

# Carotid Sinus Hypersensibility: A Concept on the Way\*

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## ABSTRACT

This review examines the pathogenesis of Carotid Sinus Hypersensitivity (CSH) by describing the different hypotheses that exist in literature. One hypothesis is that CSH is not a disease of the heart but of the central and/or peripheral nervous system where the heart is the victim. CSH is associated with cognitive disturbance, vasovagal syncope and orthostatic hypotension. Another hypothesis is that the CSH is considered a syndrome or a symptom associated with different diseases so as to require a diagnostic framework. This Hypothesis is the basis of frequent recurrences that are associated with the pacemaker implant therapy only.

**Keywords:** Syncope; Autonomic Nervous System; Pacing; Physiology

## 1. Introduction

Carotid sinus hypersensitivity (CSH) becomes a disease when it becomes responsible of falls and syncope. In this case the diction carotid sinus syndrome (CSS) is preferred. These are the cases handled, whether cardio-inhibitory type or even if mixed, with implantation of pacemaker (PM). In contrast with this therapeutic progress, the pathophysiology of the syndrome remains poorly understood. Indeed, the question arises whether the answer in bradycardia and hypotension that you get with the massage of the carotid sinus (MSC) is really just a physiological response that grows up to cause symptoms when the age progresses. And this occurs mainly in males.

## 2. CHS and Heart

In the collective idea, the CSH is considered a heart disease. Post hoc propter hoc: it is obvious if the remedy is just that, the cause is related. The CSS has been considered symptom of sino-atrial disease [1,2]. Indeed, patients with CSS usually have normal sinus node function [3], nor is there a clear demonstration that the heart is patient when the subject is symptomatic carrier (CSS) or asymptomatic to CSH. The heart is the victim of a spiteful neurologic reflex which sometime shows a prevalent vasodepressor response [4]. This does not concern the sinus node and suggests that the abnormality lies in some place of the reflex arc or at the level of the carotid sinus receptors [5]. The asymmetrical response between left

and right MSC gives the concept support of a pathological reflex arc rather than of a target organ.

The CSH has a population over 65 years from 14% to 39% [6], CSS hacks in 16% of males and 8% of females [7]. It is without any doubt that, at the same old age, the subjects suffering from CSH and who are symptomatic for CSS are minority. This puts them outside the “normal” and proposes a strong evidence in favour of specific pathology.

## 3. CHS and Brain

Patients with cognitive diseases, particularly with dementia, show strong association with the CSH, particularly in dementia with lewy bodies (DLB) [8]. This observation does not appear to be significantly related to ageing. Other morbid conditions such as Alzheimer’s disease (AD) do not have the same link. A degenerative hypothesis tries to unify the Central Nervous System degenerative diseases (AD, DLB and Parkinson’s disease ) when are associated with CSH. The degeneration of medullary autonomic nuclei which regulate baroreflex responses for accumulation of material (*i.e.* hyperphosphorylate-tau protein or fibrillar amyloid-beta) [9] allows to think of a progressive involvement of cognitive, motor and autonomic activities [8].

## 4. CHS and Peripheral Nervous System

So, if the therapeutic problem finds a solution replacing the electrical activity of the heart does not mean that this is the only form of therapy.

The stripping of adventitia from the common carotid

\*Any conflict of interest declared.

artery, the bifurcation, and for about 1 cm up both the external and internal carotid arteries was used to treat CSS with excellent results due to the fact that 85% of patients remained asymptomatic. The experience is limited to two surgical groups and a few cases [10-12]. The more recent review [13] refers to 130 procedures. The data is very significant: it is demonstrated that patients remain asymptomatic and this is index that alternative therapeutic answers there are. May be they are more complex and more difficult to implement, less documented, but certainly no less reliable, than PM implantation [10-13]. However, what we need to discuss here is the pathophysiological significance of the surgical intervention. Solving the problem of CSH and CSS modifying the reflex arc and acting on peripheral receptor, it provides an indication on the beginning site of the disease. It indicates that the event is not in the pathological effector organ, *i.e.* the heart. And that is enough to move the pathophysiological interest on the peripheral or central nervous system.

It is in this perspective that we can read some cases properly investigated who proposed a relationship between CSH and the complete or partial denervation of the sternocleidomastoid muscle [14,15] associated with a defect of proprioceptive information flow. The central nervous system, without any information about the contractile state of the muscles, interprets any information coming from the carotid sinus receptors only as an abrupt increase in blood pressure and reacts by a dramatic decrease in heart rate and/or blood pressure. This issue is not yet sufficiently supported by adequate survey, although numerically sporadic observations, relating to cervical pathology that has involved the carotid sinus, may intrude on this line of thought. [16-18].

## 5. CHS and Dysautonomia

Finally an association is reported between CSH and cardiovascular dysautonomia, witnessed by the changed baroreflex [19,20] and by the presence of orthostatic hypotension (OH). The matter of this link comes from patients studied with both MSC and tilt test. Carotid sinus hypersensitivity and OH are common conditions affecting patients with syncope and falls which are likely to co-exist in such individuals. The differences in haemodynamic response patterns to active stand and HUT in CSH subjects could be the result of an age-associated delay in sympathetic responses [21]. Indeed, the simultaneous presence of CSH and OH is present in 55% of patients selected for syncope in a single study (SYSTEMA I—Malmo) [22] but there are not still enough data to say that these subjects have a pathology of autonomic nervous system or that do not have a random association between the two diseases. In a population of over 60s, collected in different cases and selected for

syncope and falls, the CSH was present between 12.8% and 45% and OH between 24% and 32% of subjects [23-26]. Their association did not appear strong as it was age-related [23,27].

Orthostatic hypotension and CSH are both found frequently in older people being investigated for falls. Older age, male sex and symptoms of syncope are all associated with a higher likelihood of a diagnosis of either CSH or OH. Unlike OH, the prevalence of CSH varies widely between different study populations, possibly due to demographic differences.

In conclusion, it can certainly affirm that some patients, males and elderly, suffer simultaneously or more frequently in the progression of either diseases associated with CSH: OH and dementia. The absence of true associations between OH and dementia with CSH does not support the theory of an association leading to a common mechanism between the three conditions [21].

This can certainly mean that these three diseases have a high incidence in male elder but because those who are affected are a minority you can't exclude that a progressively degenerative disease is working in these patients up to affect different organs and systems.

The data is not demonstrated but is a fascinating hypothesis on which to build longitudinal studies.

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