

Sensory Aprosodia With Left Hemiparesis From Subcortical Infarction

Right Hemisphere Analogue of Sensory-Type Aphasia With Right Hemiparesis?

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• We report a case of sensory aprosodia with left hemiparesis following an ischemic infarction of the right thalamus and posterior limb of the internal capsule. Bedside evaluation, confirmed by special quantitative tests, demonstrated normal spontaneous affective prosody and gesturing with marked impairment of affective repetition and comprehension of affective prosody and gestures. A left hemiparesis with sensory loss was also present. This combination of deficits appears to represent the right-side analog to the unusual syndrome of Wernicke-type aphasia with right hemiparesis occasionally observed following left subcortical injury, thus providing further support for the hypothesis that the functional-anatomic organization of affective language in the right hemisphere mirrors that of propositional language in the left.

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Recent and compelling evidence has demonstrated that lesions involving the right hemisphere may compromise the affective components of language, while leaving the propositional intact.¹⁻⁴ Thus, the concept of "language" being a dominant function of the left hemisphere is no longer tenable. Rather, the propositional elements of language are a dominant function of the left hemisphere, while

the affective elements of language are probably a dominant function of the right hemisphere. Evaluation of clinical material and cerebral blood flow maps also suggests that the functional-anatomic organization of the affective components of language in the right hemisphere—encompassing affective prosody and emotional gesturing—is homologous to the organization of propositional language in the left hemisphere.^{1,2,5-8} Therefore, disorders of affective language, called *aprosodias*,¹ can be classified by the same specific modifiers used in aphasia terminology, eg, motor, global, and transcortical sensory. Furthermore, recovery patterns from affective deficits following right hemisphere injury also appear to mimic recovery patterns from propositional deficits following left hemisphere injury.⁶

The right hemisphere analogy also extends to the role of subcortical structures in language. Numerous investigators have documented aphasia-like syndromes in patients with lesions involving the left thalamus⁹⁻²⁰ or left internal capsule and striatum²¹⁻²⁵; likewise, there have been scattered reports of aprosodia following subcortical injury.^{1,8,26} Although subcortical aphasias share many features with the traditional cortical syndromes, atypical combinations of deficits can be observed. For instance, a right hemiparesis associated with a sensory-type aphasia may be encountered from a lesion involving the left thalamus or striatum and posterior limb of the internal capsule^{14,17,21,22,25} but, to our knowledge, has not been described from a predominantly cortical lesion. By *sensory-type aphasias*,

we refer to disorders characterized by impaired comprehension with fluent but paraphasic speech. Since repetition of propositional language is preserved in most reported cases of subcortical sensory-type aphasia,^{14,15,18,27} the overall clinical presentation suggests a transcortical syndrome. However, occasional reports have documented subcortical sensory-type aphasias that are more Wernicke-like,^{15,21,22} ie, with impaired repetition. Unlike the cortical aphasic syndromes, comprehension and repetition deficits in the subcortical cases tend to be moderate and usually show good recovery. In this article, we report the first case of the right hemisphere analogue of sensory-type aphasia with hemiparesis—sensory aprosodia with left hemiparesis.

REPORT OF A CASE

A 49-year-old, left-handed woman was admitted to Parkland Memorial Hospital, Dallas, after sudden onset of a flaccid left hemiplegia with dysarthria. Her medical history was remarkable for obesity, hypertension, and adult-onset diabetes. The patient was alert, oriented to time, person, and place, and had no aphasic deficits except for mild anomia.

Within ten days, the patient showed a predilection upper motor-neuron pattern of weakness with spasticity in the left lower extremity. There was little motor recovery in the left arm. Facial movements were full bilaterally. Marked hemianesthesia was present over the entire left side, including face and torso, to pin and temperature. Graphesthesia and position sense were severely compromised in the left hand. Hemianopsia was not present, even on double simultaneous stimulation of the visual fields.

The patient's speech was normally fluent

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without paraphasias. There was a mild anomia for parts of objects, which resolved rapidly during hospitalization. Comprehension and repetition of propositional language were completely intact. Spontaneous affective prosody and gesturing involving the face and right limbs were normal, but affective-prosodic repetition was extremely poor; the patient's voice was flat and monotonic in stark contrast to her spontaneous affective prosody. Likewise, auditory comprehension of affective prosody was severely impaired. The patient was unable to identify sadness, happiness, boredom, anger, surprise, or neutral on formal testing and could not visually discern various facial emotions when "pantomimed" by the examiner. These findings were consistent with the syndrome of sensory agraphia.¹

The patient was discharged on the 17th hospital day and reexamined four weeks later. A predilection pattern of weakness was still present in the left lower extremity. Limited movement was possible in the proximal left upper extremity, but the forearm and hand remained paralyzed. In the left leg there was greater motor activity distally than proximally. Facial movements were intact. Pin and temperature sensations were still impaired over the left extremities and torso, but no longer involved the left side of the face. Graphesthesia and position sense were now intact on the left side, and stereognosis was unimpaired if the examiner manually assisted the patient in palpating objects that were placed in her left hand.

The patient's propositional language was fully normal. Bedside assessment of affective language confirmed the persistence of a sensory agraphia. In addition, special tests were conducted to quantify the patient's deficits in processing affective language.

SPECIAL TESTS

Spontaneous Affective Prosody

In nontonal languages such as English, varying the fundamental frequency (Fo) and its harmonics during voicing to produce intonational contours is probably the most important acoustical parameter for imparting affect into speech.²⁸ The Fo variation of the patient's spontaneous speech was quantitatively measured on a pitch analyzer (PM Pitch Analyzer, Voice Identification Inc, Somerville, NJ) using the procedure described by Ross et al.²⁹ When an utterance is played into the input of the pitch analyzer, a tracing of the Fo in hertz and intensity in decibels over time is displayed on a cathode-ray tube. By using programmable cursors, the means and SDs for the Fo and decibel contours for an utterance can be measured. With the aid of a computer (PDP 11/23, Digital Inc, Maynard, Mass), the means and SDs of ten spontaneous utterances recorded during the interview were mathematical-

ly combined to give a *grand mean* and SD. The coefficient of variation (CV) of the grand mean was then calculated and multiplied by 100 to obtain an overall measurement of Fo variability (Fo-CV%). The Fo-CV% score for spontaneous speech was then compared with a value compiled previously from six controls whose Fo-CV% group mean was $17.0 \pm 3.1\%$.²⁹ The patient's Fo-CV% for spontaneous speech was 16%, yielding a Z score of -0.32 when compared with controls, thereby substantiating that spontaneous affective prosody, as measured by Fo-CV%, was normal.

Affective-Prosodic Repetition

The Fo variation for repetition was assessed with the sentence "I am going to the other movies," using a standard stimulus tape with six affective tokens: neutral, happy, sad, angry, bored, and surprised. The patient was given two attempts at each token. All 12 utterances were analyzed on the pitch analyzer as outlined above, and a grand mean Fo-CV% was calculated. The Fo-CV% score for affective-prosodic repetition compiled from six controls was $25.7 \pm 4.0\%$.²⁹ The patient's Fo-CV% was 12%, yielding a Z score of -3.43. Furthermore, the patient's Fo-CV% did not overlap with any control value (range, 20% to 32%), thus substantiating marked impairment of affective-prosodic repetition.

Auditory Comprehension of Affective Prosody

Auditory comprehension involving emotions was formally assessed with the sentence "I am going to the other movies," using a standard stimulus tape containing the same six affective tokens used in the repetition task. Each token appeared twice in randomized order during the test. The patient was asked to identify the affect in each of the 12 tokens by selecting the correct response from a written list. Prior to testing, the examiner coached the patient with the list; no primary visual or reading deficits were evident that could have interfered with testing. Auditory comprehension of affective prosody was again found to be severely impaired. The patient gave correct responses on only two of 12 trials. Normal performance based on 12 controls is 12 of 12 or occasionally 11 of 12 correct. Considering there were six affects to choose from, the patient's performance was at chance.

A second test was administered to evaluate deficits in attitudinal com-

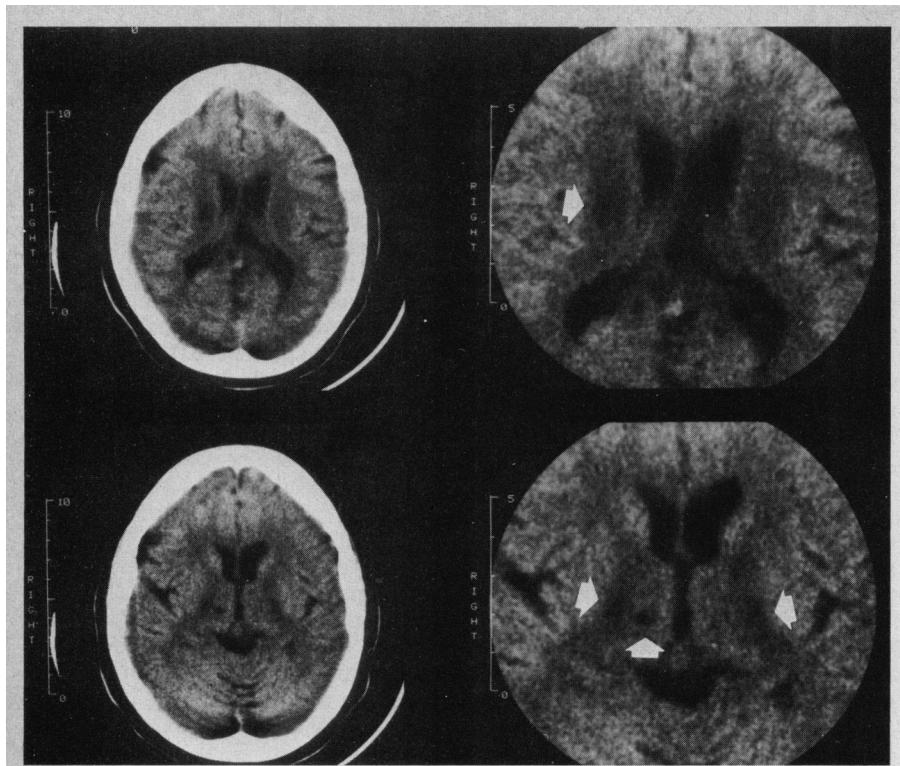
prehension. The patient was asked to classify 20 audio-taped stimuli that, depending on their prosodic content, could be interpreted literally or ironically. For instance, the sentence "I had a great day today" can be articulated using enthusiastic prosody, which affirms the literal intent of the statement, or using ironic prosody, which implies an opposite intent. Enthusiastic and ironic versions for ten sentences were randomly presented to the patient. After each stimulus, she was asked to respond "yes" or "no" to a verbal question constructed to discriminate the attitudinal intent of the sentence. For example, the stimulus "The weather is so pleasant" was paired with the question "Is the weather nasty?" Prior to formal testing, the task was explained to the patient by coaching with sample sentences and questions. The patient correctly identified only 13 of 20 stimuli. Normal scores on this test based on a control sample of 22 subjects are 20 of 20 (n = 16) or 19 of 20 (n = 6) correct. Thus, the patient's performance was close to chance levels (10 of 20 correct), substantiating that she had a profound deficit in discriminating the attitudinal, in addition to the emotional, aspects of affective prosody.

Visual Comprehension of Emotional Gesturing

Comprehension of gesturing was tested by having the patient examine six line drawings of faces depicting the six emotions outlined above. She was then asked to match, after initial coaching, each of the six drawings with the correct descriptor from a written list that was also read aloud to her. She failed to correctly identify angry, neutral, surprised, and happy but did identify sad and bored. This performance, two of six correct, was highly deviant, since normal subjects (n = 120) respond correctly $95 \pm 5\%$ of the time and never misidentify happy (Y. W. Walston, MA, unpublished thesis, University of Texas Health Science Center at Dallas, 1982).

Computed Tomographic (CT) Scans

A CT scan performed on the day of admission showed a subtle hypodensity involving the right thalamus and posterior limb of the internal capsule. A second scan performed 6½ weeks poststroke delineated an ischemic infarction that involved the right thalamus and extended into the posterior limb of the internal capsule (Figure). In addition, a circumscribed



Computed tomographic scan 6½ weeks poststroke. Right panels are 2:1 zooms of left panels. Arrow heads point to recent ischemic infarction involving right thalamus and posterior limb of internal capsule, present on both cuts, and to possible infarction involving posterior limb of left internal capsule, which is seen on only one cut. Remainder of scan was completely normal.

hypodense area, possibly representing a lesion, involving the midportion of the left internal capsule was visualized on a single CT cut (Figure). If present, the lesion was clinically "silent," since no right-sided elementary findings or pseudobulbar palsy were evident on neurologic examination, and the patient repeatedly denied any past symptoms consistent with a stroke.

COMMENT

The results from both the bedside and quantitative examination of affective language confirmed that our patient had a sensory aphasias. Although the patient is left-handed, most likely she has standard rather than mixed hemispheric dominance for language. While it could be argued that the mild anomia observed during the first examination represented a manifestation of mixed dominance, we believe the source of the anomia was related to her occasional inattentiveness during testing, since difficulty with naming occurred only during those time periods and was usually a perseverative error. Moreover, her intact comprehension and expression of propositional language, coupled with the constellation of severe affective

deficits, strongly imply a normal lateralization pattern.

Although study of aphasias at both the cortical and subcortical levels is relatively novel, an association between left subcortical injury and propositional language disorders has been suspected for nearly 50 years.²⁰ Only recently, however, has clinical evidence for subcortical aphasias been widely reported and acknowledged. Despite growing evidence for a specific language function of the basal ganglia and thalamus, questions persist on basic mechanisms. Early on, skeptics argued that the language deficits resulting from subcortical injury actually represented the impairment of cortical processing by mass effects.^{15,30,31} While mass effects are certainly a factor in many cases of subcortical hemorrhage and tumor, they cannot account for the language disorders reported in circumscribed, nonedematous hemorrhages and ischemic infarctions involving the thalamus or basal ganglia and internal capsule. Whether the language deficits following subcortical injury represent true aphasias or merely the loss of "support functions" for cortical processing is not known. While we do not offer new evidence to solve this

ongoing debate, we accept that subcortical lesions, in and of themselves, can produce aphasic syndromes that may have features not normally associated with the classic cortical aphasias. For example, in contrast to Broca's aphasia, speech recovery from a subcortical motor-type aphasia is not typically associated with telegraphic speech,²² suggesting that primary linguistic processing may be intact. On the other hand, the comprehension deficits observed following subcortical lesions do not appear to differ from those seen in the traditional cortical syndromes. However, only the subcortical variety of sensory-type aphasias are accompanied by hemiparesis.^{14,17,21,22,25} Although these aphasias are usually characterized as transcortical sensory, there are a few cases in which the deficits are Wernicke-like. The lesions in this instance have involved thalamus¹⁵ or basal ganglia.^{21,22,25} A right hemiparesis has also been a constant associated feature due to internal capsule involvement.

Since aphasic syndromes occur following circumscribed, left subcortical lesions, one would predict that homologous injuries in the right hemisphere would cause aphasias. In fact, at least four cases of purely subcortical lesions have been reported in the literature with motor, transcortical motor, or transcortical sensory aphasias.^{1,8,26} The CT scan in the present study showed no indication that the cortex or adjacent white matter was affected by infarction. Thus, our patient's sensory aphasias and left hemiparesis were the direct consequence of a purely subcortical lesion involving the right thalamus and posterior limb of the internal capsule.

Our case supplements previous evidence that the right thalamus and basal ganglia play a role in modulating the affective components of language.^{1,8,26} Moreover, the combination of sensory aphasias with left hemiparesis appears to represent the right-side analog to the unusual Wernicke-hemiparesis syndrome occasionally observed following left subcortical injury, thus lending support to the hypothesis that the functional-anatomic organization of affective language in the right hemisphere mirrors that of propositional language in the left.^{1,2,5,8}

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Behavioral Manifestations of Central Pontine Myelinolysis

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• A young woman with a clinical history and magnetic resonance imaging scan consistent with central pontine myelinolysis came to medical attention because of prominent behavioral symptoms. Marked clinical recovery occurred despite persistent radiologic abnormalities. Rapid correction of hyponatremia was probably related to the development of the central pontine myelinolysis. A normal computed tomographic scan and the absence of brain-stem signs delayed accurate diagnosis.

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Central pontine myelinolysis (CPM) was first described by Adams et al¹ in 1959. Early descriptions empha-

sized its grave prognosis.² More recent reports have documented survival and recovery.³ Ventral pontine involvement with subacute onset of cerebellar, cranial nerve, and pyramidal tract dysfunction is the typical presentation.⁴

We describe a patient in whom reversible behavioral abnormalities constituted the major manifestation of CPM. One purpose of this article is to alert physicians to the possible behavioral presentation of CPM. Another is to reemphasize the relationship between rapid correction of hyponatremia and CPM. We also forward some thoughts on the pathophysiology of these behavioral changes.

REPORT OF A CASE

A 29-year-old right-handed professional woman was in excellent health until her return from Mexico. She developed intractable nausea, anorexia, and diarrhea for one week, leading to a 2.2-kg (5-lb) weight loss.

Results of a clinical examination at that time were normal, except for orthostatic hypotension. Laboratory test results disclosed the following values: serum sodium, 114 mEq/L (114 mmol/L); potassium, 1.9 mEq/L (1.9 mmol/L); arterial blood gases demonstrated respiratory alkalosis; her alanine aminotransferase level was elevated to 128 U/L; and aspartate aminotransferase, to 115 IU/L. *Campylobacter* was grown from her stools. There was no history of major psychiatric disease in the patient or her family.

She received 2 L of normal saline with potassium supplementation. Seventeen hours later, her sodium level was 133 mEq/L (133 mmol/L), and her potassium level was 4.2 mEq/L (4.2 mmol/L). She recov-

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