Abstract: Atrial fibrillation (AF) is a common cardiac rhythm disturbance and its incidence is increasing. Radiofrequency catheter ablation (RFCA) is a highly successful therapy for treating AF, and its use is becoming more widespread; however, with its increasing use and evolving technique, known complications are better understood and new complications are emerging. Computed tomography (CT) of the pulmonary veins, or more correctly, the posterior left atrium (LA), has an established role in precisely defining the complex anatomy of the LA and pulmonary veins preablation and has an expanding role in identifying the myriad of possible complications postablation. The purposes of this article are: to review AF and RFCA; to discuss CT evaluation of the LA and pulmonary veins preablation; and to review the complications of RFCA focusing on the role of CT postablation.

Key Words: atrial fibrillation, pulmonary veins, left atrium, radiofrequency catheter ablation, CT

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ATRIAL FIBRILLATION

The most common of the sustained cardiac rhythm disturbances, atrial fibrillation (AF) is a supraventricular tachyarrhythmia that has an overall prevalence of 0.4%, but increases in incidence with age.1,2 Although rare in children, in adults, the incidence nearly doubles every 10 years affecting approximately 5% of the population over 65 years.3 It is estimated that approximately 2.2 to 2.5 million people in the United States are affected by AF which has significant clinical and economic consequences, accounting for as many as one-third of yearly cardiac dysrhythmia hospitalizations.4

AF is an important overall marker for cardiovascular risk and a major risk factor for stroke related to its 2 main complications: hemodynamic compromise and formation of thromboemboli.3 The loss of the atrial component of stroke volume (“atrial kick”), combined with heart rates that are either too fast or too slow to maintain an adequate cardiac output, leads to hemodynamic compromise, poor left ventricular function, and heart failure.3 The left atrial appendage (LAA) has been documented as the source of thrombi in 90% to 100% of nonrheumatic AF.3–6 Not only do up to 20% of all ischemic strokes occur in AF patients, but AF patients also have an 18 times higher rate of systemic arterial emboli than the general population.3–6

Normally, the sinoatrial node fires by self-excitation eliciting a single electrical impulse. This impulse rapidly spreads across the right atrium (RA) along defined electrical pathways and to the left atrium (LA) via Bachman’s Bundle.7 Synchronous atrial contraction forces blood into the ventricles. The speed of the electrical impulse is slowed by the atroventricular (AV) node before the impulse continues to the Bundle of His and is propagated through the interventricular septum via the right and left bundle branches causing synchronous ventricular contraction (Fig. 1).7

AF occurs when multiple ectopic electrical foci fire independently sending the AV node as many as 300 discharges per minute.7 The irregular ventricular response depends on the refractoriness of the AV node, vagal and sympathetic tone, and the presence of accessory pathways resulting in heart rates ranging from 30 to over 300 beats per minute.4,8 Although regular R-R intervals are possible, on an electrocardiogram (ECG), AF is characterized by a lack of P waves which are replaced by fibrillatory waves of varying morphology and frequency, with an irregularly irregular, often rapid, ventricular rhythm.4

Terminology describing AF can be confusing. Lone AF, accounting for 45% of AF cases, is defined as AF occurring in patients under 60 years of age without underlying cardiopulmonary disease.9,10 Paroxysmal AF (PAF) lasts less than 7 days and terminates spontaneously; whereas, persistent AF lasts at least 7 days and lasts indefinitely unless cardioverted. Both PAF and persistent AF can be recurrent, occurring more than once.4 With increasing age, recurrent episodes of PAF tend to become persistent as the LA undergoes electrical and structural remodeling.4 Permanent AF lasts longer than a year and sinus rhythm is not possible.4 Isolated AF is defined as AF occurring without associated atrial tachycardia or atrial flutter.4

Acute causes of AF include: recent surgery especially cardiothoracic surgery, acute myocardial infarction,
myocarditis, pulmonary embolism or other acute pulmonary disease, hyperthyroidism, electrocution, stimulants such as caffeine or alcohol or increased sympathetic or parasympathetic tone. Treatment of the underlying conditions can resolve the AF. However, AF is also associated with underlying structural heart disease, particularly mitral valvular disease, hypertension, and coronary artery disease. Other independent risk factors include: male sex, white race, age, diabetes, smoking, and obesity. With the increasing incidence of obesity, particularly childhood obesity, the incidence of AF is expected to significantly increase.

Ectopic foci responsible for the initiation of AF have been identified in the walls of the superior vena cava (SVC), both atria, the crista terminalis, ostium of the coronary sinus, interatrial septum, and the muscular sleeves of the distal pulmonary veins. The importance of the pulmonary veins in the initiation of AF is now well established. The myocardium of the LA extends a variable length into the distal pulmonary veins with the myocardial sleeves of the superior and left pulmonary veins longer than those of the inferior and right pulmonary veins. Over 90% of ectopic beats initiating AF arise from the pulmonary veins, 50% from the left superior pulmonary vein alone. Therefore, the pulmonary veins and posterior atrial wall have become important targets of interventional therapies.

**RADIOFREQUENCY CATHETER ABLATION**

Although treatments for AF include direct electrical cardioversion or chemical cardioversion with antiarrhythmic agents, these are of limited success, with AF often refractory to or recurrent after the treatment. In addition, these require the use of long-term anticoagulation therapy. Newer therapies such as the surgical Cox-Maze procedure, cryoablation and radiofrequency catheter ablation (RFCA) are aimed at causing anatomic scars to disrupt electrical communication between the ectopic foci of the pulmonary veins and the LA body. If successful, long-term anticoagulation is unnecessary.

RFCA, which is predominantly used for PAF, less often for persistent AF, is still under investigation and is a rapidly evolving therapy. As originally described by Haissaguerre et al in 1994, point ablation of site-specific arrhythmogenic foci within the walls of the distal pulmonary veins has subsequently proven to have a success rate of approximately 47%, often requiring repeat procedures and multiple veins, and has been associated with a high risk of pulmonary vein stenosis. The trend since then has been away from identifying the specific site of origin or trigger point of AF, and to increase the number of ablation lesions, thus increasing the volume of ablated or electrically isolated substrate for the AF, the left atrial myocardium. Circumferential, also known as segmental, ablation of the extrastriatal region of the pulmonary vein(s) increased the success rate to 67%. To minimize the potential of recurrence of AF and the need for repeat ablation procedures, more recent advances involve posterior left atrial ablation which is circumferential ablation of the pulmonary venous inflow vestibules bilaterally. Success rates in patients without underlying structural heart disease have increased to 88%, resulting in the more widespread clinical use of RFCA for AF (Fig. 2). The 2005 worldwide RF catheter compilation reported that the numbers of AF ablations have increased every year since 1995 when 18 patients underwent the procedure to a total of 8745 patients in 181 centers. Circumferential extrastriatal ablation and posterior left atrial ablation for segmental isolation of the pulmonary veins are the current most widely used techniques; point ablation within the distal pulmonary veins has been abandoned.

Technically, RFCA has several procedural variations, understanding the common features and challenges are important for both pre-RFCA and post-RFCA computed tomography (CT) evaluation. The procedure time is long, typically lasting several hours, and is performed with the patient under general anesthesia. This requires endotracheal or oro-tracheal intubation and the use of high frequency ventilation to minimize respiratory motion. Transesophageal echocardiography (TEE) can be performed preoperatively or intraoperatively to exclude the presence of LAA thrombus, a contraindication to the procedure.
Patients are typically in or cardioverted to sinus rhythm before the ablation. During the cardiac cycle, not only is the LA actively contracting, but so are the distal pulmonary veins which have phasic variation. The orientation, caliber, and flow volumes through the pulmonary veins' ostia are dynamic over the R-R interval.⁴⁵,⁴⁷,⁴⁸ Under fluoroscopic guidance, using a transfemoral approach, the LA is accessed indirectly via the RA through either a patent foramen ovale or transeptal (fossa ovalis) puncture.⁸,⁴⁹

The ablation catheter has to be precisely manipulated within the moving LA circumscribing each ostium or vestibule with contiguous, but not overlapping ablation lesions. Current ablation electrodes only cover a few millimeters of space, thus, depending on the circumference of the pulmonary veins' ostia or vestibules, atria require varying numbers of individual ablation lesions to create the intended, contiguous, circumferential lesion. There is an institutional variability in the choice of an ablation electrode with more recent trends including both the use of larger electrodes with higher RF power and smaller electrodes with lower RF power.²³ During an ablation, lesion depth, extent, and volume of ablated tissue are directly related to the size of the ablation electrode (3.5 to 8 mm), the RF power delivered (20 to 85 W, 55°C), and the amount of time (15 to 30 s) the active electrode is applied to the endocardium for each lesion.³⁰–³³

The use of pre-RFCA CT or magnetic resonance imaging (MRI) to show the 3-dimensional anatomy of the posterior LA has obviated the need to perform retrograde pulmonary venography during the ablation. The 3-dimensional LA models created in CT or MRI are referenced during the ablation, but real-time intraoperative imaging is still required. Intracardiac echocardiography is used to help insure direct contact between the ablation electrode and the moving endocardial surface as the RF energy is applied.³⁴,³⁵ A sensor mounted near the distal electrode of the ablation catheter which uses synchronous extracorporeal magnetic fields accurately chronicles the progress of the ablation catheter in 3-dimensional space but does not show anatomic detail. In an attempt to solve this, in some laboratories, these electromagnetic maps are fused with the preoperative 3-dimensional CT or MRI anatomic models, to yield electroanatomic maps. However, the efficacy of using this static representation of a dynamic environment is unproven and under investigation (Fig. 3).²⁸,³⁶

**PRE-RFCA CT EVALUATION**

**Technique**

Since its first published descriptions, multidetector CT (MDCT) angiography of the LA and pulmonary veins (PV CT) for the evaluation of AF patients before RFCA has become widely accepted. It rapidly and accurately illustrates the complex 3-dimensional anatomy
of the posterior LA, distal pulmonary veins, and the adjacent mediastinum providing the necessary anatomic information for successful ablation. MRI is comparable with CT for the definition of posterior left atrial anatomy with the choice of imaging technique influenced by patient and institutional factors.37 Preablation, the main goals of scanning are to delineate and display the left atrial and distal pulmonary venous anatomy in a 3-dimensional model and to identify any variant anatomy or significant incidental that may interfere with the ablation. All patients at our site are scanned approximately 24 hours before ablation and because RFCA patients typically have PAF, their rhythm may be AF or sinus at the time of CT. ECG gated technique is used for patients in sinus rhythm and non-ECG gated technique is used for patients in AF. Four slice and higher scanners allow accurate characterization of posterior atrial and pulmonary vein anatomy with both ECG gated and non-ECG gated scans.34 The increased gantry speed and larger detectors of the 16-slice and 64-slice scanners offer the advantages of decreased scan time, decreased cardiac motion, and nearly isotropic data sets which improve image quality, even without gating.

The use of dual barrel injectors is preferred, as the saline flush helps reduce artifact from dense contrast in the SVC and RA. We avoid test bolus timing in patients scanned while in AF as, in our experience; this frequently results in suboptimal LA opacification presumably due to underlying heart rate variability (and therefore altered transit time) between timing bolus evaluation and scan time. Scan parameters for ECG gated and non-ECG gated scan techniques on 16 and lower slice scanners are detailed elsewhere.34,38–41 Our technique on the 64-slice scanner for non-ECG gated examinations is equivalent to that on the 16-slice scanner. However, for ECG gated examinations on the 64-slice scanner, we found that modifying our injector protocol to the 3-phase scanning bolus used for coronary artery CT angiography works well. In our experience, test bolus timing for the aortic root instead of the LA, allows for the potential of slow LAA filling without compromising LA enhancement.

Following image acquisition, the ECG gated images are reconstructed at the desired phase of the R-R interval (usually 85%). The axial source images are reviewed and 3-dimensional models of the LA, LAA and distal pulmonary veins are created from both epicardial (extra-atrial) and endocardial (intra-atrial) vantages. Ostial or pulmonary vein measurements are performed using multiplanar reformatted images orthogonal to the pulmonary vein ostia. Maximum pulmonary vein ostial diameters occur during late atrial diastole (approximately 85% R-R) and minimum diameter occurs during atrial systole (approximately 15% R-R)28 (Fig. 4). Using steady state free precession cine MRI, Lickfett et al28 reported a 32.5% phasic change in pulmonary vein ostial diameters and a 7.2-mm mean phasic change in ostial positions. Using ECG gated MDCT, Choi et al27 documented similar phasic variation of the pulmonary vein ostia.

Because of the dynamic nature of the pulmonary veins, we advocate noting the phase that measurements were obtained in for gated examinations and measuring on the same phase in subsequent examinations. For non-ECG gated examinations, although the pulmonary veins can be accurately identified and 3-dimensional models can be made, there is no way to determine which phase or phases of the cardiac cycle were occurring during scan acquisition or if the same phase(s) were imaged on subsequent examinations.

**Interpretation**

Important information includes the identification of the number and configuration of pulmonary veins including the presence of accessory, conjoined, or ostial branches. These were once considered rare before the introduction of 3-dimensional-MDCT for the delineation of pulmonary venous anatomy.39,40 Even the original MRI literature describing the use of MRI for the

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**FIGURE 4.** "Pulmonary vein phasic variation": cross sections of the RSPV ostium obtained from orthogonal oblique reformats at 15% (A) and 85% (B) of the R-R interval showing the normal caliber changes between atrial systole and diastole in a patient with PAF pre-RFCA. Note the dense contrast in the SVC in this scan that was obtained before the availability of dual barrel injectors with a saline flush.
delineation of pulmonary venous anatomy only referred to its usefulness for identifying the 4 pulmonary veins. Conventional anatomy, occurring in only 60% to 70% of the population, regardless if they are AF patients, is 4 pulmonary veins. Typically, the right superior pulmonary vein (RSPV) drains the right upper (RUL) and middle lobes (RML), the right inferior pulmonary vein (RIPV) drains the right lower lobe (RLL), the left superior pulmonary vein (LSPV) drains the left upper lobe (LUL) including the lingula (LNG) and the left inferior pulmonary vein (LIPV) drains the left lower lobe.

When variations occur, the right side tends to be complex and has one or more accessory veins, whereas the left side tends to be more simplified often having the veins of the left lung converge proximal to the atrio-pulmonary venous junction into a short or long common trunk which drains into the LA (Fig. 5). Conjoined veins rarely occur on the right or bilaterally. An accessory vein has its own independent atrio-pulmonary venous junction separate from the superior and inferior pulmonary veins; whereas an ostial branch does not have an atrio-pulmonary venous junction, but empties into the proximal portion of a vein within 5 mm of the atrio-pulmonary venous junction. Accessory veins are important as their ostia at their atrio-pulmonary venous junctions are typically small, exposing them to increased risk of stenoses. Similarly, because ostial branches drain into a vein within 5 mm of the atrio-pulmonary venous junction, they are also at risk, but less so now that point ablations within the distal pulmonary veins are no longer performed. Anatomically, the correct way to identify accessory veins or ostial branches is to investigate the lung windows and verify the vein origin, not where the pulmonary vein enters the heart. This is clinically important, because if complications such as hemodynamically significant pulmonary venous stenosis or thrombosis occur, the associated pulmonary parenchymal abnormalities will be reflected in the segment or lobe of lung drained by the affected vein.

Accessory veins are much more common on the right. Lacomis et al reported accessory veins in 33% of both AF and non-AF patients, with 87% of them occurring on the right. Marom et al reported similar findings with a 28% incidence of accessory veins with 100% of these on the right side. The 2 most common accessory veins are the right middle (which drains all or part of the RML), accounting for 55% to 93% of accessory right-sided veins; and the superior segment RLL vein accounting for 28% of accessory right-sided veins (Fig. 6). Multiple separate veins can also drain the basilar segments of the RLL. Often, any third vein interposed between the RSPV and RIPV has been generically termed a “right middle vein,” regardless of which lobe it was draining. From the electrophysiologists’ perspective, this is true, as during an ablation, they are only viewing the ostia of the pulmonary veins at the atrio-pulmonary venous junction. In addition, Tsao et al included ostial branches as accessory veins accounting for their reported 84% incidence of “right middle” veins.

The other most notable accessory vein on the right is the “top vein,” which enters the roof of the LA superomedial to the RSPV (Fig. 7). In our series, the top vein drained either the superior segment RLL or the posterior segment RUL or a combination of the two.
Accessory veins are much more infrequent on the left; usually draining all or part of the lingula. These also on occasion have been referred to as "middle veins." Any abnormality of the LA or the interatrial septum should also be reported including LA diverticulum, interatrial septal aneurysms, and atrial septal defects (Fig. 8). ECG-gating and isovoxel data sets are allowing more detailed evaluation of the interatrial septum, roof, and anterior LA walls. It is typical to see a thin septa extending from the fossa ovalis region anteriorly and superiorly along the atrial wall and/or a variable sized, blind-ending, diverticulalike outpouching near the RSPV from the antero-superior portion of the LA, both presumably vestigial remnants (Fig. 9). The LAA should be scrutinized for the presence of thrombi but TEE remains the standard of reference for their exclusion.

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It is crucial to alert the electrophysiologist to systemic venous variants, particularly azygous continuation of the inferior vena cava where the RA is not accessible inferiorly as for technical reasons, transeptal puncture and posterior left atrial navigation cannot be performed from a superior approach (Fig. 10). Other anatomic variants which are clinically important but do not preclude RFCA include anomalous veins such as a Scimitar vein (partial anomalous pulmonary venous
return to the inferior vena cava), partial anomalous pulmonary venous return to the RA, SVC, or left brachiocephalic vein. Persistence of a left-sided SVC is important owing to its course toward the coronary sinus along the ligament of Marshall between the ostia of the left pulmonary veins and the LAA, thus along a typical ablation line. Any other anatomic abnormalities or variants in the organs or vasculature adjacent to the posterior LA, particularly if they have any mass effect on the posterior LA, including esophageal dilatation and descending aortic aneurysms, are particularly noteworthy.

Incidental findings are reported at approximately 15%, similar to the rate for coronary artery CT angiography and have included: lung carcinoma and indeterminate lung nodules, pneumonia, bronchiectasis, emphysema, pulmonary fibrosis, sarcoid, enlarged lymph nodes, coronary artery disease, thoracic aortic aneurysms, hiatal hernias, pleural and pericardial effusions. Additionally, extrathoracic findings have included: hepatocellular carcinoma, adrenal and renal lesions (Fig. 11).41,45,46

**POST-RFCA CT EVALUATION**

The goal of post-RFCA imaging is to evaluate for complications of the procedure. There are no established recommendations for post-RFCA CT follow-up. Some sites acquire PV CT at one or more intervals, typically between 3 to 12 months post-RFCA to screen for PV stenosis; whereas, others only acquire CT if clinical suspicion of a complication arises.

The catheter compilation survey revealed an overall 6% major complication rate, but with the increased use of RFCA, and the more extensive ablation techniques, the incidence and variety of complications is anticipated to increase with the full gamut of possible complications probably not yet encountered.22,25,30,31,50 Minor complications include: small pleural and pericardial effusions including postpericardiotomy syndrome; mild, not hemodynamically significant pulmonary vein stenosis; and local complications related to femoral vein catheterization including catheter site hematomas and arteriovenous fistula.32,33,51

Major complications can manifest themselves hyperacutely in the electrophysiology laboratory; acutely within hours to days of the ablation; subacutely within weeks of the ablation; or delayed, months after the ablation. Major complications include: pulmonary vein dissection or cardiac perforation causing hemopericardium and tamponade; severe pulmonary vein stenosis or thrombosis causing venous infarcts, veno-occlusive disease, and pulmonary artery hypertension; coronary spasm causing coronary ischemia; thromboembolism.
causing stroke, transient ischemic attack, retinal occlusion, myocardial infarction, pulmonary emboli, systemic emboli; phrenic nerve palsy or paralysis; aspiration pneumonia; cutaneous radiation damage; and bleeding associated with anticoagulation. More recently reported complications include: LA intramural edema, LA intramural hematoma, LA dissection, disseminated intravascular coagulation, and atrioesophageal fistula (AEF). Radiologists should particularly familiarize themselves with 2 of the major complications that can present acutely but typically have a subacute or delayed presentation: pulmonary venous stenosis/thrombosis and AEF. The interpreting radiologist should be informed by the referring physician of any history of pulmonary vein ablation to correctly consider the presence of such complications and, given the consequences of delayed diagnosis in both of these entities, vigilance by both is warranted.

Pulmonary Vein Stenosis

Approximately 1% to 10% of patients undergoing RFCA for AF develop some degree of pulmonary vein stenosis; although the true prevalence is uncertain, this contradicts reports of much higher prevalence rates in the early literature related to the definition of pulmonary vein stenosis and imaging modalities for diagnosis. The abandonment of point ablations for segmental and posterior left atrial ablations, the use of intracardiac echocardiography, and decreased ablation temperatures and energy delivery, have all contributed to the decreased incidence of moderate to severe stenosis to approximately 1.4% or less in experienced hands. The latter is an important point, as higher rates have been reported in novices, and the use of RFCA for AF is continuing to expand to more centers.

Pulmonary vein stenosis has been documented as long as 2 years postablation. When severe, it can result in complete thrombosis of the pulmonary vein resulting in venous infarcts, pulmonary veno-occlusive disease and pulmonary artery hypertension. Although the presence of venous infarcts often announces itself with sharp, pleuritic chest pain and hemoptysis, the symptoms of pulmonary vein stenosis before the onset of hemoptysis are typically nonspecific consisting of dyspnea, cough, and vague chest pain. The symptoms are often present for weeks to several months and findings on both chest radiographs and ventilation perfusion (V/Q) scans are also nonspecific contributing to errant or delayed diagnosis. Chest radiographs are either normal or can show localized patchy airspace opacities representing edema or venous infarcts, with or without pleural effusions. There are V/Q mismatches with perfusion defects in the areas of normal ventilation. The parenchymal opacities on chest radiographs and the perfusion defects on V/Q scans occur in the lobe(s) or segment(s) of lung drained by the affected vein(s).
radiograph and V/Q scan findings has contributed to the not infrequent phenomenon of patients with moderate to severe pulmonary vein stenosis initially misdiagnosed and treated for bronchitis, pneumonia, or other parenchymal lung processes, and pulmonary embolism. The delayed and incorrect diagnoses also contribute to the uncertainty of the true prevalence of post-RFCA pulmonary vein stenosis.

Similar to pre-RFCA imaging, both CT and MRI are used for evaluation of the pulmonary veins post-RFCA, but in our experience, CT has the advantage of versatility of rapid evaluation for many of the other potential complications. ECG-gated PV CT is ideal, but if pulmonary vein stenosis was not clinically suspected, and a PV CT was not obtained, the findings of severe stenosis should be identifiable on a standard enhanced chest CT, particularly with the typical thin slice, isovoxel, helical acquisitions that are routine today. Caution for over diagnosing mild stenosis is warranted if a non-ECG gated acquisition has been obtained given the known variability of pulmonary vein size throughout the cardiac cycle. If a pre-RFCA scan is available, it should be used as a baseline. Accurate identification of the location of the stenosis and measurement of the length of the stenosis are readily accomplished with PV CT. Absence of contrast within one of the pulmonary veins is likely thrombosis but may represent severe stenosis and subtotal occlusion. Packer et al in their series of 23 patients with stenoses noted that some pulmonary veins which appeared occluded on CT were still patent on venography. New pulmonary vein narrowing, particularly in association with ancillary findings is worrisome for severe stenosis.

Ancillary findings include: infiltration of the adjacent mediastinal fat secondary to edema; enlarged reactive lymph nodes; peripheral parenchymal lung opacities representing venous infarcts; and localized septal thickening related to veno-occlusive disease and local pulmonary arterial hypertension (Fig. 12). Pulmonary vein balloon angioplasty and stenting is emerging as a treatment option for symptomatic pulmonary vein stenoses postablation with rapid amelioration of symptoms (Fig. 13). Recurrence of symptoms is suggestive of either in-stent or in-segment restenosis and early experience by both Packer et al and Qureshi et al suggest the recurrence rate is high ranging from 47% to 57% (Fig. 14). The accuracy of CT for detecting in-stent restenosis has not been established.

**Atrioesophageal Fistula**

AEF is a known rare but devastating complication of surgical posterior left atrial ablation related to thermal injury of the esophagus through the LA wall during the procedure. In 2004, Pappone et al reported the first 2 cases, from 2 different centers, after circumferential pulmonary vein RFCA; 1 patient died of multiple embolic events and the other survived after emergency cardiac and esophageal surgery. Since 2004, there have been a total of 4 reported cases of AEF and one of esophageal perforation without AEF following RFCA; however, AEF is thought underreported. Mortality rates are high, exceeding 50%. Causes of death include: massive air emboli, overwhelming sepsis, and massive hematemesis. Delay in the diagnosis has contributed to the high mortality. AEF

![Figure 12: “Pulmonary vein stenosis and thrombosis”: axial CT source images (A, B) and coronal reformat (C) show absence of contrast within the LSPV and LIPV consistent with severe PV stenosis or thrombosis in a patient who was admitted to a hospital with dyspnea and hemoptysis. Note the infiltrative changes in the mediastinal fat (A–C), areas of consolidation consistent with venous infarcts with adjacent marked pleural thickening (A, D), and localized septal and intralobular septal thickening (D). Subsequent history revealed the patient had undergone RFCA for AF with point ablations in the LSPV and LIPV 4 months earlier. At pulmonary venography, the LSPV was thrombosed and the LIPV was severely stenosed. The calcifications within the areas of infarct (a-arrows) are in small branch veins related to the longstanding nature of the venous occlusion.](image-url)
reported secondary to a thermal injury of the posterior LA with disruption of the LA as the inciting event has not been reported.

Multiple factors account for the risk of esophageal injury during ablation; these relate to the anatomy and ablation methods. The anatomic relationships putting the esophagus at risk are well described by several authors and are briefly reviewed here.47,73,74 The esophagus and

FIGURE 14. “In-stent restenosis”: oblique reformat (A) and cross-section (B) through the stent 1 year after patient in Figures 12 and 13 underwent stenting of the LIPV shows new soft tissue thickening surrounding the contrast within the stent consistent with neointimal hyperplasia and in-stent restenosis. The patient was complaining of increasing dyspnea prompting the CT evaluation.

FIGURE 13. “Treatment of pulmonary vein stenosis”: axial CT source images (A, C) and cross-sectional image through the stent (B) 6 months after patient in Figure 12 underwent successful angioplasty and stenting of the LIPV, showing widely patent stent (A, B). In (C), note that although the LSPV is still occluded, the LUL infarcts and adjacent pleural thickening have retracted, and the septal and intralobular septal thickening have improved.
the posterior LA are both thin-walled, typically < 5 mm thick.47,73,74 A thin layer of fat is usually interposed between the esophagus and the posterior LA, which may serve as an insulator; there are also variable bare areas where there is direct posterior left atrial-esophageal wall contact.47,73,74 The exact location of the esophagus along the posterior LA is not constant, although it usually takes 1 of 2 routes: running vertical and parallel to the left-sided veins or obliquely from the LSPV towards the RIPV; in addition, there is dynamic peristaltic motion of the esophagus.47,73,74

The evolution of RFCA away from point ablations within the veins, to circumferential pulmonary vein ablation, and posterior left atrial ablation is thought to account for the rising incidence of AEF, as no cases of AEF had been reported when point ablations were performed.31,50,55,69,73,74 Of note, though the ablation temperature and energy delivery had initially decreased as reports of pulmonary vein stenoses were increasing, as the target shifted away from the pulmonary veins and to the posterior left atrial wall, the size of the ablation electrodes trended up. The larger ablation electrodes capable of delivering higher energy with higher temperatures have been implicated as causal.31,50,55 However, we recently encountered an AEF at our site that occurred with a smaller 3.5-mm-tip catheter while delivering lower energies and temperatures. Operator experience may also have a role but the reported cases of AEF to date have included high volume centers with experienced electrophysiologists. Repeat ablation procedures or crossing ablation lines are also implicated as increasing the risk of injury.31,49,50,64–69

AEF has been diagnosed 2 days to 3 weeks postablation. In the reported cases, retrospectively, some type of symptom, usually nonspecific chest pain, occurred much earlier, often immediately after the ablation.31,50,55,68 The clinical signs and symptoms vary and can wax and wane as the localized injury progresses from the initial injury to an inflammatory esophagitis, mediastinitis, and AEF. Dysphagia, fever, and mild elevations in the white blood cell count are early signs.31,50,55,68 Pleuritic chest pain, higher fevers, and higher white blood cell counts occur as the mediastinitis progresses.31,50,55,68 When the posterior left atrial wall is breached, air, and bacteria from the esophagus can enter the LA and systemic circulation. Signs of endocarditis and sepsis predominate followed by signs of systemic embolism which may be either air or septic.31,50,55,68 The clinical course can rapidly deteriorate with mental status changes, stroke, seizures, hematemesis, and ensuing cardiovascular collapse.31,50,55,68

TEE and esophagoscopy with CO₂ inflation are contraindicated if there is clinical suspicion of AEF because of the risk of massive systemic air embolism.30,69 Chest CT with thin section collimation and intravenous contrast is the modality of choice for diagnosis. If oral contrast is used, it should be water-soluble only. The findings are those of mediastinitis, and/or endocarditis, but there are few CT images in the literature.68 Early findings may be subtle and careful comparison with the pre-RFCA examination is warranted. The mediastinitis is centered on the posterior left atrial-esophageal region; findings include: infiltrative changes in the mediastinal fat and small fluid collections or gas locules interposed between the esophagus and posterior LA (Fig. 15). Contrast extravasation from the LA has not been reported. The defect in the anterior wall of the esophagus...
was visualized in the esophageal perforation case. As the process evolves, the wall of the posterior LA may become thickened and irregular or develop a pseudoaneurysm; gas locules can occur within the pericardial space, or within the wall or lumen of the posterior LA (Fig. 16). Although small pleural or pericardial effusions are common post-RFCA findings, pleural or pericardial enhancing loculations which rapidly accumulate should raise suspicion.

In addition, head and abdominal CTs are used to evaluate the neurologic symptoms, fever, elevated white count, and abdominal symptoms related to embolic events. Head CT can show infarcts or septic emboli. Abdominal CT can show solid organ or bowel infarcts or other sequelae of embolic events.

Emergent treatment requires medical management of the sepsis combined with surgical exploration and operative repair of both the posterior left atrial wall and, similar to a Boerhaave syndrome, resection and diversion of the esophagus while the mediastinitis is treated. Earlier diagnosis of the primary esophageal injury, before the inflammatory process extends to the adjacent atrial wall, may decrease the morbidity and mortality. Bunch et al successfully managed the solitary esophageal perforation with temporary stenting of the esophagus using a self-expanding plastic stent.

CONCLUSIONS

CT of the pulmonary veins has evolved as a practical imaging modality to evaluate AF patients undergoing RFCA. Preablation, it is used to reliably define the often complex 3-dimensional anatomy of the posterior LA and distal pulmonary veins providing the necessary anatomic information for successful ablation. The importance of CT for postablation evaluation of complications continues to increase. As the techniques for catheter ablation of AF continue to change and evolve, and as the use of catheter ablation extends to more centers, radiologists must be able to recognize the imaging findings of known complications while staying alert for potential emerging ones.

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