



REVIEW ARTICLE

Pathophysiology of neurally-mediated syncope[☆]



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KEYWORDS

Neurally-mediated syncope;
Active standing;
Tilt test;
Baroreflex

Abstract

Introduction: Neurally-mediated syncope (NMS) is defined as a transient loss of consciousness due to an abrupt and intermittent drop in blood pressure (BP).

Objectives: This study describes the putative pathophysiological mechanisms giving rise to NMS, the role of baroreflex (BR), and the interaction of its main haemodynamic variables: heart rate (HR) and BP.

Development: Episodic dysregulation affects control over the haemodynamic variables (HR and BP) mediated by BR mechanisms. During active standing, individuals experience a profound transient drop in systolic BP (SBP) due to the effect of gravity on the column of blood and probably also because of reflex vasodilation. Abnormalities in the BR in NMS could be due to a more profound drop in BP upon standing, or to delayed or incomplete vasoconstriction resulting from inhibited or delayed sympathetic activity.

Conclusions: Sympathetic hyperactivity is present in patients with NMS at rest and before syncope. During active standing or passive tilting, excessive tachycardia may be followed by bradycardia and profound hypotension. Recovery of SBP is delayed or incomplete.

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PALABRAS CLAVE

Síncope neuralmente mediado;
Ortostatismo activo;

Fisiopatología del síncope neuralmente mediado

Resumen

Introducción: El síncope neuralmente mediado (SNM) se define como una pérdida súbita y transitoria del estado de alerta debido a una caída brusca de la presión arterial (PA).

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Prueba de inclinación;
Barorreflejo

Objetivos: Describir los mecanismos putativos fisiopatológicos responsables del SNM, el papel del barorreflejo (BR) y la interacción de sus variables hemodinámicas principales: frecuencia cardíaca (FC) y PA.

Desarrollo: Existe una desregulación episódica en el control de las variables hemodinámicas (FC y PA) mediadas por el barorreflejo. Durante la bipedestación activa existe una caída profunda y transitoria de la PA sistólica (PAS) debida a la acción de la gravedad sobre la columna de sangre y probablemente también a una vasodilatación refleja producida por inhibición del reflejo vasosimpático. Las anomalías del BR en el SNM pueden ser debidas a una mayor intensidad de la caída de la PA al ponerse de pie o a una vasoconstricción retardada o incompleta debido a un reflejo vasosimpático insuficiente o retardado.

Conclusiones: Los pacientes con SNM tienen en reposo y antes del síncope un estado de hiperactividad simpática. Durante el ortostatismo activo o la inclinación pasiva hay taquicardia excesiva seguida de bradicardia e hipotensión severa. La recuperación de la caída de la PAS está retardada o incompleta.

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Introduction

Neurally-mediated syncope (NMS) is defined as a sudden and transient loss of consciousness with spontaneous recovery. It is due to generalised brain hypoperfusion resulting from an abrupt and intense drop in blood pressure (BP).¹ Initial orthostatic hypotension is defined as a short episode (20-30 s) occurring 5 to 10 seconds after standing involving an immediate decrease in systolic BP (SBP) of ~40 mm Hg, and/or diastolic BP of ~20 mm Hg.^{2,3} It is very frequent among young patients and is reported as a cause of syncope in 3.4% of those affected. It has the highest incidence among all causes of situational fainting.⁴ Prevalence of syncope is high in the general population; its incidence shows a bimodal distribution with peaks during adolescence and in patients older than 25. It is slightly more prevalent in women, with a peak of 47% vs 31% in teenage boys.^{5,6}

The Framingham study reported an incidence of syncope which increased in patients older than 70 years, both men and women.⁷ Incidence rose from 5.7 per 1000 person-years in 60 to 69-year-old men to 11.1 in 70 to 79-year-old men.^{8,9} However, in elderly patients, cumulative incidence on syncope is more difficult to obtain, since collecting data is more complicated.^{7,8}

In vasovagal or NMS, there is an intermittent and sudden dysregulation in the activity of the autonomous nervous system which causes a drop in BP, heart rate (HR), and brain perfusion.¹⁰

Orthostatic BP decreases with age, and this occurs in 14% to 20% of all elderly patients.^{2,9} Its pathophysiology has shown to be multifactorial, with baroreflex (BR) dysfunction being the most frequently mentioned putative cause.¹¹ The array of symptoms during orthostatic hypotension manifests more frequently in young adults. This could be explained because a drop in systemic BP causes a more pronounced decline in cerebral blood flow than in elderly adults.¹²

Beginning in adulthood, autonomic preganglionic neurons are lost at a rate of 5% to 8% per decade. This becomes symptomatic when neuronal loss reaches 50%. In elderly adults, a head-up tilt test provokes modifications which

suggest a decrease in central and peripheral autonomic reactivity; changes in BP and HR are more pronounced during active standing than during a passive tilt test. BP drops abruptly, and the increase in HR is higher; BR activity can be clearly observed, although changes are less pronounced in the elderly than in young adults. Elderly adults also display a slight or null increase in HR, an early decrease in BP, and delay in the increase in peripheral resistance and HR recovery, all of which increases the likelihood of vasovagal syncope in this age group.¹³

Furthermore, humoral response to orthostasis changes with age. The renin-angiotensin system seems to be less active in BP regulation during orthostasis, although resting catecholamine values seem to be higher in elderly adults. Similar increases have been observed in young adults in response to orthostasis.¹³

NMS is clinically characterised by initial prodromal symptoms which can manifest up to one minute before the event and include diaphoresis, pallor, nausea, abdominal discomfort, and yawning; these are followed by visual or auditory symptoms and difficulty concentrating, among others.^{10,14}

NMS is a significant cause of morbidity and is responsible for 1% to 2% of all emergency department consultations.¹⁵ Its hospital costs in the United States amount to 2.4 billion US dollars per year.¹⁶ Scores for NMS on quality of life scales indicate that the impact of the disease is similar to that caused by more severe chronic illnesses such as epilepsy.¹⁷

Diagnosis of NMS is fundamentally based on the clinical history and the physical examination, including BP measurement during orthostasis. Currently, there are 2 methods for evaluating the response to postural changes: active standing and the head-up tilt test. The sensitivity and specificity of the head-up tilt test are difficult to determine given the methodological differences in the way that it is administered. The lack of a gold standard makes it difficult to distinguish between normal and abnormal results. However, studies with healthy volunteers and patients with a typical history of neurally mediated syncope have reported a specificity of about 90% and a sensitivity ranging from 32% to 82%.¹⁸ When a facilitating agent is used, sensitivity increases

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