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 forceful muscle contractions that we call tetanus.
What is the difference between tetanus and tetany?

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 and the contraction is forceful and sustained. For this to happen, the muscle needs to be stimulated at a frequency equal to the reciprocal of the contraction period in seconds.

Hypocalcemia increases neuromuscular excitability by reducing the amount of depolarization necessary to initiate changes in the Na and K conductance that produce an action potential [1]. Thus, the carpopedal spasm that occurs in hypocalcemia is evidence of increased neuromuscular excitability and is 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.

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References:

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Reviewer's comments:
In agreement with the author, I would like to add that though hypocalcemia is a common cause of tetany, an excess of phosphate (high phosphate-to-calcium ratio) could also trigger muscular spasms. Milk-and-alkali tetany is an example of this imbalance.

Goetz AA. Milk-alkali syndrome with jaundice and tetany. California Medicine 1958; 89: 136-139
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**Comments by the Guest Editor:**

1. The description of tetanizing frequency could be improved. You may say: the muscle needs to be stimulated at a frequency greater than or at least equal to the reciprocal of the contraction period in seconds.

2. Tetanic contractions need not be forceful as all motor units in a muscle need not fire at the same time. They are sustained.

3. The description of the mechanism of hypocalcemic tetany is confusing. I think it is due to an increase in sodium channel permeability as a consequence of hypocalcemia.

**Conflict of interests:** The author E.S. Prakash was my student from 2001 through 2006. We have worked and published several articles together.

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