relatively late stage of the disease, but more complex ones, for example, inference or following a conversation, virtually disappear.

Further results indicate that cognition, memory, attention and the ability to manipulate ideas may also cause linguistic decline. These are linked to frontal, parietal and temporal (FPT) lobes where cellular changes are most severe. Language deficits can happen in isolation of thought, but thought cannot be impaired in isolation of language deficits.

**DISCUSSION**

Dementias fall into two types: cortical and subcortical. In cortical dementia, damage is sustained by the cortical area, for example, the gray matter distributed over the surface of the cerebral cortex and may give rise to Alzheimer's disease which accounts for approximately 50% of all cases of dementia. This means that approximately 35 million people worldwide have Alzheimer's disease and these estimates are expected to reach 100 million by 2050 if cure is not found (Town and Kassel, 2015). In addition to personality changes, Alzheimer's symptoms may include language deficit, memory loss, problems performing new tasks using knowledge already familiar to the patient. As the disease progresses, the patient shows symptoms of confusion, irritability, aggression, and mood fluctuations and ends with a total loss of long-term memory (Sugunendran, 2020). By contrast, sub-cortical dementia is the result of damage to the sub-cortical white matter, for example, the brain tissues located in the deep layers of the cerebral cortex. This accounts for 1/3 of PD patients who experience kinesia paradoxa (difficulty in performing simple movement) with problems in walking and speaking. As the disease progresses, PD patients' speech may become muttered and unintelligible. Language changes are more subtle in Parkinson's than in Alzheimer's disease.

The study of dementia offers psycholinguists an opportunity to study the language-cognition interface and to determine how far they are dependent on, or independent of, each other. Investigating language production and comprehension in patients with dementia forms the essence of exploring the subtle relationship between syntax and semantics and between semantics, knowledge of the real world and reasoning abilities. Diagnosis of patients with dementia or Wernicke's aphasia has always been elusive. In both cases patients display similar symptoms of using empty words, (examples, this, thing, etc.) and nonsense phrases in their speech. Similarly, patients with Wernicke's aphasia and dementia have problems using functors in particular; for instance, the former uses a subset of functors vis-à-vis emptier ones such as and, whereas the latter is more likely to use a broader range of functors. This difference helps distinguish aphasia from senile dementia of Alzheimer's type (SDAT).

**Wernicke's aphasia or Alzheimer's dementia**

It is not easy to determine whether patients have genuine latent knowledge of their linguistic output since it is not enough to rely on the patient's language deficits to know if
these alterations belong to Wernicke’s aphasia or they are of the type language-plus-cognitive alterations generated by Alzheimer’s dementia. In this respect, the patient’s history of a more general cognitive and behavioral deterioration may be helpful. If such history is available, chances are we are dealing with Alzheimer’s dementia. The patient’s language reflects both cognitive and linguistic impairments. However, if impairment is sudden with no history of memory or other cognitive problems, then we are dealing with Wernicke’s aphasia.

A brain degenerative, progressive complex, Alzheimer’s disease is the result of deterioration in attention abilities; it diminishes neurons and causes them to waste away (degenerate) and die without replacement. That is why it is the most common cause of dementia — a progressive deterioration in the ability to think and exercise common behavioral and social skills that disrupts a person’s ability to function independently. Compelling evidence is accumulating within the interactive/connectionist school of language processes, where attention plays the role of a controlling factor within the language system, indicating that language impairments in patients with dementia of Alzheimer’s type (DAT) are caused by specific attentional deficits (Stemmer and Whitaker, 1998). Some of these patients are unable to inhibit partially stimulated information, which causes them to mispronounce out-of-the-way words during reading and large priming and to have inhibitory effects in lexical decision tasks (Balota and Ferraro, 1993, 1996).

In the early stages of (SDAT) the patient forgets recent events or conversations and keeps repeating himself in a loop. At a later stage, a person with Alzheimer’s disease will develop mild anoma and severe amnesia (memory loss) and may lose the ability to carry out routine tasks (like doing up his buttons) and his ability to speak (Martin, 2006). With his speech muttered, broken and incomprehensible, the patient appears to be laboring under the disability to express himself; consequently, he may become aggressive and paranoid with totally unpredictable behavior. Describing the extent of the suffering his mother had to endure after she was inflicted with Alzheimer’s disease, Jerry Beller says:

"Not content to kill, Alzheimer’s tortured his Mom for years before killing her. It robbed her memory and damaged her brain, where she repeated herself in a continuous loop each time thinking she was saying it for the first time. As the disease advanced, the neurological disorder destroyed her mind and body" (Beller, 2020).

**Subcortical dementia**

Subcortical dementia manifests itself more commonly in Parkinson’s disease which generally hinders the ability to speak. It is a progressive nervous system disorder that debilitates movement. The symptoms start gradually, sometimes with a barely noticeable tremor in just one hand. As the disease progresses, tremors become more noticeable, but the disorder also commonly causes stiffness and slows down movement. The disease impacts the brain primary motor cortex and makes the patient slow in everything including the control of his organs of speech as shown in Figure 2. Note that the thought of cognitive degeneration is not rare with PD, it is even common before symptoms and unlike free recall, recognition memory may be unimpaired, which suggests that retrieval may be more problematic than encoding (Martin 2006, Whitworth and Whitworth, 2015). That is why patients suffer from dysarthria (difficulty of producing speech sounds) as well as dysgraphia (the inability to write). Micrographia (writing in very small letters and inappropriate space) is not uncommon. This is an indication of the loss of muscle
control without any damage to the brain representation of language.

Cognitive changes

Cognitive changes happen in 1/3 of the patients with Parkinson’s disease who also have dementia. These patients suffer from memory problems and may have difficulty in operating knowledge they already have and in addition to cognitive deficits, subtle language changes may also be part of the complex manifestations of PD. Errors on morphological endings are an indication of dementia which is not simply due to inattention. Additional lexical problems in patients with Parkinson’s include difficulty of learning new words (syntactic, semantic or phonological alexia). Linguistic disorder appears also in lexical selection errors although developmental alexia has frequently been described in subjects not suffering from neurological disorders (Shallice and Cooper, 2011). Recent studies suggest that what used to be described as bradyphrenia, or slow thinking, (poor cognitive abilities subtle alterations and deficit in attention, memory and procedural learning) are, at least partially, AD related. Furthermore, similarities between the cognitive disorders of PD and those caused by frontal lesions suggest that deficits in so-called executive functions are part of the clinical picture of PD (Pillon et al., 1986).

With their senses and mental abilities still intact, patients with PD may have problems processing sentences. Recent studies point out that subcortical structures involved in PD are not confined to language impairment but may affect language processes as well (Crosson, 1992). Such findings seem to bear out the claim that language deficit is a strong indication of PD. Memory and attention problems seem to be related to poor distribution of dopamine to areas of the prefrontal lobe involved in cortical networks for sentence comprehension (Grossman et al., 1992). (Dopamine is a natural chemical that occurs in the human body and controls its movements and emotional responses. It is vital for the individual’s physical and mental wellbeing).

Cortical dementia

Manifested in Alzheimer’s disease, patients with cortical dementia experience aphasia-like symptoms as damage is sustained by both the temporal and the frontal lobes. The patient’s speech lacks cohesion and is repetitive, empty and characterized by too many topic shifts. Though word order remains intact, patients display syntactic, lexical and morphological errors with wrong subject-verb agreement.

The question that remains yet to be answered is whether the grammar of the patient is affected by his dementia or by the loss of memory, inattention and lack of self-monitoring. Admittedly, the answer to this question is not an easy task. I believe we must deal with each individual case and linguistic deficit separately. The patient starts to show lexical problems in the early stages of dementia. The “tip-of-the-tongue” situations are clear indication that the missing word has not been erased from the patient’s lexicon. Later, the patient, unprompted, regains the missing word. Patients remember some information, though not always accurate, about the word like the number of syllables, sounds, letters, and stress patterns. Remembering the missing word without re-learning it means that the problem is one of retrieval of representation rather than in the representation itself.

Note that in patients with dementia, it is not possible to use the-tip-of-the-tongue data to be sure whether the problem is with retrieving the words or with the expunging of the actual words themselves. As Controversial as it is, one way of resolving this issue is to see if a given word is constantly missing across modalities (that is, from the writing system as well as the auditory system) since patients with Alzheimer’s disease exhibit a consistent loss of lexical items. Another way of telling whether the word is lost from the lexicon is by a word-to-picture matching test where even the wrong choices may furnish some information about the patient’s knowledge of the correct word. Suppose the patients were presented with a picture of a pigeon and five words to choose from. If the patient chose a semantically related word to the picture, this would indicate that the picture was meaningful for him though he could not choose the correct lexical item which is a question of difficulty in access rather than a lost representation as shown in Figure 3.

As has been reported in a number of cases, consistency in performance is extremely important where the same names that DAT patients failed to recognize in a picture-name task were the ones they failed to recognize in a word-to-picture matching test (Huff et al., 1986). Consistency in performance is in flagrant contrast with the absence of consistency across a range of semantic tests in a population of aphasic patients with impaired language comprehension (Butterworth et al., 1984). Consistency measures suggest that DAT patients suffer from a real breakdown of semantic representation, whereas in aphasic patients, retrieval impairment is more likely than a semantic breakdown. However, since consistency has not been thoroughly tested, these effects need to be duplicated.

In demented patients, paragrammatic errors (errors that evidence disturbed grammatical representation) occur regularly. Errors may include omissions, substitutions, repetitions, wrong inflectional endings, etc. Such errors, commonly referred to as agrammatism, indicate disturbance in the grammar itself.

In Alzheimer’s disease, automatic language abilities are spared until quite an advanced stage of the disease. Patients can read aloud with no difficulty even when they are making errors in naming and having empty speech. Patients may have problems with irregularly spelled, low frequency
words. This may distinguish patients with Alzheimer's disease from those with Wernicke's aphasia who try to evade them (Obler and Gjerlow, 1999). However, patients still exhibit semblance of normal behavior: thanking, complimenting, greeting their acquaintances, etc. However, in the progression of their demented illness, patients are likely to develop difficulties in observing principles of appropriate quantity, relevance and manner. Aspects of conversational abilities are impaired to various degrees and conversation breakdown may be developmental.

The intricacies of knowing whether impairment is caused by an access problem or degraded representations are all too evident in patients with AD. It has been argued that these patients suffer from a diminished semantic knowledge. However, priming effects, as suggested by most studies of DAT patients, seem to have significant impact on retrieval impairment (Chenery, 1996). Possible reasons for this irregularity of results are that most priming studies with DAT patients have used only associatively-linked words, and that the presentation environments of some studies have been inadequate to guarantee the contribution of automatic diffusion of activation to the priming effects, and that still other studies have found incredibly enormous priming effects (hyperpriming).

Hyperpriming is indicative of a representational deficit under the assumption that degraded representations benefit more from spreading activation provided by the prime than unaffected semantic representation (Chertkow et al., 1989). However, in her review, Chenery (1996) has argued that even when these factors are taken into consideration, the weight of evidence still favors an account in terms of an access impairment. Additionally, finer-grained studies are needed to resolve the remaining inconsistencies and to determine the functional locus of semantic impairment in DAT patients.

CONCLUSION

In subcortical dementias, Parkinson's disease may result in speech problems (not language). Subtle problems can be seen in naming, lexical selection, list generation, affix morphology and sentence comprehension. In cortical dementias Alzheimer's disease, language problems are more pronounced. Phonology, surface-level syntax and reading aloud are relatively spared, whereas lexicon and semantics are severely impaired. Simple pragmatic abilities are spared until relatively late in the course of the disease. More complex ones, for example, inference or monitoring what the other participant in a conversation already knows disappear.

Non-language aspects of cognition, examples, memory, attention and the ability to manipulate ideas contribute heavily to apparent linguistic decline. These are linked to frontal, parietal and temporal (FPT) lobes where cellular changes are most severe. Language can be impaired independently of thought, but thought cannot be impaired without language impairment.
REFERENCES


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