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Electroencephalography in Primary Progressive Aphasia and Apraxia of Speech

Rene L. Utianski, PhD¹, John N. Caviness, MD², Gregory A. Worrell, MD¹, Joseph R. Duffy, PhD¹, Heather M. Clark, PhD¹, Mary M. Machulda, PhD³, Jennifer L. Whitwell, PhD⁴, Keith A. Josephs, MD MST MSc¹

¹Department of Neurology, Mayo Clinic, Rochester, MN

²Department of Neurology, Mayo Clinic, Phoenix, AZ

³Department of Psychiatry & Psychology, Mayo Clinic, Rochester, MN

⁴Department of Radiology, Mayo Clinic, Rochester, MN

Abstract

Background: Past research has demonstrated that electroencephalography (EEG) is sensitive to what we now know as Primary Progressive Aphasia (PPA); however, the EEG profiles of patients with Primary Progressive Apraxia of Speech (PPAOS) and PPA, in the context of current consensus criteria, have not been studied.

Aims: The primary goal of this study was to explore the EEG profiles of patients of the nonfluent/agrammatic variant of PPA (agPPA) and PPAOS.

Methods and Procedures: Three patients with agPPA and five patients with PPAOS (two with aphasia) completed a head MRI scan and clinical EEG recording. Clinical radiologists and electrophysiologists reviewed respective imaging, blinded to clinical diagnosis.

Outcomes and Results: Patients with PPAOS who did not have aphasia had normal EEGs, while those with aphasia demonstrated theta slowing. Patients with agPPA also showed theta slowing, with one exception. MRI scans showed non-specific, age-related changes across clinical presentations.

Conclusions: This preliminary study suggests theta slowing is consistent with neurodegenerative aphasia, but not isolated apraxia of speech. EEG is a low-cost mechanism to identify possible biomarkers for use when clinical severity limits behavioral examinations or expert examiners are unavailable.

Keywords

Frontotemporal dementia; Primary Progressive Aphasia; Primary Progressive Apraxia of Speech; Electroencephalography (EEG)

Introduction

The use of electroencephalography (EEG) has expanded from identifying and characterizing seizure disorders to differentiating many different cerebral functions. Past research has demonstrated that EEG is sensitive to dementia associated with Alzheimer's disease (de Waal et al., 2011), Parkinson's disease (Caviness et al., 2007), and Lewy bodies (Briell et al., 1999), and what we now know as Primary Progressive Aphasia (PPA) (Mesulam, 1982) at varying stages of the disease processes. Nonetheless, the EEG profiles of patients with Primary Progressive Apraxia of Speech (PPAOS) and PPA, in the context of current consensus criteria (Gorno-Tempini et al., 2011), have not been studied.

PPA is a term that encompasses a group of neurodegenerative syndromes characterized by progressive language impairment (Mesulam, 2001). The nonfluent/agrammatic variant of PPA (agPPA) is characterized by grammatical errors in speech and writing and, not infrequently, apraxia of speech (Gorno-Tempini et al., 2011). When AOS, and not aphasia, is the initial manifestation of neurodegenerative disorders it is referred to as primary progressive apraxia of speech (PPAOS) (Josephs et al., 2012). In the context of PPAOS, some patients eventually develop a milder aphasia (PPAOS with aphasia). It is unclear if the initial or combination of features is indicative of underlying pathology or relates to the anticipated progression of the neurodegenerative disorder that develops.

The primary goal of this study was to improve our understanding of the EEG profiles of patients with agPPA and PPAOS. Both agPPA and PPAOS have been related to underlying tauopathies (e.g. corticobasal degeneration, progressive supranuclear palsy) (Grossman, 2010; Josephs et al., 2006; Mesulam et al., 2014). However, apraxia of speech is associated with damage to areas of the motor cortex (e.g. supplementary motor area), but is not fully localizable when the stroke literature is considered. Aphasia on the other hand is related to focal, cortical damage (e.g. Broca's area). Recent studies of *in-vivo* tau deposition in agPPA and PPAOS patients suggested cortical spread of tau uptake was associated with aphasia rather than apraxia of speech (Utianski, Whitwell, Schwarz, Duffy, et al., 2018; Utianski, Whitwell, Schwarz, Senjem, et al., 2018). We therefore hypothesized that patients with an isolated apraxia of speech (i.e. PPAOS) will have normal EEGs, while those who have aphasia will have abnormal EEGs (Mesulam, 1982).

Methods

The study was approved by Mayo Clinic's Institutional Review Board; all patients were native English speakers and gave written consent. Between October 2016 and March 2018, three patients with agPPA and five patients with PPAOS (two with aphasia) completed behavioral (speech, language, and neurological) examinations, a 3.0 Tesla volumetric head MRI scan, and clinical EEG recording.

Details of the clinical exam are consistent with those reported in a recent study (Utianski, Duffy, et al., 2018). Briefly, language examinations included the Western Aphasia Battery-Revised (Kertesz, 2007), from which the Aphasia Quotient (AQ) served as a composite measure of global language ability; the Token Test, Part V (De Renzi & Vignolo, 1962)

indexed verbal comprehension of complex instructions; the Boston Naming Test, short form (BNT) (Lansing, Ivnik, Cullum, & Randolph, 1999) served as a sensitive measure of confrontation naming ability; and the Northwestern Anagram Test (NAT) (Weintraub et al., 2009) as a measure of grammar integrity. Patients were required to perform abnormally on at least two measures of language functioning for a diagnosis of aphasia (note, not all measures are reported here). Thorough examinations of the oral mechanism, and auditory characterization of speech output, were completed to determine the presence, nature, and severity of motor speech disorder(s). Data were reviewed independently by two certified speech-language pathologists who reached a consensus diagnosis, utilizing previously reported operational definitions of PPA variants and PPAOS (Botha et al., 2015; Gorno-Tempini et al., 2011).

Neurologic examinations included the Montreal Cognitive Assessment (MoCA) (Nasreddine et al., 2005) to index general cognitive ability; the limb apraxia subtest of the WAB to assess limb ideomotor apraxia; the Movement Disorders Society-sponsored version of the Unified Parkinson's Disease Rating Scale motor subsection (UPDRS III) (Goetz et al., 2008) to index parkinsonism; and the Progressive Supranuclear Saccadic Impairment Scale (PSIS) to evaluate saccades.

Clinical radiologists reviewed the MRI scans and were blinded to clinical diagnosis. EEG recordings were collected with XLTEK, utilizing standard 10-20 positions, a sampling rate of 256Hz, and a 1-200Hz bandpass filter, during relaxed wakefulness. A clinical electrophysiologist reviewed all EEG recordings, blinded to clinical diagnosis, and made judgments of overall function. Posterior dominant rhythms (PDR) were visually identified. The clinical interpretations were reviewed for reliability by the primary author, a trained research electrophysiologist. No discrepancies were noted.

Results

Demographic information and results of behavioral examinations are reported in Table 1. Overall performance on language measures was consistent with the presence or absence of aphasia in the respective clinical groups, as they were used in making such determination. Three patients (all PPAOS) presented with dysarthria that was mild-moderate in severity. Limb praxis ranged from normal functioning to moderately impaired. Scores on the MDS-UPDRS III suggested a range from minimal to moderate Parkinsonism. Saccade functions ranged from normal to markedly impaired (per the PSIS). The patients did not meet clinical criteria for any other neurologic diagnosis at testing, including, but not limited to, progressive supranuclear palsy (Hoglinger et al., 2017; Litvan et al., 1996), corticobasal syndrome (Armstrong et al., 2013; Boeve, Lang, & Litvan, 2003), or Alzheimer's disease dementia (Albert et al., 2011; Dubois et al., 2014; McKhann et al., 1984).

MRI and EEG results are detailed in Table 2. Briefly, patients with PPAOS who did not have aphasia had normal EEGs, while those with aphasia demonstrated theta slowing. Theta slowing was defined as intermittent focal theta (4-7 Hz), or low amplitude diffuse theta, not expected for the patient's age or mental status (i.e. awake). Patients with agPPA also showed theta slowing, with one exception who demonstrated excess beta activity. MRI scans showed

non-specific, age-related changes across clinical presentations. All patients had generalized cerebral and cerebellar atrophy on MRI. Chronic small vessel ischemic changes were noted in five patients and leukoaraiosis was noted in four patients. Ex vacuo dilation of the ventricular system was noted in two patients, one with agPPA and one with PPAOS.

Discussion

This preliminary study suggests that clinical EEGs may be sensitive to the presence of aphasia, a cortically mediated process, in patients with PPAOS and PPA, but not reflect changes associated with isolated apraxia of speech. Our findings are consistent with those reported in Mesulam's seminal paper on PPA (Mesulam, 1982). Mesulam described EEG findings associated with PPA, noting left temporal slowing in all patients at some point in the disease course (Mesulam, 1982). In the current cohort, all but one patient (4/5) with aphasia, whether in the context of AOS or not, had theta slowing noted in the left temporal region. Disease duration was somewhat reduced in the patient for whom theta slowing was absent (P1) compared to the other agPPA patients; it is possible a shorter disease duration accounts for the absence of theta slowing. In fact, P1 (agPPA) demonstrated excess beta activity, an EEG pattern sometimes associated with medications (e.g. benzodiazepines); interestingly, this patient was not on any such medications at the time of exam. Alternative explanations for this finding have not yet been identified.

In Mesulam's study (Mesulam, 1982), one patient was described as having an "aphasic-apraxic disorder" and demonstrated frontotemporal sharp and slow waves, also seen in one of our patients with PPAOS with aphasia (P5). Patients with PPAOS who did not have aphasia had normal EEGs (P6-P8). Given the severity of AOS in these patients ranged from mild to severe, the differences between the patients with agPPA and PPAOS do not appear to be an issue of severity, but rather related to the clinical presentation.

Given past research in dementia (Briell et al., 1999; Caviness et al., 2007; de Waal et al., 2011), we expected to see a reduced PDR. This does not appear to be the case in either PPA or PPAOS, as the PDR for all patients in this cohort was within normal limits. PDR does not appear to have an obvious association with clinical syndrome or severity of symptoms. However, the patient with the longest disease duration (P3; 7 years) additionally demonstrated frontal predominant delta activity and lacked a clear PDR; it is possible PDRs become disrupted later in the course of disease progression and is associated with longevity of disease rather than severity of clinical presentation. Longitudinal study of these patients will allow us to confirm this speculation empirically.

MRI findings in this cohort were used as a comparison for neuroimaging correlates of clinical presentation. Overall, the patterns on visual inspection appear undifferentiated between the patient groups and, in fact, are visually indistinguishable among the patient cohorts. All patients had generalized cerebral and cerebellar volume loss on MRI. Chronic small vessel ischemic changes were noted in five patients and leukoaraiosis was noted in four patients. The aforementioned patterns on MRI are consistent with those noted in normal aging; however, assessing their potential relationship to EEG findings (e.g. risk factor) in this patient population is of interest in future studies. Ex vacuo dilation of the ventricular

system was noted in two patients, one with agPPA and one with PPAOS; the significance of this finding is unknown, especially whether it relates to clinical presentation.

Importantly, the diagnosis of agPPA and PPAOS were made based on the clinical presentations. The absence of imaging-supported criteria for agPPA can be considered a limitation of the present study, although imaging support is secondary to the clinical diagnoses in the diagnostic criteria. An additional limitation of the study is the small sample size, which of course minimizes the degree to which some of the data can be interpreted. Future studies will utilize a larger sample size and quantitative EEG.

EEG is able to evaluate cortical physiological activity many times faster than other imaging techniques, in a non-invasive, cost-effective manner, and may be more specific than MR. EEG may provide possible diagnostic biomarkers in this population which may be important when the clinical diagnosis is difficult to make secondary to the severity of the clinical presentation or when expert opinions are not available to facilitate differential diagnosis. EEG findings may also give clues to the pathophysiology. Both agPPA and PPAOS have been associated with corticobasal degeneration (CBD) and progressive supranuclear palsy (PSP). Previous research of EEGs in CBD and PSP have yielded mixed results, including normal EEGs and frontal intermittent rhythmic delta activity in both clinical presentations, with focal slow waves (Tashiro et al., 2006) and asymmetric PDR (Vion-Dury, Rochefort, Michotey, Planche, & Ceccaldi, 2004) additionally seen in CBD. Whether EEG findings reflect the underlying pathology or predict the disease trajectory is an empirical question. Certainly, if EEG findings relate to underlying pathology, it may qualify patients for pharmacological interventions. If it relates to the clinical presentation, it may affect the approach to symptomatic treatment (i.e. motor speech versus language) and could potentially serve as a biological outcome measure for treatment studies. Recruitment is ongoing to determine if these preliminary trends remain true in a larger cohort of patients and longitudinally.

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Disclosure of interest

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Table 1

Clinical, demographic, speech, language, and neurologic data for each patient. Notes: Disease duration = time from reported onset to exam; agPPA = nonfluent/agrammatic variant of Primary Progressive Aphasia; PPAOS = Primary Progressive Apraxia of Speech; F = Female; M = Male; severity was rated on 0-4 scale (0 = normal; 4 = severe); MoCA = Montreal Cognitive Assessment; WAB AQ = Western Aphasia Battery Aphasia Quotient; BNT = Boston Naming Test, short form; TT = Token Test; NAT = Northwestern Anagram Test; MDS-UPDRS III = the Movement Disorders Society- sponsored version of the Unified Parkinson's Disease Rating Scale, motor subsection; PSIS = Progressive Supranuclear Saccadic Impairment Scale. Maximum score for each assessment is noted in the column header; higher scores indicate better performance, with the exception of the MDS-UPDRS III and PSIS where higher scores reflect greater impairment.

Patient	Diagnosis	Sex	Age	Disease Duration (years)	Aphasia/AOS/ Dysarthria Severity (/4)	MOCA (/30)	WAB AQ (/100)	BNT/(15)	TT (/22)	NAT (/10)	WAB Praxis (/60)	MDS-UPDRS III (/120)	PSIS (/5)
P1	agPPA	F	69	2	1.5/0/0	21	92.7	14	DNT	0	53	13	0
P2	agPPA	M	51	4	3/0/0	DNT	DNT	0	DNT	DNT	DNT	4	0
P3	agPPA	F	59	7	2/2/0	DNT	DNT	DNT	DNT	DNT	40	19	2
P4	PPAOS with aphasia	F	75	4	2/4/1	19	71.8	DNT	14	7	45	35	4
P5	PPAOS with aphasia	M	68	5	1/2.5/0	25	97.1	13	18	5	59	5	2
P6	PPAOS	M	81	2	0/1/1	29	97.8	15	18	10	58	18	1
P7	PPAOS	M	82	8	0/4/2	26	96.6	13	19	9	51	55	0
P8	PPAOS	M	73	2	0/1/0	30	98.8	15	19	10	59	8	1

Table 2

MRI and EEG findings for each patient. Notes: MRI=head magnetic resonance imaging scan; EEG=electroencephalographic recording; PDR=posterior dominant rhythm; agPPA= nonfluent/agrammatic variant of Primary Progressive Aphasia; PPAOS=Primary Progressive Apraxia of Speech.

Patient	Diagnosis	MRI Findings	EEG PDR (Hertz)	Clinical EEG Read
P1	agPPA	<ul style="list-style-type: none"> •Moderate generalized cerebral and cerebellar volume loss. •Ex vacuo dilatation of the ventricular system. •Moderate chronic small vessel ischemic changes. •Minimal leukoatrophy. 	10	Excess beta, generalized (questionable medication effect)
P2	agPPA	<ul style="list-style-type: none"> •Moderate generalized cerebral and cerebellar volume loss, more prominent in the frontal and anterior temporal lobes. 	9-10	Intermittent left temporal theta slowing
P3	agPPA	<ul style="list-style-type: none"> •Mild to moderate generalized cerebral and mild cerebellar volume loss. •Mild chronic small vessel ischemic changes. 	Diffuse 8-9 Hertz without a clear PDR	Diffuse theta: frontal predominant delta activity, maximal right
P4	PPAOS with aphasia	<ul style="list-style-type: none"> •Moderate generalized cerebral and cerebellar volume loss. •Mild chronic small vessel ischemic changes. •Mild leukoatrophy. 	10	Intermittent left temporal theta
P5	PPAOS with aphasia	<ul style="list-style-type: none"> •Moderate cerebral and cerebellar volume loss, slightly more prominent in the parietal lobes. •Moderate leukoatrophy. 	11	Intermittent sharp left frontotemporal theta
P6	PPAOS	<ul style="list-style-type: none"> •Moderate generalized cerebral and cerebellar volume loss. •Moderate chronic small vessel ischemic changes. 	10	Normal
P7	PPAOS	<ul style="list-style-type: none"> •Moderate severe generalized cerebral and cerebellar volume loss. •Ex vacuo dilatation of the ventricular system. •Mild to moderate chronic small vessel ischemic changes. 	8-9	Normal
P8	PPAOS	<ul style="list-style-type: none"> •Mild to moderate generalized cerebral volume loss. •Mild cerebral leukoatrophy. 	10-11	Normal