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White Matter Tracts Critical for Recognition of Sarcasm

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Abstract

Sarcasm is commonly used to express criticism in a non-aggressive or humorous way. Failure to understand that the speaker is being sarcastic can lead to important miscommunications. Although numerous studies have identified impaired sarcasm comprehension in neurological impaired patients, few have attempted to identify lesions that lead to impaired sarcasm. Several gray matter structures seem to be critical for processing sarcasm, including right prefrontal cortex, superior temporal cortex, thalamus, and basal ganglia. In this study we tested the hypothesis that percent damage to specific white matter tracts (including those connect the critical gray matter structures), age, and education together predict accuracy in sarcasm comprehension. Using multivariable linear regression, with age, education, and percent damage to each of 8 white matter tracts as independent variables, and percent accuracy on sarcasm recognition as the dependent variable, we developed a model for predicting sarcasm recognition. Percent damage to the sagittal stratum had the greatest weight, and was the only independent predictor of error rate on sarcasm. Results indicate that: (1) sagittal stratum has an important role in the network underlying sarcasm comprehension; (2) sagittal stratum lesions are likely to cause deficits in understanding sarcasm and may require innovative therapies to address this disability.

Keywords

Sarcasm; Stroke; Cognitive Empathy; Sagittal Stratum; Attitudinal Prosody

Introduction

Sarcasm is used in everyday communication, often to express criticism or annoyance but in a relatively non-aggressive or humorous way (Dews & Winner, 1995; McDonald, 1999). Understanding that another person is being sarcastic, rather than sincere, from the spoken stimulus requires a number of distinct phonological and cognitive processes. Suppose one hears the sentence, “That is a pretty dress,” spoken in a sarcastic tone of voice. First, one has to understand the literal meaning of the sentence by processing the syntax and semantics of the stimulus. Then one has to detect the emotional prosody – the variation in pitch, stress, loudness, duration – of the components of auditory stimulus that makes it a sarcastic tone of voice. These prosodic features that are recognized as sarcastic include a wider range in the pitch or fundamental voice frequency, greater emphatic stress, briefer pauses, and lengthening of syllables or intensified loudness, relative to sincere speech (Anolli, Ciceri, & Infantino, 2000; Blanc & Dominey, 2003; Gelinas-Chebat & Chebat, 1992; Rockwell, 2007). Having recognized the sarcastic tone of voice, one can reject the literal meaning of the sentence and consider alternative meanings or intents of the speaker. Making an inference about what another person thinks or believes (e.g. about the dress) is sometimes referred to as cognitive empathy. Additionally, one often makes an inference about how the person feels (e.g. annoyed) – an act of affective empathy. Thus, the apparently “simple” task of recognizing whether or not a sentence is sarcastic or sincere (which neurologically healthy individuals do without really thinking about it) may depend on a complex set of phonological and cognitive, and thus neural, mechanisms.

Often there are visual cues, such as facial expressions or gesture that augment the tone of voice in conveying sarcasm (Williams, Burns, & Harmon, 2009). Visual paralinguistic cues that a person is being sarcastic include either an increase or decrease in the range and intensity of facial expressions; widened or rolling eyes; rapid blinking, grimaces or smirks (Attardo, Eisterhold, Hay, & Poggi, 2003; Rockwell, 2001). However, modulations in the tone of voice, particularly changes in the pitch contour, convey stronger cues than visual or contextual cues about the paradoxical meaning intended to be conveyed by the speaker, relative to the content of the spoken utterance (Rockwell, 2007). Both prosodic and visual cues are used by the speaker to convey to the listener that the message is not sincere. The speaker *wants* the listener to understand the irony of the message. Contextual cues that are inconsistent with the content of the speaker’s utterance can also facilitate recognition of sarcasm (Harada et al., 2009; Shany-Ur et al., 2012), although recognition of sarcasm through contextual cues is developmentally later and more complex than recognizing sarcasm through prosody (Laval & Bert-Eboul, 2005). For example, a dress that is clearly not pretty may help the listener understand the sarcasm in our example. Comprehension via contextual cues may require generation of multiple alternative meanings of the sentence, and then selection of the meaning most appropriate for the context (Thomas, 1995).

Failure to understand sarcasm (either from paralinguistic cues or contextual cues) can lead to important miscommunications. Individuals who misunderstand a sarcastic sentence as sincere will interpret the speaker’s meaning as the opposite to what was intended. This failure of communication is not uncommon after right hemisphere stroke, traumatic brain injury (TBI), schizophrenia, autism, and dementia (Bara, Bucciarelli, & Geminiani, 2000;

Champagne, Virbel, Nespoulos, & Joannette, 2003; Channon, Pellieff, & Rule, 2005; Channon et al., 2007; Dennis, Purvis, Barnes, Wilkinson, & Winner, 2001; Kipps, Nestor, Acosta-Cabronero, Arnold, & Hodges, 2009; Leitman, Ziwich, Pastenak, & Javitt, 2006; McDonald, 2006; Martin & McDonald, 2004; Rajendran, Mitchell, & Rickards, 2005; Shamay-Tsoory, Tomer, Berger, Goldsher, & Aharon-Peretz, 2005; Pell, 2006a; Pell, 2006b; Pell, 2007). However, most studies have failed to identify the specific cognitive processes that are impaired in association with particular lesions. Many have mixed visual and auditory paralinguistic cues, and/or contextual cues, so that it is difficult to assess the relative impairment in processing each type of cue.

Sarcasm is one type of intellectual or attitudinal prosody – prosody that conveys information about the speaker's attitude about the content of the utterance, such as enthusiasm, skepticism, doubt, fervor (Monrad-Kohn, 1963). Attitudinal prosody can be distinguished from intrinsic or linguistic prosody (e.g. questions versus statements), emotional prosody (e.g. anger versus sadness), and inarticulate prosody (e.g. moans, sighs) (Boss, 1996). Despite the recognition that brain lesions can lead to impaired recognition of attitudinal prosody, there have not been many attempts to identify the neural mechanism underlying comprehension of attitudinal prosody. Mitchell and Ross (2013) reviewed the available literature on the neuroanatomy of attitudinal prosody, including recognition of sarcasm. On the basis of evidence from functional imaging and lesion studies primarily of emotional prosody, they concluded that comprehension of attitudinal prosody may be mediated primarily by right posterior superior lateral temporal cortex, with contributions from right basal ganglia, thalamus, and limbic regions including medial prefrontal cortex and amygdala. However, they found few studies that directly evaluated the neuroanatomy of attitudinal prosody. The few studies that have directly evaluated attitudinal prosody have obtained results consistent with these conclusions. For example, Eviator and Just (2006) carried out an fMRI study of healthy controls listening to discourse, followed by literal, metaphoric, or ironic (sarcastic) statements. Compared to literal or metaphoric utterances, comprehension of ironic utterances was associated with activation in right middle and superior temporal cortex. A role of right superior temporal cortex in sarcasm comprehension is plausible as posterior superior lateral temporal cortex is critical for phonological processing, and the right posterior lateral temporal cortex has been implicated in processing paralinguistic information (Van Lancker & Sidtis, 1992) and affective prosody (Heilman, Scholes, & Watson, 1975; Tompkins & Mateer, 1985; Ross & Monnot, 2008; Dara, Gottesman, & Hillis, 2012). Pride et al. (2014) also found that adults with neurofibromatosis type I were impaired in understanding sarcasm, and this impairment was associated with decreased gray matter volume within right superior temporal cortex. Shamay-Tsoory et al. (2005) studied 30 patients with traumatic brain injury, 7 patients with tumors, and 4 with stroke, divided into those with right, left, or bilateral prefrontal or posterior lesions, on sarcasm, emotional prosody, and other related tasks. They found that patients with right prefrontal cortex lesions were most impaired in recognition of sarcasm, relative to healthy controls or patients with left prefrontal lesions, or left or right posterior lesions. However, traumatic brain injury is not an entirely focal injury. Likewise, Channon et al. (2007) studied 43 patients with tumors (mostly glioma, mostly bilateral), 1 with abscess, and 1 with stroke, including 23 with frontal lesions and 22 with posterior lesions, and found that frontal

lesions, but not posterior lesions, were associated with impaired comprehension of sarcasm and other “mentalizing” tasks. Rankin et al. (2009) also found, in a voxel-based morphometry study of individuals with neurodegenerative disease, that poorer sarcasm comprehension was associated with reduced volume in bilateral posterior parahippocampal gyri, temporal poles, and right medial frontal pole. Only individuals with semantic dementia (a form of frontotemporal lobar degeneration) in that study were significantly impaired in comprehension of simple sarcasm relative to controls. Furthermore, several areas identified in these studies and by Mitchell and Ross (2013) - right prefrontal cortex, medial frontal pole/orbitofrontal cortex, temporal pole, and amygdala - are important components of the neural network underlying empathy, and damage to these limbic areas results in impaired empathy (Eslinger, 1998; Shamay-Tsoory, Tomer, Berger, & Aharon-Peretz, 2003; Shamay-Tsoory, Tomer, Goldsher, Berger, & Aharon-Peretz, 2004; Shamay-Tsoory, Aharon-Peretz & Perry, 2009; Rankin, Kramer, & Miller, 2005; Rankin et al., 2006; Hurlemann et al., 2010; Hooker, Bruce, Lincoln, Fisher, & Vinogradov, 2011; Leigh et al., 2013; Brunnleib, Munte, Tempelmann, & Heldmann, 2013; Hillis, 2014), along with right anterior cingulate cortex, anterior insula, and thalamus; Leigh et al., 2013; see Hillis, 2014 for review).

Shany-Ur et al. (2012) found that individuals with behavioral variant frontotemporal dementia (another form of frontotemporal lobar degeneration associated with prominent atrophy in right frontal, insular, and temporal cortex) are impaired in all aspects of comprehension of sarcasm and lies (insincere statements), including prosody recognition, perspective taking, and emotional reading of the speaker. They were not impaired in comprehending sincere statements relative to controls. Pell (2006b; 2007) also directly studied attitudinal prosody in chronic right hemisphere stroke patients, and found them to be impaired, relative to controls; but did not investigate specific lesion-deficit associations.

Like individuals with frontotemporal lobar degeneration, individuals with right hemisphere stroke or other focal lesions often have impairments in recognition of affective prosody (Dara et al., 2012; Ross & Monnot, 2008), facial expression (Blonder, Bowers, & Heilman, 1991; Tippet, Davis, Gomez, Trupe, & Hillis, 2014); integration of contextual cues and generation of multiple meanings (Beeman, 1993; Tompkins & Mateer, 1985; Weylman, Brownell, Roman, & Gardner, 1989), and affective empathy, including perspective-taking (Shamay-Tsoory et al., 2003; 2004; 2005; Leigh et al. 2013; Hillis, 2014). Therefore, it is not surprising that they are also impaired in recognition of sarcasm (Mitchell & Ross 2013). However, in studying acute right hemisphere stroke patients, we have been unable to identify specific associations between cortical lesions and impaired sarcasm. One reason that it may be difficult to confirm the association between acute deficits in sarcasm and acute lesions in anterior temporal pole, frontal pole, and parahippocampal gyrus is that these areas are not frequently affected by stroke. Therefore, there may be low power to identify independent effects of these areas. Likewise, these areas are frequently atrophied in frontotemporal lobar degeneration (FTLD); there may be other areas, not frequently affected in FTLD, that are also important for sarcasm, but undetected by voxel-based morphometry studies of FTLD due to low variation in volume of tissue in these areas.

Functional imaging studies of healthy controls complement lesion studies by identifying all the areas that are engaged during a task, such as processing sarcastic versus sincere

statements. In a coordinate based meta-analysis using data from 354 participants in studies of comprehension of non-literal language (including sarcasm, but also metaphor, idioms, and so on), Bohrn, Altmann, and Jacobs (2012) found peak activations in bilateral inferior frontal cortex, bilateral medial frontal cortex, and large parts of left temporal cortex, as well as a region near left amygdala, associated with processing all types of figurative language (including sarcasm) across studies. However, as noted, fMRI studies that contrast comprehension of sarcasm with comprehension of metaphor and other figurative language identify activation in right superior and middle temporal cortex specifically associated with sarcasm. (Eviator & Just, 2006) or amygdala (Uchuyama et al. 2012).

The gray matter areas that have been identified as important to comprehension of sarcasm presumably operate as a network. On this hypothesis, damage to the white matter tracts that connect these components of the network should also disrupt comprehension of prosody. The sagittal stratum is a large bundle of white matter fibers that provides widespread connections from the temporal and frontal cortex to subcortical structures and brainstem. While there are additional white matter tracts that connect individual areas that have been implicated in prosody, we predicted that damage to the sagittal stratum would cause the most severe impairment in sarcasm, given that sarcasm must rely on an interaction between phonological processing in lateral temporal cortex and areas critical for cognitive perspective-taking, such as medial prefrontal cortex. We tested this hypothesis in 24 patients with acute right hemisphere ischemic stroke, who were evaluated on sarcasm recognition on the basis of prosodic cues alone and evaluated for degree of damage to white matter tracts within 24 hours of admission to the hospital for acute ischemic right hemisphere stroke. This approach allowed us to evaluate the relationship between the deficit in recognition of sarcastic prosody and damage to white matter tracts before the opportunity for substantial reorganization of structure-function relationships or recovery.

Methods and Materials

Participants

We evaluated data from a consecutive series of 24 patients who had met the following criteria for inclusion: (1) acute ischemic stroke in the right hemisphere; (2) able and willing to provide informed consent to participate in the study. Patients additionally had none of the following criteria for exclusion: (1) inability to have MRI due to pregnancy, implanted ferrous metal, claustrophobia, or weight >300 pounds; (2) reduced level of consciousness, delirium, or on-going sedation; (3) neurological disease other than stroke. The research was conducted in accordance with the Declaration of Helsinki.

Imaging

The MRI protocol included the following sequences: diffusion weighted imaging (DWI) and Apparent Diffusion Coefficient (ADC) to evaluate for acute infarct, fluid-attenuated inversion recovery (FLAIR) to evaluate for old stroke, T2 weighted imaging to evaluate for edema, Susceptibility Weighted Imaging (SWI) to evaluate for hemorrhage, MPRAGE for registration, and 3D time-of-flight angiography of the intracranial vessels. We acquired all sequences using single-shot spin-echo echo-planar imaging, in the transverse plane, with

whole brain coverage. DWI was obtained with a b-value of 1000 (s/mm²) and a least diffusion weighting (b0).

Image analysis

The boundary(s) of acute stroke lesion(s) was defined by a threshold of > 30% intensity increase from the unaffected area in the DWI. Then, a neurologist (KO), who as masked to the behavioral testing, manually modified the boundary of the lesion to avoid false-positive and false-negative areas, using RoiEditor (www.MRIstudio.org) [Oishi *et al.* 2009]. Then the least DWI (b0) with T₂-weighted contrast was transformed to the JHU-MNI-b0 atlas using affine transformation, followed by large deformation diffeomorphic metric mapping [LDDMM; Oishi, *et al.* 2009]. The resultant matrices were applied to the stroke lesion for normalization. A customized version of the JHU-MNI Brain Parcellation Map (cmrm.med.jhmi.edu) was then overlaid on the normalized lesion map to determine the percentage volume of each of the white matter tracts (stria terminalis; fornix; right inferior frontooccipital fasciculus; sagittal stratum; posterior thalamic radiation; superior frontooccipital fasciculus; superior longitudinal fasciculus; uncinate fasciculus) that was affected by the acute stroke in each individual (Figure 1), using DiffeoMap (www.MRIstudio.org).

Sarcasm Testing

Patients underwent testing of attitudinal prosody within 24 hours of admission to the hospital, using stimuli from the Aprosodia Battery (Ross & Monnot, 2008; see also Mitchell & Ross, 2013). Briefly, stimuli consisted of 40 neutral sentences (e.g. “*This looks like a safe boat*”) spoken in either a sincere tone of voice or a sarcastic tone of voice. Patients or controls were asked to state whether the person was being sarcastic or sincere.

Statistical Analysis

We used multivariate linear regression analysis to identify the independent predictors of severity of sarcasm recognition impairment (error rate in detecting sarcasm). The following independent variables were entered as potential predictors: percentage of damage to right stria terminalis, fornix; right inferior frontooccipital fasciculus; sagittal stratum; posterior thalamic radiation; superior frontooccipital fasciculus; superior longitudinal fasciculus; uncinate fasciculus; age; and education. Other white matter tracts, such as body, genu, and splenium of corpus callosum were excluded because fewer than four patients had damage to those tracts.

We then evaluated differences between stroke patients with lesions in the sagittal stratum and patients without lesions in the sagittal stratum, with regard to error rate in sarcasm, error rate in recognition of sincerity, error rate in total attitudinal prosody (recognition of sarcasm plus recognition of sincere prosody), percent correct perspective-taking (see Leigh *et al.* 2013), age, and education using unpaired t-tests.

Results

The prediction model contained all of the nine predictors except posterior thalamic radiation. The model was statistically significant, $F(9, 12) = 2.95$, $p = .042$, and accounted for approximately 46% of the variance in sarcasm recognition error rate ($r^2 = .689$, adjusted $r^2 = .456$). Error rate in recognition of sarcasm was primarily predicated by degree of damage to the sagittal stratum. The raw and standardized regression coefficients of the predictors and with their correlations with sarcasm recognition error rate are shown in Table 1. The degree of damage to the sagittal stratum received the strongest weight in the model. No other predictors, other than sagittal stratum, were independently and significantly associated with sarcasm error rate. The standardized coefficients for each of the variables are shown in Table 1.

In addition, we compared patients with and without damage to the sagittal stratum in terms of accuracy rate in recognition of sarcasm, sincerity, total attitudinal prosody (sarcasm + sincerity), total affective prosody comprehension (in which they were required to point to the label of the emotion corresponding to emotions conveyed in neutral sentences, monosyllables, or asyllabic utterances on the basis of prosodic cues), and a test of affective empathy. As shown in Table 2, patients with right sagittal stratum lesions were significantly different from those without lesions in the sagittal stratum only on sarcasm recognition (21% vs. 50% correct; $t=2.8$; $p=.021$) and total attitudinal prosody (57% vs. 67% correct; $t=2.4$; $p=0.035$).

Discussion

We found that some participants with acute right hemisphere stroke, like individuals with frontotemporal lobar degeneration, are significantly impaired in recognizing sarcastic tone of voice. Our results complement those of previous authors. We have not only confirmed that right hemisphere lesions can disrupt recognition of attitudinal prosody, as reported by Pell (2006b; 2007), Shamay-Tsoory et al. (2005), and others, but have also shown that disruption of specific white matter tracts correlates with impaired sarcasm recognition. The degree of damage to right sagittal stratum was the primary determinant of accuracy in sarcasm detection. Accuracy in recognition of sarcasm was inversely correlated with the degree of disruption of the sagittal stratum, after controlling for age, education, and damage to other white matter tracts.

The sagittal stratum is a large bundle of white matter fibers that serve to connect frontal, cingulate, temporal, parietal, and occipital cortical regions to thalamus and other deep structures. It carries both afferent and efferent fibers between thalamus and cortex. Given that thalamus (particularly the medial and lateral geniculate nuclei) is essential for processing both auditory and visual information that would be crucial for detecting prosody and facial expression to cortical regions, it makes sense that damage to this white matter tract would disrupt recognition of sarcasm in the absence of contextual cues.

We are not aware of other studies that have identified lesions in white matter tracts associated with impairments in recognition of attitudinal prosody. However, Mitchell and

Ross (2013) identified impaired affective prosody after lesions of the right thalamus. We also identified impaired affective empathy in some individuals with right thalamic strokes. It is plausible that lesions in specific nuclei of the thalamus (e.g. medial geniculate nucleus) and their connections at least acutely disrupt processing of auditory cues that would normally allow one to detect insincerity in tone of voice.

Several other factors contributed to the model for predicting accuracy of sarcasm recognition, although no other individual factor was independently and significantly associated with performance. Age was a negative predictor, while education was a positive predictor. That is, younger age and higher education contributed to accuracy in sarcasm recognition along with disruption of specific white matter tracts (see Table 1), but were not significant predictors after controlling for other variables.

The clinical implications of our findings are straightforward. Individuals with deep right hemisphere stroke, particularly involving sagittal stratum, should be evaluated for recognition of sarcasm. It is possible that those with impairments in this important form of communication can be helped to regain the ability to recognize prosodic cues. Alternatively, family and friends can be counseled to avoid sarcasm to prevent misunderstandings.

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Figure 1. A representative diffusion weighted image in this study with an acute infarction in the sagittal stratum

Infarction is indicated by transparent red. Eight white matter structures analyzed in this study are shown with colored contours.

st: stria terminalis; fx: fornix; ifo: inferior fronto-occipital fasciculus; ss: sagittal stratum; ptr: posterior thalamic radiation; sfo: superior fronto-occipital fasciculus; slf: superior longitudinal fasciculus; and unc: uncinate.

Table 1
The Multivariable Model Associated with Percent Accuracy in Recognition of Sarcasm

Variable	Unstandardized Coefficients		Standardized Coefficients	t	Significance
	Beta	Standard Error			
Constant	0.282	0.374		.754	0.465
Age	−0.002	0.003	−0.115	−.576	0.575
Education	0.024	0.018	0.256	1.356	0.200
Stria Terminalis	−2.333	18.028	−0.228	−.129	0.899
Fornix	18.470	58.673	0.329	.315	0.758
Inferior Frontooctipital Fasciculus	−0.796	0.673	−1.059	−1.183	0.260
Sagittal Striatum	−5.768	2.492	−1.081	−2.315	0.039*
Superior Frontooctipital Fasciculus	.459	0.332	0.702	1.381	0.192
Superior Longitudinal Fasciculus	0.090	0.464	0.089	.194	0.850
Uncinate	0.550	0.619	0.713	.895	0.388

* significant at an alpha level of $p < 0.05$

Table 2

Differences Between Participants with and without Disruptions of the Sagittal Striatum

Variable	Participants with Sagittal Striatum Lesions	Participants without Sagittal Striatum Lesions	t	p-value
Age	53.9 years	54.9 years	−24.0	0.813
Education	14.4 years	14.1 years	.324	0.751
Attitudinal Prosody % Correct	57.1% correct	67.4% correct	−2.38	0.035*
Sincere % Correct	84.2% correct	84.4% correct	−.032	0.976
Sarcastic % Correct	21.7% correct	50.3% correct	−2.81	0.021*
Empathy % Correct	68.4% correct	77.7% correct	−1.25	0.252
Total Prosody Comprehension % Correct	51.5% correct	50.1% correct	.188	0.854

* significant at an alpha level of $p < 0.05$