

A Proposed Physiologic Role for Pulse Synchronized Contractions (PSCs): Expert Opinion

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Pulse Synchronized Contractions, Windkessel Hypothesis, Arteries.

Previously, we have provided contrary evidence [1-3] to the Windkessel Hypothesis [4]. Windkessel describes the behavior of the smooth muscle wall of large arteries as passive elastic tubes being rhythmically distended by pulsatile pulse pressure changes [4]. We have shown that the smooth muscle wall of large arteries is able to generate fast rhythmic electrical waves [5] and in vivo the smooth muscle wall of the large arteries is capable of undergoing rapid contractions at the rate of the heartbeat [1-3,6]. Denoted as pulse synchronized contractions, or PSCs, these contractions were found to be neurogenic in origin and sensitive to neurotoxins such as tetrodotoxin or lidocaine [1-3,6] and were not produced by a movement artifact from the pulse wave or heartbeat. The pacemaker for PSCs resides in the right atrial appendage [1].

We proposed that PSCs represent a modified platform to understand the etiology of cardiovascular diseases, allowing for the potential development of new therapeutic targets. A major question is, what might be the physiologic role of PSCs?

Interestingly, Englesbe et al. [7] have shown that in cardiac transplant patients an increase in aortic aneurysms is noted. With cardiac transplant, neural connectivity from the right atrium down the aorta would be disrupted. With disruption of these neural connections, we propose that PSCs may be eliminated. The

phasing of the PSC with the pulse wave [1,2,6] is such that the PSC contraction and pulse wave upstroke occur near simultaneously and PSCs may serve to limit the distension of the vessel walls from the pulse wave. Thus, with elimination of PSCs the aortic wall may undergo greater shearing forces, leading to damage and potentially aneurysms which may lead to dissection. We believe this hypothesis warrants further evaluation.

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