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Original article

A NARRATIVE REVIEW ON NEURALLY MEDIATED SYNCOPE.

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Abstract.

In this correspondence, we aim to provide a comprehensive analysis of the pathophysiology underlying reflex syncope, encompassing vasovagal syncope, carotid sinus syndrome, and situational syncope. Additionally, we seek to enhance understanding by offering clarification on the terminology employed in this context.

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1. Introduction:

Syncope, a prevalent clinical issue, presents a significant challenge for both cardiologists and general practitioners, with an estimated annual incidence ranging from 1.3 to 2.7 events per thousand individuals [1]. A considerable number of episodes remain unreported or come to the attention of family physicians at a later stage, who, in the majority of instances, provide appropriate reassurance [2]. A higher prevalence of worrisome episodes presents itself in the Emergency Department (ED), constituting approximately 1% of the overall workload [3]. In numerous nations, individuals seeking medical attention for syncope commonly undergo hospitalisation as inpatients after presenting at the Emergency Department (ED). This approach is often associated with substantial expenses and aims to conduct a comprehensive diagnostic evaluation to identify the underlying cause of syncope. However, it is frequently observed that this endeavour does not yield the desired diagnosis [4]. In this suboptimal condition, there is subsequent reappearance of syncope and mortality that is potentially preventable [5].

Syncope, also known as a syncopal episode, is medically characterised as a temporary and self-limiting loss of consciousness (LOC) that occurs suddenly and lasts for a brief period. It is accompanied by spontaneous, prompt, and full recovery. Syncope is clinically defined by the occurrence of global cerebral hypoperfusion [2]. It is imperative to differentiate syncope from other conditions characterised by transient loss of consciousness, such as seizures, hypoglycemia, catalepsy, or aborted sudden cardiac death. In the majority of instances, a comprehensive medical history and pertinent data regarding the precipitating circumstance facilitate the determination of the underlying cause. In order to prevent any potential misunderstandings, it is advised that the term "syncope" should not be employed interchangeably with the phrase "transient loss of consciousness" in medical and academic contexts. The term "presyncope" or "near-syncope" is utilised in medical discourse to characterise a condition that bears resemblance to the prodromal phase of syncope, yet does not culminate in a loss of consciousness [2]. It is imperative to emphasise that uncertainties persist regarding whether the pathophysiological mechanisms underlying presyncope are equivalent to those observed in syncope.

The aetiology of syncope can be broadly categorised into reflex syncope (neurally mediated),

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syncope resulting from orthostatic hypotension (OH), and cardiac syncope, in accordance with the European Practise Guidelines. Reflex syncope (RS) is widely recognised as the predominant aetiology of syncope, regardless of the patient's age or the specific clinical environment. Epidemiological studies have demonstrated that respiratory syncytial infection is prevalent among the younger demographic, while cardiovascular aetiologies are more frequently observed among older individuals [6]. Syncope is widely recognised as a result of cerebral hypoperfusion, stemming from a complex interplay of both central and peripheral mechanisms [3]. Given the variability in both the frequency and severity of syncope among patients with RS, a wide array of treatment modalities exists, encompassing lifestyle modifications as well as cardiac ganglion ablation. In this scholarly review, the aim was to provide a concise synopsis of the assessment and management of Rett Syndrome.

1.1. Introducing Reflex Syncope:

Reflex syncope, also known as neurally mediated syncope, is a temporary loss of consciousness that occurs due to a decrease in systemic arterial blood pressure caused by reflexive dilation of blood vessels or a decrease in heart rate, or a combination of both. The condition is influenced by alterations in the transmission of sensory and motor signals within the autonomic nervous system. Reflex syncope encompasses vasovagal syncope, carotid sinus syncope, and situational syncope [1, 5]. Reflex syncope is a prevalent medical condition with a high incidence rate. Nearly everyone appears to have either encountered or observed an episode. Nevertheless, limited research has been conducted regarding the prevalence of this condition among the general population, especially when compared to other factors that may result in temporary loss of consciousness [6, 7]. The identification and differentiation of reflex syncope from other medical conditions can pose challenges, especially when examining epidemiological data within the medical and academic context. Furthermore, the precise delineation of syncope often exhibits considerable heterogeneity or may be entirely absent, thereby rendering the comparison of data across various studies arduous and occasionally unfeasible [6].

2. Types:

Among the three primary classifications of syncope, namely neurally mediated/reflex syncope, orthostatic hypotensive syncope, and cardiac syncope, it is noteworthy that neurally mediated (reflex) syncope stands as the prevailing type, constituting approximately two-thirds of reported cases. The various subtypes of neurally mediated syncope are delineated as follows:

2.1. Vasovagal syncope:

Vasovagal syncope typically arises in response to abrupt emotional stress, extended periods of sedentary or upright posture, inadequate fluid intake leading to dehydration, or exposure to elevated ambient temperatures. However, it is important to note that vasovagal syncope may also manifest spontaneously, devoid of any identifiable trigger. Vasovagal syncope is the prevailing form of syncope observed in the younger patient population, with a higher incidence among females compared to males. However, it is important to dispel the misconception that this condition exclusively affects the younger demographic, as it can also manifest in the elderly [7]. Typically, it is commonly accompanied by preceding symptoms such as nausea, malaise, fatigue, and diaphoresis, with subsequent slow recuperation. If the episode of syncope persists for a duration exceeding 30 to 60 seconds, it is frequently accompanied by clonic movements and urinary incontinence [7].

2.2. Carotid sinus hypersensitivity:

Carotid sinus hypersensitivity refers to an atypical reaction to carotid massage, primarily observed in individuals aged 50 and above. Spontaneous carotid sinus syndrome is characterised by the occurrence of syncope in response to specific stimuli that elicit carotid sinus activation, including head rotation, head extension, shaving, or the application of a constrictive collar. It is an infrequent aetiology of syncope, accounting for approximately 1% of cases [8]. On the other hand,

induced carotid sinus syndrome is a significantly prevalent condition that manifests as carotid sinus hypersensitivity in patients experiencing unexplained syncope, without identifiable triggers. The anomalous response primarily occurs as a result of carotid massage, rather than spontaneously. In the latter scenario, carotid sinus hypersensitivity serves as an indicator of a compromised sinus node or atrioventricular node, which demonstrates an inability to tolerate any form of inhibition [8]. The affected lymph node is the primary aetiology of syncope, as opposed to carotid sinus hypersensitivity itself, and carotid massage serves as a diagnostic procedure that reveals conduction abnormalities. Therefore, carotid sinus massage is recommended in instances of unexplained syncope, irrespective of contextual stimuli. This examination entails the application of consistent pressure on each carotid bifurcation, located just below the mandibular angle, in a sequential manner for a duration of 10 seconds. The procedure is conducted at the patient's bedside and can be performed with the patient in either supine or erect positions during tiltable testing. Opting for the erect position enhances the test's sensitivity [8].

2.3. Situational syncope:

Situational syncope, also known as reflex syncope, is a medical condition characterised by a transient loss of consciousness. This condition is triggered by specific reflexes that occur in various circumstances, including micturition (urination), defecation (bowel movement), coughing, weightlifting, laughing, or deglutition (swallowing). The reflex may be initiated by a receptor located on the visceral wall, such as the bladder wall, or by the act of straining that leads to a decrease in venous return [8].

2.4. Postexertional syncope:

Although syncope during physical exertion may indicate a concerning cardiac aetiology, postexertional syncope commonly manifests as a variant of vasovagal syncope. When physical activity is discontinued, the return of venous blood to the heart through peripheral muscular contraction is halted [8]. However, the cardiac muscle remains susceptible to the surge of catecholamines triggered by physical exertion, leading to hypercontraction within an unoccupied cardiac chamber. This elicits a vagal reflex. Postexertional syncope can also manifest in individuals with hypertrophic obstructive cardiomyopathy or aortic stenosis, conditions characterised by a small left ventricular cavity that is less capable of accommodating the decreased preload following physical exertion, thereby increasing the likelihood of complete obliteration.

3. Diagnosis:

The diagnosis is primarily established through comprehensive medical history assessment, meticulous physical examination, which may involve carotid sinus massage, electrocardiogram (ECG), and any necessary investigations to exclude the presence of notable structural heart disease [1, 9]. The incorporation of data pertaining to precipitating factors, prodromal manifestations of impending loss of consciousness, observations provided by individuals present during the episode, and subsequent recovery and symptoms experienced after the syncopal event allows for a highly plausible diagnosis to be inferred in numerous instances [2]. Vasovagal syncope is diagnosed based on the correlation between common predisposing factors, prodromal symptoms resulting from autonomic system activation (such as sensations of warmth, abdominal discomfort, sweating, nausea, and paleness), and a post-syncopal phase marked by profound fatigue and the potential for syncope recurrence or intense light-headedness upon early post-episode orthostatic challenge [9]. Myoclonic movements may manifest towards the termination of unconsciousness and necessitate differentiation from convulsive seizures.

Situational syncope is typically readily identified based on the distinct circumstances in which it manifests. Carotid sinus syncope is frequently observed in geriatric individuals, with a notable absence of evident mechanical stimulation of the carotid sinus. The diagnosis of this condition relies on a positive outcome from carotid sinus mas-

sage. In geriatric individuals, the identification of reflex syncope can pose challenges due to the infrequent occurrence of a typical clinical manifestation and the potential overlap of various syncope aetiologies. In this clinical scenario, it is frequently necessary to consider the differential diagnosis of syncope and evaluate the patient's response to the head-up tilt test or carotid sinus massage [10]. In paediatric and adolescent populations, it is crucial to not overlook the potential arrhythmic aetiologies of syncope associated with channelopathies, despite the prevalent occurrence of neurocardiogenic syncope. Therefore, it is imperative to systematically exclude these channelopathies as part of the diagnostic evaluation.

4. Tests for diagnosis:

4.1. Head-up tilt test:

The head-up tilt test employs passive alterations in body position to induce orthostatic stress and ultimately elicit reflex syncope [11]. The prevalence of positive test results in individuals diagnosed with vasovagal syncope is notably elevated, with rates ranging from 65% to 92% [11]. Nevertheless, among individuals exhibiting non-typical manifestations of reflex syncope, the rate of positive outcomes stands at a mere 51-56%, even subsequent to pharmacological stimulation via nitrates or isoproterenol. The diminished diagnostic yield of this test has resulted in a decline in its overall acceptance and utilisation. However, it may be necessary to employ this technique in order to differentiate reflex syncope from delayed orthostatic hypotension, orthostatic tachycardia syndrome, and psychogenic pseudosyncope. It may also be indicated in scenarios where reflex syncope is probable but not definitive, and, infrequently, in distinguishing reflex syncope from seizure [11]. Notably, recent scholarly investigations have emphasised the significance of the head-up tilt test in identifying a predisposition to hypotension in reflex syncope, a condition that could potentially inform the decision-making process regarding pacemaker therapy [12].

4.2. Carotid sinus massage:

The identification of carotid sinus syndrome necessitates the replication of unprovoked symptoms and, furthermore, the presence of clinical characteristics consistent with a reflexive syncope mechanism in patients. In such circumstances, carotid sinus massage typically demonstrates a prolonged period of asystole exceeding 6 seconds. The incidence of carotid sinus syndrome, as delineated in this study, stands at 8.8% when carotid sinus massage was administered subsequent to the primary assessment in individuals aged 40 years and above, presenting with syncope that aligns with a reflex mechanism [12].

4.3. Adenosine triphosphate injection:

The expeditious administration of adenosine triphosphate (ATP) has shown potential in identifying geriatric individuals who are prone to reflex syncope and could potentially derive advantages from cardiac pacing. Nevertheless, the available evidence pertaining to the efficacy of this diagnostic test remains constrained, and its utilisation outside of a research context is not advised [12].

Monitoring strategies:

The acquisition of an electrocardiogram (ECG) during an episode of syncope is considered the definitive method for establishing a symptom-rhythm correlation, thus serving as the most reliable diagnostic approach. In the context of reflex syncope, this type of correlation is especially valuable in the process of excluding alternative causes and evaluating the role of bradycardia/asystole in the occurrence of syncope. The classification of implantable loop recorder-documented syncopal episodes, as proposed by the International Study on Syncope of Uncertain Etiology (ISSUE), holds significant relevance in the differentiation between reflex syncope and various forms of arrhythmic syncope [13]. Prolonged surveillance employing an implantable loop recorder may be warranted in the initial stages of diagnostic evaluation when reflex syncope is suspected but not definitively established in the absence of cardiac pathology. In patients with a heightened risk profile, it may be employed subsequently in the diagnostic process,

in cases where a comprehensive assessment fails to yield a definitive diagnosis.

5. Management:

There are existing clinical guidelines pertaining to the management of syncope. The latest guidelines on the assessment and treatment of syncope, as published by the American College of Cardiology, American Heart Association, and Heart Rhythm Society, are currently undergoing the peer-review process [14]. Readers are directed to the aforementioned documents for an extensive discourse on the clinical management of reflex syncope [14]. Several salient points are delineated herein. The fundamental aspect of reflex syncope management entails ensuring patient comprehension and, whenever feasible, the implementation of measures to circumvent the precipitating factors associated with their syncope episodes. Sufficient hydration, encompassing elevated sodium consumption (assuming no apprehension regarding underlying hypertension), is frequently advised in medical practise. Furthermore, notwithstanding the overall good health of the majority of patients with vasovagal syncope (VVS), it is imperative to consistently contemplate the potential presence of an underlying pathological condition. The avoidance of triggers, whenever feasible, is of utmost importance in the medical context. Therefore, individuals presenting with cough syncope should undergo a comprehensive assessment to determine the underlying aetiology of their coughing episodes [14]. The primary therapeutic approach for cough management involves alleviating symptoms. Additionally, it is often imperative to advise patients to refrain from smoking and minimise exposure to air pollution as these factors can significantly impact respiratory health. When the attainment of full cessation of coughing proves to be unattainable, patients are advised to assume a recumbent position, if feasible, during the occurrence of a paroxysm of coughing. While it is not always foolproof in preventing syncope, this measure does aid in mitigating injuries associated with syncope episodes.

The optimal pacing strategy for vasovagal syncope (VVS) continues to be a subject of ongoing debate within the medical and academic communities. Brignole et al., in the ISSUE-2 and ISSUE-3 trials, observed that patients exhibiting significant bradycardia, as documented by an implantable loop recorder (ILR) during spontaneous clinical occurrences, demonstrated a reduced frequency of recurrences when managed with permanent pacing in comparison to the control group [15]. Moreover, it was observed that cardiac pacing demonstrated optimal efficacy in cases of bradycardia where tilt-table testing did not indicate a significant inclination towards vasodepressor physiology. Based on these observations, it can be inferred that individuals with vasovagal syncope (VVS) who exhibit significant symptoms and documented cardioinhibition during spontaneous events, typically through the use of an insertable cardiac monitor or implantable loop recorder (ILR), and do not show susceptibility to vasodepression as determined by tilt-table testing, may potentially experience positive outcomes from pacing interventions [15]. Nevertheless, the investigation predominantly centred on geriatric individuals (i.e., those aged 40 years and above, with a majority being 60 years or older). Moreover, the prospective evaluation of this observation remains unfinished.

6. List of abbreviations:

ED- Emergency Department
LOC- loss of consciousness
OH- orthostatic hypotension
RS- Reflex syncope
ECG- electrocardiogram
ATP- adenosine triphosphate
ISSUE- International Study on Syncope of Uncertain Etiology
VVS- vasovagal syncope
ILR- implantable loop recorder

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10. References.

1. Ganzeboom KS, Mairuhu G, Reitsma JB, et al. Lifetime cumulative incidence of syncope in the general population: a study of 549 Dutch subjects aged 35–60 years. J Cardiovasc Electrophysiol 2006;17:1172–6.
2. Olde Nordkamp LRA, van Dijk N, Ganzeboom KS, et al. Syncope prevalence in the ED compared to general practice and population: a strong selection process. Am J Emerg Med 2009;27:271–9.
3. Moya A, Sutton R, Ammirati F, et al. Guidelines for the diagnosis and treatment of syncope (version 2009). Eur Heart J 2009;30:2631–71.
4. Bartoletti A, Fabiani P, Adriani P, et al. Hospital admission of patients referred to the Emergency Department for syncope. A single-hospital prospective study based on the application of the European Society of Cardiology guidelines on syncope. Eur Heart J 2006;27:83–8.
5. Del Rosso A, Ungar A, Maggi R, et al. Clinical predictors of cardiac syncope at initial evaluation in patients referred urgently to a general hospital. Heart 2008;94:1620–6.
6. Sutton R, van Dijk N, Wieling W. Clinical history in management of suspected syncope: a powerful diagnostic tool. Cardiol J 2014;21:651–7.
7. Wieling W, van Dijk N, de Lange FJ, et al. History taking as a diagnostic test in patients with syncope: developing expertise in syncope. Eur Heart J 2015;36:277–80.
8. Sheldon R. Syncope diagnostic scores. Prog Cardiovasc Dis 2013;55:390–5.
9. Zaidi A, Clough P, Cooper P, et al. Misdiagnosis of epilepsy: many seizure-like attacks have a cardiovascular cause. J Am Coll Cardiol 2000;36:181–4.
10. Shen WK, Sheldon RS, Benditt DG, et al. ACC/AHA/HRS Guideline for the evaluation and management of syncope. Circulation 2017.
11. Wieling W, Thijs RD, Linzer M, et al. Great expectations: what patients with unexplained syncope desire. J Intern Med 2015.
12. O'Dwyer C, Bennett K, Langan Y, et al. Amnesia for loss of consciousness is common in vasovagal syncope. Europace 2011;13:1040–5.
13. van Dijk N, Boer KR, Colman N, et al. High diagnostic yield and accuracy of history, physical examination and ECG in patients with transient loss of consciousness in FAST: the fainting assessment study. J Cardiovasc Electrophysiol 2008;19:48–55.
14. Daccarett M, Jetter TL, Wasmund SL, et al. Syncope in the emergency department: comparison of standardized admission criteria with clinical practice. Europace 2011;13:1632–8.
15. Brignole M, Arabia F, Ammirati F, et al. Standardized algorithm for cardiac pacing in older patients affected by severe unpredictable reflex syncope: 3-year insights from

the Syncope Unit Project 2 (SUP 2) study. Europace 2015.