4. Consensus Committee of the American Academy of Neurology. Consensus statement on the definition of orthostatic hypotension, pure autonomic failure, and multiple system atrophy. *Neurology* 1996;46:1470.
5. Mitchell AW, Luheshi LM, Barker RA. Skin and platelet α -synuclein as peripheral biomarkers of Parkinson's disease. *Neurosci Lett* 2005;381:294–298.
6. Ikemura M, Saito Y, Sengoku R, et al. Lewy body pathology involves cutaneous nerves. *J Neuropathol Exp Neurol* 2008;67:945–953.
7. Mitsui J, Saito Y, Momose T, et al. Pathology of the sympathetic nervous system corresponding to the decreased cardiac uptake in 123I-metaiodobenzylguanidine (MIBG) scintigraphy in a patient with Parkinson disease. *J Neurol Sci* 2006;243:101–104.

Kate El Bouzidi, MB, ChB
Susan Duncan, MD,
FRCP
Ian R. Whittle, PhD,
FRCS
Christopher R. Butler,
PhD, MRCP

LESIONAL REFLEX EPILEPSY ASSOCIATED WITH THE THOUGHT OF FOOD

A 44-year-old right-handed woman was walking in the Scottish highlands. Upon unwrapping her lunch, she had a focal seizure with witnessed onset on the right side of the face and secondary generalization. Postictally, she was aphasic with a right hemiparesis. She was airlifted to hospital. Sodium valproate was commenced, increasing to 700 mg twice daily, and she was discharged home. Three weeks later, the smell of food triggered another seizure and she was admitted to the neurology unit where carbamazepine was introduced (200 mg twice daily).

The next morning, the patient had a simple partial seizure after eating a spoonful of porridge and 3 more when eating lunch, a snack, and dinner. Thereafter, most meals triggered seizures, as did other food-related stimuli such as being offered a piece of cake, seeing her visitors pass around food at her bedside, and smelling the hospital dinner trolley. Fifty-four seizures occurred over the next 14 days and 50 were related to food. The episodes typically lasted 80 seconds and were characterized by a tingling sensation in the tongue and right-sided facial and tongue movements. Consciousness was unimpaired.

Thirty-four seizures occurred during the act of eating, always early on in the meal while the patient still felt hungry. Fifteen were precipitated by the sight or smell of food alone, 1 while browsing a recipe book, and 3 when she could smell her pet dog's food. One seizure was provoked by discussing cooking and another was triggered by the mint flavor of toothpaste. A single nocturnal seizure was accompanied by a strong feeling of nausea. The 2 other seizures associated with nonfood stimuli were related to stressful situations such as discussing a call from the doctor. Neither chewing movements nor speaking induced seizures.

Interictal EEG was normal. MRI revealed a non-enhancing lesion in the left premotor strip (figure).

The seizures were refractory to medical therapy. An awake frontoparietal craniotomy was performed with electrocorticography. Epileptiform activity was identified in the left frontal operculum, anterior and inferior to the lesion (figure). A subtotal resection

was performed. Histopathology revealed a WHO grade IV glioblastoma. No reflex seizures occurred after surgery.

Discussion. We present a case of symptomatic reflex seizures triggered by food-related stimuli, with detailed neuroimaging and electrophysiologic localization of the epileptic focus. Reflex epilepsies are characterized by seizures consistently induced by a specific stimulus. Typically, they are idiopathic and generalized. Recognized stimuli include flashing lights, music, reading, toothbrushing, and eating. Over 200 cases of idiopathic eating epilepsy have been reported, many from South Asia, with a male preponderance and onset typically in the second decade of life.¹ In all cases, the act of eating was required to provoke seizures, which were focal or generalized. Of 128 cases in which treatment response is described, 48 (37.5%) became seizure-free, 64 (50%) achieved partial control of seizures, and 16 (12.5%) showed no benefit.^{1,2}

There are far fewer reported cases of symptomatic eating epilepsy. The associated pathologies include congenital malformations, vascular abnormalities, postinfective lesions, and 1 neoplasm: an astrocytoma. Lesions involved the opercula, amygdalae, and temporoparietal lobes.^{3,4} Most cases were refractory to medical therapy.

Rarer still are cases of reflex epilepsy triggered by the thought of a specific stimulus. One report described a man with temporal lobe seizures induced by thinking of his childhood home.⁵ Another patient with a left temporal focus on EEG had seizures when brushing his teeth but also on thinking of a toothbrush.⁶ To our knowledge, there have been no reports of seizures precipitated by the thought of food or hunger alone.^{1,3}

Several mechanisms have been postulated for eating epilepsy, from somatosensory and motor stimulation to gastric distension.^{2,3} Primary taste areas in the anterior insula and frontal operculum are activated by taste and olfactory stimuli but also the anticipation of food.³ Taste processing is refined in the secondary taste areas of the amygdala and orbitofrontal cortex where a greater number of neurons receive multimodal inputs.³ Functional MRI has demon-

(A) Transverse T2-weighted MRI scan with arrow indicating the lesion in the left frontal cortex (L = left). (B) Three-dimensional reconstruction of the patient's brain with superimposed graphics showing the location of the tumor (yellow) and intraoperative cortical electrodes. The electrodes that recorded epileptiform activity are shown in red. (C) Intraoperative electrocorticography revealed epileptiform activity in the leads indicated by the asterisk.

strated activation in the primary and secondary taste regions in response to the visual presentation and imagination of food.⁷ Subjective reports of appetite in healthy subjects correlate with activity in the left frontal operculum and insula.⁷

In this case, seizures were specifically triggered by food-related stimuli in the context of hunger. The finding of a seizure focus in the left frontal operculum, adjacent to the tumor, is consistent with the hypothesis that activation of this region by appetite triggered seizure activity that then propagated to the surrounding cortex and was manifest clinically as a motor seizure.

From the Division of Clinical Neurosciences (K.E., S.D., I.R.W.), Western General Hospital, Edinburgh; and Department of Clinical Neurology (C.R.B.), University of Oxford, UK.

Disclosure: Dr. El Bouzidi reports no disclosures. Dr. Duncan has received travel expenses and honoraria for lectures or educational activities not funded by industry; serves on the editorial board of *Epilepsy and Behavior*; has received speaker honoraria from UCB and Eisai Inc.; and receives research support from the British Epilepsy Association. Prof. Whittle has served/serves as an expert medical advisor for Ark Therapeutics and Archimedes Pharma; has received

funding for travel and speaker honoraria from Archimedes Pharma; serves as Associate Editor of the *British Journal of Neurosurgery*, on the editorial board of *Acta Neurochirurgica*, and on the international editorial board of *Neurosurgery*; and receives research support from the Medical Research Council, the NIH (R01 EB004155-01A2 [Co-PI]), Chief Scientist's Office (Scotland), Experimental Cancer Medicine Centre/Cancer Research UK, and the Melville Trust; and has held stock in GlaxoSmithKline. Dr. Butler holds a lectureship funded by the National Institute for Health Research, UK, and has received additional research funding from the Patrick Berthoud Charitable Foundation and the Mrs. Dale Medical Research Foundation.

Received April 7, 2009. Accepted in final form November 16, 2009.

Address correspondence and reprint requests to Dr. Christopher Butler, Department of Clinical Neurology, University of Oxford, Level 6, West Wing, John Radcliffe Hospital, Oxford OX3 9DU, UK; chris.butler@clneuro.ox.ac.uk

Copyright © 2010 by AAN Enterprises, Inc.

1. Nagaraja D, Chand RP. Eating epilepsy. *Clin Neurol Neurosurg* 1984;86:95–99.
2. Senanayake N. Eating epilepsy: a reappraisal. *Epilepsy Res* 1990;5:74–79.
3. Remillard GM, Zifkin BG, Andermann F. Seizures induced by eating. *Adv Neurol* 1998;75:227–240.

4. Robertson WC, Fariello RG. Eating epilepsy associated with a deep forebrain glioma. *Ann Neurol* 1979;6:271–273.
5. Martinez O, Reisin R, Andermann F, Zifkin BG, Sevlever G. Evidence for reflex activation of experiential complex partial seizures. *Neurology* 2001;56:121–123.
6. Navarro V, Adam C, Petitmengin C, Baulac M. Toothbrush-thinking seizures. *Epilepsia* 2006;47:1971–1973.
7. Porubska K, Veit R, Preissl H, Fritsche A, Birbaumer N. Subjective feeling of appetite modulates brain activity: An fMRI study. *Neuroimage* 2006;32:1273–1280.

Meet the New *Neurology*[®] Editor-in-Chief at the Annual Meeting

All AAN meeting attendees are welcome to meet the new Editor-in-Chief, Robert A. Gross, MD, PhD, FAAN, and the Associate Editors of the journal. This event is running in conjunction with the 62nd Annual Meeting of the American Academy of Neurology. For more information on the Annual meeting, visit <http://www.aan.com/go/am10>.

Breakfast With the *Neurology*[®] Editors

Thursday, April 15, 2010

7:00 AM–9:00 AM

Metro Toronto Convention Centre

255 Front Street West

Toronto, Ontario M5V 2W6

Room 714B

AAN Membership is Your Ticket to Annual Meeting Savings

If you're planning on attending the 62nd American Academy of Neurology Annual Meeting in Toronto, April 10 through 17, 2010, you should know that an AAN membership can save you nearly \$400 on Annual Meeting registration, program fees, and education products—that's just about the cost of membership dues alone!

On top of that, AAN membership provides additional benefits and savings you can enjoy throughout the year, such as deep discounts on education programs and products, tools for your practice, free advocacy training opportunities, free subscriptions to *Neurology*[®] and *Neurology Today*[®], and much more.

Renew your 2010 membership today at www.aan.com/dues or call (800) 879-1960 or (651) 695-1940 (international)! Early registration deadline ends March 10, 2010.

**If you are not an AAN member, join today! Visit www.aan.com/membership or call (800) 879-1960 or (651) 695-1940 (international) to become a member and take advantage of the member-only discounts on Annual Meeting registration and course fees.*