OPEN ACCESS Freely available online

Journal of Clinical & Experimental Cardiology

Commentary

Embryological Development of Cardiac Muscle Sleeves and Pulmonary Veins

Ian Bredeloux*

Department of Cardiology, Université de Tours, Tours, France

ABOUT THE STUDY

Atrial fibrillation in humans can be induced and maintained by ectopic activity in the cardiac muscle sleeves of the pulmonary vein. Any study of the pulmonary veins must take into account how they differ from the left atrial cardiac muscle. In this article, we try to encapsulate the physiological processes that lead to ectopic electrical activity in animal pulmonary veins. We stress that the beginning of triggered or automated activity may include the activation of several signalling pathways that affect both myocyte electrophysiology and the mechanisms of excitation-contraction coupling. We also gather information on the large-scale structure of cardiac muscle sleeves as well as current research that show cellular heterogeneity may help to distinguish between pulmonary vein and left atrial heart muscle and to produce arrhythmogenic occurrences.

Studies of mouse embryological development provide the most convincing evidence for the distinctions between cardiac muscle in the Left Atrium (LA) and the Posterior Ventricle (PV). The PV myocardium's origins can be distinguished from the LA myocardium's by tracking the transcription factors Pitx2 and Tbx5. In short, after the formation of the LA and the construction of the venous link between the heart and the lung, the PV myocardium develops from mesenchyme tissue at the base of the PV. Proliferation and migration through the vein to intra-lobular capillaries in the lung follow the differentiation of this mesenchyme to the myocyte phenotype over the course of a fairly little time (24 h). Similar mechanisms determine when PV myocardium develops in the human heart. A myocardial sleeve is only formed by the proliferation and migration of NKX2-5 positive and TH18 negative cells along the vein when the early embryonic single PV is connected to the LA. The single initial PV in the 110-day human embryo has split into four distinct PV ostia that drain into the LA body. The cardiac muscle sleeves are visible in all four veins and, at this time, only go as far as the hilum outside of the developing lung. The cardiac muscle sleeves of an adult human PV barely extend 1-2 cm from the PV ostia. Adult PV in other big animals, such as the pig, sheep, and dog, frequently exhibits this characteristic. On the other hand, adult rodents like mice, rats, guinea pigs, and rabbits still have their PV cardiac muscle sleeves, which extend into veins into the lungs. Together, these findings show that the PV myocytes and LA myocytes have distinct embryological origins. It is therefore very feasible that these two cell groups also operate physiologically differently, which would make PV cardiomyocytes more likely to initiate electrical ectopic activities that encourage AF. Several studies have been conducted on the processes responsible for the development of arrhythmia in PV especially those that do not only affect the PV but also result in aberrant action potentials. In fact, the electrophysiology, calcium management, innervation, and other characteristics of PV cardiac muscle make it especially well-suited for triggered action.

However, in addition to these well-known arrhythmogenic pathways, the PV structure and unique physiological characteristics of cells provide access to other arrhythmia-inducing mechanisms. One arrhythmogenic process unique to the PV is illustrated by CAA. Further research into PV cardiomyocyte cell physiology, particularly how adrenergic pathways control these cells, is necessary to understand the precise mechanics of CAA induction. The authenticity of fibre direction and the heterogeneity of the cell population, which are unique to PV myocardium and contribute to its propensity for arrhythmia, can be included. Finally, recent findings from fundamental research imply that one or more pharmaceutical strategies may be able to cause electrical severance of the PV from the LA. By inhibiting the propagation of PV ectopic electrical activity to the LA, this provides for the first time cutting-edge research opportunities for the identification of molecular targets and the subsequent development of more specialized pharmaceutical therapies for AF.

Correspondence to: Ian Bredeloux, Department of Cardiology, Université de Tours, Tours, France, E-mail: ian.bredeloux@univ-tours.fr

Received: 28-Nov-2022, Manuscript No. JCEC-22-21244; **Editor assigned:** 01-Dec-2022, PreQC No. JCEC-22-21244 (PQ); **Reviewed:** 19-Dec-2022, QC No. JCEC-22-21244; **Revised:** 26-Dec-2022, Manuscript No. JCEC-22-21244 (R); **Published:** 02-Jan-2023, DOI:10.35248/2155-9880.23.13.765.

Citation: Bredeloux I (2023) Embryological Development of Cardiac Muscle Sleeves and Pulmonary Veins. J Clin Exp Cardiolog.13:765.