Cerebellar induced differential polyglot aphasia: A neurolinguistic and fMRI study

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Abstract

Research has shown that linguistic functions in the bilingual brain are subserved by similar neural circuits as in monolinguals, but with extra-activity associated with cognitive and attentional control. Although a role for the right cerebellum in multilingual language processing has recently been acknowledged, a potential role of the left cerebellum remains largely unexplored.

This paper reports the clinical and fMRI findings in a strongly right-handed (late) multilingual patient who developed differential polyglot aphasia, ataxic dysarthria and a selective decrease in executive function due to an ischemic stroke in the left cerebellum. fMRI revealed that lexical-semantic retrieval in the unaffected L1 was predominantly associated with activations in the left cortical areas (left prefrontal area and left postcentral gyrus), while naming in two affected non-native languages recruited a significantly larger bilateral functional network, including the cerebellum. It is hypothesized that the left cerebellar insult resulted in decreased right prefrontal hemisphere functioning due to a loss of cerebellar impulses through the cerebello-cerebral pathways.

1. Introduction

During the past decades a substantial amount of clinical and experimental research has been dedicated to the functional organization of the bilingual brain and the neural networks subserving language processing in bi- or multilinguals in comparison to monolinguals. Findings from these studies have reported that essentially monolinguals and bilinguals process languages in the same neural fashion, but with extra-activity associated with cognitive and attentional control (Abutalebi & Green, 2007, 2016). This extra-activity is usually associated with some specific factors related to second language (L2) processing. Indeed, much of the available literature on the neurobiology of multilingualism indicates that the neural representation and organization of language is the product of a complex process depending on various factors such as age of language acquisition, level of proficiency and level of exposure (Abutalebi, 2008; Perani & Abutalebi, 2005). A more divergent network is associated with late acquisition of the L2 language (Liu & Cao, 2016) and less proficiency (Kotz, 2009). As outlined by Abutalebi and Green (2007), a non-native language which is not processed with the same ease as L1 is less automatized in neurocognitive terms and as such in need of increased cognitive control (i.e., language control). These language control mechanisms allow multilinguals to adequately suppress one language while communicating in another and to flawlessly switch between several target languages.

Converging evidence from clinical and experimental neuroimaging studies shows that the neural system subserving language control and selection processes consists of a widely distributed general cognitive control system mainly involving the bilateral dorsolateral prefrontal areas (specifically the middle and inferior frontal gyri), the anterior cingulate cortex, the bilateral inferior parietal lobules, and subcortical structures such as the basal ganglia, the thalamus, and the cerebellum....
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bellar loop involved in language and executive control (Stoodley & Schmahmann, 2009) in bilingual language processing has been much less explored.

The cerebellum is linked to all the key regions of the language control network and in their adaptive control model (Green & Abutalebi, 2013), Green and Abutalebi (2013) attribute a role in “opportunistic planning” to the cerebellum during multilingual lan-

guage processing. This model attributes a prominent role to the cere-

bellar - left prefrontal connection in using more readily available L1 words/structures to convey meaning in a less proficient language (Green & Abutalebi, 2013).

Functional imaging studies using sentence production and comprehension tasks have to elucidate this view but, as hypothesized (Abutalebi & Green, 2016), it is plausible that cerebellar activation mediates the prediction of future input (L2 processing) based on past knowledge (L1 structures/vocabulary) (Ito, 2008). The ability to make predictions entails maintaining an ongoing representation, which ensures resistance to interference (Abutalebi & Green, 2016). A number of studies have reported changes in cerebellar grey matter density in bilingual speakers correlated to proficiency (bilateral left Crus I/II and right lobule V (Platikas, Johnstone, & Marinis, 2014)); and the density in the right posterior vermis might be of the left posterior vermis (Zou, 2014).

Clinical findings might contribute to our knowledge about the cerebellar role in multilingualism, but bilingual or polyglot aphasia is a diverse and complex phenomenon that is still poorly understood (Paradis & Libben, 2014).

A variety of aphasia symptoms and recovery patterns have been observed in bilingual/multilingual strokes in language-critical regions (Lorenzen & Murray, 2008). Although parallel recovery typically occurs in most of the multilingual cases, a number of non-parallel recovery patterns have been documented in the literature (Fabbro, 2001, Green and Abutalebi (2008) argued that non-parallel recovery in multilingual aphasia is due to disruption of the language control network. One such pattern of non-parallel recovery is involuntary and uncontrolled ‘pathological language mixing and switching’ (Marién, Abutalebi, Engelborghs, & De Deyn, 2005; Kong, Abutalebi, Lam, & Weekes, 2014). Damage to the frontal-subcortical circuit can lead to pathological language switching and mixing, and even to fixation on one single language (Green & Abutalebi, 2008).

Kong et al. (2014) related pathological language mixing and switching to an impairment of executive functions, suggesting a shared fronto-basal ganglia network between the domain-general executive system and language control.

We report the clinical and functional neuroimaging findings in a strongly right-handed multilingual patient who following a left cere-

bellar stroke developed aphasia in each of the six languages he acquired as a late polyglot, while his mother tongue (L1) remained largely unaffected (differential polyglot aphasia). Pathological fixation on one language has been previously reported after subcortical damage (Agioti, Beltramello, Girardi, & Fabbro, 1996; Agioti & Fabbro, 1993), and after damage to the language-dominant temporal lobe (Ku, Lachmann, & Nagler, 1996). After a stroke affecting the left basal ganglia, a 68-year-old right-handed woman developed bilingual aphasia affecting expression in her mother tongue (Venetian) more than in her second language (Italian) while comprehension was preserved in both languages (Agioti & Fabbro, 1993; Agioti et al., 1996). Left temporal lobe damage, on the other hand, resulted in a loss of all expressive and receptive second language skills, leaving his mother tongue fully intact (Ku et al., 1996). In our case, the pathological fixation on his mother tongue was linked to damage to the left cerebellum. A hypothesis is put forward to explain the selective disruption of the non-native languages due to left cerebellar stroke.

2. Case report

2.1. History

A 72-year-old right-handed man was admitted to hospital after acute onset of language disturbances, balance problems, vertigo, and vomiting. On admission, the clinical neurological examination revealed left-sided ataxia with a strong tendency to fall over to the right side. He could stand up straddled. He was not able to understand or express himself in any other but his maternal language (English (L1)) that was unaffected, apart from mild word-finding difficulties for low-frequency words and mild ataxic dysarthria (slurred speech):

“I was watching television at my apartment in Antwerp when sud-

denly the room seemed to spin around violently. I tried to stand but was unable to do so. I felt a need to vomit and managed to crawl to the bathroom to take a plastic bowl. My next instinct was to call the emergency services, but the leafl that I have outlining the services was in Dutch and for some reason, I was unable to think (or speak) in any other language than my native English. I have lived in Antwerp for many years and use Dutch (Flemish) on a day-to-day basis. I called my son-in-law, who speaks fluent English and he drove me to Middelheim Hospital. We normally speak English when together. I understood none of the questions asked of me in Dutch by hospital staff and they had to be translated back to me in English. My speech was slurred. I had lost some words, I was aware of that, but I cannot recall which words. I made no attempt to speak any of the other languages I know, and in the first hours of my mishap happening, I do not think I realized that I had other languages.”

Medical history consisted of arterial hypertension, type 2 diabetes mellitus and a right occipital infarction 10 years before the current stroke. He had an educational level of 12 years (grammar school) and had worked as a war and political correspondent for British, US and Australian newspapers in several countries for more than 40 years. He mastered seven languages: English (maternal language; L1), French (learned at school from age 11 onwards, L2), German (learned at school from age 13 onwards, L3), Slovene (L4) and Serbo-Croat (L5) (learned by means of a crash course at age 24), Hebrew (Ivrit, learned during an intensive course at age 28, L6), and Dutch (moved to live in Belgium from age 35 onwards, L7). He used English (L1), Dutch (L7) and French (L2) on a nearly daily basis. He was in regular contact with friends in Belgrade and Berlin with whom he communicated in Serbo-Croat (L5) and German (L3). He read the Serbian and German press online and followed several forums that talk of the old Yugoslavia, its politics and economics.

T2-weighted axial FLAIR MRI of the brain showed an inhomogeneous hyperintense signal in the territory of the medial branch of the left PICA slightly encroaching upon the posterior portion of the lower medulla at the left (gracile and cuneate nuclei) consistent with a recent infarction in the vascular territory of the left PICA (Fig. 1A–C). An old vascular lesion in the left occipital lobe (Fig. 1D and E) and some periventricular white matter lesions were found as well (Fig. 1F). Diffusion-weighted MRI (axial images) confirmed a hyperintense signal in the territory of the medial branch of left PICA (Fig. 2A–C) with involvement of the medial portion of left dentate nucleus. Based on the

2 All cerebellar anatomy terminology is in accordance with Schmahmann, Doyon, Petrides, Evans, and Toga (2000).
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