RECURRENT EPISODES OF SYNCOPE IN ELDERLY MAN WITHOUT STRUCTURAL HEART DISEASE

EPISÔDIOS RECORRENTES DE SÍNCOPE EM HOMEM IDOSO SEM CARDIOPATIA ESTRUTURAL

Dr. Raimundo Barbosa Barros Fortaleza – Ceará - Brazil

Prezado Prof Andrés, veja que caso interessante (não é a primeira vez que acontece).Este paciente após toda investigação abaixo foi encaminhado para o Hospital de Messejana para investigação de síncopes recorrentes de causa desconhecida. O colega que o atendeu na admissão realizóu uma simples manobra de compressão dos seios carotídeos (que já devia ter sido realizada) e esclareceu o diagnóstico.
Gostaria de compartilhar com os colegas do foro e chamar a atenção para o valor desta ferramenta, muitas vezes esquecida, na investigação de síncope em pacientes idosos sem cardiopatia estrutural.

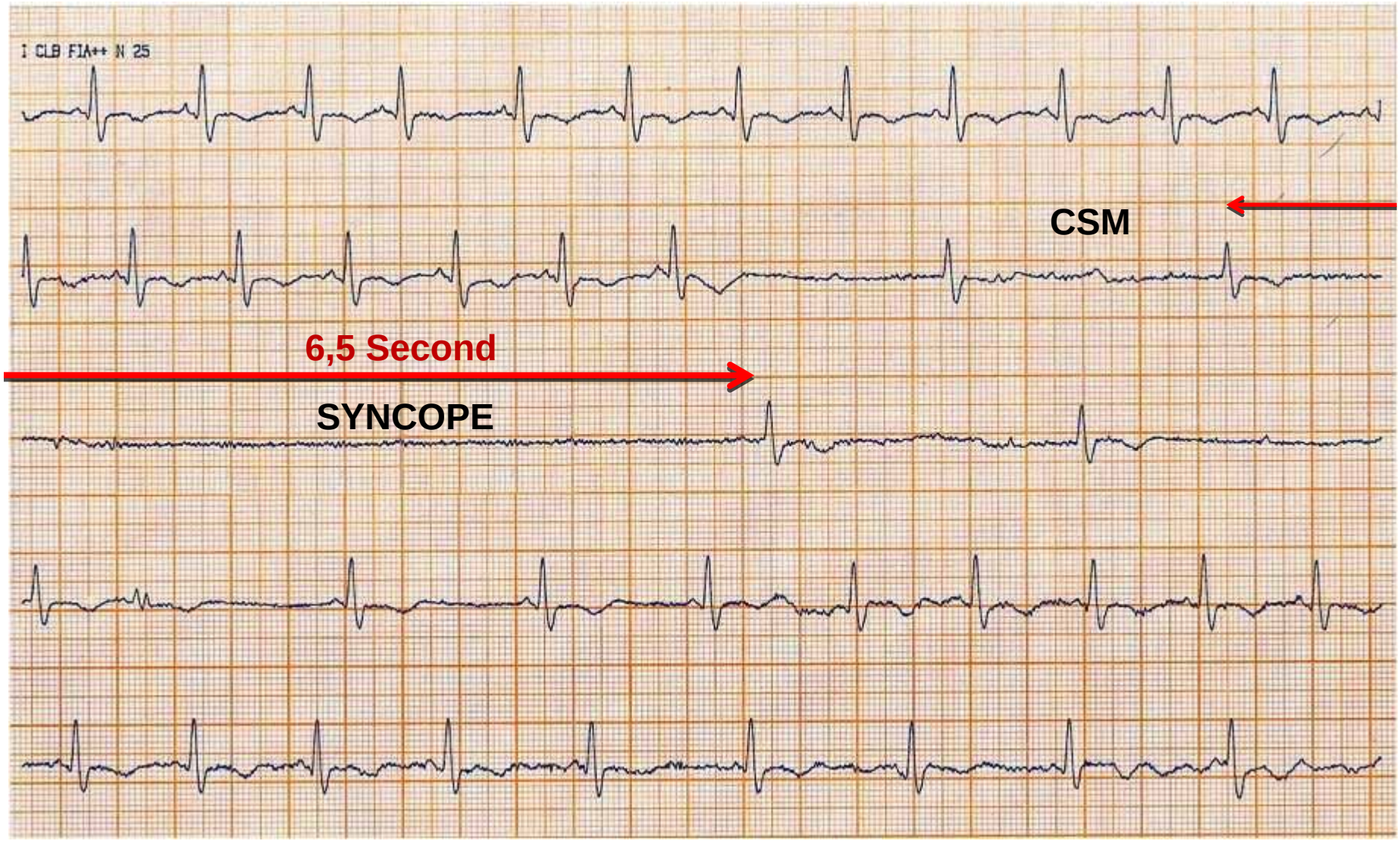
Homem idoso 75a, sem cardiopatia estrutural com síncopes recorrentes e inexplicadas precedidas de curtos pródromos
HOLTER 24h (x2) – Normal;
ETT (x2) – Normais.;
Cintilografia miocárdica – Normal;
Coronariografia – Normal;
TC de crânio – Normal;
EPS –> SNRT – N, Sem taquicardias indutíveis com estimulação programada; HV = 70ms.

Pergunta: Ele é portador da forma cardioinibitória? Vasodepressora o de uma forma mista?
Um abraço
Raimundo Barbosa Barros

Dear Professor. Andrés, see what interesting case (do not the first time that happens).
This old man patient after all following below investigatigation was sent to Messejana´s Hospital for investigation of recurrent unknown origin syncope episodes.

The colleague who served in the admission performed a simple frequently forgotten maneuver: the compression/ stimulation of the carotid sinus or carotid sinus massage *(that already should have been made) and clarified the diagnoses.*
I would like to share with colleagues of the forum and draw attention to the value of this tool, often overlooked in the investigative study of syncope in elderly patients without structural heart disease.
History: 75y.o. old man without structural heart disease with unexplained recurrent syncopes episodes preceded by short prodromos.
HOLTER monitoring 24 (x2) : Normal;
ETT (x2) "Normal;
Myocardic Scintigraphy: "Normal.;
Coronary angiography "Normal.;
Skull tomography: Normal.;
EPS: SNRT N, No tachycardias with programmed stimulation; HV = 70ms.

Question: it is carrying the cardioinhibitory syncope type?; the vasodepressor type? or the mixed type?
A hugs for all Raimundo Barbosa Barros


I CLB FIA++ N 25
CSM
6,5 Second
SYNCOPE

COLLEAGUES OPINIONS

You demonstrated carotid sinus hypersensitivity. A tilt table test might better sort out the associated problem of a vasodepressor component.

Foi demonstrado hipersensibilidade do seio carotídeo. Um teste da cama inclinada seria melhor para esclarecer se o paciente possui um componente vasodepressor associado.

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Respuesta Cardioinhibitoria al masaje de los senos carotideos.

Cardioinibitory response to Carotid Sinus Massage (CSM)

Carlos Rodriguez MD crartuza@hotmail.com

Estimado Andrés: Este caso de Raimundo demuestra que en la evaluación del síncope sigue habiendo dificultades. Es interesante ver los numerosos estudios realizados en este provecto. No es infrecuente observar esto. Hasta ahora la história clínica es lo mas importante (El interrogatorio es diagnóstico en aproximadamente el 50% de los casos). En este caso no está descripta las circunstancias que ocurrían los episodios sincopales. Si la la história clínica nos hace sospechar de hipersensibilidad del seio carotídeo es mandatório el masage del seno carotídeo en ancianos. La compresión del seno carotídeo demuestra la causa del síncope.

El ECG nos va a demostrar si hay o no cardiopatía. Un ECG normal nos descarta cardiopatía. Obviamente tenemos que mirarlo cuidadosamente porque puede existir un síndrome del QT prolongado. Hay que tener registro continuo del ECG y de la presión arterial como hizo Raimundo. Por supuesto, antes debemos descartar que existan soplos carotídeos. El riesgo de complicaciones neurológicas consecuencia del masage del seio carotídeo es mínimo. Nosotros de rutina lo realizamos al comienzo del test de la cama inclinada porque en la posición ortostática se incrementa la sensibilidad (J Am Coll Cardiol 1986; 7:158–162).

Saludos, Oscar Pellizón MD Argentina.

Dear Andrés: The case of Raimundo shows that the evaluation of syncope difficulties remains. It is interesting to see the many studies in this Draft. It is not uncommon to see this. Until to day the clinical history is most important (The interview is diagnosed in approximately 50% of cases). In this case is not described the circumstances that occurred syncopal episodes. If the clinical history leads us to suspect Carotid Sinus Hypersensitivity *is mandatory to Carotid Sinus Massage (CSM) in the elderly. CSM shows the cause of syncope. The ECG will show us whether or not heart disease. A normal ECG rule out heart disease. Obviously, we must to look carefully the ECG because there may be a long-QT syndrome. It should be a continuous recording of ECG and blood pressure as did Raimundo. Of course, we must first exclude any carotid bruits. The risk of neurological complications following the* Carotid Sinus Massage (*CSM) is minimal. We routinely perform the test at the beginning of the Till test because the standing position increases the sensitivity (J Am Coll Cardiol 1986, 7:158-162).Regards, Oscar MD Pellizón Argentina.*

FINAL COMMENTS AND THEORETICAL CONSIDERATIONS
COMENTÁRIOS FINAIS E CONSIDERAÇÕES TEÓRICAS

By Andrés Ricardo Pérez-Riera MD

Carotid Sinus Hypersensitivity (CSH)

Synonyms: Charcot-Weiss-Baker Syndrome - Weiss-Baker Syndrome -Carotid Sinus Syncope - Carotid Sinus Syndrome

According to the current European Society of Cardiology guidelines, CSH is diagnosed when carotid sinus massage elicits ≥3 s asystole, a fall in systolic blood pressure of ≥50 mmHg, or both, with symptoms[1]. The current criteria for CSH are too sensitive, explaining the reported high prevalence of CSH in the general older population.

Although baroreceptor function usually diminishes with age, some people experience hypersensitive carotid baroreflexes. For these individuals, even mild stimulation to the neck results in marked bradycardia and a drop in blood pressure. CSH predominantly affects older males. It is a potent contributory factor and a potentially treatable cause of unexplained falls and neurocardiogenic syncopal episodes in elderly people.[2,3] Yet, CSH is often overlooked in the differential diagnosis of syncope. CSH, orthostatic hypotension, and vasovagal syncope are common conditions that are likely to coexist in patients with syncope and falls[4]. Pathophysiology Hypersensitivity of the carotid sinus body, such that stimulation of the carotid sinus produces hypotension and/or syncope through an exaggerated baroreceptor-mediated reflex involving the nerve of Hering and the medulla Typical triggering factors are shaving, head turning, or tight collars Syncope can occur with or without accompanying bradycardia An important cause of falls and syncope in the elderly; carotid sinus hypersensitivity is rare before the age of 50 Mechanisms responsible for the syndrome are as yet poorly understood

1. **Krediet CT, Parry SW, Jardine DL, Benditt DG, Brignole M, Wieling W. The history of diagnosing carotid sinus hypersensitivity: why are the current criteria too sensitive? Europace. 2011 Jan;13:14-22.**
2. **Parry SW, Steen N, Bexton RS, Tynan M, Kenny RA. Pacing in elderly recurrent fallers with carotid sinus hypersensitivity: a randomised, double-blind, placebo controlled crossover trial. *Heart*. May 2009;95(5):405-9.**
3. **Gillespie LD, Robertson MC, Gillespie WJ, Lamb SE, Gates S, Cumming RG, et al. Interventions for preventing falls in older people living in the community. *Cochrane Database Syst Rev*. Apr 15 2009;CD007146.**
4. **Tan MP, Newton JL, Chadwick TJ, Parry SW. The relationship between carotid sinus hypersensitivity, orthostatic hypotension, and vasovagal syncope: a case-control study. *Europace*. Dec 2008;10:1400-5.**

The carotid sinus reflex plays a central role in blood pressure homeostasis. Changes in stretch and transmural pressure are detected by baroreceptors in the heart, carotid sinus, aortic arch, and other large vessels. Afferent impulses are transmitted by the carotid sinus, glossopharyngeal, and vagus nerves to the **nuclei tractus solitarius** and the **para-median nucleus** in the brain stem. Efferent limbs are carried through sympathetic and vagus nerves to the heart and blood vessels, controlling HR and vasomotor tone. In CSH, mechanical deformation of the carotid sinus (located at the bifurcation of the common carotid artery) leads to an exaggerated response with bradycardia or vasodilatation, resulting in hypotension, presyncope, or syncope.

Baroreceptors are sensors located in the blood vessels of several mammals[1]. They are a type of mechanoreceptor that detects the pressure of blood flowing through them, and can send messages to the central nervous system to increase or decrease total peripheral resistance and cardiac output. Baroreceptors act immediately as part of a negative feedback system called the baroreflex[2]. as soon as there is a change from the usual mean arterial blood pressure, returning the pressure to a normal level. They are an example of a short-term blood pressure regulation mechanism. Baroreceptors detect the amount of stretch of the blood vessel walls, and send the signal to the nervous system in response to this stretch[2].The nucleus tractus solitarius in the medulla oblongata recognizes changes in the firing rate of action potentials from the baroreceptors, and influences cardiac output and systemic vascular resistance through changes in the autonomic nervous system. Baroreceptors can be divided into two categories: high-pressure arterial baroreceptors and low-pressure baroreceptors (also known as cardiopulmonary [3] or volume receptors[4].

1. **Stanfield, CL; Germann, WJ. (2008) Principles of Human Physiology, Pearson Benjamin Cummings. 3rd edition, pp.424.**
2. **Stanfield, CL; Germann, WJ. (2008) Principles of Human Physiology, Pearson Benjamin Cummings. 3rd edition, pp.427.**
3. **Levy, MN; Pappano, AJ. (2007) Cardiovascular Physiology, Mosby Elsevier. 9th edition, pp.172.**
4. **Stanfield, CL; Germann, WJ. (2008) Principles of Human Physiology, Pearson Benjamin Cummings. 3rd edition, pp.430-431.**

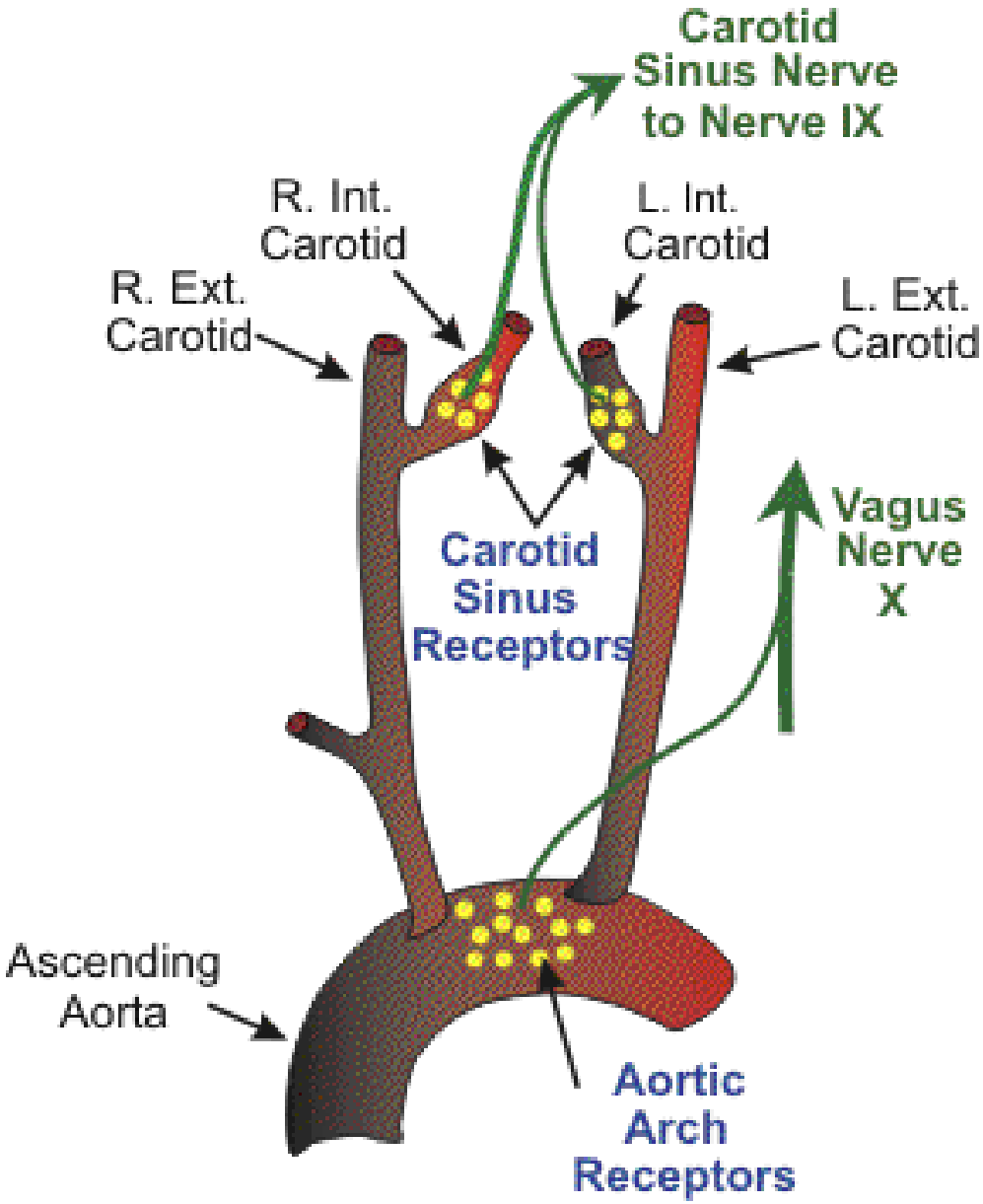
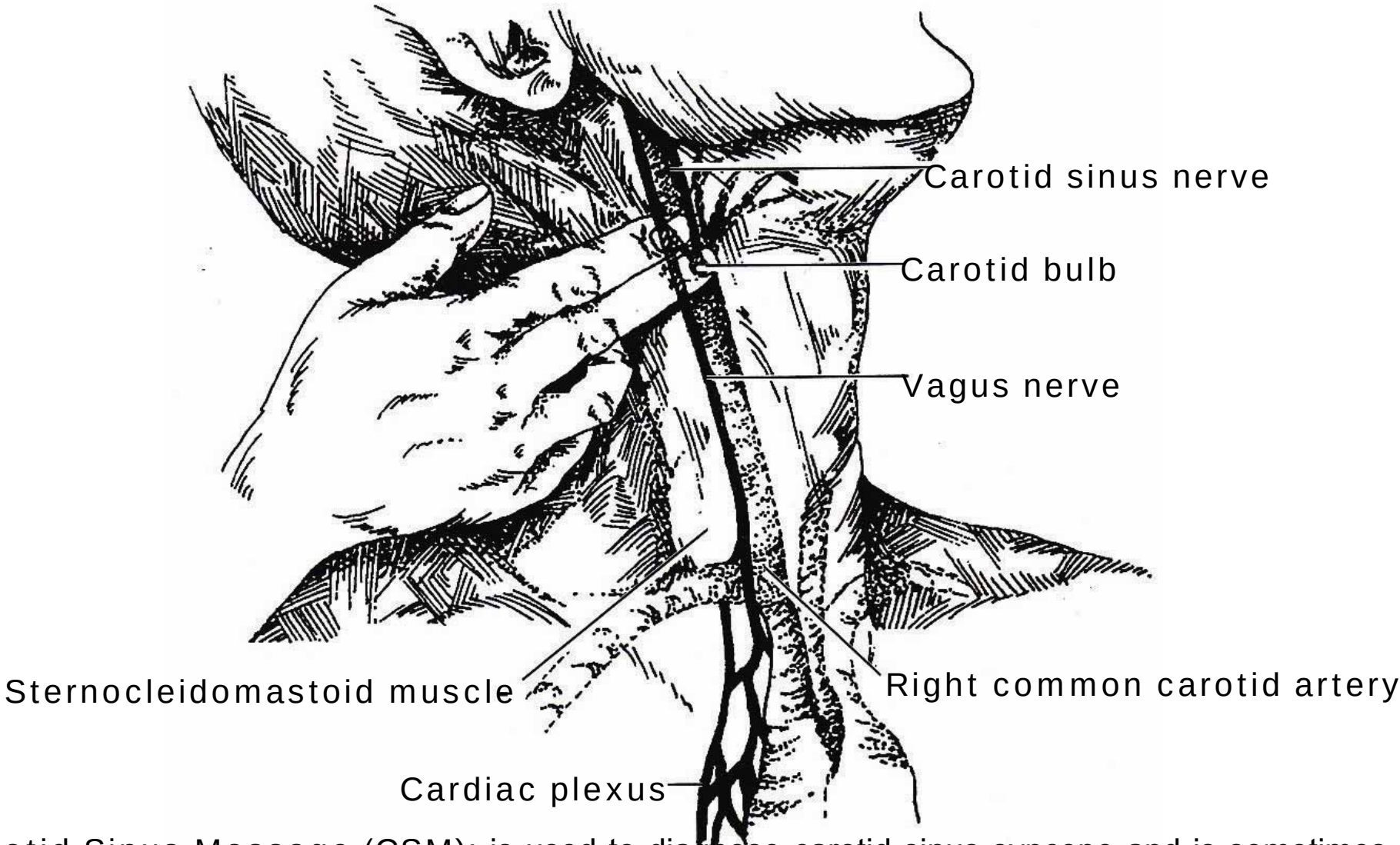


Figure 1. Location and innervation of arterial baroreceptors.

The carotid sinus is a localized dilation of the internal carotid artery at its origin, the common carotid artery bifurcation.



Carotid Sinus Massage (CSM): is used to diagnose carotid sinus syncope and is sometimes useful for differentiating supraventricular tachycardia (SVT) from VT. Like the valsalva maneuver, it is a therapy for SVT. It is less effective than pharmaceutical management of SVT with verapamil or adenosine though is still the preferred first-line of treatment in a hemodynamically stable patient.

Arterial (high-pressure) baroreceptors:
are present in the aortic arch and the carotid sinuses of the left and right internal carotid arteries. The baroreceptors found within the aortic arch enable the examination of the blood being delivered to all the blood vessels via the systemic circuit, and the baroreceptors within the carotid arteries monitor the blood pressure of the blood being delivered to the brain. Arterial baroreceptors are stimulated by pressure changes in the arteries. The baroreceptors can identify the changes in the blood pressure, which can increase or decrease the HR. They are sprayed nerve endings that lie in the tunica adventitia of the artery, not drug-binding molecules as the term *receptor* may suggest. A change in the mean arterial pressure induces depolarization of these sensory endings, which results in action potentials (APs). These APs are conducted to the central nervous system by axons and have a direct effect on the cardiovascular system through autonomic neurons[1]. Hormone secretions that target the heart and blood vessels are affected by the stimulaton of baroreceptors. If blood pressure falls, such as in hypovolaemic shock, baroreceptor firing rate decreases. Signals from the carotid baroreceptors are sent via the glossopharyngeal nerve (cranial nerve IX). Signals from the aortic baroreceptors travel through the vagus nerve (cranial nerve X)[2]. If the arterial pressure is severely lowered, the baroreflex is activated[3]. Baroreceptors respond very quickly to maintain a stable blood pressure, but they respond only to short-term changes. Over a period of 1–2 days, they will reset to a new value[4]. Thus, in people with essential hypertension the baroreceptors behave as if the elevated blood pressure is normal and aim to maintain this high blood pressure. The receptors then become less sensitive to change[5].

1. **Stanfield, CL; Germann, WJ. (2008) Principles of Human Physiology, Pearson Benjamin Cummings. 3rd edition, pp.424-425.**
2. **Bray, JJ; Cragg, PA; Macknight, ADC; Mills, RG. (1999) Lecture Notes on Human Physiology, Blackwell Publishing. 4th edition, pp.379.**
3. **Guyton, AC; Hall, JE. (2006) Medical Physiology, Elsevier Saunders. 11th edition, pp.258.**
4. **Guyton, AC; Hall, JE. (2006) Medical Physiology, Elsevier Saunders. 11th edition, pp.211.**
5. **Levy, MN; Pappano, AJ. (2007) Cardiovascular Physiology, Mosby Elsevier. 9th edition, pp.171.**

Low-pressure baroreceptors

The low-pressure baroreceptors, are found in large systemic veins, in pulmonary vessels, and in the walls of the right atrium and ventricles of the heart[1]. The low-pressure baroreceptors are involved with the regulation of blood volume. The blood volume determines the mean pressure throughout the system, in particular in the venous side where most of the blood is held.

The low-pressure baroreceptors have both circulatory and renal effects; they produce changes in hormone secretion, resulting in profound effects on the retention of salt and water; they also influence intake of salt and water. The renal effects allow the receptors to change the mean pressure in the system in the long term.

Denervating these receptors 'fools' the body into thinking that it has too low blood volume and initiates mechanisms that retain fluid and so push up the blood pressure to a higher level than it would otherwise have.Studies in humans have suggested that autonomic degeneration with accumulation of hyperphosphorylated tau or alpha-synuclein in neurones in medulla may impair central regulation of baroreflex responses and predispose elderly patients to CSH. However, the exact mechanism and site of abnormal sensitivity is unknown. The exaggerated response may be due to changes in any part of the reflex arc or the target organs.

1. **Stanfield, CL; Germann, WJ. (2008) Principles of Human Physiology, Pearson Benjamin Cummings. 3rd edition, pp.430-431.**

Elderly patients with this condition may deny syncope and present with recurrent unexplained falls. CSM, ideally with noninvasive phasic blood pressure monitoring, should be routinely performed in elderly patients with unexplained bradycardic or hypotensive symptoms. Clinically, 3 subtypes of CSH have been described

1. The cardioinhibitory subtype (CI): comprises 70-75% of cases. The predominant manifestation is a decreased heart rate, which results in sinus bradycardia, AV block, or asystole due to vagal action on sinus and atrioventricular nodes. This response can be abolished with atropine.
2. The vasodepressor subtype (VD): 5-10% of cases. The predominant manifestation is a vasomotor tone decrease without a change in heart rate. It is necessary a reduction in systolic blood pressure exceeding 50 mm Hg independent of heart rate slowing. The significant resulting drop in blood pressure is due to a change in the balance of parasympathetic and sympathetic effects on peripheral blood vessels. This response is not abolished with atropine. Symptom reproduction is more likely with the CI than the VD subtypes[1].
3. The mixed subtype comprises 20-25% of cases. A decrease in HR and vasomotor tone occurs. The prevalence of CSH is increased in elderly patients with hip fractures, only in those who present with an unexplained fall and report a history of syncope or unexplained falls in the past. The vasodepressor/mixed forms account for the majority of CSH responses in the group of unexplained fallers[2].

1. **Tan MP, Newton JL, Reeve P, Murray A, Chadwick TJ, Parry SW. Results of carotid sinus massage in a tertiary referral unit--is carotid sinus syndrome still relevant? Age Ageing. 2009 Nov;38:680-686.**
2. **Sachpekidis V, Vogiatzis I, Dadous G, Kanonidis I, Papadopoulos C, Sakadamis G. Carotid sinus hypersensitivity is common in patients presenting with hip fracture and unexplained falls. Pacing Clin Electrophysiol. 2009 Sep;32:1184-90.**

TERMINOLOGY DEFINITIONS

Syncope is a sudden transient loss of consciousness associated with loss of postural tone. "Blackout spells," "passing out," or "fainting" are terms occasionally used by patients and refer to syncope only if associated with loss of consciousness. Syncope must be differentiated from other states of altered consciousness, such as cardiac arrest, coma, seizures, vertigo, dizziness, lightheadedness, and presyncope. *Cardiac arrest* is a sudden loss of consciousness without spontaneous recovery, requiring electrical or pharmacologic cardioversion. *Coma* is a generalized, prolonged, and extreme depression of cerebral function. *Epilepsy* or *seizures* are generally characterized by convulsions during the loss of consciousness. The postictal period is prolonged after a seizure and characterized by drowsiness, headache, confusion, and other neurologic symptoms. Convulsions can occur with syncope, but generally only after unconsciousness has lasted for a few seconds or longer; tongue biting or incontinence of bowel or bladder is rare. Vertigo, dizziness, lightheadedness, or presyncope are characterized by a subjective sense of motion or a feeling of inability to remain standing. These symptoms are not accompanied by loss of consciousness and thus are readily distinguished from syncope.

The terms *spontaneous carotid sinus syndrome* and *induced carotid sinus syndrome* have been introduced to categorize patients who are presumed to have CSH.

The term *spontaneous carotid sinus syndrome* refers to a clinical situation in which the symptoms can be clearly attributed to a history of accidental mechanical manipulation of the carotid sinuses (eg, taking pulses in the neck, shaving) and CSH is reproduced by CSM. Spontaneous carotid sinus syndrome is rare and accounts for about 1% of causes of syncope.

The term *induced carotid sinus syndrome* refers to a clinical situation in which a patient has no clear history of accidental mechanical manipulation of the carotid sinuses and has a negative result from workup for syncope, except for a hypersensitive response to CSM, which can be attributed to the patient's symptoms. Induced carotid sinus syndrome is more prevalent than spontaneous carotid sinus syndrome and accounts for the bulk of patients with an abnormal response to CSM observed in the clinical setting.

Frequency

CSH is found in 0.5-9.0% of patients with recurrent syncope. CSH is observed in up to 14% of elderly nursing home patients and 30% of elderly patients with unexplained syncope and drop attacks.

Mortality/Morbidity CSH is associated with an increased risk of falls, drop attacks, bodily injuries, and fractures in elderly patients.

Rates of total mortality, SD, MI, or stroke are unaffected by the presence of CSH.

Gender

CSH is more common in males than in females.

Age

CSH is predominantly a disease of elderly people; it is virtually unknown in people younger than 50 years.

History

Although many patients remain asymptomatic, the following are symptoms of CSH:

Recurrent dizziness, near-syncope, recurrent syncope[1], non accidental, unexplained falls[2].

Symptoms produced by head turning or wearing garments with tight-fitting collars

Neck tumors, extensive neck scarring secondary to radical dissection or radiation fibrosis or neck trauma. Possible prodrome or retrograde amnesia for the syncope events.

1. **Kuo FY, Hsiao HC, Chiou CW, Liu CP. Recurrent syncope due to carotid sinus hypersensitivity and sick sinus syndrome. *J Chin Med Assoc*. Oct 2008;71:532-535.**
2. **Parry SW, Steen N, Bexton RS, Tynan M, Kenny RA. Pacing in elderly recurrent fallers with carotid sinus hypersensitivity: a randomised, double-blind, placebo controlled crossover trial. *Heart*. May 2009;95:405-409.**

Tan et al[1] comparing autonomic function measured by heart rate variability (HRV) and baroreflex sensitivity in patients with symptomatic CSH and asymptomatic individuals with and without CSH.

The authors studied 22 patients with symptomatic CSH, 18 with asymptomatic CSH, and 14 asymptomatic older individuals without CSH.

Non-invasive measurements of HR and BP were obtained during 10 min of supine rest.
Low frequency (LF), high frequency (HF), and total power spectral density (PSD) for HRV were determined using the autoregressive method.
The baroreflex slope (BRS) and baroreflex effectiveness index (BEI) were determined using the sequence method for baroreflex sensitivity.

There were significant increases in the LF-HRV, total PSD, LF:HF, normalized (nu) LF-HRV, down ramp BEI, and total BEI in the symptomatic CSH group compared with non-CSH controls.
The asymptomatic CSH group had significantly higher LF-HRV, total PSD, nuLF-HRV, and LF:HF, as well as up, down, and total BRS and BEI than non-CSH control participants.
This study has demonstrated an association between CSH with increased resting sympathetic activity and baroreflex sensitivity regardless of the presence of symptoms, indicating the presence of autonomic dysregulation in individuals with CSH.

These findings therefore suggest that CSH is part of a generalized autonomic disorder but do not differentiate between asymptomatic and symptomatic individuals.

1. **Tan MP, Kenny RA, Chadwick TJ, Kerr SR, Parry SW.Carotid sinus hypersensitivity: disease state or clinical sign of ageing? Insights from a controlled study of autonomic function in symptomatic and asymptomatic subjects. Europace. 2010 Nov;12:1630-1636.**

Causes: CSH is associated with the following: Male sex, advanced age, hypertension, CAD, orthostatic hypotension, vasovagal syncope, Alzeimer and Parkinson disease, dementia with Lewy body [1;2] Concurrent medication with digitalis, beta-blockers, and methyldopa.

Diagnosis

Laboratory Studies

The initial diagnostic workup for CSH should rule out the following:

Vasovagal syncope
Orthostatic hypotension
Situational syncope
Sick sinus syndrome
Cardiogenic syncope
Other causes of syncope (eg, neurogenic, metabolic, psychogenic)

Any patient with syncope should be evaluated with the following:

A carefully elicited history
A thorough physical examination
An ECG,

Procedures: Carotid sinus massage is the diagnostic maneuver of choice, but the technique has not been standardized. A commonly accepted massage method includes the following 4 steps:

Place the patient in the supine position with the neck slightly extended. The patient should lie supine for a minimum of 5 minutes before carotid sinus massage is applied.

Massage over the point of maximal carotid impulse, medial to the sternomastoid muscle at the upper border level of the thyroid cartilage.

Massage for 5 seconds on both sides, with a 1-minute interval between massages.

1. **Ballard C, Shaw F, McKeith I, Kenny R. High prevalence of neurovascular instability in neurodegenerative dementias. *Neurology*. Dec 1998;51:1760-2.**
2. **Kenny RA, Shaw FE, O'Brien JT, Scheltens PH, Kalaria R, Ballard C. Carotid sinus syndrome is common in dementia with Lewy bodies and correlates with deep white matter lesions. *J Neurol Neurosurg Psychiatry*. Jul 2004;75:966-71.**

Continuously monitor surface ECG and blood pressure. Phasic, noninvasive, beat-to-beat blood pressure monitoring is preferred over using a cuff measurement.

A massage is considered to have a positive result if any of the following 3 criteria are met:

1. Asystole exceeding 3 seconds (indicates cardioinhibitory CSH)
2. Reduction in systolic blood pressure exceeding 50 mm Hg independent of heart rate slowing (indicates vasodepressor CSH)
3. Combination of the above (indicates mixed CSH),

A less frequently used method consists of carotid sinus massage performed for 5 seconds on each side in the supine and 60º positions using the head-up tilt table. Substantial evidence shows that sensitivity and diagnostic accuracy of carotid sinus massage can be enhanced by performing the test with the patient in an upright position. Furthermore, the endpoint of a 50 mm Hg reduction in systolic blood pressure may be achieved with tilt, but not when supine.

Do not perform a carotid sinus massage if the patient is known to have transient ischemic attack, stroke, or myocardial infarction in the preceding 3 months. History of VT/VF, or carotid bruit on auscultation are relative contraindications to carotid sinus massage.

Some authors describe the use of carotid Doppler ultrasonography to guide carotid sinus massage in patients who have a carotid bruit on auscultation. Carotid sinus massage is performed only in patients with a carotid bruit when there is less than 70% stenosis on Doppler examination.

Although carotid sinus massage is usually a benign bedside procedure, a few case reports describe rare neurological deficit symptoms following the massage. Currently, the estimated incidence of neurological complications is less than 0.2%.

A single case report describes the induction of coronary artery spasm by carotid sinus massage. Similarly, rare case reports describe the induction of atrial or ventricular arrhythmias by carotid sinus massage. Carotid massage has its greatest clinical utility in elderly patients aged 60-80 years.

The positive predictive value of carotid massage remains undefined. Therefore, a clinician who finds a sensitive carotid sinus should consider other prognostically important causes of syncope and the presence of comorbid conditions.

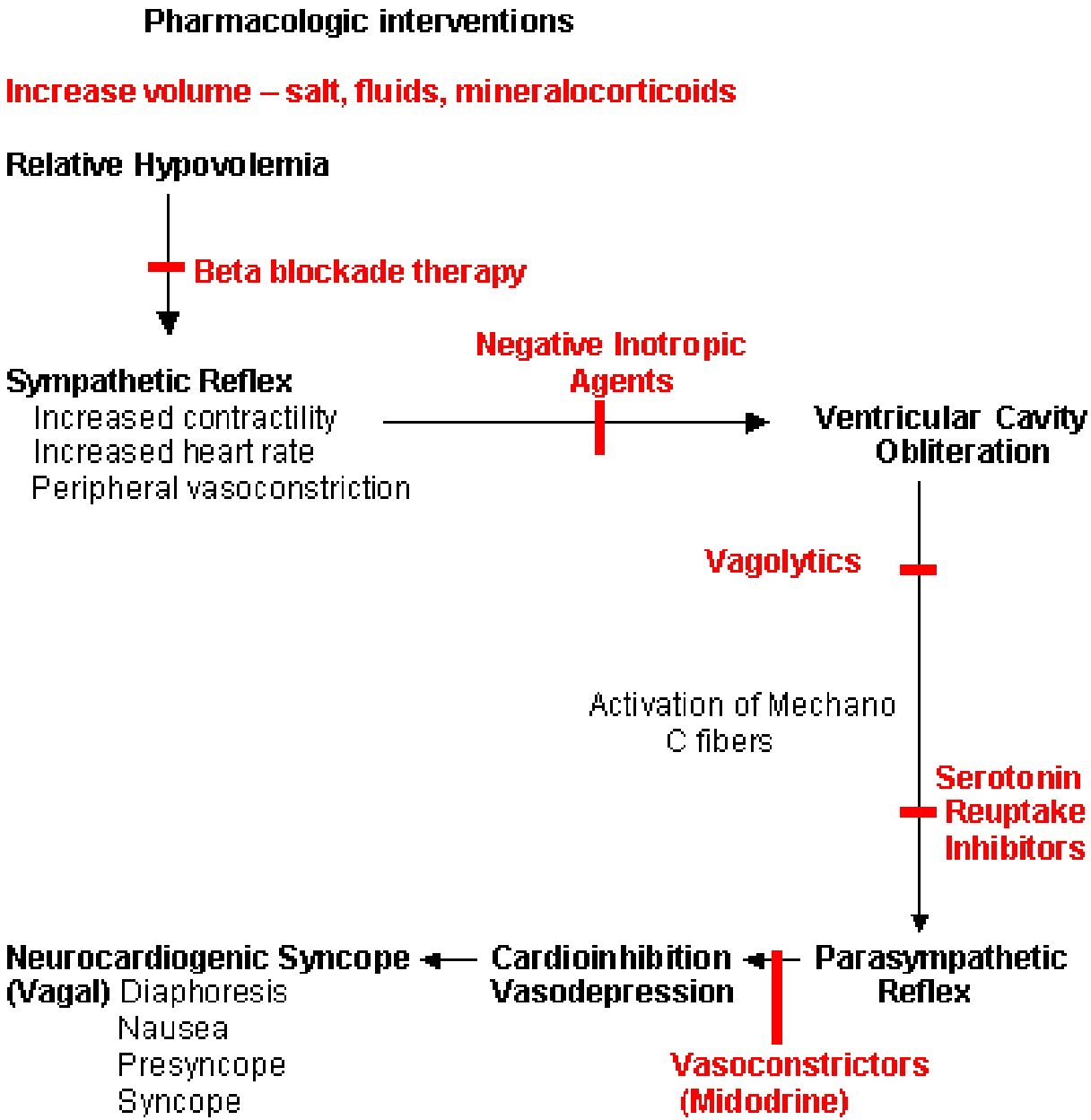
Treatment: of CSH is based on the frequency, severity, and consequences of each patient's symptoms. An individual without another cardiovascular disease should increase salt intake and drink more fluids containing electrolytes. Most patients can be treated with education, lifestyle changes, expectancy, and routine follow-up. Volume maintenance can control the vasodepressor form of CSH, preventing syncopal episodes by maintaining adequate central volume. No general activity restrictions are necessary. Patients should be aware of prodromal symptoms of presyncope or syncope. In such circumstances, immediately assuming supine posture is recommended to prevent syncope and/or falls. Precipitating events, such as wearing tight neck collars or sudden rotating neck movements, should be avoided. A few individuals who have incapacitating and recurrent symptoms may need:

1) Pharmacotherapy has been used to treat recurrent, symptomatic conditions. First line of therapy is maintaining adequate intravascular volume with sufficient fluid and salt intake. If this is not sufficient, then the addition of a mineralocorticoid may be helpful. Of the specific drugs that have been used, beta adrenergic blocking drugs have had the best results although not all studies have demonstrated a positive response. Although a variety of pharmacological agents has been used empirically to treat recurrent, symptomatic CSH, no single agent has been unequivocally proven to provide long-term effectiveness in large-scale, randomized controlled trials. Some observers have successfully used the serotonin reuptake inhibitors sertraline and fluoxetine in patients who were unresponsive to dual-chamber pacing. A randomized, controlled pilot study showed that treatment with midodrine, an alpha-1 agonist, could significantly decrease the rate of symptoms and the degree of hypotension in the vasodepressor form of CSH.

Treatment with midodrine was also associated with an elevation of a mean 24-hour ambulatory blood pressure level[1]. Midodrine induces arterial and venous capacitance constriction and has minimal cerebral and cardiac effects. It is indicated for the treatment of symptomatic orthostatic hypotension. Fludrocortisone is another agent that can be used in this setting. The US FDA has not yet approved these agents for the management of CSH.

The proposed sites of action of the various pharmacologic options are shown in Paul Levine[2] work on following slide Figure 3.

1. **Moore A, Watts M, Sheehy T, et al. Treatment of vasodepressor carotid sinus syndrome with midodrine: a randomized, controlled pilot study. *J Am Geriatr Soc*. Jan 2005;53:114-8.**



From Levine P - Role of Pacing in the Management of Neurocardiogenic
http://www.fac.org.ar/scvc/llave/arritmia/levine1/levine1i.htm

PACING THERAPY

2) Permanent pacemaker implantation is generally considered an effective treatment for cardioinhibitory CSH and mixed forms of CSH. Current American College of Cardiology/American Heart Association/Heart Rhythm Society clinical practice guidelines consider permanent pacing therapy to be a class I indication (ie, general agreement exists that the therapy is effective and useful) in patients with recurrent syncope caused by carotid sinus stimulation in the absence of any drug that depresses the sinus node or atrioventricular conduction. Permanent pacing is considered a class IIa indication in patients with recurrent syncope without clear, provocative events and with a hypersensitive cardioinhibitory response. Permanent pacing is discouraged in patients with a hypersensitive cardioinhibitory response to carotid sinu s stimulation in the absence of symptoms1.The consensus is that dual chamber pacing (DDD, DVI, DDI) is optimal in the patients. However, VVI mode is also effective in preventing recurrent syncope in some patients. AAI and VDD modes are considered inappropriate. Cardiac pacing has little or no effect on the vasodepressor type of CSH and may not reduce the frequency of falls in patients with CSH. Permanent pacing may diminish but not entirely eliminate the symptoms in CSH.

Surgery remains an option for a patient with a neck tumor that is compressing the carotid sinus. Consult an electrophysiologist or cardiologist to rule out cardiac arrhythmia and evaluate the patient for pacemaker implantation. Obtain a surgical consultation and evaluation if the patient has a neck tumor that is compressing the carotid sinus.

1. ***Epstein AE, DiMarco JP, Ellenbogen KA, Estes NA 3rd, Freedman RA, Gettes LS, et al. ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices) developed in collaboration with the American Association for Thoracic Surgery and Society of Thoracic Surgeons. J Am Coll Cardiol. May 27 2008;51(21):e1-62***