Review Article

Safety and Complications of Pulsed Field Ablation for Atrial Fibrillation: Biophysics, Diagnosis, and Management

Alejandro Velasquez,[1] Gabriel Odozynski,[2] Heloisa Gonzaga Menezes de Moraes,[3] Leandro Ioschpe Zimerman,[4,5] Andre d'Avila[2,6]

Beth Israel Deaconess Medical Center,[1] Boston, Massachusetts – USA
Harvard-Thorndike Electrophysiology Institute – Electrophysiology Institute,[2] Boston, Massachusetts – USA
Escola Bahiana de Medicina e Saúde Pública – Unidade Acadêmica Brotas,[3] Brotas, BA – Brazil
Hospital de Clínicas de Porto Alegre,[4] Porto Alegre, RS – Brazil
Associação Hospital Moinhos de Vento,[5] Porto Alegre, RS – Brazil
Hospital SOS Cardio,[6] Florianópolis, SC – Brazil

Abstract

Pulsed field ablation (PFA) uses irreversible electroporation to preferentially target myocardial tissue during atrial fibrillation (AF) ablation while limiting collateral injury to adjacent structures. This narrative review summarizes complications associated with PFA, focusing on the mechanisms, incidence, diagnosis, prevention, and management of procedural and energy-specific adverse events. Evidence was drawn from 14 major trials, registries, and postapproval studies selected for the comprehensiveness of their safety data. Complications were classified as vascular, cardiac, pulmonary, hematologic, kidney, neurological, or gastrointestinal. Despite substantial heterogeneity in study design, reported complication rates were generally low. PFA appears to markedly reduce traditional thermal complications, with no cases of pulmonary vein stenosis or atrioesophageal fistula reported in the major studies reviewed. However, PFA-associated adverse events include intravascular hemolysis, hemoglobinuria-associated acute kidney injury, coronary vasospasm, arrhythmias, vasovagal responses, and transient phrenic nerve injury. Clinical neurological events, such as stroke and transient ischemic attack, remain uncommon, although silent cerebral lesions are detected on postprocedural magnetic resonance imaging. Preventive strategies include careful vascular access, intracardiac echocardiography-guided transseptal puncture, strict periprocedural anticoagulation, limiting the number of applications, adequate catheter-tissue contact, hydration, and nitroglycerin administration when ablating near coronary arteries. Management of vagal responses requires readiness for temporary pacing or anticholinergic therapy. PFA represents a major advance in the safety of AF ablation by reducing severe thermal injuries, although electroporation-related complications require specific attention. Safe adoption requires prevention and management strategies informed by complication mechanisms as real-world use expands.

Keywords

Irreversible Electroporation Therapy; Atrial Fibrillation; Catheter Ablation; Electroporation

Mailing Address: Alejandro Velasquez •
Beth Israel Deaconess Medical Center, 330 Brookline Avenue, Suite ES 217 (Department of Graduate Medical Education) / Deaconess Building, Suite 306, Boston, MA 02215
E-mail: avelasq4@bidmc.harvard.edu
Manuscript received May 14, 2026, revised manuscript August 23, 2026, accepted September 16, 2026
Editor responsible for the review: Mauricio Scanavacca

DOI: https://doi.org/10.36660/abc.20260389i

Introduction

Catheter ablation has become a first-line rhythm control strategy for symptomatic paroxysmal or persistent atrial fibrillation (AF).[1] Historically, thermal modalities, including radiofrequency ablation (RFA) and cryoballoon ablation, have been the standard approaches to pulmonary vein isolation (PVI). Their nonselective thermal effects can cause collateral injury to adjacent structures, resulting in rare but serious complications, such as atrioesophageal fistula, pulmonary vein (PV) stenosis, and persistent phrenic nerve palsy.[1-3]

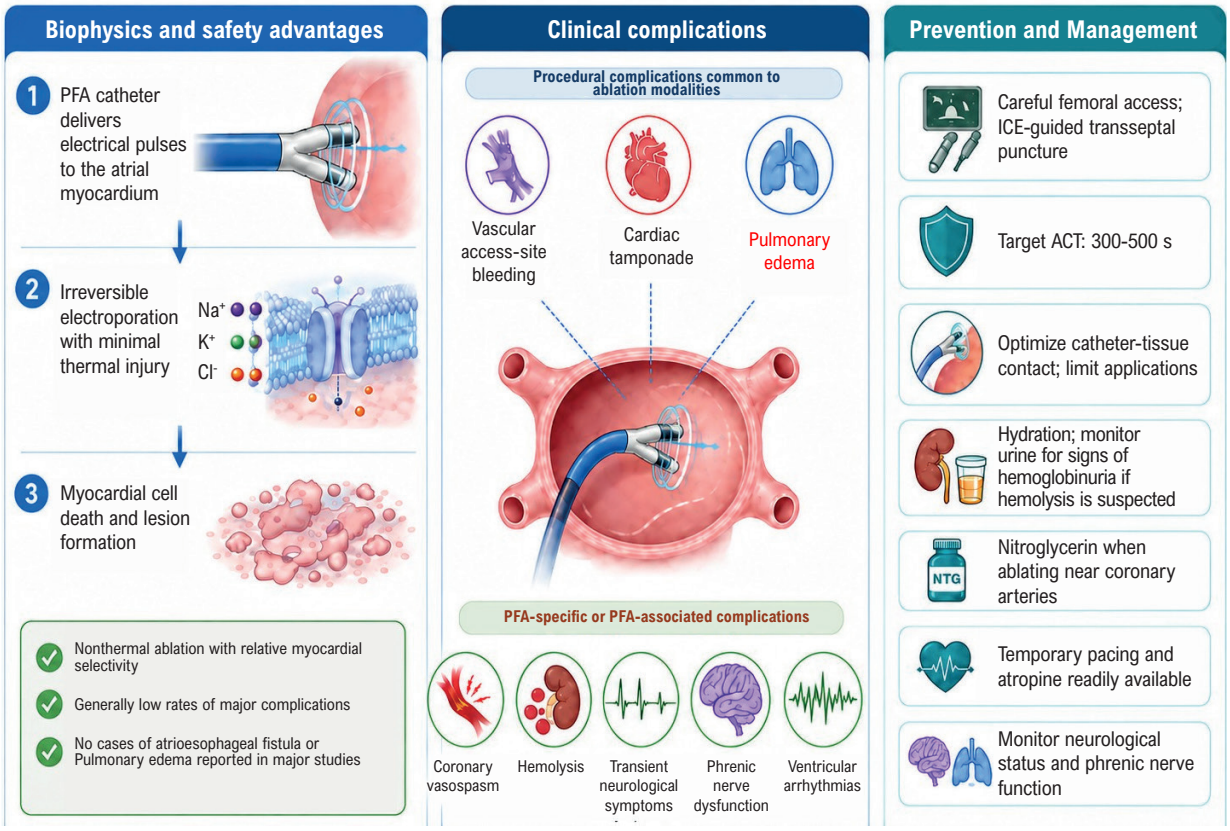
Pulsed field ablation (PFA) has emerged as a nonthermal alternative. Randomized trials and registries have demonstrated noninferior efficacy, shorter procedure times, and low rates of major complications compared with thermal ablation. Freedom from recurrent atrial arrhythmias at 12 months has been comparable to that achieved with conventional thermal ablation, indicating that the favorable safety profile of PFA is accompanied by sustained clinical effectiveness.[2-8] Nevertheless, PFA carries both energy-specific and procedural risks, despite markedly reducing thermal complications (Central Illustration). Commercially available PFA platforms differ in their waveforms, electrode configurations, and catheter designs, which may influence their complication profiles.

This narrative review examines 14 major trials and registries, focusing on the clinical presentation, prevention, and management of complications associated with PFA (Table 1).[2-15]

Methods

This narrative review provides an evidence-based synthesis consistent with established recommendations for narrative review reporting. The authors searched PubMed

Central Illustration: Safety and Complications of Pulsed Field Ablation for Atrial Fibrillation: Biophysics, Diagnosis, and Management



Arq Bras Cardiol. 2026; 123(10):e20260389

Overview of PFA and its mechanism of action, associated procedural and energy-specific complications, and prevention and management strategies based on current evidence and safety protocols. ACT: activated clotting time; ICE: intracardiac echocardiography; NTG: nitroglycerin; PFA: pulsed field ablation.

for studies published between 2019, when the first clinical trials of PFA were published, and 2026, using combinations of the terms "pulsed field ablation," "irreversible electroporation," "atrial fibrillation," "complications," "safety," and "adverse events." Eligible studies included peer-reviewed, English-language randomized trials, multicenter registries, and postapproval studies in humans reporting procedural safety outcomes. Reference lists of eligible studies and previous reviews were manually searched for relevant publications on mechanisms and safety, including case reports of rare complications. Four authors independently screened major trials and registries. Inclusion was determined by consensus based on clinical relevance, sample size, and the comprehensiveness of safety reporting, with priority given to the largest available datasets. Reported incidence ranges represent the lowest and highest estimates across the included studies rather than pooled or weighted estimates.

Biophysical and technical foundations of pulsed field ablation

Biophysics and mechanism of action

PFA relies on irreversible electroporation (IRE), a nonthermal mechanism in which high-voltage, ultrashort electrical pulses, delivered at 1,500-2,000 V over microseconds to milliseconds, create permanent nanopores in the lipid bilayer of target cells.[2-12] Above a critical electric field threshold, irreversible membrane injury causes cell death without substantial thermal injury. In contrast, RFA destroys tissue primarily through coagulative necrosis induced by resistive heating.

A key advantage of PFA is its relative tissue selectivity, as different cell types have different IRE thresholds. Cardiomyocytes are highly susceptible at electric field strengths of approximately 400-600 V/cm, whereas vascular smooth muscle, phrenic nerve, esophageal, and PV tissues require substantially higher field strengths to undergo IRE.

Table 1 – Major trials, studies, and registries reporting complication rates associated with PFA. Primary safety endpoints are provided to illustrate differences in definitions across studies

Study	n	Primary safety endpoint	Complication rate
PULSED AF[2]	300	Freedom from serious procedure- and device-related adverse events	0.7%
ADVENT[3]	305*	Serious device- and procedure-related adverse events within 7 days; atrioesophageal fistula or PV stenosis at any time	2.1%
inspIRE[4]	226	Early-onset primary adverse events within 7 days	0.0%
PULSE-EU[5]	48	Early (< 7 days) and late (> 7 days) major device- or procedure-related adverse events	2.0%
MANIFEST-PF[6]	1,568	Composite of acute and delayed major adverse events	Major: 1.9%; minor: 4.0%
MANIFEST-17K[7]	17,642	Composite of acute and delayed major adverse events	Major: 0.98%; minor: 3.21%
MANIFEST-US[8]	41,968	Composite of acute and delayed major adverse events	Major: 0.62%; minor: 2.10%
AdmIRE[9]	277	Prespecified primary safety events within 7 days; cardiac tamponade or perforation within 30 days; atrioesophageal fistula within 90 days; PV stenosis within 12 months	2.9%
VOLT CE Mark[10]	150	Serious device- and/or procedure-related adverse events within 7 days	2.7%
PFA vs ACB vs ARF[11]	214*	Composite of major adverse events	2.3%
FARADISE[12]	1,158	Serious device- and procedure-related adverse events from day 7 through 12 months	1.5%
VOLT-AF IDE[13]	392	Device- and/or procedure-related adverse events within 7 days	1.9%
ADVANTAGE AF[14]	260	Prespecified primary safety events within 7 days; cardiac tamponade or perforation within 30 days; atrioesophageal fistula or PV stenosis within 12 months	2.3%
AVANT GUARD[15]	257*	Composite of serious device- and procedure-related adverse events through 12 months in the combined randomized PFA group and PFA-assigned safety cohort	5.1%

**Number of patients in the PFA arm. CBA: cryoballoon ablation; DVT: deep vein thrombosis; PFA: pulsed field ablation; PV: pulmonary vein; RFA: radiofrequency ablation.*

These differences help explain the low rates of clinically significant esophageal injury and the absence of reported PV stenosis following PFA.[3,5-14]

Pulse characteristics and complication patterns

Pulse width and waveform design are critical determinants of efficacy and safety. Microsecond pulses remain the clinical standard, despite their association with substantial skeletal muscle stimulation. Symmetric biphasic pulses reduce neuromuscular stimulation compared with monophasic waveforms, thereby limiting skeletal muscle activation and patient discomfort during conscious sedation. Contemporary systems deliver coordinated pulse bursts separated by interpulse intervals, limiting heat accumulation and off-target effects.[8-10,16]

The energy delivery protocol also influences complication patterns. Increasing the number of pulses may enhance lesion depth but increase collateral Joule heating and microbubble formation, potentially raising the risk of gas embolization and associated neurological events, including stroke and silent cerebral lesions.

Catheter design

PFA platforms vary substantially in catheter architecture. Available designs include multielectrode single-shot arrays,

such as pentaspline and fixed- or variable-loop circular designs; balloon-based systems; spherical or lattice-tip catheters; and focal devices. These platforms differ in electrode number and configuration, catheter size, adjustability, tissue contact assessment, mapping integration, and intended application, ranging from PVI to focal ablation and substrate modification.[1-14,15] Such differences may influence lesion formation, procedural efficiency, blood-pool exposure to the electric field, and complication risk. Safety findings should therefore be interpreted in the context of the specific platform rather than generalized to PFA as a homogeneous technology.[17-19]

Monopolar configurations, which deliver energy between a catheter tip and a dispersive pad, can produce deeper lesions but greater skeletal muscle stimulation and pain, as can monophasic waveforms. Bipolar configurations can confine the electric field and reduce collateral neuromuscular activation. Lesion geometry also depends on catheter design, orientation relative to myocardial fibers, and tissue contact. Although PFA is less dependent on contact than thermal ablation, even small increases in catheter-tissue distance can substantially increase the voltage required for lesion formation, raising the risk of bubble formation and localized thermal injury.[16,17,20]

Complications of pulsed field ablation

Vascular complications

Vascular and access-related complications remain among the most common adverse events following ablation, although they are not specific to PFA. Reported incidence estimates for individual complications range from 0.002% to 3.000% across major PFA studies (Table 2). These events are related to vascular access and transseptal puncture rather than electroporation itself and also occur with other ablation modalities. Although PFA systems use larger sheaths, ranging from 8.5F to 17F, sheath size alone has not been independently associated with major vascular events.[21,22]

Bleeding and access-site complications

Groin hematoma is the most common access-related complication (0.3%-3.0%) and is generally managed conservatively with compression and pressure dressings; transfusion is rarely required. Pseudoaneurysm (< 0.3%) and arteriovenous fistula (< 0.3%) are less common and are diagnosed by duplex ultrasonography. Small pseudoaneurysms may resolve spontaneously or with ultrasound-guided compression, whereas thrombin injection or surgery is reserved for larger or refractory lesions. Retroperitoneal hemorrhage is rare (0.002%-0.070%) but potentially life-threatening and typically results from femoral puncture above the inguinal ligament. One fatal case was reported among more than 40,000 patients in MANIFEST-US.[8]

Thrombotic and embolic complications

Thrombotic and embolic complications, including deep vein thrombosis (0.004%-0.060%) and air embolism (< 1%), remain uncommon. Uninterrupted periprocedural anticoagulation with direct oral anticoagulants or vitamin K antagonists, together with intraprocedural anticoagulation targeting an activated clotting time (ACT) of 300-500 s, substantially reduces thromboembolic risk. Routine flushing of sheaths and catheters with heparinized saline helps prevent air entrapment.

Ultrasound-guided vascular access is a key preventive strategy, improving vessel identification and first-pass cannulation while reducing inadvertent arterial puncture.[22,23] A randomized trial showed that vascular closure devices were superior to figure-of-eight sutures alone after large-bore sheath removal, reducing time to ambulation and minor bleeding.[24]

Aortic wall injury

Transient late gadolinium enhancement of the descending aortic wall has been reported after PFA near the posterior left atrium (LA). Unlike access-related vascular injury, this finding occurred at rates similar to those observed with thermal ablation (33% vs. 43%; p = 0.52) and resolved completely by 3 months, without aneurysm, dissection, or stenosis, suggesting a nonspecific effect.[25] Aortic dissection associated with retrograde ventricular access is a mechanical rather than energy-related complication and was not reported in the 14 reviewed trials. Ultrasound-guided access and preprocedural aortic imaging may help mitigate this risk.[26]

Cardiac complications

Reported incidence estimates for individual cardiac adverse events range from 0.01% to 2.00% across the reviewed studies (Table 3). These events include procedural complications that are not specific to PFA, such as cardiac tamponade and pericardial effusion, as well as PFA-associated events, including pericarditis, coronary vasospasm, arrhythmias, and complications involving cardiac implantable electronic devices (CIEDs).

Cardiac tamponade and pericardial effusion

Cardiac tamponade is primarily related to catheter manipulation and transseptal puncture rather than PFA energy delivery. Nevertheless, it remains an important complication, with reported incidence estimates of 0.7%-1.6%.[2-8] Tamponade presents as an acute pericardial effusion with hemodynamic compromise and requires urgent percutaneous pericardiocentesis. Pericardial effusions without hemodynamic compromise occurred in 0.12%-2.00% of cases and were managed conservatively.

Proficiency in intracardiac echocardiography (ICE) is essential for transseptal access with the larger sheaths used in PFA. A spiral guidewire technique that allows serial dilation of the interatrial septum under ICE guidance is strongly recommended.[27] ICE also facilitates early recognition of tamponade and prompt intervention.

Table 2 – Vascular complications associated with PFA

Complication	Incidence	Mechanism	Clinical presentation	Management
DVT	0.004%-0.06%	Sheath-related vascular injury and venous stasis	Limb pain and swelling	Anticoagulation
Retroperitoneal hemorrhage	0.002%-0.07%	Puncture above the inguinal ligament, resulting in blood accumulation in the noncompressible retroperitoneal space	Hypotension, flank pain, and a substantial decrease in hemoglobin	CT confirmation; management ranging from conservative care to endovascular or surgical repair
Arteriovenous fistula	0.02%-0.3%	Inadvertent arterial puncture during venous cannulation	Continuous bruit, palpable thrill, and limb swelling	Compression, thrombin injection, or surgical repair
Air embolism	0.038%-0.3%	Air entry during catheter manipulation and sheath exchanges	Neurological symptoms or hemodynamic changes	Prevention of further air entry, supplemental oxygen, and Trendelenburg positioning; hyperbaric oxygen therapy in severe cases
Pseudoaneurysm	0.1%-0.3%	Vessel wall disruption creating a communication between the vascular lumen and the extravascular space	Pain, swelling, a pulsatile mass, and a palpable thrill	Compression or thrombin injection; surgical repair in refractory cases
Bleeding or hematoma	0.3%-3%	Large-bore sheath insertion causing blood extravasation into surrounding tissues	Findings ranging from minor ecchymosis to expanding groin hematomas	Conservative management with manual compression and pressure dressings; transfusion in severe cases
Total vascular complications*	0.25%-8.1%	—	—	—

*Incidence estimates represent ranges across the reviewed studies. Complications are listed in ascending order of the upper limit of the incidence range; ties are resolved using the lower limit. *Range of overall vascular complication rates reported across the 14 reviewed trials and registries. CT: computed tomography; DVT: deep vein thrombosis; PFA: pulsed field ablation.*

Pericarditis

Pericarditis following PFA is uncommon, occurring in 0.06%-2.00% of cases, and may result from an inflammatory response to lesion formation.[28] A biomarker study demonstrated greater myocardial injury after PFA than after RFA, consistent with a larger ablated surface area, but a milder systemic inflammatory response. This finding may help explain the generally self-limited course of pericarditis after PFA.[29] Most cases responded to medical therapy or required no specific intervention. Limiting the number of applications per procedure is the primary preventive strategy.

Coronary vasospasm and acute coronary syndrome

Coronary vasospasm is a rare but important PFA-specific complication, reported in 0.10%-0.14% of cases and more frequently than with RFA.[6-8,14] Most cases occur when energy is delivered near coronary arteries, particularly during mitral isthmus ablation near the left circumflex artery or cavotricuspid isthmus ablation near the right coronary artery.[30,31] By contrast, routine PVI has not been associated with an increased anatomical risk of coronary vasospasm because the PVs are not in close proximity to major coronary arteries. The mechanism likely involves autonomic dysregulation triggered by the electric field and hemolysis-mediated nitric oxide (NO) scavenging. Cell-free hemoglobin released during PFA reacts with NO, reducing its local bioavailability and promoting coronary smooth muscle hypercontractility.[32]

Most cases are subclinical and manifest as transient angiographic coronary narrowing without electrocardiographic changes or hemodynamic compromise. Symptomatic presentations include ST-segment elevation, chest pain, hypotension, and ventricular arrhythmias. In MANIFEST-17K, one patient developed ventricular fibrillation (VF) requiring resuscitation and defibrillation; in ADVANTAGE AF, one patient ultimately underwent percutaneous coronary intervention.[7,14]

Table 3 – Cardiac complications associated with PFA

Complication	Incidence	Mechanism	Clinical presentation	Management
CIED-related complications	0.01%-0.08%	Electromagnetic interference and catheter manipulation near device components	Lead dislodgement or device dysfunction detected during interrogation	Pacemaker reimplantation; backup ventricular pacing during the procedure
Coronary vasospasm	0.1%-0.14%	Autonomic modulation and NO scavenging near coronary arteries	Subclinical angiographic narrowing; symptomatic cases may present with ST-segment changes, chest pain, or arrhythmias	IV nitroglycerin; intracoronary nitroglycerin in refractory cases
MI or ACS	0.05%-0.4%	Coronary vasospasm or thrombosis; the mechanism remains incompletely understood	ST-segment elevation and angiographic evidence of coronary involvement	PCI; IV nitroglycerin
Cardiac tamponade	0.7%-1.6%	Catheter manipulation and transseptal puncture	Acute pericardial effusion with hemodynamic compromise, including hypotension, tachycardia, and obstructive shock	Percutaneous or surgical pericardial drainage
Pericarditis	0.06%-2%	Mild inflammatory response following nonthermal lesion creation	ECG changes, pericardial effusion, a pericardial friction rub, and chest pain	Anti-inflammatory therapy and monitoring
Pericardial effusion	0.12%-2%	Catheter manipulation and an inflammatory response	Small effusion detected by echocardiography without hemodynamic compromise	Conservative monitoring; pericardiocentesis if hemodynamically significant
Total cardiac complications*	0.6%-4.16%	—	—	—

*Incidence estimates represent ranges across the reviewed studies. *Range of overall cardiac complication rates reported across the 14 reviewed trials and registries. ACS: acute coronary syndrome; CIED: cardiac implantable electronic device; ECG: electrocardiogram; IV: intravenous; MI: myocardial infarction; NO: nitric oxide; PCI: percutaneous coronary intervention; PFA: pulsed field ablation.*

Intravenous nitroglycerin rapidly reverses vasospasm in most cases. For procedures targeting regions adjacent to coronary arteries, prophylactic intravenous nitroglycerin is recommended. One reported regimen consists of a 3,000-μg bolus followed by 2,000-μg boluses at 2-min intervals after energy delivery begins.[33] Procedural preparation should include the availability of nitroglycerin at doses of 2,000-4,000 μg and interventional cardiology support for refractory cases.

Arrhythmias

PFA carries an intrinsic, timing-dependent proarrhythmic risk: energy delivery during the ventricular vulnerable period, corresponding to the T wave, may induce VF, particularly with monophasic waveforms. R-wave-synchronized gating minimizes this risk.[34] Additional mechanisms include arrhythmias triggered by coronary vasospasm (see subsection "Coronary vasospasm and acute coronary syndrome") and PFA-associated bradycardia (see subsection "Vasovagal responses"). Rare cases of unexplained sudden death were reported in MANIFEST-US (0.019%), predominantly within 3 days of the procedure; causality remains uncertain.[8]

Interactions with cardiac implantable electronic devices

CIED-related complications were reported in 0.01%-0.08% of cases.[7,8] High-voltage PFA pulses may cause electromagnetic interference, whereas catheter manipulation may lead to lead dislodgement or fracture. Two device-

specific mechanisms warrant attention. First, inductive coupling of PFA energy to CIED leads may trigger VF through an R-on-T mechanism (see subsection "Arrhythmias"). Second, high-voltage pulses may directly damage internal circuitry, causing generator dysfunction or software reset and potentially necessitating device replacement, independently of arrhythmic events.[35-37] Preventive measures include preprocedural and postprocedural device interrogation, reprogramming to prevent oversensing, avoidance of contact with CIED components, backup ventricular pacing, and continuous electrocardiographic monitoring.[38-41]

Pulmonary complications

Reported incidence estimates for pulmonary complications range from 0.028% to 1.500% (Table 4). These events are largely procedural rather than PFA-specific. No cases of PV stenosis were reported following PFA.[1-8]

Pulmonary edema and pneumonia

Pulmonary edema (0.1%-1.5%) is attributed to periprocedural fluid overload, particularly during longer procedures involving higher irrigation volumes. Patients with preexisting heart failure require careful fluid monitoring. Treatment includes intravenous diuretics and noninvasive ventilation. Pneumonia (0.028%-0.070%) is rare and is more closely associated with hospitalization or aspiration than with PFA itself.[7,8]

Guidewire and catheter entrapment

Guidewire or catheter entrapment within an inferior PV is a rare mechanical complication unrelated to energy delivery and was not reported in the 14 reviewed trials. However, a case series described three events among 229 procedures (1.3%), presenting as a sudden loss of catheter mobility after distal advancement into a small branch.[42] Forceful traction should be avoided because of the risk of dissection or tamponade. The preferred approach is to retract the assembly into the sheath to restore coaxial alignment before applying gentle traction; balloon-assisted or surgical retrieval may be required if these maneuvers fail.

Hemolysis and kidney complications

Reported incidence estimates for hemolysis and kidney complications, among the most characteristic PFA-specific adverse events, range from 0.009% to 0.760% (Table 5). Electroporation of circulating erythrocytes directly links the biophysical properties of PFA to these downstream clinical manifestations.

Hemolysis

Despite its relative myocardial selectivity, PFA causes dose-dependent intravascular hemolysis because erythrocytes are susceptible to electroporation at clinically used electric field strengths. High-energy pulses disrupt erythrocyte membranes, releasing cell-free hemoglobin into the circulation.[32] Inadequate tissue contact during ablation may increase blood-pool exposure to the electric field, amplifying this effect. Hemolysis rates also differ substantially across PFA platforms. A prospective study of 245 patients comparing four commercially available systems reported an overall hemolysis incidence of 16.7%, with rates of 37.0%, 26.1%, and 14.7% for pentaspline, circular-array, and lattice-tip catheters, respectively. No clinically apparent kidney impairment was observed.[17]

Hemolysis ranges from subclinical biochemical abnormalities, including decreased haptoglobin and

Table 4 – Pulmonary complications associated with PFA

Complication	Incidence	Mechanism	Clinical presentation	Management
PV stenosis	No cases reported	Progressive ostial narrowing secondary to remodeling induced by energy delivery	Dyspnea and hemoptysis months after ablation	PV stenting; no cases requiring treatment reported with PFA
Pneumonia	0.028%-0.07%	No direct causal relationship with PFA; likely related to hospitalization or aspiration	Fever, leukocytosis, and respiratory symptoms	Antibiotic therapy
Pulmonary edema	0.1%-1.5%	Periprocedural fluid overload and transient atrial dysfunction following extensive ablation	Dyspnea and hypoxemia within hours to days after ablation	IV diuretics and NIV
Total pulmonary complications*	0.028%-1.5%	—	—	—

*Incidence estimates represent ranges across the reviewed studies. *This range reflects the lowest and highest incidence estimates for the pulmonary complications listed above across the 14 reviewed trials and registries. IV: intravenous; NIV: noninvasive ventilation; PFA: pulsed field ablation; PV: pulmonary vein.*

increased indirect bilirubin or lactate dehydrogenase, to clinically significant events involving a measurable decline in hemoglobin or a need for transfusion. Hemoglobinuria, identified visually or by urine dipstick testing, indicates a greater hemolytic burden and may progress to hemoglobinuria-associated acute kidney injury (AKI), the most severe manifestation. Reported incidence varies widely according to diagnostic thresholds and screening methods. Clinically significant cases may be underestimated because few studies systematically assess hemolysis. Limiting PFA applications and optimizing tissue contact are the primary preventive strategies. Postprocedural laboratory monitoring may guide management.[17,43]

Hemolysis-induced acute kidney injury

Hemoglobinuria-associated AKI occurs in 0.02%-0.40% of cases.[2-10] Cell-free hemoglobin is filtered by the kidneys and can cause tubular injury, oxidative stress, and inflammation.[44,45] The serum creatinine components of the Kidney Disease: Improving Global Outcomes definition of AKI include an increase of at least 0.3 mg/dL within 48 h or an increase to at least 1.5 times baseline within 7 days.[46] Most cases are subclinical, although hematuria, hemoglobinuria, or oliguria may occur. Management includes carefully titrated intravenous hydration and, rarely, temporary hemodialysis. The prognosis is generally favorable, with complete recovery.

Neurological and autonomic complications

Reported incidence estimates for individual neurological complications range from 0.06% to 2.00% across the reviewed studies, excluding silent cerebral lesions and vasovagal responses (Table 6). Stroke and transient ischemic attack (TIA) occur at rates comparable to those observed with thermal ablation. Silent cerebral lesions and vasovagal responses warrant separate consideration because of differences in their mechanisms, detection, and reporting.

Stroke and transient ischemic attack

Stroke (0.1%-2.0%) and TIA (0.08%-0.40%) are among the most serious neurological complications of catheter ablation.[2-8] Although the nonthermal mechanism of PFA may reduce coagulum formation, microbubble generation, preexisting LA thrombus, and debris released during catheter manipulation remain important sources of emboli.[18]

AVANT GUARD was paused after 6 procedure-related strokes, a higher number than anticipated. These events were attributed to the enrollment of older, higher-risk patients and to procedural factors. Following protocol changes, including stricter screening, enhanced anticoagulation measures, and an ACT target of ≥ 350 s, the rate of safety events in the ablation arm decreased from 6% to 3%, with no further strokes reported after the trial resumed.[15] Prevention relies on uninterrupted anticoagulation, an intraprocedural ACT target of 300-500 s, preprocedural exclusion of LA thrombus by transesophageal echocardiography, avoidance of rapid catheter withdrawal, and flushing of sheaths and catheters with heparinized saline.

Silent cerebral lesions

Silent cerebral lesions (SCLs) are subclinical ischemic brain injuries detected by diffusion-weighted magnetic resonance imaging (DW-MRI) in the absence of neurological symptoms. Reported incidence estimates across PFA trials range from 5.0% to 20.5%.[2-7] A systematic review and network meta-analysis of 86 trials involving 10,456 patients estimated a pooled incidence of 14.4% in PFA cohorts, with no statistically significant difference compared with thermal ablation.[47] Detection rates depend on whether imaging is performed systematically or in response to symptoms, with higher rates reported in cohorts undergoing routine imaging.

However, this pooled estimate may obscure substantial differences between catheter platforms. Reported SCL

Table 5 – Hemolysis and kidney complications associated with PFA

Complication	Incidence	Mechanism	Clinical presentation	Management
Hemolysis	0.009%-0.76%	Dose-dependent electroporation of erythrocyte membranes, resulting in intravascular hemolysis and cell-free hemoglobin release	Subclinical laboratory abnormalities, including decreased haptoglobin and increased LDH and indirect bilirubin; anemia	Limiting the number of pulses; postprocedural laboratory monitoring
AKI	0.02%-0.4%	Hemoglobin-mediated tubular injury, oxidative stress, and kidney inflammation	Increased serum creatinine meeting KDIGO criteria; hematuria, hemoglobinuria, or oliguria	IV hydration; temporary hemodialysis in severe cases
Total hemolysis and kidney complications*	0.009%-0.76%	—	—	—

*Incidence estimates represent ranges across the reviewed studies. *This range reflects the lowest and highest incidence estimates for the hemolysis and kidney complications listed above across the 14 reviewed trials and registries. AKI: acute kidney injury; IV: intravenous; KDIGO: Kidney Disease: Improving Global Outcomes; LDH: lactate dehydrogenase; PFA: pulsed field ablation.*

Table 6 – Neurological and autonomic complications associated with PFA

Complication	Incidence	Mechanism	Clinical presentation	Management
Stroke	0.1%-2%	Embolization of microbubbles, air, or tissue debris during catheter manipulation	Focal neurological deficits, aphasia, or visual disturbances	Uninterrupted anticoagulation and a target ACT of 300-500 s for prevention; prompt neurological evaluation
TIA	0.08%-0.4%	Same as stroke	Transient neurological deficits resolving within 24 h	Same as stroke
PNI	0.06%-1.3%	PFA delivery near the phrenic nerve causing membrane hyperpolarization; rarely, direct nerve injury	Often asymptomatic; positional dyspnea or diaphragmatic dysfunction detected by CMAP monitoring	Usually self-limited; limiting applications near the RSPV for prevention
SCLs	5%-20.5%§	Systemic embolization of microbubbles, air, or tissue debris during catheter manipulation	Subclinical lesions detected by DW-MRI	A 10-s pause between applications and strict periprocedural anticoagulation for prevention
Vasovagal responses	~30%-60%†	Activation of extracardiac ganglionated plexi near the pulmonary veins	Bradycardia, sinus pauses, AV block, hypotension, or asystole during energy delivery	Prophylactic anticholinergic therapy; initiation of PVI at the RSPV; temporary cardiac pacing when needed
Total neurological complications*	0.06%-2%	—	—	—

*Incidence estimates represent ranges across the reviewed studies. *This range reflects the lowest and highest incidence estimates for the neurological complications listed above, excluding silent cerebral lesions and vasovagal responses, across the 14 reviewed trials and registries. §Silent cerebral lesions were excluded from the total neurological complication range because they were reported only in studies using systematic postprocedural DW-MRI screening. †Vasovagal responses were excluded from the total neurological complication range because of inconsistent reporting across studies and their often transient nature. ACT: activated clotting time; AV: atrioventricular; CMAP: compound motor action potential; DW-MRI: diffusion-weighted magnetic resonance imaging; PFA: pulsed field ablation; PNI: phrenic nerve injury; PVI: pulmonary vein isolation; RSPV: right superior pulmonary vein; SCL: silent cerebral lesion; TIA: transient ischemic attack.*

rates are low with pentaspline catheters (3.0%-8.8%),[48,49] intermediate with circular multielectrode array catheters (8.9%-22.0%),[19] and markedly higher with variable-loop circular catheters (66.7%-85.0%). The latter have also been associated with an atypical distribution involving the posterior circulation and, in one cohort, early study termination following a disabling stroke.[19,50] The discrepancy between low rates of clinical stroke (< 1%) and the appreciable frequency of magnetic resonance imaging-detected SCLs may reflect the small, often subcortical size of most lesions, which may not produce overt neurological deficits.[19]

PFA-specific prevention protocols remain undefined, although platform-specific workflow adjustments appear promising. In one trial involving a variable-loop catheter, workflow modifications that included a 10-s pause between energy applications were followed by a decrease in SCL incidence, from four of the first six participants to four of the subsequent 33 participants.[50] This finding supports the interval between applications as a potential risk mitigation strategy. Strict periprocedural anticoagulation is also recommended based on evidence from thermal ablation.

Phrenic nerve injury

Phrenic nerve injury (PNI) was reported in 0.06%-1.30% of cases across major PFA trials. However, systematic screening was often absent, and detection relied primarily on symptoms or hemidiaphragm elevation on chest radiography.[2-8] A prospective study using the more sensitive approach of compound motor action potential (CMAP) monitoring detected diaphragmatic paralysis in 40.6% of patients. Incomplete PNI remained present at the end of the procedure in 18.8%, and one patient had persistent

dysfunction at 3 months.[51] The proposed mechanism is phrenic nerve membrane hyperpolarization, particularly during ablation near the right superior PV (RSPV). Transient PNI is self-limited, whereas persistent palsy requires monitoring for respiratory compromise. Limiting applications near the RSPV and avoiding deep catheter placement within the vein are recommended preventive measures.[51-53]

Vasovagal responses

Vasovagal responses, including bradycardia, sinus pauses, atrioventricular block, hypotension, and asystole during energy delivery, occur more frequently with PFA than with thermal ablation, affecting approximately 30%-60% of procedures.[54,55] In a retrospective study, vagal responses occurred in 41% of PFA procedures compared with 5.1% of RFA procedures.[56] The mechanism involves direct depolarization of extracardiac ganglionated plexi near the PVs. Unlike ganglionic necrosis induced by RFA, PFA preserves structural integrity while triggering acute autonomic activation.[57]

A prospective study demonstrated that initiating PVI at the RSPV rather than the left superior PV reduced vasovagal responses from 78% to 13%, likely by interrupting input from the anterior right ganglionated plexus to the sinoatrial node.[58] This effect also extended to subsequent ablation of the left inferior PV (28% vs. 0%) but not the right inferior PV. The study did not directly evaluate initiating ablation at an inferior PV. Management of vasovagal responses includes temporary cardiac pacing when needed. Prophylactic atropine (0.3-1.0 mg) or glycopyrrolate (0.2 mg) before venous puncture may reduce the incidence and severity of these events.[59,60]

Gastrointestinal complications

Neither atrioesophageal fistula nor significant esophageal injury was reported across the 14 reviewed PFA studies, including those involving postprocedural endoscopy.[2-8] In MANIFEST-17K, endoscopy performed in 29 patients identified esophageal edema in 4 (13.8%), without clinical sequelae.[7] At least one case of esophageal ulceration following PFA has been reported, although preprocedural endoscopy was not performed to exclude a preexisting lesion.[61] Preclinical porcine studies involving direct esophageal PFA delivery demonstrated only transient mucosal changes. A recent study by Nies et al. identified small, nontransmural lesions at 24 h that resolved by 14 days.[62,63] Esophageal sparing may reflect natural retraction during energy delivery and reversible electroporation of nontarget tissues exposed to subthreshold electric field strengths. These findings support omitting routine esophageal monitoring.

Clinical implications and limitations

By substantially reducing the risk of thermal injury, PFA has enabled more extensive lesion sets, including posterior wall isolation and ablation of targets outside the PVs. Nevertheless, PFA has changed the distribution of procedural risks rather than eliminated them. Coronary vasospasm, hemolysis-associated AKI, arrhythmias, and CIED interactions require continued attention as adoption expands. Institutional protocols should incorporate targeted preventive measures, including nitroglycerin before isthmus ablation, hydration and hemolysis monitoring during prolonged procedures, and CIED interrogation with backup pacing available.

Interpretation of current safety data is limited by substantial heterogeneity. Much of the evidence comes from high-volume centers that adopted PFA early, with inconsistent complication definitions, screening practices, and follow-up durations. Direct comparisons between platforms remain limited. Prospective multicenter registries with standardized definitions and follow-up beyond 1 year are needed to establish the durability of PFA's safety advantages and identify late complications.

Conclusion

PFA represents a major advance in the safety of AF ablation by reducing severe thermal injuries, although procedural risks persist and electroporation-related complications require specific attention. Safe adoption depends on prevention and management strategies informed by the mechanisms of these complications and the characteristics of each platform. As real-world use expands, standardized safety reporting and long-term follow-up will be essential to clarify differences among platforms and identify uncommon or delayed adverse events.

Author Contributions

Conception and design of the research: Velasquez A, Silva GO, d'Avila A; Data collection: Velasquez A, Silva GO, Moraes HGM; Data analysis and interpretation: Silva GO, Moraes HGM, d'Avila A; Manuscript drafting: Velasquez A, Silva GO, Moraes HGM, d'Avila A; Critical revision of the manuscript for important intellectual content: Velasquez A, Silva GO, Moraes HGM, Zimerman LI, d'Avila A.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

There were no external funding sources for this study.

Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This article does not contain any studies with human participants or animals performed by any of the authors.

Use of Artificial Intelligence

During the preparation of this work, the author(s) used Google Gemini and OpenAI ChatGPT for image creation

and grammatical correction. After using this tool/service, the author(s) reviewed and edited the content as needed and take full responsibility for the content of the published article.

Availability of Research Data

The underlying content of the research text is contained within the manuscript.

References

1. Joglar JA, Chung MK, Armbruster AL, Benjamin EJ, Chyou JY, Cronin EM, et al. 2023 ACC/AHA/ACCP/HRS Guideline for the Diagnosis and Management of Atrial Fibrillation: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2024;149(1):e1-e156. doi: 10.1161/CIR.0000000000001193.
2. Verma A, Haines DE, Boersma LV, Sood N, Natale A, Marchlinski FE, et al. Pulsed Field Ablation for the Treatment of Atrial Fibrillation: PULSED AF Pivotal Trial. Circulation. 2023;147(19):1422-32. doi: 10.1161/CIRCULATIONAHA.123.063988.
3. Reddy VY, Gerstenfeld EP, Natale A, Whang W, Cuoco FA, Patel C, et al. Pulsed Field or Conventional Thermal Ablation for Paroxysmal Atrial Fibrillation. N Engl J Med. 2023;389(18):1660-71. doi: 10.1056/NEJMoa2307291.
4. Duytschaever M, Potter T, Grimaldi M, Anic A, Vijgen J, Neuzil P, et al. Paroxysmal Atrial Fibrillation Ablation Using a Novel Variable-Loop Biphasic Pulsed Field Ablation Catheter Integrated with a 3-Dimensional Mapping System: 1-Year Outcomes of the Multicenter inspIRE Study. Circ Arrhythm Electrophysiol. 2023;16(3):e011780. doi: 10.1161/CIRCEP.122.011780.
5. Turagam MK, Neuzil P, Petru J, Funasako M, Koruth JS, Skoda J, et al. AF Ablation Using a Novel "Single-Shot" Map-and-Ablate Spherical Array Pulsed Field Ablation Catheter: 1-Year Outcomes of the First-in-Human PULSE-EU Trial. Heart Rhythm. 2024;21(8):1218-26. doi: 10.1016/j.hrthm.2024.04.102.
6. Turagam MK, Neuzil P, Schmidt B, Reichlin T, Neven K, Metzner A, et al. Safety and Effectiveness of Pulsed Field Ablation to Treat Atrial Fibrillation: One-Year Outcomes from the MANIFEST-PF Registry. Circulation. 2023;148(1):35-46. doi: 10.1161/CIRCULATIONAHA.123.064959.
7. Ekanem E, Neuzil P, Reichlin T, Kautzner J, van der Voort P, Jais P, et al. Safety of Pulsed Field Ablation in More than 17,000 Patients with Atrial Fibrillation in the MANIFEST-17K Study. Nat Med. 2024;30(7):2020-9. doi: 10.1038/s41591-024-03114-3.
8. Turagam MK, Aryana A, Day JD, Dukkipati SR, Hounshell T, Nair D, et al. Multicenter Study on the Safety of Pulsed Field Ablation in Over 40,000 Patients: MANIFEST-US. J Am Coll Cardiol. 2026;87(2):172-93. doi: 10.1016/j.jacc.2025.10.051.
9. Reddy VY, Calkins H, Mansour M, Wazni O, Di Biase L, Bahu M, et al. Pulsed Field Ablation to Treat Paroxysmal Atrial Fibrillation: Safety and Effectiveness in the AdmIRE Pivotal Trial. Circulation. 2024;150(15):1174-86. doi: 10.1161/CIRCULATIONAHA.124.070333.
10. Tilz RR, Chierchia GB, Gunawardene M, Sanders P, Haqqani H, Kalman J, et al. Safety and Effectiveness of the First Balloon-in-Basket Pulsed Field Ablation System for the Treatment of Atrial Fibrillation: VOLT CE Mark Study 6-Month Results. Europace. 2025;27(4):euaf072. doi: 10.1093/europace/euaf072.
11. Kueffer T, Stettler R, Maurhofer J, Madaffari A, Stefanova A, Iqbal SUR, et al. Pulsed-Field vs Cryoballoon vs Radiofrequency Ablation: Outcomes after Pulmonary Vein Isolation in Patients with Persistent Atrial Fibrillation. Heart Rhythm. 2024;21(8):1227-35. doi: 10.1016/j.hrthm.2024.04.045.
12. Boersma LVA, Széplaki G, Russo AD, García-Bolao I, Efremidis M, Szegedi N, et al. Real-World Experience with the Pentaspline Pulsed Field Ablation System: One-Year Outcomes of the FARADISE Registry. Europace. 2025;27(9):euaf182. doi: 10.1093/europace/euaf182.
13. Lo M, Gambhir A, Sundaram S, Sanders P, DeLurgio D, Trivedi A, et al. Safety and Effectiveness of a Novel Balloon-in-Basket Pulsed-Field Ablation Catheter for the Treatment of Paroxysmal and Persistent AF: Volt-AF IDE Trial Acute Results. Heart Rhythm. 2025;22(10):2524-33. doi: 10.1016/j.hrthm.2025.04.037.
14. Reddy VY, Gerstenfeld EP, Schmidt B, Nair D, Natale A, Saliba W, et al. Pulsed Field Ablation for Persistent Atrial Fibrillation: 1-Year Results of ADVANTAGE AF. J Am Coll Cardiol. 2025;85(17):1664-78. doi: 10.1016/j.jacc.2025.03.515.
15. Wazni OM, Chun KJ, Amin A, Anic A, Ajijola OA, Gibson DN, et al. A Randomized Controlled Trial of Pulsed Field Ablation vs Antiarrhythmic Therapy as a First-Line Treatment for Persistent Atrial Fibrillation: The AVANT GUARD Trial Rationale and Design. Heart Rhythm O2. 2025;7(3):561-74. doi: 10.1016/j.hroo.2025.12.011.
16. Sugrue A, Maor E, Munoz FDC, Killu AM, Asirvatham SJ. Cardiac Ablation with Pulsed Electric Fields: Principles and Biophysics. Europace. 2022;24(8):1213-22. doi: 10.1093/europace/euac033.
17. Mountantonakis SE, Beccarino N, Patel H, Castillo A, Siddiqui T, Bhatt M, et al. Incidence of Laboratory-Defined Severe Intravascular Haemolysis Across Commercially Available Pulsed Field Ablation Technologies for Atrial Fibrillation. Europace. 2025;27(9):euaf185. doi: 10.1093/europace/euaf185.
18. Sánchez-Gómez JM, Ortiz-Fernández Á, Soler-Martínez J, Gabaldón-Pérez A, Iraola-Viana D, Martínez-Brotons Á, et al. Incidence of Brain Lesions Following Pulsed Field Ablation for Atrial Fibrillation. Heart Rhythm. 2026;23(1):8-15. doi: 10.1016/j.hrthm.2025.02.020.
19. Miyazaki S, Kawamura I, Iwasa Y, Negishi M, Tateishi R, Honda M, et al. Different Incidence and Size of Silent Strokes after Pulsed Field Ablation with Circular Shaped Ablation Catheters. Circ Arrhythm Electrophysiol. 2025;18(5):e013719. doi: 10.1161/CIRCEP.125.013719.
20. Tabaja C, Younis A, Hussein AA, Taigen TL, Nakagawa H, Saliba WI, et al. Catheter-Based Electroporation: A Novel Technique for Catheter Ablation of Cardiac Arrhythmias. JACC Clin Electrophysiol. 2023;9(9):2008-23. doi: 10.1016/j.jacep.2023.03.014.
21. Benali K, Khairy P, Hammache N, Petzl A, Costa A, Verma A, et al. Procedure-Related Complications of Catheter Ablation for Atrial Fibrillation. J Am Coll Cardiol. 2023;81(21):2089-99. doi: 10.1016/j.jacc.2023.03.418.
22. Lee HJ, Lee SH, Park S, Kim MJ, Kim J, Kim JY, et al. Incidence, Predictors, and Management of Femoral Vascular Complications Following Catheter Ablation for Atrial Fibrillation: A Systematic Duplex Ultrasound Study. Biomedicines. 2025;13(2):314. doi: 10.3390/biomedicines13020314.
23. Krimphoff A, Urbanek L, Bordignon S, Schaack D, Tohoku S, Chen S, et al. The Impact of Ultrasound-Guided Vascular Access for Catheter Ablation of Left Atrial Arrhythmias in a High-Volume Centre. J Interv Card Electrophysiol. 2024;67(5):1247-55. doi: 10.1007/s10840-024-01779-x.
24. Dashwood A, Ha FJ, Healy S, Puchalski R, Cheng SH, Tian B, et al. Vascular Closure Device Following Pulsed Field Ablation for Atrial Fibrillation: A Randomized Controlled Trial. Pacing Clin Electrophysiol. 2025;48(11):1294-300. doi: 10.1111/pace.70051.
25. Cochet H, Nakatani Y, Sridi-Cheniti S, Cheniti G, Ramirez FD, Nakashima T, et al. Pulsed Field Ablation Selectively Spares the Oesophagus during Pulmonary Vein Isolation for Atrial Fibrillation. Europace. 2021;23(9):1391-9. doi: 10.1093/europace/euab090.
26. Ishida S, Takemoto Y, Kimata R, Yagami K. Iatrogenic Acute Type A Aortic Dissection during Catheter Ablation for Idiopathic Ventricular Premature Contraction. Oxf Med Case Reports. 2024;2024(9):omae110. doi: 10.1093/omcr/omae110.

27. Tzeis S, Gerstenfeld EP, Kalman J, Saad EB, Shamloo AS, Andrade JG, et al. 2024 European Heart Rhythm Association/Heart Rhythm Society/Asia Pacific Heart Rhythm Society/Latin American Heart Rhythm Society Expert Consensus Statement on Catheter and Surgical Ablation of Atrial Fibrillation. Europace. 2024;26(4):euae043. doi: 10.1093/europace/euae043.

28. Isenegger C, Arnet R, Jordan F, Salis M, Knecht S, Krisai P, et al. Incidence of Acute Pericarditis after Pulsed-Field Ablation for the Treatment of Atrial Fibrillation. Europace. 2024;26(7):euae180. doi: 10.1093/europace/euae180.

29. Osmancik P, Bacova B, Hozman M, Pistkova J, Kunstatova V, Sochorova V, et al. Myocardial Damage, Inflammation, Coagulation, and Platelet Activity during Catheter Ablation Using Radiofrequency and Pulsed-Field Energy. JACC Clin Electrophysiol. 2024;10(3):463-74. doi: 10.1016/j.jacep.2023.11.001.

30. Zhang C, Neuzil P, Petru J, Funasako M, Hala P, Kopriva K, et al. Coronary Artery Spasm during Pulsed Field vs Radiofrequency Catheter Ablation of the Mitral Isthmus. JAMA Cardiol. 2024;9(1):72-7. doi: 10.1001/jamacardio.2023.4405.

31. Farnir FIP, Chaldoupi SM, Hermans B, Johannessen A, Haugdal MA, Ruwald MH, et al. Ablation of Cavotricuspid Isthmus-Dependent Atrial Flutter Using a Focal Monopolar Pulsed-Field Ablation Catheter: Feasibility, Periprocedural Coronary Spasms and Conduction Disorders. Heart Rhythm. 2026;23(1):105-13. doi: 10.1016/j.hrthm.2025.07.001.

32. Popa MA, Venier S, Menè R, Della Rocca DG, Sacher F, Derval N, et al. Characterization and Clinical Significance of Hemolysis after Pulsed Field Ablation for Atrial Fibrillation: Results of a Multicenter Analysis. Circ Arrhythm Electrophysiol. 2024;17(10):e012732. doi: 10.1161/CIRCEP.124.012732.

33. Malyshev Y, Neuzil P, Petru J, Funasako M, Hala P, Kopriva K, et al. Nitroglycerin to Ameliorate Coronary Artery Spasm during Focal Pulsed-Field Ablation for Atrial Fibrillation. JACC Clin Electrophysiol. 2024;10(5):885-96. doi: 10.1016/j.jacep.2023.12.015.

34. Bradley CJ, Haines DE. Pulsed Field Ablation for Pulmonary Vein Isolation in the Treatment of Atrial Fibrillation. J Cardiovasc Electrophysiol. 2020;31(8):2136-47. doi: 10.1111/jce.14414.

35. Goldar G, Sifuentes A, Vargas C, Farjo P, Dominic P, Bailin S. Asynchronous Pulsed Field Ablation-Induced Ventricular Fibrillation. Heart Rhythm. 2026;23(5):1023-6. doi: 10.1016/j.hrthm.2025.10.032.

36. Takahashi K, Kuwahara T, Makita T, Arisuda Y, Kadono K, Oshio T. Irreversible Intracardiac Defibrillator Circuit Damage Induced by Pulsed-Field Ablation: Mechanistic Insights. HeartRhythm Case Rep. 2026;12(3):344-9. doi: 10.1016/j.hrcr.2025.12.012.

37. Nair DG, Gabrah K, Doty B, Nair G, Reddy VY. Cardiac Implantable Electronic Device Software Reset and Damage after Pulsed Field Ablation Using a Monopolar Lattice-Tip Catheter. Heart Rhythm. 2026;23(5):1027-31. doi: 10.1016/j.hrthm.2025.09.027.

38. Koop BE, Albrecht EM, Lecy E, Kippola J. Pulsed Field Ablation in Patients with Cardiac Implantable Electric Devices: Vigilance is Warranted. Pacing Clin Electrophysiol. 2026;49(1):58-66. doi: 10.1111/pace.70097.

39. Iacopino S, Tondo C, Bianchi S, Russo AD, Maggio R, Rovaris G, et al. Safety and Feasibility of Pulsed Field Ablation with a Pentaspline Catheter in Patients with Cardiac Implantable Electronic Devices: A Multicentre Experience. Europace. 2025;27(8):euaf132. doi: 10.1093/europace/euaf132.

40. Chen S, Chun JKR, Bordignon S, Tohoku S, Urbanek L, Schaack D, et al. Pulsed Field Ablation-Based Pulmonary Vein Isolation in Atrial Fibrillation Patients with Cardiac Implantable Electronic Devices: Practical Approach and Device Interrogation (PFA in CIEDs). J Interv Card Electrophysiol. 2023;66(8):1929-38. doi: 10.1007/s10840-022-01445-0.

41. Wan EY, Rogers AJ, Lavelle M, Marcus M, Stone SA, Ottoboni L, et al. Periprocedural Management and Multidisciplinary Care Pathways for Patients with Cardiac Implantable Electronic Devices: A Scientific Statement from the American Heart Association. Circulation. 2024;150(8):e183-96. doi: 10.1161/CIR.0000000000001264.

42. Shoda M, Takami M, Imamura K, Fukuzawa K. Guidewire Entrapment in Pulmonary Veins during Pulsed Field Ablation of Atrial Fibrillation: A Case Series. HeartRhythm Case Rep. 2026;12(7):707-12. doi: 10.1016/j.hrcr.2026.04.002.

43. Bruss J, Kueffer T, Tanner H, Noti F, Haeberlin A, Thalmann G, et al. Hemolysis Induced by Pulsed-Field Ablation of Atrial Arrhythmias: A Comparative Analysis of Current Systems. J Cardiovasc Electrophysiol. 2025;36(10):2498-506. doi: 10.1111/jce.70049.

44. Venier S, Vaxelaire N, Jacon P, Carabelli A, Desbiolles A, Garban F, et al. Severe Acute Kidney Injury Related to Haemolysis after Pulsed Field Ablation for Atrial Fibrillation. Europace. 2023;26(1):euad371. doi: 10.1093/europace/euad371.

45. Mohanty S, Casella M, Compagnucci P, Torlapati PG, Della Rocca DG, La Fazia VM, et al. Acute Kidney Injury Resulting from Hemoglobinuria after Pulsed-Field Ablation in Atrial Fibrillation: Is it Preventable? JACC Clin Electrophysiol. 2024;10(4):709-15. doi: 10.1016/j.jacep.2023.12.008.

46. Kellum JA, Lameire N; KDIGO AKI Guideline Work Group. Diagnosis, Evaluation, and Management of Acute Kidney Injury: A KDIGO Summary (Part 1). Crit Care. 2013;17(1):204. doi: 10.1186/cc11454.

47. Hu X, Li W, Ren B, Zeng R. Incidence of Silent Cerebral Events Detected by MRI in Patients with Atrial Fibrillation Undergoing Pulsed Field Ablation vs Thermal Ablation: A Systematic Review and Network Meta-Analysis. Heart Rhythm. 2026;23(1):32-43. doi: 10.1016/j.hrthm.2025.04.008.

48. Patel C, Gerstenfeld EP, Gupta SK, Winterfield J, Woods C, Natale A, et al. Comparison of Cerebral Safety after Atrial Fibrillation Using Pulsed Field and Thermal Ablation: Results of the Neurological Assessment Subgroup in the ADVENT Trial. Heart Rhythm. 2024;21(11):2103-9. doi: 10.1016/j.hrthm.2024.05.048.

49. Reinsch N, Füting A, Höwel D, Bell J, Lin Y, Neven K. Cerebral Safety after Pulsed Field Ablation for Paroxysmal Atrial Fibrillation. Heart Rhythm. 2022;19(11):1813-8. doi: 10.1016/j.hrthm.2022.06.018.

50. Laskov V, Hozman M, Malikova H, Lauer D, Kremenova K, Herman D, et al. Cerebrovascular Ischemic Lesions after Pulsed Field Ablation for Atrial Fibrillation Using Variable-Loop Ablation Catheter. JACC Clin Electrophysiol. 2026;12(4):814-23. doi: 10.1016/j.jacep.2025.12.018.

51. Chéhirlian L, Koutbi L, Mancini J, Hourdain J, Richard-Vitton R, Wilkin M, et al. High Incidence of Phrenic Nerve Injury in Patients Undergoing Pulsed Field Ablation for Atrial Fibrillation. Heart Rhythm. 2025;22(12):e1206-13. doi: 10.1016/j.hrthm.2025.08.010.

52. Oda Y, Nogami A, Igarashi D, Usui R, Uno K. A Case Report of Persistent Phrenic Nerve Injury by Pulsed Field Ablation: Lessons from Anatomical Reconstruction and Electrode Positioning. HeartRhythm Case Rep. 2025;11(8):763-6. doi: 10.1016/j.hrcr.2025.05.015.

53. Howard B, Haines DE, Verma A, Kirchhof N, Barka N, Onal B, et al. Characterization of Phrenic Nerve Response to Pulsed Field Ablation. Circ Arrhythm Electrophysiol. 2022;15(6):e010127. doi: 10.1161/CIRCEP.121.010127.

54. Chen S, Narayan SM, Boveda S, Neven K, Ruwald MH, Martinek M, et al. 2025 International Expert Practical Guide on the Use of the Pentaspline Pulsed Field Ablation System in Atrial Fibrillation Ablation Procedures. Circ Arrhythm Electrophysiol. 2025;18(8):e013977. doi: 10.1161/CIRCEP.125.013977.

55. Zhou M, Yang H, Sun H, Yu M, Si D, He Y. Pulse Field Ablation: An Update on Energy-Specific Adverse Events. Rev Cardiovasc Med. 2025;26(10):44442. doi: 10.31083/RCM44442.

56. Sebag F, Roeun SR, Vincent MV, Durand JD, Miled MM, Simeon ES, et al. Comparison of Vagal Effect during Pulmonary Vein Isolation with Radiofrequency and Pulse Field Ablation: A Retrospective Study. Europace. 2025;27(Suppl 1):euaf085.345. doi: 10.1093/europace/euaf085.345.

57. van Zyl M, Khabsa M, Tri JA, Ladas TP, Yasin OZ, Ladejobi AO, et al. Open-Chest Pulsed Electric Field Ablation of Cardiac Ganglionated Plexi in Acute Canine Models. J Innov Card Rhythm Manag. 2022;13(7):5061-9. doi: 10.19102/icrm.2022.130704.

58. Del Monte A, Della Rocca DG, Pannone L, Vetta G, Fernández MC, Marcon L, et al. Pulsed Field Ablation of the Right Superior Pulmonary Vein Prevents Vagal Responses Via Anterior Right Ganglionated Plexus Modulation. Heart Rhythm. 2024;21(6):780-7. doi: 10.1016/j.hrthm.2024.01.040.

59. Vetta G, Della Rocca DG, Cespon-Fernandez M, Del Monte A, Magnocavallo M, Mene R, et al. Prophylactic Administration of Glycopyrrolate Prevents Vasovagal Responses Induced by Pulsed Field Ablation in Patients with Atrial Fibrillation. Europace. 2024;26(Suppl 1):euae102.194. doi: 10.1093/europace/euae102.194.

60. Santangeli P, Rosso R, Pachon JC. Managing Vagal Responses Induced by Pulsed Field Ablation: Go Right First? Heart Rhythm. 2024;21(6):788-9. doi: 10.1016/j.hrthm.2024.02.029.

61. Maan A, Chacko P, Kobeissy A, Koruth J. Esophageal Injury after Pulmonary Vein Isolation Using Pulsed Electric Fields. Heart Rhythm. 2026;23(4):800-4. doi: 10.1016/j.hrthm.2026.01.021.

62. Neven K, van Es R, van Driel V, van Wessel H, Fidder H, Vink A, et al. Acute and Long-Term Effects of Full-Power Electroporation Ablation Directly on the Porcine Esophagus. Circ Arrhythm Electrophysiol. 2017;10(5):e004672. doi: 10.1161/CIRCEP.116.004672.

63. Nies M, Koruth JS, Mlček M, Petru J, Tibenská VC, Watanabe K, et al. Is the Esophagus Spared during Pulsed Field Ablation? Early Histopathology and in Vivo Esophageal Retraction. Heart Rhythm. 2026;23(4):788-99. doi: 10.1016/j.hrthm.2025.06.033.

This is an open-access article distributed under the terms of the Creative Commons Attribution License