Taylor & Francis
Taylor & Francis Group

[Original article]

Pocket haematoma after cardiac electronic device implantation in patients receiving antiplatelet and anticoagulant treatment: a single-centre experience

Gültekin Gunhan DEMIR, MD; Gamze Babur GULER, MD; Ekrem GULER, MD; Hacı Murat GÜNEŞ, MD; Filiz KIZILIRMAK, MD; İbrahim Oğuz KARACA, MD; Mehmet Onur OMAYGENÇ, MD; Beytullah ÇAKAL, MD; Erkam OLGUN, MD; Umeyr SAVUR, MD; Ersın IBISOGLU, MD; Irfan BARUTÇU, MD; Fethi KILIÇASLAN, MD

Medipol University Medicine Faculty, Cardiology Department, Istanbul, Turkey.

Objective In modern cardiology practice, implantation of cardiac electronic devices in patients taking anticoagulant or antiplatelet therapy is a common clinical scenario. Bleeding complications are of particular concern in this patient population and pocket haematoma is one of the most frequent complications. We sought to determine the relationship between periprocedural antiplatelet/anticoagulant therapy and pocket haematoma formation in patients undergoing cardiac implantable electronic device (CIED) implantation.

Methods We conducted a retrospective study including 232 consecutive patients undergoing CIED implantation in the department of cardiology of the Medipol University Hospital. Patients were divided into six groups: clopidogrel group (n = 12), acetylsalicylic acid (ASA) group (n = 73), ASA + clopidogrel group (n = 29), warfarin group (n = 34), warfarin + ASA group (n = 21) and no antiplatelet-anticoagulant therapy group as the control group (n = 63). CIED implantations were stratified under four subtitles including implantable cardioverter/defibrillator (ICD), cardiac resynchronization therapy (CRT), permanent pacemaker and the last group as either device upgrade or generator replacement.

Results The mean age of the patients was 63 ± 14 years and 140 patients were male (60.3%). A pocket haematoma was documented in 6 of 232 patients (2.6%). None of the patients with pocket haematoma needed pocket exploration or blood transfusion. The type of the device did not have a significant effect on pocket haematoma incidence ($P = 0.250$). Univariate logistic regression showed that platelet level and ASA plus clopidogrel use were significantly associated with haematoma frequency after CIED implantations, respectively (OR: 0.977, CI 95% [0.958-0.996]; OR: 16.080, CI 95% [2.801-92.306]). Multivariate analysis revealed that dual antiplatelet treatment ($\beta = 3.016$, $P = 0.002$, OR: 2.410, 95% CI [3.042-136.943]) and baseline platelet level ($\beta = -0.027$, p:0.025, OR: 0.974, 95% CI [0.951-0.997]) were independent risk factors for pocket haematoma formation.

Conclusion Dual antiplatelet therapy and low platelet levels significantly increased the risk of pocket haematoma formation in patients undergoing CIED implantations.

Keywords *Pocket haematoma – implantable cardioverter defibrillator – cardiac resynchronization therapy – permanent pacemaker.*

INTRODUCTION

The number of patients undergoing cardiac electronic device implantation is increasing worldwide and many of these patients are already receiving some kind of antiplatelet or anticoagulant therapy. Despite technological advancements in device and pharmacological therapy, bleeding complications remain as a worrisome issue for the operator and the patient. Small vessel bleeding within the device pocket causes pocket haematoma. Most of the pocket haematomas are small and may be managed conservatively[1,3]. Clinically significant pocket haematoma are associated with prolonged hospital stay, increased risk of infection, interruption of antiplatelet or anticoagulant therapy and further surgery including revision or evacuation[3]. The incidence of pocket haematoma represents a wide range and results obtained from prior randomized

Address for correspondence:

Gültekin Günhan Demir, M.D.,
Dept. of Cardiology, Medipol University Hospital,
Birlik Mah. Bahçeler Cad. No:5 Esenler 34214 Istanbul, Turkey.
E-mail: ggdemir@gmail.com

Received 31 December 2015; revision accepted for publication 8 February 2016.

http://dx.doi.org/10.1080/00015385.2017.1281539

trials and meta-analysis conflict with recent real-life population-based studies which demonstrate lower rates of pocket haematoma in spite of increased overall complication rates[1,2]. Dual anti-platelet therapy and heparin bridging strategy are well-known predictors for pocket haematoma[3-5].

We aimed to investigate the frequency and predictors of pocket haematoma in patients undergoing cardiac implantable electronic device (CIED) implantations and taking antiplatelet/anticoagulant agents.

METHODS

Patient characteristics and study design

In this retrospective observational study, we enrolled 232 patients undergoing CIED implantation or replacement in our institution between November 2012 and July 2014. Device-related procedures were categorized in four subtitles including *de novo* implantable cardioverter/defibrillator (ICD), cardiac resynchronization therapy (CRT), permanent pace-maker implantations and another group covering generator replacement, device upgrade or lead revision. Exclusion criteria were defined as age less than 18 years, use of novel anticoagulant agents (dabigatran, rivaroxaban, apixaban etc.) or antiplatelet agents (prasugrel, ticagrelor), combination of warfarin and clopidogrel therapy, lead extraction procedure or procedures involving epicardial access.

We reviewed detailed medical records of each patient before and after device implantation including any follow-up visit after the index procedure. Data about complications were collected on review of all patient charts.

Patients were divided into six main groups as follows according to the treatment before the index procedure: clopidogrel group, acetylsalicylic acid (ASA) group, ASA + clopidogrel group, warfarin group, warfarin + ASA group. Patients not taking any form of antiplatelet or anticoagulant therapy were defined as the control group.

Based on results from previous studies and the meta-analysis, pocket haematomas were defined and classified as follows: minor haematomas as any haematoma that may cause local pain and could be managed conservatively with a sandbag and without requirement of blood transfusion or interruption of antiplatelet/anticoagulant therapy[1,5,6]. Clinically significant pocket haematoma was defined as any haematoma necessitating blood transfusion, interruption of antiplatelet/anticoagulant therapy, prolonged hospitalization and further pocket surgery including revision or evacuation. Prolonged hospitalization was defined as a delay in discharge or rehospitalization within 24 hours after the index procedure. Interruption of antiplatelet or anticoagulant therapy was defined as reversal or withholding of warfarin or implicated antiplatelet agent due to haematoma.

Ongoing clopidogrel therapy was not discontinued in any patients irrespective of concomitant ASA therapy at the time of surgery. In patients receiving warfarin treatment, the international normalized ratio (INR) on the day of surgery was expected to be less than 3.0 value.

All device-related procedures were performed with subclavian venous access to the right heart by one board-certified electrophysiologist in our institution. The study was approved by the local ethics committee.

Statistical analysis

Mean ± standard deviation (SD) and median, [range] were used for continuous variables, while percentages were used for categorical variables. Normal distribution was tested with the Kolmogorov-Smirnov test. Pearson's chi-square test was used to test the categorical variables. Univariate logistic regression analysis was used to explore the association between all factors and haematoma frequency. The variables with a significant association with haematoma frequency by univariate analysis were included into multivariate logistic regression (forward stepwise model) for further analysis. *P*-value of < 0.05 was considered significant for all tests. The Statistical Package for the Social Sciences (SPSS version 11.0, SPSS Inc., Chicago, IL, USA) was used.

RESULTS

A total of 232 patients were enrolled in the study. The baseline patient and procedural characteristics of the study population are shown in table 1. The mean age of the patients was 63 ± 14 years and 140 of the patients (60.3%) were male. Two hundred and thirty-two procedures consisted of 90 ICD implantations (38.8%), 54 CRT implantations (23.3%), 61 permanent pace-maker implantations (26.3%) and a sum of 27 procedures including generator replacement, device upgrade or lead revision (11.6%).

Of 232 patients, 12 patients (5.2%) were on clopidogrel treatment, 73 patients on ASA treatment (31.5%), 29 patients on dual antiplatelet (ASA + clopidogrel) treatment (12.5%), 21 patients on warfarin plus ASA treatment (9.1%) and 34 patients on warfarin treatment (14.5%). Sixty-three patients (27.1%) in the control group were not taking any form of anticoagulant or antiplatelet therapy.

Pocket haematoma was documented in 6 of 232 patients (2.6%). Only one of 6 pocket haematomas was a clinically significant pocket haematoma which required prolonged hospital stay and interruption of dual antiplatelet therapy while the remaining five minor pocket haematomas were managed conservatively. None of the pocket haematomas required blood transfusion

Table 1 The baseline procedural characteristics of patients

Age (years)	63 ± 14
Gender (male %)	60.3% (140)
Ejection fraction (%)	35 [70-15]
Creatinine (mg/dl)	1 [6.4-0.5]
Platelets	221 ± 65
INR	2.02 ± 0.57
Hypertension (%)	72.8% (169)
Diabetes mellitus (%)	28.4% (66)
ICD implantation (%)	38.8% (90)
CRT implantation (%)	23.3% (54)
Pacemaker implantation(%)	26.3% (61)
Device upgrade (%)	11.6% (27)
Drug therapy	
Clopidogrel (%)	5.2% (12)
ASA (%)	31.5% (73)
Clopidogrel + ASA (%)	12.5% (29)
Warfarin + ASA (%)	9.1% (21)
Warfarin (%)	14.7% (34)
Control (%)	27.2% (63)

ASA: acetylsalicylic acid, CRT: cardiac resynchronization therapy, ICD: implantable cardioverter/defibrillator, INR: international normalized ratio.

Table 2 Univariate regression analysis of haematoma frequency

	OR	CI 95%
Age (years)	1.03	0.966-1.104
Gender (%)	0.65	0.128-3.291
Ejection fraction (%)	0.98	0.925-1.030
Creatinine (mg/dL)	1.7	0.941-3.066
Platelets	*0.98*	*0.958-0.996*
INR	2.42	0.146-40.060
CRT implantation (%)	1.67	0.298-9.394
Pacemaker implantation (%)	2.9	0.569-14.753
Device upgrade (%)	1.47	0.166-13.097
Clopidogrel use (%)	3.91	0.420-36.386
Clopidogrel + ASA use (%)	*16.08*	*2.801-92.306*
Warfarin + ASA use (%)	2.06	0.229-18.509

ASA: acetylsalicylic acid, CRT: cardiac resynchronization therapy.

or further surgery. Pocket haematomas developed in 4 patients on dual antiplatelet therapy (66.7%) whereas the remaining two haematomas were in the clopidogrel group and warfarin plus ASA group, respectively (16.7% vs 16.7%). The type of the device or procedure was not associated with haematoma formation ($P = 0.250$). Similarly, age and gender difference was not associated with pocket haematoma formation.

Univariate logistic regression showed that baseline platelet level, ASA + clopidogrel use were significantly associated with pocket haematoma frequency after CIED implantations, respectively, as shown in table 2 (OR: 0.977, CI 95% [0.958-0.996]; OR: 16.080, CI 95% [2.801-92.306]). A multivariate logistic regression analysis was performed to determine the independent risk factors of pocket haematoma. Among these, an independent association with disease was found for ASA + clopidogrel use ($\beta = 3.016$, $P = 0.002$, OR: 20.410, 95% CI [3.042-136.943]) and platelet level ($\beta = -0.027$, $P = 0.025$, OR: 0.974, 95% CI [0.951-0.997]) (Hosmer and Lemeshow test; $P = 0.809$, Nagelkerke's R square: 0.372) (table 3).

DISCUSSION

Expanding indications for cardiac device therapy and increased numbers of patients receiving antiplatelet/anticoagulant agents underscore better management and definition of complications in this common patient population. Pocket haematoma may lead to serious morbidity and occasionally mortality via prolonged hospitalization, interruption of anticoagulant/antiplatelet therapy, infection, further surgery including revision, evacuation or extraction[6].

Pocket haematoma definitions used in previous studies represent prominent diversity therefore the range for pocket haematoma incidence is wide (2.9-9.5%) unlike other complications of cardiac rhythm device surgery[7].When these factors are taken into account, we preferred to use the definitions utilized in

Table 3 Multivariate analysis for independent predictors of pocket haematoma frequency after cardiac device implantation

	Beta	*P*	OR	95% CI
ASA + clopidogrel	3.016	0.002	20.41	3.042-136.943
Platelet levels	–0.027	0.025	0.97	0.951-0.997

Hosmer and Lemeshow test; $P = 0.809$; Nagelkerke's R square: 0.372 model significance.
$P < 0.001$.

the meta-analysis[1] for pocket haematoma and categorized into two subheadings namely "minor pocket haematoma" and "clinically significant pocket haematoma". A recent population-based cohort study from Denmark containing 5,918 patients reported a pocket haematoma rate of 2.3% although frequency of overall complications almost reached 10% [2]. David *et al*. compared heparin bridging strategy (HBS) with continued warfarin treatment in patients at high risk for thromboembolic events and found that continued warfarin treatment was associated with decreased incidence of clinically significant pocket haematoma[8].This relationship was in accordance with the meta-analysis and HBS accounted for the nearly 20-fold increased risk of pocket haematoma formation.Furthermore, another recent meta-analysis by Du et al. offered continuous oral-anticoagulant treatment as the best strategy with respect to lower risk of bleeding and rare thromboembolism when compared with HBS or interrupted oral-anticoagulant therapy[9]. The relatively low number rates of pocket haematoma observed in recent studies may be attributed to increased operator experience and abandonment of heparin-bridging strategy. Similarly, our study demonstrates a pocket haematoma rate of 2.6% which is consistent with the literature[10].

The present study showing that dual antiplatelet therapy significantly increases pocket haematoma risk is consistent with previous studies including the meta-analysis of Bernard *et al.*[1,11]. In a review it is suggested that discontinuation of dual antiplatelet therapy, in particular clopidogrel, 4-5 days before device surgery decreases pocket haematoma risk[6]. Accumulating evidence and the most recent ESC cardiac pacing guidelines suggest that dual antiplatelet therapy for primary prevention of cardiovascular events can safely be interrupted before CIED surgery but established management of patients with recent DES or other indications mandating dual antiplatelet therapy is challenging and requires individualized management[7,12]. Optimal management of antiplatelet therapy in patients with recently implanted drug-eluting stents (DES) before cardiac device surgery is still no man's land. It is largely left to the operator's discretion to balance the risk of thrombotic complications such as late-stent thrombosis and bleeding risks in patients on dual antiplatelet therapy.

Previous studies including meta-analyses classified patients on either aspirin or clopidogrel alone as single antiplatelet therapy group and this group was not associated with an increased risk of haematoma formation[1,8]. Kutinsky *et al*. subdivided this group into clopidogrel or ASA alone subgroups. As expected, each group solely was not associated with increased haematoma risk (4.2% for aspirin and 11.1% for clopidogrel) but combination of these two antiplatelet drugs dramatically and significantly increased the risk (24.2%) for pocket haematoma[3].Similarly, we detected that dual antiplatelet therapy maintained perioperatively caused increased pocket haematoma formation and one of these was a clinically significant pocket haematoma. These findings suggest a summative antiplatelet effect of ASA and clopidogrel on platelet count and functions.

Thrombocytopenia was thoroughly investigated in just one of previous studies and the presence of moderate to severe thrombocytopenia was found to be associated with increased pocket haematoma complication[12].

Table 4 Comparison of patients with and without haematoma according to drug therapy and procedural characteristics

	Haematoma (+)	Haematoma (–)	*P*
Drug therapy			
Clopidogrel	1 (16.7%)	11 (4.9%)	*0.001*
ASA	0 (0%)	73 (32.3%)	
ASA + clopidogrel	4 (66.7%)	25 (11.1%)	
ASA + warfarin	1 (16.7%)	20 (8.8%)	
Warfarin	0 (0%)	34 (15%)	
Control	0 (0%)	63 (27.9%)	
Devices			
ICD	0 (0%)	90 (39.8%)	0.250
CRT	2 (33.3%)	52 (23%)	
Permanent pacemaker	3 (50%)	58 (25.7%)	
Device upgrade	1 (16.7%)	26 (11.5%)	

ASA: acetylsalicylic acid, CRT: cardiac resynchronization therapy, ICD: implantable cardioverter/defibrillator.

So, we included data of baseline thrombocyte count in our study and obtained a similar result with the mentioned study although our definition of low platelet levels was not necessarily thrombocytopenia. We found that baseline platelet level was associated with pocket haematoma formation. Despite the fact that our patient population did not contain any patient with severe thrombocytopenia, the association we detected between low platelet levels and increased pocket haematoma formation may be attributed to the suppression of platelet functions as a result of antiplatelet usage.

A retrospective study by Thal *et al.* reported that patients under dual antiplatelet therapy have increased risk of pocket haematoma and half of these patients needed pocket revision and evacuation. This relationship was supported by a prospective trial though the percentage of patients requiring surgical evacuation was much lower (6.6%) [13]. In our study the patient who developed a clinically significant pocket haematoma was receiving dual antiplatelet therapy and required prolonged hospitalization and interruption of antiplatelet therapy but surgical revision or evacuation was not needed.

We categorized patients into 6 groups according to the antiplatelet/anticoagulant therapy or combination. Among the 6 groups, dual antiplatelet therapy was the only group found to be associated with increased risk of pocket haematoma. Cano *et al.* followed a similar strategy and divided patients into 5 groups and two of them contained an oral anticoagulant plus enoxaparin bridging strategy. Enoxaparin bridging strategy and dual antiplatelet therapy were found associated with increased haematoma risk[5]. Unlike that study we continued oral anticoagulant therapy both in warfarin and warfarin + ASA therapy groups if they had a high risk of thromboembolic events (annual risk ≥ 5%) and discontinued warfarin in low-risk patients as recommended by current literature[3,6,14]. On the day of surgery the mean INR was 2.02 in our patient population, and one patient in the warfarin + ASA group developed a minor pocket haematoma while no patient in the warfarin group developed a haematoma. Although abolishment of HBS led to fewer patient groups according to antiplatelet/anticoagulant therapy combinations, it may be considered as the primary rationale for lower pocket haematoma rates compared with previous studies.

Apart from well-established risk factors dual antiplatelet therapy and HBS, several other risk factors have been identified in recent studies individually such as female gender, obesity, chronic renal insufficiency, cephalic vein cut-down, operator experience and high-volume centre, number of leads, emergency or out-of-hours, system upgrade, type of device, procedure duration, etc. and these factors should be investigated with randomized, prospective trials in order to establish definitive conclusions[2,15].

Our study may be regarded as an extension of previous studies with supportive results[16]. Despite accumulated data about bleeding complications following cardiac device surgery, there are still gaps in many topics including standard pocket haematoma definition, optimal management of antiplatelet and anticoagulant therapy, bleeding risks with newer antiplatelet and anticoagulant agents, timing for surgical intervention. A recent study investigated bleeding risks after CIED implantation with uninterrupted dabigatran in comparison with warfarin and obtained similar risks[17].In spite of this promising result, the risk was increased when combined with antiplatelet drugs which emphasizes that combination therapy of anticoagulant and antiplatelet therapy including novel agents will keep challenging operators in the near future.

STUDY LIMITATIONS

The main limitations of the present study are data collection from a single institution and the retrospective observational design which consequently leads to bias possibility and presence of multiple confounders. Similarly, performance of all device procedures by a single senior operator and his awareness of preoperative antiplatelet/anticoagulant therapy shall be considered as another limitation. On the other hand, it could be an advantage for the elimination of operator-dependent bleeding complications. The sample size of the divided subgroups is relatively small in comparison with previous studies. All of the CIED implantations were performed by using direct subclavian puncture, actually none of the previous studies but Kutinsky *et al.* demonstrated superiority of cephalic vein access[3].

CONCLUSIONS

Dual antiplatelet therapy and baseline low platelet levels significantly increased the risk of pocket haematoma formation in patients undergoing CIED implantations. Examination of baseline thrombocyte count and interruption of dual antiplatelet therapy whenever possible may be helpful in the prevention of pocket haematoma formation following cardiac device surgery.

CONFLICT OF INTEREST: none.

REFERENCES

1. Bernard ML, Shotwell M, Nietert PJ, Gold MR. Meta-analysis of bleeding complications associated with cardiac rhythm device implantation. *Circ Arrhythm Electrophysiol* 2012; **5**: 468-74.
2. Kirkfeldt RE, Johansen JB, Nohr EA, Jørgensen OD, Nielsen JC. Complications after cardiac implantable electronic device implantations: an analysis of a complete, nationwide cohort in Denmark. *Eur Heart J* 2014; **35**: 1186-94.
3. Kutinsky IB, Jarandilla R, Jewett M, Haines DE. Risk of hematoma complications after device implant in the clopidogrel era. *Circ Arrhythm Electrophysiol* 2010; **3**: 312-8.
4. Tompkins C, Cheng A, Dalal D, Brinker JA, Leng CT, Marine JE, Nazarian S, Spragg DD, Sinha S, Halperin H, Tomaselli GF, Berger RD, Calkins H, Henrikson CA. Dual antiplatelet therapy and heparin "bridging" significantly increase the risk of bleeding complications after pacemaker or implantable cardioverter-defibrillator device implantation. *J Am Coll Cardiol* 2010; **55**: 2376-82.
5. Cano O, Osca J, Sancho-Tello MJ, Olagüe J, Castro JE, Salvador A. Morbidity associated with three different antiplatelet regimens in patients undergoing implantation of cardiac rhythm management devices. *Europace* 2011; **13**: 395-401.
6. Korantzopoulos P, Letsas KP, Liu T, Fragakis N, Efremidis M, Goudevenos JA. Anticoagulation and antiplatelet therapy in implantation of electrophysiological devices. *Europace* 2011; **13**: 1669-80.
7. European Society of Cardiology (ESC); European Heart Rhythm Association (EHRA), Brignole M, Auricchio A, Baron-Esquivias G, Bordachar P, Boriani G, Breithardt OA, Cleland J, Deharo JC, Delgado V, Elliott PM, Gorenek B, Israel CW, Leclercq C, Linde C, Mont L, Padeletti L, Sutton R, Vardas PE. 2013 ESC guidelines on cardiac pacing and cardiac resynchronization therapy: the task force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). *Europace* 2013; **15**: 1070-118.
8. Birnie DH, Healey JS, Wells GA, Verma A, Tang AS, Krahn AD, Simpson CS, Ayala-Paredes F, Coutu B, Leiria TL, Essebag V; BRUISE CONTROL Investigators. Pacemaker or defibrillator surgery without interruption of anticoagulation. *N Engl J Med* 2013; **368**: 2084-93.
9. Du L, Zhang Y, Wang W, Hou Y. Perioperative anticoagulation management in patients on chronic oral anticoagulant therapy undergoing cardiac devices implantation: a meta-analysis. *Pacing Clin Electrophysiol* 2014; **37**: 1573-86.
10. Sridhar AR, Yarlagadda V, Yeruva MR, Kanmanthareddy A, Vallakati A, Dawn B, Lakkireddy D. Impact of haematoma after pacemaker and CRT device implantation on hospitalization costs, length of stay, and mortality: a population-based study. *Europace* 2015; **17**: 1548-54.
11. Dai Y, Chen KP, Hua W, Zhang JT, Zhang S. Dual antiplatelet therapy increases pocket hematoma complications in Chinese patients with pacemaker implantation. *J Geriatr Cardiol* 2015; **12**: 383-7.
12. Chen HC, Chen YL, Guo BF, Tsai TH, Chang JP, Pan KL, Lin YS. Thrombocytopenia, dual antiplatelet therapy, and heparin bridging strategy increase pocket hematoma complications in patients undergoing cardiac rhythm device implantation. *Can J Cardiol* 2013; **29**: 1110-7.
13. Thal S, Moukabary T, Boyella R, Shanmugasundaram M, Pierce MK, Thai H, Goldman S. The relationship between warfarin, aspirin, and clopidogrel continuation in the peri-procedural period and the incidence of hematoma formation after device implantation. *Pacing Clin Electrophysiol* 2010; **33**: 385-8.
14. Ahmed I, Gertner E, Nelson WB, House CM, Dahiya R, Anderson CP, Benditt DG. Continuing warfarin therapy is superior to interrupting warfarin with or without bridging anticoagulation therapy in patients undergoing pacemaker and defibrillator implantation. *Heart Rhythm* 2010; **7**: 745-9.
15. Said SM, Esperer HD, Hahn J, Bollmann A, Richter S, Rauwolf T, Prondzinsky R, Schmeisser A, Braun-Dullaeus RC. Influence of oral antiplatelet therapy on hemorrhagic complications of pacemaker implantation. *Clin Res Cardiol* 2013; **102**: 345-9.
16. Dreger H, Grohmann A, Bondke H, Gast B, Baumann G, Melzer C. Is antiarrhythmia device implantation safe under dual antiplatelet therapy? *Pacing Clin Electrophysiol* 2010; **33**: 394-9.
17. Jennings JM, Robichaux R, Mcelderry H T, Plumb VJ, Gunter A, Doppalapudi H, Osorio J, Yamada T, Kay GN. Cardiovascular implantable electronic device implantation with uninterrupted dabigatran: comparison to uninterrupted warfarin. *J Cardiovasc Electrophysiol* 2013; **24**: 1125-9.