

## BENIGN EEG PATTERNS: IS THERE MORE TO LEARN?

**Prevalence of Benign Epileptiform Variants.** Santoshkumar B, Chong JJ, Blume WT, McLachlan RS, Young GB, Diosy DC, Burneo JG, Mirsattari SM. *Clin Neurophysiol* 2009;120(5):856–861. **OBJECTIVE:** There are numerous distinctive benign electroencephalographic (EEG) patterns which are morphologically epileptiform but are non-epileptic. The aim of this study was to determine the prevalence of different benign epileptiform variants (BEVs) among subjects who underwent routine EEG recordings in a large EEG laboratory over 35 years. **METHODS:** We retrospectively studied the prevalence of BEVs among 35,249 individuals who underwent outpatient EEG recordings at London Health Sciences Centre in London, Ontario, Canada between January 1, 1972 and December 31, 2007. The definitions of the Committee on Terminology of the International Federation of Societies for EEG and Clinical Neurophysiology (IFSECN) were used to delineate epileptiform patterns (Chatrian et al. A glossary of terms most commonly used by clinical electroencephalographers. *Electroenceph Clin Neurophysiol* 1974;37:538–48) and the descriptions of Klass and Westmoreland [Klass DW, Westmoreland BF. Nonpileptogenic epileptiform electroencephalographic activity. *Ann Neurol* 1985;18:627–35] were used to categorize the BEVs. **RESULTS:** BEVs were identified in 1183 out of 35,249 subjects (3.4%). The distribution of individual BEVs were as follows: benign sporadic sleep spikes 1.85%, wicket waves 0.03%, 14 and 6 Hz positive spikes 0.52%, 6 Hz spike-and-waves 1.02%, rhythmic temporal theta bursts of drowsiness 0.12%, and subclinical rhythmic electrographic discharge of adults in 0.07%. **CONCLUSION:** The prevalence of six types of BEVs was relatively low among the Canadian subjects when compared to the reports from other countries.

## COMMENTARY

One of the most common consultations that occurs in an epileptologist's clinic involves an unexpected or anomalous EEG report; for instance, a physician sends a patient with a classic syncopal episode for an EEG and then is taken aback when the results are consistent with, or even diagnostic of, epilepsy. The uneasy physician, who sensibly hesitates to make the diagnosis of epilepsy, sends both the patient and the EEG to an epileptologist, who finds a benign paroxysmal or sharp pattern on the tracing. Seventy years after the discovery of the EEG and its widespread validation as a useful diagnostic procedure, why does this happen? There are several potential reasons, including: (1) shrinking time to train doctors to read EEGs, within modern, busy neurology residency programs, (2) the inherent subjectivity of pattern recognition applied to a complex signal, and (3) the undoubtedly ambiguous appearance of various morphological patterns, some of which can even give pause to the experienced electroencephalographer.

Current problems in misreading EEGs may have their origins in the initial applications of electroencephalography—a procedure that was enthusiastically received in the 1940s and 1950s as a revelatory diagnostic tool for conditions from psychosis to migraine. Early clinical EEG studies associated certain EEG patterns with specific diseases, producing pub-

lished reports such as: “EEG patterns in \_\_\_\_\_ [insert name of disease],” with little or no use of an appropriate comparison population and sometimes with no direct knowledge of the patients being recorded. This practice led to overblown claims for the diagnostic specificity of the electroencephalogram. In addition, unusual or puzzling symptoms sometimes were linked with morphologically sharp or paroxysmal patterns thought to suggest the presence of epilepsy. A 1963 paper, for example, reported the findings of 5,000 consecutive EEGs, describing 253 patients with “psychomotor variant” waves (i.e., rhythmic temporal theta discharges), associated with “ictal symptoms, such as fainting attacks, crying spells, rage attacks” and other “epileptoid” [sic] phenomena (1). In the early 1950s, an EEG with 14 and 6 positive spikes was even produced by defense attorneys as purported evidence of an organic brain disorder to explain a homicide by their client (2).

The EEG patterns included in the study by Santoshkumar et al., reviewed here, are the most common and easily characterized EEG variants initially thought to be associated with epilepsy, but which eventually proved to be present just as often in healthy people. The fact that these patterns are infrequently identified in routine recordings probably has contributed to their durability as a speciously abnormal finding. These benign EEG variants were debated and identified as controversial patterns, and today standard EEG textbooks include them as normal patterns and as variants only insofar as they are not seen in the majority of EEGs. The study by Santoshkumar et al. does nothing to add yet another nail in the coffin of the

pathogenicity of benign epileptiform variants, as it is simply a survey of their incidence in an unselected population of patients referred to an outpatient EEG laboratory. As such, the study's greatest value may lie in the implications about the methods used in contemporary, routine EEG recording and reading.

The prevalence figures the investigators reported are remarkably low, ranging from 0.07% for subclinical rhythmic electrographic discharges of adults (SRDA) to 1.85% for benign sporadic sleep spikes (i.e., benign epileptiform transients of sleep, or BETS). In comparison, in 1977 White et al. found BETS (also known as small sharp spikes) in 20% of consecutive patients referred for diagnostic EEGs for a variety of symptoms and disorders (3). They also studied a cohort of healthy young teens as a comparison group and discovered BETS in 24% of them. Similarly, in 1966 Lombroso et al. prospectively studied the EEGs of 212 healthy young teens, looking for 14 and 6 positive spikes, which they called "ctenoids" (4). Their subjects were deliberately prevented from sinking into any but the lightest sleep stages by loud noises during the recording. Using this unusual protocol, an amazing 58% of the teens were said to show the pattern.

Early EEGs were recorded with referential montages, generally using linked ears or unilateral ear references. Most of the benign patterns under consideration have widespread, shallow fields and are generally poorly seen or not seen at all with the bipolar montages that have more commonly been in use in recent years. The EEGs showing a high incidence of 14 and 6 positive spikes recorded by Lombroso et al., for example, were performed exclusively with referential montages.

As the pathological basis for these patterns became increasingly discredited, EEG assessments simultaneously changed. The EEGs reviewed by Santoshkumar et al. date from 1972 to 2007, so the great majority of them were read by expert electroencephalographers who most likely accepted the benign nature of the discharges. Thus, these experts may not have felt impelled to take the time to enter into the database ev-

ery pattern not seen in a so-called typical or normal EEG; thus these benign epileptiform variants may often have been ignored. Because of the retrospective nature of the study, the readers presumably were not specifically reviewing the EEGs looking for these patterns.

The problem of misleading or incorrect EEG interpretation has not entirely been solved by the eventual clarification of these variants, however. Many erroneous EEG interpretations do not arise from these patterns, but rather from more subtle over-reading of sharply formed normal patterns. Among a group of patients with clinically obvious syncope or proven pseudoseizures, Benbadis et al. reviewed 15 EEGs that had been read as containing epileptiform activity (5). Only one EEG showed wicket spikes, while other activity which was spuriously interpreted as epileptiform consisted of hypnagogic hypersynchrony, hyperventilation induced slowing, or most commonly, simply fluctuations of sharply contoured background activity. The answer to such misinterpretations can be solved only through better education in EEG pattern recognition and assessment—during and after basic neurological training.

by Donna C. Bergen, MD

## References

1. Gibbs FA, Rich CL, Gibbs EL. Psychomotor variant type of seizure discharge. *Neurol* 1963;13:991–998.
2. Schwade ED. Matricide with EEG evidence of thalamic or hypothalamic disorder. *Dis Nerv Syst* 1953;14:18.
3. White JC, Langston JW, Pedley TA. Benign epileptiform transients of sleep. Clarification of the small sharp spike controversy. *Neurol* 1977;27:1061–1068.
4. Lombroso CT, Schawartz IH, Clark DM, Muench H, Barry J. Ctenoids in healthy youths. Controlled study of 14- and 6-per-second positive spikes. *Neurol* 1966;17:1152–1158.
5. Benbadis SR, Tatum WO. Overinterpretation of EEGs and misdiagnosis of epilepsy. *J Clin Neurophysiol* 2003;20:42–44.