

Evaluation of the defibrillation threshold in atrial fibrillation by transoesophageal cardioversion using a biphasic impulse

Fryderyk Prochaczek¹, Hanna Winiarska¹, Piotr Kończakowski¹, Renata Pyka¹, Damian Dziuk¹, Jerzy Gałęcka²

¹Chair and i Division of Cardiology, Medical Academy of Silesia, Tychy, Poland

²Instytut Techniki i Aparatury Medycznej ITAM, Zabrze, Poland

Abstract

Introduction: Recurrent atrial fibrillation (AF) in the setting of haemodynamic disturbances requires frequently repeated cardioversions, which is associated with the risk of myocardial damage. It is thus necessary to identify methods which can minimise the cardioverter impulse energy.

Aim: To define the defibrillation threshold in recent-onset AF using a biphasic impulse, following an infusion of magnesium, potassium, and amiodarone.

Methods: Transoesophageal cardioversion was performed in 32 patients with AF lasting ≤ 48 hours, in whom prior administration of 40 mEq K⁺, 4.0 g MgSO₄ and 300 mg amiodarone did not restore sinus rhythm. Cardioversion was performed under short intravenous anaesthesia using a biphasic impulse travelling from a multi-annular oesophageal electrode to two electrodes on the anterior chest wall. The initial energy was set to 1 J, which was subsequently increased according to the following protocol: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50 and 70 J.

Results: Electrical cardioversion following the administration of electrolytes and amiodarone restored sinus rhythm in all the patients (100% efficacy). The mean defibrillation threshold was 12.9 \pm 14.3 J, with a minimal effective energy of 1 J and a maximum effective energy of 70 J. The defibrillation threshold was in the range from 1 to 10 J in 75% of the patients. The mean cumulative energy transferred between electrodes during evaluation of the defibrillation threshold was 39.7 J (SD, 38.8).

Conclusions: Transoesophageal cardioversion using a low-energy (mean, 12.9 J; range, 1–70 J) biphasic impulse, following the intravenous administration of potassium chloride and amiodarone, was 100% effective in restoring sinus rhythm in AF.

Key words: atrial fibrillation, transoesophageal cardioversion, defibrillation threshold in atrial fibrillation

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Introduction

The clinician often has to deal with cases of atrial fibrillation (AF) lasting several hours, which is accompanied by a moderate haemodynamic compromise, often leading to weakness, fatigue, vertigo or chest pain. The physician's therapeutic decision must be made quickly, not only due to the clinical symptoms associated with arrhythmia, but also because of the risk of thromboembolic complications, which arises if arrhythmia remains untreated for more than 48 hours.

An intravenous infusion of potassium chloride and magnesium sulphate may restore sinus rhythm; if it does not, however, the physician must decide whether to use pharmacological or electrical cardioversion. In order to safely perform pharmacological cardioversion, it is necessary to assess cardiac function, which may be difficult to accomplish on or shortly after admission. Drugs which may be used even in symptomatic heart failure include amiodarone, dofetilide and ibutilide [1-4]. Amiodarone has an especially favourable safety profile in short-term treatment [2]. It can also be administered

Address for correspondence:

Fryderyk Prochaczek, Katedra i Oddział Kliniczny Kardiologii ŚAM, Wojewódzki Szpital Specjalistyczny nr 1, ul. Edukacji 102, 43-100 Tychy, Poland, tel.: +48 32 325 43 18, fax: +48 32 325 42 84, e-mail: fred@itam.zabrze.pl

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to hypertensive patients with myocardial hypertrophy [5]. Dofetilide and ibutilide are unavailable in Poland for pharmacological cardioversion in AF, as these drugs have not been registered for use.

Should a pharmacological attempt to restore sinus rhythm fail, the physician's options are limited to transthoracic or transoesophageal electrical cardioversion. The initial energy commonly used in transthoracic cardioversion in AF is 200 J [6, 7]. The electrical field thus generated encompasses not only the atria, but also the ventricular myocardium. It is possible to decrease the energy required for successful defibrillation by using a biphasic impulse, especially during transoesophageal cardioversion, which allows a further energy reduction [8-10]. This reduction in transoesophageal cardioversion is possible due to the fact that the electric field generated in the atria is more homogeneous, as it bypasses the other structures of the heart – it is contained between the active electrode, posterior to the left atrium (i.e. in the oesophagus), and the surface electrodes on the anterior chest wall [9]. The current guidelines for transthoracic cardioversion allow the use of biphasic impulses; they do not, however, specify the energy levels at which defibrillation should be initiated [4]. There are, similarly, no such guidelines for low-energy transoesophageal cardioversion.

The goal of this study was to determine the defibrillation threshold (DFT) in recent-onset AF by transoesophageal cardioversion using biphasic impulses. An additional goal was to determine the energy level which was effective after a single impulse in at least 75% of patients.

Methods

Patients

Patients with an identifiable onset of arrhythmia within 40 hours were qualified for the evaluation of DFT. Patients with haemodynamic compromise and/or a heart rate above 130 beats/minute were initially included in the study. The initial therapy comprised an intravenous administration of 40 mEq potassium chloride and 4.0 g magnesium sulphate within 3 hours, and, if sinus rhythm was not restored, an intravenous infusion of 300 mg amiodarone over 1 hour. Thirty two patients, in whom sinus rhythm was not restored despite treatment and persistent haemodynamic disturbances and accelerated heart rate indicated the need for electric cardioversion, were ultimately included in the study. The study population consisted of 16 male and 16 female patients with a mean age of 61.6 years (35–79). The clinical characteristics of the study population are presented in Table I. Written consent for the evaluation of DFT was acquired from the Bioethics Committee of the Medical University of Silesia (NN-043030/94).

Transoesophageal cardioversion

Transoesophageal cardioversion was performed in patients after obtaining their written consent. All subjects received an intravenous bolus of 5000 IU heparin prior to cardioversion. Echocardiography was performed in 30/33 patients, in order to rule out the presence of thrombi or echogenic blood.

According to the established protocol, impulses started at 1 J and were subsequently repeated at higher energy levels at 1-minute intervals until sinus rhythm was

Cardioversion threshold protocol in AF<48 h

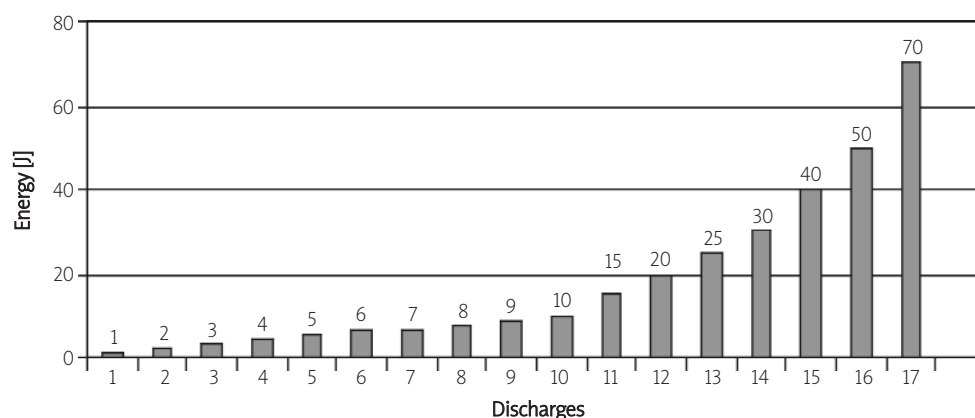


Figure 1. Study protocol for DFT determination

restored or a level of 70 J reached (Figure 1). The 1-minute intervals between impulses were intended to allow cell membranes focally injured by the previous impulse to return to the resting state [11].

The active electrode (anode) used in cardioversion was an oesophageal, five-ring defibrillation electrode (ITAM, Zabrze; ring radius, 6 mm; total surface area, 7 cm²). Self-adhesive, gel-filled passive electrodes (surface area, 50 cm²) were placed on both sides of the sternum in the 4th intercostal spaces, in electrocardiographic points C1 and C3. The oesophageal electrode was inserted to a depth determined by a modified Roth formula: depth=(height [cm])/5–2 [cm], after pretreatment with Dormicum (midazolam). The pretreatment reduced the period of anaesthesia required for insertion of the electrode and gave the investigator time to ensure that the electrode was correctly positioned (ECG recording from defibrillation electrodes or x-ray control). The oesophageal electrode and both passive electrodes were connected to an adapter (ITAM, Zabrze) possessing two adjustable metal surfaces, on which the defibrillator cables were placed. In order to ensure synchronisation of the impulse with the R wave, the defibrillator's ECG input was connected to three surface ECG electrodes. Following the placement of the oesophageal electrode at the appropriate depth for cardioversions, the patient was given short-acting anaesthetics (intravenous fentanyl and thiopental). Cardioversion was performed by biphasic impulse using a Lifepak 12 cardioverter-defibrillator.

The lowest energy level to restore sinus rhythm within 30 seconds of an impulse was considered as the DFT [12]. The cumulative amount of energy applied during all cardioversions was calculated for each patient. Following cardioversion, patients were monitored for dysphagia, as well as ST abnormalities, conduction disturbances and supraventricular or ventricular arrhythmias.

Table I. Clinical characteristics of study population

Underlying condition	Prevalence [number of pts.]
Arterial hypertension	19 (59.4 %)
Coronary heart disease	4 (12.5 %)
Valvular lesion	5 (15.6 %)
AF without apparent cause	3 (9.4 %)
Other	1 (3.1 %)

Table II. Number of patients with given thresholds in the study group (n=32)

EFFECTIVE ENERGY [J]	1	2	3	4	5	6	8	10	20	30	50	70
Number of patients	1	1	1	3	6	1	1	10	5	1	1	1

Statistical analysis

Statistical analysis was performed using the Shapiro-Wilk test, with the goal of determining the energy level required to restore sinus rhythm after a single impulse in 75% of subjects.

Results

Neither symptoms of peripheral venous irritation nor arrhythmia were noted during the administration of potassium or amiodarone. Transoesophageal cardioversion using a biphasic impulse restored sinus rhythm in all subjects; 23/32 returned to sinus rhythm after impulses of 1 to 10 J. Table II presents the distribution of successful cardioversions by energy level; Figure 2 depicts an example of an ECG recorded during cardioversion.

The mean DFT was found to be 12.9±14.3 J (Table III). This table also includes the mean cumulative energy during DFT determination in recent-onset AF. Two of 32 subjects complained of dysphagia after cardioversion; this symptom disappeared after one day of treatment

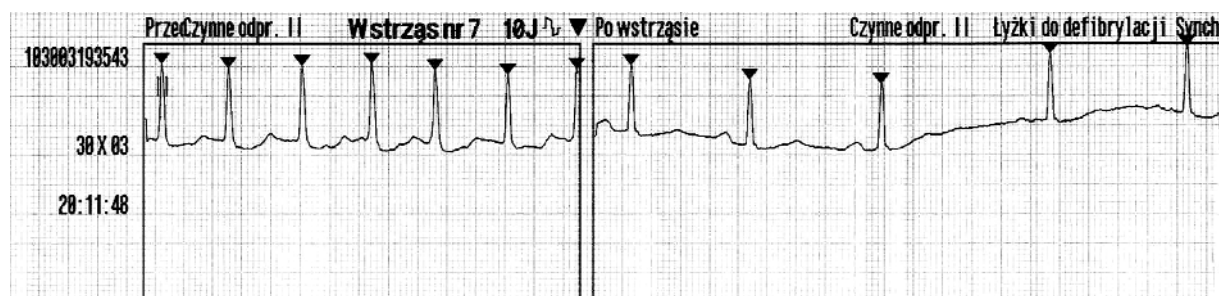


Figure 2. An example showing successful AF termination with 10 J cardioversion. Same energy was effective in 75% of pts. in whom first attempt was successful. ECG recording at 25 mm/s

Table III. Mean DFT and total energy values and associated side effects

N	Mean DFT [J]	Mean total energy [J]	ST elevation	Premature ventricular contractions	Dysphagia
32	12.9 (1–70) SD=14.3	39.7 (1–300) SD=38.8	0/32	0/32	2/32

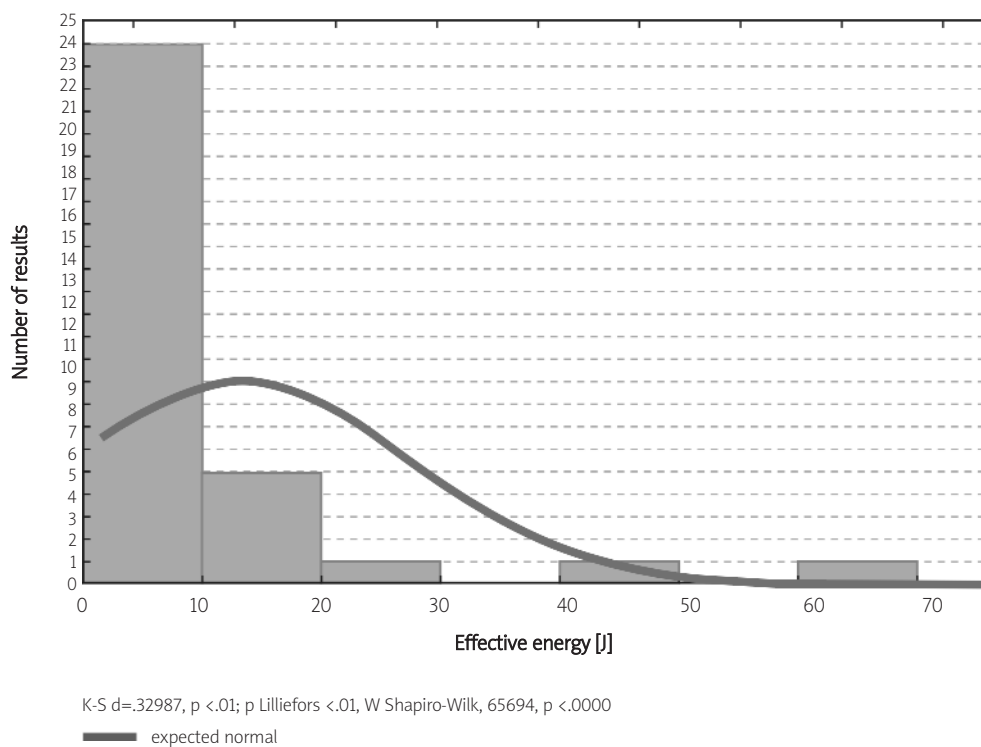
with an H₂-blocker and alkalinising agent. The cumulative amount of energy applied in these patients was 250 and 300 J, respectively. Statistical analysis revealed that an impulse energy of 10 J was sufficient to restore sinus rhythm after a single impulse in 75% of subjects (Figure 3). The mean cumulative energy applied in patients successfully defibrillated at 10 J was 7.2 J (1–55 J).

Discussion

The decision of whether and how to restore sinus rhythm in AF should be based on established therapeutic methods [4]. ESC guidelines name a few drugs that have been proven to be useful, effective and safe – of these, propafenone and amiodarone are available in Poland. Propafenone fulfils class I criteria in terms of utility and effectiveness, but only if it is possible to exclude left ventricular dysfunction [4]. If the functional state of the heart is unknown, only amiodarone, which may

also be administered in clinically symptomatic heart failure, can be used. Amiodarone is considered as class II indication with respect to utility and effectiveness at the highest level of evidence (A), and was thus administered to our patients.

Patients in whom pharmacological treatment is unsuccessful require electrical cardioversion – either transthoracic or transoesophageal. In our study, we used transoesophageal cardioversion and determined the AF DFT in patients in whom potassium, magnesium and amiodarone were ineffective. We demonstrated a high efficacy of transoesophageal cardioversion. The 100% success rate observed for transoesophageal cardioversion in AF using biphasic impulses up to 70 J may be a result of both the initial electrolyte stabilisation and lowering of DFT by the administration of amiodarone [4, 13]. Poleszak et al. reported an identical (100%) efficacy of transoesophageal cardioversion in AF lasting less than 48

**Figure 3.** Successful shocks at given energy levels in 32 study patients

hours, despite the fact that electrolytes and amiodarone were not administered prior to cardioversion and monophasic impulses were used [8].

The mean DFT in our study (12.9 J) was surprisingly low compared to the value (47.1 mA) reported by Poleszak et al. for the monophasic impulse [8]. This discrepancy may be a result of both the administration of amiodarone and the utilisation of a biphasic impulse. Experimental studies have demonstrated that drugs which decrease the difference between the resting membrane potential and threshold potential in excitable myocytes lower current density required for defibrillation. Amiodarone has been shown to lower the DFT by up to 20% [14]. Applying a biphasic impulse decreases the DFT in ventricular fibrillation by approximately half in comparison to a monophasic impulse (suppressed sinusoid) [15]. These data indicate that the low DFT observed in our study can be primarily attributed to the effect of the biphasic impulse.

Interestingly, while DFT values ranged from 1 to 70 J, high-energy impulses (30, 50 and 70 J) were only necessary in 3 subjects. In transthoracic cardioversion using a biphasic impulse these are often the initial values; when a monophasic impulse is used, cardioversion usually begins with an impulse of 200 J [4, 6, 7]. The main advantage of transoesophageal over transthoracic cardioversion is a result of the fact that the latter is very imprecise in directing the generated electrical field towards the atria. This is related to the highly variable electrical resistance of tissues located between the defibrillation electrodes [11]. The electrical field thus generated has a broad range and encompasses regions of the heart particularly susceptible to injury – the sinoatrial and atrioventricular nodes and ventricular myocardium. Due to this lack of precision, classical cardioversion can lead to post-defibrillation arrhythmias, sinus bradycardia, atrioventricular block and ST-segment elevation, none of which were observed in our patients defibrillated transoesophageally.

Most DFT values (22/32) fell in the range of 1 to 10 J. Such low DFT levels have been traditionally reserved for endocavitary cardioversion, in which the impulse is conducted between electrodes located in the right atrium and the coronary vein or in the right atrium and on the chest surface [12, 16, 17]. This comparison confirms the opinion that transoesophageal cardioversion using a biphasic impulse is a low-energy procedure [9, 18].

The most effective and ethical method of determining DFT is to evaluate it during an isolated

episode of AF, commencing with expected sub-threshold energy levels. Our study implemented such a protocol. The protocol, which called for the application of several consecutive impulses, must have, however, contributed to the low observed value of DFT, since each subsequent impulse decreases tissue resistance, and therefore increases the proportion of atrial cells simultaneously at a similar stage of their action potential, which, in turn, decreases the amount of energy required for defibrillation [15]. In our patients, successfully defibrillated at levels up to 10 J (mean cumulative energy, 7.2 J), this influence was probably limited, but noteworthy, due to the fact that it can have implications for the determination of the initial impulse energy in patients undergoing transoesophageal defibrillation.

The determination of AF DFT using our protocol was a time-consuming task for the physicians and subjected some patients to a large cumulative energy exposure. None of the 3 patients with respective cumulative energy levels of 220, 250 and 300 J presented any ECG abnormalities indicating post-defibrillation myocardial injury. This observation is consistent with the 2001 ESC guidelines which underline the wide safety margin between the energy levels required for effective cardioversion and levels which can significantly damage the myocardium [4, 19, 20].

There are, however, data indicating that myocardial injury is related to the amount of energy applied, which suggests that the heart should be protected from excessive amounts of energy [15, 21, 22]. The damage inflicted by an impulse is thought to correspond not only to the total amount of energy applied, but also to the frequency with which consecutive impulses are repeated [15]. Even the minimum effective impulse energy causes focal damage to cell membranes, which can be visualised by electron microscopy and disrupts the function of intercellular junctions. An excessive amount of energy, in turn, inhibits cellular respiration by damaging mitochondria, which leads to the activation of anaerobic metabolic pathways. This damage is especially evident in patients in whom cardioversion is frequently repeated.

Endoscopic studies indicate that transoesophageal cardioversion can damage the oesophageal mucosa [8, 23]. Two patients in our study, in whom large cumulative amounts of energy were applied, complained of heartburn, which did not pose a therapeutic problem, as it was eased by the administration of an alkalisant agent and a H₂-blocker. Poleszak et al. similarly observed dysphagia in 3 of 112 patients subjected to transoesophageal cardioversion of AF [8].

Our analyses indicate that, when our protocol is applied, a 75% success rate for cardioversion was achieved at an energy level of 10 J, which corresponds to a cumulative energy of 55 J. Can this efficacy be expected in everyday clinical practice, if impulses commence at 10 J? In order to answer this question, it is necessary to study a similar group of patients, applying a smaller number of impulses. Such an abridged protocol could include subsequent impulses at 20 and 70 J, which would limit the cumulative energy applied to the heart to a maximum of 100 J.

The observation that cardioversion was, in some cases, successful at very low energy levels (1–2 J) indicates that the suggested protocol should include an initial impulse of 1 J for the Zoll M Series defibrillator and 2 J for the Medtronic Lifepak 12. The initial impulse may itself be sufficient to restore sinus rhythm, and, in the worst case (i.e. defibrillation threshold 70 J), would increase the cumulative energy applied to the heart by only 2%.

It is necessary to formulate a set of convenient, effective and safe therapeutic recommendations for doctors on duty in Polish centres treating patients with AF. Low-energy transoesophageal cardioversion, which limits the negative impact on the heart, seems to be an attractive alternative to transthoracic cardioversion.

Conclusions

1. Transoesophageal electric cardioversion using a biphasic impulse, performed after the administration of electrolytes and amiodarone, was successful in 100% of recent-onset AF cases. An impulse of 10 J, with a cumulative energy of 55 J, was successful in 75% of patients.
2. The observed defibrillation thresholds in recent-onset AF by transoesophageal cardioversion using a biphasic impulse (mean 13 J) indicate that this method can be considered a low-energy therapy with limited adverse effects on the heart.

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Ocena progu defibrylacji migotania przedsionków impulsem dwufazowym przy użyciu kardiowersji przezprzewodowej

Fryderyk Prochaczek¹, Hanna Winiarska¹, Piotr Kończakowski¹, Renata Pyka¹, Damian Dziuk¹, Jerzy Gałęcka²

¹Katedra i Oddział Kliniczny Kardiologii, Medical Academy of Silesia, Tychy

²Instytut Techniki i Aparatury Medycznej ITAM, Zabrze

Streszczenie

Wstęp: Nawracające migotanie przedsionków (AF) przy obecnych zaburzeniach hemodynamicznych wymusza wielokrotne powtarzanie kardiowersji, co niesie z sobą ryzyko uszkodzenia mięśnia sercowego. Wskazane jest zatem poszukiwanie metod, które pozwalają na użycie możliwie najniższych energii impulsu kardiowertującego.

Cel: Określenie rzeczywistego progu defibrylacji krótkotrwałego AF impulsem dwufazowym, po przygotowaniu infuzją magnezu, potasu i amiodaronu.

Metodyka: Kardiowersję drogą przezprzewodową przeprowadzono u 32 pacjentów z AF \leq 48 godz., u których uprzednie dożylnie podanie 40 mEq K⁺, 4,0 g MgSO₄ oraz 300 mg amiodaronu nie przywróciło rytmu zatokowego. Kardiowersje wykonywano w krótkotrwałej narkozie dożylniej, wykorzystując impuls dwufazowy przepływający pomiędzy wielopierścieniową elektrodą przewodową a dwiema elektrodami umieszczonymi na powierzchni klatki piersiowej. Rozpoczynano od energii 1 J, zwiększając stopniowo zgodnie z protokołem: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50 i 70 J.

Wyniki: Po podaniu elektrolitów i wlewie amiodaronu kardiowersja elektryczna przywróciła rytm zatokowy u wszystkich badanych (skuteczność 100%). Średni próg defibrylacji wyniósł 12,9 \pm 14,3 J przy najniższej skutecznej energii – 1 J i najwyższej – 70 J. U 75% pacjentów próg defibrylacji mieścił się w przedziale od 1 do 10 J. Średnia energia przepływająca między elektrodami w czasie realizacji protokołu oznaczania progu defibrylacji wyniosła 39,7 J (SD=38,8).

Wniosek: Kardiowersja przezprzewodowa poprzedzona podaniem dożylnym wlewem chlorku potasu i podaniem amiodaronu pozwoliła uzyskać 100 % skuteczność umiarawiania AF przy zastosowaniu impulsu dwufazowego o małej energii wynoszącej średnio 12,9 J (1–70 J).

Słowa kluczowe: migotanie przedsionków, kardiowersja przezprzewodowa, próg defibrylacji migotania przedsionków

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Adres do korespondencji:

Fryderyk Prochaczek, Katedra i Oddział Kliniczny Kardiologii ŚIAM, Wojewódzki Szpital Specjalistyczny nr 1, ul. Edukacji 102, 43-100 Tychy, tel.: +48 32 325 43 18, faks: +48 32 325 42 84, e-mail: fred@itam.zabrze.pl

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