

# Benefits from catheter ablation in patients with pulmonary hypertension: Recent advances

Stepan Havranek, Zdenka Fingrova, Milan Dusik, Vladimir Dytrych, David Ambroz, Pavel Jansa

2<sup>nd</sup> Department of Internal Medicine — Cardiovascular Medicine, 1<sup>st</sup> Faculty of Medicine, Charles University, General University Hospital in Prague, Prague, Czech Republic\*

## Correspondence to:

Prof. Stepan Havranek, MD, PhD,  
Department of Internal Medicine  
— Cardiovascular Medicine,  
1<sup>st</sup> Faculty of Medicine,  
Charles University,  
General University Hospital  
in Prague,  
U Nemocnice 2, 128 08, Prague,  
Czech Republic,  
phone: +420723085848,  
e-mail: stepan.havranek@vfn.cz  
Copyright by the Author(s), 2024  
DOI: 10.33963/v.phj.101246

## Received:

May 29, 2024

## Accepted:

June 20, 2024

**Early publication date:** June 28, 2024

## ABSTRACT

Supraventricular tachycardia (SVT) is a frequent complication of pulmonary hypertension (PH). The most prevalent SVTs are atrial fibrillation (AF) and typical atrial flutter (AFL), followed by focal and macroreentrant atrial tachycardia (AT) and nodal arrhythmia (AV nodal reentry tachycardia or AV reentry tachycardia). SVT is frequently associated with functional deterioration and right ventricular failure in PH patients. According to some data, reestablishing sinus rhythm is associated with clinical improvement. Catheter ablation of typical AFL, nodal tachyarrhythmias, or other less complex focal ATs have been shown to be feasible, acutely effective, and safe in patients with PH. However, the long-term clinical outcome is modified by the recurrence of index arrhythmia and the onset of a new SVT. Due to right atrial dilatation, technical issues can arise when ablation is carried out. The role of catheter ablation in patients with AF or more complex AT is even less effective. The results mirror the success rate in the general AF population with non-paroxysmal AF. However, the data is limited, and electrophysiological procedures are also more often complicated by specific adverse events in a severely frail population. Despite these limitations, catheter ablation is the treatment of choice in less complex SVT, but the indications for AF ablation must be more individualized.

**Key words:** atrial fibrillation, atrial tachycardia, catheter ablation, pulmonary hypertension

## INTRODUCTION

Pulmonary hypertension (PH) can develop in multiple clinical conditions. It can be categorized according to the clinical presentation, pathological findings, and hemodynamic characteristics [1, 2]. Various treatment strategies have been established in patients with PH that improve hemodynamics, exercise capacity, and quality of life [1, 2]. Yet despite these advances, PH remains a progressive disease with a generally inauspicious prognosis.

## EPIDEMIOLOGY OF SUPRAVENTRICULAR TACHYCARDIAS IN PULMONARY HYPERTENSION

Supraventricular tachycardias (SVTs) have been frequently observed, with a cumulative incidence of 10%–29% in patients with both idiopathic [3] and secondary PH [4–7], including chronic thromboembolic PH, either

inoperable [4, 6] or treated with pulmonary endarterectomy [8].

The most prevalent SVTs are atrial fibrillation (AF) and atrial flutter (AFL) [3–6, 9]. We can also detect other paroxysmal SVTs in the PH population [7]. However, some series have indicated that AF and AFL are equally common [4, 6, 9]. Some data has shown an excessive AF prevalence [7, 13]. In the study by Fingrova et al. [7], AF made up 68% of all detected SVTs. It seems that the prevalence of AF reflects the regularity of follow-ups and active focus on the detection of both symptomatic and silent persistent AF. High AF prevalence could also result from including more potentially dormant combined post- and pre-capillary PH (Cpc-PH). The prevalence of AF increases by more than 50% when the postcapillary component is manifested [7, 10].

When patients with isolated pre-capillary PH (Ipc-PH) were analyzed, the highest

\*This institution is part of the European Reference Network for Rare Lung Diseases (ERN-Lung)

occurrence of all SVTs (mostly AF) among patients with pulmonary arterial hypertension (PAH) was associated with congenital heart disease (prevalence of 35%) [7]. The highest proportion of AF was identified among patients with lung disease, and PAH associated with connective tissue disease (AF prevalence of 87% and 89% of all SVTs, respectively) [7].

### **PATHOPHYSIOLOGICAL MECHANISMS OF SUPRAVENTRICULAR TACHYCARDIAS IN PULMONARY HYPERTENSION**

Apart from the precise mechanisms of SVT (i.e., AV nodal re-entry tachycardia [AVNRT], AV re-entry tachycardia, or typical AFL, triggers of AF in pulmonary veins), an arrhythmogenic mechanism of complex atrial tachycardias, including AF, in the population of PH patients appears to be more complicated. This more complex situation could predict ablation outcomes in patients with PH. The evidence supports the right atrial (RA) substrate for complex atrial arrhythmia: PH leads to an upstream enlargement of the RA due to right ventricular overload and functional impairment [11]. A long-standing PH is associated with progressively slowing conduction, reduced tissue voltage, and regions of electrical silence in the RA [12]. Finally, modulations of the autonomic system and an elevated right heart filling pressure may trigger and perpetuate related arrhythmia [13, 14].

The arrhythmogenic mechanism does not have to be related only to RA. It has been shown that the left-sided substrate could play a role in the arrhythmogenesis of complex atrial arrhythmia including AF, even in isolated precapillary PH [15]. When a post-capillary component of PH is present, the arrhythmia mechanisms are most likely different from Ipc-PH and more likely correspond to the arrhythmogenic substrate in left heart disease [16–18]. In patients with PH due to left heart disease, AF is prevalent in up to 60% [10].

Therefore, it is not surprising that the increase in the prevalence of SVT in the Cpc-PH group is mainly due to a higher occurrence of persistent or permanent AF. The high burden of persistent or permanent arrhythmia may result from LA remodeling [19, 20]. On the other hand, patients with AF have frequently decreased atrial contraction, atrioventricular asynchrony, and a rapid heart rate with a reduction of diastolic filling, which may represent the cause of an increase in pulmonary wedge pressure itself [17, 21–24].

### **CLINICAL SIGNIFICANCE**

It has also been demonstrated that SVT in patients with PH is frequently associated with functional deterioration and right ventricular failure [3–6, 9, 11]. Therefore, it is unsurprising that SVT development has also been investigated as a predictor of mortality [3, 4, 25]. Olsson et al. [4] identified that the estimated survival rate after diagnosing PH was reduced in patients with permanent AF compared

to patients with transient episodes or without arrhythmia. Another study confirmed that SVT in patients with idiopathic PAH presages substantial morbidity and may be a determinant of mortality [3]. Fingrova et al. [7] however showed in a multivariate analysis that rather than SVT manifestation, advanced age, male gender, deterioration of functional parameters, and a larger RA diameter were stronger predictors of mortality [7].

A strategy attempting sinus rhythm restoration in patients with idiopathic PH appears to improve the clinical outcome [3, 5]. For these reasons, European Society of Cardiology/European Respiratory Society guidelines for the management of PH promote a rhythm control strategy with cardioversion and antiarrhythmic drugs for some PH patients with SVT [2]. However, limited and often controversial data about the management of arrhythmias in the PH population exists. No data comparing prospectively rate and rhythm control strategies is available. In general, antiarrhythmic drugs may not be a feasible option because of their frequent lack of efficacy, harmful inotropic and chronotropic properties in the setting of right ventricle dysfunction (including beta-blockers or verapamil), severe side effects, and interaction with specific therapy for PH [26, 27]. The Ic class of antiarrhythmic drugs are contraindicated in patients with prior myocardial infarction and left ventricular systolic dysfunction. No data is available on structurally abnormal right heart patients [28]. On the other hand, widespread use of the Ic class has been recorded in multiple observational analyses. There is also no trial data investigating the safety and efficacy of amiodarone use in PH. Treatment with amiodarone could be complicated by pulmonary infiltration in 1%–2% of patients [26]. In a study by Soon et al. [27], a disproportionately high number of patients with idiopathic PAH suffered from amiodarone-induced thyroid dysfunction, with severe consequences. Amiodarone is a potent inhibitor of CYP 450 isoenzymes and glycoprotein P efflux transporters.

We speculate that amiodarone administration might influence levels of specific therapy, i.e., bosentan levels, as suggested by Reddy et al. [28].

### **CATHETER ABLATION OF TYPICAL ATRIAL FLUTTER AND LESS COMPLEX SUPRAVENTRICULAR TACHYARRHYTHMIAS**

Catheter ablation is the treatment of choice in PH, as with the general population for AVNRT, AV re-entry tachycardia, typical AFL, and focal ATs. Several retrospective studies, despite a limited number of patients, have reported that radiofrequency catheter ablation (CA) of typical AFL, nodal tachyarrhythmias, or other less complex focal ATs is feasible, acutely effective, and safe in patients with PH [5, 6, 29–36]. **Table 1** presents results from previously published cohorts.

However, the long-term clinical outcomes after CA have been found to be less positive and more divergent. Bradfield et al. reported that only 5/10 patients with acutely successful CTI ablation were completely arrhyth-

**Table 1.** Acute and long-term efficacy of catheter ablation in patients with pulmonary hypertension and supraventricular tachycardia

Authors	Patient cohort	Ablated arrhythmias	Acute efficacy	Complications	Any arrhythmia recurrence (index or newly diagnosed) during long-term follow-up
Tongers et al. 2006	8 patients with AFL/AVNRT	Typical AFL 5 AVNRT 3	Typical AFL 5/5 AVNRT 3/3	No complication	AFL 1/5 (redo in 1 patient) AVNRT 1/3 (AFL ablation in 1 patient) 24-month follow-up
Showkathali et al. 2011	22 patients with typical AFL	Typical AFL 22	Typical AFL 22/22	No complication	Typical AFL 3/22 3-month follow-up
Luesebrink et al. 2012	38 patients with typical AFL	Typical AFL 38	Typical AFL 38/38	No complication	Data not reported
Bradfield et al. 2012	12 patients with typical AFL	Typical AFL 14	12/14 (2 redo)	Procedural death 1 Right-sided heart failure 1	Typical AFL 5/10 3-month follow-up
Kanmanthareddy et al. 2014	43 patients with AT	Typical AFL 20 AF 10 AV node 13	Data not reported	Data not reported	Typical AFL 17/20 AF 10/10 1-year follow-up
Bandorski et al. 2014	32 patients with AT	Typical AFL 14 AT 13 AVNRT 4 AV node 1	Typical AFL 12/14 AT 4/13 AVNRT 4/4 AV node 1/1	Data not reported	Data not reported
Malaczynska-Rajpold et al. 2016	7 patients with AT	Typical AFL/other CTI-dependent MRAT 9 WPW 1	Typical AFL/CTI-dep MRAT 6/9 (3 redo) WPW 1/1	No complication	Typical AFL/CTI-dep MRAT 3/6 (3 redo) Follow-up period not reported
Zhou et al. 2021	60 patients with ablated SVT	Typical AFL 33 AVNRT 16 MRAT 8 AVRT 5 Focal AT 3	Typical AFL 32/33 AVNRT 15/16 MRAT 4/8 AVRT 5/5 Focal AT 3/3	AV block 1 Stroke 1 Pseudoaneurysm 1 Arterio-venous fistula 1	AFL 24/32 AVNRT 0/15 MRAT 1/4 AVRT 0/5 Focal AT 0/3 Median follow-up 36 (3–108) months
Kamada et al. 2021	23 patients with SVT	Typical AFL 18 MRAT 5 Focal AT 3 Non-sustained AT 3 AVNRT 1	Typical AFL 17/18 All ATs 8 /11 (failure in 3 non-sustained ATs) AVNRT 1/1	Bradycardia (junctional) after ablation of MRAT	All SVTs 12/12 (9 AT and 3 AF) Recurrence of index arrhythmia in 2 patients after ablation of AT
Havranek et al. 2023	74 patients with AF or AT	AF 38 AFL 23 AVNRT 3 Another AT 10	AF 36/38 AFL 21/23 Another AT 9/10	Severe vagal reaction 1 Pericardial effusion 1 Arterio-venous fistula 1 Low cardiac output 2 Sudden death during 24hr after ablation 1	AF 19/38 AFL 4/23 AVNRT 0/3 Other AT 4/10 Median follow-up 13 (12–36) months
Satish et al. 2023	38 patients with SVT	Typical AFL 29 AT 15 MRAT 7 AF 5 AVNRT 4	Typical AFL 4/5 AT 9/15 MRAT 4/7 AF 4/5 AVNRT 3/4	Death secondary to respiratory failure triggered by sedation during AF ablation 1 Postprocedural decompensation 9 (Typical AFL 5, AT 2, AVNRT 2)	Typical AFL 5/20 AT 2/7 MRAT 1/2 AF 4/4 AVNRT 0/2
Boyle et al. 2024	20 patients with AF	AF 28	Not reported	Groin hematoma 1 Tamponade and stroke 1 Phrenic nerve injury 1	After a single procedure, 50% After multiple procedures, 30% 3-year follow-up

Abbreviations: AF, atrial fibrillation; AFL, atrial flutter; AT, atrial tachycardia; AV, atrioventricular; AVNRT, atrioventricular nodal re-entry tachycardia; AVRT, atrioventricular re-entry tachycardia; CTI, cavo-tricuspid isthmus; MRAT, macro re-entry atrial tachycardia; SVT, supraventricular tachycardia; WPW, Wolff–Parkinson–White syndrome

mia-free after 3 months, and three patients had recurrent arrhythmias different from their initial typical AFL [29]. In 23 patients ablated for typical AFL and other organized SVTs, arrhythmia occurred in 12 patients during a 5-year follow-up, of whom a new onset of arrhythmia was seen in 10 cases [30]. On the other hand, a recent retrospective study found a more favorable outcome in 32 patients with successful ablation of typical AFL, with a recurrence rate of ~20% during a follow-up of 3–108 months. However, the proportion of recurrence of index arrhythmia and onset of new arrhythmias were not provided [33]. No recurrence of typical AFL was observed in a recent study by Havranek et al. [37]. This finding is far more favorable than some pre-

viously reported long-term data [29, 30]. The authors speculate that RFCA with 3D-electroanatomical mapping and direct visual control using intracardiac echocardiography could be responsible for such an outcome. Unfortunately, the recurrence of different arrhythmias in 4/23 AFL patients in both study arms was noticed during the follow-up, which concurs with previous results [30, 31].

Some technical challenges can arise when carrying out ablation due to right atrial and tricuspid annular dilatation in PH hearts. The same cases were acutely unsuccessful in these studies due to enormous RA dilatation. In parallel, significantly longer procedures, fluoroscopy, and total ablation times have been demonstrated in PH ablations [29,

31]. RA dilatation has some other consequences. The cycle length of a typical AFL in PH patients is prolonged [36, 37] compared to the general population [38]. Therefore, given the inherent difficulties with the diagnosis and ablation of typical AFL in PH, it is worth considering the use of 3D electroanatomical mapping, long steerable sheaths, and intracardiac echocardiography to improve the success rate and reduce the procedure time by enhancing the reach of tissue contact. Our experience shows that the most useful approach combines long steerable sheaths and intracardiac echocardiography. Steerable sheaths are regularly used in our center for PH patients. Another limitation could be venous access. Some anatomical abnormalities of femoral veins are typically found. Puncture of veins under ultrasound control might reduce complication rates.

Even though CA of AVNRT, focal AT, or typical AFL in patients with PH is generally a safe procedure, some procedure-related severe adverse events have been recognized [29, 33, 39]. Therefore, CA appears safe even in a population of frail PH patients when performed by experienced operators.

Reducing procedural time, preventing volume overload, eliminating hypotension, and experience with abnormal venous and right heart anatomy in PH are the cornerstones of developing the prevention of ablation-related complications.

### **CATHETER ABLATION OF ATRIAL FIBRILLATION AND COMPLEX ATRIAL TACHYARRHYTHMIAS**

Unlike typical AFL and paroxysmal SVT, data about ablation in left atrial-related arrhythmia, including AF and more complex AT, has only rarely been reported [17, 39, 41–43]. **Table 1** sets out the details. When looking at the acute efficacy and long-term outcome of CA in macro re-entrant atrial tachycardias, results are worse than in typical AFL, AVNRT, or focal AT. The long-term effect was positive only in ~50 % of patients [33, 36, 39].

The role of CA in the general population is well established. The standard of care is pulmonary vein isolation (PVI). As mentioned above, PH primarily results in structural and electrical remodeling in the right heart, which likely renders non-pulmonary vein triggers more critical, meaning the ablation strategy should be modified. The result of triggers arising distal to standard ablation sites is also allied to the condition's progressive nature. Patients with PH are likely to have a higher recurrence rate following AF ablation. PH has been found to be an independent risk factor for late recurrence [40].

The first retrospective series of 10 patients with PH ablated for AF has just been published in 2024 by Kanmanthareddy et al. [35]. They report pulmonary vein isolation with additional left atrial lesions in all patients, combined with cavotricuspid isthmus ablation in 4 subjects. All patients had significant left-atrial substrate defined by CARTO mapping. In all 10 patients, a recurrence of AF was docu-

mented during a 12-month follow-up. A similar experience was reported by Satish et al. [39]. Furthermore, Boyle et al. [41] published three case reports with patients suffering from AF and complex AT. Long-term failure of AF ablation was reported in one patient. In a second, repeat CA for focal AT was finally successful. In a third patient, multiple procedures led to the elimination of AF and AFL.

The first prospective randomized multicenter study, published in 2023, showed no benefit of an extensive first-line bi-atrial CA of all potentially arrhythmogenic substrates on top of selective ablation of index clinical arrhythmia in patients with AF or AT [37]. In the overall study, the recurrence of any arrhythmia was recognized in 27/71 ablated patients during a median 13 months (range 12–36) follow-up. This study also enrolled 38 patients with AF with the highest procedural failure. Arrhythmia-free survival was only 47%. CA was not primarily successful in two AF patients due to abnormal venous tortuosity and extremely dilated RA preventing successful transseptal puncture. The extensive ablation was intended to reduce a long-term arrhythmia recurrence rate by ablating all inducible arrhythmias, including those that did not manifest clinically before the procedure, and by preventative modification of arrhythmogenic substrate for potentially new and currently non-inducible arrhythmias.

The most recent data has just been published by Boyle et al. [42], who retrospectively analyzed the results of CA of AF in 20 patients recruited from 4 high-volume ablation centers. Twenty-eight ablations were performed in 20 referred subjects. The pulmonary veins were isolated in all 20 index ablation procedures and reisolated in 7/8 repeat procedures (88%). Two procedures were performed with cryoablation, and the rest with radiofrequency energy. The cavotricuspid isthmus was ablated in 13 patients (46%). Additional extrapulmonary vein triggers (posterior wall, left atrial roof, ligament of Marshall, mitral isthmus, coronary sinus floor, and focal left atrial scar) were targeted in 11 patients (39%). No ablation was performed in RA other than the cavotricuspid isthmus. Three-year AF-free survival was 50% after a single procedure, and 70% after multiple procedures.

It is worth underscoring that many adverse events were recorded in the two most extensive studies [37, 42]. In the cohort reported by Havranek et al. [37], apparent procedure-related complications were recognized in 3 patients: a prolonged severe vagal reaction during sheath removal at the end of the procedure with the need for short cardiopulmonary resuscitation; peri-procedural progression of conservatively treated pericardial effusion; and surgically treated arteriovenous fistula. Three more adverse events could probably be related to CA. Of these, two patients manifested low cardiac output after the procedure, which led to prolonged hospitalization in one patient and progression to terminal heart failure and death in the second patient. One patient died suddenly (pulseless electrical activity) the day after the ablation of AVNRT without evi-

dence of any periprocedural complication as assessed by autopsy. Hypoxia and end-stage heart failure were most likely responsible. The other three patients manifested severe sinus bradycardia and sinus arrest episodes after the termination of persistent arrhythmia; however, ablation in these patients was not done in proximity to the sinus node. In the second series [42], procedural complications occurred in 4 patients (14%). Three of these were considered severe: pericardial tamponade after double transeptal puncture together with stroke in the same patient, possibly because of reversal of anticoagulation immediately after left atrial ablation, and phrenic nerve injury. The only minor complication was a groin hematoma. Peri-procedural death secondary to acute mixed respiratory failure triggered by procedural moderate sedation for ablation of AF was also reported by Satish et al. [39]. To prevent a procedure-related bleeding complication, precise management of periprocedural anticoagulation should be applied. In our center, all PH patients are admitted one day before the planned procedure, and anticoagulation is precisely managed [43]. According to our experience, ablation with uninterrupted vitamin K antagonists with INR 2-3 is safe in patients with PH. When patients are treated with direct oral anticoagulants, we follow a protocol of minimally interrupted strategy, skipping a single dose on the procedure day without any low molecular weight heparin bridging.

In contrast to well-defined, organized SVTs (AVNRT, AFL, and less complex AT), the long-term results of AF ablation seem inauspicious. On the other hand, these results are comparable to the general population with AF, particularly given that nearly half of patients had persistent AF. The relatively high arrhythmia recurrence rate was similar to that in non-paroxysmal AF in non-PH patients with structural heart disease [44, 45]. A recent meta-analysis reported a pooled median success rate of 66.7% (95% CI, 60.8%–72.2%) after a single CA for non-paroxysmal AF [44]. Beyond PVI, several trigger and left atrial substrate modification ablation strategies have been proposed to improve success in non-paroxysmal AF. However, the randomized control trial STAR AF II indicated that adjunctive RF ablation strategies did not improve outcomes over PVI alone but were associated with higher fluoroscopy and procedure times [45]. A lack of benefit from an extensive ablation has also been found in patients with PH and AF or complex AT [37].

### **ABLATION FAILURE AND LIMITING FACTORS**

In many studies, a significant number of patients scheduled for CA of SVT were not eventually ablated due to some procedural failure. The leading reason was the non-inducibility or presence of only non-sustainable index arrhythmia, which precludes adequate mapping and leads to empirical ablation or makes ablation impossible. Secondly, more than index arrhythmia or multifocal tachycardia could be recognized, and ablation is not feasible or must be exten-

ded afterwards. Thirdly, the placement of catheters was not successful because of abnormal venous anatomy and an extremely dilated RA.

The next concern about the CA of SVT in PH is the high number of patients with different induced or spontaneous arrhythmia from tachycardia during screening [37]. Several explanations can be offered. Firstly, the coexistence of multiple arrhythmias in individual patients is quite common in patients with PH [37]. Secondly, one arrhythmia can “convert” to a different one, either spontaneously or by administering antiarrhythmic drugs. Thirdly, the dilatation and hypertrophy of the RA modify the typical pattern of arrhythmia on a standard ECG. It has been reported that aside from the prolongation of arrhythmia CL, both broader and smaller amplitude flutter waves have been found in patients with AFL and PH [46]. In the referred cohort, a typical ECG pattern was seen in only 42% of PH patients, compared to 100% of controls. A surface ECG is not a reliable predictor of CTI dependence, likely due to the severe right heart enlargement associated with PAH. Therefore, surface ECG pattern and AFL CL are of limited clinical utility in determining the origin of AFL in PH patients. Finally, 3D electroanatomical mapping allows for a more precise diagnosis of current arrhythmia. Although typical AFL can frequently be found as the first manifestation of SVT in patients with PH, AF is even more prevalent [3–6]. Importantly, new-onset arrhythmias (i.e., different from their index tachycardia) have been observed in 30%–48% of recurrent cases [29, 30, 37].

The frailty of patients with PH and SVTs is also an essential limit of electrophysiological procedures. Induction of arrhythmia is one of the typical factors leading to hemodynamic deterioration. Patients with PH frequently have hypoxia. General anesthesia could increase the single procedure success rate of CA of complex AT and shorten fluoroscopy or procedural time without increasing procedural complications [47]. However, there is concern about severe complications related to general anesthesia in patients with PH. PH is a serious condition, and the induction of general anesthesia can incur additional hemodynamic stress. It is also known that patients with severe PH have increased rates of delayed extubating, heart failure, and mortality after surgery in general [48, 49]. On the other hand, conscious sedation may result in accidental hypoventilation episodes with their consequences.

The final concern is stiff left atrial syndrome, a well known phenomenon with a prevalence of 1.4%–8% typically occurring several years after extensive complex catheter ablation in the left atrium [48–51]. There is a risk of further aggravation of PH with left atrial stiff syndrome after extensive catheter ablation.

This potentially long-term side effect of extensive left atrial ablation in PH patients is one of the arguments against routine extensive ablation in the left atrium in that population.

## CONCLUSIONS

CA for typical AFL, focal AT, and other paroxysmal SVT is an effective treatment despite PH. However, the long-term prognosis is limited mainly by the onset of new SVTs. CA for AF is also feasible in patients with PH, with similar success rates to those with non-paroxysmal AF. Complication rates may be higher than in non-PH patients, and specialized anesthetic and procedural management is likely needed to optimize safety.

Therefore, CA appears safe even in a population of frail PH patients when performed by experienced operators.

## Article information

**Conflict of interest:** None declared.

**Funding:** Supported by the Ministry of Health, Czech Republic — conceptual development of research organization (General University Hospital in Prague — VFN, 00064165).

**Open access:** This article is available in open access under Creative Commons Attribution-Non-Commercial-No Derivatives 4.0 International (CC BY-NC-ND 4.0) license, allowing to download articles and share them with others as long as they credit the authors and the publisher, but without permission to change them in any way or use them commercially. For commercial use, please contact the journal office at [polishheartjournal@ptkardio.pl](mailto:polishheartjournal@ptkardio.pl).

## REFERENCES

1. Simonneau G, Montani D, Celermajer D, et al. Haemodynamic definitions and updated clinical classification of pulmonary hypertension. *Eur Respir J*. 2019; 53(1): 1801913, doi: [10.1183/13993003.01913-2018](https://doi.org/10.1183/13993003.01913-2018), indexed in Pubmed: [30545968](https://pubmed.ncbi.nlm.nih.gov/30545968/).
2. Humbert M, Kovacs G, Hoeper MM, et al. 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. *Eur Heart J*. 2022; 43(38): 3618–3731, doi: [10.1093/eurheartj/ehac237](https://doi.org/10.1093/eurheartj/ehac237), indexed in Pubmed: [36017548](https://pubmed.ncbi.nlm.nih.gov/36017548/).
3. Wen Li, Sun ML, An P, et al. Frequency of supraventricular arrhythmias in patients with idiopathic pulmonary arterial hypertension. *Am J Cardiol*. 2014; 114(9): 1420–1425, doi: [10.1016/j.amjcard.2014.07.079](https://doi.org/10.1016/j.amjcard.2014.07.079), indexed in Pubmed: [25217453](https://pubmed.ncbi.nlm.nih.gov/25217453/).
4. Olsson KM, Nickel NP, Tongers J, et al. Atrial flutter and fibrillation in patients with pulmonary hypertension. *Int J Cardiol*. 2013; 167(5): 2300–2305, doi: [10.1016/j.ijcard.2012.06.024](https://doi.org/10.1016/j.ijcard.2012.06.024), indexed in Pubmed: [22727973](https://pubmed.ncbi.nlm.nih.gov/22727973/).
5. Ruiz-Cano MJ, Gonzalez-Mansilla A, Escribano P, et al. Clinical implications of supraventricular arrhythmias in patients with severe pulmonary arterial hypertension. *Int J Cardiol*. 2011; 146(1): 105–106, doi: [10.1016/j.ijcard.2010.09.065](https://doi.org/10.1016/j.ijcard.2010.09.065), indexed in Pubmed: [21056484](https://pubmed.ncbi.nlm.nih.gov/21056484/).
6. Tongers J, Schwerdtfeger B, Klein G, et al. Incidence and clinical relevance of supraventricular tachyarrhythmias in pulmonary hypertension. *Am Heart J*. 2007; 153(1): 127–132, doi: [10.1016/j.ahj.2006.09.008](https://doi.org/10.1016/j.ahj.2006.09.008), indexed in Pubmed: [17174650](https://pubmed.ncbi.nlm.nih.gov/17174650/).
7. Fingrova Z, Ambroz D, Jansa P, et al. The prevalence and clinical outcome of supraventricular tachycardia in different etiologies of pulmonary hypertension. *PLoS One*. 2021; 16(1): e0245752, doi: [10.1371/journal.pone.0245752](https://doi.org/10.1371/journal.pone.0245752), indexed in Pubmed: [33471824](https://pubmed.ncbi.nlm.nih.gov/33471824/).
8. Havranek S, Fingrova Z, Ambroz D, et al. Atrial fibrillation and atrial tachycardia in patients with chronic thromboembolic pulmonary hypertension treated with pulmonary endarterectomy. *Eur Heart J Suppl*. 2020; 22(Suppl F): F30–F37, doi: [10.1093/eurheartj/suaa096](https://doi.org/10.1093/eurheartj/suaa096), indexed in Pubmed: [32694951](https://pubmed.ncbi.nlm.nih.gov/32694951/).
9. Daliento L, Somerville J, Presbitero P, et al. Eisenmenger syndrome. Factors relating to deterioration and death. *Eur Heart J*. 1998; 19(12): 1845–1855, doi: [10.1053/euhj.1998.1046](https://doi.org/10.1053/euhj.1998.1046), indexed in Pubmed: [9886728](https://pubmed.ncbi.nlm.nih.gov/9886728/).
10. Rottlaender D, Motloch LJ, Schmidt D, et al. Clinical impact of atrial fibrillation in patients with pulmonary hypertension. *PLoS One*. 2012; 7(3): e33902, doi: [10.1371/journal.pone.0033902](https://doi.org/10.1371/journal.pone.0033902), indexed in Pubmed: [22439013](https://pubmed.ncbi.nlm.nih.gov/22439013/).
11. Pietra GG, Capron F, Stewart S, et al. Pathologic assessment of vasculopathies in pulmonary hypertension. *J Am Coll Cardiol*. 2004; 43(12 Suppl S): 25S–32S, doi: [10.1016/j.jacc.2004.02.033](https://doi.org/10.1016/j.jacc.2004.02.033), indexed in Pubmed: [15194175](https://pubmed.ncbi.nlm.nih.gov/15194175/).
12. Medi C, Kalman JM, Ling LH, et al. Atrial electrical and structural remodeling associated with longstanding pulmonary hypertension and right ventricular hypertrophy in humans. *J Cardiovasc Electrophysiol*. 2012; 23(6): 614–620, doi: [10.1111/j.1540-8167.2011.02255.x](https://doi.org/10.1111/j.1540-8167.2011.02255.x), indexed in Pubmed: [22269035](https://pubmed.ncbi.nlm.nih.gov/22269035/).
13. Folino AF, Bobbo F, Schiraldi C, et al. Ventricular arrhythmias and autonomic profile in patients with primary pulmonary hypertension. *Lung*. 2003; 181(6): 321–328, doi: [10.1007/s00408-003-1034-x](https://doi.org/10.1007/s00408-003-1034-x), indexed in Pubmed: [14749936](https://pubmed.ncbi.nlm.nih.gov/14749936/).
14. Schrier RW, Bansal S. Pulmonary hypertension, right ventricular failure, and kidney: different from left ventricular failure? *Clin J Am Soc Nephrol*. 2008; 3(5): 1232–1237, doi: [10.2215/CJN.01960408](https://doi.org/10.2215/CJN.01960408), indexed in Pubmed: [18614776](https://pubmed.ncbi.nlm.nih.gov/18614776/).
15. Fingrova Z, Havranek S, Ambroz D, et al. The left atrial substrate plays a significant role in the development of complex atrial tachycardia in patients with precapillary pulmonary hypertension. *BMC Cardiovasc Disord*. 2019; 19(1): 157, doi: [10.1186/s12872-019-1142-z](https://doi.org/10.1186/s12872-019-1142-z), indexed in Pubmed: [31253083](https://pubmed.ncbi.nlm.nih.gov/31253083/).
16. Crijns HJ, Tjeerdsma G, de Kam PJ, et al. Prognostic value of the presence and development of atrial fibrillation in patients with advanced chronic heart failure. *Eur Heart J*. 2000; 21(15): 1238–1245, doi: [10.1053/euhj.1999.2107](https://doi.org/10.1053/euhj.1999.2107), indexed in Pubmed: [10924313](https://pubmed.ncbi.nlm.nih.gov/10924313/).
17. Maisel WH, Stevenson LW. Atrial fibrillation in heart failure: epidemiology, pathophysiology, and rationale for therapy. *Am J Cardiol*. 2003; 91(6A): 2D–8D, doi: [10.1016/s0002-9149\(02\)03373-8](https://doi.org/10.1016/s0002-9149(02)03373-8), indexed in Pubmed: [12670636](https://pubmed.ncbi.nlm.nih.gov/12670636/).
18. Middlekauff HR, Stevenson WG, Stevenson LW. Prognostic significance of atrial fibrillation in advanced heart failure. A study of 390 patients. *Circulation*. 1991; 84(1): 40–48, doi: [10.1161/01.cir.84.1.40](https://doi.org/10.1161/01.cir.84.1.40), indexed in Pubmed: [2060110](https://pubmed.ncbi.nlm.nih.gov/2060110/).
19. Spach MS, Josephson ME. Initiating reentry: The role of nonuniform anisotropy in small circuits. *J Cardiovasc Electrophysiol*. 1994; 5(2): 182–209, doi: [10.1111/j.1540-8167.1994.tb01157.x](https://doi.org/10.1111/j.1540-8167.1994.tb01157.x), indexed in Pubmed: [8186887](https://pubmed.ncbi.nlm.nih.gov/8186887/).
20. Ausma J, Wijffels M, Thoné F, et al. Structural changes of atrial myocardium due to sustained atrial fibrillation in the goat. *Circulation*. 1997; 96(9): 3157–3163, doi: [10.1161/01.cir.96.9.3157](https://doi.org/10.1161/01.cir.96.9.3157), indexed in Pubmed: [9386188](https://pubmed.ncbi.nlm.nih.gov/9386188/).
21. Gillette PC, Smith RT, Garson A, et al. Chronic supraventricular tachycardia. A curable cause of congestive cardiomyopathy. *JAMA*. 1985; 253(3): 391–392, doi: [10.1001/jama.253.3.391](https://doi.org/10.1001/jama.253.3.391), indexed in Pubmed: [3965793](https://pubmed.ncbi.nlm.nih.gov/3965793/).
22. Hsu LF, Jaïs P, Sanders P, et al. Catheter ablation for atrial fibrillation in congestive heart failure. *N Engl J Med*. 2004; 351(23): 2373–2383, doi: [10.1056/NEJMoa041018](https://doi.org/10.1056/NEJMoa041018), indexed in Pubmed: [15575053](https://pubmed.ncbi.nlm.nih.gov/15575053/).
23. Cha YM, Redfield MM, Shen WK, et al. Atrial fibrillation and ventricular dysfunction: a vicious electromechanical cycle. *Circulation*. 2004; 109(23): 2839–2843, doi: [10.1161/01.CIR.0000132470.78896.A8](https://doi.org/10.1161/01.CIR.0000132470.78896.A8), indexed in Pubmed: [15197156](https://pubmed.ncbi.nlm.nih.gov/15197156/).
24. Morillo CA, Klein GJ, Jones DL, et al. Chronic rapid atrial pacing. Structural, functional, and electrophysiological characteristics of a new model of sustained atrial fibrillation. *Circulation*. 1995; 91(5): 1588–1595, doi: [10.1161/01.cir.91.5.1588](https://doi.org/10.1161/01.cir.91.5.1588), indexed in Pubmed: [7867201](https://pubmed.ncbi.nlm.nih.gov/7867201/).
25. Smith B, Genuardi MV, Koczo A, et al. Atrial arrhythmias are associated with increased mortality in pulmonary arterial hypertension. *Pulm Circ*. 2018; 8(3): 2045894018790316, doi: [10.1177/2045894018790316](https://doi.org/10.1177/2045894018790316), indexed in Pubmed: [29969045](https://pubmed.ncbi.nlm.nih.gov/29969045/).
26. Effect of prophylactic amiodarone on mortality after acute myocardial infarction and in congestive heart failure: meta-analysis of individual data from 6500 patients in randomised trials. Amiodarone Trials Meta-Analysis Investigators. *Lancet*. 1997; 350(9089): 1417–1424, indexed in Pubmed: [9371164](https://pubmed.ncbi.nlm.nih.gov/9371164/).
27. Soon E, Toshner M, Mela M, et al. Risk of potentially life-threatening thyroid dysfunction due to amiodarone in idiopathic pulmonary arterial hypertension patients. *J Am Coll Cardiol*. 2011; 57(8): 997–998, doi: [10.1016/j.jacc.2010.09.059](https://doi.org/10.1016/j.jacc.2010.09.059), indexed in Pubmed: [21329849](https://pubmed.ncbi.nlm.nih.gov/21329849/).
28. Reddy SA, Nethercott SL, Khialani BV, et al. Management of arrhythmias in pulmonary hypertension. *J Interv Card Electrophysiol*. 2021; 62(2): 219–229, doi: [10.1007/s10840-021-00988-y](https://doi.org/10.1007/s10840-021-00988-y), indexed in Pubmed: [33821385](https://pubmed.ncbi.nlm.nih.gov/33821385/).

29. Bradfield J, Shapiro S, Finch W, et al. Catheter ablation of typical atrial flutter in severe pulmonary hypertension. *J Cardiovasc Electrophysiol.* 2012;23(11):1185–1190, doi: [10.1111/j.1540-8167.2012.02387.x](https://doi.org/10.1111/j.1540-8167.2012.02387.x), indexed in Pubmed: [22734591](https://pubmed.ncbi.nlm.nih.gov/22734591/).
30. Kamada H, Kaneyama J, Inoue YY, et al. Long term prognosis in patients with pulmonary hypertension undergoing catheter ablation for supraventricular tachycardia. *Sci Rep.* 2021;11(1):16176, doi: [10.1038/s41598-021-95508-3](https://doi.org/10.1038/s41598-021-95508-3), indexed in Pubmed: [34376719](https://pubmed.ncbi.nlm.nih.gov/34376719/).
31. Luesebrink U, Fischer D, Gezgin F, et al. Ablation of typical right atrial flutter in patients with pulmonary hypertension. *Heart Lung Circ.* 2012;21(11):695–699, doi: [10.1016/j.hlc.2012.06.005](https://doi.org/10.1016/j.hlc.2012.06.005), indexed in Pubmed: [22795737](https://pubmed.ncbi.nlm.nih.gov/22795737/).
32. Showkathali R, Tayebjee MH, Grapsa J, et al. Right atrial flutter isthmus ablation is feasible and results in acute clinical improvement in patients with persistent atrial flutter and severe pulmonary arterial hypertension. *Int J Cardiol.* 2011;149(2):279–280, doi: [10.1016/j.ijcard.2011.02.059](https://doi.org/10.1016/j.ijcard.2011.02.059), indexed in Pubmed: [21420184](https://pubmed.ncbi.nlm.nih.gov/21420184/).
33. Zhou B, Zhu YJ, Zhai ZQ, et al. Radiofrequency catheter ablation of supraventricular tachycardia in patients with pulmonary hypertension: feasibility and long-term outcome. *Front Physiol.* 2021;12:674909, doi: [10.3389/fphys.2021.674909](https://doi.org/10.3389/fphys.2021.674909), indexed in Pubmed: [34220537](https://pubmed.ncbi.nlm.nih.gov/34220537/).
34. Bandorski D, Schmitt J, Kurzlechner C, et al. Electrophysiological studies in patients with pulmonary hypertension: A retrospective investigation. *Biomed Res Int.* 2014;2014:617565, doi: [10.1155/2014/617565](https://doi.org/10.1155/2014/617565), indexed in Pubmed: [24977152](https://pubmed.ncbi.nlm.nih.gov/24977152/).
35. Kanmanthareddy A, Reddy YM, Boolani H, et al. Incidence, predictors, and clinical course of atrial tachyarrhythmias in patients with pulmonary hypertension. *J Interv Card Electrophysiol.* 2014;41(1):9–14, doi: [10.1007/s10840-014-9928-5](https://doi.org/10.1007/s10840-014-9928-5), indexed in Pubmed: [25005454](https://pubmed.ncbi.nlm.nih.gov/25005454/).
36. Małaczyńska-Rajpold K, Komosa A, Błaszyk K, et al. The management of supraventricular tachyarrhythmias in patients with pulmonary arterial hypertension. *Heart Lung Circ.* 2016;25(5):442–450, doi: [10.1016/j.hlc.2015.10.008](https://doi.org/10.1016/j.hlc.2015.10.008), indexed in Pubmed: [26643289](https://pubmed.ncbi.nlm.nih.gov/26643289/).
37. Havranek S, Fingrova Z, Skala T, et al. Catheter ablation of atrial fibrillation and atrial tachycardia in patients with pulmonary hypertension: a randomized study. *Europace.* 2023;25(5):euaad131, doi: [10.1093/euro-pace/euad131](https://doi.org/10.1093/euro-pace/euad131), indexed in Pubmed: [37178136](https://pubmed.ncbi.nlm.nih.gov/37178136/).
38. Havránek S, Simek J, Stovíček P, et al. Distribution of mean cycle length in cavo-tricuspid isthmus dependent atrial flutter. *Physiol Res.* 2012;61(1):43–51, doi: [10.33549/physiolres.932204](https://doi.org/10.33549/physiolres.932204), indexed in Pubmed: [22188106](https://pubmed.ncbi.nlm.nih.gov/22188106/).
39. Satish T, Chin K, Patel N. Outcomes after supraventricular tachycardia ablation in patients with group 1 pulmonary hypertension. *Cardiol Res.* 2023;14(5):403–408, doi: [10.14740/cr1556](https://doi.org/10.14740/cr1556), indexed in Pubmed: [37936620](https://pubmed.ncbi.nlm.nih.gov/37936620/).
40. Zhang YQ, Zhang FL, Wang WW, et al. The correlation of pulmonary arterial hypertension with late recurrence of paroxysmal atrial fibrillation after catheter ablation. *J Thorac Dis.* 2018;10(5):2789–2794, doi: [10.21037/jtd.2018.04.92](https://doi.org/10.21037/jtd.2018.04.92), indexed in Pubmed: [29997941](https://pubmed.ncbi.nlm.nih.gov/29997941/).
41. Boyle TA, Daimee UA, Simpson CE, et al. Left atrial ablation for the management of atrial tachyarrhythmias in patients with pulmonary hypertension: A case series. *Heart Rhythm Case Rep.* 2022;8(4):275–279, doi: [10.1016/j.hrcr.2022.01.012](https://doi.org/10.1016/j.hrcr.2022.01.012), indexed in Pubmed: [35497477](https://pubmed.ncbi.nlm.nih.gov/35497477/).
42. Boyle TA, Ha B, Haq I, et al. Atrial fibrillation ablation in patients with pulmonary hypertension: Multicenter experience. *Heart Rhythm.* 2024, doi: [10.1016/j.hrthm.2024.04.056](https://doi.org/10.1016/j.hrthm.2024.04.056), indexed in Pubmed: [38621500](https://pubmed.ncbi.nlm.nih.gov/38621500/).
43. Liao JN, Chan YH, Kuo L, et al. Optimal anticoagulation in elderly patients with atrial fibrillation: Which drug at which dose? *Kardiol Pol.* 2022;80(2):128–136, doi: [10.33963/KP.a2022.0046](https://doi.org/10.33963/KP.a2022.0046), indexed in Pubmed: [35167115](https://pubmed.ncbi.nlm.nih.gov/35167115/).
44. Voskoboinik A, Moskovitch JT, Harel N, et al. Revisiting pulmonary vein isolation alone for persistent atrial fibrillation: A systematic review and meta-analysis. *Heart Rhythm.* 2017;14(5):661–667, doi: [10.1016/j.hrthm.2017.01.003](https://doi.org/10.1016/j.hrthm.2017.01.003), indexed in Pubmed: [28434446](https://pubmed.ncbi.nlm.nih.gov/28434446/).
45. Verma A, Jiang Cy, Betts TR, et al. Approaches to catheter ablation for persistent atrial fibrillation. *N Engl J Med.* 2015;372(19):1812–1822, doi: [10.1056/NEJMoa1408288](https://doi.org/10.1056/NEJMoa1408288), indexed in Pubmed: [25946280](https://pubmed.ncbi.nlm.nih.gov/25946280/).
46. Matsuo S, Yamane T, Tokuda M, et al. Prospective randomized comparison of a steerable versus a non-steerable sheath for typical atrial flutter ablation. *Europace.* 2010;12(3):402–409, doi: [10.1093/europace/eup434](https://doi.org/10.1093/europace/eup434), indexed in Pubmed: [20083483](https://pubmed.ncbi.nlm.nih.gov/20083483/).
47. Di Biase L, Conti S, Mohanty P, et al. General anesthesia reduces the prevalence of pulmonary vein reconnection during repeat ablation when compared with conscious sedation: results from a randomized study. *Heart Rhythm.* 2011;8(3):368–372, doi: [10.1016/j.hrthm.2010.10.043](https://doi.org/10.1016/j.hrthm.2010.10.043), indexed in Pubmed: [21055479](https://pubmed.ncbi.nlm.nih.gov/21055479/).
48. Bellotti A, Arora S, Gustafson C, et al. Predictors of post-induction hypotension for patients with pulmonary hypertension. *Cureus.* 2022;14(11):e31887, doi: [10.7759/cureus.31887](https://doi.org/10.7759/cureus.31887), indexed in Pubmed: [36579234](https://pubmed.ncbi.nlm.nih.gov/36579234/).
49. Lai HC, Wang KY, Lee WL, et al. Severe pulmonary hypertension complicates postoperative outcome of non-cardiac surgery. *Br J Anaesth.* 2007;99(2):184–190, doi: [10.1093/bja/aem126](https://doi.org/10.1093/bja/aem126), indexed in Pubmed: [17576968](https://pubmed.ncbi.nlm.nih.gov/17576968/).
50. Witt CM, Fenstad ER, Cha YM, et al. Increase in pulmonary arterial pressure after atrial fibrillation ablation: incidence and associated findings. *J Interv Card Electrophysiol.* 2014;40(1):47–52, doi: [10.1007/s10840-014-9875-1](https://doi.org/10.1007/s10840-014-9875-1), indexed in Pubmed: [24532114](https://pubmed.ncbi.nlm.nih.gov/24532114/).
51. Yang Y, Liu Q, Wu Z, et al. Stiff left atrial syndrome: A complication undergoing radiofrequency catheter ablation for atrial fibrillation. *J Cardiovasc Electrophysiol.* 2016;27(7):884–889, doi: [10.1111/jce.12966](https://doi.org/10.1111/jce.12966), indexed in Pubmed: [26920815](https://pubmed.ncbi.nlm.nih.gov/26920815/).