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Age-accelerated psychomotor slowing in temporal lobe epilepsy

Connie Sung[a,1], **Jana E. Jones**[b,*], **Daren C. Jackson**[b,2], **Yui Chung Chan**[c,3], **Fong Chan**[d,4], **Michael Seidenberg**[e,5], **Bruce P. Hermann**[b,2]

[a] *Department of Counseling, Educational Psychology and Special Education, Michigan State University, Erickson Hall, 620 Farm Lane Room 455, East Lansing, MI 48824, United States*
[b] *Department of Neurology, University of Wisconsin School of Medicine and Public Health, 1685 Highland Ave, Madison, WI 53705, United States*
[c] *Department of Counseling Psychology and Guidance Services, Teachers College, Room 1008, Ball State University, Muncie, IN 47306, United States*
[d] *Department of Rehabilitation Psychology and Special Education, University of Wisconsin, 403 Education Building, 1000 Bascom Mall, Madison, WI 53706, United States*
[e] *Department of Psychology, Rosalind Franklin School of Medicine and Science, 3333 Green Bay Road, North Chicago, IL 60064, United States*

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Summary Cognitive and psychomotor slowing is a complication of epilepsy and is less often a focus of investigation relative to other cognitive domains (e.g., memory). A diversity of tasks has been used to examine psychomotor slowing in epilepsy, but it remains unknown whether the degree of epilepsy-related slowing is fixed or is exacerbated with increasing task demand. Also unknown is to what degree age related slowing is accelerated in epilepsy. Participants with temporal lobe epilepsy ($n = 50$) were compared to healthy controls ($n = 69$) across three tasks of psychomotor speed with varied complexity. Performance was examined as a function of group (epilepsy, controls), task complexity (simple, intermediate, complex), and chronological age. The results showed that speed of performance declined across the epilepsy and control participants as a function of task complexity. Epilepsy participants were significantly slower than controls across the three tasks, and there was a significant three-way interaction (group by task complexity by age). These results demonstrate that psychomotor slowing is related to

* Corresponding author. Tel.: +1 608 262 5481; fax: +1 608 263 0412.
E-mail addresses: csung@msu.edu (C. Sung), jejones@neurology.wisc.edu (J.E. Jones), jackson@neurology.wisc.edu (D.C. Jackson), ychan@bsu.edu (Y.C. Chan), chan@education.wisc.edu (F. Chan), michael.seidenberg@rosalindfranklin.edu (M. Seidenberg), hermann@neurology.wisc.edu (B.P. Hermann).
[1] Fax: +1 517 353 6393.
[2] Fax: +1 608 263 0412.
[3] Fax: +1 765 285 5455.
[4] Fax: +1 608 262 8108.
[5] Fax: +1 847 578 8758.

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task complexity in both epilepsy and healthy control participants, always greater in epilepsy participants, and there is a significant age acceleration of psychomotor slowing in the epilepsy group that is magnified by complex tasks.

Introduction

The potential neurocognitive complications of chronic temporal lobe epilepsy are generally well appreciated and may include problems in memory, language, and executive function (Cahn-Weiner et al., 2009; Dodrill, 2004; Helmstaedter and Elger, 2009; Jokeit et al., 2000; Mameniskiene et al., 2006; Riley et al., 2010). While psychomotor speed has been a less frequent focus of attention as a core complication of epilepsy, it appears to be a broadly represented abnormality that has been reported in investigations of neuropsychological function in adults with chronic epilepsy (Piazzini et al., 2006) as well as children with established cryptogenic localization-related (van Mil et al., 2010) and uncomplicated epilepsies (Boelen et al., 2005). Psychomotor slowing has also been reported in children and adults with new onset epilepsies prior to initiation of medications (Oostrom et al., 2003; Prevey et al., 1998; Taylor et al., 2010). Furthermore, especially in adults, psychomotor slowing worsens over time and exceeds the rate of change in many other cognitive abilities (Baker et al., 2011; Hermann et al., 2006). In addition, psychomotor slowing is an appreciated complication of seizure medications (Eddy et al., 2011; Loring et al., 2007; Park and Kwon, 2008), an impact that may be added to the intrinsic slowing that can be observed antecedent to medication initiation in pediatric and adult new onset epilepsies (Oostrom et al., 2003; Taylor et al., 2010).

Normal aging is well known to be associated with slowing of psychomotor speed (Era et al., 2011), an effect that has been attributed to age-related disruption of both cerebral white (Lu et al., 2011) and grey matter (see Seidler et al., 2010 for a review). How normal aging processes may impact already affected cognitive systems in epilepsy remains an area of active investigation, with little work devoted to characterizing the added impact of age to already evident psychomotor slowing.

Complicating the issue further is the diversity of tasks used in this literature. Psychomotor speed has been measured using a wide range of psychomotor tasks that vary in their complexity and relation to real world situations. Available tasks range from simple and uncomplicated unimanual tasks to more complicated bimanual assembly tasks, and include tests of simple and complex reaction time, motor dexterity, coding, assembly and other tasks. For instance, in a sample of 65 adults with temporal lobe epilepsy who participated in a comprehensive neuropsychological evaluation, Wang et al. (2011) found that 86% of the sample had some form of cognitive impairment and of those 69% had deficits in psychomotor speed as measured by the Purdue Pegboard. Exner et al. (2002) examined a small sample of adults with frontal lobe epilepsy ($n = 16$) and temporal lobe epilepsy ($n = 16$) and reported psychomotor slowing in both groups when compared to healthy controls on the Trail Making Test (A and B). On the other hand, in another study of individuals with frontal lobe epilepsy (FLE), temporal lobe epilepsy, and controls employing the Trail Making Test of the Delis-Kaplan Executive Function System, McDonald et al. (2005) reported no difference in motor speed between individuals with temporal lobe epilepsy and healthy controls groups, and this finding may be attributed to younger age of the sample. Nevertheless, there has been no systematic investigation of psychomotor slowing as a function of task demand and complexity, the degree to which these task demands may exert differential effects on participants with epilepsy vs. healthy controls, and whether normal age-related changes in speeded performances are exacerbated aging individuals with epilepsy.

In this investigation we administered three psychomotor tasks of increasing complexity to individuals with epilepsy and healthy controls, measures that have been widely used to assess psychomotor speed and function in various settings (Grigsby et al., 2008; Sachdev et al., 2005; Wright et al., 2008). Based on the existing research on psychomotor slowing and epilepsy, we hypothesized the following: (a) individuals with epilepsy would perform more slowly across all tasks than controls; (b) the impairment in the epilepsy group would be magnified as a function of task complexity; and (c) age-accelerated progression of this impairment would be observed in the epilepsy group relative to controls.

Methods

Participants

A total of 119 research participants were the focus of this investigation including 50 individuals with temporal lobe epilepsy and 69 healthy controls. Initial selection criteria for the participants with epilepsy included: (a) chronological age between 18 and 63 years, (b) WAIS-III IQ > 69, (c) complex partial seizures of definite or probable temporal lobe origin based on consensus conference review, (d) no MRI abnormalities other than atrophy on clinical interpretation, and (e) no other neurological disorder. Temporal lobe epilepsy was defined by continuous video/EEG monitoring demonstrating temporal lobe seizure onset of spontaneous seizures, clinical semiology in conjunction with interictal EEGs, clinical neuroimaging, and developmental and clinical history. Initial selection criteria for the controls included: (a) chronological age between 18 and 63, (b) WAIS-III Full Scale IQ > 69, (c) either a friend, relative, or spouse of the participant with epilepsy, (d) no current substance abuse, or medical or psychiatric condition that could affect cognitive functioning, and (e) no episode of loss of consciousness greater than 5 min, identified developmental learning disorder, or repetition of a grade in school. This project was reviewed