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Chapter

Cardiovascular Magnetic Resonance Imaging of Atrial Fibrillation: An Advanced Hemodynamic Perspective

Mankarman Ghuman, Hansuk Kim, Hana Sheitt and Julio Garcia

Abstract

Atrial fibrillation (AF) patients can be referred to cardiac magnetic resonance imaging (MRI) for an accurate assessment of cardiac function and left atrial structure. Cardiac MRI is the gold standard for the quantification of heart volumes and allows the noninvasive tissue characterization of the heart. In addition, advanced flow assessment can be achieved using 4D-flow MRI to elegantly depict the hemodynamic efficiency of the left atrium (LA) and left ventricle (LV) throughout the cardiac cycle. Patients with AF may have occult LV disease and thrombus formation. Biomarkers based on 4D-flow MRI may unmask the presence of LA/LV disease by quantifying 3D stasis, flow distribution, and vortex formation. These biomarkers have proved to characterize AF stages, to complement standard risk scores, and bring new insights on heart hemodynamic performance. This chapter aims to present a standard cardiac MRI protocol for atrial fibrillation and the innovative usefulness of advanced flow imaging in clinical settings.

Keywords: cardiac flow, atrial fibrillation, 4D-flow magnetic resonance imaging (MRI), hemodynamic biomarkers

1. Introduction

Imaging and quantifying various characteristics of blood flow throughout the heart is essential in modern-day cardiology. Accurate image representations help accurately identify many known types of cardiovascular diseases. Atrial fibrillation (AF) is one of the most common types of atrial arrhythmias encountered in adults, which can be characterized by an irregular and rapid heartbeat with uncoordinated atrial activation and ineffective atrial contraction [1, 2]. The Framingham Heart Study reported that AF developed in 37% of the population after the age of 55 years [3]. AF can be detected and/or confirmed through various image modalities since it originates in the atrial chambers of the heart. This arrhythmia causes multiple simultaneous electrical signals
Atrial Fibrillation (AF) is characterized by abnormal electrical activity within the atria leading to irregular electrocardiogram (ECG) patterns and atrial activity, loss of coordinated atrial contractions, and inadequate ventricular filling [4]. A patient with AF may experience symptoms, the most common of which include palpitations, shortness of breath, fatigue, dizziness, and chest pain [4].

AF can be caused by various factors. Individuals with previously existing cardiovascular diseases show higher signs of developing AF [4]. A common cause can be atrioventricular (AV) dyssynchrony, in which the normal AV contraction experiences delays due to the irregular conduction of the AV node [5]. AV dyssynchrony can be the reason behind the more common symptoms of AF since it directly affects the atria, causing it to operate in a fast and disorganized matter. There is a small fraction of patients who do not have previously existing heart problems yet still show signs of AF [4]. For these patients, AF can be a result of lifestyle choices, such as diet, lack of exercise, and smoking [6]. A recent multi-institutional study reported that 19% of patients newly diagnosed with AF (41% women) had an acute AF precipitant including mainly cardiac surgery (22%) and pneumonia (20%) followed in minor portion by myocardial infarction, pulmonary embolism, thyrotoxicosis, or alcohol intoxication [7].

A common mechanism for developing AF involves thrombogenicity. An increase in thrombogenicity within the heart can be a result of AF or can further increase the incidence of AF in patients who do not yet have it [8]. Intracardiac thrombi can be found in the atrial walls among patients with AF since the fast and irregular contractions of the atria can cause stress on the endometrial walls, resulting in damage, which promotes a hemostatic pathway to induce thrombus formation [9]. Another mechanism in AF involves hemodynamics. Specifically, decreased left ventricular (LV) hemodynamics can be affiliated with AF [10]. Maintaining adequate blood flow is important for overall cardiovascular health. Patients with AF typically show decreased hemodynamics; their low cardiac output can be attributed to irregular atrial contractions, causing their blood to begin pooling in the atria.

Mechanisms like increased thrombogenicity and decreased LV hemodynamics in patients with AF can lead to manifestations like stroke and ischemia. Increased thrombogenicity in the heart could cause a decrease in blood flow to the brain, resulting in a stroke [11]. A stroke would be a severe outcome as it drastically reduces overall body function and ability. Individuals with AF are much more likely to have a stroke in their lifetime than others since they have repeated incidents of irregular heartbeat [11]. This key symptom is what drives the formation of thrombi and can obstruct cardiac output. AF is associated with an increased incidence of stroke by a factor of four in men and 5.7 in women [12]. The risk of death increases by a factor of 2.4 among men and by a factor of 3.5 among women [13]. Reduced left atrium (LA) function increases the risk of blood stasis and clot formation in the LA, especially the left atrial appendage (LAA), which is a small extension of the LA. The LAA structure has high anatomical variability and has an important endocrine function. Its separation from the LA body promotes a blood turnover dependent on the systolic contraction. The loss of the LAA contraction during AF contributes to the increment of blood stasis and thrombus formation [14]. The CHA2DS2-VASc score considers the patient’s history of congestive heart failure, hypertension, diabetes mellitus, stroke, vascular disease, age (between 65 and 74 years and >75 years), and sex. It is currently used for the stroke risk stratification of AF patients and for the recommendation of anticoagulant therapy [1, 2, 15]. However, the CHA2DS2-VASc score does not include individual physiologic factors, which limit its prediction power.

Another manifestation, ischemia, can be a result of decreased LV hemodynamics. Ischemia is a condition observed when there is an obstruction of blood flow to a part of the body or organ. An ischemic stroke, a combination of the two manifestations, could
also be a possibility for individuals with AF as an obstruction in the circulatory system slows down cardiac output and causes low oxygen levels [16]. AF directly influences decreased cardiac output and can, therefore, be a leading factor in ischemic strokes seen in patients [1, 2]. At an early stage, an AF episode lasts <7 days of onset, and then, sinus rhythm is restored. This stage is known as paroxysmal AF. As severity progresses, AF episodes can last beyond 1 week. This stage is known as persistent AF. If the event does not terminate, it is considered as permanent AF. Persistent AF continues unless it is interrupted by electrical or pharmacological cardioversion, and it is associated with increased atrial fibrosis than paroxysmal AF [17]. Among patients undergoing cardioversion, up to 20% develop recurrent AF, and it becomes difficult to restore sinus rhythm [18].

There are many types of image modalities used to accurately diagnose several types of cardiovascular diseases. AF in patients can be identified using dedicated technology like electrocardiogram (ECG), echocardiogram (echo), cardiac computed tomography (CT), and cardiac magnetic resonance imaging (MRI). ECG is the best tool for recording electrical signals in the heart that correspond to the contraction and relaxation of the atria and ventricles. By performing ECG tests over long periods of time, a physician can recognize if there are any atrial arrhythmias like AF present by assessing the electrical patterns corresponding to the patient’s heartbeat [19]. In patients that do not display visible signs of arrhythmias, yet present with symptoms, a Holter monitor can be used to assess any irregular heartbeats the patient may experience in a 24-hour period [20]. In addition to an echo and ECG, a CT scan can be used for detecting AF in patients. Unlike an ECG, a cardiac CT is a scan of the heart that shows any calcium deposits in the coronary arteries and chambers of the heart. Used mainly on patients presenting with symptoms of coronary heart disease, a CT is a good indicator of any atherosclerotic plaque buildup. For a patient with AF, a CT angiogram is preferred as it accurately shows the interior of the heart [21]. Through this test, common AF manifestations like fast/disorganized atrial activation may be observed. An echo operates by implementing sound waves to create real-time images of the patient’s heart and can be useful in observing valve function and cardiac muscle health. In patients with AF, an echo will show how the heart contracts, and through that, observers will be able to notice how atrial contractions appear irregular and rapid [22]. An echo is accurate in displaying valve function and can show any abnormalities that may be associated with them, as well as with cardiac contractions.

Cardiac MRI has emerged as a valuable tool for interrogating the underlying substrate in AF patients. MRI uses a magnetic field to capture incoming radio waves from hydrogen atoms in various cells that respond to the magnification [23]. Like a CT scan, an MRI can provide highly accurate anatomical visualization. A cardiac MRI can additionally help analyze manifestations like atrial fibrosis and fat buildup around the heart walls. In addition to traditional MRI, four-dimensional flow (4D-flow) MRI is a very accurate and versatile way of visualizing and determining the size of biomarkers, including 3D stasis, vortices, changes in pressure, as well as flow distribution. 4D-flow MRI imaging helps achieve the precise visualization of the heart chambers, where manifestations of AF, including thrombus formations and hemodynamic efficiency, can be seen. The next section will introduce more in detail how new 4D-flow MRI approaches can accurately outline the progression of the AF.

2. Cardiovascular magnetic resonance for atrial fibrillation

In recent years, the innovation in interventional therapies and cardiac imaging for AF has motivated great interest and attention to a deeper understanding of the
atrial anatomic structure and function. In addition, the innovations in 3D blood flow assessment have revealed a new light in the effect that AF has on heart hemodynamics. The standard cardiac magnetic resonance protocol for AF aims to provide a detailed assessment of LV/LA structure and function (Figure 1) [24].

There is a clinical indication for pulmonary vein assessment preablation procedure [24–26]. The postablation imaging remains optional. The LA has a highly complex structure with close interaction between the anatomical, structural, and functional aspects. The LA imaging assessment aims to characterize the two parts of the LA: the posterior-superior inflow (venous) and the anteroinferior outflow (vestibular). Both the contrast-enhanced (CE) and noncontrast techniques can be used for the evaluation of the pulmonary veins. Noncontrast cardiac-triggered imaging with respiratory navigation-gating balanced steady-state free precession (bSSFP) can provide high-quality images in a short time for assessing the basic clinical questions [27]. CE can be performed with both the extracellular and blood pool contrast agents. The intravascular half-time life of the extracellular chelates ranges between 60 and 120 s [28]. The extracellular contrast agents have a rapid leakage into the interstitial space that reduces the enhancement of both arteries and veins shortly after injection. To obtain reasonable-quality images, it is recommended to initiate the acquisition immediately after the first pass of the contrast agent. A more recent strategy includes the use of ferumoxytol as blood pool agent, which has a much longer half-life, facilitating ultrahigh spatial resolution of both the arterial and the venous systems [29]. The voxel size difference achieved with ferumoxytol is one order of magnitude smaller compared with traditional acquisitions [30]. It is recommended to perform the breath-held 3D CE angiogram in the coronal projection encompassing the pulmonary veins and LA [24]. The use of an oblique plane centering the pulmonary veins can reduce the slab thickness but will lead to less coverage of the LA. When the patient has irregular rhythm, the ECG gating should be synchronized with systole. Three volumetric acquisitions are recommended: (1) precontrast, (2) first pass, and (3) after contrast administration. The precontrast acquisition serves as a reference for subtraction. A time-resolved multiphase (acquisition and contrast started simultaneously) angiogram can provide an isolated pulmonary phase image for reconstruction and integration with common ablation mapping software. Contrast should be injected at a 2–3-mL/s rate for an optimal result. Image slice thickness can be 1–2 mm with an in-plane resolution of 1–1.5 mm. An isotropic configuration is
preferred (i.e., 1 × 1 × 1 mm). A slab of 60–80 slices typically covers a normal heart, and the number of slices can be increased to encompass the volume of interest. Standard 2D phase-contrast acquisitions can be added to quantify flow through each pulmonary vein. Late-gadolinium enhancement can also be added to assess the LA wall for fibrosis [31].

3. Advanced hemodynamic assessment in AF using 4D-flow

Four-dimensional flow (4D-flow) has been developed to achieve a comprehensive acquisition of blood flow through the heart [32, 33]. Phase-contrast flow-encoding acquisitions are performed in all three spatial dimensions of space and time along the cardiac cycle (3D + time = 4D). This technique has existed since earlier 1990s; however, computational and hardware limitations limited its clinical applicability [34]. During the last decade, both computational power and hardware development have allowed for a realistic integration into clinical settings. In recent years, a scientific consensus and acquisition recommendations have been published with the purpose of standardizing the acquisition and analysis of 4D-flow data [35–37]. Acquisition parameters are optimized to provide the best possible imaging accuracy in each protocol. For AF imaging, the slab acquisition can be sagittal or axial with whole-heart coverage using isotropic spatial resolution (2–2.5 mm). Retrospectively ECG-gated acquisition with 30 phases is advised for adequate coverage of the cardiac cycle. A respiratory navigator can be used to reduce respiratory motion. Acceleration methods, such as parallel imaging or compressed sensing, can be used to achieve an acquisition time between 5 and 10 min. A sample of acquisition is illustrated in Figure 2.

There have been efforts to quantify 4D-flow measurements to improve diagnosis and evaluation of disease and risk assessment of AF (Table 1).

An initial application of 4D-flow is the generation of phase-contrast angiogram (PC MRA), which can be obtained by multiplying the velocity magnitude and the cine magnitude volumes. One of the advantages of PC MRA versus the CE MRA is that it can be obtained without contrast and allow us to obtain comparable anatomic characterization for the pulmonary veins (Figure 3). One limitation is the limited characterization of the LAA. The primary method of characterizing hemodynamics in LA, from 3D blood flow velocities, is to take an average from all voxels through a whole

![Figure 2](image-url)

**Figure 2.** Acquisition planning of 4D-flow. Region of interest covers the whole heart, as illustrated by the blue rectangle. The acquisition requires electrocardiogram gating and respiratory control, as it is shown by the small orange rectangle. Velocity encoding in each direction of the volume of interest is used to obtain velocity phases, which are subtracted from encoding reference to calculate blood flow velocities within the volume (X, Y, Z). The cardiac cycle average magnitude facilitates the anatomic visualization of the heart.
cardiac cycle or peak velocity. Although there is some contradiction between studies, most of the recent studies characterizing AF blood flow with relatively large cohorts agree that there is a significant decrease in mean and peak flow velocity in LA, even in paroxysmal AF patients with sinus rhythm [39–42, 44]. Most notably, the increase
in CHA_{2}DS_{2}-VASc score has been associated with reduced mean LA velocity [40, 41], which suggests that 4D-flow measurement may be able to improve risk assessment.

Kinetic energy, which is proportional to the mean square of velocity, was also markedly lower in AF patients than in controls [44]. Similarly, energy loss is also reduced (Figure 4). Left atrial flow stasis map proposed by Markl et al. [39] focuses on the flow
stagnation at individual voxel. This method counts the number of time frames under threshold velocity (0.1 m/s) at each voxel, which is supposed to increase the chance of thrombosis. The result can be shown as a map projected on the MRA image as well as the average ratio relative to the one whole heart cycle. Several studies have consistently reported flow stasis to be elevated in AF patients both in sinus rhythm and in fibrillation [39, 41, 42, 45]. An example of flow stasis is displayed in Figure 5.

In addition, flow patterns through the pulmonary vein into the LA have been studied [42]. The 3D asymmetrical configuration of the systemic pulmonary veins allows the development of vortical flow patterns during early diastolic LV filling while avoiding/reducing blood stasis [42]. AF patients often show LA inflow fragmentation and vortex formation in the LA (see Figure 6). Increment of vortex size can be observed in paroxysmal AF, and it is associated with higher risk score. Similarly, decreased LA velocity and increased LA blood flow stasis have also been reported in the LAA [41, 44]. However, 4D-flow MRI special resolution may not facilitate the accurate segmentation the LAA [35]. Despite the latter, 4D-flow parameters have shown excellent reliability and reproducibility in AF patients [45]. LA peak velocity and vorticity were found to be more reproducible and independent of physiological biomarkers than LA mean velocity, LA vortex volume, and blood flow stasis.

Figure 5.
Left atrial stasis maps. Sample of a patient 3D stasis map pre- and postablation showing regions with elevated stasis (red arrows). Larger stasis regions are indicators of possible thrombus formation. The region near the left atrial appendage typically shows elevated stasis after ablation.

Figure 6.
Evolution of atrial vortex formation in atrial fibrillation. A vortex typically forms during left atrial inflow and tends to disappear during ejection. However, in atrial fibrillation patients, small vortices remain in the atrium during the cardiac cycle.
There was an approach to associate risk factors with LA flow characteristics [46]. This study presented patients with moderate to high CHA\textsubscript{2}DS\textsubscript{2}-VASc scores have impaired LA flow parameters even though they have restored from arrhythmia or have no AF history. Recently, a novel sequence to evaluate 3D hemodynamics with a fully self-gated and free-running sequence, called 5D flow, has been proposed [47]. This method extracts cardiac and respiratory signals from SI projection signals, eliminating the need for ECG gating, and adds the respiratory phase as the fifth dimension by sorting acquired scan lines according to respiratory signal. This method can be extended to be used in AF patients by replacing the respiratory dimension with the RR length dimension to tackle the variability of arrhythmic heartbeats [48]. The study successfully found a correlation between flow parameters and AF burden with reasonable scan time (<10 min).

4. Conclusion

In conclusion, advances in cardiac magnetic resonance imaging can facilitate the assessment of cardiac function and left atrial structure. This chapter aimed to introduce a standard cardiac MRI protocol for atrial fibrillation. Advanced hemodynamics using 4D-flow can improve the assessment of the left atrium flow patterns and efficiency throughout the cardiac cycle. Novel flow biomarkers such as 3D stasis, kinetic energy, or vortex formation may unmask the presence of LA/LV disease in atrial fibrillation.

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Conflict of interest

The authors have no conflict of interest.
Author details

Mankanman Ghuman, Hansuk Kim, Hana Sheitt and Julio Garcia*
University of Calgary, Calgary, Alberta, Canada

*Address all correspondence to: julio.garciaflores@ucalgary.ca

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