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Clinical Observations

Burst Suppression Pattern on Electroencephalogram Secondary to Valproic Acid–Induced Hyperammonemic Encephalopathy

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ABSTRACT

BACKGROUND: Valproic acid may induce hyperammonemic encephalopathy. Various electroencephalogram (EEG) abnormalities have been documented in association with this condition, but not burst suppression, an abnormal EEG pattern that is associated with severe encephalopathy. **METHODS:** Serial EEGs, clinical observations, and laboratory findings were analyzed. **PATIENT DESCRIPTION:** This 13-year-old girl with autism and intractable epilepsy experienced increased seizures; her valproic acid dose was increased and other antiepileptic drugs were administered. She became lethargic, and her EEG showed a burst suppression pattern. Her ammonia concentration was increased to 101 μmol/L and her valproic acid level was increased to 269.9 mg/L. Valproic acid was discontinued and carnitine was administered. Subsequently she became more alert, her ammonia concentration decreased, and her EEG changed from a burst suppression pattern to a continuous pattern. Within three days, she was back to her baseline level of functioning. **CONCLUSIONS:** Valproic acid-induced hyperammonemic encephalopathy can produce a burst suppression EEG patternin the patient's.

Keywords: antiepileptic drug adverse effects, burst suppression EEG, hyperammonemia, hyperammonemic encephalopathy, valproic acid

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Valproic acid may induce hyperammonemic encephalopathy, a condition characterized by acutely impaired consciousness with confusion and lethargy, focal or bilateral neurological symptoms or signs, and increased seizure frequency. These initial signs can progress to ataxia, stupor, and coma. Electroencephalogram (EEG) findings in individuals with valproic acid-induced hyperammonemic encephalopathy include generalized slow waves, often frontally predominant, and frontal-maximal triphasic waves.[1-8] Spikes or sharp waves have also been reported, with varying distributions.[1,2,4,6] These findings may be a reflection of the seizure diathesis that led to treatment with valproic acid and may be present both before the hyperammonemic encephalopathy and after it has resolved. Status epilepticus has also been documented.[5,9] Burst suppression, an EEG pattern that occurs in association with severe encephalopathies, has not been previously described as a consequence of hyperammonemic encephalopathy. We describe a girl with a burst suppression pattern on EEG because of valproic acid-induced hyperammonemic encephalopathy.

Patient Description

This 13-year-old girl with intractable epilepsy experienced an increased seizure frequency manifesting with head drops, and her valproic acid dose was increased to 1575 mg twice per day. Her medical history was significant for autism and behavioral problems for which she received risperidone. She was not taking any other antiepileptic medication. At her baseline she was ambulatory and spoke a few words.

Two weeks after the increase in the valproic acid dose, she had an episode of emesis at home after which she was poorly responsive and experienced frequent seizures. She was admitted to another hospital and there was given 10 mg of diazepam rectally followed by 4 mg of lorazepam; she was then loaded with 20 mg/kg of fosphenytoin followed by an additional 6 mg/kg of fosphenytoin. Because the seizures persisted she was given 10 mg/kg of phenobarbital.

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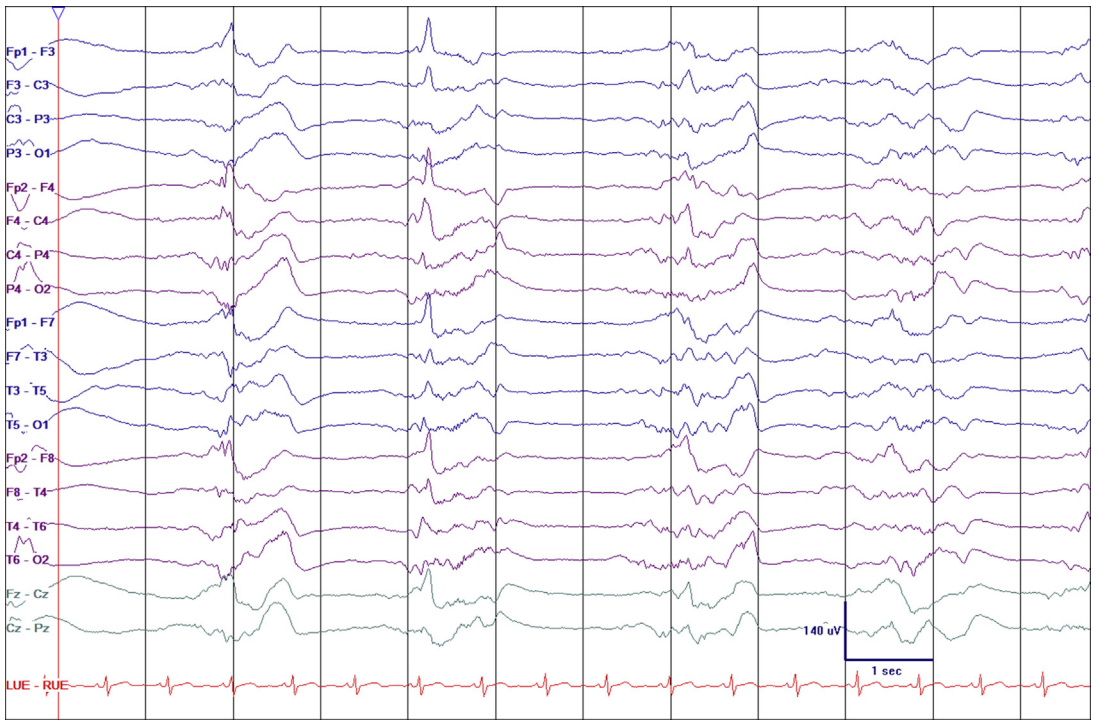

FIGURE 1.
Electroencephalogram recorded on the day of admission showing a burst suppression pattern. (The color version of this figure is available in the online edition.)

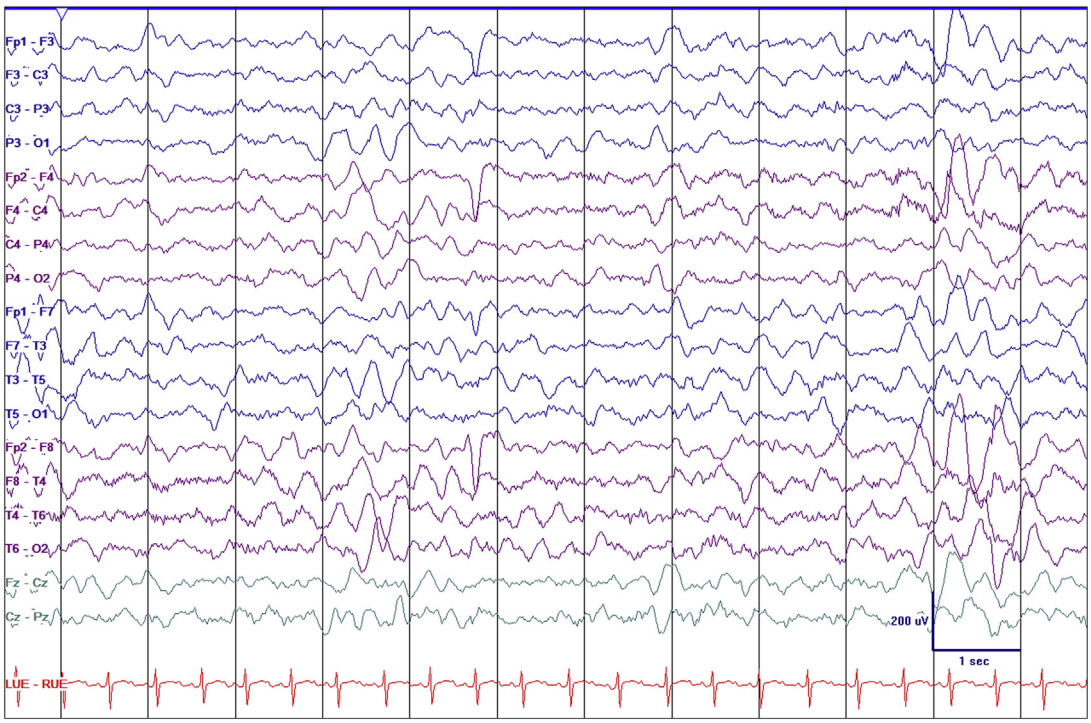

FIGURE 2.
Electroencephalogram recorded during wakefulness, after the burst suppression pattern had resolved and she had returned to her baseline neurological status. The EEG is continuous, but there is generalized background slowing. (The color version of this figure is available in the online edition.)