

Affective aprosodia from a medial frontal stroke[☆]

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Accepted 13 January 2004

Abstract

Background and objectives. Whereas injury to the left hemisphere induces aphasia, injury to the right hemisphere's perisylvian region induces an impairment of emotional speech prosody (affective aprosodia). Left-sided medial frontal lesions are associated with reduced verbal fluency with relatively intact comprehension and repetition (transcortical motor aphasia), but persistent affective prosodic defects associated with right medial frontal lesions have not been described. **Methods.** We assessed the prosody of a man who sustained a right medial frontal cerebral infarction seven years prior. **Results.** While propositional speech expression was normal including syntactic prosody, the patient was impaired at expressing emotions using prosody. His comprehension and repetition of prosody were also impaired but less so than expression. **Conclusions.** Right medial frontal lesions can induce an affective aprosodia that primarily impairs expression.

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1. Introduction

“It is not what you said, but how you said it.” Normally, speech may simultaneously transmit two types of messages, verbal and prosodic. Whereas, the verbal message is conveyed by a complex code requiring semantic, lexical, syntactic, and phonemic processing, the prosodic message is conveyed by changes in pitch, amplitude, tempo, and rhythm. In European languages, such as English, prosody can be used linguistically, primarily at the syllable and word levels to mark stress and can be used to signal whether an utterance is statement or question (i.e., declarative versus interrogative). Prosody, at the sentence level and involving principally changes in fundamental frequency, can also be used to convey emotional messages (Monrad-Krohn, 1947; Paul, 1909; Pell, 1999).

Broca (1865) noted that in more than 90% of people, the left hemisphere is dominant in mediating what Hughlings Jackson (1915) later called propositional

speech. Hughlings Jackson (1915), however, observed that even non-fluent aphasic patients from left hemisphere lesions could imbue their simple utterances with emotional content by using affective intonations and suggested that the right hemisphere might be important in mediating affective prosody. This postulate, however, was not investigated until about 25 years ago when Heilman, Scholes, and Watson, 1975 and Tucker, Watson, and Heilman, 1977 reported patients with right hemisphere lesions who were not aphasic, but who could not comprehend (Heilman et al., 1975) or express (Tucker et al., 1977) emotions using affective prosody. Subsequently, Ross and Mesulam (1979) reported two patients who could not express affective prosody but could comprehend affective prosody. Ross (1981) then suggested that these affective prosodic deficits be called aprosodia. He suggested that the types of affective prosody disturbances seen with right hemisphere damage might mirror the types of aphasia syndromes observed with left hemisphere damage and described some patients with right posterior perisylvian lesions who demonstrated affective prosody comprehension disorders and other patients with right anterior perisylvian lesions who were impaired at expressing affective prosody but had spared comprehension of affective prosody. Other studies, however, have suggested that not all

[☆] This work was supported by NIDCD P50-DC03888, the Department of Veteran Affairs and the State of Florida Memory Disorder Clinics.

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patients fit into this localization paradigm (Wertz, Henschel, Auther, Ashford, & Kirshner, 1998).

Lichtheim (1885) reported a patient who had non-fluent speech, but who was able to comprehend and repeat speech. Subsequently, there have been other reports of similar patients. Although some of these patients have left dorsolateral frontal convexity lesions that are above Broca's area (frontal-opercular region), there have been multiple reports of this syndrome being induced by strokes in the distribution of the left anterior cerebral artery and in these reports the critical area injured appeared to be the supplementary motor area (Bogousslavsky, Assal, & Regli, 1987; Freedman, Alexander, & Naeser, 1984; Pai, 1999; Masdeu, Schoene, & Funkenstein, 1978; Rubens, 1975; Ziegler, Kilian, & Deger, 1997). Whereas Bell, Davis, Morgan-Fisher, and Ross (1990) reported a child who had transiently impaired expressive prosody from a right frontal lesion, we could find no reports of adults with right medial frontal damage who demonstrated persistent aprosodia and thus the purpose of this report is to present such a case.

2. Methods

2.1. Subject

On September 25, this retired railroad machinist, who was at that time 76 years old, was admitted to the Veterans Affairs Medical Center, Gainesville, Florida, for evaluation of a stroke. On the day of admission, while sitting and watching television, he had the onset of a transient loss of speech followed by slurring of speech. When he attempted to stand, he could not and fell over, but had no loss of consciousness. The patient had no history of a prior stroke, but did have a history of coronary artery disease, including two myocardial infarctions, stable angina with ischemic cardiomyopathy associated with a reduced ejection fraction, and mitral regurgitation. He also had a history of polycythemia. His medications included digoxin, lisinopril, metoprolol, furosemide, aspirin, niacin, cimetidine, allopurinol, isosorbide dinitrate, and nitroglycerine.

A general examination during admission revealed a fourth heart sound, as well as a grade 2/6 systolic murmur at the left sternal border. Although his admission neurological examination was reported to be normal, the patient was later noted by the neurology consult to have a mild left hemiparesis that was maximal in his left shoulder with a left-sided extensor plantar response. The neurology consult also noted that the patient had slurred speech, an abnormal gait, a left-sided visual grasp, and decreased saccades to the left. There was some question as to whether he had a left hemianopia and possibly some mild left-sided inattention or neglect.

The patient had an MRI taken 2 days after his admission (see Fig. 1), which revealed a lesion of the right supplementary motor area (Brodmann's area 6) and the anterior cingulate gyrus (Brodmann's area 24). The forceps minor with its input to the genu of the corpus callosum was also probably involved. An echocardiogram revealed an ejection fraction of 20% and a Holter monitor revealed an episode of ventricular tachycardia. Because the physicians taking care of him thought that he was at risk for another embolus due to his poor ejection fraction and intermittent arrhythmia, he was anticoagulated with warfarin and treated with amiodarone.

Shortly after we opened a new rehabilitation center, this patient was seen as a possible rehabilitation candidate (January 2002). Since being discharged from the hospital in 1994, his dysarthria and left arm weakness almost completely resolved. A neurological examination at that time revealed no evidence of hemispatial neglect. He had a very mild left facial droop and decreased sensation in his feet in a stocking-like pattern. His strength was normal except for the finger extensors of his left hand. He also had difficulty making fine finger movements with his left hand. He walked slowly and turned en block; otherwise, this 83 year old man had a normal neurological examination. He performed normally on the Geriatric Depression Scale (Yesavage et al., 1982–83), but had constructional apraxia as determined by the Rey–Osterrieth Complex Figure task (Osterrieth, 1944). He scored 23 on the Mini-Mental State Examination (Folstein, Folstein, & McHugh, 1975), but right hemisphere lesions can induce constructional apraxia (drawing the intersecting pentagons) and frontal lobe lesions can reduce working memory (spell “world” backwards). Our patient performed incorrectly on both of these tests. If these errors were excluded his score would be in the normal range (27). While the patient did not demonstrate florid left-sided neglect, he did make an error when copying the left, but not right pentagon and when spelling “world” backwards he misplaced the letter “o” which is on the left side of the word.

The wife of this patient told one of the authors (S.L.) that after this patient's stroke in '93 his voice was quieter, and “that everything sounded the same no matter how he was feeling.” To learn if this man with a right hemisphere stroke had affective aprosodia, he underwent a series of prosody tests (Table 1). The Florida Emotion Expressive Battery (unpublished, University of Florida) has subjects perform a series of tests; the first three assess peoples' ability to imitate syntactic and emotional prosody, and the other three to produce syntactic and emotional prosody to command. In all the expressive prosody tests there are 20 test items and for the affective prosody tests the five emotions assessed are sad, happy, anger, fear, and neutral. The order in which these emotions are tested is randomized. Performance

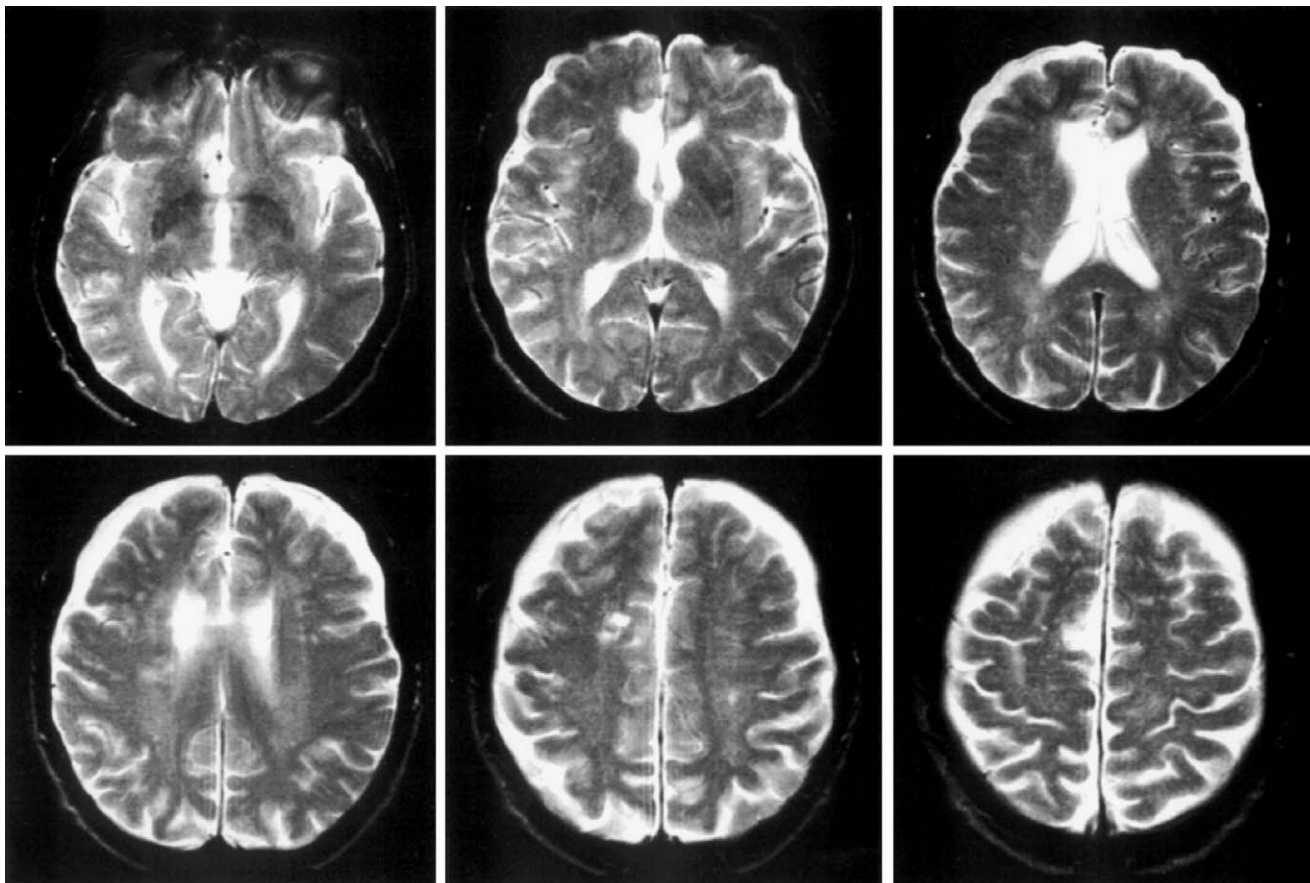


Fig. 1. MRI of subject revealing right medial frontal lesion.

on all prosody assessments was evaluated by a panel of three experienced judges, expert in the evaluation and treatment of aprosodia. When asked to imitate a sentence that contains syntactic prosody (question versus statement) the patient was 70% accurate (Table 1). When asked to imitate a semantically neutral sentence using emotional prosody, he was 60% accurate. In the third imitative test he was asked to repeat sentences where the semantic content was either incongruent or congruent with the requested emotional prosody (e.g., Repeat “All the puppies are dead.” with a sad tone.) He was 30% accurate on this task.

When the patient was asked to produce a sentence to command using syntactic prosody, he was able to correctly produce questions and statements 100% of the time, but when he was asked to produce affective prosody with a verbally neutral sentence he was only 25% accurate. When asked to inflect a sentence with an incongruent prosody, he demonstrated 0% accuracy. Thus, it appears that his main expressive problem was with spontaneously intoning affective prosody.

To assess his ability to comprehend affective prosody, he was given the Florida Affect Battery (Bowers, Blonder, & Heilman, 1991). This battery tests a patient's

ability to identify spoken affective prosody as well as emotionally expressive faces. In one prosody task, he was presented with series of paired sentences spoken either using the same or different affective prosody (e.g., both spoken in a happy tone or one spoken in a happy tone and the other a sad tone) and he had to decide if the two sentences were intoned with the same or different emotional prosody. He was 70% accurate on this test, while normal subjects in his age range get 97.8% (SD 4.6) correct. When asked to name the emotional intonation when the semantic content was neutral, he was 50% accurate, but normal subjects are 83% (SD 13.0) accurate. When the affective prosody conflicted with the semantic content of the message and he was asked to name the emotion expressed by the affective prosody, he was 21% accurate whereas normal subjects perform with 61.7% (SD 12.6) accuracy. In contrast to his impaired comprehension of emotional prosody, he performed normally on affective facial discrimination tasks. For example, when asked to discriminate emotional faces he was 85% accurate; normal subjects perform with 87.8% (SD 9.8) accuracy. When he was asked to name the type of emotion displayed on a face he was 90% accurate and normal controls are 87% (SD 8.7) accurate.

Table 1
Test results of affect comprehension and discrimination as well as prosody (emotional and non-emotional) expression

Test	Pretreatment
<i>Florida Affect Battery</i>	
Identity Discrimination	90
Affect Discrimination	85
Name Affect	90
Select Affect	95
Match Affect	Discontinued
Non-emotional Prosody Discrimination	31
Emotional Prosody Discrimination	70
Name Emotional Prosody	50
Conflicting Prosody	36
Match Emotional Prosody to Emotional Face	35
Match Emotional Face to Emotional Prosody	Not tested
<i>Expressive Battery</i>	
Non-emotional Prosody Imitation	70
Emotional Prosody Imitation (neutral semantic content)	60
Emotional Prosody Imitation (semantics congruent/ incongruent)	30
Emotional Prosody to Command (semantics congruent/incongruent)	0
Emotional Prosody to Command (neutral semantic content)	25
Non-emotional Prosody to Command	100

3. Discussion

When seen in our rehabilitation center 7 years after he sustained a right medial frontal lobe stroke that damaged portions of his supplementary motor area and the anterior cingulate gyrus, the patient had evidence of affective aprosodia. Because he was not aphasic and he was flawless at producing non-emotional prosody (e.g., questions versus statements) to command, but was dramatically impaired at producing emotional prosody, it appears he had an affective prosodic expressive deficit. His ability to comprehend and repeat emotional prosody was also impaired, but perhaps not as impaired as his expression. Although this patient did have some hearing loss, he was able to repeat the words in sentences well and could repeat syntactic prosody. Thus, his aprosodia could not be attributed to decreased hearing.

Left hemisphere superio-medial frontal lesions that involve the supplementary motor area can induce aphasia. Usually these patients are non-fluent but have intact comprehension and flawless repetition. This aphasic syndrome has been called transcortical motor aphasia (Caplan, 2003). Observation of the patient reported here suggests that lesions of the right hemisphere's superio-medial frontal cortex can induce affective, but not syntactic aprosodia. Unlike patients with left-sided medial frontal lesions who have intact speech repetition and comprehension and thus are classified as transcortical motor aphasia, our patient's repetition and comprehension of emotion prosody was

not normal. Thus, his prosody disturbance induced by right medial frontal hemisphere damage did not entirely mirror the transcortical motor aphasia syndrome observed with left medial frontal hemisphere damage. However, the profile of a greater impairment in spontaneous affective prosodic expression than in repetition or comprehension is similar to the profile observed with transcortical motor aphasia.

The reason why a right superio-medial frontal lesion would produce a mixed aprosodia, with impairments of expression, comprehension, and repetition, is not entirely known, but there are several possibilities. One possibility is that after his initial stroke 7 years prior to our evaluation, he had another stroke that impaired his repetition and comprehension of prosody. However, there are several observations that are inconsistent with this hypothesis. First, since his stroke in 1993, he has had no other new episodes of neurological impairment and most often strokes in other regions would induce other deficits. Second, after he was evaluated by us he fell and hit his head. Because he was being treated with anticoagulants, a CT scan was performed. The interpretation of this scan by a radiologist, who is not an author of this paper was that there was no evidence of a new cerebral infarction, intracranial hemorrhage, or mass, but there was evidence of his old cerebral infarction in the right frontal lobe.

Another possible explanation for his mixed aprosodia is a callosal disconnection. Prior studies have suggested that affective prosody is incorporated into propositional speech by means of an interhemispheric mechanism (Klouda, Robin, Graff-Radford, & Cooper, 1988; Ross, Thompson, & Yenkosky, 1997; Speedie, Coslett, & Heilman, 1984). Based on his MRI scan there is a high probability that this patient's stroke damaged some of the fibers crossing through the anterior portion of the corpus callosum inducing a partial callosal disconnection. This callosal disconnection might have dissociated verbal output mediated by the left hemisphere from the expression of emotional prosody mediated by the right hemisphere. Whereas this explanation might account for the impaired expression and repetition of affective prosody, it could not account for the impaired comprehension of emotional prosody.

As mentioned above, left frontal lesions induce an impairment of verbal fluency (Caplan, 2003) and functional imaging studies in normal subjects reveal that verbal fluency is associated with activation of this area (Crosson et al., 1999). Functional imaging studies have also revealed that emotionally salient stimuli activate the anterior cingulate gyrus (Lane et al., 1998; Taylor, Phan, Decker, & Liberzon, 2003; Teasdale et al., 1999). Several mechanisms have been proposed to explain why left medial frontal lesions reduce fluency, including: verbal motor akinesia, inertia of or impaired access to concept formations (semantic representations), and impaired

access to the verbal output lexicon (Gold et al., 1997). Right medial frontal lesions might induce a parallel impairment of emotional prosody (e.g., prosodic emotional akinesia, inertia of or impaired access to emotional concepts or to affective echoic representations which are stored in the right hemisphere).

According to Jurgens (1986), the different human non-verbal emotional vocal utterances (e.g., laughing, shrieking, and moaning) and emotional intonation patterns (e.g., scolding, lamenting, and caressing) can be shown to have their acoustic and emotional counterparts in the vocal repertoire of the monkey. In the monkey the anterior cingulate cortex seems to be responsible for the volitional control of emotional vocal utterances. Thus, non-verbal emotional vocal utterances such as affective prosody might be the human homologue of monkey's emotional vocalizations. While the emotional vocalizations in monkeys most often appear to mediated bilaterally, in some *Macaca mulatta* this function appears to be lateralized (Sutton, Trachy, & Lindeman, 1981).

Both lesion and functional imaging studies have suggested that the anterior cingulate gyrus is important for mediating attention and hemispheric arousal (Heilman, Watson, & Valenstein, 2003). Attention to important stimuli and right hemisphere arousal are critical for the processing and experiencing of emotional stimuli. Patients with right hemisphere lesions that reduce right hemisphere arousal and induce inattention often show manifestations of the neglect syndrome, but when tested this man did not show evidence of neglect. In addition, if his right hemisphere was hypo-aroused we would have expected other functions mediated by the right hemisphere to also be impaired and yet he performed normally on the affective facial tests. These observations suggest that his problem was not a global right hemisphere processing deficit or a general cognitive deficit.

When monkeys received horseradish peroxidase injections in the anterior cingulate cortex at the level of the genu of the corpus callosum, a region yielding emotional vocalizations when electrically stimulated and the homologue of the area that was injured in our patient, retrogradely labeled neurons were found at the cortical level within auditory association cortex (e.g., the banks of the superior temporal sulcus, Jurgens, 1983). A functional imaging study also suggests that the medial frontal regions might be a critical element in an auditory attentional network (Benedict et al., 1998) and hence, lesions of this medial frontal-auditory association cortex network might impede repetition and comprehension of affective prosody which is heavily dependent upon the function of the right auditory cortex.

Adolphs, Damasio, and Tranel (2002) studied 66 patients with strokes and found that in addition to posterior lesions impairing the recognition of emotional

prosody, frontal lesions were also associated with emotional prosodic comprehension deficits. The finding that the frontal lobe might be important in the comprehension of emotional prosody is supported by positron emission tomography (PET) studies that demonstrated right frontal activation when normal subjects were comprehending emotional prosody (George et al., 1996; Imaizumi et al., 1997). These lesion and functional imaging studies suggest that the right hemisphere might be organized differently than the left, such that whereas the left is highly modular, the right contains many more distributed representations and this patient's focal right frontal medial lesion might have interrupted a distributed network important for expression, comprehension, and imitation of emotional prosody.

Cancelliere and Kertesz (1990) as well as Karow and Connors (2003) also studied brain lesioned subjects ability to express and comprehend emotional prosody. Both of these studies reported that injury to the basal ganglia also appears to impair these functions. Although our patient did not injury his basal ganglia, he did injure the supplementary motor area, which is strongly connected with the basal ganglia (i.e., putamen) and the mixed aprosodia described by these authors might also be related to medial frontal (supplementary motor) dysfunction.

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