

## &gt; THE PATIENT

56-year-old man

## &gt; SIGNS &amp; SYMPTOMS

- Increased heart rate
- Weakness
- Intense sweating
- Horseradish consumption

## &gt; THE CASE

A 56-year-old physician (CUL) visited a local seafood restaurant, after having fasted since the prior evening. He had a history of hypertension that was well controlled with lisinopril/hydrochlorothiazide.

The physician and his party were seated outside, where the temperature was in the mid-70s. The group ordered oysters on the half shell accompanied by mignonette sauce, cocktail sauce, and horseradish. The physician ate an olive-size amount of horseradish with an oyster. He immediately complained of a sharp burning sensation in his stomach and remarked that the horseradish was significantly stronger than what he was accustomed to. Within 30 seconds, he noted an increased heart rate, weakness, and intense sweating. There was no increase in nasal secretions. Observers noted that he was very pale.

About 5 minutes after eating the horseradish, the physician leaned his head back and briefly lost consciousness. His wife, while supporting his head and checking his pulse, instructed other diners to call for emergency services, at which point the physician regained consciousness and the dispatcher was told that an ambulance was no longer necessary. Within a matter of minutes, all symptoms had abated, except for some mild weakness.

## THE DIAGNOSIS

Ten minutes after the event, the physician identified his symptoms as a horseradish-induced vasovagal syncope (VVS), based on a case report published in *JAMA* in 1988, which his wife found after he asked her to do an Internet search of his symptoms.<sup>1</sup>

## THE DISCUSSION

■ **Horseradish's active component** is isothiocyanate. Horseradish-induced syncope is also called *Seder syncope* after the Jewish Passover holiday dinner at which observant Jews are required to eat "bitter herbs."<sup>1,2</sup> This type of syncope is thought to occur when horseradish vapors directly irritate the gastric or respiratory tract mucosa.

■ **VVS commonly manifests for the first time at around age 13 years**; however, the timing of that first occurrence can vary significantly among individuals (as in this case).<sup>3</sup> The afferent aspect of a VVS episode is poorly understood, while the efferent aspect has been elucidated. A transient inhibition of the sympathetic nervous system results in vasodilatation, hypotension, and a temporary increase in vagal tone with bradycardia,<sup>4-7</sup> which leads to cerebral underperfusion and loss of consciousness.

■ **The loss of consciousness** may be caused by an emotional trigger (eg, sight of blood, cast removal,<sup>8</sup> blood or platelet donations<sup>9,10</sup>), a painful event (eg, an injection<sup>11</sup>), an orthostatic trigger<sup>12</sup> (eg, prolonged standing), or visceral reflexes such as swallowing.<sup>13</sup> In approximately 30% of cases, loss of consciousness is associated with memory loss.<sup>14</sup> Loss of

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➤ Vasovagal syncope may have developed as a trait through evolution, although modern medicine treats it as a disease.

consciousness with VVS may be associated with injury in 33% of cases.<sup>15</sup>

■ **The recovery with awareness** of time, place, and person may be a feature of VVS, which would differentiate it from seizures and brainstem vascular events. Autonomic prodromal symptoms—including abdominal discomfort, pallor, sweating, and nausea—may precede the loss of consciousness.<sup>8</sup>

### An evolutionary response?

VVS may have developed as a trait through evolution, although modern medicine treats it as a disease. Many potential explanations for VVS as a body defense mechanism have been proposed. Examples include fainting at the sight of blood, which developed during the Old Stone Age—a period with extreme human-to-human violence—or acting like a “possum playing dead” as a tactic designed to confuse an attacker.<sup>16</sup>

■ **Another theory involves clot production** and suggests that VVS-induced hypotension is a defense against bleeding by improving clot formation.<sup>17</sup>

■ **A psychological defense theory** maintains that the fainting and memory loss are designed to prevent a painful or overwhelming experience from being remembered. None of these theories, however, explain orthostatic VVS.<sup>18</sup>

■ **The brain defense theory** could explain all forms of VVS. It postulates that hypotension causes decreased cerebral perfusion, which leads to syncope resulting in the body returning to a more orthostatic position with increased cerebral perfusion.<sup>19</sup>

■ **The patient** in this case was able to leave the restaurant on his own volition 30 minutes after the event and resume normal activities. Ten days later, an electrocardiogram was performed, with negative results. In this case, the use of a potassium-wasting diuretic exacerbated the risk of a fluid-deprived state, hypokalemia, and hypotension, possibly contributing to the syncope. The patient has since “gotten back on the horseradish” without ill effect.

### THE TAKEAWAY

Consumers and health care providers should be aware of the risks associated with consump-

tion of fresh horseradish and should allow it to rest prior to ingestion to allow some evaporation of its active ingredient. An old case report saved the patient from an unnecessary (and costly) emergency department visit. **JFP**

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