

The influence of ablation power reduction associated with oesophagus location on pulmonary vein isolation results in patients with paroxysmal atrial fibrillation: six-month follow-up

Piotr Buchta¹, Krzysztof Myrda¹, Michał Skrzypek², Adam Wojtaszczyk¹, Barbara Budzyn¹, Mariusz Gąsior¹

¹3rd Chair and Clinical Department of Cardiology, Silesian Centre for Heart Diseases, Zabrze, Poland

²Department of Biostatistics, School of Public Health in Bytom, Medical University of Silesia, Katowice, Poland

Abstract

Background: Catheter ablation of atrial fibrillation (AF) could be associated with a thermal oesophageal (EO) injury. To avoid this complication intraluminal EO temperature monitoring and ablation power reduction at the areas with excessive heating could be used. However, the reduced energy could limit the ablation lesion depth, without creation of lasting transmural scar and influence on long-term ablation results.

Aim: The primary goal was to evaluate the homogeneity of forced ablation power reduction due to excessive EO heating in different parts of the left atrium. The secondary goal was to assess the influence of power reduction in different EO locations on long-term AF recurrence.

Methods: We examined retrospectively 109 consecutive patients with symptomatic, medically refractory paroxysmal AF, who underwent pulmonary vein isolation using radiofrequency ablation. In 40.4% of the patients the EO course was central (group B) left atrium posterior wall, in 31.2% it was left sided (group A), and in 28.4% it was right sided (group C).

Results: The maximal measured temperature (41.0 ± 1.0 vs. 39.2 ± 1.5 vs. $40.6 \pm 0.7^\circ\text{C}$) and forced ablation power (15.9 ± 5.6 vs. 23.5 ± 6.1 vs. 17.4 ± 5.7 W) differed significantly according to the EO course (A, B, C, respectively). In six-month follow-up 76.15% of patients were free of arrhythmias. There was no statistically significant difference between groups (A-C) regarding the AF recurrence rate: 32.4% vs. 20.5% vs. 19.4% ($p = 0.37$).

Conclusions: The maximal intraluminal EO temperatures and the necessary level of power reduction during AF ablation are inhomogeneous in different parts of the left atrium, but they are not associated with different six-month follow-up results.

Key words: atrial fibrillation, catheter ablation, oesophageal injury

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INTRODUCTION

Atrial fibrillation (AF) is the most common cardiac arrhythmia. Catheter ablation is one of the standard treatment options. However, the oesophageal wall is adjacent to the ablated left atrium (LA), especially the posterior wall and the pulmonary vein (PV) ostia. Thus, ablation could be associated with transient oesophageal erythema and dangerous complications such as: creation of oesophageal ulcerations or aorto-oesophageal fistulas and perioesophageal nerve injury

(peri-ENI, e.g. acute pyloric spasms and gastric hypomotility). The incidence of oesophageal injury after radiofrequency (RF) ablation of AF has been reported to range from 2.9% to 50% (mostly as localised erythema), depending on the RF power settings and ablation strategies [1–13].

To avoid these complications, real-time intraluminal oesophageal temperature monitoring during ablation with the oesophageal temperature probes advanced to the level of the LA could be used. Additionally, the probe could be a marker

Address for correspondence:

Piotr Buchta, MD, PhD, 3rd Chair and Clinical Department of Cardiology, Silesian Centre for Heart Diseases, ul. M. Skłodowskiej-Curie 9, 41–800 Zabrze, Poland, e-mail: piotr.buchta@gmail.com

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of the oesophageal lumen location in relation to PV ostia [3, 12, 14–17]. It was reported that the use of temperature monitoring significantly reduces the oesophageal injury ratio (6% vs. 36%, $p < 0.006$) without any serious complications related to the probe [3, 12]. In a few small studies, it was suggested that the use of an oesophageal temperature probe per se appears to be a risk factor for the development of endoscopically detected oesophageal lesions, mostly due to acting with an “antenna effect” or causing mechanical injury [16, 18, 19]. However, creation of oesophageal lesions was associated with higher temperature achieved during ablation [18], and potential mechanical injury during pre-procedural transoesophageal echo could not be excluded. In the study on thermal effects of the probes, the temperature rise in the oesophageal wall and probe seemed to be primarily produced by thermal conduction, and the proposed notion of the “antenna effect” producing satellite oesophageal lesions during AF ablation was not evident if the metallic parts were not accidentally grounded [19].

Halm et al. [5] reported that oesophageal lesions occurred in patients with an oesophageal temperature $> 41^{\circ}\text{C}$, with the odds of an oesophageal lesion increased by a factor of 1.36 for every 1°C increase in oesophageal temperature. Thus, in the areas with excessive heating ablation, power should be reduced. However, the reduced energy could influence ablation lesion depth, without creation of lasting transmural scar. Incomplete block lines are associated with a higher recurrence rate of AF.

The primary goal of our analysis was to determinate whether the forced ablation power reduction due to excessive oesophageal heating is inhomogeneous in different parts of the LA. The secondary goal was to assess the influence of power reduction in different oesophageal locations on long-term AF recurrence in patients with paroxysmal AF.

METHODS

Patient population

We examined 109 consecutive patients with symptomatic, medically refractory paroxysmal AF. The patients underwent pulmonary vein isolation (PVI) using radiofrequency (RF) alternating current ablation. The subjects were retrospectively analysed.

Mapping and ablation procedure

Prior to the procedure, written informed consent was obtained from all patients. Transoesophageal echocardiography was performed one day before ablation to exclude thrombus and potential embolic material formation. The ablation was performed under general anaesthesia using sevoflurane and/or propofol infusion under an anaesthetist's supervision. A diagnostic quadripolar catheter was placed into the right ventricle apex (RVA) and decapolar catheter into the coronary sinus (CS). Single or double transseptal puncture was performed, and

unfractionated heparin was administered to maintain an activated clotting time of 300–350 s. The oesophageal temperature was measured during the ablation with an oesophageal temperature probe (SensiTherm, St. Jude Medical).

Mapping and ablation were performed using CARTO 3 (Biosense Webster, Diamond Bar, CA, USA) or Ensite Velocity system (St. Jude Medical Inc., St. Paul, MN, USA) after integration of a three-dimensional (3D) model of the anatomy of the LA and PVs obtained from pre-interventional computed tomography (CT). Prior to ablation, the LA anatomy was reconstructed with an ablation catheter (ThermoCool, Biosense Webster; IBI Therapy Cool Flex, St. Jude Medical, Inc.) and circular mapping catheter (Lasso, Biosense Webster, Diamond Bar, CA; Optima, St. Jude Medical Inc., St. Paul, MN, USA), and finally integrated with the CT image. Adjustment of images was achieved through landmarks and surface integration.

The initial RF generator setting consisted of an upper catheter tip temperature of 43°C , a maximal RF power of 35 W, and an irrigation flow rate of 30 mL/min. In RF, application was to the posterior wall, and the initial RF generator setting consisted of a maximal RF power of 20 W. The ablation line was performed at the atrial side of the PV antrum and depended primarily on the operator's decision. The RF energy was reduced if the oesophageal temperature rose when ablating the posterior wall of the LA. If the temperature rose to $> 39^{\circ}\text{C}$, the ablation was stopped immediately and the energy was further reduced.

Catheter navigation was supported in all patients with a steerable sheath (Agillis, St. Jude Medical). The procedural endpoint was considered as the electrophysiologically proven bidirectional block for the ablation lines with either a circular or mapping (ablation) catheter using a “pace-and-ablate” strategy, confirmed > 20 min after last RF application. After proving bidirectional block of the PV, test for inducibility with burst pacing from CS (with 300 ms, 250 ms, and 200 ms) was performed. If AF was induced, electrical cardioversion was performed. In cases of induced or previously documented typical atrial flutter, cavotricuspid isthmus (CTI) was additionally ablated.

All patients underwent LA voltage mapping with healthy tissue considered with > 0.5 mV, and border zone between 0.2 mV and 0.5 mV. If potential arrhythmogenic substrate was identified, additional lines were ablated, usually a “box” lesion and isolation of the posterior wall or septal lines.

The course of the esophagus was categorised vertically into three segments of the posterior wall (left-sided area with PVs ostia, mid-area, right-sided area with PVs ostia) (Fig. 1) and estimated during a procedure based on a fluoroscopic view of the temperature probe and ablation catheter localisation.

Anti-arrhythmic treatment was discontinued post-interventionally and beta-blockers were administered if there were no contraindications. Patients were administered oral



Figure 1. The location of the oesophagus close to left atrium posterior wall. Superimposition of a grid with vertical columns; A — left-sided area with pulmonary vein (PV) ostia; B — mid-area; C — right-sided area with PV ostia with oesophageal course distributions categorised into segments of left atrium posterior wall [%]

anticoagulants for more than six months. Patients were started on proton pump inhibitors and continued for four weeks. Oesophagoscopy was performed if the oesophageal temperature rose to $> 41^{\circ}\text{C}$.

Follow-up

Holter electrocardiography was performed after three and six months. The first three months were considered as a blanking period. If symptoms occurred outside the recording period, patients were requested to contact our centre or the referring physician to obtain electrocardiogram documentation. Episodes of AF and/or macro-re-entrant atrial tachycardia lasting > 30 s were considered as recurrences.

Statistical analysis

To describe patient baseline characteristics, we used both means with standard deviations and medians with interquartile ranges, and frequencies with percentages for categorical variables. When comparing between groups defined by the oesophagus course, we tested for differences using the Kruskal-Wallis test. Normal distribution of data was assessed by means of the Shapiro-Wilk test. All tests were interpreted as two-sided with a 5% level of significance. All statistical analyses were performed using SAS software (version 9.4 SAS Institute, Cary, North Carolina, USA).

RESULTS

The mean age of the study population was 58.2 ± 9.9 years, 63.3% were male, 48.6% patients had hypertension, 15.6% diabetes, 19.3% coronary artery disease, mean left ventricular ejection fraction was $53 \pm 4.4\%$, and body mass index was 29.7 ± 4.2 . The oesophageal locations differed significantly; in the majority of patients (40.4%) the oesophageal courses were central LA posterior wall, in 31.2% left sided, and 28.4% right sided (Fig. 1).

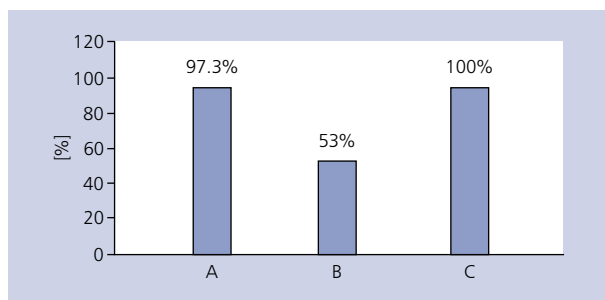


Figure 2. Prevalence of significant oesophageal temperature increase defined as $> 39^{\circ}\text{C}$ in different oesophageal courses; A — left-sided area with pulmonary vein (PV) ostia; B — mid-area; C — right-sided area with PV ostia [%]

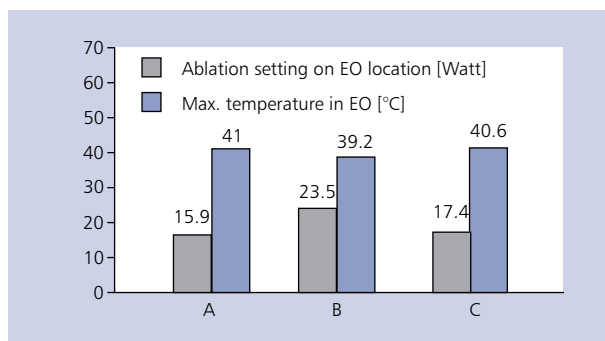


Figure 3. Power setting and maximal temperature on oesophagus (EO) according to course in relation to left atrium posterior wall; A — left-sided area with pulmonary vein (PV) ostia; B — mid-area; C — right-sided area with PV ostia

The median value of the maximum oesophageal temperature was 40.21°C (min. 37.0°C ; max. 42.5°C , LQ 39.55°C ; UQ 41.25°C). In the whole population, significant temperature increase was associated with forced reduction of the ablation power to a median value of 20 W (LQ 15 W; UQ 25 W). In four patients burst pacing after PVI induced arrhythmias with the need for additional ablation lines. In eight patients, substrate modification was performed based on the finding of low-voltage and scar areas in voltage-mapping. In all patients with right-sided oesophageal course the temperature rose to more than 39°C despite initial ablation power reduction; the lowest ratio of significant temperature increase was noted in the middle-course of the oesophagus; in the whole population a temperature $> 39^{\circ}\text{C}$ was noticed in 80.56% (Fig. 2). The maximal observed oesophageal temperature differed significant between groups: 41.0 ± 1.0 vs. 9.2 ± 1.5 vs. $40.6 \pm 0.7^{\circ}\text{C}$, respectively ($p < 0.01$). The mean power settings on oesophagus locations (A, B, C, respectively) were: 15.9 ± 5.6 vs. 23.5 ± 6.1 vs. 17.4 ± 5.7 W (Fig. 3) and differed significantly between central and sided locations ($p < 0.01$ for A vs. B and

B vs. C), without significant difference between sided locations ($p = 0.66$ for A vs. C). PV variants occurred: left common ostium in 18.6%; three right PV ostia in 11.6%; four and more right PVs in 12.8% of patients. The rest of the patients had two right- and two left-sided PVs. However, we did not observe any correlation between the type of PV ostia and measured oesophageal temperatures ($p = \text{NS}$).

Ablation was performed by two experienced electrophysiologists. Complete PVI was achieved in all patients. The mean fluoroscopy time was 12.2 ± 7.3 min with a mean 580.5 ± 378.5 mGy exposure dose. The median RF energy application was time 2880 s. In 12% of patients, temperature rose to $> 41^\circ\text{C}$, and oesophagoscopy was performed identifying local erythemas. No serious iatrogenic oesophageal injuries were not found in any of the patients studied.

In six-month follow-up 76.15% patients were free of arrhythmias. There was no difference between groups (A, B, C) regarding the AF recurrence rate: 32.4% vs. 20.5% vs. 19.4% ($p = 0.37$). 50% of patients with arrhythmia inducible after PVI had AF episodes in follow-up, in contrast to the rest of the patients (22.9%), but the difference was not statistically significant ($p = 0.24$).

DISCUSSION

In our study, we retrospectively analysed 109 consecutive patients with symptomatic, medically refractory paroxysmal AF, who underwent PVI using RF ablation. The ablation procedural aspects and the six-month outcomes were compared according to the oesophageal course in relation to the LA posterior wall.

In the cohort, the majority of patients had the central course of oesophagus in relation to LA. These findings are in line with relations observed in the previous studies of Bunch et al. [20] and Kottkamp et al. [21] but are in contrast to the studies of Kiuchi et al. [12] with the majority of patients with left-sided oesophageal courses in relation to the LA posterior wall [17]. Unexpectedly, despite initial power reduction and mostly central oesophageal location, we noticed a significant temperature increase to $> 39^\circ\text{C}$ in 80% of patients. This finding is comparable with previously published data [17] and emphasises the relevance of caution in LA posterior ablation with support of oesophageal temperature monitoring in all patients. The explanation for this further temperature increase could be based on the observation that the oesophageal temperature gradually increases by $1\text{--}3^\circ\text{C}$ within 10–20 s even after cessation of RF energy application [14]. Hence, to prevent a temperature increase to values related with higher risk of oesophageal ulceration [5] we stopped the applications at 39°C . Unfortunately, even by energy reduction to ≤ 10 W in 8% of patients one could expect significant temperature increase [3].

Not surprisingly, as the majority of the patients in our cohort underwent only PVI, we observed significantly higher temperature at the side areas of LA resulting in higher forced power reduction. The presence of temperature increase in

all patients with right-sided oesophageal location could be associated with the wall thinness in this area. In particular, the lower parts of the LA are at increased risk because it was shown that the distances between oesophagus and LA posterior wall are shorter behind the inferior than the superior parts of the LA, where the posterior LA is mostly in contact with the oesophagus [17, 21].

Di Biase et al. [22] reported an increased rate of PV reconnection in patients ablated with lower power settings. This procedure failure may be the result of poor electrode–tissue contact, insufficient power delivery, or excessive convective cooling [23]. In our study, we used steerable Agilis sheaths, which may strengthen the electrode-to-tissue contact, with creation of deeper lesions but with potentially higher risk of oesophageal injury. However, Halm et al. [5] observed no oesophageal lesions if the maximal intraluminal oesophageal temperature in patients ablated with steerable sheath technology was kept below 41.8°C . Thus, with respect to the taken temperature cut-off value for RF cessation, ablation with an Agilis sheath, especially with constant force measurement, could be considered as safe and without higher risk of injury [7].

The reduction of ablation power, with tissue heating to sub-lethal temperatures, can result in reversible loss of myocardial electrical activity. The return of PV conduction is associated with histopathological evidence of non-transmural lesions along the ablation line [23]. Therefore, we expected higher recurrence of AF in patients ablated with reduced energy, forced by significant oesophagus intraluminal temperature increase. However, we did not find statistically important differences in follow-up outcomes between defined oesophageal locations. The relatively high recurrence rate in the left-sided course could be associated with the thicker atrial wall in this location, but one cannot exclude other re-conduction sites not related to the posterior wall of the LA because they are probably distributed equally around the PV circular lesion. This follow-up data are consistent with a previously published study, which reported that catheter ablation using oesophageal temperature monitoring and active ablation power settings changes may reduce the incidence of oesophageal injury but without increasing the incidence of AF recurrence [12].

Limitations of the study

The study has several important limitations. First, the sample size was relatively small. Second, the analysis was performed retrospectively in a non-randomised, single-centre study. However, the uniform AF ablation protocol is an advantage. Third, oesophageal endoscopy both before and after AF ablation could not be performed. Therefore, we could not confirm whether the observed oesophageal erythema was caused by transoesophageal echocardiography before AF ablation or RF energy application. Finally, in some cases the oesophageal location could be underestimated, especially in patients with a hiatal hernia.

CONCLUSIONS

The maximal intraluminal oesophageal temperatures and the necessary level of power reduction during AF ablation are inhomogeneous in different parts of the LA, but they are not associated with different six-month follow-up results.

Conflict of interest: none declared

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Wpływ położenia przetyku względem tylnej ściany lewego przedsionka na wyniki izolacji żył płucnych u chorych z napadowym migotaniem przedsionków: obserwacja 6-miesięczna

Piotr Buchta¹, Krzysztof Myrda¹, Michał Skrzypek², Adam Wojtaszczyk¹, Barbara Budzyn¹, Mariusz Gąsior¹

¹III Katedra i Oddział Kliniczny Kardiologii, Śląskie Centrum Chorób Serca, Zabrze

²Katedra Biostatystyki Wydział Zdrowia Publicznego w Bytomiu, Śląski Uniwersytet Medyczny, Katowice

Streszczenie

Wstęp: Przeszkórna ablacja migotania przedsionków (AF) może się wiązać z powikłaniem pod postacią termicznego uszkodzenia przetyku. Ryzyko jego wystąpienia może zostać zmniejszone poprzez monitorowanie temperatury w przetyku, z redukcją mocy ablacji przy jej nadmiernym wzroście. Jednak zmniejszenie mocy może potencjalnie wiązać się z mniejszą penetracją energii, brakiem trwałej pełnościenności zmiany i gorszym efektem odległym zabiegu.

Cel: Pierwszorzędowym celem badania była ocena, czy stopień koniecznej redukcji energii jest jednorodny w różnych obszarach lewego przedsionka. Drugorzędowym celem była ocena wpływu redukcji energii ablacji na częstość nawrotów AF w obserwacji odległej.

Metody: Analizie retrospektywnej poddano 109 kolejnych pacjentów z objawowym, opornym na farmakoterapię napadowym AF, u których wykonano izolację żył płucnych prądem o wysokiej częstotliwości. U 40,4% chorych przetyk przebiegał w obszarze pośrodkowym tylnej ściany lewego przedsionka (grupa B), u 31,2% lewostronnie (grupa A), a u 28,4% prawostronnie (grupa C).

Wyniki: Maksymalna zmierzona temperatura ($41,0 \pm 1,0$ vs. $39,2 \pm 1,5$ vs. $40,6 \pm 0,7^\circ\text{C}$) oraz zredukowana moc ablacji ($15,9 \pm 5,6$ vs. $23,5 \pm 6,1$ vs. $17,4 \pm 5,7$ W) różniły się istotnie między poszczególnymi położeniami przetyku (kolejno A, B, C). W obserwacji 6-miesięcznej u 76,15% chorych nie zanotowano żadnej arytmii. Częstość nawrotów AF nie różniła się istotnie statystycznie między grupami (A, B, C): 32,4% vs. 20,5% vs. 19,4% ($p = 0,37$).

Wnioski: Zarówno maksymalna temperatura wewnątrz przetyku, jak i konieczny stopień redukcji mocy ablacji AF nie są jednorodne w różnych obszarach lewego przedsionka, nie wpływają jednak na wynik zabiegu w obserwacji 6-miesięcznej.

Słowa kluczowe: migotanie przedsionków, ablacja przezskórna, uszkodzenia przetyku

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Adres do korespondencji:

dr n. med. Piotr Buchta, III Katedra i Oddział Kliniczny Kardiologii, Śląskie Centrum Chorób Serca, ul. M. Skłodowskiej-Curie 9, 41–800 Zabrze,
e-mail: piotr.buchta@gmail.com

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