detailed in vivo examination of brain injuries, which have radically improved lesion-symptom mapping methods. Whilst spatial specificity has improved over history, from large areas of damage to millimetre precision, there is an underlying issue that is rarely addressed in lesion-symptom mapping research, which relates to the fact that damage to a given area of the brain is not random but constrained by the brain vasculature. Standard lesion-symptom mapping does not take this inherent statistical structure of patients' lesions into account. The aim of this study was to uncover this lesion statistical structure and to relate it to the language and cognitive impairments in a group of seventy left hemisphere, post-stroke, chronic aphasic cases. We applied a data reduction method, varimax rotated principal component analysis, to the patients' brain lesion maps. The underlying structure in the lesion maps revealed 20 components of which 17 were interpretable, with most of them reflecting the distribution of middle cerebral artery (MCA) sub-branches. In addition, we extracted the underlying statistical structure from a neuropsychological test battery that consisted of 21 language and cognitive assessments, revealing a four factor behavioural solution, reflecting: phonological ability, semantic ability, executive-demand and speech fluency. We used stepwise regression in order to predict behavioural factors from the principal lesion components and found significant models for all four core abilities (all p's < 0.001). For each model, we projected the beta weights into the brain space. Phonological ability was predicted by two components, which were located in the inferior longitudinal fasciculus, posterior segment of the arcuate fasciculus and inferior frontal gyrus. Three components significantly predicted semantic ability and were located in the anterior temporal lobe extending to the medial temporal lobe, supramarginal gyrus, and angular gyrus. Executive-demand was predicted by two components covering dorsal edges of the MCA, while speech fluency was predicted by two components that were located in the middle frontal gyrus, precentral gyrus, and subcortical regions (putamen and thalamus). The identified lesion territories map very closely with detailed angiography studies and, for the first time, we have linked these to core behavioural deficits.

Meaning: Prosody, Social and Emotional Processes

C35 No Acoustic Evidence from RHD for a Right Hemisphere Role in Prosody Production: A Meta-Analysis

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The right hemisphere (RH) is often thought to have a special role in prosody comprehension and production in general, and affective prosody in particular. If RH structures support prosody wholly or in part, we would expect acoustic differences between the productions of people with right hemisphere damage (RHD) and non brain-damaged (NBD) controls. Such differences have been reported, but the literature is mixed, and spans many years and experimental design types. To get perspective on the scope of these results, we conducted a systematic review and meta-analysis of acoustic measures of prosody production in people with RHD. We searched PubMed, PsychINFO, Web of Science and Google Scholar with the terms: (prosody OR intonation OR inflection OR intensity OR pitch OR fundamental frequency OR speech rate OR voice quality) AND (RHD OR right hemisphere) AND (stroke) AND (acoustic). Of the resulting articles, we selected only empirical studies with an N>2 that quantified acoustic measures of production and included a control group of non brain-damaged (NBD) participants. From the remaining articles, we calculated standardized mean differences (d) for fundamental frequency (F0), Intensity, Speech Duration, Pause Duration, Speech Rate, and Vowel Duration. We also noted whether the task was free speech production or constrained production, and whether the task targeted linguistic or emotional prosody. Our search produced 47 papers. Of these, only 10 met our inclusion criteria, and of these 2 reported from the same study. Mean sample N's were: RHD: 12.15 (SD 10.92), NBD: 14.35 (SD 11). Mean age in years was: RHD: 58.94 (SD 16.7), NBD: 51.76 (SD 14.58). We found no significant impact of RHD on acoustic measures of F0 (d = -0.35, se = 0.29, p = 0.23, N = 9), nor of Intensity (d = -2.98, se = 2.84, p = 0.29, N = 4). No other features had enough papers (N > 3) to warrant a meta-analysis. Of the moderating factors (Task and Prosody Type) only Task had a significant impact (d = -13.08, se = 2.89, p < 0.0001), but this only for intensity, and a single study with a small N (RHD = 8, NBD = 7) and no age-matching drove this surprisingly large effect (Cook's d = 0.9). There was no evidence of publication bias measured by regression analysis of funnel plots for F0 (z = 1.5937, p = 0.1110). Intensity did show evidence of publication bias (z = -4.1582, p < .0001), but this result was driven entirely by a single study out of 4 total. Taken at face value, the literature does not support a special role for the RH in prosody production. However, our study points to a greater problem in the field: studies with acoustic measures of prosody production in RHD are few, and sample sizes too small considering the heterogeneity of the population, to assess even medium effect sizes (power analyses indicate 30-100 participants per group required). We advocate responsible data sharing and standardized automated procedures for the extraction of acoustic features.

Writing and Spelling

C36 Selective involvement of posterior perisylvian regions in sublexical processing: Evidence from brain tumor patients

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