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Symptom of Pulmonary Vein and Cardiac Disease

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Abstract

The description of atrial fibrillation (AF) as a functional electrical condition ignores the significant underlying structural abnormalities. The muscular sleeve of the atrium and the pulmonary vein (PV) undergo microstructural alterations, resulting in a weak foundation for AF maintenance. Current data indicate that this arrhythmia typically requires a trigger for start and a weak electrophysiological or anatomical substrate for maintenance, despite the lack of knowledge regarding the anatomical and functional foundations of AF. Whether the trigger mechanisms are focused improved automaticity, prompted activity, or micro re-entry from cardiac tissue is currently unknown. AF onset can be sped up by both sympathetic and parasympathetic stimuli, which appear to also play a role in AF maintenance. A mechanism that may involve cellular ageing, apoptosis, and subsequent atrial fibrosis and inflammation is linked to both new-onset and recurrent AF, according to growing clinical data.

Keywords: Atrial fibrillation • Triggers • Pulmonary vein • Structural remodelling

Introduction

There is still a lack of knowledge regarding the pathophysiology of atrial fibrillation (AF). Given that mitral valve disease, hyperthyroidism, hypertension, coronary artery disease, and other pathological conditions can accompany this arrhythmia, and can also occur in a healthy heart, a condition known as "lone AF." The multifactorial pathogenesis of AF is suggested by current research. It has been demonstrated that the majority of cases of paroxysmal AF are caused by triggers from PVs and non-PV sites. However, ectopic foci may not necessarily be necessary for the onset and maintenance of AF. As a result of the combination of refractory period and conduction velocity, decreased wavelength allowed for the maintenance of multiple concurrent re-entry circuits, which led to the development of AF. The progression of the disease and the notion that "AF begets AF" have been linked to the idea of electrical remodelling and the associated variations in ion channel number and function. The presence of a vulnerable atrial structural substrate with areas of conduction block, which causes spatial separation of the wavelets and encourages re-entry, has been linked to the persistence of the arrhythmia, or chronic AF. In chronic AF, atrial remodelling occurs, which moves sites with the highest dominant frequency (rotors) from the PV region toward the left atrium (LA) or right atrium (RA) and complicates wave propagation. However, it is still unknown if the structural remodelling with interstitial fibrosis and myolysis features or an increase in autonomic tone are pro-fibrillatory variables or if AF can be caused by or created as a symptom of advanced age or underlying cardiac disease [1].

Description

Multidetector computed tomography and magnetic resonance imaging, among other non-invasive imaging techniques, have recently demonstrated that the pulmonary vein (PV) architecture of a patient varies. One sign of this variation is the number of PVs; Some patients have common trunks while others have five distinct ostia. A common left or right pulmonary vein is present in 25% of patients, with the left PV appearing more frequently than the right PV. The presence of "additional" PVs, the most common of which is a distinct right middle PV draining the middle lobe of the lung, is another common finding that can be found in up to 26% of patients. The ostial diameter of this right middle PV is smaller than that of the other veins. 74% of our 35 heart specimens had the traditional configuration of four orifices, and 31% were close to a short vestibule or funnel-shaped common vein, according to our anatomical analysis. Five venous orifices were present in 17% of patients, and 9% of them shared a left or right vein. In the conventional pattern, the right inferior PV follows the intercaval region, while the right superior PV follows the intersection of the right atrium and superior caval vein. The right PVs' orifices are located close to the atrial plane. The group led by Michel Hassaguerre demonstrated that atrial muscle sleeves that extend from the LA myocardium to the PV are essential for establishing the LA's electrical connection to the PVs. The majority (or 80%) of focal triggers are found in this myocardium. When the refractory period and/or conduction velocity are decreased, they are able to produce propagating wavelets, which may lead to re-entrant circuits and AF. These authors noticed that PV ectopy was not evenly distributed. Currently, the electrical separation of all PVs is the primary strategy of any transcatheter ablation procedure intended to treat AF. In laboratories with a lot of experience, this method has a relatively high success rate (> 85%) for treating patients with paroxysmal AF. Patients with persistent or permanent AF, according to some facilities, have a 65 percent success rate, though biatrial defragmentation and anatomical linear ablation with the goal of ending AF are typically required in addition to pulmonary vein isolation [2].

It was discovered that the organization of the myocyte bundles within the sleeves was quite complex. The arrangement of muscle fascicles appeared to be mesh-like and consisted of bundles that ran in a circular orientation and were connected to bundles that ran in a longitudinal orientation, in contrast to previous research. Anisotropic conduction across the bundles is triggered by this configuration, which could act as a focused trigger or micro re-entry, according to our hypothesis. The PVs' "AF initiation" function may also be actively supported by the presence of scattered fibrosis patches. The conditions necessary to sustain re-entry from either numerous wavelets or single high frequency rotors with fibrillatory conduction are all facilitated by atrial structure remodeling, myocyte sarcolemmal ion channels, and intercellular communication between myocytes. Myocytes undergo microscopic and molecular changes during remodeling, which are linked to altered protein production of ion channel constituents. Additionally, the atria undergo macroscopic structural changes, such as dilatation and/or fibrosis, during remodelling [3].

The reorganization of sarcolemmal ion channels causes variations in

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atrial action potentials. The repolarization time course is unable to respond to rate variations, refractory times are decreasing, and repolarization is accelerating. The primary alterations in action potentials are as follows: Reduced repolarization and refractory periods make it possible to form smaller re-entrant circuits and reduce the wavelength. Heterogeneous remodelling promotes fibrillatory conduction by promoting inhomogeneous sluggish conduction and block. The concept of "electrical remodelling" has been supported by a number of studies that sought to identify the action potential aberrations and alterations in ionic current that are associated with AF. Even though electrical remodelling of the sarcolemmal ion channels during AF causes action potentials of short duration with short refractory periods and possibly a decrease in sodium current, these changes alone may only partially explain AF susceptibility but not the onset of the arrhythmia. Another approach that may provide the substrate necessary to maintain re-entry in one or more small circuits is through gap junction cell coupling remodelling. The observed ionic changes and the altered gap junction cell coupling provide scant evidence for the onset of the arrhythmia [4].

The significance of atrial structural changes in providing the arrhythmia's vulnerable substrate has been the focus of the concept of a "second component," which is distinct from the electrophysiological changes associated with AF. It is unclear whether these changes, which occur at the levels of myocytes and extracellular matrix, take place prior to or subsequent to the onset of the arrhythmia. A clinical connection exists between abnormalities in the function of the sinus node (SN). Researchers first hypothesized that the SN might be to blame for the arrhythmia's persistence due to this result and the SN's characteristically slow conduction. Davies and Pomerance assert that short-term AF and long-term AF have distinct SNs. These researchers claim that while short-term AF has a normal SN, long-term AF has a lower proportion of specialized myocytes. The fibrotic changes in the node and atria could be caused by the arrhythmia and its subsequent disruption of chamber function, according to the authors. The proportion of nodal fibers in the sinus rhythm and AF patient groups, according to other authors, is comparable. The clinical data that are currently available indicate that the SN is probably passive during AF because atrial impulses enter the SN at a rate that is significantly faster than its intrinsic frequency. The link between SN dysfunction and AF is likely caused by disorders that simultaneously affect the atria and SN, rather than AF itself being caused by SN pathology [5].

Conclusion

Atrial fibrillation's complexity as a disease is probably due to multiple aetiopathogenic pathways. Current data indicate that this arrhythmia typically requires a trigger for start and a weak electrophysiological or anatomical substrate for maintenance, despite the lack of knowledge regarding the anatomical and functional foundations of AF. Whether the trigger mechanisms are focused improved automaticity, prompted activity, or micro re-entry from cardiac tissue is currently unknown. AF induction may be aided by parasympathetic and sympathetic stimuli, which appear to play important roles in AF maintenance.

In order to encourage anatomical re-entry or anchor rotors, the significance of structural discontinuities and diverse fiber orientation transmutably along the cardiac bundles in the fibrillatory process and the maintenance of AF is emphasized. Even though electrical remodelling is reversible, structural remodelling, which includes interstitial fibrosis and cellular deterioration, is irreversible. However, it still promotes re-entrant circuits and AF. A mechanism that may involve cellular ageing, apoptosis, and subsequent atrial fibrosis and inflammation is linked to both new-onset and recurrent AF, according to growing clinical data.

Acknowledgement

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Conflict of Interest

None.

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