A cautionary tale of carotid sinus massage for narrow QRS tachycardia

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A 72-year-old woman with a history of metabolic syndrome presented with sudden-onset rapid palpitations and vertigo beginning 2 h earlier. She denied previous chest pain, shortness of breath, syncope, or heart failure. On admission, blood pressure was 143/87 mmHg, and heart rate, 112 bpm. Cardiopulmonary examination was unremarkable. Laboratory tests including renal function, electrolytes, thyroid function, and blood formula were normal. An echocardiogram showed normal biventricular function and the absence of valvular heart disease or pericardial effusion. The initial electrocardiogram (Fig. 1A) showed regular, narrow QRS tachycardia with evidence of supraventricular activity, best visible in leads V1–V6. The rs' pattern in leads V1–V6 suggests atrial tachycardia — either atypical atrial flutter, focal atrial tachycardia, or atypical atrioventricular (AV) nodal re-entrant tachycardia — with 2:1 AV conduction. To establish the diagnosis, we performed carotid sinus massage (CSM) that did not modify the heart rate or modulate AV conduction. Right after CSM termination, the patient developed shortness of breath and mild chest pain, and a new electrocardiogram was immediately performed (Fig. 1B). Following CSM, the “paired” or “group beating” appearance of QRS corresponded to improved 3:2 AV conduction with a Wenckebach phenomenon, resulting in a mean ventricular rate of 149 bpm (= atrial rate [224 bpm] × AV conduction ratio [2:3]). Atrial activity (F waves) is best seen in leads V1–V6, at an unchanged rate (224 bpm, F-F = 268 ms) and an AV sequence of an initial atrial wave normally conducted (184 ms), a second F-wave conducted with a prolonged interval (216 ms), and a third F-wave that is blocked; then the cycle of events is reinitiated. Alternating QRS morphology with intermittent incomplete right bundle-branch block is due to the Ashman phenomenon, where the right bundle branch is refractory due to the shorter R-R interval of the preceding cycle [1]. CSM elicits an intense activation of the baroreceptor reflex [2]. The afferent limb includes nerve fibres present in the carotid sinus (CS) that join the glossopharyngeal nerve to directly stimulate medullary centres. The efferent limb of the reflex is twofold: a) by vagal excess due to activation of parasympathetic fibres to the heart and b) by sympathetic inhibition through attenuation of the tonic discharge of sympathetic nerves trafficking from the stellate ganglion to the heart. When CS pressure is increased and maintained, intense parasympathetic stimulation and complete sympathetic inhibition can occur for some seconds. A rebound response after withdrawal of CS pressure is due to simultaneous withdrawal of the parasympathetic brake and return of the sympathetic nerve discharge together with enhanced liberation of catecholamines by the adrenal glands [2]. Deepak et al. [3] described a case of ventricular fibrillation induced by CSM, concluding that vagally mediated rebound sympathetic activity and catecholamine release could have led to ventricular ectopy and tachyarrhythmia. One should also note a distinct mechanism of supraventricular tachycardia acceleration after CSM: that of CSM-induced elimination of a rate-related bundle branch block in the presence of a participating ipsilateral accessory pathway [4]. To our knowledge, this is the first report of rebound facilitation in AV conduction induced by CSM for narrow QRS tachycardia. Although our patient remained stable and enhanced AV conduction was only transient, clinicians should be aware of this rare possibility and ensure resuscitation equipment is readily available while performing vagal manoeuvres.

References

Figure 1. A. An electrocardiogram showing regular narrow QRS tachycardia, with evidence of atrial activity (+), best visible in leads V1–V6. The rs’ pattern (•) in leads V1–V6 suggests atrial tachycardia with 2:1 atrioventricular conduction; B. An electrocardiogram following carotid sinus massage: the “group beating” appearance of QRS corresponds to 3:2 atrioventricular conduction. Alternating QRS morphology with intermittent incomplete right bundle-branch block is due to the Ashman phenomenon.

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Conflict of interest: none declared

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