Mini Review
Volume 10:6, 2022

Journal of Cardiovascular Diseases & Diagnosis

ISSN: 2329-9517 Open Access

Atrial Fibrillation is Triggered and Maintained by Anatomical Structures

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Abstract

The considerable underlying structural abnormalities are not taken into account by the description of atrial fibrillation (AF) as a functional electrical condition. Microstructural alteration of the muscular sleeve of the atrium and pulmonary vein (PV) creates a weak foundation for the maintenance of AF. Despite a lack of knowledge about the anatomical and functional underpinnings of AF, current data show that this arrhythmia typically needs a trigger for start and a weak electrophysiological and/or anatomical substrate for maintenance. It is currently unknown if the trigger mechanisms involve micro re-entry from cardiac tissue, prompted activity, or focused improved automaticity. Both sympathetic and parasympathetic stimuli, which also appear to be involved in AF maintenance, can promote AF onset. Inflammation is linked to both new-onset and recurrent AF through a mechanism that may involve cellular ageing, apoptosis, and ensuing atrial fibrosis, according to growing clinical data.

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

Introduction

Understanding the pathophysiology of atrial fibrillation is still lacking (AF). Given that this arrhythmia can coexist with a number of pathological conditions (such as mitral valve disease, hyperthyroidism, hypertension, coronary artery disease, etc.) and can also occur in a healthy heart, a condition known as "lone AF," current research points to a multifactorial pathogenesis for AF. It has been demonstrated that triggers coming from both PVs and non-PV sites account for the majority of cases of paroxysmal AF. Ectopic foci, however, might not necessarily be required for the onset and upkeep of AF. Decreased wavelength alone (a product of refractory period and conduction velocity) permitted the maintenance of several concurrent re-entry circuits, resulting in the formation of AF. The idea of electrical remodelling and the variations in ion channel number and function that go along with it have been linked to the progression of the illness and the idea that "AF begets AF." The persistence of the arrhythmia, or so-called chronic AF, has been linked to the presence of a vulnerable atrial structural substrate with areas of conduction block, which causes spatial separation of the wavelets and encourages re-entry. Atrial remodelling, which occurs in chronic AF, makes wave propagation more complex, multiplies sites with the highest dominant frequency (known as rotors), and moves these sites from the PV region towards the left atrium (LA) and/or right atrium. It is still unknown, nevertheless, whether the structural remodelling with interstitial fibrosis and myolysis features or a rise in autonomic tone are pro-fibrillatory variables or whether AF can be created or produced as a symptom of advanced age or underlying cardiac disease [1-5].

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Date of Submission: 03-June-2022, Manuscript No. JCDD-22-74808; **Editor assigned:** 06-June-2022, PreQC No. JCDD-22-74808; **Reviewed:** 17-June-2022, QC No. JCDD-22-74808; **Revised:** 23-June-2022, Manuscript No. JCDD-22-74808; **Published:** 30-June-2022, DOI: 10.37421/2329-9517.2022.10.500

Triggers of Atrial Fibrillation

Pulmonary veins

A variety of non-invasive imaging methods, including multidetector computed tomography and magnetic resonance imaging, have recently revealed that patient architecture of the pulmonary vein (PV) varies. The number of PVs is one manifestation of this variation; some patients have 5 separate ostia, whilst others have common trunks. 25% of patients have a common left or right pulmonary vein, with the left PVs appearing more frequently than the right ones. Another typical finding that can be found in up to 26% of patients is the presence of "additional" PVs, the most prevalent of which is a distinct right middle PV draining the middle lobe of the lung. This right middle PV's ostial diameter is smaller than that of other veins. Our anatomical investigation of 35 heart specimens revealed that 74% of them had the traditional configuration of four orifices, with 31% of them being located in the vicinity of a short vestibule or funnel-shaped common vein. 17% of patients had five venous orifices, and 9% shared a common vein on the left or right. The right superior PV follows the intersection of the right atrium and superior caval vein in the conventional pattern, while the right inferior PV follows the intercaval region. In close proximity to the atrial plane are the orifices of the right PVs. It is crucial to prove that the LA is electrically connected to the PVs through atrial muscle sleeves that stretch from the LA myocardium to the PV, as the group of Michel Hassaguerre proved. This myocardium is where the majority (80%) of focal triggers are found. They can produce propagating wavelets, which may result in re-entrant circuits and AF when there is a reduction in the refractory period and/or conduction velocity. These writers noticed that PV ectopy was distributed unevenly. Currently, the main approach of any transcatheter ablation operation that aims to treat AF is the electrical separation of all PVs. This method is associated

with a comparatively high success rate (> 85%) in patients with paroxysmal AF in laboratories with substantial expertise. Some facilities claim that patients with chronic AF (persistent or permanent) have a 65% success rate, although this typically necessitates additional treatments such biatrial defragmentation and anatomical linear ablation with the goal of terminating AF in addition to pulmonary vein isolation [6-8].

It was discovered that the myocyte bundles' organisation within the sleeves was quite intricate. In contrast to earlier studies, the arrangement of muscle fascicles appeared to be mesh-like and was composed of bundles that ran in a circular orientation and interconnected with bundles that ran in a longitudinal orientation. According to our hypothesis, such a configuration causes anisotropic conduction across the bundles, which alone could serve as a focused trigger or micro re-entry. Patchy patches of fibrosis, which were also found, may actively participate in the PVs' "AF initiation" function [5].

Structural Remodels that Aid in the Development and Maintenance of Af

Remodeling of the atrial structure, myocyte sarcolemmal ion channels, and intercellular communication between myocytes all contribute to the circumstances required to sustain re-entry from either numerous wavelets or single high frequency rotors with fibrillatory conduction. Remodeling entails microscopic and molecular changes in the structure and function of myocytes linked to changed protein production of ion channel constituents, as well as macroscopic structural changes in the atria such as dilatation and/or fibrosis. Atrial action potentials vary as a result of remodelling of sarcolemmal ion channels. Repolarization is accelerating, refractory times are getting shorter, and the repolarization time course isn't able to react to rate variations. These are the main alterations in action potentials. Repolarization and refractory period reductions result in a reduction in wavelength and enable the formation of small re-entrant circuits. With inhomogeneous sluggish conduction and block, heterogeneous remodelling encourages fibrillatory conduction. Several investigations, that sought to pinpoint the ionic current alterations and action potential aberrations connected to AF have supported the idea of "electrical remodelling." Although sarcolemmal ion channel electrical remodelling during AF results in short duration action potentials with short refractory periods and maybe a decrease in sodium current, these alterations alone may only partially account for AF susceptibility but not the onset of the arrhythmia. Gap junction cell coupling remodelling could be another way that provides the substrate required to maintain re-entry in one or more tiny circuits [9].

The modification of gap junction cell coupling and the observed ionic alterations offer scant evidence for the beginning of the arrhythmia. The idea of a "second component," separate from the electrophysiological changes linked to AF, has focused on the significance of atrial structural changes in providing the arrhythmia's vulnerable substrate. It is unclear whether these modifications, which are found at the myocyte and extracellular matrix levels, occur before or after the onset of the arrhythmia [10].

Sinus Node Function

Sinus node (SN) function abnormalities and AF have a clinical relationship. This result, along with the SN's characteristically slow conduction, first prompted researchers to hypothesise that the SN might be responsible for perpetuating the arrhythmia. The SN is different between long-term AF and short-term AF, claim Davies and Pomerance. According to these scientists, long-term AF has a lower proportion of specialised myocytes while short-term AF has a normal SN. The authors speculate that the arrhythmia and subsequent disturbed function of the chambers may be the cause of the fibrotic alterations in the node and atria. Other authors demonstrated that the percentage of nodal fibres in the sinus rhythm group and the AF patient group is comparable. The SN is likely passive during AF, according to the clinical data currently available, with atrial impulses entering the SN at a pace that is substantially quicker than its intrinsic frequency. Instead than SN pathology per se contributing to AF, the link between SN dysfunction and AF is likely caused by disorders that simultaneously impact the SN and atria [11,12].

Conclusion

Multiple aetiopathogenic pathways most likely contribute to the complexity of atrial fibrillation as a disease. Despite a lack of knowledge about the anatomical and functional underpinnings of AF, current data show that this arrhythmia typically needs a trigger for start and a weak electrophysiological and/or anatomical substrate for maintenance. It is currently unknown if the trigger mechanisms involve micro re-entry from cardiac tissue, prompted activity, or focused improved automaticity. Parasympathetic and sympathetic stimuli, which both appear to be important in AF maintenance, can promote AF induction.

The relevance of structural discontinuities and diverse fibre orientation transmurally along the cardiac bundles in the fibrillatory process and the maintenance of AF are highlighted, encouraging anatomical re-entry or anchoring rotors. Even though structural remodelling, which includes interstitial fibrosis and cellular deterioration, is irreversible, it nevertheless promotes re-entrant circuits and AF despite electrical remodelling being reversible. Inflammation is linked to both new-onset and recurrent AF through a mechanism that may involve cellular ageing, apoptosis, and ensuing atrial fibrosis, according to growing clinical data.

Conflict of Interest

The authors confirm that this article content has no conflicts of interest.

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How to cite this article: Brunzel, Darnell. "Atrial Fibrillation is Triggered and Maintained by Anatomical Structures." J Cardiovasc Dis Diagn 10 (2022): 500.