



Point of view

What is the difference between tetanus and tetany?

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Here is a common dialog between a physiology teacher and an undergraduate student during a physiology examination:

Teacher: What is the difference between tetanus and tetany?

Student: Tetanus is a disease caused by *Clostridium tetani* whereas tetany is a consequence of hypocalcemia.

Teacher: You are correct.

My comments: While there is nothing really wrong with the above dialog, it reads as though tetanus and tetany are two different things. My point is, in a mechanistic sense, there is little difference between tetanus and tetany but this is not commonly noted. Let me explain.

The clinical condition "tetanus", caused by *Clostridium tetani*, is characterized by painful muscle spasms and rigidity; this is because the toxin tetanospasmin which *C. tetani* produces is a powerful inhibitor of the release of glycine, an inhibitory neurotransmitter, from Renshaw cells in the spinal cord. When this happens, unrestrained high frequency discharge of alpha motor neurons results in sustained skeletal muscle contractions that we call tetanus.

Experimentally, for example with a frog sciatic nerve-gastrocnemius muscle preparation, we "tetanize" the muscle by stimulating the sciatic nerve supplying the muscle (or the muscle itself) at high frequencies (called tetanizing frequencies); in other words, we are exciting α -motor neurons supplying the muscle at high frequencies. Strictly speaking, a muscle is said to be tetanized completely if there is no relaxation between successive contractions. To be tetanized, skeletal muscle needs to be stimulated at a frequency greater than or at least equal to the reciprocal of the contraction period in seconds.

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Hypocalcemia is said to increase neuromuscular excitability by reducing the magnitude of depolarization necessary to initiate changes in the Na and K conductance that produce an action potential [1]. The carpopedal spasm that occurs in hypocalcemia is evidence of increased neuromuscular excitability and must be due to increased discharge of motor neurons supplying the corresponding muscles. Indeed, Mullin and colleagues [2] have demonstrated the occurrence of opisthotonus, pleurothotonus, and rigidity of abdominal musculature in experimental dogs in which hypocalcemia was induced by injecting calcium poor solutions into the cisterna magna. Thus, the neuromuscular consequences of tetanotoxin and hypocalcemia are similar.

Conflict of interest: the author is the editor of Medical Physiology Online.

References:

1. Ganong WF. Chapter 2. Excitable Tissue: Nerve; In: *Review of Medical Physiology*, 22nd edition, Mc Graw Hill Co, New York, 2005.
2. Mullin FJ, Hastings AB, and Lees WM. Neuromuscular responses to variations in calcium and potassium concentrations in the cerebrospinal fluid. *Am J Physiol* 1938; 121: 477-481.

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Conflict of interest: E.S.Prakash invited me to review this manuscript. He was my student from 2001 through 2006. We have worked and published several articles together.

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Correction (incorporated 30 Jan 2008):

The correct citation of the paper by Mullin FJ et al should be *Am J Physiol* 1938; 121: 719-727, and not *Am J Physiol* 1938; 121: 477-481 as mentioned in the original article above. I thank P Pavithran, Student advisor for Medical Physiology Online for alerting me of this error.
