Europace (2004) **6**, 292–295

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CASE REPORT

Carotid sinus massage during evaluation for transient loss of consciousness: just a positive test?

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Submitted 2 February 2004, and accepted after revision 14 March 2004

KEYWORDS
carotid sinus massage;
syncope;
ventricular ectopy

Abstract An electrocardiographic recording obtained during diagnostic evaluation of recurrent transient loss of consciousness in a 53-year-old man is presented. Carotid sinus massage (CSM), having elicited a ventricular asystole of >5 s duration was deemed to have provided a possible diagnostic basis for syncope. However, apart from the pause and somewhat unexpectedly, CSM also suppressed preexisting frequent ventricular ectopy. Explanations for this unexpected finding can only be considered speculative, but include direct CSM-induced parasympathetic suppression of ectopic activity at intra-ventricular pacemaker sites, concomitant diminution of sympathetic neural activity at ectopic sites, or interruption of 'linking' of normal ventricular activation to initiation of premature ventricular contractions.
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Introduction

Carotid sinus massage (CSM) is an important diagnostic manoeuvre for evaluation of syncope in older patients [1]. Pressure at the site of bifurcation of the common carotid artery has long been known to elicit a reflex parasympathetic response in the heart. The physiological outcomes are negative chronotropic and dromotropic effects, principally evident at the sinus and AV nodes, respectively. Additionally, peripheral vascular dilation (i.e., vasodepressor effect) may also be observed, and is thought to be due in part to diminution of sympathetic neural tone [2].

CSM is undertaken by applying firm massage for 5–10 s at the anterior margin of the sternocleidomastoid muscle at the level of the cricoid cartilage. Initially, this is undertaken with the patient in the supine position but it may be necessary to repeat the test with the patient upright in order to derive the greatest diagnostic benefit. After 1 or 2 min a second massage is applied on the opposite side if the initial CSM failed to yield a 'positive' result. It is generally agreed that a 'positive' finding,

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[1] Dr Ermis was supported by a grant from the Midwest Arrhythmia Research Foundation (Edina, Minnesota).

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doi:10.1016/j.eupc.2004.03.002

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suggesting the presence of carotid sinus syndrome, is either a ventricular pause $\geq$ 3 s and/or a fall of systolic blood pressure $\geq$ 50 mmHg, or reproduction of spontaneous symptoms [1—3].

Findings in this report illustrate that the neural reflex associated with CSM has multiple facets, and the impact of the manoeuvre extends beyond the sinus and atrio-ventricular (AV) nodes.

Case

A 53-year-old man was brought to the emergency room for evaluation of an episode of transient loss of consciousness. The medical history indicated that he had experienced frequent similar but less severe spells over 2—3 years. Apart from these episodes, his past medical, social and family histories were unremarkable. He was not being treated with any medication at the time. Physical examination was normal except for an irregular heart rhythm. Blood count, electrolytes and cardiac enzymes were normal. A 12-lead electrocardiogram showed sinus rhythm with frequent premature ventricular complexes. He was admitted to the telemetry unit for observation and cardiac monitoring. During subsequent diagnostic evaluation, CSM was performed.

The rhythm strip provided (Fig. 1) was obtained during right sided CSM. Left-sided CSM did not alter the cardiac rhythm. The finding of an induced asystole $>$ 5 s duration was deemed consistent with carotid sinus syndrome, and pacemaker therapy has been effective. However, suppression of frequent ventricular ectopy during CSM was unexpected, and the understanding of this observation is the basis of this communication.

Discussion

The rhythm strip recorded prior to CSM shows a baseline sinus rhythm with frequent premature

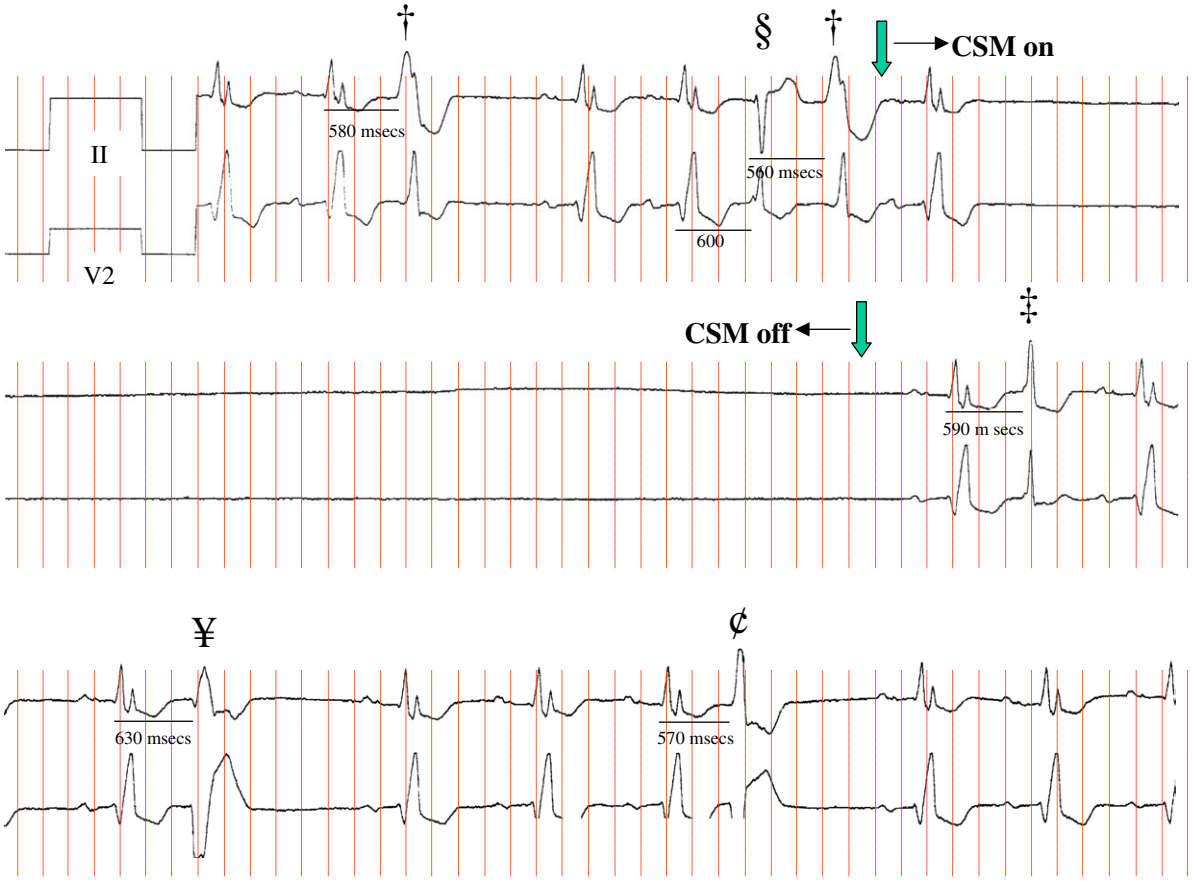


Figure 1 Rhythm strip (lead II above, lead V2 below) obtained during right sided CSM. The recording is reproduced at 25 mm/s, 10 mm/mV. Arrows indicate the timing when CSM was initiated and terminated. Note that the frequent polymorphic ventricular premature complexes (each form labelled with †, §, ‡, ¥ and ¢) which were consistently present both before and after CSM, disappeared during the massage-induced ventricular asystolic period. The variable coupling intervals of ectopic beats to the onset of the preceding QRS are indicated.

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ventricular complexes of several morphologies (Fig. 1). Initiation of right sided CSM triggered a ventricular asystolic pause of approximately 9.2 s duration. This finding constitutes a 'positive' CSM response [1] and suggests that carotid sinus syndrome may contribute to spontaneous symptoms in this individual despite his relatively young age. However, in addition, CSM unexpectedly suppressed ventricular ectopy that had been consistently present prior to CSM, and returned after termination of the manoeuvre.

Sinus pauses, sinus arrest, and transient high grade AV block are anticipated events with CSM in patients with carotid sinus hypersensitivity. These elements of the cardiac conduction system are densely infiltrated with parasympathetic nerve endings, and the physiological effects of CSM are attributed primarily to enhanced vagal efferent activity. On the other hand, parasympathetic control of cellular automaticity and conduction typically diminishes substantially in more distal aspects of the conduction system. Thus, CSM suppression of ventricular ectopy is a rare observation in the literature [4]. However, insight into potential parasympathetic influence on conduction system properties is provided by reports examining the impact of increased parasympathetic tone on ventricular tachycardia. This topic has been the subject of case reports [5—11] which support the notion that in selected cases certain infra-AV nodal sites may be sensitive to vagal manoeuvres.

Several explanations for CSM suppression of ventricular ectopy can be offered, but all are speculative. First, elements of the intra-ventricular Purkinje network are believed to be common sources of ventricular ectopy, and it is generally accepted that there is substantial inter-individual variation of parasympathetic neural penetration within these fibres [12,13]. Conceivably, in certain patients CSM-induced enhanced parasympathetic tone, might slow 'phase 4' depolarization in potential pacemaker cells. Second, diminution of sympathetic neural activity is also known to occur during CSM, and is believed to account for the vasodilation (i.e., vasodepressor) commonly accompanying carotid sinus stimulation [2]. Conceivably, if operative in the heart, this same sympathetic 'withdrawal' might diminish susceptibility to catecholamine supported triggered activity. Finally, a non-neural mechanism can be considered. Specifically, it is possible that the ventricular ectopic beats in this patient are 'linked' to the normally conducted ventricular events. Thus, in the absence of normal cardiac activation, the ectopic event could no longer occur. In this context, both local reentry as well as a mechanically induced ectopic beat could be contemplated.

The mechanism of CSM suppressed ventricular ectopy in this case cannot be stated with certainty. However, careful review of the rhythm strip provides two observations that diminish the likelihood of a 'linking' phenomenon. First, the coupling interval between various ventricular ectopic beats and preceding conducted beats varies (Fig. 1). This finding implies that tight coupling between the two phenomena does not exist in this patient. Second, accounting for the several morphologies of ectopic beats is difficult in a 'linking' scenario. In essence, one would need to hypothesize an extremely complex mechanism in order to explain the observed morphology differences.

In summary, the CSM observation in this patient demonstrates once again the wide range of manifestations of neural reflex activity in the heart.

Acknowledgements

The authors would like to thank Wendy Markuson and Barry LS Detloff for their technical support.

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