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EDITORIAL

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Pulse synchronized contractions: Rhythmic contractions in large arteries in synchrony with the heart beat

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The classical view of the smooth muscle wall of large arteries is that they behave as passive elastic tubes. According to this view, the arteries are rhythmically distended by the pulse wave versus undergoing rhythmic activation during the cardiac cycle. Data obtained over the past several decades have shown this description not to be correct. In dogs, cats, rabbits, rats and man rhythmic contractions occur in synchrony with the cardiac cycle in large conduit arteries. These contractions, denoted pulse synchronized contractions (PSCs), are neurogenic in origin with their pacemaker in the right atrium. The phasing of the PSC has the upstroke of the contractions during the rising phase of the pulse wave. This has been suggested to serve to limit vessel wall distension by the pulse wave. Further evaluation of PSCs is required to understand the clinical-pathologic role that they may play.

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One of the principal platforms for understanding the behavior of the cardiovascular system is that the smooth muscle wall of large arteries does not undergo rhythmic activation (i.e., contractions) during the cardiac cycle; but, rather, they behave as passive elastic tubes.This was described by the Windkessel Hypothesis [1]. This hypothesis persists as the principal description of the large arteries even though data have been provided for several decades showing that it is incorrect. Indeed, in studies with dogs,cats, rabbits, rats and humans rhythmic arterial tension oscillations have been shown to occur in synchrony with the heartbeat [2-9]. This activity, which has been denoted pulse synchronized contractions or PSCs, has been demonstrated in the aorta, coronary arteries, femoral arteries, pulmonary arteries, carotid arteries and brachial arteries [2-9].

The pacemaker for PSCs resides in the right atrium, postulated to allow for excellent coordination between the pulse wave and PSCs [2]. PSCs are neutrally-mediated showing sensitivity to the neural blocker tetrodotoxin in animals [2-5]. Based on the phasing of the PSC to the pulse wave, with the contraction upstroke slightly preceding the upstroke of the pulse wave, it has been postulated that they serve to reduce the Laplacian Forces acting on the vessels, thus serving a protective function for the vessel wall [2,3].

A fundamental dilemma in science is the trade-off between potential premature adoption of new hypotheses versus being entrenched in existing dogma. We believe that demonstration of PSCs in multiple animal models, as well as in man, as well as across several large conduit arteries, reaches the threshold for the phenomena to be recognized. It is time to evaluate the potential clinical relevance of PSCs in the field of cardiology.

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