ESC European Society of Cardiology

Europace (2020) **0**, 1–10
doi:10.1093/europace/euaa209

CLINICAL RESEARCH

Oesophageal Probe Evaluation in Radiofrequency Ablation of Atrial Fibrillation (OPERA): results from a prospective randomized trial

Katharina Schoene [1,2]*, Arash Arya[1], Friederike Grashoff[2], Helge Knopp[3], Alexander Weber[4], Matthias Lerche[1], Sebastian König[1], Sebastian Hilbert[1], Simon Kircher[1], Livio Bertagnolli[1], Borislav Dinov[1], Gerhard Hindricks[1,2], Ulrich Halm[5], Markus Zachäus[5], and Philipp Sommer[6]

[1]Department of Electrophysiology, Heart Center, University of Leipzig, Struempellstrasse 39, 04289 Leipzig, Germany; [2]Leipzig Heart Institute GmbH, Leipzig, Germany; [3]Helios Klinikum Leisnig, Leisnig, Germany; [4]KMG Klinikum Güstrow, Güstrow, Germany; [5]Parkkrankenhaus Leipzig, Leipzig, Germany; and [6]Herz- und Diabetes Zentrum NRW, Ruhr University Bochum, Bad Oeynhausen, Germany

Received 8 March 2020; editorial decision 30 June 2020; accepted 10 July 2020

Aims The aim of the study was to determine the incidence of oesophageal lesions after radiofrequency ablation (RFA) of atrial fibrillation (AF) with or without the use of oesophageal temperature probes.

Methods and results Two hundred patients were prospectively randomized into two groups: the OPERA+ group underwent RFA using oesophageal probes (SensiTherm™); the OPERA− group received RFA using fixed energy levels of 25 W at the posterior wall without an oesophageal probe. All patients underwent post-interventional endoscopy and Holter-electrocardiogram after 6 months. (Clinical.Trials.gov: NCT03246594). One hundred patients were randomized in OPERA+ and 100 patients in OPERA−. The drop-out rate was 10%. In total, 18/180 (10%) patients developed endoscopically diagnosed oesophageal lesions (EDEL). There was no difference between the groups with 10/90 (11%) EDEL in OPERA+ vs. 8/90 (9%) in OPERA− ($P = 0.62$). Despite the higher power delivered at the posterior wall in OPERA+ [28 ± 4 vs. 25 ± 2 W ($P = 0.001$)], the average EDEL size was equal [5.7 ± 2.6 vs. 4.5 ± 1.7 mm ($P = 0.38$)]. The peak temperature did not correlate with EDEL size. During follow-up, no patient died. Only one patient in OPERA− required a specific therapy for treatment of the lesion. Cumulative AF recurrence after 6 (3–13) months was 28/87 (32%) vs. 34/88 (39%), $P = 0.541$.

Conclusion This first randomized study demonstrates that intraoesophageal temperature monitoring using the SensiTherm™ probe does not affect the probability of developing EDEL. The peak temperature measured by the thermoprobe seems not to correlate with the incidence of EDEL. Empiric energy reduction at the posterior wall did not affect the efficacy of the procedure.

Keywords Ablation • Atrial fibrillation • Oesophageal lesion • Endoscopically diagnosed oesophageal lesions • Atrio-oesophageal fistula

* Corresponding author. Tel: +49 341 865 1413; fax: +49 341 865 1460. *E-mail address*: schoene_katha@t-online.de

What's new?

- In this first prospective randomized study, we could demonstrate that the use of intraoesophageal temperature monitoring (SensiTherm™) does not affect the incidence of endoscopically diagnosed oesophageal lesions.
- Empiric reduction of ablation energy did not result in impaired efficacy of the procedure with identical success rates in both groups.

Introduction

Catheter ablation has become a standard therapy for treatment of atrial fibrillation (AF).[1] Fortunately, overall rate of severe complications due to ablation for AF is low.[2] However, a recent large study out of the USA evaluating 60 203 admissions of patients for AF ablation between 2010 and 2015 reported an early mortality in 0.46% of cases.[3] Nevertheless, atrio-oesophageal fistula (AEF) still occur with an estimated incidence of 0.05–0.15%.[4] With a mortality of >80%, it remains the most serious complication.[5,6] Endoscopically diagnosed oesophageal lesions (EDEL) after AF ablation can progress to oesophageal perforation and AEF.[7] In case of an initial deep ulcer, 4.2% of these lesions progressed to perforation or AEF.[8] The use of oesophageal probes for temperature monitoring during radiofrequency ablation (RFA) for AF is controversially discussed. According to the Consensus Statement on catheter and surgical ablation of AF, the use of an oesophageal temperature probe for guiding energy delivery is 'reasonable' with a IIa recommendation and level of evidence class C.[1] There is no data from randomized studies evaluating the value of using intraoesophageal thermoprobes.

Therefore, the aim of the present study was to analyse the impact of oesophageal probes in RFA of AF with respect to post-interventional oesophageal complications.

Methods

Study design

The sample size was calculated for non-inferiority of the OPERA+ group with regards to the incidence of EDEL. From 2017 to 2019 at the heart centre of Leipzig, 200 patients with an indication for RFA of AF were prospectively randomized in a 1:1 ratio into two groups. The process was an on-site randomization using sealed opaque envelopes. The OPERA+ group underwent RFA using an oesophageal probe (SensiTherm™, FIAB, Firenze, Italy) to measure the intraluminal oesophageal temperature during ablation; energy titration was at the operators' discretion with a given temperature cut-off which should not be exceeded. The OPERA− group received RFA using fixed energy levels of 25 W when ablating the posterior left atrial wall without using an oesophageal probe (*Figure 1*). All patients underwent post-interventional endoscopy and a 6 months follow-up including re-evaluation of oesophageal complications (primary endpoint) and assessment of rhythm stability (secondary endpoint). The study was registered in Clinical.Trials.gov (NCT03246594). It was approved by the local ethics committee and performed in accordance with the declaration of Helsinki. Written informed consent was obtained from all patients before enrolment in this study.

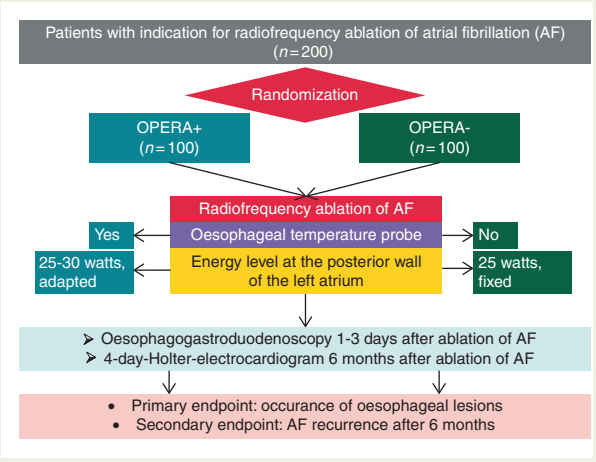

Figure 1 Study flowchart.

Ablation procedure

With more than 1200 RFA for AF per year, the Heart Center Leipzig is a high-volume centre.

Our ablation approach has been described previously.[9] In brief, interventional RFA of AF was performed under deep sedation with propofol infusion. After transseptal access, the pulmonary veins (PVs) were isolated ipsilateral in pairs with a circumferential lesion using point-by-point ablation. The ablation catheter was an irrigated tip catheter using radiofrequency as the energy source and a contact pressure of 10–30 g. The ablation lines were placed at an antral level and circumferential around the left- and right-sided PVs. Additional ablation lines were performed at the discretion of the operator as a substrate-based approach.[9] Mapping was performed using 3D mapping systems: EnSite® Precision (Abbott/St. Jude Medical, St. Paul, MN, USA) or CARTO® (Biosense Webster, Baldwin Park, CA, USA). Intracardiac ultrasound guidance was not used.

Oesophageal temperature measurement and titration of energy during ablation

During ablation of patients in the OPERA+ group, the intraoesophageal temperature was measured by an intraluminal oesophageal probe (SensiTherm™, FIAB, Firenze, Italy). The SensiTherm™ probe is approved for the surveillance of the oesophageal temperature during left atrial ablation procedures. This multisensor probe has a body of 7 French and 5 superficially located olive-shaped electrodes. The three middle electrodes are for temperature measurement and the proximal and distal electrodes can be used for sensing and pacing. This unsteerable probe has a soft silicone tip to avoid traumatic injury during intraoesophageal placement. To ensure maximal exposure to radiofrequency energy the probe was permanently adjusted according to the current position of the ablation catheter. Energy level of radiofrequency at the beginning of ablation at the posterior wall was 25 W and was increased to 30 W in absence of temperature rise above 40°C. The OPERA− group received RFA using a limit of energy of 25 W at the posterior left atrial wall without using an oesophageal probe. The cut-off temperature for oesophageal heating to stop RFA was 41.0°C. Post-interventionally, every patient was treated with 40 mg Pantoprazol per day for 4 weeks.

Post-ablation oesophageal endoscopy

Every patient underwent post-interventional oesophagogastroduodenoscopy (EGD) 1–3 days after the ablation procedure by a gastroenterologist experienced in evaluation of radiofrequency-induced thermal lesions.

Endoscopically detected oesophageal lesions approximately at 30 cm ab oral close to the left atrium (EDEL) were photo-documented and classified according to Deneke *et al.*[7]: erythema and erosion ≤5 mm were defined as EDEL 1 and lesions or ulcers >5 mm with or without perforation as EDEL 2. The endoscopists were blinded to the respective OPERA group.

Follow-up

Every patient underwent 4-day-Holter-electrocardiogram (ECG) 6 months after PV isolation (PVI). Documented atrial arrhythmia >30 s was classified as a recurrence. The time period up to 3 months after PVI was defined as a blanking period. Thereafter, any ECG-documented AF or left atrial flutter was registered and counted as a procedural failure.

Statistics

All statistical tests were performed using SPSS version 26.0 (SPSS, Chicago, IL, USA). Each categorical variable is expressed as number and percentage of patients. Continuous data are reported as mean ± standard deviation. Follow-up time is given as median (interquartile range). The groups were compared using the χ^2 test or Fisher's exact test for categorical variables and the Wilcoxon rank-sum test for continuous variables. Recurrence rates of AF were estimated by Kaplan–Meier analysis. A two-sided *P*-value <0.05 was considered to indicate statistical significance.

Results

Patient characteristics

Two hundred patients with an indication for ablation of AF were randomized. A total of 180 patients [119 male (66%), mean age 62.7 ± 9.8 years and 111 (62%) with persistent AF] were included in the study. Patient characteristics stratified by randomization are shown in *Table 1* and were well balanced between the groups. The drop-out rate of 10% (10/100 in each group) was mostly due to incidental findings in preinterventional imaging, withdrawal of consent for the study or the EGD.

Procedure data

The procedural data are shown in *Table 2*. There were no differences between the groups OPERA+ and OPERA− with respect to the total procedure time (130 ± 42 min vs. 134 ± 44 min, not significant), the total ablation time (35 ± 20 min vs. 36 ± 20 min, not significant), ablation at the posterior wall (96.7% vs. 98.9%, not significant), placement of additional ablation lines (24.4% vs. 17.8%, not significant), and the maximal energy level at the anterior part of the lesion (36.3 W vs. 36.2 W, not significant). With regard to the energy level at the posterior wall of the left atrium, the minimal energy was significantly lower in OPERA− compared to OPERA+ (23.3 W vs. 24.2 W, $P < 0.001$). There was a trend to more frequent linear lesions at the posterior wall in OPERA+ which may have contributed to the higher incidence of EDEL—without reaching the level of statistical significance. The oesophagus position as projected in the 3D mapping system was close to the left PVs in 47% of the patients. The ablation related complication rate was low and equally distributed between the groups (*Table 2*).

In one patient of OPERA+, the oesophageal probe had to be removed before ablation due to the need of invasive ventilation.

Primary endpoint—incidence of oesophageal lesions

The mean time between ablation procedure and EGD was 1.3 ± 0.8 days. In total, 18/180 (10%) patients developed EDEL (*Table 3*). There was no difference between the groups with 10/90 (11%) EDEL in OPERA+ vs. 8/90 (9%) in OPERA− ($P = 0.62$). The distribution of EDEL 1 (≤5 mm)/EDEL 2 (>5 mm) was similar

Table 1 Baseline characteristics

Variables	All patients (*n* = 180)	OPERA+ (*n* = 90)	OPERA− (*n* = 90)	*P*-value
Age (years)	63 ± 10	63 ± 9	63 ± 11	0.93
Male sex, *n* (%)	119 (66.1)	60 (66.7)	59 (65.6)	0.88
Paroxysmal AF, *n* (%)	69 (38.4)	31 (34.4)	38 (42.2)	0.28
Persistent AF, *n* (%)	111 (61.7)	59 (65.6)	52 (57.8)	0.28
EHRA class	2.5 ± 0.6	2.5 ± 0.6	2.5 ± 0.6	0.81
NYHA class	1.3 ± 1.2	1.2 ± 1.2	1.4 ± 1.2	0.30
CHA_2DS_2 VASc score	2.6 ± 1.5	2.6 ± 1.5	2.6 ± 1.5	0.92
Hypertension, *n* (%)	156 (86.7)	79 (87.8)	77 (85.6)	0.66
Diabetes mellitus, *n* (%)	32 (17.8)	15 (16.7)	17 (18.9)	0.70
Prior stroke, *n* (%)	14 (7.8)	9 (10)	5 (5.6)	0.27
Peripheral arterial disease, *n* (%)	7 (3.9)	5 (5.6)	2 (2.2)	0.44
Coronary heart disease, *n* (%)	35 (19.4)	18 (20)	17 (18.9)	0.85
Left ventricular ejection fraction (%)	55 ± 10	56 ± 10	54 ± 11	0.39
Body mass index (kg/m^2)	30.1 ± 5.4	29.8 ± 5.4	30.4 ± 5.5	0.51
History of gastrointestinal disease, *n* (%)	34 (18.9)	15 (16.7)	19 (21.1)	0.45
Gastro-oesophageal reflux disease, *n* (%)	12 (6.7)	3 (3.9)	9 (10)	0.03

Values are presented as mean ± standard deviation or *n* (%).
AF, atrial fibrillation; EHRA, European Heart Rhythm Association symptom scale; NYHA, New York Heart Association symptom scale; CHA_2DS_2-VASc, congestive heart failure, hypertension, age >75 years, diabetes mellitus, prior stroke, transient ischaemic attack or thromboembolism, vascular disease, age 65–74 years, sex category (female).

Table 2 Procedure data

Variables	All patients (*n*=180)	OPERA+ (*n*=90)	OPERA− (*n*=90)	*P*-value
Total procedure time (min)	132 ± 43	130 ± 42	134 ± 44	0.68
Total ablation time (min)	36 ± 20	35 ± 20	36 ± 20	0.66
Ablation at posterior wall, *n* (%)	176 (97.8)	87 (96.7)	89 (98.9)	0.62
Minimal energy at posterior wall (W)	23 ± 3	23 ± 4	24 ± 2	<0.001
Maximal energy at posterior wall (W)	26 ± 3	28 ± 4	25 ± 2	<0.001
Maximal energy at anterior wall (W)	36 ± 3	36 ± 3	36 ± 2	0.84
Maximum of oesophageal temperature	–	40.7 ± 1.4	–	not applicable
Oesophagus position next to left PV's/in the mid/next to right PV's (%)	–	47/25/28	–	not applicable
Additional ablation lines, *n* (%)	38 (21.1)	22 (24.4)	16 (17.8)	0.27
Count of prior ablation procedure[a]	1.3 ± 0.6	1.3 ± 0.6	1.3 ± 0.6	0.79
Total complication rate, *n* (%)	13 (7.2)	5 (5.6)	8 (8.9)	0.39
Arteriovenous fistula/pseudoaneurysm/stroke	3/8/2	1/2/2	2/6/0	0.23

Values are presented as mean ± standard deviation or *n* (%).
[a]Study procedure included.

Table 3 Incidence of post-interventional oesophageal lesions

Variables	All patients (*n* = 180)	OPERA+ (*n* = 90)	OPERA− (*n* = 90)	*P*-value
Oesophageal lesions in total, *n* (%)	18 (10)	10 (11.1)	8 (8.9)	0.62
EDEL 1, *n* (%)	13 (7.2)	6 (6.7)	7 (7.8)	0.39
EDEL 2, *n* (%)	5 (2.8)	4 (4.4)	1 (1.1)	0.39
EDEL size (mm)	5.2 ± 2.3	5.7 ± 2.6	4.5 ± 1.7	0.38
Time between ablation and EGD (days)	1.3 ± 0.8	1.3 ± 0.7	1.3 ± 0.9	0.99

Values are presented as mean ± standard deviation or *n* (%).
EDEL, endoscopically detected oesophageal lesions; EDEL 1, erythema and erosion ≤5 mm; EDEL 2, lesion or ulcers >5 mm with or without perforation; EGD, oesophagogastroduodenoscopy.

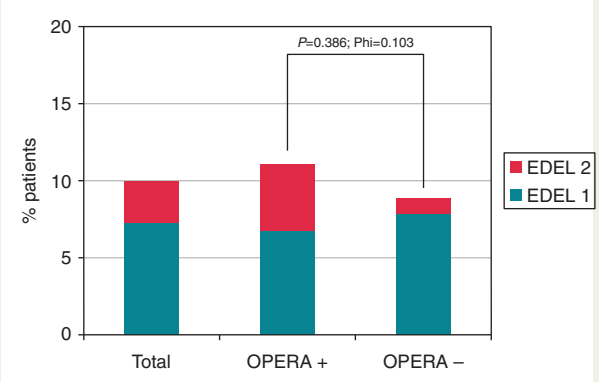

Figure 2 Occurrence of endoscopically detected oesophageal lesions and its distribution between the groups. EDEL 1, erythema and erosion ≤5 mm; EDEL 2, lesion or ulcers >5 mm with or without perforation.

between the groups (6/4 vs. 7/1, $P = 0.39$) (*Figure 2*). The average oesophageal EDEL size was equal [5.7 ± 2.6 vs. 4.5 ± 1.7 mm ($P = 0.38$)]. The peak temperature measured in OPERA+ was 40.7 ± 1.4°C. Endoscopically diagnosed oesophageal lesions were also seen in cases when the peak temperature was <39°C. No correlation was found between the maximum intraluminal temperature and the probability of oesophageal damage in EGD (*Figure 3*).

With regard to logistic regressions analysis, none of the tested patient characteristics or procedural parameters was predictive for developing EDEL (*Table 4*).

Oesophageal lesions during follow-up

During follow-up, no patient died. One patient in OPERA− received a prophylactic endovac therapy for a severe oesophageal ulceration. Basically, endovac therapy is a minimally invasive method for the treatment of anastomotic leakage. The gastrointestinal cavum can be drained by a vacuum sponge and supports the granulation process for healing. All patients were treated with 40 mg Pantoprazol per day for 4 weeks.

Secondary endpoint—atrial fibrillation recurrence

Cumulative AF recurrence after 6 (3–13) months was 28/87 (32%) vs. 34/88 (39%), $P = 0.541$ (*Figure 4*). In the OPERA+ group, five

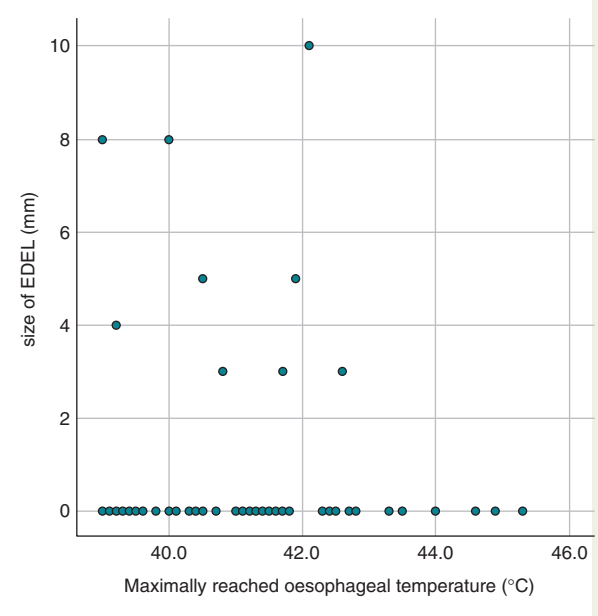

Figure 3 Correlation between the maximum intraluminal temperature and the lesion diameter of oesophageal damage in EGD. One patient had an EDEL without temperature rise above 39.0°C. EDEL, endoscopically detected oesophageal lesion; EGD, oesophagogastroduodenoscopy.

RFA for AF with and without the use of an intraoesophageal temperature probe. The main finding of this study is that intraoesophageal temperature monitoring using the SensiTherm™ probe does not affect the incidence of EDEL. Empiric reduction of the ablation energy did not result in impaired efficacy of the procedure with identical success rates in both groups.

Oesophageal temperature probes

Recently, two retrospective studies comparing the occurrence of EDEL after RFA for AF with or without continuous oesophageal temperature measurement using the SensiTherm™ probe revealed controversial results.[10,11] Müller *et al.*[10] enrolled 80 patients and found a significantly higher incidence of EDEL in the temperature probe group (30% vs. 2.5%). Furthermore, the use of an oesophageal temperature probe was an independent predictor for the occurrence of EDEL. The hypothesis is that non-insulated metallic components of the temperature probe attracting electric current can lead to heat transfer to the oesophagus.[12] On the other hand, Kiuchi *et al.*[11] reported a significantly lower incidence of EDEL in the temperature probe group in a total of 160 patients (0% vs. 7.5%). The discrepancy of these results underlines the need for randomized studies.

Table 4 Univariable logistic regression analysis identifying predictors for the presence of EDEL after ablation of AF

Variables	Odds ratio (95% CI)	*P*-value
Age (years)	1.032 (0.977–1.090)	0.255
Male gender	1.028 (0.366–2.887)	0.958
Paroxysmal atrial fibrillation	1.026 (0.378–2.788)	0.959
Persistent atrial fibrillation	0.974 (0.359–2.646)	0.959
Left ventricular ejection fraction (%)	1.008 (0.960–1.059)	0.752
CHA_2DS_2Vasc score	1.186 (0.866–1.623)	0.288
Body mass index (kg/m^2)	0.996 (0.909–1.090)	0.925
Prior gastrointestinal disease	1.764 (0.583–5.336)	0.341
Count of ablation procedures performed	0.803 (0.315–2.041)	0.644
Procedure time (min)	0.997 (0.985–1.009)	0.634
Ablation time (min)	1 (0.999–1)	0.761
Minimal energy at posterior wall (W)	0.931 (0.803–1.080)	0.347
Maximal energy at posterior wall (W)	1.028 (0.892–1.186)	0.7
Energy at anterior wall (W)	0.910 (0.748–1.106)	0.343
Oesophagus position next to left PV's vs. in the mid/next to right PV's	1.105 (0.297–4.117)	0.881
Maximum of oesophageal temperature (°C)	1.010 (0.662–1.541)	0.963
Patients with oesophageal temperature ≥39°C	0.961 (0.568–1.625)	0.882
Time for placement of the temperature probe	0.99 (0.986–1.011)	0.832

AF, atrial fibrillation; CI, confidence interval; EDEL, endoscopically detected oesophageal lesions.

patients were on antiarrhythmic drugs after the 3-month blanking period and four patients in the OPERA− group (*P* = not significant).

Discussion

This is the first prospective randomized study comparing the incidence of oesophageal lesions as a surrogate for fistula formation after

Halbfass *et al.*[13] investigated S-shaped temperature probes with insulated thermocouples (S-Cath™). In that retrospective study, the incidence of EDEL between the groups with or without temperature monitoring was equal (7.5% vs. 10%). A comparison of multisensor vs. single sensor probes in a study with 20 patients found no significant difference in the occurrence of EDEL, although the multisensory probe provided a greater sensitivity of temperature rising.[14]

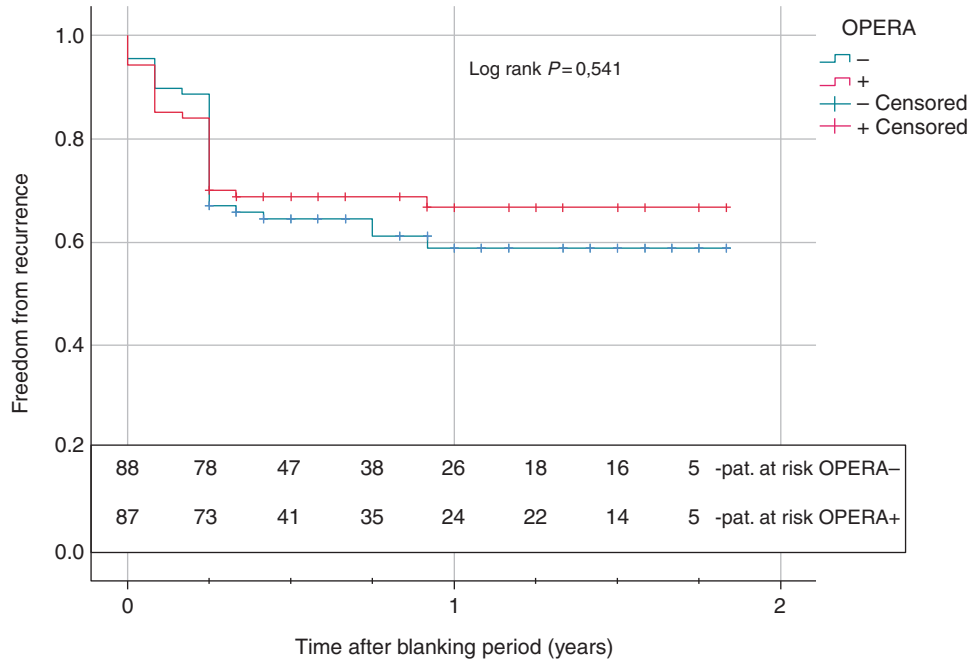

Figure 4 Recurrence of atrial fibrillation or left atrial flutter after a 3-month blanking period.

Intraoesophageal temperature level

In our study, the peak oesophageal temperature during ablation was not predictive for the development of EDEL. Despite repositioning the temperature probe according to the position of the ablation catheter, also in the absence of temperature rise above 39°C, EDEL was seen in EGD. A mismatch of the broad and flat oesophagus and the thin SensiTherm™ probe leading to incomplete temperature registration might be one reason. However, the precise mechanism of oesophageal injury is not fully understood. Besides the heating theory, ischaemic injury caused by thermal occlusion of end arterioles are also discussed.[15]

An observational study of 185 consecutive patients undergoing post-interventional EGD after RFA for AF reported an intraoesophageal temperature cut-off. In that study by Halm *et al.*[16] EDEL were only found in case of oesophageal temperature >41.0°C. Furthermore, the odds ratio of 1.36 for EDEL per degree of temperature increase supported the oesophageal heating theory as one possible mechanism for oesophageal injury.

John *et al.*[17] investigated the effect of oesophageal cooling by injection of ice-cold 0.9% saline into the upper oesophagus in 76 consecutive patients undergoing box isolation at the posterior wall. There was no statistical difference in the incidence of any oesophageal thermal lesions between the two groups (55.3% vs. 64.9%, $P = 0.40$), whereby there was a trend towards more severe oesophageal lesions in the non-cooled group (29% vs.13.5%, $P = 0.10$). However, the general incidence of EDEL seemed to be relatively high in this study. A meta-analysis with 494 patients using oesophageal cooling to protect the oesophagus from thermal injury during RFA reported identic findings with a reduction of the severity of the EDEL.[18]

Recently, a novel oesophageal infrared thermography system was investigated by Deneke *et al.*[19] in the HEAT-AF study. They could demonstrate a higher peak oesophageal temperature in patients with EDEL in comparison to patients without oesophageal lesions. Further studies are needed to establish appropriate cut-offs for guiding energy titration during AF ablation.

In the light of the results of our study, it remains questionable if small and thin electrode designs are capable of providing adequate coverage of the oesophageal width or if these electrodes will always just be able to reflect a small and frequently not representative portion of the oesophageal tissue being at risk close to the posterior wall of the left atrium.

Energy level at the posterior wall

The maximal energy level at the posterior wall in our study was 26 ± 3 W reflecting a usual ablation strategy over the last years.[20,21] Nevertheless, using high-power short-duration (HPSD) ablation became more and more popular in the current years. The basic idea of the HPSD approach is to destroy tissue by restrictive heating. The avoidance of distant conductive heating might be an advantage in reducing collateral injuries like oesophageal complications.

In a recently published meta-analysis including over 11 000 ablations used 45–50 W for 2–10 s at the posterior wall oesophageal temperature monitoring was performed in over 99%.[22] Post-interventional EGD as a routine was not performed. In this study, one patient experienced an AEF treated by surgery (<0.01%). The rate of AEF in a subgroup with lower power (35 W for 20 s) at the posterior wall was higher [3/2538 (0.12%)]. However, it must be pointed out, that the time period of this registry was >10 years including changes in ablation tools like contact force or mapping

systems. Furthermore, two of the three AEF occurred in a subset of patients without oesophageal temperature monitoring and a fluoless ablation procedure.

The QDOT-FAST Trial reported very HPSD lesions with 90 W for 4 s in a cohort of 52 patients.[23] The average power applied was 85.4 ± 6.7 W. Oesophageal temperature monitoring and post-interventional EGD were not performed. Interestingly, reconnection of the PVs occurred predominantly at the posterior wall.

Theoretically, HPSD creates more ideal lesion geometries especially for the posterior wall since the lesions seem to be more shallow which could protect neighbouring structures as the oesophagus.[24] Some recent studies have called into question the benefits of HPSD ablations in preventing oesophageal lesions. Barbhaiya *et al.*[25] found severe oesophageal temperature increases with HPSD of 50 W for 6 s, and caution that significant temperature increases will be undetected when lesions are >20 mm away from a temperature sensor. However, randomized studies focusing on oesophageal lesions under HPSD ablation are needed.

Clinical implications

What do these results mean for our daily practice? First, part of the EDEL occurred without measurable temperature increase. Second, we could not avoid any EDEL when using the probe. Maybe we underestimated the complexity of the anatomical structure oesophagus—but the clinical benefit using this device seems to be limited. Maybe approaches like oesophageal deviation or different lesion geometries (HPSD ablation) are more effective in protecting the oesophagus.

Limitations

(i) The most relevant limitation of this study is that we had to use EDEL as a surrogate parameter for oesophageal injury. Maybe other pathomechanisms exist for AEFs which are not starting with thermal damage. Using AEF as the primary endpoint would have resulted in study with several thousand patients given the very low incidence of this complication. (ii) Data about the lesion size index or ablation index at the posterior wall was not assessed in this study. (iii) A pre-ablation EGD was not performed. However, EDEL occurred in typical locations (30 cm ab oral) due to the anatomic relation to the left atrium. (iv) Due to different oesophagus motility, the applicability of our study results to patients undergoing AF ablation in general anaesthesia might be limited.

Conclusions

In this first prospective randomized study, intraoesophageal temperature monitoring using the SensiTherm™ probe for AF ablation does not affect the incidence of EDEL: the peak temperature measured by the thermoprobe seems not to correlate with the incidence of EDEL. Empiric energy reduction at the posterior wall did not affect the efficacy of the procedure.

Conflict of interest: P.S. is member of the Advisory Board of Abbott. All other authors have declared no conflict of interest.

Data availability

The data underlying this article will be shared on reasonable request to the corresponding author.

References

1. Calkins H, Hindricks G, Cappato R, Kim YH, Saad EB, Aguinaga L *et al.* 2017 HRS/EHRA/ECAS/APHRS/SOLAECE expert consensus statement on catheter and surgical ablation of atrial fibrillation. *Europace* 2018;**20**:e1–160.
2. Cappato R, Calkins H, Chen SA, Davies W, Iesaka Y, Kalman J *et al.* Prevalence and causes of fatal outcome in catheter ablation of atrial fibrillation. *J Am Coll Cardiol* 2009;**53**:1798–803.
3. Cheng EP, Liu CF, Yeo I, Markowitz SM, Thomas G, Ip JE *et al.* Risk of mortality following catheter ablation of atrial fibrillation. *J Am Coll Cardiol* 2019;**74**:2254–64.
4. Ghia KK, Chugh A, Good E, Pelosi F, Jongnarangsin K, Bogun F *et al.* A nationwide survey on the prevalence of atrioesophageal fistula after left atrial radiofrequency catheter ablation. *J Interv Card Electrophysiol* 2009;**24**:33–6.
5. Mohanty S, Santangeli P, Mohanty P, Di Biase L, Trivedi C, Bai R *et al.* Outcomes of atrioesophageal fistula following catheter ablation of atrial fibrillation treated with surgical repair versus esophageal stenting. *J Cardiovasc Electrophysiol* 2014;**25**:579–84.
6. Singh R, Landa EJ, Machado C. Atrial-esophageal fistula after catheter ablation: diagnosing and managing a rare complication of a common procedure. *Am J Case Rep* 2019;**20**:557–61.
7. Halbfass P, Pavlov B, Müller P, Nentwich K, Sonne K, Barth S *et al.* Progression from esophageal thermal asymptomatic lesion to perforation complicating atrial fibrillation ablation: a single-center registry. *Circ Arrhythm Electrophysiol* 2017;**10**:e005233.
8. Yarlagadda B, Deneke T, Turagam M, Dar T, Paleti S, Parikh V *et al.* Temporal relationships between esophageal injury type and progression in patients undergoing atrial fibrillation catheter ablation. *Heart Rhythm* 2019;**16**:204–12.
9. Rolf S, Kircher S, Arya A, Eitel C, Sommer P, Richter S *et al.* Tailored atrial substrate modification based on low-voltage areas in catheter ablation of atrial fibrillation. *Circ Arrhythm Electrophysiol* 2014;**7**:825–33.
10. Müller P, Dietrich JW, Halbfass P, Abouarab A, Fochler F, Szöllösi A *et al.* Higher incidence of esophageal lesions after ablation of atrial fibrillation related to the use of esophageal temperature probes. *Heart Rhythm* 2015;**12**:1464–9.
11. Kiuchi K, Okajima K, Shimane A, Kanda G, Yokoi K, Teranishi J *et al.* Impact of esophageal temperature monitoring guided atrial fibrillation ablation on preventing asymptomatic excessive transmural injury. *J Arrhythm* 2016;**32**:36–41.
12. Nguyen DT, Barham W, Zheng L, Dinegar S, Tzou WS, Sauer WH. Effect of radiofrequency energy delivery in proximity to metallic medical device components. *Heart Rhythm* 2015;**12**:2162–9.
13. Halbfass P, Müller P, Nentwich K, Krug J, Roos M, Hamm K *et al.* Incidence of asymptomatic oesophageal lesions after atrial fibrillation ablation using an oesophageal temperature probe with insulated thermocouples: a comparative controlled study. *Europace* 2017;**19**:385–91.
14. Tschabrunn CM, Silverstein J, Berzin T, Ellis E, Buxton AE, Josephson ME *et al.* Comparison between single- and multi-sensor oesophageal temperature probes during atrial fibrillation ablation: thermodynamic characteristics. *Europace* 2015;**17**:891–7.
15. Cummings JE, Schweikert RA, Saliba WI, Burkhardt JD, Brachmann J, Gunther J *et al.* Assessment of temperature, proximity, and course of the esophagus during radiofrequency ablation within the left atrium. *Circulation* 2005;**112**:459–64.
16. Halm U, Gaspar T, Zachäus M, Sack S, Arya A, Piorkowski C *et al.* Thermal esophageal lesions after radiofrequency catheter ablation of left atrial arrhythmias. *Am J Gastroenterol* 2010;**105**:551–6.
17. John J, Garg L, Orosey M, Desai T, Haines DE, Wong WS. The effect of esophageal cooling on esophageal injury during radiofrequency catheter ablation of atrial fibrillation. *J Interv Card Electrophysiol* 2020;**58**:43–50.
18. Leung LW, Gallagher MM, Santangeli P, Tschabrunn C, Guerra JM, Campos B *et al.* Esophageal cooling for protection during left atrial ablation: a systematic review and meta-analysis. *J Interv Card Electrophysiol* 2019;doi:10.1007/s10840-019-00661-5.
19. Deneke T, Nentwich K, Berkovitz A, Sonne K, Ene E, Pavlov B *et al.* High-resolution infrared thermal imaging of the esophagus during atrial fibrillation ablation as a predictor of endoscopically detected thermal lesions. *Circ Arrhythm Electrophysiol* 2018;**11**:e006681.
20. Reddy VY, Shah D, Kautzner J, Schmidt B, Saoudi N, Herrera C *et al.* The relationship between contact force and clinical outcome during radiofrequency catheter ablation of atrial fibrillation in the TOCCATA study. *Heart Rhythm* 2012;**9**:1789–95.
21. Reddy VY, Dukkipati SR, Neuzil P, Natale A, Albenque JP, Kautzner J *et al.* Randomized, controlled trial of the safety and effectiveness of a contact

force-sensing irrigated catheter for ablation of paroxysmal atrial fibrillation: results of the TactiCath Contact Force Ablation Catheter Study for Atrial Fibrillation (TOCCASTAR) study. *Circulation* 2015;**132**:907–15.
22. Winkle RA, Mohanty S, Patrawala RA, Mead RH, Kong MH, Engel G *et al.* Low complication rates using high power (45-50 W) for short duration for atrial fibrillation ablations. *Heart Rhythm* 2019;**16**:165–9.
23. Reddy VY, Grimaldi M, De Potter T, Vijgen JM, Bulava A, Duytschaever MF *et al.* Pulmonary vein isolation with very high power, short duration, temperature-controlled lesions: the QDOT-FAST trial. *JACC Clin Electrophysiol* 2019;**5**:778–86.
24. Bourier F, Duchateau J, Vlachos K, Lam A, Martin CA, Takigawa M *et al.* High-power short-duration versus standard radiofrequency ablation: insights on lesion metrics. *J Cardiovasc Electrophysiol* 2018;**29**:1570–5.
25. Barbhaiya CR, Kogan EV, Jankelson L, Knotts RJ, Spinelli M, Bernstein S *et al.* Esophageal temperature dynamics during high-power short-duration posterior wall ablation. *Heart Rhythm* 2020;**17**:721–7.