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## Short communication

## Unilateral 14 and 6 Hz positive bursts \*

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**Summary** We describe the unilateral occurrence of 14 and 6 Hz positive bursts in successive EEGs in a 25-year-old woman following surgical resection of a left parietal arteriovenous malformation which had caused a left parieto-temporal intracerebral hematoma.

This is only the second reported case of unilateral 14 and 6 Hz positive bursts. This could represent either a normal pattern seen unilaterally because of a skull defect or be a manifestation of neuronal damage.

**Key words:** 14 and 6 Hz positive bursts; Unilateral bursts; Breach rhythm

Since their original description by Gibbs and Gibbs (1951), it is now generally agreed that 14 and 6 Hz positive bursts represent a benign pattern, seen most commonly during drowsiness and light sleep, with a peak incidence between 13 and 15 years (Lombroso et al. 1966). They are most prominent over the posterior temporal areas and seen best with wide interelectrode distances. They can be asynchronous and asymmetric, but unless infrequent, are bilateral (Klass and Westmoreland 1985). We report a patient who after a craniotomy for removal of an arteriovenous malformation (AVM) showed frequent, strictly unilateral, high amplitude 14 and 6 Hz positive bursts ipsilateral to the skull defect.

**Case report**

A previously healthy 25-year-old right-handed woman presented in July 1987 with headaches; head CT scan showed a left parieto-temporal intracerebral hemorrhage with slight mass effect. The clot was evacuated following a left parieto-temporal craniotomy. Postoperatively, she had a mild right hemiparesis and a dysphasia which subsequently resolved. An angiogram revealed a small AVM (2×2.5 cm) in the left parietal region, 1 cm deep to the cortical surface. This was resected in April 1988 without complication. Her last follow-up was in September 1990, when she had a normal neurological examination and no seizures on phenytoin. Her head CT then revealed

encephalomalacia in the posterior portion of the left temporal lobe and posterior basal portion of the left parietal lobe.

**EEG studies**

The recordings were made on 21-channel instruments with electrodes placed according to the international 10–20 system, and utilizing referential, longitudinal bipolar and transverse bipolar montages. In all recordings, both the ipsilateral ear and Cz were used as references. In the last recording, an additional neck-chest reference was used. The first EEG, performed in April 1988, had a waking background of 10 Hz, better developed over the right hemisphere, persistent polymorphic delta activity in the left temporal area, left anterior and midtemporal spikes, a left centro-midtemporal breach rhythm, and a few unilateral left-sided 14 and 6 Hz positive bursts seen mostly during drowsiness and light sleep. A repeat study in February 1990 was unchanged with unilateral left-sided positive bursts. A 2 h recording performed in March 1990 revealed more than 200 positive bursts seen exclusively over the left hemisphere. On a neck-chest reference, the amplitude of the discharges varied between 40 and 120  $\mu$ V and were maximal at T5 (Fig. 1). Most of the bursts had a frequency of 14 Hz, although a few 6 Hz positive bursts were also seen.

Computerized spectral analysis was done on the right and left central and posterior temporal areas referenced to a neck-chest electrode. Filter bandpass was 0.1–50 Hz with a sampling rate of 200 Hz. Twenty-four 2.5 sec artifact-free epochs were selected during deep drowsiness for power spectral analysis. Fast Fourier transformation was applied to each of the epochs and the transformed data averaged to produce a final spectral analysis of the 60 sec sample. The total and relative power of the spectrum in the delta (1–3.9 Hz), theta (4.0–7.9 Hz), alpha (8.0–12.9 Hz), beta<sub>1</sub> (13.0–17.5 Hz) and beta<sub>2</sub> (17.6–25 Hz) bands were calculated. We found a higher absolute power over the left-sided derivations with a left/right ratio varying across frequency bands between 2.2 and 3.5 at the central area and between 1.2 and 2.3 at the posterior temporal location. The

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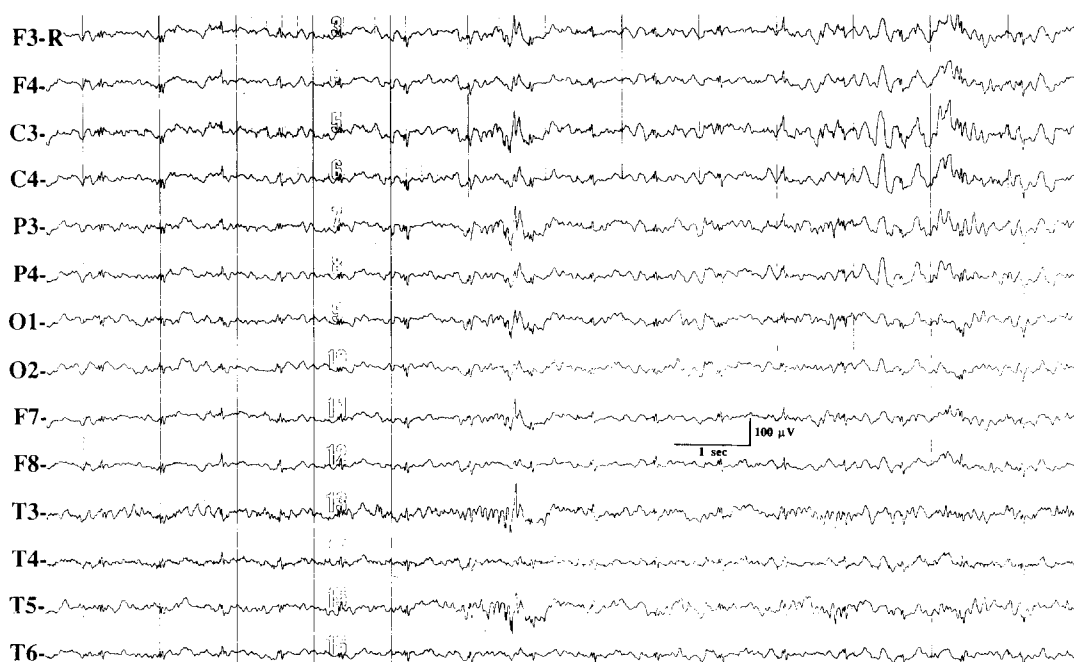


Fig. 1. EEG in deep drowsiness showing unilateral 14 Hz positive burst over the left hemisphere. These discharges are widely distributed with a maximum amplitude over the posterior temporal area. R = neck-chest reference.

relative power over right- and left-sided derivations was similar across frequency bands.

## Discussion

This case is of interest because of the strictly unilateral 14 and 6 Hz positive bursts seen on repeated EEG recordings. There is only one previous well documented case report of unilateral positive bursts in a 33-year-old man who underwent a right temporo-parietal craniotomy for evacuation of a posterior parietal hematoma following a penetrating fragment wound (Wyler and Chatrian 1972). His record showed 14 and 6 Hz positive bursts mostly confined to the side of the craniotomy although rarely seen synchronously over the opposite hemisphere but with a much lower amplitude. The authors described the case because of its unusual EEG features but did not offer an explanation for its occurrence.

The presence of unilateral positive bursts in these cases may result from one or more of the following: (1) these discharges can occasionally be present strictly unilaterally; (2) they are seen unilaterally because of the skull defect; (3) the unilateral occurrence of these bursts is a manifestation of neuronal damage.

In the series of Gibbs and Gibbs (1951), 9% of 201 patients with positive bursts had unilateral discharges but no data were provided about the number of discharges seen in those records. This is important because their rate of occurrence varies amongst individuals from a high of 1 in every 6 sec of drowsiness or sleep to a low of 1 in 30 min (Lombroso et al. 1966). It has been our experience and that of others (Klass and Westmoreland 1985) that cases with unilateral discharges have but a few bursts in their records. It appears unlikely, given the number of discharges seen in this patient and their persistent unilaterality on successive recordings, that this finding represents a normal variant.

Since both reported patients had a craniotomy with bursts ipsilateral to the skull defect, they may be enhanced unilaterally by increased current flow due to the skull defect and parenchymal loss. This finding was not previously described in the records of adult patients with skull defects (Cobb et al. 1979). In addition, a breach

rhythm is usually confined to 1 or 2 electrodes with a sharp fall-off in amplitude (Cobb et al. 1979). In this patient, the 14 and 6 Hz positive bursts had a wide distribution maintaining their maximal amplitude over the posterior temporal derivations. Cobb et al. (1979) found that in patients with skull defects, the amplitude of the normal alpha and beta activity was never increased by a factor of more than 3-fold when compared to the side with an intact skull, and that these rhythms were always detectable on the non-operated side. In this patient, the spectral analysis revealed that the absolute power in the beta<sub>1</sub> band on the left was approximately 2-fold greater than that on the right side. With discharges of up to 120 μV in amplitude, one would expect the discharges to be seen also on the non-operated side.

Unilateral discharges could be a manifestation of neuronal damage. Both reported patients with this finding had intracerebral hemorrhages in the parieto-temporal area. Although Gibbs and Gibbs (1951) suggested that 14 and 6 Hz positive bursts are of subcortical origin, their generator is still unknown. Depth recordings revealed that the bursts are better developed and of higher voltage in depth leads while maintaining the same frequency and configuration as in scalp recordings (Walker and Marshall 1963; Niedermeyer et al. 1967). If these bursts were subcortical in origin, they could be seen unilaterally in cases with cortical damage as a release phenomenon from cortical inhibitory mechanisms, as suggested by Grossman (1952). If they were cortical in origin (Metcalf 1963) with diencephalic/brain-stem interactions, then a disinhibitory mechanism from deep structures or an excitatory mechanism to cortical structures could explain their unilateral occurrence.

Although no final conclusion can be reached concerning this finding, it suggests that structural lesions affecting specific cortical or subcortical structures could activate the occurrence of 14 and 6 Hz positive bursts.

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