

Defecation Syncope – A Poorly Described Phenomenon that should not be at the ‘Bottom’ of Your Differential

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Abstract Syncope is widespread and responsible for three to five percent of US emergency room visits. While clear cardiac or orthostatic factors are identified in a majority of cases, situational syncope is infrequently described in the literature despite case reports dating back to the early 20th century. Here we present a case of an 84 year old with recurrent hypotension and bradycardia related to defecation. We hope to expand the knowledge around this subject and enhance earlier recognition and treatment.

Keywords Syncope, Defecation, Micturation, Autonomic Failure

1. Introduction

Syncope is a widespread phenomenon with an incidence between three and six percent over 10 years[1] and is responsible for three to five percent of US emergency room visits[2]. While clear cardiac factors (including arrhythmias and valvular disease) or orthostatic factors (including drugs and hypovolemia) are identified in a majority of cases many times the cause is not ultimately obvious. Vasovagal syncope, or carotid hypersensitivity, is a common cause of syncope. However, situational syncope is not as well described. Typical triggers include any actions that cause a Valsalva response (expiring against a closed glottis)[3] and include coughing, swallowing, micturation and defecation. While well known, with case reports dating back to the early 20th century[4], defecation syncope is infrequently described in the literature.

2. Case Presentation

Here we present a case of an 84 year old woman with coronary artery disease, hypertension and hyperlipidemia, presented to the emergency department after a syncopal event. She had no history of diabetes mellitus or thyroid disease. A recent Echo showed normal biventricular function. She lived independently.

She had been complaining of significant constipation and was, in fact, at a drug store buying a laxative when she felt

unwell, went to the bathroom and had a syncopal event while straining.

On her arrival she felt well and had little recall of the event. She denied angina, dyspnea, palpitations or infectious symptoms. She did not appear post-ictal. Her blood pressure was 197/78, her pulse was 78 and regular and her oxygen saturation of 98% on room air. There was no significant postural change in her blood pressure or pulse upon standing. She was in no distress.

The remainder of her physical exam revealed abdominal fullness and significant hard stool in her rectal vault. Bloodwork demonstrated normal troponin, TSH and fasting glucose.

Her medications at home included: metoprolol 25 mg orally twice daily, hydralazine 12.5 mg orally three times daily, felodipine 10 mg once daily, ramipril 5 mg orally twice daily, ASA 81 mg orally once daily and simvastatin 40 mg orally once daily. All doses had been stable for several months. Her blood pressure had been somewhat difficult to control, evidenced by the fact that she was on four different anti-hypertensive agents.

ECG obtained in the emergency department showed normal sinus rhythm. Abdominal x-ray revealed large amounts of stool throughout her colon from the rectum to the hepatic flexure “Figure 1”. There were no signs of obstruction and no masses.

Upon stimulation with an enema, the patient promptly became bradycardic (heart rate of 36; “Figure 2”) and hypotensive (blood pressure of <80 mmHg systolic) requiring treatment with intravenous fluids and atropine.

The patient was admitted to the Cardiac Care Unit for cardiac monitoring. During her admission she experienced several further episodes of presyncope while straining to pass bowel movements (see “Figure 3”). An aggressive

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bowel protocol was initiated (with the exemption of any enemas). Due to the difficulty in resolved our patient’s constipation, a computed tomography scan of her abdomen and pelvis was ordered to rule out any obstruction. The CT

confirmed severe constipation with dilation of her colon (maximum diameter 9.5 cm). There were no masses or signs of obstruction.

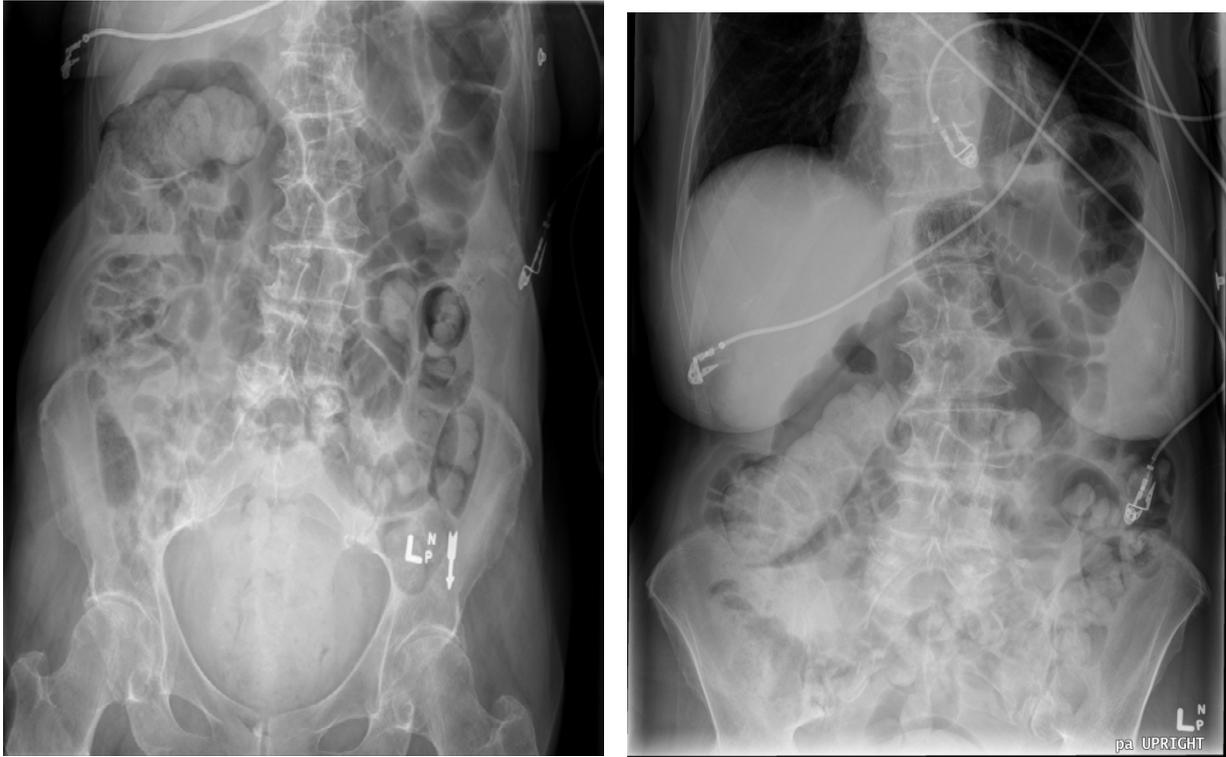


Figure 1. An abdominal X-rays of our patient showing significant fecal loading throughout the colon

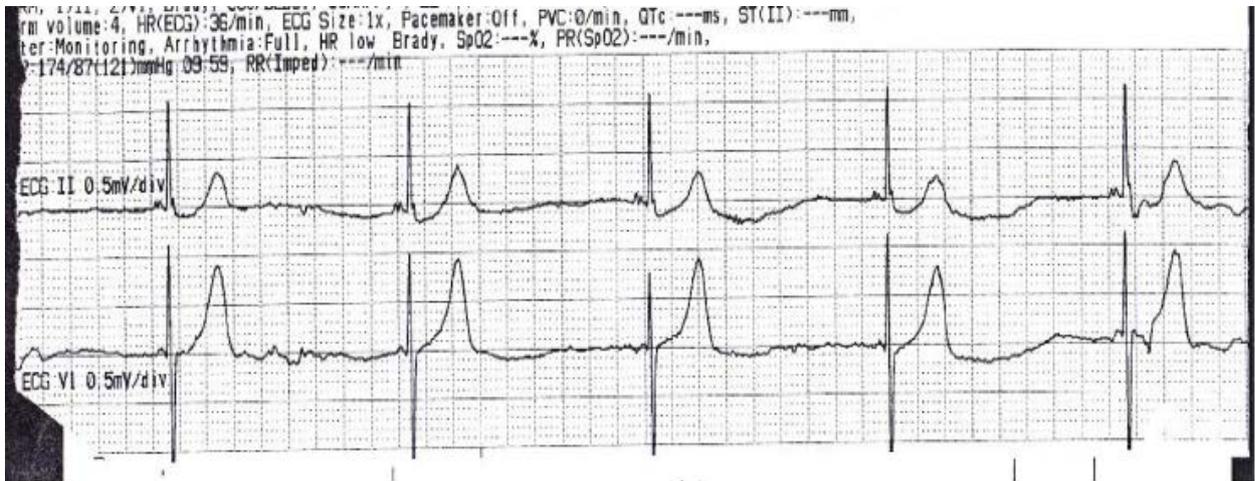


Figure 2. A rhythm strip demonstrating marked sinus bradycardia with a rate of 36 beats per minute

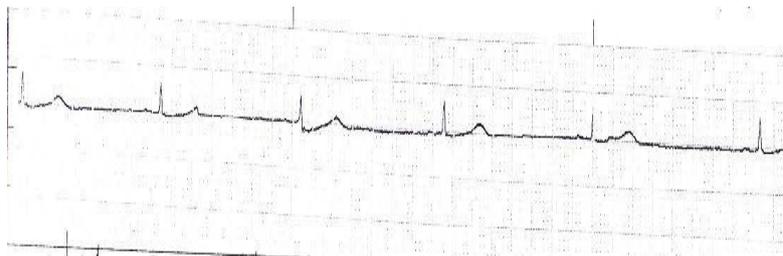


Figure 3. A subsequent episode of bradycardia which occurred during this patient’s admission to the Cardiac Care Unit

The General Surgery service was consulted given the size of her colon. However, they felt she was not at risk for perforation and decided to treat her conservatively.

Her calcium channel blocker (CCB) was also discontinued as constipation is a rare, but well described, side effect of CCB therapy.

She was followed until her constipation had resolved and it was felt safe to discharge her home.

3. Discussion

Neurocardiogenic syncope is common in the general population with a prevalence of up to 3-3.5% [5]. It has a bimodal distribution with peaks in the early teens and the 8th decade of life. The differential diagnosis is large and includes all types of syncope with etiologies including (but not limited to) valvular heart disease, arrhythmias, thromboembolic events, hypovolemia, drugs, autonomic failure and seizures.

Defecation syncope (DS) and micturition syncope (MS) may be more common than previously described owing to the lack of patient reporting for fear of embarrassment or other factors. It has been estimated by some that defecation and micturition may precipitate up to 4% of cardiovascular events and up to 22% of venous thromboembolic events [6].

DS is a phenomenon that is more common in women and may take years to appropriately diagnose [7, 8]. It can occur at any time throughout the day, generally has an associated GI prodrome and tends not to be aggravated by EtOH, all of which are key differences between DS and MS [9].

One often used test for situational syncope is the Tilt Table Test/Head Up Tilt Test [10]. In susceptible patients, this may induce postural symptoms, while the patient is in a carefully monitored environment ("Figure 4"). However, this has been found to be not only cumbersome, but unreliable in the diagnosis of DS [8, 11]. Molnar and Somberg instead propose a heart rate recovery index (HRR) that better predicts those at risk for DS. The HRR involves measuring a patient's heart rate at maximum exercise and then again one minute after exercise cessation. The authors found a significantly greater drop in among those with neurocardiogenic syncope with a sensitivity of greater than 80%.

Diagnosis of this condition is important as it may be a sign of underlying chronic disease. Several studies have suggested that DS may be an early form of autonomic failure [12, 13]. One case series reported central abnormalities in the autonomic nervous system or local abnormalities in the innervation of capacitance vessels of the lower limbs and/or splanchnic vessels in patients with DS [12].

Another series of 20 patients with DS found about half of the patients with "unknown cause" for their syncope to have significant underlying comorbidities including hypothyroidism, panhypopituitarism and severe CAD or multiple co-existing diseases such as one patient with diabetes and SLE. More importantly this group had quite a

high mortality rate of 35% over the 24 months of follow up [13].

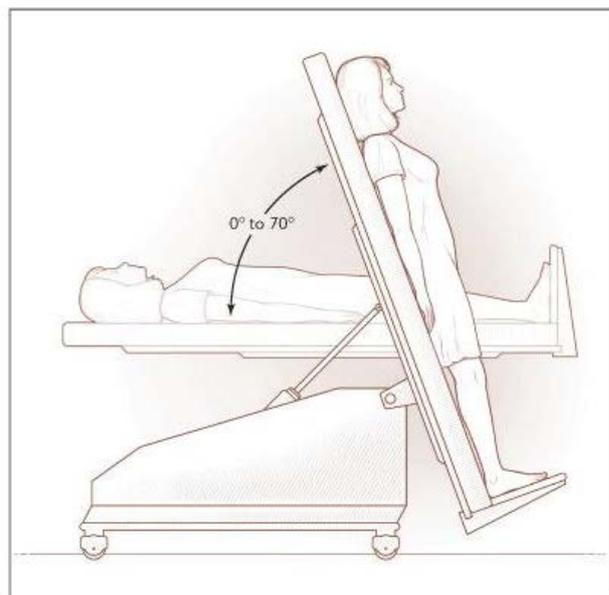


Figure 4. The Tilt Table Test attempts to cause syncope by having the patient lie flat on a special table or bed while connected to ECG and blood pressure monitors. The table then creates a change in posture from lying to standing

Treatment of these patients is difficult and many recur, although less than with other types of vasovagal syncope [8]. Correcting any underlying cause (such as constipation in our case) or treating co-morbid conditions should be the mainstay of therapy.

Constipation is a well known, although infrequent cause of syncope in patients taking calcium channel blockers [14]. This effect is usually associated with non-dihydropyridine calcium channel blockers (and most specifically high doses of verapamil). While the mechanism is not entirely understood, one study showed that in healthy control patients, CCB related constipation may be related to inhibition of the sigmoid myoelectric response to a meal [15]. Other potential mechanisms included reduced colonic transit and increased water absorption. This study, in contrast to the accepted dogma, involved both nifedipine (a dihydropyridine CCB) as well as verapamil (a non-dihydropyridine CCB) and found that the effect was most pronounced with nifedipine.

Our patient was taking felodipine (another dihydropyridine) and the discontinuation of this medication may have aided in the relief of her constipation.

However, the prevalence of constipation in the geriatric community is quite high (up to 41%) [16]. Further, only part of this effect can be attributed to side effects of medications. Other potentially reversible causes of constipation include decreased physical activity and exercise. It is thus paramount to consider the whole patient and look for both pharmacological and non-pharmacological ways to improve constipation.

An offending medication or set of circumstances may not be found in each patient. In those with no clear risk factors of constipation there is some evidence to suggest that they may respond well to steroid supplementation. Others will go on to eventually require permanent pacing[12].

One interesting solution may be to change our toileting position. Squatting (instead of sitting) has been shown to decrease intrathoracic pressure while straining and possibly reduce the risk of syncope[17].

Overall, a careful examination for other signs of autonomic failure (postural hypotension, urinary incontinence, impotence or trouble with body temperature, etc) should be undertaken as well, taking into account the pre-test probability in a given patient. A targeted evaluation for other entities described above may also be reasonable given the risk factors present in a given patient.

Our patient did not have any objective evidence of postural instability, endocrine disease (hypopituitarism, hypothyroidism or diabetes mellitus) or autoimmune disease. We did not pursue a work up beyond that point.

4. Conclusions

Defecation is an important cause of syncope, especially in the elderly population who are more at risk of constipation. There are several potential causes of constipation. They include reversible causes (such as medication side effects and immobility) and non-reversible causes (such as underlying neurological or endocrine disease).

While treatment can be difficult, it can also be an elegant solution to a disruptive and harmful problem for patients. Further, DS may be a clue to more serious underlying pathology and should not be overlooked.

REFERENCES

- [1] Soteriades ES, Evans JC, Larson MG, Chen MH, Chen L, Benjamin EJ and Levy D. Incidence and Prognosis of Syncope. *N Engl J Med* (2002); 347(12): 878-933.
- [2] Shen WK, Decker WW, Smars PA, Goyal DG, Walker AE, Hodge DO, Trusty JM, Brekke KM, Jahangir A, Brady PA, Munger TM, Gersh, BJ, Hammill SC and Frye RL. Syncope Evaluation in the Emergency Department Study (SEEDS). A Multidisciplinary Approach to Syncope Management. *Circulation* (2004); 110:3636-3645.
- [3] Fenton AM, Hammill SC, Rea RF, Low PA, Shen W. Vasovagal Syncope. *Annals of Internal Medicine* (2000); 133(9): 714-725
- [4] Cameron, T. Epilepsy and constipation. *Br. Med.J.* (1911). 2: 920.
- [5] Savage DD, Corwin L, McGee DL, Kannel WB and Wolf PA. Epidemiologic features of isolated syncope: the Framingham Study. *Stroke* (1985); 16:626–629.
- [6] Culic V. Triggering of cardiovascular incidents by micturation and defecation. *International Journal of Cardiology* (2006); 109(2): 277-279
- [7] Park J, Jang SY, Yim HR, On YK, Huh J, Shin D, Kim JH and Kim JS. Gender Difference in Patients with Recurrent Neurally Mediated Syncope. *Yonsei Medical Journal* (2010); 51(4): 499-503
- [8] Bae MH, Kang JK, Kim NY, Choi WS, Kim KH, Park SH, Lee JH, Yang DH, Park HS, Cho Y, Chae S and Jun J. Clinical Characteristics of Defecation and Micturation Syncope Compared with Common Vasovagal Syncope. *PACE* (2012); 35:341-347
- [9] Komatsu K, Sumiyoshi M, Abe H, Kohno, Hayashi H, Sekita G, Tokano T, Nakazato Y and Daida H. Clinical Characteristics of Defecation Syncope Compared with Micturation Syncope. *Circulation Journal* (2010); 74:307-311
- [10] Kenny RA, Ingram A, Bayliss J and Sutton R. Head up tilt: a useful test for investigating unexplained syncope. *Lancet* (1986); 1:1352 - 1355.
- [11] Molnar J and Somberg JC. Neurocardiogenic syncope. *Cardiology* (2009); 114:47-49
- [12] Allan L, Johns E, Doshi M, Kenny RA and Newton JL. Abnormalities of sympathetic and parasympathetic autonomic function in subjects with defaecation syncope. *Europace* (2004); 6:192-198
- [13] Kapoor WN, Peterson J and Karpf M. Defecation Syncope: A symptom with multiple etiologies. *Arch Intern Med* (1986); 146: 2377-2379
- [14] Elliot WJ and Ram CV. Calcium channel blockers. *J Clin Hypertens (Greenwich)* (2011). 13(9): 687-689
- [15] Bassotti G, Calcara C, Annese V, Fiorella S, Roselli P, Morelli A. Nifedipine and verapamil inhibit the sigmoid colon myoelectric response to eating in healthy volunteers. *Dis Colon Rectum* (1998). 41(3): 377-380.
- [16] Van Dijk, KN, de Vries CS, van den Berg, PB, Dijkema, AM, Brouwers JRGB and de Jonh-van den Berg. Constipation as an adverse effect of drug use in nursing home patients: an overestimated risk. *Br J Clin Pharmacol* (1998); 46(3): 255-261
- [17] Sikirov BA. Cardio-vascular Events at Defecation: Are They Unavoidable. *Medical Hypotheses* (1990); 32: 231-233