Analyzing the oral speech of an Alzheimer affected person: A Case Study

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Abstract
This paper describes the case study of former British Prime Minister Harold Wilson’s oral interventions (1964-1970 and 1974-1976), potentially affected by Alzheimer disease (AD) at the time of resignation (Garrard, 2009). To pursue this research, the oral interventions of Harold Wilson are examined. One advantage of analyzing Harold Wilson is that all his Parliamentary interventions are available online (Hansard transcripts) and the context in which the speech is produced is held constant across the different years. The amount of data is divided into random-selected 25,000 words for each year of Wilson’s 10-years at office (1964-1970 and 1974-1976); totalling 250,000 tokens. To determine the extent of differences in the linguistic patterns before and after being AD affected, bigrams and trigrams are analysed in order to investigate whether different periods exhibit characteristic oral features. We are confident that this longitudinal study and pattern analysis might provide us with some new understanding of the evolution and progression of language deterioration in AD.

Keywords: Clinical linguistics, corpus linguistics, AD, speech analysis, language deterioration

1. Introduction
Alzheimer’s disease (AD) is a progressive, degenerative disease of the brain, which causes thinking and memory to become seriously impaired. It is the most common form of dementia. The disease was first identified by Dr. Alois Alzheimer in 1906. He described the two hallmarks of the disease: plaques –numerous tiny dense deposits scattered throughout the brain which become toxic to brain cells at excessive levels– and tangles which interfere with vital processes eventually “choking” off the living cells. As well, when brain cells degenerate and die, the brain markedly shrinks in some regions.

As the disease progresses, nerve cells in several brain areas shrink and die, including cells that normally produce critical neurotransmitters 1, the chemical messengers that relay brain signals from one nerve cell or neuron 2 to another. Acetylcholine 3 is a neurotransmitter that is deficient in people with AD. As nerve cells continue to die, the brain itself shrinks and the wrinkles along its surface become smoother.

AD is a progressive and irreversible decline in a range of cognitive abilities, typically beginning with episodic memory (Galton et al., 2000). The most striking early symptom is memory loss,

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1 A substance (such as norepinephrine or acetylcholine) that transmits nerve impulses from one cell to another across a synapse.
2 One of the cells that constitute nervous tissue, that have the property of transmitting and receiving nervous impulses.
3 A neurotransmitter released at autonomic synapses and neuromuscular junctions.
usually manifest as minor forgetfulness that becomes steadily denser with illness progression. The preclinical stage of AD is inconspicuous and there are almost by definition no reliable and valid symptoms and signs which would allow a very early diagnosis before the manifestation of irreversible deficits (Förstl and Kurz, 1999).

More and more researchers are paying closer attention to the early stages of AD, when there is a greater chance of enabling patients to lead normal lives longer. Positive results may improve diagnostic techniques and drugs (e.g. cholinergic drugs) that can slow the degenerative brain disease right in its initial phases. New ways of detecting AD are among the biggest breakthroughs, since treatment is most effective early on. Until recently scientists thought the only way to be sure of a diagnosis was by examining brain tissue –after the patient had died.

No doubt that early diagnosis is quite critical. The primary symptom of mild cognitive impairment (MCI) is persistent trouble recalling information and events. The challenge and importance of pre-diagnostic AD detection is very much related to MCI, where patients may be aware of their symptoms and able to report on their own cognitive dysfunctions. Some studies have already demonstrated that the use of patients’ language samples dating back years, or even decades, before the clinical confirmation of AD might be a useful source for detecting MCI (Snowdon, 2003; Garrard et al., 2005; Garrard, 2009; Lancashire and Hirst, 2009).

2. Language and AD

There are many evidences that suggest that language is very vulnerable to MCI at the earliest stages of AD (Groves-Wright et al., 2004; Forbes-McKay and Venneri, 2005; Garrard et al., 2005). Language decline is probably one of the fastest and most predominant changes in primary progressive aphasia present at the earliest stages of AD, and associated with global cognitive deficits: decreased speech output, reduced conversational initiation, echolalia 4 and changes in the pragmatics of conversation.

Various authors have examined and compared the language in normal aged people with AD affected ones. Major changes were found in vocabulary richness (Maxim and Bryan, 1994): in normal aging, the mental lexicon continues expanding indefinitely; however, lexical retrieval becomes slower. In contrast, AD affected people have more serious problems in lexical retrieval and the mental lexicon becomes eventually inaccessible.

Maxim and Bryan (1994: 46) also found differences in the use of indefinite words (i.e. thing, matter, something, someone, etc.); a modest increase is found in healthy normal aging, while patients with AD show a dramatic overuse of indefinite words (Nicholas et al., 1985).

Word repetition and phrase repetition have also been found different between normal aging and AD; healthy people evidence some increase; however, AD patients show greater increase in repeating words and phrases (Nicholas et al., 1985).

Regarding syntax, Bates et al. (1995) discovered significant differences in normal aging compared to AD; where normal aging has no effect on the range and variability of syntactic structures used, AD patients underused passives and embedded sentences (Ellis, 1996).

Discourse also exhibits differences between normal aging and AD. Data obtained from AD patients reveal incoherence, sentence shortening and uncontrollable repetition of a particular

4 The repetition of vocalizations made by another person.
response, such as a word, phrase, or gesture, despite the absence or cessation of a stimulus (Harnish and Neils-Strunjas, 2008).

3. Research aim

Previous research (Cantos, 2010) points towards the hypothesis that Harold Wilson might have been aware of his MCI, and might have started using various surrogate measures of his cognitive reserve as an attempt to cope with his cognitive dysfunctions. These substitute measures might have included the use of pre-existing cognitive processing approaches or enlist compensatory approaches such as (i) accessing more common and frequent words; and (ii) using repetitively “prefabricated” chunk of words. Wilson might have had more cognitive reserve and was “more successful” than other AD affected people at coping with the same amount of brain damage. This might also explain why his fellow MP’s at the House, press, etc., did not realise anything about his onset of AD.

The possibility to identify and measure the very earliest stage of AD could provide important insights into cognitive reserve (Garrard, 2009). The cognitive reserve model suggests that the brain actively attempts to cope with brain damage by using pre-existing cognitive processing approaches or by enlisting compensatory approaches (Stern, 2003). Individuals with more cognitive reserve would be more successful at coping with the same amount of brain damage. Thus, the same amount of brain damage or pathology will have different effects on different people. The concept of cognitive reserve provides a ready explanation for why many studies have demonstrated that higher levels of intelligence and of educational and occupational attainment are good predictors of which individuals can sustain greater brain damage before demonstrating functional deficit. Rather than positing that these individuals’ brains are grossly anatomically different than those with less reserve (e.g. they have more synapses), the cognitive reserve hypothesis posits that they process tasks in a manner that allows them to cope better with the brain damage (Stern, 2006). Since the duration of this pre-symptomatic period reflects the capacity of the reserve, any significant change would clearly be of further interest if any factors (e.g. diet, education, job, etc.) were revealed to be correlated with it.

A high-priority objective of this article is to focus on one of Wilson’s surrogate measures of his cognitive reserve as an attempt to cope with his cognitive dysfunctions: repetitive use of “prefabricated” chunk of words. We are confident that a longitudinal analysis of this substitute measure might shed new light in pre-diagnostic AD detection.

4. Methodology

4.1. Materials

The corpus has been compiled using Harold Wilson’s oral outcomes extracted from the Hansard. The Hansard contains transcripts of all spoken activity in the two Houses of Parliament. Although it cannot and does not always report every single word said by an MP, departures for verbatim are seldom noticeable and may reflect some deletions of repeated words and fillers as well as corrections of departures from grammatical convention (Garrard, 2009: 258).

Ten comparison periods were chosen: 1964-1970 and 1974-1976. More precisely, we randomly extracted 25,000 tokens for each period. Each period-sample begins at a random sentence-boundary in the period-subcorpus and continued up to the first sentence boundary after 25,000 words. Thus, the total corpus for analysis accounts for 250,000 tokens.
To focus our analysis on spontaneous speech, we deleted written questions/answers and statements, as these are normally read from texts that may not even have been written by the speaker. So we limited the data to spontaneous speech, containing verbal exchanges (responses, follow-up questions and subsequent exchanges).

4.2. Method

This research is based on a case study. A case study may show a correlation between two or more variables/factors, and whether or not a causal relationship can also be proven. Case study is an ideal methodology when a holistic, in-depth investigation is needed (Feagin et al., 1991), and it is particularly useful in depicting a holistic portrayal of a person’s behaviour, in our case: the repetitive use of “prefabricated” chunk of words of a single individual at the earliest stages of AD or more precisely at the pre-clinical stages of AD.

Furthermore, our case study method involves an in-depth, longitudinal examination of a single case, contrasting ten periods: 1964-1970 and 1974-1976; where 1964 is Harold Wilson’s first year at office (not affected by AD) and 1976, the year of Harold Wilson’s resignation (potentially affected by AD and showing some signs of MCI).

4.3. Procedure

If Harold Wilson’s resignation was, as hypothesised, influenced by his growing awareness of incipient cognitive decline, then we would expect consistent difference in his output across years. Additionally, Wilson might have started using various surrogate measures of his cognitive reserve as an attempt to cope with his cognitive dysfunctions.

Our analysis focuses on one of the potentially used surrogate measures: repetitive use of “prefabricated” chunk of words. More precisely, we shall concentrate on the commonest sub-sequences of 2 and 3 items across years.

5. Results

In order to analyse the usage of the most common bigrams and trigrams, we implemented the assumptions of Burrows’ Delta (Burrows, 2006), and usages of the commonest n-grams were compared across years. Tab. 1 lists the 30 most frequently used bigrams in the corpus (250,000 tokens), together with the relative frequencies in each of the years. Values are displayed for the entire corpus and for bigrams spoken by Harold Wilson. Comparison of the ten sets (10 years) of values using the Friedman test revealed no difference \( \chi^2(9)=8.829, p=0.453 \).

A second set of n-grams was assembled by selecting the 30 most commonly used trigrams. An equivalent set of values relating to these items is displayed in Tab. 3. Using these data, Friedman’s test was compatible with the ten sets of values originating from different samples \( \chi^2(9)=17.776, p=0.038 \).

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5 Burrows’s Delta is a unitary measure of textual difference. It has been used extensively for automated authorship attribution. We have applied it here, in a simplified version, to detect a single person’s speech dissimilarities across time.
Multiple pairwise comparisons using Wilcoxon’s test revealed statistical differences between 7 of the 45 possible pairings (see Tab. 3).

Although the p-values of Tab. 3 are not conclusive between the trigram usage of Harold Wilson at the year of resignation and the previous years at office, two trends are discernible. The first is that significant contrasts occur within the years 1966 vs. 1968 and 1967 vs. 1968. The second is that three out the seven significant differences (42.86%) are found between 1976 and three other previous years (1966, 1967 and 1970). This might evidence some kind of overuse of trigrams towards the end of Wilson’s stay at office, before resignation. This finding was not apparent in bigram usage (see Tab. 1).
This points that a more consistent analysis on the usage of n-grams with longer sub-sequences (>3 words) might strengthen the inference that Wilson’s started to overuse “prefabricated” chunk as a surrogate measures in order to cope with his cognitive dysfunctions. Tab. 4 shows...
the total amount of $n$-grams used by Harold Wilson (frequency $\geq 3$; $n=2$ to 12). Comparison of the ten 10 years of values using the Friedman test revealed difference $[\chi^2(9)=49.1, p=0.000]$. The comparison of $n$-gram repetition with years is very revealing and statistically significant. Another enlightening finding is the near absence of 9-grams and complete absence 10-grams and bigger $n$-grams between 1964 and 1975, Wilson showed some preference for the use of these long “prefabricated” chunks in 1976. Note also that 7-gram and 8-gram usage between 1964 and 1975 is similar to 11-gram and 12-gram usage in 1976 respectively. Fig. 1 visualises this evidence and it becomes apparent how Wilson’s usage of $n$-grams is seems clearly atypical, with a clear trend of overusing them.

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Table 4: Distributions of $n$-grams (frequency $\geq 3$; $n=2$ to 12) by year

Figure 1: Log-transformed distribution data of $n$-grams (frequency $\geq 3$; $n=2$ to 12) by year
6. Conclusions

In this research, we have assumed, as Garrard (2009), that 1976 is a period prior to Wilson’s symptom onset of AD, during which the gradual accumulation of neurodegenerative pathology is not yet accompanied by any apparent cognitive difficulties. Furthermore, we understand that spontaneous language can reveal early signs of AD: potential pre-clinical markers of cognitive dysfunction.

These two assumptions have led us to analyse the spontaneous speech of former British Prime Minister Harold Wilson, by contrasting his ten years at office: 1964-1970 and 1974-1976, studying the repetitive use of “prefabricated” chunks ($n$-grams), as a possible sign of his incipient AD affection.

The result findings have revealed changes in the usage of a $n$-grams that might point towards the presence of some degenerative neuropathology in Harold Wilson at the time of his resignation:

1. Possible changes in the organization and structuring of the mental lexicon: an overall higher usage rate of fixed structures (2-12 grams).
2. More precisely, bigram usage did not reveal significant difference in usage across year.
3. Trigrams usage output significant differences [$\chi^2(9)=17.776, p=0.038$]. Multiple pairwise comparisons revealed statistical differences between 7 of the 45 possible pairings. Three out the seven significant differences (42.86%) are found between 1976 and three other previous years (1966, 1967 and 1970). This might show some kind of overuse of trigrams just before resignation.
4. A more consistent analysis on the usage of $n$-grams with longer sub-sequences (>3 words; $n=2$ to 12) strengthens the hypothesis that Wilson might have started to overuse “prefabricated” chunk as a surrogate measures in order to cope with his cognitive dysfunctions. The comparison of $n$-gram repetition with years is very revealing and statistically significant [$\chi^2(9)=49.1, p=0.000$].
5. Another important finding is the near absence of 9-grams and complete absence of 10-grams and bigger $n$-grams between 1964 and 1975; Wilson showed some preference for the use of these long “prefabricated” chunks in 1976.
6. 7-gram and 8-gram usage between 1964 and 1975 is similar to 11-gram and 12-gram usage in 1976 respectively.
7. Likely symptoms of less lexical and grammatical complexity/variety due to the higher resort to more common and frequent phrase structures (higher type repetition rate).

These findings also points towards the hypothesis that Harold Wilson might have been aware of his MCI, and that he might have started using various surrogate measures of his cognitive reserve as an attempt to cope with his cognitive dysfunctions. These substitute measures might have included the use of pre-existing cognitive processing approaches or enlist compensatory approaches such as (i) accessing more common and frequent $n$-grams; and (ii) using repetitively “prefabricated” chunk of words.

We are confident that Wilson had more cognitive reserve and was “more successful” than other AD affected people at coping with the same amount of brain damage. This might also explain why his fellow MP’s at the House, press, etc., did not realise anything about his onset of AD.
References


