

## PALILALIA AND EXECUTIVE DYSFUNCTION IN SCHIZOPHRENIA: A THREE-CASE STUDY<sup>1</sup>

**Rodophe Prosper MAAH**  
University of Douala, Cameroun  
[marop86@yahoo.com](mailto:marop86@yahoo.com)

**Abstract:** Palilalia is a repetitive speech disorder that has considerably been studied. However, the causes of this disorder are still unclear. This paper discusses the cognitive hypothesis that could shed light on the explanation of palilalia in schizophrenia. Three patients with palilalia deliberately participated to the study; they were all diagnosed with schizophrenia at the Psychiatric Center of the Yaoundé Jamot Hospital (PCYJH). The methods used are essentially empirical: no experimental method (MRI or Scan) was used to investigate the brain of participants. A Dictaphone was used to collect spontaneous linguistic data. The corpus presented revealed two types of palilalia (spasmodic and atonic). Both types of palilalia described were slightly different from the original description by Sterling (1924). As far as the explanation of the disorder is concerned, it is conjectured that palilalia can be related to a disorder in the activation process which is one of the executive functions involved in language production mechanism.

**Key words:** Palilalia, executive function, activation processes, inhibitory processes, schizophrenia.

**Résumé :** La palilalia qui est un trouble de répétition de la parole a fait l'objet de plusieurs études. Cependant, ses causes restent encore inconnues. Dans cet article, nous présentons une hypothèse cognitive qui pourrait apporter plus de lumière sur l'explication des causes de ce trouble en schizophrénie. Pour ce faire, trois patients diagnostiqués de schizophrénie par les médecins du Centre Psychiatrique de l'Hôpital Jamot de Yaoundé (CPHY) ont volontairement participé à cette étude. Les méthodes utilisées dans le cadre de cette étude sont essentiellement empiriques (aucune méthode expérimentale comme l'IRM ou scanner n'a été utilisée). Le discours des patients était enregistré à l'aide d'un Dictaphone, puis transcrit phonétiquement. Les données obtenues de nos patients indiquent deux types de palilalia (spasmodique et atonique). Ces derniers types de palilalia sont légèrement différents de ceux antérieurement décrits par Sterling (1924). Quant aux causes de la palilalia, nous avons postulé que la palilalia peut être due à un dysfonctionnement dans le processus d'activation qui constitue l'une des fonctions exécutives impliquées dans les mécanismes de production du langage.

**Mots clés :** Palilalie, fonction exécutive, processus d'activation, processus of inhibition, schizophrénie.

---

<sup>1</sup> **Acknowledgements:** I am particularly grateful to Dr Jean Pierre Kamga Olen, the Head of the Psychiatric Center of the Jamot Hospital Yaounde and all the staff who approved this initiative. I am also grateful to all the patients who deliberately accepted to participate to this experience.

## Introduction

This paper investigates the cause of palilalia in three schizophrenic patients, using a cognitive approach. Reports on the origin of palilalia rather seem diverging than converging to the same explanation, though bringing together elements that significantly contribute in getting closer to the truth. While it is widely attested that palilalia could be associated with neurological damage like basal ganglia calcification and a motor speech disorder, etc. (Lapointe & Horner, 1981; Benke & Butterworth, 2001), studies on other repetitive disorders (Kipps & Hodges, 2013; Bossikass et al., 2014) associate executive dysfunctions with perseveration and echolalia, and suggest that palilalia should be, analogically to perseveration and echolalia, considered as resulting from deficits of inhibitory processes. Since brain investigations of patients with schizophrenia do not show any neuroanatomical abnormality as it is the case in aphasia, one is tempted to argue that palilalia is associated with executive dysfunctions. In addition to the fact that the relation between palilalia and executive functions has received less attention, we want to investigate the relationship between activation process and palilalia.

### ▪ Background of study

Palilalia is a rare speech disorder characterized by a compulsive and abnormal repetition of utterances. Dubois (2001, p.341) defines it as a language disorder characterized by a spontaneous repetition of the same utterance over and over again. Palilalia is also defined in terms of its acoustic features as a disorder of speech characterized by the compulsive repetition of a sentence, phrase, word, or syllable with an increasing rapidity and a decreasing voice volume (Fergusson & Boller, 1977; Lapointe & Horner, 1981; Ikeda & Tanabe, 1992; Benke & Betterworth, 2001; Yankovsky & Treves, 2002). Palilalia is widely known as an acquired speech disorder. However, it has been mostly identified in aphasia and most degenerative disorders. It can result from a cerebrovascular damage (Merzbach, 1928, Yasuda et al, 1990), infarcts (Abe et al, 1993) or other neurologic pathologies like epilepsy (Van Bogaert, 1934; Alajouanine et al., 1959; Benke & Butterworth, 2001; Yang-je cho et al, 2009). It can also be observed in Alzheimer disease (Braanten et al, 2006; Barkat-Defradas et al, 2008;Garno, Tempini, et al, 2011; Azevedo et al, 2011), encephalitis (Leyser, 1923; Sterling, 1924),Creutzfeldt-Jacob disease, disease of white matter (Asai et al, 2001; Walterfang et al (2005) and Parkinson and Pick diseases (Mary & levy, 1992; Ikeda & Tanabe, 1992). Some reports also indicate cases of palilalia inschizophrenia and schizophrenia-like (Marie & Levy, 1925; Lapointe& Horner, 1981 Yasuda et al, 1990; Insel, 2010). Palilalia is considered by some authors as a language disorder (Dubois, 2001). Unlike the latter, Critchley (1929), Boller (1972), Boller (1977), Lapointe (1981) Benke & Butterworth (2001), Ikeda & Tanabe (2002), Yang-je Cho et al (2008), amongst others, rather describe palilalia as a speech disorder, since the symptom does not affect the structure of the repeated utterance. Meanwhile, the disorder is, to a certain extent, considered as a fluency

disorder (Ludlow & Loucks, 2003; Chang et al, 2009; Bertutletti, 2012; Lavoie & O'conner, 2012).

Palilalia should, however, be distinguished from other repetitive disorders such as verbal perseveration, stereotypy and echolalia. While perseveration designates an inappropriate repetition of a behavior or an utterance in a repetition task, echolalia refers to the repetition of a speaker's verbal expression in a form of echo. Stereotypy of speech is a segment uttered repeatedly and frequently in almost every situation of conversation (Neary et al, 1998). Reports from the literature point out two types of palilalia: spasmodic palilalia or heterolalia and atonic palilalia or homolalia (Sterling, 1924; Ikeda & Tanabe, 1992; Benke & Butterworth, 2001). According to these researchers, spasmodic palilalia would refer to a compulsive reiteration of utterances with increasing rate, decrease in voice loudness, and content change after interruption, while atonic palilalia would be characterized by the reiteration of utterances with a constant rate, occasionally altering with periods of silence and content maintenance after interruption. Though widely spread, the distinction between the types of palilalia is still questionable. Tools from acoustic methods would address the gap between both spasmodic and atonic palilalia and shed more insights on the origin of the disorder. This raises the question of whether the types of palilalia can yield different explanations of the syndrome.

Previous reports on the explanation of palilalia highlight many causes, the most usual being default in basal ganglia and a motor speech disorder (Lapointe & Horner, 1981; Benke & Butterworth, 2001). The problem with these studies is that brain investigations suggest diverging hypotheses on the explanation of palilalia. In Ikeda & Tanabe (1992), spasmodic palilalia is associated with disturbances of the basal ganglia, damage to the median frontal lobe and atrophy of brain stem, while atonic palilalia is related to fronto-temporal convexity (Norman & Shallice, 1986; Berthier et al, 1996; Neary et al, 1998). In Yankovsky & Treves (2002), patients' fMRI revealed lesions on the left frontal lobe, which led to the conclusion that palilalia is caused by a dysfunction on the left frontal lobe. Another account of palilalia comes from Yang-je Cho et al (2009) where the MRI showed a lesion along the left superior and posterior frontal gyrus, the cortical and subcortical structures. Paramedian thalamic and midbrain infarcts and extra pyramidal system have also been associated with palilalia (Marie & Levy, 1925; Boller et al, 1973; Yasuda et al, 1990). Levelt (1989) points out to control malfunctions at the level of articulators. According to this hypothesis, different verbal repetitions result from various levels of speech system, some at the level of motor speech assembly and others from an additional source at a more central level (Sterling, 1924; Butterworth, 1992; Benke & Butterworth, 2001). This, in other words, relates palilalia to disturbances of the control system (Butterworth, 1992). Obviously, most of these studies relied on experimental brain investigation methods of patients whose electroencephalogram (EEG) or functional magnetic resonance imaging (fMRI) disclosed brain lesions or atrophy of one organ or another. However, researches from the cognitive and neurocognitive domains suggest that motor inhibition problems could be the most plausible explanation

of palilalia and other repetitive disorders like perseveration and as we are putting forward in this paper.

## 1. Methods

The methodological approach used in this paper is mainly empirical; data were collected using a recorder, then transcribed. The transcription was crosschecked by a phonetician.

### 1.1 Research material and methods

Twenty-one patients, that is, eighteen men and three women hospitalized participated to the study. Participants all deliberately signed a consent form, their care givers also signed the same consent form. They were all met at the Psychiatric Center of the Yaoundé Jamot Hospital (PCYJH) in Cameroon. Diagnosis and symptom profile were investigated by psychiatrists using the Structured Clinical Interview for DSM-IV (First et al., 1997). The age interval between patients was 15 – 48 years, with general mean average of 20.23 years. As far as the education background is concerned, all participants attended at least secondary school and they were French speakers.

Palilalia was observed in three participants out of twenty-one. Patient one (BAS) was a 46-year-old-man diagnosed with simple schizophrenia. He was met on the fourth day of hospitalization and was a speaker of basaa', a Cameroonian Bantu language. He was admitted into the hospital for a relapse caused by an interruption of medical treatment. He exhibited a single case of palilalia all through the interview, see example (1). No iteration was observed during the naming task. Patient two (NSS) was a seventeen-year-old young man who was diagnosed with paranoid schizophrenia. He was interviewed on the third day of hospitalization and was a French speaker. He presented a case of palilalia as indicated in (2). Patient three (BMS) was a man aged 41 and was hospitalized for his ninth relapse. He was diagnosed with hebephrenic schizophrenia twenty years before, and he spoke French. Unlike the first two patients, patient three was very productive. Twenty four (24) cases of palilalia were observed in his speech. He was interviewed on the third day. Examples (3), (4) and (5) indicate the types of iterations uttered by BMS. It is worth noting that the three patients were already under treatment at the moment of the interviews.

### 1.2 Procedures

The approaches used are mainly naturalistic, that is observation and interview. A language test was also used whereby patients were expected to recognize and name objects or images. Patients were received in the investigator's office, and they were given a seat at a two meters distance from the table of the investigator. Observation checklists were filled using Information contained in the patients' medical file, those provided by their guardian and the one collected by observation. There was no specific topic for the interview; the conversation depended on each patient since we needed the conversation to be as natural as possible. Each patient was interviewed only once. The interviews

were recorded using a dissimulating Dictaphone. The record file was transferred to a computer and saved as a VLC file, then transcribed orthographically. The linear frequencies of the iterative speech were obtained using the acoustic “Pratt” software.

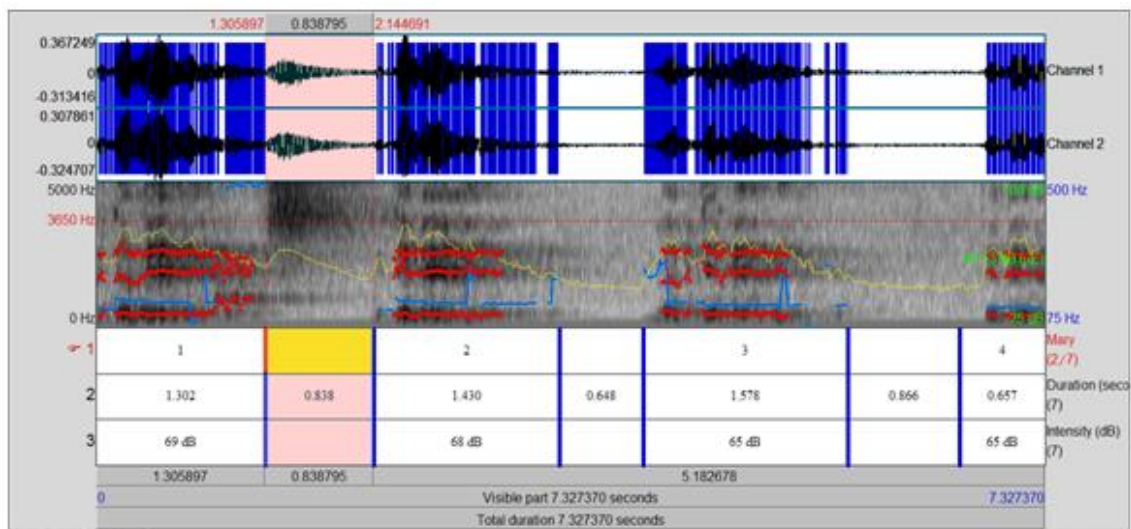
## 2. Results

Reiterations were observed in both spontaneous speech and naming objects. BAS exhibited a single case of palilalia, the symptom occurred in a spontaneous speech as illustrated in (1). No repetition was observed in naming objects or images, or in the repetition task.

(1)

Mbálèbé ì, mbálè 6é ì, mǎmbálè 6é ì, mbálè 6é ì  
 Truth not that truth not that, I say truth not that, truth not that  
 “That is not true that is not true I say that is not true, that is not true<sup>2</sup>”

There are four reiterations of the same sentence without any change in the content of the repetition. There is no change of content in the repetition after interruption, indicating that we are in presence of *homolalic* or *atonic palilalia* as indicated in figure 1.



- Dark= speech stream /HZ
- Blue column = repetitions /ms
- White spaces = pauses / ms
- Yellow curve = Frequency / voice volume
- Red line = highest frequency

Fig 1: Acoustic characteristics of BAS's repetitive speech

<sup>2</sup>This translation was provided by linguists who are native speakers of basaa.



Three main analyses can be carried out from figure 1: first of all, the yellow curve on the spectrogram indicating the frequency in db or the loudness of the reiteration shows that the volume of the repetitive sentence is decreasing, shifting down from 69 db in the first repetition to 65 db in the fourth repetition. Secondly, there are periods of silence between repetitions. The duration of these silences increases considerably from the first to the last repetition. The third observation concerns the duration of each of the iterations which increases from one iteration to another, except the last repetition. Also, the frequency decreases within the same phrase and increases after a period of silence. In sum, the above iterative speech is characterized by a decrease in rate and loudness, and periods of silence. This was also observed in NSS as indicated in (2).

(2)

Mhm ! J'ai dit c'est mon sang éééé....Elle connaît, elle connaît que c'est mon sang, elle connaît<sup>3</sup>...

In this example, two phrases are repeated, resulting in what Lapointe & Horner (1981) called "phrase reiteration within a reiterated phrase (Prp). The first iterated phrase is "c'es tmon sang", while the second one is "elle connaît que". In terms of acoustic features, the above example shares the same characteristics with (1). Among the twenty-four (24) cases of palilalia observed in BMS, only three examples are provided here, that is (3), (4) and (5). All examples occurred in spontaneous speech. However, (5) also occurred sixteen times (16) in naming task and eight (8) times in spontaneous speech.

(3)

900000 milliards<sup>4</sup>... (34 times) was the iterated segment.

This was repeated thirty four times (34) in an increasing rate and increasing voice volume. No silence was observed, except when the patient was breathing. Also, BMS did not show any change in the content of repetition as illustrated in figure 2 below.

<sup>3</sup>Mhm! I said it's my blood eeee.... She does know that it's my blood

<sup>4</sup>"900 000 billions"

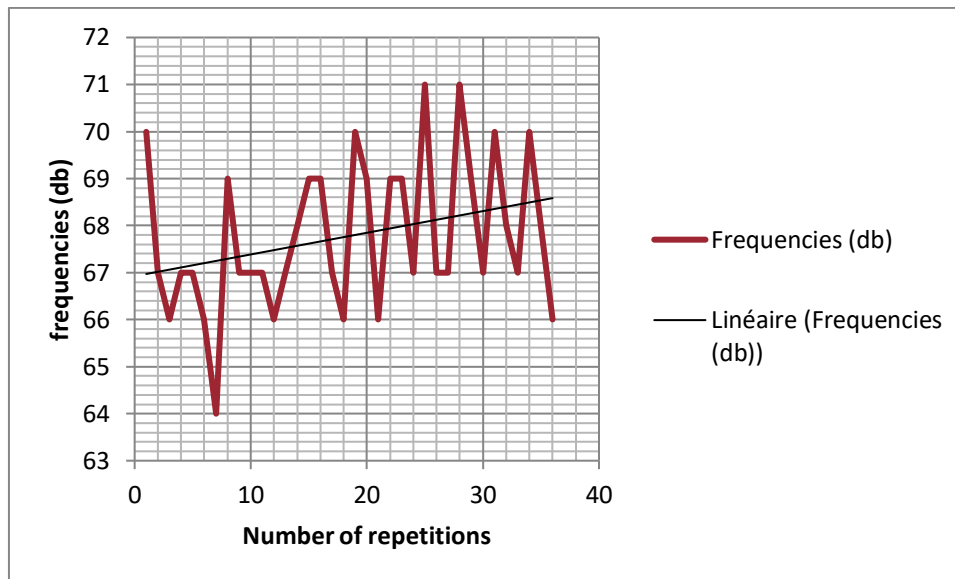


Fig 2: Graphic representation of linear frequency of (3)

Figure 2 represents the linear frequency of 34 iterations of the same number (900 000 milliards) uttered by BMS in a natural conversation. Within this repetitive speech, seven breaths could be observed, dividing the 34 iterations into eight sub groups. The capital observation is that the frequency of the first iteration after each breath is considerably higher than those preceding the breaths. Also, the frequency of the first iteration after the breath depends on whether the breath is brief or long. The dark line indicating the linear frequency shows that the voice volume is increasing (from 67 to 68.5 db).

(4)

900 000 milliards n-tier fois<sup>5</sup>.

In (4), the number of phrase is repeated up to thirteen times in an increasing rate and voice volume. The only moment of silence observed is when the patient is breathing. In (5) below, BMS reiterates the word (5) *Tirétirétirétiré* [tire]), "to pull". Repetition occurs all through the identification task without any change in the content. Every palilalia utterance of this word ends with an unidentified word or syllable [ek]. As far as the rate and loudness are concerned, they are all increasing as indicated in the diagram below.

<sup>5</sup>900 000 billions n-times

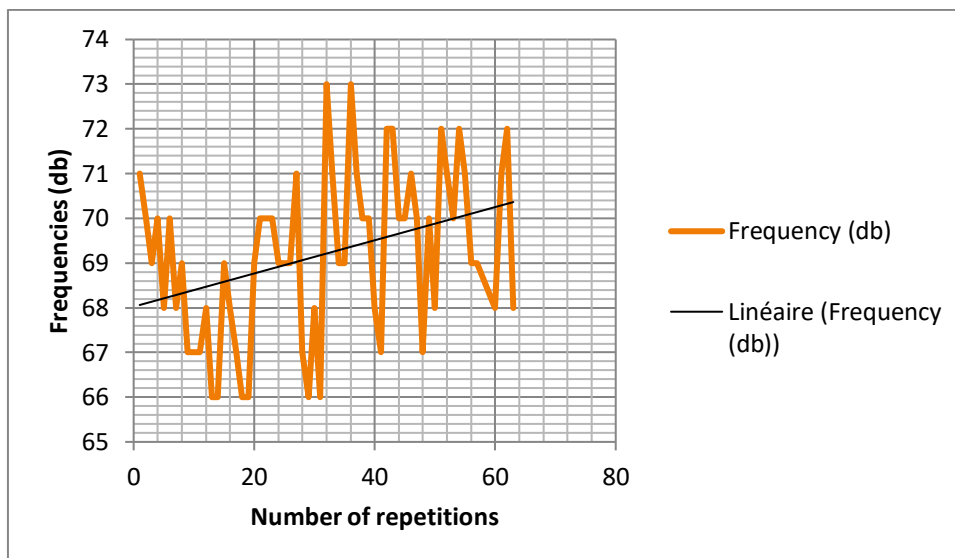


Fig 3: Graphic representation of the linear frequency of (5)

Three observations can be made from this diagram: firstly, while the lowest frequency is 66 db, the highest one is 73 db. Secondly, these frequencies surprisingly fall within the 13<sup>th</sup> and the 36<sup>th</sup> repetitions, while the highest frequency was expected to occur at the first iteration and the lowest one at the final iteration. Thirdly, the dark line indicating the linear frequency shows that in general, the voice volume is increasing, instead of decreasing.

As far as the reiteration types are concerned, four reiteration types were identified in the corpus presented here following the model designed by Lapointe & Horner (1981).

- One-word (W) reiteration, e.g. Tire as in (5).
- Phrase reiteration (P), e.g. mbálè bé i, mbálè bé ì<sup>6</sup>, as in (1)
- Phrase reiteration within a reiterated phrase, e.g. C'est mon sang, elle connaît que c'est mon sang, elle connaît que c'est mon sang.
- Number reiteration, e.g. 900 000 billions.

### 3. Discussion

Structurally speaking, four types of reiteration were observed, that is the one-word reiteration, the phrase reiteration, the sentence reiteration within a reiterated phrase and the number reiteration. This can shed more light on the typology of palilalia in a sense that the typology of reiteration is likely to determine the classification of palilalia. When the repeated segment is a word or a number, the iteration can occur severally in an increasing rate and voice volume. But when the repeated segment is a phrase or a phrase within a repeated phrase, the palilalic utterance will be short of no longer than six iterations realized in a decreasing rate and voice volume and altering with periods of silence. Assuming that long iterated segments result in atonic palilalia while

<sup>6</sup>The translation of the repeated segment here is "That is not true".



short repeated ones yield in spasmodic palilalia, the classification of palilalia will bring in the problem of the management of energy during the repetition.

### 3.1 *The types of palilalia*

Based on the classification of Sterling (1924) attested in Ikeda & Tanabe(1992) and Benke & Butterworth (2001) who identify two types of palilalia (spasmodic and atonic), it is difficult to categorize the above examples because they don't fall in either of the two types. The acoustic analysis of the above iterative speech showed differences within the types of palilalia. Firstly, BAS and NSS combine characteristics of the two types of palilalia described by the above mentioned authors. Decrease in voice loudness from spasmodic palilalia and periods of silence from the atonic type were found in both (1) and (2). The third characteristic of both utterances is a "decrease in rapidity" which, to the best of our knowledge, has not been described before. BAS and NSS can be considered, to some extent, as a type of palilalia characterized by compulsive repetitions of utterances with a decrease in rate and voice volume, and periods of silence. It is worth noting that these characteristics could be signs of tiredness or loss of energy. This in turn results in a kind of monotony of speech which assumingly cannot exceed six iterations; otherwise the patient will get out of breath. Secondly, examples (3), (4) and (5) do not confirm the categorization described in the literature either. The problem lies on the voice volume which is increasing, instead of decreasing as in Sterling (1924), Ikeda & Tanabe (1992) and Benke & Butterworth (2001). The corpus indicates that spasmodic palilalia is not uttered with a decrease in voice volume, revealing specific characteristics of palilalia that had not been addressed before.

The distinction between two types of palilalia is not contested in this paper, instead, the specific characteristics of each type of the disorder need to be reconsidered. It is now clear that the designation "atonic" which means lack of energy fits the type of palilalia described, that is, compulsive repetitions of utterances with a decrease in rate and voice volume, and periods of silence. As opposed to the wide spread description of atonic palilalia (constant rate, periods of silence and content maintenance after interruption), the current corpus clearly indicates the gradual loss of energy (decrease in voice volume and rapidity, increase in pauses duration) through repetitions. As indicated above, this loss of energy could be explained by the characteristics of the linguistics element repeated by the patient. The longer the reiterated segment, the faster the loss of energy. As far as spasmodic palilalia is concerned, it was observed that instead of a decrease in voice volume, participants show an increase in loudness all through the repetitions. This highlighting the original meaning of the word "spasmodic" (compulsive contraction of muscles), increase in loudness and rate are direct result of the compulsivity of speech. This being the case, the classification of palilalia should be reconsidered. The energy-base opposition between the two types of palilalia can be a guide to our explanation of palilalia, raising the question of whether those differences are pertinent to the origin of the disorder.

### 3.2 On the origin of palilalia

Though not converging, hypotheses highlighting neuroanatomical problems such as defaults in basal ganglia (Lapointe & Horner, 1981; Benke & Butterworth, 2001), problems of white matter (Walterfang et al, 2005), calcification of the frontal lobe and atrophy of the brain stem (Norman & Shallice, 1986) have provided materials for the cognitive explanation of palilalia. It is now known that frontal lobes determine the ability to initiate thought and actions and coordinate them with the mental objective to realize them. Also, default in inhibitory processes caused by a reduction in basal ganglia affects motor processing that is implicated in initiating motor processes (Lavoie & O'Conner, 2012). Accounts from the literature indicate that repetitive speech disorders like perseveration, echolalia and palilalia could directly result from deficits of executive functions (Lopez Villegas, 1996; Hemple, 2001, Kipps & Hodges, 2005; Bossikass et al, 2014). Defaults of executive functions could in turn be associated with problems in the frontal lobe and the calcification of basal ganglia (Oliviera & Oliviera, 2013). It is suggested that repetitive speech disorders are associated with a disorder in inhibitory processes (Villegas, 1996; Hemple, 2001; Kipps & Hodges, 2005). It is also stipulated that deficits in inhibitory processes include palilalia (Vitkovich & Hemptreys, 1991; Cohen & Dehaen, 1998; Lavoie & O'Conner, 2012). But when examined closely, palilalia seems to be different from stereotypes, echolalia and perseveration. While stereotypes are characteristics of filled pauses and not necessarily related to executive functions, perseveration and echolalia are respectively associated with conception deficits and default of inhibition. The claim of this hypothesis is that failure to inhibit the realized segment can result in the exteriorization of the same element over and over again.

The problem with this hypothesis is that inhibitory processes involve significant and sustained control abilities, or self-awareness (Norman & Shallice 1986; Moses et al. 2004; Brunet 2010; Barkley 2012). The assumption that palilalia is associated with deficit of inhibition implies that patients could be able to self-regulate their speech, because most of them are conscious of their repetitions (Lapointe & Horner, 1981). Since they are not able to self-regulate their speech, that means palilalia is not related to inhibitory processes. Also, the linear frequency of the above described repetitive speech indicates that voice volume is increasing for spasmodic palilalia and decreasing for atonic palilalia. This is not the case when a disorder is associated with inhibitory disorder, because inhibitory processes are not associated with problems of energy. For instance, perseveration is uttered with a constant voice volume but it is related to inhibitory processes (Villegas, 1996; Hemple, 2001; Kipps & Hodges, 2005). Instead, palilalia seems to be uttered with more energy than ordinarily. Such compulsive utterance cannot be inhibited even when there is no deficit in the process. Inhibitory processes do not actually function in a unitary way. It is hypothesized that motor processes are regulated by two antagonist mechanisms functioning alternatively to achieve the same goal (Moses et al, 2004). These

processes include Activation processes and inhibitory processes. While speaking, syllables, words and phrases are released by activation processes and automatically inhibited when uttered.

The other alternative is, however, more conceivable whereby a dysfunction in activation processes can provide a plausible explanation to palilalia. By definition, palilalia is a compulsive repetitive disorder. This compulsive characteristic results from an energetic contraction of the activation system. This, in turn, can induce a hyper activation of the same segment yielding in a compulsive iterative speech. In this case, the disorder is more associated with problems of energy, the length of the iterated segment, and contractions. Thus, the duration of the contraction depends on the energy supply at the moment of the contraction and the length of the segment to be uttered: when the quantity of energy at the moment of contraction is poor and the segment to be uttered long, the resulting repetitive utterance is realized with a decrease in rapidity, voice volume and sometimes altering with periods of silence (atonic palilalia); when the energy supply is rather exceeding, this results in a compulsive iterative speech produced with increase rapidity and voice volume, that is spasmodic palilalia. Activation processes are involved in a number of disorders that include cognitive, physical, and social behaviors. Based on the above explanations, palilalia would not directly related to a neurophysiological problem as discussed in cases of aphasia, dementia, front temporal atrophy or the calcification of basal ganglia, etc. because its presence in schizophrenia where brain investigations do not generally disclose any neurophysiological anomaly. This is an evidence that the origin of palilalia could be somewhere else. Concerning the cognitive origin of palilalia, the compulsive characteristic of palilalia provides arguments against the hypothesis of a deficit in inhibitory processes which do not involve changes in rapidity or voice volume. Thus, our assumption is that palilalia can originate from a disorder in the activation processes of executive functions, resulting a compulsive contraction of activation processes with implications in speech flow characterized by spasmodic or atonic iterations.

## Conclusion

This paper aimed at providing an explanation to palilalia, a compulsive iterative speech disorder identified in schizophrenic patients. It was originally hypothesized that defaults in activation processes can be the explanation to the disorder. The acoustic characteristics of the utterances disclosed two opposing types of palilalia: instead of the original characteristics of palilalia, an energy-based description of palilalia was disclosed our data, bringing in arguments for compulsive contractions of activation mechanisms. In sum, it is argued that palilalia may neither be related to a neurophysiological problem nor may it be related to deficits in inhibitory process as discussed in previous works, but to compulsive contractions of activation processes, one of the most important executive functions involved in language production. However, our study is mainly empirical that is, it relies on naturalistic methods. This can bias our findings, for we did not use any experimental method to investigate the cognitive

capacities of our patients. Therefore, further studies on this topic need to bring in a language test that can help identify cognitive deficits.

### Bibliographic references

- ABE Keiko Ritsuko Yokoyama and SHIRO Yorifuji. 1993. "Repetitive speech disorders resulting from Infarcts in the Paramedian Thalamic and Midbrain", *Journal of Neurology, Neurosurgery and Psychiatry*, 56: 1024-1026.
- ALAJOUANINE Thomas et al. 1959. "Palilalie Paroxystique et vocalisations itératives au cours de crises Epileptiques par lésion intéressant l'aire motrice supplémentaire", *Revue Neurologique*, 101:685-699.
- American Psychiatric Association. 1994. *Diagnostic and statistical manual of mental disorders. DSM-IV 4th ed.* Washington, DC: American Psychiatric Association.
- ASAI Yuka. et al. 2001. "Alpha-like activity in terminal stage of Creutzfeldt-Jakob disease", *Acta NeurolScand*; 104: 118-122.
- AZEVEDO Jacinto et al. 2011. "Clozapine-induced palilalia?" *Acta Neuropsychiatrica*, 24: pp.122-124.
- BARKAT-DEFRADAS et al. 2008. « Les troubles du langage dans la Maladie d'Alzheimer ». 27 journées d'études sur la Parole, Jun 2008, Avignon, France. Pp. Actes électroniques non numérotés, hal-00321233.
- BENKE Thomas & BETTERTWORTH Brian. 2001. "Palilalia and Repetitive Speech: Two case studies", *Brain and Language*, 78:62-81.
- BERTHIER Marcello et al. 1996. "Obsessive-compulsive Disorder associated with Brain lesions: Clinical phenomenology, cognitive function, and anatomic correlates", *The American Academy of Neurology*, 47: 353-36.
- BERTULETTI Laure. 2012. *Impact d'une rééducation orthophonique des fonctions exécutives sur le langage oral chez les sujets aphasiques*, dumas-00728808, version 1.
- BOLLER Francis et al. 1975. "Palilalia", *British Journal of communication disorders*, 10: 92-97.
- BOLLER Francis et al. 1973. „Family Palilalia", *Neurology*, 23: 1117-1123.
- BOSSIKASS Nicolas et al. 2014. "Fluency in Parkinson Disease: disease duration cognition status and age". *ArqNeuropsiquir*, 72: 349-355.
- BRAATEN Alyssa et al. 2006. "Neurocognitive differential diagnosis of dementing diseases: Alzheimer's dementia, Vascular Dementia, frontotemporal dementia, and major depressive disorder". *Intern. J. Neuroscience*, 116 : 1271-1293.
- BRUNET, Stéphane., (2010), *Les Persévérations verbales: Interprétations cognitives chez la personne aphasique et mise en lien avec les théories de l'effet de priming*. Travail de tutorat concernant le stage spécialisé, Université Libre de Bruxelles.
- BUTTERWORTH Brian. 1992. "Disorders of phonological encoding", *Cognition*, 42: 261-286.

- BUTTERWORTH Brian. 1980b. "Evidence from pauses. In B. Butterworth (Ed.)", *Language production, Vol. 1: Speech and talk*. London: Academic Press.
- BUTTERWORTH Brian. 1980a. "Some Constraints on models of Language Production". In B. Butterworth (Ed.), *Language production, Vol. 1: Speech and talk*. London: Academic Press.
- CHANG et al. 2009. "Similarities in speech and white matter characteristics in idiopathic developmental stuttering and adult-onset stuttering», *Journal of neurolinguistics*: 1-15.
- COHEN Leonard. & Dehaene Stanislas. 2011. "The Unique Role of the Visual Word form area in Reading", *Trends in Cognitive Sciences*, 15 (6): 254-62.
- COHEN Leonard. & Dahaene, Stanislas. 1998. "Competition between past and present, Assessment and interpretation of verbalperseveration". *Brain*, 21: 1641-1659.
- CRITCHLEY, E. M. R. 1998. "Speech disorders of Parkinsonism A review". *Journal of Neurology, Neurosurgery and Psychiatry*, 44: 751-758.
- CRITCHLEY Michel. 1927-1928. "On Palilalia", *Journal of Neurology, Neurosurgery and Psychiatry*, 18:23-31.
- DUBOIS Jean et al. 2001. *Dictionnaire de linguistiques, Larousse*, 2<sup>nd</sup> Edition.
- FERGUSSON Jams. & Boller Francis. 1977. "A different form of Agraphia: Syntactic writing errors in patients motor speech disorders", *Brain and language*, 4: 382-389.
- GARNO Tempini et al. 2011. "Classification of primary progressive Aphasia and its variants", *Neurology*, 76: 1006-1014.
- HEMPLE Amelia. et al. 2001. "PET findings and neurophysiological deficits in a case of Fahr's disease", *Psychiatry Res*, 108: 133-140.
- IKEDA Manabu & Tanabe Hadime. 1992. "Two forms of Palilalia: A clinicoanatomical study", *Behavioural Neurology*, 5, 241-246.
- INSEL Thomas Roland. 2010. "Rethinking Schizophrenia". *Nature*, 468: 186-193.
- KIPPS Christopher & Hodges John. 2005. "Cognitive assessment for clinicians", *J NeurolNeurosrg Psychiatry*, 76: 22-30.
- LAPOINTE Leonard & Horner Jennifer. 1981. "Palilalia: A descriptive study of pathological repetitive utterances", *American speech- language- hearing association*, 0022: 34-38.
- LAVOIE Marc & O'conner Kieron. 2012. *Neurocognitive Aspects of Tourette Syndrome and Related Disorders When Things Go Wrong - Diseases and Disorders of the Human Brain*, Dr. Theo Mantamadiotis (Ed.), ISBN: 978-953-51-0111-6, InTech, Available from: <http://www.intechopen.com/books/when-things-gowrong-diseases-and-disorders-of-the-human-brain/neurocognitive-aspects-of-tourette-syndrome-and-related-disorders>.
- LEYSER Edith. 1923. „Die zentralen Dysarthrien und ihre Pathogenese“, *Klinische Wochenschrift*, 2:2176-2179.
- LEVELT, W. J., Praamstra, P., Meyer, A. S., Helenius, P., & Salmelin, R. (1998), "AnMEG study of picture naming. *Journal of Cognitive Neuroscience*, 10(5): 553-567.



- LEVY Michael, Miller Bryan Lee., & Cummings Jason. 1998. Frontal and Frontotemporal dementia. In J. H. Growdon & M. N. Rosser (Eds.), *The dementias*. Boston, MA: Butterworth-Heinemann, 45-65.
- LUDLOW, Christy. & Loucks Torrey. 2003. "Stuttering: a dynamic motor control disorder", *Journal of Fluency Disorders*, 28: 273-295.
- MARIE Pierre. 1992. « Palilalie et syndrome Parkinsonien par Encephalite epidemique ». *Rev Neurol (Paris)*, 29:66.
- MARIE Pierre & Levy G. 1925. Un singulier trouble de la parole. *Monde Med* 1925; 35: 6-80.
- OLIVIERA Michel & Oliviera Francis. 2013. "Basal Ganglia Calcification as putative cause for cognitive decline", *Dement Neuropsychology*, 7: 151-154.
- MERZBACH Alexander. 1928. "Die Sprachiteration en und ihre Lokalisation bei Herderkrankungen des Gehirns". *Journal für Psychologie und Neurologie*, 36 :211-316.
- MOSES M S et al. 2004. « I'm sitting here filling Aphasic! A study of recurrent perseveration errors elicited in unimpaired speakers", *Brain and language*, 89: 157-173.
- NEARY David et al. 1998. "Fronto-temporal lobar Degeneration: A consensus on clinical diagnostic criteria". *Neurology* 51: 1546-1554.
- NORMAN Donald Arthur & Shallice Tim. 1986. "Attention to action: Willed and automatic control of behavior", In R. J. Davidson, G. E. Schwartz, & D. Shapiro (Eds.), *Consciousness and self-regulation* (Vol.4). New York: Plenum.
- STERLING William. 1924. « Palilalie et le Symptôme 'Linguosalivairé' dans le Parkinsonisme Encéphalique », *Revue Neurologique*, 32:205-220.
- VAN Bogaert. 1934. "Ocular paroxysms and Palilalia", *The Journal of Nervous and Mental Diseases*, 80:48-61.
- VITKOVITCH & Humphreys Glyn. 1991. "Perseverant responding in speeded naming of pictures. It's in the links", *Journal of Experimental Psychology: Learning, Memory and Cognition*, 17: 664-680.
- WALTERFANG Mark. 2005. "Disease of white matter and schizophrenia-like psychosis", *Australian and New-Zealand Journal of Psychiatry*, 39: 746-756.
- WOOD Eric Francis. 1980. "Patterns of regional cerebral blood flow during attempted reading aloud by stutterers both on and off haloperidol medication: Evidence for inadequate left frontal activation during stuttering", *Brain and Language*, 9: 141-144.
- YANG-JE cho et al. 2009. "Palilalia, Echolalia, and Echopraxia-Palipraxia as Ictal manifestations in a patient with left frontal lobe Epilepsy", *Epilepsia*, 50(6):1616-1619.
- YANKOVSKY Alexander & Treves Adrian. 2002. *Postictal mixed transcortical aphasia*. *Seizure*, 11: 278-279.
- YASUDA Akiguchi et al. 1990. "Paramedian Thalamic and Midbrain Infarcts associated with Palilalia", *Journal of Neurology, Neurosurgery and Psychiatry*, 53:797-9.