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Silent pauses in aphasia

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ABSTRACT

Pauses may be studied as an aspect of the temporal organization of speech, as well as an index of internal cognitive processes, such as word access, selection and retrieval, monitoring, articulatory planning, and memory. Several studies have demonstrated specific distributional patterns of pauses in typical speech. However, evidence from patients with language impairment is sparse and restricted to small-scale studies. The aim of the present study is to investigate empty pause distribution and associations between pause variables and linguistic elements in aphasia. Eighteen patients with chronic aphasia following a left hemisphere stroke were recruited. The control group consisted of 19 healthy adults matched for age, gender, and years of formal schooling. Speech samples from both groups were transcribed, and silent pauses were annotated using ELAN. Our results indicate that in both groups, pause duration distribution follows a log-normal bimodal model with significantly different thresholds between the two populations, yet specific enough for each distribution to justify classification into two distinct groups of pauses for each population: short and long. Moreover, we found differences between the patient and control group, prominently with regard to long pause duration and rate. Long pause indices were also associated with fundamental linguistics elements, such as mean length of utterance. Overall, we argue that post-stroke aphasia may induce quantitative but not qualitative alterations of pause patterns during speech, and further suggest that long pauses may serve as an index of internal cognitive processes supporting sentence planning. Our findings are discussed within the context of pause pattern quantification strategies as potential markers of cognitive changes in aphasia, further stressing the importance of such measures as an integral part of language assessment in clinical populations.

1. Introduction

Empty pauses –i.e. empty gaps during speech usually referred to as silent pauses– gradually drew the attention of both linguists and psychologists, as they begun to realize that aspects like frequency and duration may follow specific patterns, assuming that pauses could serve specific purposes during speech (see Rochester, 1973 for a review).

Despite the actual absence of words during pausing, silence is considered to provide important information about a speaker's internal processing. Early studies on speech and reading suggest that pauses tend to appear in various durations, in different types of narration, and seem to follow specific patterns. Among the first scholars who attempted a psycholinguistic explanation, Lounsbury (1954) considered pause types as "*mutually exclusive sorts of events which differed from each other in location, duration and function, independent or possibly complementary*" (as described in Rochester, 1973, p. 54). Thus, pauses of short duration serve breathing and articulation, while longer pauses tend to reflect internal cognitive processes. More specifically, several studies in various types of speech indicated that long pauses may occur before words of low contextual probability (Goldman-Eisler, 1958a, 1958b); therefore they arguably reflect cognitive load on word production at the level of lemma access and selection (Butterworth, 1979). Lemma is considered to be a body of syntactical/morphological information linked to each lexical concept (Levelt, Roelofs and Meyer,

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1999). According to Levelt's model, accurate production of a single word first requires activation of a lexical concept and then the selection of the appropriate lemma. Pauses also tend to be produced prior to less frequent words, thus unveiling possible difficulties in word production at the level of word-form retrieval and encoding (see Beattie and Butterworth, 1979; Griffin and Bock, 1998). Moreover, there is some evidence that hesitations may be also linked to speech organization and syntactic planning (Butterworth, 1976), especially when they appear between utterances, i.e. at the beginning of a sentence (Goldman-Eisler, 1958a, 1958b).

Several studies have demonstrated that pausing may be affected by neurological conditions. Individuals with Parkinson's disease often present with increased silent intervals, especially at the onset of conversational speech production, compared to neurologically intact speakers (Goberman et al., 2005; Goberman and Elmer, 2005). Differentiated pause patterns and higher than normal overall pause rates have also been reported in patients with primary progressive aphasia (Ballard et al., 2014; Mack et al., 2015; Teichmann et al., 2013). Finally, individuals with amyotrophic lateral sclerosis and Friedreich's Ataxia have been shown to produce longer pauses in conversational speech compared to controls (Green et al., 2004; Rosen et al., 2003). These studies clearly show prominent differences in pausing between healthy participants and pathological populations. Interestingly, these studies have also demonstrated how pausing measures may be used as diagnostic markers and/or as markers of cognitive changes over time (see Ballard et al., 2014; Fraser et al., 2014; Mack et al., 2015; Yunusova et al., 2016). Taken together, these findings suggest that pauses could be an integral aspect of cognitive assessment in clinical practice and research.

Few studies have thus far thoroughly investigated pauses in stroke-induced aphasia. Despite the fact that even contemporary aphasia textbooks refer to pauses or hesitations, the focus mostly remains on speech rate as a measure of speech output integrity (e.g., Efthymiopoulou et al., 2017; Fossett et al., 2016). To the best of our knowledge, there are sparse findings on the relationship between pauses and aphasia, mainly derived by small-scale studies, focusing either on pause location in relation to speech elements, or pause frequency. Among the first studies, Butterworth (1979) stressed the importance of pauses in an attempt to interpret impaired word selection. More specifically, he analyzed an interview speech sample of a patient with jargon aphasia, in order to investigate the relationship between pauses and neologisms. Speech analysis revealed that longer pauses often precede neologisms and verbal paraphasias, thus supporting the hypothesis that silent intervals in speech could be considered as an indicator of impaired lexical search, while other language functions, such as syntax, remain relatively intact. Following similar methodology, Panzeri et al. (1990) investigated speech samples of jargon aphasia, and confirmed Butterworth's initial observation (i.e. increased likelihood of occurrence of long pauses before neologisms) for 2 out of the 3 patients who participated in the study, while the third patient demonstrated a different pattern (higher pause frequency before real words). Given the small sample, combined with the lack of a common pause trend across patients with similar aphasic phenotypes, the desired footing for generalizing findings, which could potentially contribute to clarifying pauses' role in aphasic speech, is far from robust. Kirsner et al. (2005b) approached pausing following a different rationale, and compared eight individuals with aphasia (IWA) with thirteen healthy controls with regard to their pause distributions. The authors concluded that most IWA differ from controls in terms of several pause indices (e.g. mean duration and pause rate), and further stress the importance of log-normal distributed data for investigating such phenomena in both intact and impaired speech. Based on this approach, Hird and Kirsner (2010) analyzed speech samples from three IWA with different taxonomic profiles, in comparison to a neurologically intact speaker. They observed that all three patients, similar to the control subject, presented log-normalized bimodal distributions of pauses, including short pauses and long pauses. However, different aphasia diagnostic profiles presented with distinguished pause duration. These findings indicate that pausing distributions could serve as a quantitative index of speech output.

A critical issue of debate in the relevant literature concerns the determination of pause boundaries. Goldman-Eisler (1968) recommended the threshold of 250 ms, as a criterion for a priori distinguishing pauses into two qualitative categories that differ in duration, location and function, and thus may reflect different kinds of cognitive processes. Pauses shorter than 250 ms (i.e., short or articulation pauses) were considered to signify breathing and articulation, and consequently were treated as phenomena beyond psycholinguistic interest, while pauses longer than 250 ms (i.e., long or hesitation pauses) were assumed to reflect higher level cognitive processing such as word seeking and sentence planning. Even though the notion of the aforementioned dichotomy has been widely adopted, most studies implement arbitrary threshold values, thus creating confusion and posing impediments to the integration of published data through meta-analyses, and generally to the interpretation of pauses' meaning. On the other hand, recent studies question the implementation of a fixed threshold as a gold standard. Campione and Veronis (2002) advocated against a predetermined range for defining pauses, although they acknowledged the enormous manual effort required to take pauses shorter than 250 ms into account, and the fact that such pauses are very difficult to discriminate from occlusives. Interestingly, they analyzed their data from connected speech without using predetermined thresholds and successfully showed that disregarding extreme duration values (either very brief or very long) can lead to dismissing important information. For instance, they indicated that percentages of brief pauses (< 200 ms) vary across different languages, while very long pauses (> 2000 ms) appear only in spontaneous speech, but they are absent in reading. One could therefore argue that exclusion of particular pauses on the basis of a predetermined duration criterion constitutes a methodological bias, which could prove to be detrimental to any attempt to objectively describe the temporal components of speech. In line with the argument against selective inclusion of pauses in any kind of analysis, several authors have argued that any threshold differentiating between short and long pauses may be influenced by different variables, such as gender, age, educational level and health status (for recent studies adopting this view see Kirsner et al., 2005a; Rosen et al., 2003, among many others). Two key conclusions can be drawn from the above short literature review. First, all measurable pauses, regardless of duration, should be included in any relevant analysis, in order for a given data set to be complete. Second, predetermined thresholds differentiating types of pauses (in this context, short and long pauses) should be viewed with skepticism.

In sum, although pauses have been considered to be important aspects of language, there is no evidence with regard to their distribution in aphasia. The present study aims to investigate whether patients with aphasia exhibit a different pattern of pause duration distributions, compared to neurologically intact speakers. If the patterns between IWA and healthy individuals differ, then an aphasia producing lesion would be considered to cause a serious breakdown of the temporal organization of speech. In contrast, similar distributions would reflect preserved patterns of pauses, and supposedly temporal speech organization, despite the presence of aphasia. Nevertheless, in the latter case, there could be differences with regard to central tendency indices (e.g. mode, median, or mean) between the two groups (IWA and controls). On the basis of the main notion discussed in the Introduction, differences found in shorter pauses would relate to articulation, while differences in longer pauses could be attributed to impaired cognitive processes essential for word retrieval and sentence planning. Despite the indications that pauses may provide elucidating information concerning brain function during speech, there is only a small number studies focusing on pause location, while even fewer studies meticulously investigate pause duration distributions in normal and

pathological speech. However, no study has so far attempted to incorporate both approaches. As Kirsner et al. (2002) fairly claim, there is an obvious gap with regard to pausing studies. This may due to practical and methodological issues, such as time consuming process of manual pause annotation. A secondary aim of this study is to assess relationships between two pause modalities (short and long) and linguistic elements, such as utterances and specific word types, in an attempt to integrate previously implemented methodologies, in aphasic and typical speech.

2. Methodology

2.1. Participants

Eighteen patients with chronic aphasia following a left hemisphere stroke, 40–74 years old were recruited. Aphasia was assessed with the short form of the Boston Diagnostic Aphasia Examination (BDAE-SF; Goodglass and Kaplan, 1972), adapted in Greek (Tsapkini et al., 2009). Further neuropsychological testing included the Boston Naming Test (BNT; Kaplan et al., 1983), standardized in Greek (Simos et al., 2011), and the Controlled Oral Word Fluency (COWF; Kosmidis et al., 2004). Patients with speech rate (words/minute) lower that 40 were excluded: a pilot preliminary study including only severely non-fluent individuals showed that their speech output pattern poses great limitations to subsequent analysis, due to extremely long intervals of silence between sparse, and rather short utterances, often corresponding to single words. Structural imaging data (CT or MRI) were obtained for each patient, and lesion sites were identified and coded for 16 predetermined left hemisphere areas: the inferior and middle frontal gyri, the precentral gyrus, the inferior, middle and superior temporal gyri, the inferior parietal lobule, including the angular and supramarginal gyri, the thalamus, the insula, the supplementary motor area, the internal and external/extreme capsule fasciculi, the head and tail of the caudate nucleus, the putamen, and the globus pallidus. Selection and coding of the lesion areas was based on previously reported methodology (Kasselimis et al., 2017). The total number of affected cortical and subcortical areas served as an index of lesion extent (lesion score) (as described in Efthymiopoulou et al., 2017 and Kasselimis et al., 2013). The control group consisted of the patients' caregivers: 19 healthy adults, 45–86 years old, with no neurological or psychiatric history. The two groups were matched for age, gender, and years of formal schooling. All participants were right-handed and native speakers of Greek. Informed consent was obtained from all participants prior to participation (for detailed demographics for the two groups, see Table 1; for individual lesion data, see Table 2).

Table 1
Demographic characteristics of the two groups.

	Aphasic patients (n = 18)		Controls (n = 19)		p
	Range	Mean (SD)	Range	Mean (SD)	
Age (years)	40–74	57.94 (9.14)	45–86	63.95 (10.60)	.074[a]
Education (years)	6–20	12.28 (3.75)	6–16	10.95 (4.05)	.31[a]
Gender (n)					
Men	12		10		.51[b]
Women	7		9		

[a] Independent samples *t*-test.
[b] Chi-square.

2.2. Data analysis

Aphasic speech samples were derived from recordings of the stroke story interview during standard BDAE-SF assessment. Healthy participants were asked to provide a brief narration regarding the patient's history (i.e. the stroke incident and how it affected their lives); that was the speech sample equivalent to the stroke story. Speech samples were then transcribed, and silent pauses were annotated using ELAN (Wittenburg et al., 2006; Brugman and Russell, 2004) by two independent raters. ELAN is a professional annotation tool, specifically designed for language analysis, which allows the user to create and edit annotations in audio and video data. Its main advantage, compared to other annotation tools, is that annotations may occur in multiple levels (tiers). In our study, we created tiers to separately annotate empty pauses, utterances, verbs, nouns and paraphasias (See Fig. 1, for a multi-tier annotation sample).

Subsequently, speech samples were segmented in utterances, using primarily semantic, syntactic and intonational criteria, in accordance with speech annotation methodology proposed by Saffran et al. (1989), adapted in Greek language by Varkanitsa (2012). In line with the guidelines established by Saffran et al. (1989), pauses were not interpreted as utterance boundaries. Then, each pause, regardless of duration, was examined separately, in order to detect its location in relation to utterance (i.e. whether it appears between two utterances or within an utterance) and then to annotate what follows the pause. Following previous studies (Arévalo et al., 2007; Mack et al., 2015), we focused on major lexical categories, namely nouns and verbs. Mean Length of Utterance (MLU), number of utterances per hundred words, number of utterances containing long pauses, number of utterances containing short pauses, and number of utterances containing no pauses, were also calculated. In order to overcome the problem of pause boundaries discussed in the Introduction, no a priori boundary thresholds were used for the detection of lowest and highest admissible pause durations. On the basis of the methodology rationale put forward by Campione and Veronis (2002) and Hird and Kirsner (2010), all pause samples were log-transformed (using the natural logarithm of each pause), with a log-normal bimodal distribution pattern emerging from the histogram (bin size = .1) and further curve fitting, where the two modes may be assumed to represent short and long pause duration maxima. In order to estimate the boundary threshold value directly from our data, we applied finite-mixture modeling to distinguish between the two emerging types of pauses (95% confidence interval), as originally described in Trang et al. (2015): the two types of pauses were modeled after their sample mean and standard deviation, and subsequently mixed using a weighting parameter λ (Oakes, 1999; Schlattmann, 2009). The λ parameter and threshold were estimated through maximum likelihood via the expectation-maximization algorithm as described in Do and Batzoglou (2008), while Monte Carlo simulations were implemented to estimate confidence intervals. Statistical analysis was carried out using the R 3.3.0 software environment (R Core Team, 2013).

In order to investigate possible patterns with regard to relationships between lesion and pause variables, we conducted the following analyses: First, correlations between lesion score and pause rate (number of pauses per hundred words[1]) separately for long and short pauses. Assumption of normality and absence of non-linear relationships were first assessed by implementing Shapiro-Wilk and creating scatter graphs, respectively. Due to small sample size, and assumption violations, Spearman rho was used instead of Pearson r. Second, we

[1] Pearson correlation analyses between long pauses' frequency, short pauses' frequency (number of long pauses per minute and number of short pauses per minute, respectively), and speech rate were conducted at first. Results showed a significant negative correlation between speech rate (words per minute) z-scores and frequency of long pauses (number of pauses per minute) ($r = -.765$, $p < .05$), while no significant correlation appeared for short pauses frequency ($r = .448$, $p = .07$), for the IWA group. Even though the latter correlation failed to reach significance, we observed a correlation trend: Long pause frequency presented an inverse association with speech rate, while the opposite pattern is shown for short pauses (i.e. negative and positive correlation coefficients for long and short pauses respectively). We argue that the analogy between speech rate and short pause frequency may simply indicate that increased verbal output will inevitably be accompanied by a shift in short silent intervals between utterances. Therefore, in order to control for the speech rate variable, we used an alternative rate index, i.e. pause per hundred words, instead of pauses per minute. MLUs, and all pause-related indices were also calculated for the first 100 words, in order to ensure consistency.

Table 2
Individual lesion data for the aphasic group.

ID	Gender	IC	EC	GP	Putamen	CNh	CNt	Thalamus	SMA	PrG	Insula	IFG	MFG	IPL	STG	MTG	ITG	LS
1	F	intact	lesion	intact	intact	intact	intact	intact	intact	intact	lesion	intact	intact	lesion	lesion	lesion	lesion	5
2	F							Missing										–
3	M	intact	intact	intact	intact	intact	intact	intact	intact	intact	intact	intact	intact	lesion	lesion	lesion	lesion	4
4	M	intact	intact	intact	intact	intact	intact	intact	intact	intact	lesion	intact	intact	lesion	intact	intact	lesion	3
5	M	intact	intact	intact	lesion	intact	intact	intact	intact	lesion	lesion	lesion	lesion	intact	lesion	intact	intact	6
6	F	lesion	lesion	lesion	lesion	lesion	lesion	lesion	intact	lesion	lesion	lesion	intact	lesion	lesion	lesion	lesion	14
7	M	lesion	lesion	lesion	lesion	intact	intact	intact	intact	intact	lesion	lesion	intact	intact	lesion	lesion	intact	8
8	M	intact	lesion	intact	lesion	intact	intact	intact	intact	intact	lesion	lesion	intact	lesion	lesion	intact	intact	6
9	F	intact	intact	intact	lesion	lesion	lesion	intact	intact	lesion	lesion	lesion	intact	lesion	lesion	intact	intact	8
10	F	intact	intact	intact	lesion	lesion	lesion	intact	intact	lesion	lesion	lesion	intact	lesion	lesion	intact	intact	8
11	M	lesion	intact	intact	intact	intact	lesion	intact	intact	intact	intact	intact	intact	intact	intact	intact	intact	2
12	M							Missing										–
13	M	intact	intact	intact	intact	intact	intact	intact	intact	intact	lesion	intact	intact	lesion	intact	intact	intact	2
14	F	lesion	lesion	lesion	lesion	lesion	lesion	intact	intact	intact	lesion	intact	intact	lesion	lesion	intact	intact	9
15	M	intact	intact	intact	intact	intact	intact	intact	lesion	lesion	lesion	lesion	lesion	intact	intact	intact	intact	5
16	M	lesion	lesion	lesion	lesion	intact	intact	intact	intact	intact	intact	intact	intact	lesion	intact	intact	intact	5
17	M	intact	intact	intact	intact	intact	intact	intact	intact	intact	intact	intact	intact	lesion	lesion	lesion	lesion	4
18	M	intact	lesion	intact	intact	intact	intact	intact	intact	intact	lesion	intact	intact	lesion	lesion	lesion	intact	5

IC: internal capsule; EC: external capsule; GP: globus pallidus; CNh: caudate nucleus head; CNt: caudate nucleus tail; SMA: supplementary motor area; PrG: precentral gyrus; IFG: inferior frontal gyrus; MFG: middle frontal gyrus; IPL: inferior parietal lobule; STG: superior temporal gyrus; MTG: middle temporal gyrus; ITG: inferior temporal gyrus; LS: lesion score.

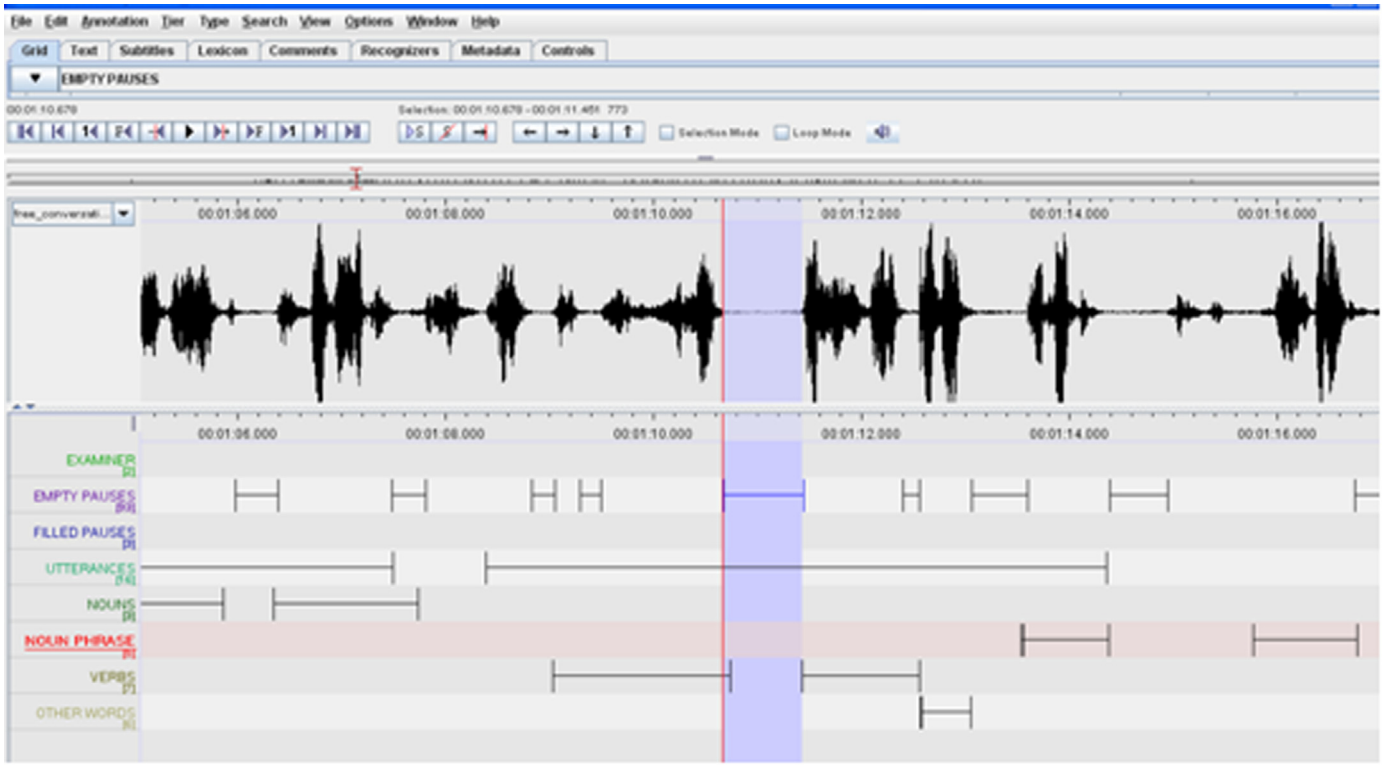

Fig. 1. Sample of multi-tier speech annotation made in ELAN. Tiers included: **Examiner** (examiner intervals that were annotated and excluded from speech analysis), **Empty Pauses** (silent pauses), **Filled Pauses** (vocalized pauses such as "um", "uh", "hmm"), **Utterances** (units of speech that are delimited by semantic, syntactic and intonational indicators), **Nouns**, **Noun Phrases (**determiner-noun, determiner-adjective-noun, adjective-noun), **Verbs** and **Paraphasias**.

compared mean pause rates (separately for short and long pauses) between IWA subgroups defined by the existence of lesion in specific regions. In order to reduce the number of comparisons and consequently ensure a degree of statistical robustness, we restricted to particular lesion loci, on the basis of two criteria: strong association with language according to contemporary literature (Petrides, 2014; Petridesand Pandya, 2008), and a reasonable numbers of participants (at least 5) in each subgroup defined by the particular locus. Candidate regions for these analyses were the inferior frontal, the superior and middle temporal gyri, the inferior parietal lobule, and the extreme/external capsule fasciculus. These regions broadly correspond to the perisylvian language network, in accordance with general consensus (Petrides, 2014; Price, 2012). It should be however noted that information about essential white matter tracts, i.e. the arcuate and superior longitudinal fasciculi, were not available, and given that these structures have been shown to be crucially involved in language (Saur et al., 2008), lack of such data poses a serious limitation with regard to our lesion analysis. Eventually, the inferior parietal lobule was not used as an independent variable due to limited number of participants in the non-lesion subgroup (n = 4). Analyses were conducted with Mann-Whitney *U* test and

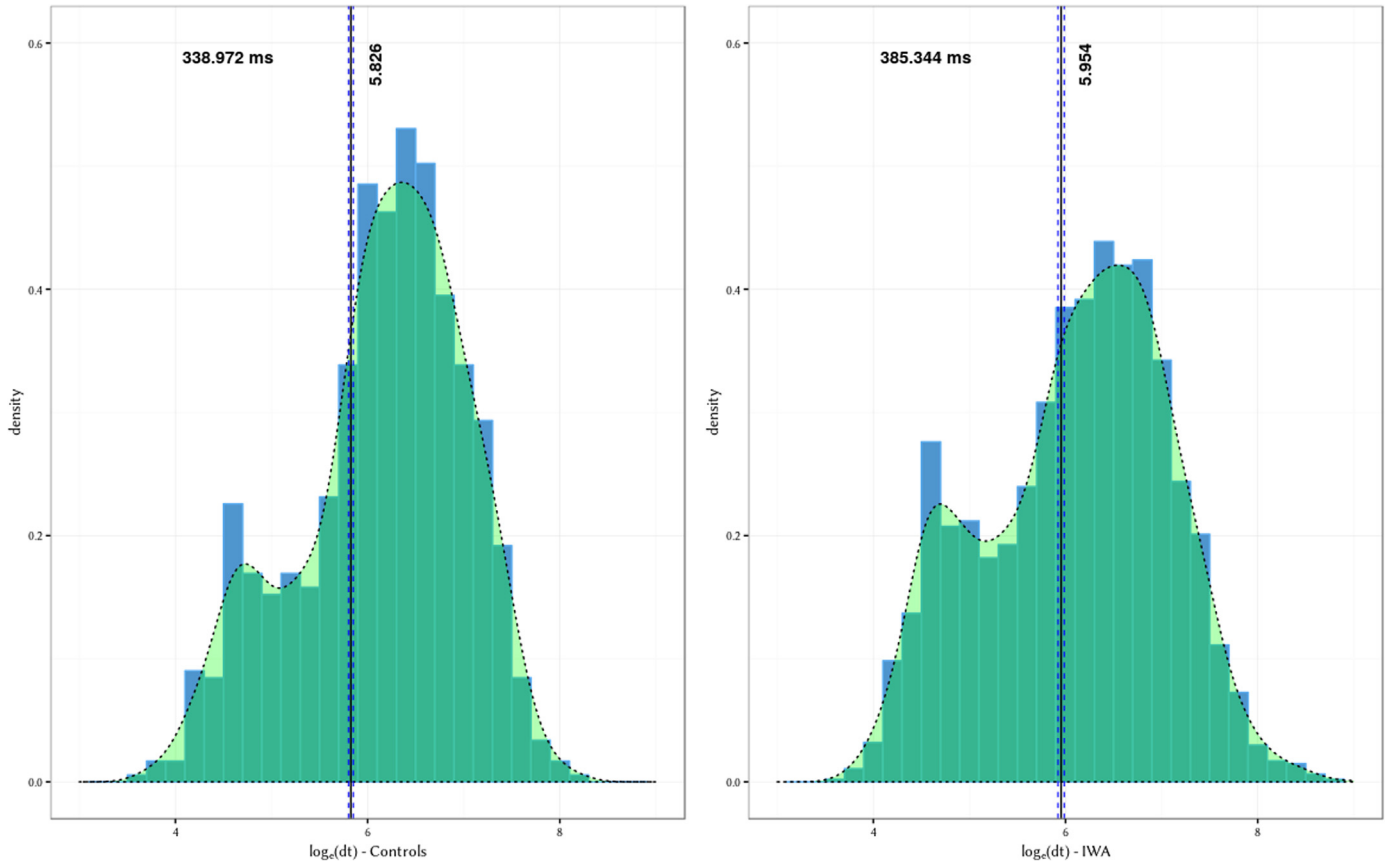

Fig. 2. Pause duration distribution in loge(dt) for the control and the IWA group. At 95% confidence, controls and IWA presented non-overlapping confidence intervals (CI) for their thresholds between short and long pauses at 5.826 (~338.972 ms, CI: 5.812–5.840) and 5.954 (~385.344 ms, CI: 5.932–5.976) respectively.

the Bonferroni correction was applied in order to avoid error type I due to multiple comparisons. The adjusted significance level used was $\alpha = .008$. In order to assess relationships between the two pause modalities (short and long) and linguistic elements, Spearman correlation analyses between long and short pause rates, speech rate (words per minute), MLUs, spontaneous naming (BNT scores), and verbal fluency (COWF scores) were conducted. Finally, we compared IWA and controls, with regard to short and long pause rate, number of short and long pauses between and within utterances, MLUs, and number of utterances containing long, short, and no pauses.

3. Results

Our results clearly show that both IWA and healthy participants fit well into a log-normal bimodal distribution model (see Fig. 2). That is, the observed distributions are the result of the combination of two types of pauses, namely short and long pauses, with medians of 4.85 (128 ms) and 6.51 (670 ms) in the log domain and 4.88 (132 ms) and 6.65 (785 ms) for the non-brain damaged participants and for IWA respectively. Log-transformed silent pauses ranged from 3.87 to 8.87 (48–6000 ms) for the IWA group and from 3.69 to 8.18 (40–3550 ms) for the control group. A further investigation of distances between indices of central tendency for the two groups revealed that the median of long pauses was significantly higher ($\mu_{2a} = 6.650$, CI95%: 6.609–6.692) for the IWA group compared to the corresponding median of the control group ($\mu_2c = 6.514$, CI95%: 6.472–6.556). The difference between the medians for short pauses did not reach statistical significance ($\mu_1a = 4.884$, CI95%:4.841–4.928; $\mu1c = 4.854$, CI95%:4.787–4.922).

The estimated threshold value between the two pause types was significantly higher for the IWA group (CI95%: 5.932–5.976) compared to controls (CI95%: 5.812–5.840). As illustrated in Fig. 2, despite both distributions being bimodal (with a clear preference towards long pauses), IWA demonstrated a higher frequency for short pauses compared to controls, while the opposite trend is evident for long pauses. However these differences did not reach statistical significance.

Spearman correlation analysis revealed a moderate positive association between lesion score and long pause rate; rho = .594, p = .015. The corresponding correlation coefficient for short pause rate failed to reach significance (rho = −.019, p = .943). M-U tests investigating possible relationships between pause rates and lesion locus yielded significant results only for the inferior frontal gyrus lesion locus. In particular, patients whose lesion included the inferior frontal gyrus were shown to demonstrate increased long pause rate (U = 4.0, p = .004), made more long pauses within utterances (U = 2.0, p = .002), and, consequently, produced more utterances containing long pauses (U = 4.5, p = .004).

For the IWA group, correlation analyses showed that long pause rate was associated with speech rate (rho = −.779, p = .0002) and MLU (rho = −.889, p = .0000008), but not with COWF or BNT scores. Correlations between the aforementioned measures and short pause rate were non-significant. For the control group, long pause rate was also associated with speech rate (rho = −.770, p = .0004), but not with MLU.

With regards to the distributional characteristics of pauses (see Table 3 for descriptive statistics), IWA exhibited a pattern similar to that of controls. Specifically, both groups produced more long pauses (66.76% for controls; 56.26% for IWA) than short pauses (33.24% for controls; 43.74% for IWA). In addition, in both groups the majority of short pauses were found before nouns and noun phrases (NPs) (26.38% for controls; 26.90% for IWA) than verbs (25.95% for controls; 20% for IWA), whereas the majority of long pauses were found before verbs

Table 3
Descriptive statistics for pause variables and linguistic elementsfor the control and the IWA group.

	Healthy individuals			Individuals with aphasia		
	Mean (SD)	**Median**	**Range (min-max)**	**Mean (SD)**	**Median**	**Range (min-max)**
Number of pauses	19.75 (8.1)	17.50	27 (11–38)	54.06 (28.5)	48.50	112 (18–130)
Number of short pauses	5.81 (6.5)	4.00	27 (0–27)	20.28 (11.7)	16.50	40 (6–46)
Number of short pauses between utterances	.50 (1.1)	.00	4 (0–4)	.39 (.8)	.00	2 (0–2)
Number of short pauses within utterances	5.44 (6.1)	4.00	25 (0–25)	18.89 (10.8)	16.50	38 (6–44)
Number of long pauses	13.94 (4.9)	12.50	20 (6–26)	33.78 (24.7)	29	101 (8–109)
Number of long pauses between utterances	6.75 (2.1)	7.00	7 (3–10)	12.67 (5.8)	12.00	19 (3–22)
Number of long pauses within utterances	6.94 (4.5)	6.50	16 (2–18)	21.11 (21.3)	16.00	91 (2–93)
Number of utterances	7.00 (2.6)	6.50	10 (4–14)	13.22 (5.4)	12.00	16 (6–22)
Mean Length of Utterance	15.92 (5.07)	15.43	18.86 (7.14–26)	8.94 (3.8)	8.44	12.61 (4.55–17.16)
Number of utterances with no pauses	2.13 (2.3)	2.00	9 (0–9)	2.39 (2.8)	1.50	11 (0–11)
Number of utterances with short pauses	3. 00 (2.3)	2.50	9 (0–9)	7.44 (3.1)	7.00	11 (3–14)
Number of utterances with long pauses	3.88 (1.4)	4.00	5 (2–7)	8.89 (4.9)	8.00	16 (2–18)
Number of nouns	18.19 (4.9)	17.00	19 (12–31)	17.44 (8.3)	18.00	31(1–32)
Short pauses before nouns	1.38 (1.75)	1.00	6 (0–6)	5.50 (3.7)	5.00	12 (0–12)
Long pauses before nouns	2.69 (1.9)	2.50	6 (0–6)	6.39 (5.4)	6.00	16 (0–16)
Number of verbs	20.50 (4.9)	21.50	19 (9–28)	19.61 (4.9)	20.00	18 (8–26)
Short pauses before verbs	2.25 (2.8)	1.00	9 (0–9)	5.06 (5.2)	4.00	20 (0–20)
Long pauses before verbs	5.44 |(2.3)	5.50	9 (1–10)	9.00 (5.9)	9.00	20.00 (0–20)
Number of paraphasias	_	_	_	6.33 (5.4)	5.00	16 (0–16)
Short pauses before paraphasias	_	_	_	1.90 (1.9)	2.00	6 (0–6)
Long pauses before paraphasias	_	_	_	2.44 (3.3)	1.00	10 (0–10)

(24.36% for controls; 18.55% for IWA) than nouns and NPs (22.46% for controls; 18.55% for IWA). However, the two groups differed in respect to pauses' location in relation to utterances. In the control group the vast majority of short pauses was found within utterances, whereas the majority of long pauses was found between utterances. The IWA exhibited the same pattern for short pauses but a reverse pattern for long pauses; in both cases, the IWA produced more pauses within utterances. Comparisons between IWA and controls using M-*U* tests, revealed significant differences with regard to total pause rate ($U = 17$, $p = .00001$), short pause rate ($U = 22$, $p = .00003$), and long pause rate ($U = 56.5$, $p = .003$), with the IWA group producing more pauses in both modalities. Regarding pauses' position, the two groups did not differ regarding the number of short pauses between utterances, while a significant difference emerged with regard to the occurrence of short pauses within utterances ($U = 24.5$, $p = .00004$), as IWA produced more short pauses than controls. Additionally, IWA produced significantly more long pauses between and within utterances ($U = 48.5$, $p = .001$; $U = 64$, $p = .006$, respectively), compared to healthy individuals. Finally, the two groups differed significantly regarding the occurrence of short pauses before nouns, with the IWA group producing significantly more pauses than controls ($U = 36$, $p = .0002$). Comparisons for short pauses before verbs and for long pauses before nouns and verbs failed to reach significance.

4. Discussion

To the best of our knowledge, this is the first study investigating pausing duration distributions in Greek aphasic connected speech. Our results show that pause distributions of both populations follow similar log-normal bimodal patterns, allowing classification of pauses into two types in both aphasic and normal speech groups: short and long pauses. This is in accordance with previous studies exploring pausing in IWA (Kirsner et al., 2005b) and neurologically intact speakers (Campione and Veronis, 2005). We argue that this finding suggests that the general pattern governing specific temporal components of connected speech, namely the occurrence of silent gaps between spoken words, may be resistant to brain damage, thus remaining unchanged after a left-lateralized stroke which otherwise severely affects language functions.

However, besides the above described similar distribution patterns, the two groups demonstrated significant differences with regard to particular pause-derived indices, namely the median for the long pauses peak and the threshold distinguishing the two types of pauses. Specifically, IWA exhibited a higher median for long pauses compared to controls. In addition, the threshold distinguishing the two types of pauses was found to be significantly higher in IWA than controls. We believe that these two findings are interrelated and reflect general deficits in spontaneous speech for IWA. Following previous literature, we argue that the threshold distinguishing the two types of pauses is dynamic and affected by several demographic and/ or pathological factors (see for example Kirsner et al., 2005a). We therefore suggest that the difference observed in the boundary thresholds may be dependent solely on central tendency indices related to long pauses, while short pauses appear to be irrelevant. We further suggest that IWA's greater median for long pauses may be interpreted by the fact that patients with aphasia need more time either for sentence planning or word finding. To support this hypothesis, we performed correlation analyses between long and short pause rates, speech rate, MLU, spontaneous naming (BNT scores), and verbal fluency (COWF scores). Long pause rate was found to be negatively correlated with both MLU and speech rate, while no significant correlations appeared for short pause rate, for the IWA group. This finding suggests that the higher median for long pauses and the interrelated higher threshold in IWA compared to controls may reflect impaired access to lexical/semantic representations and/or deficits in sentence planning. Notably, the corresponding correlation coefficients between performance on lexical access measures (i.e. BNT and COWF) and long pause rate are rather weak. Therefore, we argue that the frequency of long pauses may serve as an index of cognitive processes, not only associated to lexical access, but also heavily relying on sentence planning (see also Goldman-Eisler, 1958a, 1958b). The fact that the correlation coefficients between long pause rate and speech rate are of similar magnitude among the two groups, may serve as an indication that the association pattern between the occurrence of silent gaps and the production of spoken words is preserved. This finding, combined with the analogy of the two pause distributions (see Fig. 2), allows us to assume that IWA and neurotypical speakers do not pose qualitative differences with regard to underlining mechanisms of temporal organization of speech. On the other hand, we argue that pause patterns differ in a quantitative manner between the two groups. In general, IWA tend to produce more pauses. Moreover, the mean duration of long pauses is significantly increased in aphasia. Additionally, longer pauses tend to occur more often between but also within utterances in IWA. Taken together, these findings

suggest that IWA may follow a pause pattern which may be similar to that of healthy individuals, although impaired, as indicated by increased frequency and duration of long pauses. The fact that long pause rate is strongly and inversely associated with MLU only in IWA, further enhances the hypothesis that quantitative alterations related to long pauses may reflect an impairment in sentence planning.

Mean Length of Utterance, as the mean number of narrative words contained in one utterance (oral sentence), has been widely used in studies of pathological speech and especially in speech samples produced by patients with aphasia (Gleason et al., 1980; Wilson et al., 2010). More specifically, the length of utterances has been implemented as an important criterion to distinguish patients with aphasia in fluent and non-fluent and measure aphasia severity (Goodglass et al., 1964). Furthermore, Wagenaar et al. (1975) suggested that MLU and number of words produced are variables strong enough to support the distinction between fluency and non-fluency. Nevertheless, MLU is argued to reflect qualitative linguistic aspects, and therefore cannot be labeled solely as a fluency index. Several studies have shown that low MLUs may indicate decreased sentence complexity in IWA, regardless of whether aphasia is fluent or non-fluent (Edwards, 1995; Gordon, 2006; Rochon et al., 2000; Varkanitsa, 2012). In sum, we argue in favor of an association between elevated frequency of long pauses and impaired sentence planning, however we also acknowledge that MLU remains a multifaceted index and may reflect processes extending beyond sentence complexity.

Overall, our data support the notion that long pauses may reflect higher cognitive functions, such as temporal organization of speech, whereas short pauses may be involved in phonetic and articulatory aspects. We argue that the higher medians for long pauses (resulting in higher threshold distinguishing two types of pauses) exhibited by IWA may be attributed to limited language-related processing resources (see also Avrutin, 2006; Kolk, 1995). This hypothesis is further supported by the fact that IWA –as opposed to healthy participants- demonstrate a distinctive trend for long pause occurrence; namely the higher frequency of long pauses within utterances, which probably reflects a breakdown of language processing while the patient makes an effort to form a meaningful string of interconnected words.

Considering the neural correlates of the differences observed in silent pauses in IWA, M-U results were significant for the inferior frontal gyrus, thus indicating that this particular lesion locus may have an effect on long pause rate. In particular, our findings indicate that a lesion including the inferior frontal gyrus significantly increases long pause rate. Since we lack detailed digital neuroimaging data, no strong conclusions can be drawn. We can however speculate about this finding and attribute the observed association between the specific lesion site and long pause rate to two possible factors. First, reduced speech output, and consequently more silent gaps, could be considered an expected outcome after damage to the inferior frontal gyrus, given that the latter includes Brodmann areas (BA) 44 and 45, which are traditionally associated with speech fluency since the time of Broca (1861). Although the causal relationship between the specific lesion site and the resulting aphasia phenotype has been questioned (Kasselimis et al., 2015; Mohr et al., 1978), it is the common consensus that stroke-induced brain damage affecting, among other regions, the foot of the third frontal convolution, is strongly related to non-fluent aphasia (Henseler et al., 2014; Kreisler et al., 2000). In this sense, higher pause rates could be explained as a result of reduced speech output. However, this explanation is somewhat generic. A complementary interpretation of this finding could be related to the role of the ventrolateral prefrontal cortex (which includes BA 45) in selective retrieval (Chapados and Petrides, 2015; Kostopoulos and Petrides, 2016). In this context, impaired selective retrieval of semantic and phonological aspects of verbal information due to the destruction of the pars triangularis, could result in anomic phenomena, and therefore more long pauses, which in turn could lead to low speech rate and poor sentence planning, as shown by the inverse association of long pause rate with words per minute and MLU. Since we lack digital MRI data, these hypotheses remain to be investigated in future studies. It should be also noted that our results are not directly comparable to published data derived from studies focusing on pauses, because –to the best of our knowledge- no study has thus far investigated the relationship between specific lesion loci and pause variables in post-stroke aphasia. Studies with patients with neurodegenerative diseases have demonstrated a relationship between pauses and integrity (or lack thereof) of frontal cortical and subcortical regions (Ash et al., 2012; Mack et al., 2015; Pistono et al., 2016). However these studies utilized different methodology for quantifying pauses, and, most importantly, did not include stroke patients.

With regard to correlations between lesion extent and pause frequency, long pause rate was positively correlated with lesion score, while short pause rate was not. This finding indicates that the strength of the relationship between lesion extent and pause rate, may be dependent on pause type. A large lesion affecting cortical and subcortical regions is therefore expected to result in increased number of long pauses within a given range of produced words. Given that the extent of left-lateralized lesions is commonly related to severity of aphasia (and consequently speech output integrity), this finding is in accordance with the results reported above, and further supports the notion that an extensive lesion would most probably result in reduced speech rate and a shift in long pause rate. In any case, acknowledging the vast restrictions of the present study with regard to lesion analysis potential (i.e. non-digital data and small sample size, to name the most important ones), we reserve from further discussing lesion correlates of pause variables.

Overall, our main findings suggest that pause patterns in IWA are qualitatively similar to-, but quantitatively different from- those of neurologically intact controls. We overall argue that the present results are preliminary, do not reveal any clear-cut associations, and should be interpreted with caution. Future studies involving digital MRI data and larger samples could further elucidate possible relationships between lesion loci/extent and pause characteristics.

Similar to other researchers, we acknowledge the fact that a major constraint in the study of pausing refers to the cost of time (Kirsner et al., 2002). This is a serious drawback that prevents researchers from analyzing larger amounts of data and consequently examining the possible influence of more variables. On the other hand, we strongly believe that computational linguistics and other relevant fields may be a good asset to overcome these difficulties and that studies like the one presented in this paper clearly point towards multidisciplinary collaborations. A second issue concerns not only pauses but also speech research in general. There are numerous factors influencing speech, which more than often are not easily controlled or even measurable. However, the major difficulty may be due to lack of a consensus for a standard methodology and, therefore a comparison between studies is practically unfeasible. In this framework, and in order to overcome such impediments, we suggest that all pauses, regardless of duration, should be included in any relevant analysis. We further argue that an ante hoc threshold for differentiating between pause types based on their duration constitutes a major drawback, due to two main reasons. First, the variety of such thresholds in the literature poses great difficulty in conducting any meta-analysis, which would significantly aid in clarifying the role of silent gaps in inner language processes. Second, any predetermined threshold is arbitrary, and does not emerge from analyzing speech-derived data, but rather reflects a consensus or an individual researcher's intuition.

5. Conclusion

Although pause-derived variables could be used as valuable complementary measures to quantify speech output, aphasia literature is rather scanty in this regard. The relevant studies are sparse and mostly restrict pause analysis to frequency and location, without taking duration into consideration. A systematic measurement and characterization

of pauses in connected speech may hold rich potential for the assessment of aphasia mainly due to its high ecological validity; unlike standardized tests involving confrontational naming, repetition, sentence completion etc., measures of pausing are an aspect of a natural behavior and reflect communication efficiency. The present study shows that pause distributions of healthy and pathological speech may share common ground in terms of bimodality, thus indicating that short and long pauses are fundamental components of speech, independently of pathology, but they pose differences with regard to specific aspects such as boundary thresholds distinguishing pause types on the basis of duration, as well as central tendency indices related to long pauses. The latter may reflect deficits associated to sentence planning or access to lexical/semantic representations. Overall, our findings suggest that post-stroke aphasia does not affect the general pattern of pauses during connected speech. Nevertheless, IWA do demonstrate quantitative differences compared to neurotypical speakers, with regard to pause rate and duration. These differences are more salient for long pauses, which can be considered to be indices of internal cognitive processes supporting sentence planning. To further elucidate the role of silence in speech temporal organization, a larger sample size of spontaneous, typical and impaired, speech is desirable. The next step could be to further scrutinize the relationship between pauses and errors produced during different genres of narratives. For future studies, we suggest the adoption of two key methodological guidelines in order to further pursue such goals in pause research: unbiased, duration-independent inclusion of pauses, and no implementation of a predetermined threshold to distinguish between short and long pauses.

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