Title: Implicit-statistical learning and its relation to aphasia

Highlights

- We tested Implicit-statistical learning in aphasic patients with frontal or posterior lesions
- Implicit-statistical learning is impaired but not completely absent in aphasia
- Lesion location does not predict Implicit-statistical learning deficit in aphasia
- The magnitude of learning effect correlates to the degree of impairment in syntax and phonological working memory

Abstract

Background: Implicit-statistical learning (ISL) research investigates whether domain-independent mechanisms are recruited in the linguistic processes that require manipulation of patterned regularities (*e.g.* syntax). Two sources of evidence, behavioural and neural, recently demonstrated that syntactic processing and ISL mechanisms a) correlate in healthy populations (*e.g.* Daltrozzo, 2017, Misyak & Christiansen, 2010) and b) activate overlapping brain regions in the left frontal lobe (see Udden & Bahlmann, 2012 for review). Persons with aphasia (PWA's) suffer brain damage in regions that are vital to language processing. In support of the domain-dependence of ISL mechanisms, research shows that PWA's with frontal lobe lesions manifest convergent deficits in syntax and ISL mechanisms, but no direct correlation was performed due to small sample sizes (*e.g.* Christiansen, Kelly, Shillcock & Greenfield, 2010; Zimmerer et al., 2014). So far, ISL mechanisms in aphasic patients with temporal or parietal lobe lesions have not been systematically investigated.

Aims: We investigated two complementary hypotheses: 1) the Neural hypothesis, that PWA's with frontal lesions display more severely impaired ISL abilities than PWA's with posterior lesions and 2) the Behavioural hypothesis, that the magnitude of impairment in ISL mechanisms correlates to syntactic, but not lexical deficits in aphasia.

Methods: We tested 14 PWA's, 6 with frontal lesions and 8 with posterior lesions, and 11 controls on a visual statistical learning (VSL) task, which mirrored the traditional boundary-detection task in Saffran, Aslin and Newport (1996).

Results and discussion: We do not find support for the Neural hypothesis as patients with spared frontal regions manifest impaired ISL mechanisms. This is attributed to a) ISL mechanisms being vulnerable to other cognitive dysfunctions and/or b) ISL mechanisms anatomically extending to the posterior brain regions. Notably, ISL mechanisms are impaired, but not absent in aphasia. With regards to the Behavioural hypothesis, we provide the first empirical evidence of correlation between ISL mechanisms and syntactic, but not lexical impairment in aphasia. Unexpectedly, a correlation was also found with non-word repetition task, possibly implicating working memory as instrumental to ISL mechanisms. We discuss both the theoretical contributions to the debate of domain-independence of ISL mechanisms and clinical implications for implicit language therapy.