INTRODUCTION

The proper classification of aphasia based on clinical symptoms has been debated for well over a century. Much of the early debates centered on relating localized brain damage to a constellation of speech and language impairments. The premise behind much of this work was based on the notion that lesion-symptom mapping could reveal how language was organized in the brain (Broca, 1861, 1865; Dejarine, 1906; Marie, 1906). Although the principle for classifying aphasia based on specific symptoms has been fervently challenged (e.g. Head, 1926) it is still customary to report aphasia types in clinical studies of aphasia. Similar symptoms in sub-groups of patients suggests a similar pattern of brain damage. Nevertheless, it remains unclear if specific aphasia types can be diagnosed simply based on the location of cortical damage. One way to examine this issue would be to relate lesion patterns to aphasia types using multivariate pattern analysis (MVPA). MVPA of neuroimaging data has been successfully used to diagnose diseases such as dementia, schizophrenia, and Parkinson's disease (Orru et al., 2012). In the present study, we demonstrate how MVPA can be used to predict aphasia type in persons with chronic stroke. Unlike previous studies that perform the analysis on voxels (using MRI scans), we trained a classifier on the proportional damage to brain areas (defined with a brain atlas). In addition, we computed the loadings that reflect the contribution of each brain area to classification.

METHODS

We recorded structural MRI scans for 27 persons with left-hemisphere lesions. Lesion size and extent were normalized with the Clinical Toolbox software (Rorden et al., 2012). Each person's lesion was segmented into regions as defined by (a) Brodmann atlas (www.mricro.com) and (b) by John Hopkins University (JHU) atlas (Faria et al., 2011). We computed the proportional damage for every brain region. The diagnosis for each patient was accomplished using the Western Aphasia Battery (WAB; Kertesz, 1982); 10 individuals were classified as having Broca's aphasia, 9 persons had anomic aphasia, and 8 tested within normal limits. We predicted the aphasia type from the proportional damage using support vector machines with linear kernels (LIBSVM toolbox; Chang & Lin, 2011) for 3 pairings of aphasia types (Broca's/none, anomic/none; Broca's/anomic). At each iteration of the cross-validation procedure, two subjects (one from each aphasia type) were held out, and their aphasia type was determined after training on the remaining subjects. This process was repeated for all possible pairings of subjects. Relative contribution of each area was estimated from the weights of the linear decision boundary (LaConte et al., 2005).

RESULTS

Aphasia type was predicted above chance for all 3 binary contrasts. When using Brodmann atlas, the prediction accuracy was 93.8% (Broca's/none), 75% (anomic/none), and 67.9% (Broca's/anomic). When using JHU atlas, the corresponding prediction accuracy was 93.8%, 68.8%, and 88.3%. Figures 1 through 3 show the relative contribution of the Brodmann areas of the left hemisphere for each of the three contrasts. The areas with the largest contribution to classification are in the left temporal lobe (superior and middle temporal gyri; hippocampus/parahippocampus; primary auditory areas) as well as pre/postcentral gyri. Broca's area was also important for distinguishing Broca's aphasia from the other two types, but not for the anomic/none contrast. In the JHU atlas, the caudate nucleus and putamen were particularly important of the Broca's/anomic classification.

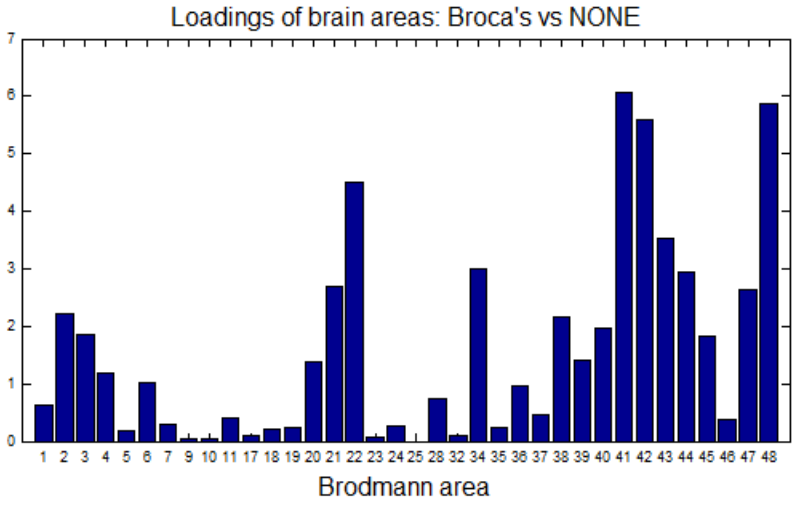
CONCLUSIONS

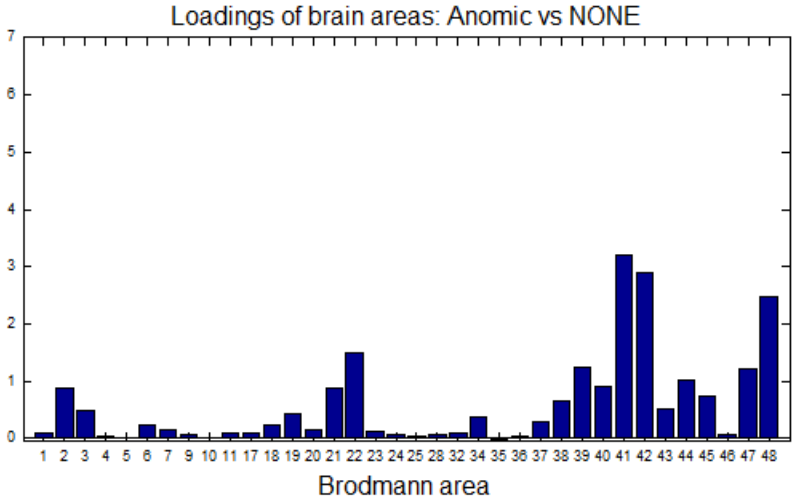
We successfully predicted the aphasia type in persons with left-hemisphere lesions. We used a combination of our lesion-mapping toolbox and a support vector machine to predict the aphasia type. Damage to the areas in left temporal lobe, pre/postcentralgyri, and Broca's area was shown to be highly relevant for accurate prediction of aphasia type.

Successful prediction of aphasia type based on lesion location suggests that patterns of brain damage are similar across patients, even among those who have anomic aphasia. Traditionally, lesion patterns in persons with anomic aphasia have been thought to be quite variable. The results reported here suggest that the damage that causes anomic aphasia is sufficiently similar across cases to allow for almost 90% classification of anomic versus Broca's aphasia. Although the successful classification of aphasia based on cortical damage may have theoretical implications, it probably has very limited value for clinical practice. Aphasia types and specific clinical symptoms are sufficiently defined based on behavioural testing and neuroimaging data add little, if any, value for characterization of clinical profiles. In contrast, neuroimaging may provide value for predicting long-term outcome in acute patients. If aphasia type can be classified based on lesion patterns, then acute MRI scans may be of potential use for making prognosis. We believe that diagnosis (i.e. classifying aphasia type) is a precursor to prognosis and that the work reported here sets a baseline for predicting long-term outcome in aphasia.

We are working on replicating the findings reported here in a larger dataset (N=86). If the current study is accepted for presentation, the results from the larger sample will also be reported. The advantage of this larger dataset is that it contains cases with all of the major aphasia types (including Wernicke's, conduction, and global aphasia).

FIGURES


Loadings of brain areas: Broca's vs NONE
0 1 2 3 4 5 6 7
1 2 3 4 5 6 7 9 10 11 17 18 19 20 21 22 23 24 25 28 32 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48
Brodmann area


Loadings of brain areas: Anomic vs NONE
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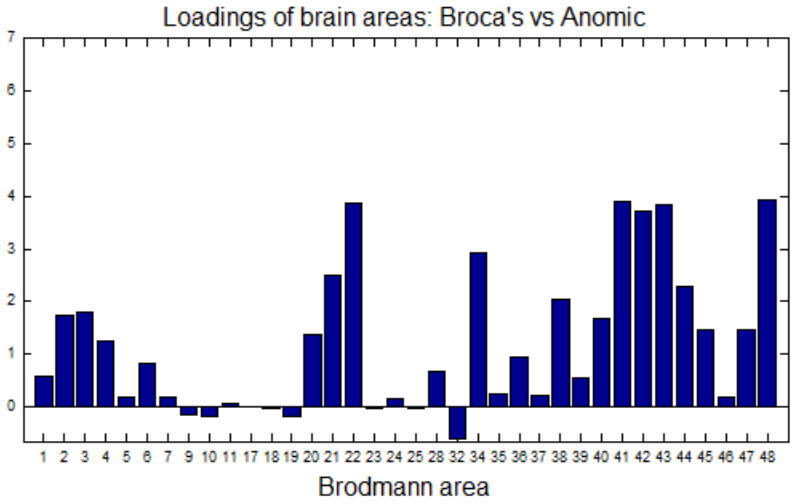
1 2 3 4 5 6 7 9 10 11 17 18 19 20 21 22 23 24 25 28 32 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48
Brodmann area


Loadings of brain areas: Broca's vs Anomic
0 1 2 3 4 5 6 7
1 2 3 4 5 6 7 9 10 11 17 18 19 20 21 22 23 24 25 28 32 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48
Brodmann area

REFERENCES

Broca, P. (1861). Remarques Sur le Siége de la Faculté Du Langage Articulé, Suivies D'une Observation D'aphémie (Perte de la Parole). *Bulletin Society Anatomique. 6*, 330–357.

Broca, P. (1865). Sur le siège de la facultlé du language articulé. *Bull Soc Anthropol., 6*, 337–393.

Chang, C.-C., Lin, C.-J. (2011). LIBSVM : a library for support vector machines. *ACM Transactions on Intelligent Systems and Technology, 2*(3), Article # 27.

Déjerine J. (1906). L'aphasie motrice: sa localisation et sa physiologie pathologique. *Presse Med, 57*, 453–457.

Faria AV, Joel SE, Zhang Y, Oishi K, van Zjil PC, Miller MI, Pekar JJ, Mori S. (2012) Atlas-based analysis of resting-state functional connectivity: evaluation for reproducibility and multi-modal anatomy-function correlation studies. *Neuroimage. 61*(3), 613-21

LaConte, S., Strother, S., Cherkassky, V., Anderson, J., Hu, X. (2005). Support vector machines for temporal classification of block design fMRI data. NeuroImage, 26, 317-329.

Marie, P. (1906). The third frontal convolution plays no special role in the function of language. *Seminale Medicale*, 26, 241-247.

Orru, G., Pettersson-Yeo, W., Marquand, A. F., Sartori, G., Mechelli, A. (2012). Using support vector machine to identify imaging biomarkers of neurological and psychiatric disease: a critical review. *Neuroscience & Biobehavioral Reviews, 36*(4), 1140-1152.

Rorden, C., Bonilha, L., Fridriksson, J., Bender, B., Karnath, H. O. (2012).Age-specific CT and MRI templates for spatial normalization. *NeuroImage, 61*(4), 957-965.