



Pulse Synchronized Contractions (PSCs): A Call to Action!



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Editorial

For over a century, the prevailing view has been that the smooth muscle wall of large conduit arteries does not undergo rhythmic contractile activity in synchrony with the cardiac cycle. This behavior was described by Otto Frank and denoted the Windkessel Hypothesis [1]. Considering the wide acceptance of the Windkessel Hypothesis, potential targets for new therapeutic agents have not considered rhythmic activation during the cardiac cycle.

In contrast to the Windkessel Hypothesis, evidence has accumulated that the smooth muscle wall of large arteries undergoes rhythmic activation in synchrony with the heartbeat. This rhythmic contractile activity has been denoted pulse synchronized contractions (PSCs) [2-6]. PSCs have been demonstrated in dogs, cats, rats, and, notably, humans [2-13]. Specifically, activity has been seen in aorta, carotid, femoral, pulmonary, and brachial arteries [2-13]. Much attention has been paid to confirming that PSCs are not due to a movement artifact secondary to the pulse wave or cardiac contractions [2,13]. PSCs are not abolished in bled animals, in which there is no pulse wave, and when animals have a dissociation between ventricular contractile activity and right atrial activity, PSCs are coupled to atrial not ventricular rate [2].

The pacemaker for PSCs resides in the right atrium [2,13], allowing speculation that this may allow coordination between cardiac and vascular contractile activity. Generation of PSCs is through a neurogenic not myogenic mechanism [2,12,13], although the specific neural pathway has not been elucidated.

Although data in contradistinction to the Windkessel Hypothesis have been published for over 50 years [2-13] and these findings have been demonstrated in several independent laboratories, PSCs have not been accepted, and, therefore, a potential therapeutic site for the treatment of certain cardiovascular diseases has not been explored. PSCs may potentially serve a protective function limiting vessel distension with the pulse wave as the upstroke of the PSC appears to slightly precede the upstroke of the pulse wave. This

specific timing of the pulse wave and PSC may serve to limit vessel distension, reducing the Laplacian forces acting on the vessel wall.

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