

## Case Reports

# Expressive Aprosody and Amusia as a Manifestation of Right Hemisphere Seizures

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**Summary:** *Purpose:* Aprosody and amusia are disorders commonly associated with right hemisphere abnormalities. They are regarded as negative phenomena and usually seen after strokes. We report a case of a patient who had both expressive aprosody and amusia as a clinical manifestation of right temporooccipital seizures.

*Methods:* A 43-year-old woman had a 1-month history of monotonic speech and difficulty singing. Her examination revealed both expressive aprosody and amusia. Magnetic reso-

nance imaging of the head was normal, but her EEG revealed several electrographic seizures of right temporooccipital origin.

*Results:* Treatment with phenytoin (PHT) almost immediately caused her speech and singing to return to baseline. A repeated EEG was normal

*Conclusions:* Seizures of right temporooccipital origin can manifest with expressive aprosody and amusia. **Key Words:** Amusia—Aprosody—Electroencephalogram—Epilepsy—Seizures.

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Disorders of prosody refer to abnormalities involving the affective components of speech that include pitch, melody, intonation, and gestural posturing (1). These abnormalities are generally categorized as either expressive or receptive and are due to lesions of the nondominant hemisphere that occur in a location homologous to those of the dominant hemisphere that lead to an impairment of propositional language (aphasia) (2).

Amusia is an impaired capacity for musical activity that cannot be explained by damage to the articulatory apparatus or to primary receptor mechanisms (3). Similar to the aphasias and the aprosodias, amusias also have been categorized as either expressive or receptive. Expressive amusia is the loss of ability to sing, write musical notation, or play an instrument. Receptive amusia is defined as “musical deafness,” including the inability to recognize familiar melodies or the loss of ability to read musical notation (4).

Both amusia and aprosody are generally considered negative phenomena and result from structural lesions such as strokes. However, we present a case of both

expressive aprosody and amusia presenting as an ictal manifestation of seizures emanating from the right temporooccipital region.

### CASE REPORT

A 43-year-old woman who was a church choir member had a 1-month history of changes in her voice. Her speech became monotonic, and she also experienced difficulty with singing. In addition, she complained of intermittent paresthesias involving the left upper extremity and facial region. Her medical history was significant for breast cancer, for which she underwent a lumpectomy in August 2000 and subsequent radiation therapy and chemotherapy with doxorubicin, paclitaxel, and cisplatin from October 2000 to January 2001.

Neurologic examination for prosody used the criteria enumerated by Ross (2). The patient had poor pitch variation and intonation, resulting in monotonic speech with poor spontaneous prosody and affective repetition. Postural gesturing was somewhat impaired, resulting in a flat affect; however, affective comprehension was intact. She had difficulty with singing because of imprecise production of pitch variation. The rest of her neurologic examination was normal. Magnetic resonance imaging (MRI) of the head with and without gadolinium and a

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lumbar puncture including cytology studies were normal. A routine 30-min EEG documented six episodes of rhythmic sharp and slow-wave activity emanating from the right temporooccipital area and lasting ~30 s each.

The patient was started on phenytoin (PHT), 100 mg, 3 times a day. The following day, her speech returned to baseline. A repeated EEG was normal. On examination she had normal prosody, and her singing abilities returned. Her left-sided paresthesias also resolved. A repeated MRI of the head 3 months later was normal.

### DISCUSSION

This is the first known report of expressive aprosody and amusia with a clinical manifestation of seizures of right temporooccipital origin. Our patient was essentially in simple partial status epilepticus, as evidenced by the frequent runs of ictal activity seen on routine EEG associated with the speech abnormalities. She had a dramatic resolution of symptoms with PHT, and this correlated with normalization of EEG findings.

Our patient had both positive (left-sided paresthesias) and negative (expressive aprosody and amusia) ictal manifestations. Left-sided paresthesias are commonly seen in seizures of right parietal origin (5). Negative ictal symptoms occur much less frequently but are well described and may include speech arrest, paralysis, blindness, or loss of hearing (6,7). The reasons for negative ictal manifestations are not well established but postulated to be secondary to either a stimulation of inhibitory cortical regions (8), a dampening of receptive abilities of sensory regions (7), or inhibition of spinal motor neurons (9).

The reason for the patient's seizures remains unclear; obviously a history of breast cancer raises the concern of brain metastasis. That she had both positive and negative ictal manifestations and that her symptoms per se indicate involvement of two distinct brain regions strongly suggests that the underlying etiology behind her spells could be brain metastasis. However, the patient had a normal lumbar puncture including cytology studies, and repeated MRI studies of the brain were normal.

Although the localization for aprosody is generally accepted to be nondominant hemisphere in origin, the precise anatomic localization for the different types of amusia is less well known.

As in the case of our patient, expressive amusia with preservation of receptive functions and without aphasia has been associated with lesions of the right hemisphere and is accompanied by an expressive aprosody (10,11). A temporary state of expressive vocal amusia can be induced after amobarbital injection into the right carotid artery (12). However, patients with dominant hemisphere lesions who develop Broca aphasia are also likely to show concomitant expressive amusia (4).

Localization for receptive musical abilities is even less well known and also has been associated with temporal lobe disease involving either or both hemispheres (4,13). Independent of disease, localization for receptive musical abilities also is dependent on factors such as inherent musical aptitude, musical training, age, and sex (3,10).

Previously described seizures associated with music include musicogenic epilepsy, a phenomenon wherein music is a precipitating stimulus for seizures (14), and musical partial seizures, wherein the ictus consists of hallucinated music (3). Both events are more frequently of right hemisphere origin; however, reflex epilepsies brought about by singing are associated with epileptiform discharges emanating from both left and right centrotemporal regions (15).

Our case shows that seizures originating from the non-dominant hemisphere can result in both aprosody and expressive amusia, and that there exists a relation, albeit poorly understood, between expressive musical abilities and the affective components of speech.

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### REFERENCES

1. Devinsky O, ed. *Behavioral neurology*. St. Louis: Mosby-Year Book, 1992.
2. Ross ED. The aprosodias. *Arch Neurol* 1981;38:561-9.
3. Brust JCM. Music and the neurologist: a historical perspective. *Ann N Y Acad Sci* 2001;930:143-52.
4. Benton AL. The amusias. In: Critchley M, Henson RA, eds. *Music and the brain*. London: William Heinemann Medical Books Ltd., 1977:378-97.
5. Van Ness PC, Lesser RP, Duchowny MS. Simple sensory seizure. In: Engel J, Pedley TA, eds. *Epilepsy: a comprehensive textbook*. Philadelphia: Lippincott-Raven, 1997:533-55.
6. Meador KJ, Moser E. Negative seizures. *J Intl Neuropsychol Soc* 2000;6:731-3.
7. Ghosh D, Mohanty G, Prabhakar S. Ictal deafness: a report of three cases. *Seizure* 2001;10:130-3.
8. Noachtar S, Luders HO. Focal akinetic seizures as documented by electroencephalography and video recordings. *Neurology* 1999; 53:427-9.
9. Matsumoto R, Ikeda A, Ohara S, et al. Nonconvulsive focal inhibitory seizure: subdural recordings from motor cortex. *Neurology* 2000;55:429-31.
10. Peretz I. Brain specialization for music. *Ann N Y Acad Sci* 2001; 930:153-65.
11. Confavreux C, Croisile B, Garassus P, et al. Progressive amusia and aprosody. *Arch Neurol* 1992;49:971-6.
12. Gordon HW, Bogen JE. Hemispheric lateralization of singing after intracarotid sodium amylobarbitone. *J Neurol Neurosurg Psychiatry* 1974;37:727-38.
13. Schuppert M, Munte TF, Wieringa BM, et al. Receptive amusia: evidence from cross-hemispheric neural networks underlying music processing strategies. *Brain* 2000;123:546-59.
14. Weiser HG, Hungerbuhler, Siegel AM, et al. Musicogenic epilepsy: review of the literature and case report with ictal single photon emission computed tomography. *Epilepsia* 1997;38:200-7.
15. Herskowitz J, Rosman NP, Geschwind N. Seizures induced by singing and recitation: a unique form of reflex epilepsy in childhood. *Arch Neurol* 1984;41:1102-3.