

2009-10-21

Hyperventilation-Induced Tetany: A Case Report and Brief Review of the Literature

Daniel Schneider

University of Massachusetts Medical School

Let us know how access to this document benefits you.

Follow this and additional works at: https://escholarship.umassmed.edu/neurol_bull

Repository Citation

Schneider D. Hyperventilation-Induced Tetany: A Case Report and Brief Review of the Literature.

Neurological Bulletin 2009;1:11-13. https://doi.org/10.7191/neurol_bull.2009.1002. Retrieved from https://escholarship.umassmed.edu/neurol_bull/vol1/iss1/3

Creative Commons License



This work is licensed under a [Creative Commons Attribution-Noncommercial-Share Alike 3.0 License](https://creativecommons.org/licenses/by-nc-sa/3.0/).

This material is brought to you by eScholarship@UMMS. It has been accepted for inclusion in Neurological Bulletin by an authorized administrator of eScholarship@UMMS. For more information, please contact Lisa.Palmer@umassmed.edu.

NEUROLOGICAL BULLETIN

FEATURING ARTICLES BY TRAINEES IN NEUROLOGY & NEUROSCIENCE

Hyperventilation-Induced Tetany: A Case Report and Brief Review of the Literature

Daniel Schneider

Department of Neurology
University of Massachusetts Medical School, Worcester, MA

The consultation for a paroxysmal event is a common diagnostic challenge for neurologists. Diagnoses such as seizure and transient ischemic attack are often in the differential and this can have significant implications for the patient, including limitations on the ability to drive, time lost performing multiple tests, and even the prescribing of unnecessary medications. The ability to recognize relatively benign syndromes before an extensive and unnecessary work-up is begun is therefore imperative in the ideal management of a patient.

In this report, I present the case of a healthy 22 year old woman who presented to the Emergency Department (ED) of the University of Massachusetts Medical Center following a low speed car accident. The preliminary differential diagnosis from the ED staff was seizure vs. conversion disorder, and a neurological consultation was requested to help guide the work-up and disposition of the patient.

Case Report

The patient was a 22 year old, healthy, right-

handed female who reported that she was driving earlier in the day when suddenly she experienced paresthesias in her hands and feet. These rapidly spread over her entire body, including her face. About 30 seconds after the paresthesias began, she found that she was unable to move her legs, then arms, and then her entire body. When she began to lose movement of her legs, she turned her car into a parking lot and continued at a low speed until she hit a guard rail at the far side of the lot. She denied any loss of consciousness or confusion after the event. She was told by a bystander that her speech was “funny,” and she believed he meant it was slurred but was uncertain. She denied incontinence or tongue-biting. She denied anxiety or panic prior to episode. She experienced some nausea and vomiting in the AM and noted, “I think I was hung over from last night”. She then elaborated that she drank about four beers and smoked some marijuana at a concert the night before and explained that this was unusual for her since she rarely drank that much or used marijuana.

There was no significant medical or psychiatric history. Her only medication was a birth

Correspondence to Daniel Schneider: daniel.schneider@umassmemorial.org

Keywords: calcium, Chvostek’s sign, hypocapnia

control pill for years. She denied any allergies to medications. She currently lived with her mother and was finishing a BA in business management. She had a long-term boyfriend who was her sole sexual partner. She denied any history of abuse or dependence on alcohol or illicit drugs. She did drink alcohol on occasion but rarely drank more than 3 beers and only about once a week on a weekend. Also she smoked marijuana rarely at social events. She denied tobacco use or other drug use. Her only significant neurological family history was ALS in her grandfather.

Her physical exam was largely unremarkable. She was afebrile and her vital signs were normal. She was appropriately dressed and well-groomed. Her neck was supple, her heart and lung exam were normal. She was alert and oriented with intact cognitive functions. Her cranial nerves were unremarkable, strength was full with intact bulk and tone and no abnormal movements noted, reflexes 2+ with downgoing toes, and gait, coordination and sensation were also normal.

Metabolic panel, including calcium, phosphorus and magnesium, was normal. Her blood count was normal. A urine HCG was negative. An EKG was performed that revealed sinus tachycardia at 104 bpm.

The most pertinent, and ultimately most revealing, test was forced hyperventilation. She was asked to hyperventilate for three minutes and after about one minute she began to experience the same paresthesias she had reported earlier and shortly thereafter experienced a spastic flexion of both hands and feet in a pattern characteristic of tetany. She reported that all these symptoms exactly replicated her presenting complaint. On further exam, she also had a positive Chvostek's sign on the left.

Discussion

Hyperventilation can be defined as a state in which breathing in excess of metabolic requirements results in hypocapnia.¹ Many medical and psychiatric conditions can lead to this condition, and there is a long literature examining the body's response to hypocapnia.¹⁻³ The individual manifestations of hypocapnia vary widely, but symptoms can include paresthesias in the face, trunk, and extremities, fasciculations, and tetany, among others.

Tetany can be understood as a hyperexcitability of the axons of peripheral nerves leading to the generation of repetitive discharges;³ it is physically manifested by spasms of the hands and feet (carpopedal), either bilaterally or unilaterally.^{1,2} The most common cause is decreased calcium ion concentration, but the literature reports numerous cases of normocalcemic tetany in the context of hyperventilation. The prevailing notion is that this is due to alkalosis causing a change in the relative amounts of bound versus free calcium ions in the plasma.³ This interpretation has been supported by the frequent observation that normocalcemic patients with tetany still present with Chvostek's sign or a positive Trousseau test, both generally considered indicative of low calcium. However, this theory has come under fire in recent years, as some have argued that other factors such as magnesium deficiency,⁴ hypoglycemia,⁵ or malfunction at the brainstem reticular formation unrelated to ionic imbalance⁶ also may be playing a role.

Once the diagnosis of hyperventilation and tetany is made, the next step is to evaluate the patient for causative factors. Many disorders have been implicated in hyperventilation and hypocapnia, including respiratory diseases (including asthma), left ventricular failure, pulmonary emboli, chronic pain, aspirin overdose, anxiety and panic states, prolonged

talking, pyrexia, pregnancy, and the second half of the menstrual cycle.^{1,7,8} Additionally, and of most relevance to my patient, there have been case reports and studies of patients presenting with hyperventilation and resulting tetany in the context of headaches, presumably due to changes in breathing in reaction to the discomfort.⁹

In my patient, the diagnosis was made when a trial of hyperventilation reproduced her symptoms; the presence of a Chvostek's sign while normocalcemic supported this interpretation. In some patients, attacks of hyperventilation-induced tetany are common (spasmophilia), and a chronic hyperventilation syndrome can develop. This is frequently associated with anxiety, but an underlying organic etiology can also be the cause.¹⁰ The treatment of this syndrome is often directed toward identifying and treating the underlying cause, but at least one small study argues that carbamazepine may be effective.⁶

In the case of my patient, the diagnosis was hyperventilation leading to paresthesias and normocalcemic tetany. This syndrome was most likely brought on by the combination of her headache and any lasting metabolic changes brought on by the alcohol the night before. As this was the only time she had ever had these symptoms, she was advised to avoid drinking to excess and to follow-up with us or her primary care doctor should the symptoms recur.

References

1. O'Sullivan G, Harvey I, Bass C, Sheehy M, Toone B, Turner S. Psychophysiological investigations of patients with unilateral symptoms in the hyperventilation syndrome. *Br J Psychiatry* 1992;160:664-667.
2. Kukumberg P. Side-specific electromyographic differences in tetany. *Lancet* 1991;337:1607.
3. Macefield G, Burke D. Paraesthesiae and tetany induced by voluntary hyperventilation. Increased excitability of human cutaneous and motor axons. *Brain* 1991;114 (Pt 1B):527-540.
4. Fehlinger R, Seidel K. The hyperventilation syndrome: a neurosis or a manifestation of magnesium imbalance? *Magnesium* 1985;4:129-136.
5. Anantharaman V. Hypoglycaemic tetany--a case report. *Singapore Med J* 1988;29:524-525.
6. Kukumberg P. Hyperventilation tetany: effect of carbamazepine. *J Neurol Neurosurg Psychiatry* 1991;54:937.
7. Gardner WN, Bass C, Moxham J. Recurrent hyperventilation tetany due to mild asthma. *Respir Med* 1992;86:349-351.
8. Moss PD, McEvedy CP. An epidemic of overbreathing among schoolgirls. *Br Med J* 1966;2:1295-1300.
9. Narchi H. A child with headaches and abnormal movements. *Eur J Pediatr* 2000;159:219-221.
10. Sarma RN, Rao KB. Tetany: An Analysis of Fifty Cases. *J Indian Med Assoc* 1963;41:588-598.

Disclosure: the authors report no conflicts of interest.

All content in Neurological Bulletin, unless otherwise noted, is licensed under a Creative Commons Attribution-Noncommercial-Share Alike License <http://creativecommons.org/licenses/by-nc-sa/3.0/> (ISSN 1942-4043)