# Clinical Case Report

## Gelastic syncope: An unusual condition

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#### **ABSTRACT**

Gelastic syncope or laughter-induced syncope is a rare disease often misdiagnosed as narcolepsy or cataplexy. We report a 54-year-old man with syncopal episodes. Each episode started after laughter, leading to light-headedness with blurring of vision and loss of consciousness for a few seconds. The episodes resolved spontaneously. The treatment of gelastic syncope is the same as that for neurally mediated syncope.

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#### INTRODUCTION

Syncope is a common clinical problem, accounting for 3.5% of emergency department visits and 6% of hospital admissions annually in the USA. Most episodes are neurally mediated, cardiac or idiopathic. Situational syncope includes post-tussive, micturition, defaecation, swallowing and Valsalva-induced syncope. Laughter-induced syncope, also known as gelastic syncope, is a rare subtype of situational syncope.

#### THE CASE

A 54-year-old man was admitted to our hospital due to recurrent syncopal episodes precipitated by laughter. The first episode occurred one year ago at a social gathering. The episodes became more frequent and the patient had four episodes in the 3 months before admission. According to witnesses, the patient would laugh wholeheartedly, then go completely limp with his eyes closed and would lose consciousness transiently. This was followed by spontaneous recovery in a few seconds. There was no history of convulsions, tongue bite, frothing from the mouth or incontinence during the episodes. The patient had learned to control his laughter and was able to interrupt the progression of his symptoms at the first warning sign of light-headedness.

The patient had diabetes mellitus and hypertension for the past 3 years. He complained of excessive snoring, daytime sleepiness and disturbed sleep. He had never experienced muscle weakness, hallucinations or sleep paralysis during the episodes. There were no diurnal lapses into sleep or sudden, irresistible sleep attacks. He was moderately obese and had nasopharyngeal crowding. The rest of the examination was normal.

The patient was investigated for the cause of these syncopal episodes. Electrocardiography, echocardiography, carotid Doppler, Holter monitoring, tilt-table testing, electroencephalography and MRI of the brain were normal. Polysomnography showed obstructive sleep apnoea with no signs of cataplexy or narcolepsy. Apnoea—

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hypopnea index was 25.9 with the lowest recorded  $O_2$  saturation being 84%. The quality of sleep was poor with sleep efficiency of 44.9%. Repeat polysomnography with automatic continuous positive airway pressure (auto-CPAP) showed complete abolition of apnoea with significant improvement in sleep efficiency to 66.5%. He was advised to take plenty of fluids orally, reduce weight, exercise regularly and use auto-CPAP at night.

#### DISCUSSION

The cause of situational syncope can usually be found by careful history taking.<sup>3</sup> Intense laughter causes repetitive forced expiration in a staccato pattern with a Valsalva-type effect. This increases intrathoracic pressure, decreases venous return and thus reduces the stroke volume. Normally, these changes are compensated by cerebrovascular autoregulation and autonomic reflexes. In neurally mediated syncope, the failure of compensatory mechanisms leads to inappropriate bradycardia and hypotension resulting in reduced cerebral perfusion and a transient loss of consciousness.<sup>4</sup>

It is important to distinguish gelastic syncope from cataplexy.<sup>5</sup> In laughter-induced syncope, a Valsalva-like phenomenon (increased intrathoracic pressure) occurs only when the patient laughs intensely.<sup>6</sup> However, in cataplexy the emotion rather than the physical act of laughing is thought to trigger an episode.<sup>7</sup> While laughter is the commonest trigger for cataplexy, attacks can also be triggered by other emotions such as fear, elation or anger.<sup>7</sup> A cataplectic attack is not followed by loss of awareness.<sup>7</sup> Moreover, a patient cannot prevent cataplectic attacks voluntarily. Also cataplexy/narcolepsy tends to begin in the second decade of life.

Gelastic syncope can be prevented with the same measures used for other neurally mediated syncope. This includes adequate hydration (2 litres of fluid per day), dietary salt supplementation, use of compression stockings, certain physical manoeuvres (gripping of hands and tensing of arms and legs) and avoidance of situations known to produce syncope. Patients refractory to these manoeuvres<sup>8,9</sup> may be given beta-blockers. For patients unresponsive to beta-blockers, midodrine may be considered.

Unlike syncope caused by cardiac or neurological diseases, gelastic syncope does not lead to increased mortality. 10

This case shows the importance of history in arriving at the correct diagnosis for the cause of syncope.

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