International Journal of Cardiology Congenital Heart Disease 19 (2025) 100562

Contents lists available at ScienceDirect

International Journal of Cardiology Congenital Heart Disease

journal homepage: www.journals.elsevier.com/international-journal-of-cardiology-congenital-heart-disease

Ventricular arrhythmias and the role of antitachycardia pacing in patients with electrical heart disease and hypertrophic cardiomyopathy

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ARTICLE INFO

Keywords:
Antitachycardia pacing
Electrical heart disease
Hypertrophic cardiomyopathy
ICD
Ventricular tachycardia

ABSTRACT

Background: Whether it is reasonable to program ATP in patients with electrical heart disease (EHD) or hypertrophic cardiomyopathy (HCM) is not thoroughly clarified. Aim of the study was to define the types of ventricular arrhythmias and evaluate the safety and efficacy of ATP activation in these patients.

Methods and results: A total of 154 patients (53.9 % male, 64.9 % secondary prevention) with EHD or HCM, who had an implanted cardioverter defibrillator (ICD) with ATP activated, were included in this retrospective analysis; comprising a median of 65.0 months of follow-up. In 39/154 (25.3 %) patients appropriate ICD therapy was delivered during the follow-up. Patients with HCM had a significantly higher incidence rate of monomorphic VTs than patients with EHD (0.21 versus 0.01 per month, 0 < 0.001). ATP terminated monomorphic VT with an efficacy of 88,2 % in 94.9 % of the occurring episodes. The incidence rate per month of torsade de pointes (TdP) tachycardia and VF was significantly higher in patients with EHD versus HCM (0.04 vs. 0.001, p=<0.001; 0.06 vs. 0.007, p=<0.001). The termination of TdP tachycardia and VF was associated with ATP in 14.0 % and 0 % (ATP efficacy of 28.3 % and 0 % respectively). The implantation for secondary prevention was associated with the occurrence of appropriate ICD therapy during the follow-up period (OR 3.94 [95%CI 1.53–10.14], p = 0.005).

Conclusion: Ventricular tachycardias in patients with HCM are primarily monomorphic and can be effectively terminated with ATP. In patients with EHD, TdP tachycardias and VF occur more frequently and are preferentially terminated by ICD shock.

No financial support of the study.

1. Introduction

Implantable cardioverter defibrillators (ICD) are widely used for primary and secondary prevention of sudden cardiac death [1,2]. Two modalities are available to terminate ventricular arrhythmias: one is the cardioversion or defibrillation and the other is antitachycardia pacing (ATP).

Although cardioversion shocks are highly effective in terminating ventricular tachyarrhythmia they may not only reduce the quality of life but may also lead to a considerable battery drain [3–5]. Furthermore, studies show that ICD shocks itself are associated with increased morbidity and mortality [6–8], although the underlying disease might play the greater role in this regard [9].

ATP programming has been shown to be effective in terminating slow and fast ventricular tachycardia avoiding painful shocks without increasing the risk of arrhythmia acceleration or arrhythmia syncope in patients with ischemic or idiopathic dilated cardiomyopathy [10–15].

In patients with other underlying cardiac diseases, especially in entities like electrical heart disease (EHD) and hypertrophic cardiomyopathy (HCM), which are commonly believed to cause primarily polymorphic VT or ventricular fibrillation (VF), ATP was programmed with restraint due to concerns about efficacy, proarrhythmic effects and arrhythmic syncope due to delayed shock delivery.

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https://doi.org/10.1016/j.ijcchd.2024.100562
Received 7 December 2024; Received in revised form 26 December 2024; Accepted 27 December 2024
Available online 28 December 2024

1.1. Aim of the study

The aim of the present study was to evaluate which type and number of ventricular arrhythmias occur in patients with EHD and HCM and to determine the efficacy and safety of ATP programming in this group of patients.

2. Methods

2.1. Patient population

This monocentric retrospective analysis included patients with long QT Syndrome, Brugada Syndrome, idiopathic ventricular fibrillation, catecholaminergic polymorphic ventricular tachycardia, early repolarisation syndrome and hypertrophic cardiomyopathy in whom an ICD was implanted for either primary or secondary prevention and at least one ATP was activated for heart rates >200 bpm. Patient charts were evaluated for the occurrence and type of arrhythmias and ICD therapy. Patients were supplied with an ICD at our center between June 1993 and May 2012 or have received their device in other centers but were followed up at our institution. After May 2012, patients with EHD or HCM were increasingly provided with a subcutaneous ICD. In order to avoid potential bias,no more patients were included in the study population after this date.

2.2. ICD implantation

ICD implantation was performed transvenously except in pediatric patients in whom epicardial fixation of the leads was preferred. Sensing and pacing thresholds were routinely determined during the implantation procedure. The performance of defibrillation threshold testing and the type and manufacturer of the implanted device depended on the decision of the implanter.

2.3. Data collection and long-term follow-up

For the retrospective analysis, baseline data at implantation and follow-up data of all patients were collected. The study protocol was approved by the ethics committee of the Technische Universität München, Munich, as the leading ethics committee for the German Heart Center Munich.

After the implantation, patients routinely underwent a first device check-up at two month, thereafter regular device interrogations were scheduled every six months. Device interrogation data were independently reviewed by two cardiologists regarding occurring atrial and ventricular arrhythmias and regarding appropriate and inappropriate ICD therapy including both ATP and shocks.

ICD therapies were classified as appropriate when they occurred in response to ventricular tachycardia or ventricular fibrillation; ICD therapies delivered because of atrial tachycardia, electromagnetic interference or oversensing were defined as inappropriate.

2.4. Statistics

Data analysis was performed using the software package IBM SPSS statistics Version 25 for Windows (IBM Corp. Ammonk, N.Y. USA). For quantitative measures means and standard deviations or median and ranges (minimum to maximum) are displayed. For categorical measures absolute and relative frequencies are shown. Univariable analysis was used to evaluate associations between baseline data and appropriate ICD therapy. Incidence rates per month were compared using the Poisson test. All statistical tests were performed two-sided on a significance level of $\alpha = 0.05$.

3. Results

3.1. Population characteristics

A total of 154 patients with ICD implantation due to EHD or HCM between June 1993 and May 2012 were included in the study. Of these, 83 (53.9 %) were male, mean age was 44.4 ± 21.9 years, 100 (64.9 %) were implanted for secondary prevention of sudden cardiac death. The follow-up was in median 65.0 months, with the date of the last follow-up in December 2020. Underlying cardiac diseases were long QT Syndrome (n = 42), Brugada Syndrome (n = 19), idiopathic ventricular fibrillation (n = 11), catecholaminergic polymorphic ventricular tachycardia (n = 3), early repolarisation syndrome (n = 3) and hypertrophic cardiomyopathy (n = 76). A single chamber-chamber device was implanted in 83 patients (53.9 %). At least one ATP was activated in all 154 patients according to the inclusion criteria.Detailed baseline demographic and clinical characteristics of the studied patients are shown in Table 1.

3.2. Ventricular arrhythmias and appropriate ICD therapy

During follow-up, ventricular arrhythmias causing appropriate ICD therapy (ATP or shock) were observed in 39 (25.3 %) patients. A total of 692 ventricular tachyarrhythmias were documented in these patients, consisting of 433 monomorphic VT episodes, 11 polymorphic VT episodes, 93 torsade de pointes tachycardias (TdP) and 155 episodes of VF.

In an univariable analysis the implantation of the ICD for secondary prevention was the only factor which was associated with the occurrence of appropriate ICD therapy due to ventricular tachyarrhythmia during follow-up (OR 3.94 [95%CI 1.53–10.14], p = 0.005). Further results of the univariable analysis are displayed in Table 2.

A total of 407 monomorphic VT occurred in patients with HCM, while 26 episodes of monomorphic VT were documented in EHD patients. The incidence rate per month of monomorphic VT was significantly higher in patients with HCM than in patients with EHD (0.21 versus 0.01, RR 0.06 [95%CI 0.04–0.09], p < 0.001). However, the incidence rate per month of TdP tachycardia and of VFwas significantly higher in patient with EHD than in patients with HCM (0.04 versus 0.001, RR 39.59 [95%CI 11.7–239.4], p < 0.001 and 0.06 versus 0.007, RR 9.50 [95%CI 5.54–17.47], p < 0.001). A total of 2 TdP tachycardias

Table 1
Baseline characteristics.

Patients n = 154	
Age at implant [years], mean ± STD	44.4 ± 21.9
Male n (%)	83 (53.9)
Cardiac Disease	
Electrical Heart Disease n (%)	78 (50.6)
Long-QT-Syndrom n (%)	42 (27.3)
Brugada Syndrom n (%)	19 (12.3)
Idiopathic VF n (%)	11 (7.1)
Early Repolsarisation n (%)	3 (1.9)
CPVT n (%)	3 (1.9)
Hypertrophic CMP n (%)	76 (49.4)
Arterial Hypertension n (%)	37 (24.0)
Diabetes n (%)	13 (8.4)
Previous myocardial infarction n (%)	6 (3.9)
Coronary artery disease n (%)	17 (11.0)
LVEF [%], median (min-max)	60 (25–78)
Medication at implant date	
AAD I n (%)	4 (2.6)
AAD II (Betablocker) n (%)	106 (68.8)
AAD III n (%)	4 (2.6)
ACE-Inhibitor n (%)	54 (35.1)
Single chamber ICD n (%)	83 (53.9)
Dual chamber ICD n (%)	67 (43.5)
CRT-ICD n (%)	4 (2.6)
Primary Prevention n (%)	54 (35.1)
Secondary Prevention n (%)	100 (64.9)
Follow-Up [month], median (min-max)	65.0 (2–299)

Table 2
Univariable analysis for the association of appropriate ICD therapy with baseline characteristics. Numbers indicate absolute and relative frequencies of patients with appropriate ICD therapy in the corresponding subgroups.

Variables (n=154 patients)	Yes	No	OR	95 % CI	p
Male n	17/83 (20.5 %)	22/71 (31.0 %)	0.57	(0.28–1.19)	0.137
Electrical heart disease n	20/78 (25.6 %)	19/76 (25.0 %)	1.03	(0.50–2.14)	0.927
Hypertrophic Cardiomyopathy n	19/76 (25.0 %)	20/78 (25.6 %)	0.97	(0.47–1.99)	0.927
Arterial Hypertension n	10/37 (27.0 %)	29/117 (24.8 %)	1.12	(0.49–2.60)	0.785
Diabetes n	2/13 (15.4 %)	37/141 (26.2 %)	0.51	(0.11–2.41)	0.397
Previous myocardial infarction n	3/6 (50.0 %)	36/148 (24.3 %)	2.00	(0.39–10.23)	0.405
Coronary artery disease n	5/17 (29.4 %)	34/137 (24.8 %)	1.40	(0.44–4.49)	0.568
Single chamber ICD n	19/83 (22.9 %)	20/71 (28.2 %)	0.76	(0.37–1.57)	0.454
Dual chamber ICD n	18/67 (26.9 %)	21/87 (24.1 %)	1.15	(0.56–2.40)	0.700
Secondary prevention n	33/100 (33.0 %)	6/54 (11.1 %)	3.94	(1.53–10.14)	0.005

and 13 episodes of VF occurred in patients with HCM, while a total of 91 TdP tachycardias and 142 episodes of VF were documented in EHD patients.

The mean cycle length of ventricular arrhythmia differed significantly between patients with EHD and patients with HCM (EHD 250.57 ± 39.46 ms vs. HCM 321.26 ± 47.19 ms; $p < 0.001$).

ATP terminated monomorphic VT with an efficacy of 88,2 % (number of effective vs. ineffective ATP 411 vs. 466, ATP termination after median 1 ATP [min – max: 1–4 ATP]; mean 1.14 ± 0.52), with 88 % (381/433) of monomorphic VT having a cycle length between 300 and 350 ms. In total 411 (94,9 %) monomorphic VT episodes were terminated by ATP.

TdP tachycardia termination was associated with ATP in 13 (14.0 %) episodes (ATP efficacy of 28.3 %, potentially effective vs. ineffective ATP 13 vs.46). Arrhythmia termination occurred after median of 1 ATP [min – max: 1–2 ATP]; mean 1.22 ± 0.44). None of the 155 episodes of VF could be terminated by ATP, with ATP being delivered during loading of the ICD in all cases (efficacy 0 %).

In total, nineteen ICD shocks were delivered to terminate monomorphic VT with a first shock efficacy of 89.5 % (number of effective vs. ineffective shock 17/2, termination after median 1 shock [min – max: 1–2 shocks]; mean 1.12 ± 0.35). In patients with an ineffective first shock, termination could be achieved with the subsequent shock. TdP tachycardia were terminated by 59 ICD shocks with a first shock efficacy of 93.2 % (number of effective vs. ineffective shock 55/4, termination after median 1 shock [min-max 1–2 shocks:]; mean 1.03 ± 0.19). To terminate VF a total of 161 shocks were delivered with an efficacy of 91.0 % (number of effective vs. ineffective shock 147/14, termination after median 1 shock [min – max: 1–4 shocks]; mean 1.25 ± 0.67).

Spontaneous termination of sustained ventricular arrhythmia happened in no episodes (0 %) of monomorphic and in 2 episodes (18.2 %) of polymorphic VT, in 12 episodes (12.9 %) of TdP tachycardia, and in 8 instances (5.2 %) of VF, respectively.

For detailed information on occurring ventricular tachycardias, their therapy and the incidence rate per month please see Table 3.

3.3. Inappropriate ATP therapies and ICD shocks

During the follow-up inappropriate ICD therapy was delivered due to diverse reasons in 18 patients (11.7 % of the study population).

In 13 patients (EHD n = 2 vs. HCM n = 11, 8.4 % of the study population) a total of 368 inappropriate ATP therapies were delivered with a significantly higher incidence rate per month in patients with HCM compared with EHD (RR 0.04 [95%CI 0.01–0.10], $p < 0.001$). Most common reason for inappropriate ATP was supraventricular tachycardia in both patient groups.

In 10 patients (EHD n = 1 vs. HCM n = 9, 6.5 % of the study population) inappropriate ATP was followed by inappropriate shock.

A total of 59 inappropriate ICD shocks were delivered in 13 patients (8.4 % of the study population, EHD n = 4 vs. HCM n = 9). Most common reason for inappropriate ICD shocks were supraventricular tachycardia in patients with HCM and T-Wave-Oversense in patients with EHD. The incidence rate per month of inappropriate ICD shocks was significantly higher in patients with EHD (RR 3.39 [95%CI 2.02–5.67], $p < 0.001$).

Detailed information on inappropriate ICD therapy is shown in Table 4.

3.4. Acceleration

Acceleration of ventricular heart rate related to inappropriate ATP occurred in 3 atrial arrhythmia episodes in 2 patients (EHD n = 0 vs.

Table 3
Occurrence of ventricular tachycardia, incidence rate per month and appropriate ICD therapy. VT = ventricular tachycardia, ATP = antitachycardia pacing, eff = effective, ineff = ineffective.

Patients	Total (n=39)	EHD (n=20)	Incidence rate per month	HCM (n=19)	Incidence rate per month	p	RR (CI)
Monomorphic VT	**433**	**26**	**0.01**	**407**	**0.21**	**<0.001**	**0.06 (0.04–0.09)**
ATP (eff/ineff)	**466 (411/55)**	**40 (14/26)**		**426 (397/29)**			
Shock (eff/ineff)	**19(17/2)**	**13(12/1)**		**6(5/1)**			
Spontaneous Termination	**0**	**0**		**0**			
Acceleration (ATP/Shock)	**5(5/0)**	**0(0/0)**		**5(5/0)**			
Polymorphic VT	**11**	**7**	**0.003**	**4**	**0.002**	**0.52**	**1.52 (0.44–5.94)**
ATP (eff/ineff)	**10(6/4)**	**2(2/0)**		**8(4/4)**			
Shock (eff/ineff)	**3(3/0)**	**3(3/0)**		**0(0/0)**			
Spontaneous Termination	**2**	**2**		**0**			
Acceleration (ATP/Shock)	**0(0/0)**	**0(0/0)**		**0(0/0)**			
Torsade de Points	**93**	**91**	**0.04**	**2**	**0.001**	**<0.001**	**39.59 (11.7–239.4)**
ATP (eff/ineff)	**46(13/33)**	**46(13/33)**		**0(0/0)**			
Shock (eff/ineff)	**59(55/4)**	**57(53/4)**		**2(2/0)**			
Spontaneous Termination	**12**	**12**		**0**			
Acceleration (ATP/Shock)	**13(13/0)**	**13 (13/0)**		**0(0/0)**			
Ventricular Fibrillation	**155**	**142**	**0.06**	**13**	**0.007**	**<0.001**	**9.50 (5.54–17.47)**
ATP (eff/ineff)	**4(0/4)**	**1(0/1)**		**3(0/3)**			
Shock (eff/ineff)	**161(147/14)**	**148(135/13)**		**13(12/1)**			
Spontaneous Termination	**8**	**7**		**1**			

Table 4
Inappropriate ICD therapy, incidence rate per month and reasons for inappropriate ICD therapy. ATP = antitachycardia pacing.

Patients	Total (n = 18)	EHD (n = 5)	Incidence rate per month	HCM (n = 13)	Incidence rate per month	p	RR (CI)
Inappropriate ATP	**368**	**4**	**0.01**	**364**	**0.27**	**<0.001**	**0.04 (0.01–0.10)**
Supraventricular Tachycardia	**292**	**4**		**288**			
T-Wave-Oversensing	**76**	**0**		**76**			
Oversensing due to lead failure	**0**	**0**		**0**			
Electromagnetic Interference	**0**	**0**		**0**			
Inappropriate Shock	**59**	**28**	**0.08**	**31**	**0.02**	**<0.001**	**3.39 (2.02–5.67)**
Supraventricular Tachycardia	**25**	**2**		**23**			
T-Wave-Oversensing	**24**	**24**		**0**			
Oversensing due to lead failure	**6**	**2**		**4**			
Electromagnetic Interference	**4**	**0**		**4**			
Inapp. ATP before inapp. Shock	**43**	**1**	**0.00**	**42**	**0.03**	**0.001**	**0.09 (0.00–0.47)**

HCM n = 2, 1.3 % of the study population) and was each followed by ICD shock, which terminated the supraventricular tachycardia in all cases.

Acceleration with appropriate ATP occurred in 15 ventricular arrhythmia episodes in 8 patients (EHD n = 4 vs. HCM n = 4, 5.2 % of the study population, number of ATP = 18). All accelerated ventricular arrhythmias were subsequently terminated by ICD shocks.

No acceleration occurred in association with ICD shocks or inappropriate ICD therapies (shocks or ATP). For further details, please see Table 3.

4. Discussion

Data about the role of antitachycardia pacing (ATP) in patients with electrical heart disease (EHD) and hypertrophic cardiomyopathy (HCM) is scarce. The main findings of our study with its very long-term follow-up of such a patient population are: 1) in patients with EHD torsade-de-pointes (TdP)-tachycardias and ventricular fibrillation (VF) occur primarily whereas in patients with HCM monomorphic ventricular tachycardia (VT) occur more often, 2) the implantation of the ICD for secondary prevention was the only factor which was associated with the occurrence of appropriate ICD therapy and 3) in patients with EHD most of the occurring ventricular arrhythmias were terminated by shock whereas ventricular arrhythmias in patients with HCM mostly were terminated by ATP and thereby ATP prevented the need for ICD shock delivery in patients with HCM in more than 94 % of episodes.

It is known from several studies that patients with EHD primarily develop fast polymorphic VT, TdP tachycardia or VF, which are often self-limiting in the case of TdP tachycardia but are highly likely to result in death when VF occurs [16–18]. The same is thought to be true for HCM, however data on the nature and specific treatment of ventricular arrhythmia in this patient group is overall scarce. However, it is shown in some studies that patients with HCM often initially experience monomorphic ventricular tachycardia, which then degenerates to ventricular fibrillation [11,19,20]. This was also observed in our study, in which patients with HCM primarily experienced monomorphic VT during the long-term follow-up, whereas patients with EHD tended to experience TdP tachycardias or VF as their primary arrhythmia. This is accompanied by the fact that the cycle length of the occurring ventricular arrhythmias was significantly longer in patients with HCM compared to those in patients with EHD.

In patients with structural heart disease the programming of ATP is well established and its painless termination of arrhythmias and the battery saving behavior is very appreciated [1,2,10,11]. This is because various studies have shown that ATP can effectively terminate ventricular tachycardias, even if they are in a high-frequency range (10–12; 15). However, this has been shown mainly for monomorphic VT in patients with ischemic cardiomyopathy or dilative cardiomyopathy. For patients with other cardiac diseases, little reliable data exists so far. In our study population of patients with EHD and HCM, monomorphic VT were terminated with very good efficacy by ATP, whereas TdP tachycardia with shorter cycle length could not be terminated with ATP to a satisfactory extent. Moreover, ATP during loading of the ICD could not terminate ventricular fibrillation in a single episode.

As a result, ventricular arrhythmias in patients with HCM, which were mostly monomorphic VT, could be terminated primarily with ATP; whereas the shock was the effective therapy for the ventricular arrhythmias, mostly TdP tachycardia or VF, in patients with EHD.

For patients with HCM, this means that a large number of ICD shocks could be saved by ATP.

Concerns, that ATP could lead to a high rate of acceleration or that inappropriate ATP could trigger VT have not been confirmed so far [21–24]. Our study showed a low rate of acceleration in this particular patient population: Inappropriate ATP in general occurred in 8,4 % of our study population and were triggered by supraventricular tachycardia. In only three episodes in two patients (1.3 % of the study population) the inappropriate ATP led to acceleration of ventricular heart rate and was followed by inappropriate shock. Acceleration due to appropriate ATP occurred in 15 ventricular arrhythmia episodes in 8 patients (5.2 % of the study population) and were all subsequently terminated by ICD shocks. No episode of supraventricular or ventricular arrhythmia was triggered by inappropriate or by appropriate ATP.

For about a decade, subcutaneous ICDs have been implanted with priority in patients with EHD and HCM [25]. The advantages for the mostly younger patients are obvious: the totally subcutaneous implantation leads to a reduction of lead complications and cardiac or systemic inflammation in the long term [25,26]. An important disadvantage is the lack of antibradycardic stimulation and the lack of ATP. Ventricular tachyarrhythmias can only be treated by shock delivery. In particular patients with HCM suffered from inappropriate shock delivery with the first generations of devices, often triggered by oversensing [27–29]. This problem has improved significantly with the latest generation of S-ICDs using the SMART pass algorithm [30]. Nevertheless, a reduction of inappropriate and appropriate shocks must continue to be the aim of further development, given that ICD shocks not only result in severe psychological impairment but possibly also increase morbidity and mortality [8,31]. Especially in this mostly young patient population these relevant issues should be kept in mind.

Given that in most cases ATP can obviously effectively terminate the initially present monomorphic ventricular tachycardias in patients with HCM and thereby prevent further shock therapy, it should carefully be considered before implantation of an ICD in these patients whether a transvenous system with its possibility of overstimulation may provide additional therapeutic benefits. Advances in ICD technology may further optimize the management of ventricular arrhythmias in patients with EHD and HCM in the future. The extravascular ICD combines subcutaneous placement of the device with a retrosternal lead, allowing for subcutaneous ATP [32]. Another interesting development are leadless systems that can be combined with S-ICDs to deliver ATP [33]. Such advances could spare HCM patients the need to switch to transvenous ICD systems when pharmacologic or ablative therapies are unsuccessful, further reducing the risks and long-term complications associated with transvenous leads. Future studies will need to show whether the ATP

efficacy of these new systems is comparable to that of transvenous ICDs, particularly in terminating monomorphic VT in patients with HCM.

4.1. Study limitations

This was a retrospective, observational cohort study analysis performed to assess the long-term follow-up in ICD in high-risk ventricular tachyarrhythmia patients. Because patients were implanted over a 9-year period, changes in guidelines for the implantation of ICDs, pharmacological antiarrhythmic therapy, and ablation procedures could have created a heterogeneous treatment. As our study was based on ICD interrogation charts, no reliable statement can be made on the history of syncope or unconsciousness. Finally, ICD settings at discharge have been modified during follow-up, but this should not affect the analysis of safety and efficacy.

5. Conclusion

Ventricular tachycardias in patients with HCM are primarily monomorphic and can be effectively terminated by ATP in the majority of cases. In patients with EHD, polymorphic tachycardias and ventricular fibrillation occur more frequently and are primarily terminated by shock delivery.

CRediT authorship contribution statement

Verena Kantenwein: Writing – review & editing, Writing – original draft, Validation, Project administration, Investigation, Formal analysis, Data curation, Conceptualization. **Herribert Pavaci:** Writing – review & editing, Writing – original draft, Validation, Data curation, Conceptualization. **Bernhard Haller:** Writing – review & editing, Methodology, Formal analysis, Conceptualization. **Marta Telishevska:** Writing – review & editing, Validation, Conceptualization. **Lena Friedrich:** Writing – review & editing, Investigation. **Maximilian Walgenbach:** Writing – review & editing, Investigation. **Carsten Lennerz:** Writing – review & editing, Visualization, Data curation. **Christof Kolb:** Writing – review & editing, Writing – original draft, Supervision, Formal analysis, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Carsten Lennerz, Lena Friedrich reports a relationship with Medtronic Inc that includes: speaking and lecture fees and travel reimbursement. Carsten Lennerz, Lena Friedrich, reports a relationship with Boston Scientific Corporation that includes: speaking and lecture fees and travel reimbursement. Carsten Lennerz, Lena Friedrich reports a relationship with Abbott Vascular Inc that includes: speaking and lecture fees and travel reimbursement. Casten Lennerz, Lena Friedrich reports a relationship with BIOTRONIK Inc that includes: speaking and lecture fees and travel reimbursement. Lena Friedrich reports a relationship with MicroPort Scientific Corporation that includes: funding grants. Christof Kolb reports a relationship with AstraZeneca Pharmaceuticals LP that includes: speaking and lecture fees. Christof Kolb reports a relationship with MicroPort Scientific Corporation that includes: speaking and lecture fees. Christof Kolb reports a relationship with Bristol Myers Squibb that includes: speaking and lecture fees. Christof Kolb reports a relationship with BIOTRONIK Inc that includes: speaking and lecture fees. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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