

# Syncope in the elderly

## *Síncope em idosos*

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### ABSTRACT

The framework of syncope presents prevalence increases with age, reaching up to 19.5 per 1,000 individuals per year, with morbidity by trauma, fractures, and worsen quality of life. Furthermore, mortality within a year from the syncope episode may reach 33% if the cause is cardiac arrest. Aging-related changes that occur in the cardiovascular reflexes and anatomical changes, in combination with the comorbidities and use of medicines, lead to increased incidence of syncope in elderly people. The identification of the cause of syncope is important for appropriate treatment, with improved quality of life, prevention of recurrence and death. The clinical method enables the diagnosis by as much as 50% of cases, and the arsenal of complementary exams available must be requested according to the clinical reasoning.

**Key words:** Syncope; Aged; Cardiovascular Diseases; Hypotension, Orthostatic.

### RESUMO

*O quadro de síncope apresenta aumento de sua prevalência com a idade, alcançando até 19,5 por 1.000 indivíduos/ano, com morbidade por trauma, fraturas e piora da qualidade de vida. Além disto, a mortalidade dentro de um ano do episódio de síncope pode chegar a 33%, se a causa é cardíaca. As alterações relacionadas ao envelhecimento que ocorrem nos reflexos cardiovasculares e alterações anatômicas, em combinação com as comorbidades e o uso de medicamentos, levam ao aumento da incidência de síncope em pessoas idosas. A identificação da causa da síncope é importante para o tratamento adequado, com melhora da qualidade de vida, prevenção de recorrência e de morte. O método clínico possibilita o diagnóstico em até 50% dos casos e o arsenal de exames complementares disponíveis deve ser solicitado de acordo com o raciocínio clínico.*

*Palavras-chave:* Síncope; Idoso; Doenças Cardiovasculares; Hipotensão Ortostática.

### INTRODUCTION

Syncope is the sudden loss of consciousness, combined with the inability of maintaining a postural tone, with immediate and spontaneous recovery without requiring electrical or chemical cardioversion. Regardless of its cause, this clinical framework is secondary to cerebral hypoperfusion, with short duration (between 10 and 20 seconds). These features allow the differentiation between syncope and other causes of transient loss of consciousness such as seizure and others. However, in the elderly, the syncope scenario can be accompanied by amnesia.<sup>1</sup>

The overall incidence rate of the first report, and not for the first syncope episode, is 6.2 per 1,000 individuals per year. There is an increase in prevalence with

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age, which almost doubles between 70 and 79 years, both in men and women, and near triples in individuals over 80 years of age, reaching the rate of 16.9 per 1,000 individuals per year in men, and 19.5 per 1,000 individuals per year in women.<sup>2</sup> This framework is responsible for 3 to 5% of emergency calls, resulting in the hospitalization of 40% of patients for the average time of 5.5 days.<sup>1</sup>

Two important factors should be considered in the prognosis: the risk of recurrence associated with trauma and risk of death. Recurrence may occur in up to one-third of individuals, and the number of episodes is the strongest predictor of recurrence. Recurrence represents an important impact on quality of life, comparable to that of chronic diseases such as arthritis, depression, and late-stage kidney disease. The main factors associated with poorer quality of life are: being a female, the high prevalence of comorbidities, the number of episodes, and pre-syncope.<sup>1</sup> In addition, there are high costs related to its research, with annual expenditures of 2,400 billion dollars and the average cost of 5,500 thousand and five hundred dollars per hospitalization.<sup>3</sup> Mortality within syncope episodes is mainly associated with structural heart disease, which is more frequent in the older population, and primary electrical heart diseases (channelopathies), which are more frequent in young individuals.<sup>1</sup> Mortality within one year may be 18 to 33% if the cause of syncope is cardiac and between 0 and 12% if the cause is non-cardiac.<sup>4</sup>

## PHYSIOLOGICAL ALTERATIONS IN THE ELDERLY RELATED TO THE SYNCOPE CLINICAL FRAMEWORK

Aging-related alterations related with heart rate, blood pressure, cerebral blood flow, baroreceptors reduced sensitivity, blood volume control in combination with comorbidities, and use of medicines lead to the increased incidence of syncope in elderly people.

Heart rate (HR) and pressure responses to orthostatism occur in three phases: a) an initial response; b) an early stage of stabilization; c) a period of prolonged orthostatism. All three stages are influenced by aging. The maximum increase of HR in the initial phase declines with age, leading to a relatively fixed frequency, regardless of posture. Despite the absence of variation

in the HR response, blood pressure and cardiac output are properly maintained to orthostatism in healthy, active, well hydrated, and normotensives elders due to the reduction in vasodilation and venous filling during the initial phase, as well as the increase in peripheral vascular resistance after prolonged orthostatism. However, in elders with hypertension, heart disease, using vasoactive drugs, anemic, or dehydrated, and those with dysautonomic circulatory disorders, these circulatory adjustments to postural changes are altered, making them vulnerable to postural hypotension and syncope or pre-syncope.<sup>5</sup>

The elderly are vulnerable to a reduction in blood volume by the excessive loss of salt by the kidneys due to plasma Renin and aldosterone decline, increase in plasma atrial natriuretic peptide, and concomitant use of diuretic therapy. Despite the increase in plasma osmolarity, a corresponding increase in thirst is not observed, which leads to chronic hypovolemia. The reduction in blood volume associated with alterations in the age-related diastolic function can lead to a situation of low cardiac output, which increases the susceptibility to orthostatic hypotension and vasovagal syncope.<sup>6</sup>

The diastolic dysfunction is quite prevalent in the elderly, even in the absence of ventricular hypertrophy. Any alteration in total blood volume and venous return can compromise the diastolic filling, even more and worse in the cardiac output. There is a relative increase in the peripheral vascular resistance in the elderly, perhaps compensatory and to maintain venous return and cardiac output. Therefore, the use of vasodilators and diuretics, opposed to this defense mechanism, facilitates the occurrence of syncope events. The same is true in the case of loss of atrial systolic function caused by arrhythmias, especially by atrial fibrillation, of high prevalence in this age group, reducing the output and predisposing to hypotension and syncope.<sup>5</sup>

The cerebral self-regulating system, which remains the circulation constant even in wide range of alterations in the systemic arterial pressure, is compromised in the event of a systemic arterial hypertension and, possibly, in old age, by anatomical alterations that lead to less vascular elasticity. It has been shown that, in this age group, there may be increased cerebrovascular resistance in the postprandial period, despite the accompanying hypotension in the digestive process, which may favor postprandial syncope.<sup>6</sup>

The baroreflex response is hypo sensitive and decreases in older age. The vagal influence on the

cardiovascular system is decreased and, despite the relative increase in sympathetic activity, the compensatory cardio acceleration and drops in blood pressure are compromised. Studies suggest that the loss of elasticity of the stiffness of arteries in old age, especially in the baro sensitive regions of the aorta and carotid, is involved with the reduction of baroreceptors' responses to pressure variations. Anatomical alterations in the sinus node also occur due to the progressive loss of cellularity and in the atrioventricular node, usually attributed to calcification of atrioventricular rings, predisposing the elderly to bradyarrhythmias and conduction disturbances, which are known causative factors of syncope events.<sup>7</sup>

## MAIN CAUSES OF SYNCOPE IN THE ELDERLY

The identification of the cause of syncope presents two important objectives:

- to obtain information about the risk of recurrence of syncope and its prognosis;
- to establish the treatment according to the cause, resulting in improved quality of life, prevention of injuries, and death.<sup>1</sup> The main causes of syncope in the elderly (Table 1) are discussed below.

**Table 1 - Main causes of syncope in the elderly**

Physiopathology classification	Causes
Neuro mediated	Vasovagal Situational Hypersensitivity of the carotid sinus
Cardiac origin	Structural cardiopathy Arrhythmias
Hypotensive syndromes	Orthostatic hypotension Postprandial hypotension
Neurological origin	Autonomic dysfunction Cerebrovascular disease
Endocrinological origin	Diabetic neuropathy Adrenal insufficiency Hypopituitarism

## Neuromediated

### Vasovagal

Before the advent of the tilt test, the vasovagal syncope was considered rare in the elderly. Although its incidence and prevalence are not well known in this population, it is estimated to be responsible for about

one-third of cases, contributing to its bimodal distribution.<sup>8</sup> The term vasovagal was first used by William Gowers in 1907 and described as the mechanism of vasovagal syncope by Thomas Lewis in 1932. The precipitating factors of the classic presentation are orthostatism or prolonged sitting position, emotional stress, pain, heat, venipuncture, postprandial period, use of alcohol, and dehydration.

In the elderly population, there is an association with the use of diuretics and vasodilators. Prodromes such as nausea, vomiting, abdominal pain, cold sweating, paleness, palpitations, and dizziness may occur, being more common in young people. After the loss of consciousness, which has an average duration of 12 seconds, tonic-clonic contractions are of short duration (< 15 s) when they happen. Some clinical findings distinguish vasovagal syncope from the epilepsy framework, with aura, headache, sleepiness, sphincter release, mental confusion, and loss of consciousness for more than five minutes being associated with the latter. The framework of vasovagal syncope presents a benign course in young people; its natural history in the elderly is unknown. There is a reduction in quality of life and association with depression, fatigue, and trauma during the loss of consciousness; however, there is no increased risk for cardiovascular morbidity, and mortality is practically null.<sup>1,8</sup>

The exact mechanism of vasovagal syncope is not well understood. However, it is postulated that the precipitating stimuli result from decreased venous return with vigorous heart contraction with ventricular filling, triggering the Bezold-Jarish reflex. This occurs through the action of mechanoreceptors (C fibers) located in the atriums, ventricles (preferably in the inferolateral wall of the left ventricle), and pulmonary artery manifesting itself with hypotension and paradoxical bradycardia resulting from the increase in the inhibitory activity of receptors and consequent parasympathetic over activity. The sudden interruption of cerebral perfusion after six to eight seconds is required for the loss of consciousness and vasodilation is the primary mechanism.<sup>1,8,9</sup>

### Situational

There are conditions that trigger the Valsalva maneuver, resulting in reduced venous return such as urination, defecation, cough, visceral pain, postprandial,

and carrying weight.<sup>1</sup> The syncope related to urination may occur in those patients with prostatic hypertrophy.

### Carotid sinus hypersensitivity

This is the extrinsic disease of the sinus knot that is characterized by pre-syncope or syncope due to an exacerbated carotid sinus reflex response. The precipitating factors are sudden movements of head and neck, cervical compressions, and even the use of a tie. Its incidence is from 35 to 40 patients/year/million individuals, with prevalence among men (man:woman ratio of 4:1) and more frequent in the elderly, especially diabetics, with carotid or coronary atherosclerosis. It is responsible for up to 20% of the syncope framework in the elderly. The hypersensitivity response of the carotid sinus may present three patterns: a) cardio inhibitory, with asystole for more than three seconds (between 18 and 24% of cases; more frequent in women); b) vasodepressor, with a fall of more than 50 mmHg in the systolic blood pressure (between 10 and 25% of cases); and c) mixed, with both components, cardio inhibitory and vasodepressor.<sup>1,10</sup>

### Cardiac origin

This cause may be responsible for 21 to 34% of syncope in the elderly. Aortic stenosis is the most common structural heart disease associated with syncope in the elderly, which occurs related to efforts or because of vasodilation by drugs or hot baths. Other causes are conditions that result in a decreased cardiac output such as hypertrophic cardiomyopathy, ischemic heart disease, heart failure, aortic dissection, cardiac tamponade, prosthetic valve thrombosis, cardiac tumors, pulmonary hypertension, and pulmonary thromboembolism.<sup>1,11</sup>

Both bradyarrhythmias and tachyarrhythmias may cause syncope as the result of a compromised cardiac output.<sup>1</sup> Elders with associated structural heart disease present a high risk. The most frequent bradyarrhythmias in this context are the sinus bradycardia with cardiac frequency < 40 bpm, sinus pauses  $\geq 3$  seconds, and advanced levels of atrioventricular blockage. Ventricular tachycardia and supraventricular tachycardia are among the tachyarrhythmias with high frequencies.

### Hypotensive syndromes

These frameworks occur due to age-related physiological and pathological alterations that result in syncope. The most prevalent in the elderly are orthostatic hypotension and postprandial hypotension.

The framework of orthostatic hypotension (OH) or postural manifests as falls, dizziness, pre-syncope, or syncope resulting in functional impairment with head injuries, bone fractures, and hospitalization.<sup>1,12</sup> In the elderly, its frequency varies between 5 and 55%<sup>13-16</sup>, with higher rates in those who reside in institutions. The definition of the classic OH is the drop of least 20 mmHg in systolic blood pressure and/or 10 mmHg in diastolic blood pressure within three minutes when taking the orthostatic position due to the decrease in venous return by the damming of up to 1,000 mL of blood in the lower extremities.<sup>1</sup>

There are other OH presentations, as the initial, when there is a drop greater than 40 mmHg with symptoms after taking the orthostatic position for less than 30 seconds, and progressive OH when the drop in blood pressure is gradual, between three and 30 min after taking the orthostatic position without bradycardia (differentiating it from the vasovagal neuro mediated response framework).<sup>1</sup> This framework has predisposing conditions such as dehydration, postprandial period, malnutrition, lack of physical fitness, drug actions, and comorbidities.<sup>1,12,13</sup> However, regardless of these conditions, OH has been associated with increased general mortality due to cardiovascular causes and is considered a predictor of cardiovascular events in the elderly.<sup>13,14,17</sup>

The framework of postprandial hypotension is a common cause of syncope in the elderly with prevalence between 25 and 38%, reaching 67%, mainly in the elderly who reside in institutions. It occurs within two hours after meals, with a drop of at least 20 mmHg in systolic blood pressure, or an absolute value less than 90 mmHg in those with systolic blood pressure of at least 100 mmHg. The pathophysiology is assigned to a sympathetic dysfunction, with inappropriate peripheral vasoconstriction and an insufficient increase in heart rate. The precipitating factors are vasodilating drugs, diets high in carbohydrates, and a high temperature of foods or environment. It can occur in the elderly without comorbidities; however, it is more frequent in elders with Parkinson's disease, diabetics, and hypertensive. This framework can result in falls, syncope, fractures, myocardial ischemia, and cerebrovascular accident; it is also an independent predictor of mortality.<sup>12,18</sup>

## Neurological causes

These causes include autonomic dysfunction frameworks, cerebrovascular diseases, and subclavian steal syndrome.

The autonomic dysfunction can be primary, secondary, or due to an adverse effect of drugs.<sup>11,19</sup> The primary forms occur in central nervous system diseases (such as Parkinson's disease, multiple systems atrophy or Shy-Drager syndrome, Huntington's disease, and Guillian-Barre syndrome) and pure primary dysfunction (Bradbury-Eggleston syndrome - by involvement of the sympathetic peripheral nervous system). The secondary occur because of aging alterations and involvement of the peripheral nervous system by diabetes mellitus, kidney disease, alcoholism, amyloidosis; infections of the nervous system by the Chagas disease, human immunodeficiency virus; metabolic diseases such as deficiency of vitamin B<sub>12</sub>, porphyria; and autoimmune diseases such as rheumatoid arthritis and others less frequent. Drugs such as antihypertensive (diuretics, vasodilators) and antidepressants can also lead to autonomic dysfunction.

The main clinical presentation of these neurological causes is the occurrence of OH, however, other signs of autonomic dysfunction may be present such as impotence, sweating, incontinence or urinary retention, dysfunction of the gastrointestinal tract, and etc.

When there are focal neurological deficits within the framework of cerebrovascular accident, the presentation can be of syncope; however, other manifestations usually accompany this framework.<sup>11</sup>

The subclavian steal syndrome was described in 1969 by Symon and is characterized by the obstruction due to malformation or atherosclerosis of the subclavian artery proximal to the spine origin, which causes retrograde flow in the left vertebral artery. Neurological symptoms such as dizziness, paresthesia, and syncope happen on the occasion of exercises performed by the left arm in one-third of cases; however, patients may present frameworks of transient ischemic attacks.<sup>11,20</sup>

## Endocrinological causes

These causes include autonomic diabetic neuropathy, chronic adrenal insufficiency, and hypopituitarism. These conditions result in OH due to autonomic dysfunction or hypovolemia, potentially leading to syncope. Conditions associated with loss of liq-

uids such as insipidus diabetes and kidney diseases with loss of salt, and conditions associated to vasoactive substances, such as pheochromocytoma and carcinoid syndrome, can also result in syncope.<sup>11</sup>

Other conditions such as convulsive frameworks and acute mental confusion (delirium) in the elderly may be mistaken for syncope, especially when associated with fluctuating levels of consciousness. In addition, there are other manifestations that are commonly diagnosed incorrectly as syncope, namely:<sup>1,5,11,19</sup>

- transient ischemic attacks of vertebrobasilar or carotid origin;
- hypoglycemia, hypoxia, hypercapnia, and other metabolic disorders;
- alcohol and other substances intoxication;
- some forms of epilepsy;
- psychiatric disorders.

## RESEARCH

The initial evaluation, which includes anamnesis, physical examination, and electrocardiogram enables the diagnosis in 23 to 50% of patients.<sup>1,21</sup> Thus, the story should be detailed, featuring every syncope episode, precipitating factors, prodromes, posture in which it occurred, what the patient was performing, and associated symptoms (nausea, skin pallor, cyanosis, sweating, muscle contractions, mental confusion, trauma). Previous records of comorbidities, use of medications, and family history are also important to establish the cause of syncope. There are questionnaires that help in the diagnosis of the cause of syncope, showing 89% sensitivity and 91% specificity for vasovagal syncope.<sup>22</sup> The physical examination must be comprehensive, with blood pressure measurement in the supine position and in three minutes in orthostatism (to detect OH), blood pressure measurement in both upper limbs (to detect likely subclavian steal), cardiovascular system proficient examination, which can identify signs of heart disease and/or arrhythmias.

When the initial evaluation does not enable a diagnosis, additional tests must be carried out guided by the clinical method.

The tilt test has the following indications:

- in the case of an odd unexplained syncopal episode of high risk (with trauma or occupational implications) or recurrent episodes in the absence of heart disease or even with its presence (when other causes have been excluded);

- when it will change the therapeutic approach;
- when the clinical method does not allow a diagnosis requiring the differentiation from epileptic, falls, and dizziness frameworks.

The tilt test came to be used for the diagnosis of vasovagal or unexplained syncope from 1986 and, since then, several publications have demonstrated its utility and reproducibility. It consists of a passive tilt by the patient after a five to 20 minutes period at rest, at an angle between 60 and 70 degrees, for 20 to 45 minutes. The patient should be under electrocardiographic and blood pressure monitoring, and fasting. If the passive phase is negative, the pharmacological drug phase can be performed with intravenous isoproterenol or sublingual aerosol nitroglycerin, at an angle, during the additional 15 to 20 minutes. The positive test can present one of the answers:

- vasovagal or neuro cardiogenic, which allows for three patterns:
  - **vasodepressor**: there is a drop in blood pressure greater than 30 mmHg with no significant change in heart rate;
  - **cardioinhibitory**: the bradyarrhythmia as a sinus pause for more than three seconds or atrioventricular block preceding hypotension;
  - **mixed**: there is hypotension preceding or concomitant to bradycardia;
- dysautonomic response:
  - postural hypotension;
  - gradual and progressive fall in blood pressure; and
- postural orthostatic tachicardizing syndrome, when there is an intolerance to posture with immediate and sustained increase of more than 30 bpm in heart rate or heart rate maintained above 120 bpm with pressure instability. Depending on the protocol used, the sensitivity varies between 51 and 75% and specificity between 92 and 94%; its use has been considered safe.<sup>1,8,9</sup>

In the elderly, the dysautonomic and vasovagal responses are the most common as the cause of syncope.

Another test is the carotid sinus massage, indicated for patients with more than 40 years of age or with a history suggestive of carotid sinus hypersensitivity. The rate of complications of this test is low (0.29%) and due to neurological frameworks. Therefore, it is contraindicated in patients with transient ischemic accident or stroke in the last three months, carotid bruit,

or carotid stenosis. The massage should be conducted for five to 10 seconds, starting on the right because the right carotid sinus is the most sensitive, under heart and blood pressure monitoring, and in a suitable environment. The positive responses may present three patterns, as discussed earlier in this article.<sup>1,10,11</sup>

In the case of history and physical examination suggesting structural heart disease, the echocardiogram must be requested. Cardiac monitoring through the dynamic electrocardiography is indicated on suspicion of arrhythmias. This monitoring can be done through the 24 h Holter system if symptoms with a frequency of at least once a week; or intermittent recording or holter if symptoms occur between one and four weeks. When the patient shows infrequent symptoms, and there is no left ventricular dysfunction, the use of an implantable device is indicated placed in the subcutaneous area, near the precordial region. Because its battery life is of 36 months, it allows for extended monitoring with 35% of diagnostic frequency. There is also the real-time monitoring, which allows the transmission of signals to a central data storing site, electronically transmitted to the attending physician.<sup>1</sup>

For the investigation of patients with syncope and severe ventricular dysfunction or with high pre-test probability, the electrophysiological study is recommended. Through it, the cardiac conduction system can be studied, verifying bradyarrhythmias and the level of blockage, and performing programmed ventricular stimulation, i.e. triggering tachyarrhythmias, which may be the cause of syncope. Thus, despite its low sensitivity and specificity, this invasive exam can identify the cause and guide the implantation of a pacemaker or cardioverter-defibrillator in selected patients.<sup>1,11,21</sup>

The stress test has its intended diagnostic use in the context of syncope during or shortly after the effort, and if there are findings that explain the syncope framework.<sup>1</sup>

Other evaluations, such as biochemical and neurological tests and psychiatric evaluation, may be performed according to the diagnostic hypothesis.

## TREATMENT

The goal of treatment is to improve the quality of life and prevent the recurrence of syncope, fractures, functional decline, and death. Thus, the treatment depends on the cause of syncope, which in the elderly is often multifactorial.

In the case of vasovagal syncope, proper hydration and avoiding precipitating factors are the first and main recommendations. Because prodromes are less frequent in the elderly, it may be difficult to resort to procedures to increase venous return and blood pressure levels such as crossing legs or lying to abort syncope. If the precipitating factor is a medicine, reducing its dose and even suspending its use may be needed. Multicenter studies have shown that the isometric exercises that avoided the recurrence of syncope did not include patients over 70 years of age. However, these exercises should also be encouraged in the elderly population despite limitations by musculoskeletal or neurological comorbidities.

The salt supplementation, which is the proper recommendation for young patients with vasodepressing syncope, is contraindicated in hypertensive elders. The use of elastic stockings in lower limbs in order to increase venous return must be stimulated, but it is not well adhered by patients. The use of class IIa artificial pacemaker is recommended in selected patients with asystolic cardioinhibitory responses, as well as in patients with carotid sinus hypersensitivity with predominantly cardioinhibitory response. There is no evidence of the practice of tilt training (orthostatic training) in the elderly and the beta blockers, used in the past without scientific evidence, are contraindicated in cases of vasovagal syncope.<sup>1,8,23</sup>

If the cause is OH, recommendations such as hydration (two to three liters per day), correction of electrolyte disturbances such as hyponatremia, avoiding precipitating factors, and salt supplementation (10 g of NaCl) if possible, are the most important. The use of fludrocortisone (0.1 to 0.3 mg/day), a mineralocorticoid that leads to sodium retention and volumetric expansion, is a class IIa recommendation. There is a withdrawal in 33% of elders due to hypokalemia, increased blood pressure, edema, and heart failure. The use of midodrine (doses of 5 to 20 mg three times daily) results in peripheral vasoconstriction due to its alpha-agonist action; it is also a class IIa recommendation. However, there is a 25% intolerance rate due to hypertension in the supine position, urinary urgency, pollakiuria, and worsening of angina and other heart diseases. Adopting a high-level headboard (more than 10°) is another recommendation (class IIb) because it prevents nocturnal polyuria, leading to a better distribution of blood volume. Abdominal compression, with the aim of improving the venous return, is also a class IIb recommendation.<sup>1,8</sup>

If the cause is a structural heart disease, the treatment will be according to its etiology and severity of the systolic dysfunction. Patients with bradyarrhythmias with repercussion, without a removable cause, with intra- or infra-hisian blockage, should be subjected to an artificial pacemaker implantation. In the case of supraventricular tachyarrhythmias, drug treatment or ablation, depending on the type of arrhythmia, is recommended. The implantation of cardioverter-defibrillator may be necessary in cases of ventricular tachycardia.<sup>1</sup>

According to the cause of syncope, a joint approach involving Neurology, Endocrinology, and/or Psychiatry may be necessary.

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