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EHRA POSITION PAPER

Management of asymptomatic arrhythmias: a European Heart Rhythm Association (EHRA) consensus document, endorsed by the Heart Failure Association (HFA), Heart Rhythm Society (HRS), Asia Pacific Heart Rhythm Society (APHRS), Cardiac Arrhythmia Society of Southern Africa (CASSA), and Latin America Heart Rhythm Society (LAHRS)

David O. Arnar (Iceland, Chair)[1]*, Georges H. Mairesse (Belgium, Co-Chair)[2], Giuseppe Boriani (Italy)[3], Hugh Calkins (USA, HRS representative)[4], Ashley Chin (South Africa, CASSA representative)[5], Andrew Coats (United Kingdom, HFA representative)[6], Jean-Claude Deharo (France)[7], Jesper Hastrup Svendsen (Denmark)[8,9], Hein Heidbüchel (Belgium)[10], Rodrigo Isa (Chile, LAHRS representative)[11], Jonathan M. Kalman (Australia, APHRS representative)[12,13], Deirdre A. Lane (United Kingdom)[14,15], Ruan Louw (South Africa, CASSA representative)[16], Gregory Y. H. Lip (United Kingdom, Denmark)[14,15], Philippe Maury (France)[17], Tatjana Potpara (Serbia)[18], Frederic Sacher (France)[19], Prashanthan Sanders (Australia, APHRS representative)[20], Niraj Varma (USA, HRS representative)[21], and Laurent Fauchier (France)[22]

ESC Scientific Document Group: Kristina Haugaa[23,24], Peter Schwartz[25], Andrea Sarkozy[26], Sanjay Sharma[27], Erik Kongsgård[28], Anneli Svensson[29], Radoslaw Lenarczyk[30], Maurizio Volterrani[31], Mintu Turakhia[32], Isreal W.P. Obel[33], Mauricio Abello[34], Janice Swampillai[35], Zbigniew Kalarus[36,37], Gulmira Kudaiberdieva[38], and Vassil B. Traykov[39]

[1]Department of Medicine, Landspitali - The National University Hospital of Iceland and University of Iceland, Reykjavik, Iceland; [2]Department of Cardiology, Cliniques du Sud-Luxembourg, Arlon, Belgium; [3]Division of Cardiology, Department of Biomedical, Metabolic and Neural Sciences, University of Modena and Reggio Emilia, Policlinico di Modena, Modena, Italy; [4]Department of Arrhythmia Services, Johns Hopkins Medical Institutions Baltimore, MD, USA; [5]Division of Cardiology, Department of Medicine, Groote Schuur Hospital and University of Cape Town, Cape Town, South Africa; [6]Department of Cardiology, University of Warwick, Warwickshire, UK; [7]Department of Rhythmology, Hôpital Universitaire La Timone, Marseille, France; [8]Department of Cardiology, The Heart Centre, Rigshospitalet, Copenhagen University Hospital, Copenhagen, Denmark; [9]Department

* Corresponding author. Tel: +3545431000; fax: +35-5436467. *E-mail address*: davidar@landspitali.is

of Clinical Medicine, Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen, Denmark; [10]Antwerp University Hospital, University of Antwerp, Edegem, Belgium; [11]Clínica RedSalud Vitacura and Hospital el Carmen de Maipú, Santiago, Chile; [12]Department of Cardiology, Royal Melbourne Hospital, Melbourne, VIC, Australia; [13]Department of Medicine, University of Melbourne, Melbourne, VIC, Australia; [14]Liverpool Centre for Cardiovascular Science, University of Liverpool and Liverpool Heart & Chest Hospital, Liverpool, UK; [15]Aalborg Thrombosis Research Unit, Aalborg University, Aalborg, Denmark; [16]Department Cardiology (Electrophysiology), Mediclinic Midstream Hospital, Centurion, South Africa; [17]Cardiology, University Hospital Rangueil, Toulouse, France; [18]Cardiology Clinic, Clinical Center of Serbia, School of Medicine, University of Belgrade, Serbia; [19]Service de Cardiologie, Institut Lyric, CHU de Bordeaux, Bordeaux, France; [20]Centre for Heart Rhythm Disorders, South Australian Health and Medical Research Institute, University of Adelaide and Royal Adelaide Hospital, Adelaide, South Australia, Australia; [21]Department of Cardiovascular Medicine, Cleveland Clinic, Cleveland, OH, USA; [22]Service de Cardiologie et Laboratoire d'Electrophysiologie Cardiaque, Centre Hospitalier Universitaire Trousseau et Université François Rabelais, Tours, France; [23]Department of Cardiology, Center for Cardiological Innovation and Institute for Surgical Research, Oslo University Hospital, Oslo, Norway; [24]Institute of Clinical Medicine, University of Oslo, Oslo, Norway; [25]Istituto Auxologico Italiano, IRCCS, Center for Cardiac Arrhythmias of Genetic Origin, Milan, Italy; [26]Heart Rhythm Management Centre, UZ Brussel-VUB, Brussels, Belgium; [27]St. George's, University of London, UK; [28]Department of Cardiology, OUS-Rikshospitalet, Oslo, Norway; [29]Department of Cardiology, University Hospital of Linkoping, Sweden; [30]Silesian Center for Heart Disease, Zabrze, Poland; [31]IRCCS San Raffaele Pisana, Roma, Italy; [32]Stanford University, Cardiac Arrhythmia & Electrophysiology Service, Stanford, USA; [33]Parktown West, 2006, Johannesburg, Gauteng, South Africa; [34]Sanatorio Finochietto, Buenos Aires, Argentina; [35]Electrophysiologist & Cardiologist, Waikato Hospital, University of Auckland, New Zealand; [36]SMDZ in Zabrze, Medical University of Silesia, Katowice, Poland; [37]Department of Cardiology, Silesian Center for Heart Diseases, Zabrze; [38]Adana, Turkey; and [39]Department of Invasive Electrophysiology and Cardiac Pacing, Clinic of Cardiology, Acibadem City Clinic Tokuda Hospital, Sofia, Bulgaria

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Asymptomatic arrhythmias are frequently encountered in clinical practice. Although studies specifically dedicated to these asymptomatic arrhythmias are lacking, many arrhythmias still require proper diagnostic and prognostic evaluation and treatment to avoid severe consequences, such as stroke or systemic emboli, heart failure, or sudden cardiac death. The present document reviews the evidence, where available, and attempts to reach a consensus, where evidence is insufficient or conflicting.

Keywords Arrhythmias • Asymptomatic • Asystole • Atrial fibrillation • Atrial tachyarrhythmias • Bradycardia • Extrasystoles • Heart failure • Stroke • Tachycardia-induced cardiomyopathy • Ventricular tachycardia • Wolff–Parkinson–White syndrome

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Introduction

The perception of individuals with heart rhythm abnormalities can be highly variable. While many patients are acutely aware of even minor heart beat irregularities, others may be completely unaware of episodes of rapid tachyarrhythmias.

Palpitations are the most common symptom reported by patients with cardiac arrhythmias of various types and duration. The term 'palpitations' refers to a subjective perception of an abnormal cardiac activity, described by patients as an uncomfortable sensation of pulsation or motion in the chest and/or adjacent areas.[1] Some may experience other symptoms, in association with a documented cardiac arrhythmia, such as fatigue, shortness of breath, dyspnoea, chest discomfort, dizziness, or syncope. These symptoms are sometimes referred to as 'atypical presentations' of a symptomatic arrhythmia.[2]

On the other hand, individuals with cardiac arrhythmias can be asymptomatic. Arrhythmias that may in certain cases be asymptomatic, such as atrial fibrillation (AF), incessant supraventricular tachycardias (SVT) and non-sustained ventricular tachycardias (NSVT) could, however, have important implications for patient outcomes.[3–7] Asymptomatic AF may lead to stroke, asymptomatic ventricular arrhythmias may result in sudden cardiac death (SCD), and all forms of sustained or repetitive tachyarrhythmias of various origins can possibly lead to deterioration of left ventricular (LV) function. Moreover, in the same patient, the same type of arrhythmia can be symptomatic in some circumstances but asymptomatic in others.[8]

It's not clear whether asymptomatic arrhythmias should be evaluated and managed differently than symptomatic arrhythmias. This is in large part because published studies on the approach to and therapy of arrhythmias have mainly included symptomatic individuals. Asymptomatic arrhythmias are rather frequent in daily practice and are generally considered to be more benign compared to those that cause symptoms and not requiring treatment. However, it is important for clinicians to recognize that there may be several exceptions and that asymptomatic arrhythmias may require a detailed evaluation and in certain cases, appropriate treatment.

Recently, there has been a rapid increase in the number of medical devices and accessories that are available directly to consumers and can aid in evaluating heart rate or even record a rhythm strip. These devices have the potential to increase the diagnostic yield of heart rhythm disturbances and increase the prevalence of asymptomatic arrhythmias, perhaps even substantially, in the coming years.

Given that the approach to asymptomatic arrhythmias is neither particularly clear nor straightforward, the European Heart Rhythm Association (EHRA), in collaboration with the Heart Failure Association (HFA), the Heart Rhythm Society (HRS), the Asia Pacific (APHRS), the Cardiac Arrhythmia Society of Southern Africa (CASSA), and the Latin American Heart Rhythm Society (LAHRS), convened a Task Force to review the clinical management of specific types of asymptomatic arrhythmias. The goal was to emphasize evidence-based approaches for risk stratification and appropriate pharmacological or non-pharmacological treatments, where evidence exists for asymptomatic arrhythmias. However, the ultimate decision on management must be made by the healthcare provider after discussion with the patient, taking into account individual factors and preferences, along with potential risks and benefits.

Preamble

Members of the Task Force were advised to perform a detailed literature review, weigh the strength of evidence for or against a particular approach, treatment, or procedure, and include estimates of expected health outcomes where data exist. Patient-specific modifiers, co-morbidities, and issues of patient preference that might influence the choice of particular tests or therapies were considered, as was the need for follow-up and not least, cost-effectiveness. With regard to issues without evidence other than clinical experience, a consensus was achieved by agreement of the expert panel after thorough discussions. This document was prepared by the Task Force with representation from EHRA, HFA, HRS, APHRS, CASSA, and LAHRS. The document was peer-reviewed by official external reviewers representing EHRA, HFA, HRS, APHRS, CASSA, and LAHRS.

Consensus statements are evidence-based when possible and derived from available published data or determined through consensus opinion where data are not available. However, the current systems of ranking level of evidence have become complicated such that their practical utility can be compromised. Therefore, we opted for an easier and a more user-friendly system of ranking using 'coloured hearts' that should allow physicians to easily assess the current status of the evidence and consequent guidance. This EHRA grading of consensus statements does not have separate definitions of the level of evidence. This categorization, used for consensus statements, must not be considered as directly similar to that used for official society guideline recommendations, which apply a classification (Class I–III) and level of evidence (A, B, and C) to recommendations.

Thus, a green heart indicates a 'should do this' consensus statement or indicated treatment or procedure that is based on at least one randomized trial, or is supported by strong observational evidence that it is beneficial and effective. A yellow heart indicates general agreement and/or scientific evidence favouring a 'may do this' statement or the usefulness/efficacy of a treatment or procedure. A 'yellow heart' symbol may be supported by randomized trials based on a small number of patients or results which are perhaps not widely applicable. Treatment strategies for which there is scientific evidence of potential harm and should not be used ('do not do this') are indicated by a red heart (*Table 1*).

Finally, this is a consensus document that includes evidence and expert opinions from several countries. The pharmacologic and non-pharmacologic antiarrhythmic approaches discussed may, therefore, include drugs that do not have the approval of governmental regulatory agencies in all countries.

Arrhythmias and symptoms

Arrhythmias may be associated with diverse symptoms, as discussed in the Introduction section. Interestingly, some individuals may be completely asymptomatic from their heart rhythm disturbances. It should also be clarified that not all individuals that experience palpitations are actually having an arrhythmia simultaneously.[9] This conflicting presentation of symptoms or the lack of them in arrhythmias is rather poorly understood. In part, this might be because many studies suffer from a lack of structured systematic assessments and survey instruments for symptoms. In addition, there is a lack of good data assessing the relationship between symptoms and arrhythmia burden. Also potential placebo

Table 1 Scientific rationale behind colored hearts recommendations

Definitions where related to a treatment or procedure	Consensus statement instruction	Symbol
Scientific evidence that a treatment or procedure is beneficial and effective. Requires at least one randomized trial or is supported by strong observational evidence and authors' consensus (as indicated by an asterisk)	'Should do this'	
General agreement and/or scientific evidence favour the usefulness/efficacy of a treatment or procedure. May be supported by randomized trials based on a small number of patients or which is not widely applicable	'May do this'	
Scientific evidence or general agreement not to use or recommend a treatment or procedure	'Do not do this'	

and nocebo effects of therapeutic interventions are not controlled for in most studies on symptoms.

There are many possible contributing factors in determining whether arrhythmias might cause symptoms or not. The type and origin of the arrhythmia likely plays a part in determining whether it is symptomatic or not. The presence of various cardiovascular disorders leading to systolic or diastolic dysfunction may also play an important role. As such, isolated premature beats from both the atria and the ventricles and short bursts of arrhythmias might be less likely to produce symptoms than sustained episodes of the same heart rhythm abnormality. There are no data however to suggest that atrial tachyarrhythmias cause fewer palpitations than ventricular tachyarrhythmias although the latter could possibly have a greater effect on blood pressure, perhaps leading to dizziness or even syncope. In tachyarrhythmias, the decreased diastolic filling time might contribute to a lowering of blood pressure and symptoms. The haemodynamic effect of a heart rhythm disturbance is also influenced by the rate of the arrhythmia, the circulating blood volume at the time of the arrhythmia, the left ventricle function, and the presence of concurrent co-morbidities. The faster the ventricular response during the arrhythmia, the more likely it is to cause symptoms and the lower the left ventricular ejection fraction (LVEF), the less likely the individual is to tolerate a sustained rapid arrhythmia. There are also indications that younger individuals may have more symptoms from arrhythmias than those who are older.[10] With bradyarrhythmias in general, it is believed that a sinus or atrioventricular (AV) pause of at least 6–7 s is needed to cause symptoms such as syncope.[11]

Many people who have arrhythmias also have structural cardiovascular disorders and are taking medications that can affect the ability of the heart to tolerate a heart rhythm disturbance. Such medications include beta-blockers, calcium channel antagonists, and various vasodilators. These drugs might accentuate a negative haemodynamic response during a tachyarrhythmia and in turn increase the probability of symptoms. They may also have an effect on chronotropic response which may also play a role in determining the degree and severity of symptoms.

Sympathetic nervous system afferents are connected to sensory mechanoreceptors which are activated by the mechanical stretch resulting from a premature ventricular contraction (PVC).[12] This causes the perception of premature beats by some patients. The autonomic nervous system modulates cardiac activity in a variety of ways and may in some cases have arrhythmogenic effects and facilitate the induction of heart rhythm disturbances.[13,14] Indeed, cardiac sympathetic denervation has been used to prevent life-threatening arrhythmias.[15] The autonomic nervous system tone may also affect the rate, persistence, and haemodynamic consequences of arrhythmias and via this mechanism possibly influence the perception of the individual's symptoms.

Pain tolerance can vary substantially amongst patients and the relationship between arrhythmias and symptoms also greatly varies between patients. For example, whereas some patients with a very high burden of PVCs (>20%) are completely asymptomatic, other patients experience uncomfortable symptoms with a single PVC. Patients with a low threshold for experiencing symptoms with arrhythmias are sometimes referred to as having 'cardiac awareness'.[16]

The pathophysiologic basis for this significant variation in threshold for symptoms is not known. It is also unknown if genetic influences play a role in whether arrhythmias cause symptoms or not. However, cultural variations between populations, ethnicity, or educational level certainly all play a role in the perception and expression of medical symptoms. There is growing evidence suggesting an association between psychosocial factors and the risk of cardiac arrhythmias.[17] The type of personality might also have an effect on the individual's perception of the arrhythmia although this relationship has not yet been well defined.

Premature atrial contractions and non-sustained atrial tachyarrhythmias

Premature atrial contractions (PACs), while common, do not always cause symptoms and many patients with PACs may be completely unaware of their occurrence.[18] The proportion of patients with PACs that experience symptoms is unknown; as are the demographic and clinical variables that predict whether a patient will have symptoms at the time of the PACs. Also unclear is the possible link between the number of PACs and the development of associated symptoms?

The occurrence of symptoms in arrhythmias is an issue that has been speculated on in the previous chapter, and the focus of this section will be to describe the clinical importance of PACs, regardless of whether they are symptomatic or asymptomatic. In this regard, it is important to recognize that most clinical trials, which have studied the clinical impact of atrial premature beats, did not categorize atrial PACs on the basis of whether they are symptomatic or not but rather their burden over a given period.[18–21] It could also be important to note that some individuals may have a heightened awareness of PACs, including patients early after having undergone AF and SVT ablations.

Over the past two decades, the rather common belief that PACs are benign and of little clinical importance has evolved considerably. Today, it is recognized that the presence of frequent PACs or shorts runs of PACs may be an independent predictor of the development of atrial tachycardia and AF.[19–21] However, the impact of having completely asymptomatic PACs is currently unknown.

Some studies have attempted to evaluate the risk of PACs on outcomes. In a study by Binici *et al.*,[19] 48-h Holter data from the Copenhagen Holter Study, which enrolled healthy middle aged men and women, assessed the relationship between PACs and outcomes of incident AF, stroke, and death. In this study, 15% of individuals without known cardiovascular disease had excessive supraventricular ectopic activity, defined in this case as a burden of 30 PACs or more per hour. After a median follow-up time of 6.3 years, excessive PACs were associated with an increased risk of both the primary endpoint of death or stroke [hazard ratio (HR) 1.64, 95% (confidence interval) CI 1.03–2.60; $P < 0.036$] and admissions for AF (HR 2.78, 95% CI 1.08–6.99; $P < 0.033$).[19]

In the same cohort from the Copenhagen study but with a longer median follow-up of 14.4 years, Larsen *et al.*[22] found that excessive atrial ectopic activity was associated with a two-fold increase in the adjusted risk of stroke. Interestingly, less than 15% of patients with a high number of PACs and stroke had a clinical diagnosis of AF prior to their stroke. Furthermore, the annual stroke risk in patients with excessive atrial ectopic activity in combination with a CHA_2DS_2-VASc score ≥2 was 2.4% per year which is in a similar range as patients with AF and a CHA_2DS_2-VASc score ≥2, supporting the view that PACs burden might actually be a possible surrogate marker for AF. Similar findings were reported by Dewland *et al.*,[21] who examined Holter data from 1260 people without previously known AF from the Cardiovascular Health Study, and found that a doubling of the hourly PAC count was associated with a 17% increase in AF risk (HR 1.17, 95% CI 1.13–1.22) and 6% increase in overall mortality (HR 1.06, 95% CI 1.03–1.09).

One explanation for this observation could be that the presence of frequent PACs identifies patients likely to develop AF, and that AF leads to an increased risk of stroke and death. A second possible mechanism for these observations is that frequent PACs alone may be a marker for a subclinical, atrial cardiomyopathy, that might promote both the development of AF and increased stroke risk.[21,23,24] This 'atrial cardiomyopathy hypothesis' proposes that the development of AF and PACs is an epiphenomenon outside the causal pathway between myopathy and stroke. Recent genetic studies showed an association between mutations in sarcomere genes and AF, a link that may be mediated through a subclinical atrial cardiomyopathy.[25–28]

Many questions remain unanswered concerning this link between PACs, AF, stroke, and increased mortality. One is whether treatment of patients with a high burden of PACs with antiarrhythmic medications or by means of catheter ablation reduces the risk of developing AF, thereby reducing stroke risk and decreasing mortality. Another issue is whether there is a clinically important cut-off for what abnormal PAC burden should be. Specific definitions of excessive supraventricular ectopic activity are lacking. As this question remains unanswered, it will also be important to do further research to define the day-to-day variability in PAC frequency and what the optimal screening test should be. At the present time, a 24-h Holter monitor is the 'gold standard' for assessing PAC frequency. According to Gladstone *et al.*, excessive ectopic activity was present when PACs burden >500 PACs/day was observed on Holter monitoring. The probability of AF increased from less than 9% among patients with PACs burden <100/24 h to over 40% in those with a PAC burden of >1500/24 h).[29] In this consensus document, we have chosen to accept that a high burden of PACs exists when they exceed 500 in 24 h.

Yet another important but unresolved question concerns the use of anticoagulation. It stands to reason that patients with an increased stroke risk profile who have a certain frequency of PACs could benefit from anticoagulation. However, the benefit/risk of such an approach requires testing in clinical trials. Regarding the significance of asymptomatic vs. symptomatic PAC in this setting, there is simply lack of data and knowledge.

A summary of studies on PACs and clinical consequences is provided in Supplementary material online, *Table S1*.

Consensus statement	Symbol	References
Patients with a high PAC burden (>500/24 h) on Holter monitor should be considered at increased risk for developing of AF and be educated on the symptoms of AF. They should undergo further evaluation for possible AF including more detailed or prolonged rhythm monitoring		[19–21], Expert consensus
Comprehensive cardiovascular risk factor modification is recommended for patients with a high PAC burden including careful control of hypertension, weight loss, and screening for sleep apnoea. In addition, evaluation for structural heart disease should be considered in selected cases.		Expert consensus
When brief episodes of AF, which *per se* would not be an indication for oral anticoagulation (OAC), are observed, the burden of PACs (>500 PACs/24 h or any episode of runs of more than 20 PACs) could add to the decision process whether anticoagulation therapy should be initiated. This decision should always be made on an individual basis.		Expert consensus
Low to moderate PAC burden without documented AF is not an indication for oral anticoagulation		Expert consensus

AF, atrial fibrillation; PAC, premature atrial contraction.

Asymptomatic ventricular pre-excitation

The prevalence of ventricular pre-excitation, also termed a delta wave or Wolff–Parkinson–White (WPW) pattern on an electrocardiogram (ECG), is estimated to be 0.1–0.3%.[30,31] The lifetime risk of SCD in symptomatic WPW syndrome has been estimated at 3–4%.[32,33] Consequently, there has been a general agreement that symptomatic pre-excitation is a Class I indication for an electrophysiology study (EPS) with a view to catheter ablation of the accessory pathway.

Individuals with asymptomatic pre-excitation, however, have a lower lifetime risk of SCD, and this has varied between 0 and 0.6% in different studies.[32–34] Thus, the approach to individuals with asymptomatic pre-excitation is not as straightforward as in those that are symptomatic and has continued to be an important topic of discussion over the past decades. One of the key issues debated has been whether or not to attempt to invasively risk stratify with an EPS and ablate the accessory pathway in those at perceived increased risk of SCD. Back in 2003, the joint AHA, ACC, and ESC guidelines did state that the positive predictive value of EPS was too low to justify routine use in asymptomatic patients.[35] However, this topic continues to be controversial and is far from being resolved.

An initial evaluation of the patient with asymptomatic pre-excitation could include an exercise stress test and/or a 24-h Holter monitor looking for an accessory pathway block with increasing heart rate and intermittent accessory pathway conduction over the 24 h. Both are indicative of a long effective refractory period (ERP) of the pathway. The individual with intermittent pre-excitation during sinus rhythm is generally considered to be at very low risk for SCD.

On the other hand, high-risk features for increased risk of SCD in patients with ventricular pre-excitation include a young age,[36] inducibility of atrioventricular tachycardia (AVRT) during EPS,[37] a short antegrade ERP of the accessory pathway (≤250 ms),[36–38] and multiple accessory pathways[37,39,40] (*Table 2*). There have been suggestions that high adrenergic states, exercise, or emotion might lead to more rapid conduction over the accessory pathway.

A few randomized studies have been performed in an attempt to evaluate the risk of sudden death in patients with suggested high-risk EPS features. In a rather small study of 73 patients, none of those who had an ablation experienced AF or VF during follow-up. However, 43% of the control patients had AVRT, 14% had AF and there was 1 aborted VF in a 22-year-old male with multiple accessory pathways.[41] In a similar randomized study of 60 children with high-risk EPS features, during follow-up 7/27 control patients had AF and there was 1 sudden death in a patient who had first presented with AF but whose parents had declined ablation. There were no patients in whom VF was the initial presentation.[42] In a meta-analysis, Obeyesekere *et al.*,[43] evaluated 20 studies including a total of 1869 patients with a mean age of 7 to 43 years. Ten SCDs occurred during 11 722 person-years of follow-up. In this analysis, seven of the studies originated from Italy and reported nine SCDs. The overall SCD risk was 1.25 per 1000 person-years, with children having a higher risk (1.93 vs. 0.86 per 1000 person-years, $P = 0.07$). There were 156 AVRTs in 9884 person-years of follow-up from 18 studies with a risk of 16 per 1000 person-years follow-up. The authors concluded that the low incidence of SCD and AVRT argued against routine EPS in most asymptomatic individuals with WPW.

Pappone *et al.*,[44] recently reported an 8-year single-centre registry data experience including 2169 patients undergoing ablation for ventricular pre-excitation including both symptomatic and asymptomatic patients. In the 1001 patients who did not have ablation, VF occurred in 1.5% of patients, virtually exclusively (13 of 15) in children (median age 11 years), and was associated with a shorter accessory pathway antegrade ERP and AVRT initiating AF but not with symptoms. In the ablation group, ablation was successful in 98.5%, and no patients developed malignant arrhythmias or VF over the 8 years of follow-up. The authors concluded that the prognosis of the WPW syndrome depended on intrinsic electrophysiological properties of the accessory pathway rather than on symptoms.

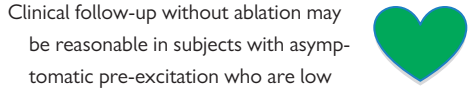
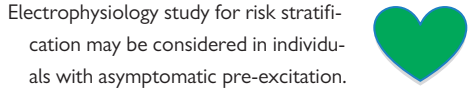
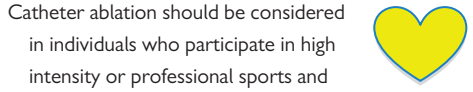
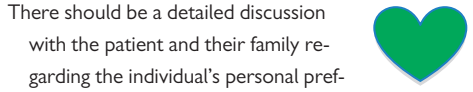
Table 2 High risk features of an antegrade accessory pathway

- Young age
- Effective refractory period of the accessory pathway <240 ms (>250 b.p.m.)
- Inducibility of atrioventricular reentrant tachycardia at EPS
- Multiple accessory pathways

b.p.m., beats per minute; EPS, electrophysiology study; ms, milliseconds.

Discussions on this subject have so far failed to reach a clear consensus. It is of importance to this discussion to understand that ablation can be performed with exceedingly low risk in the modern era and as an example there was only one major complication reported in the above registry in 2169 patients.[44] In a systematic review on risk stratification for arrhythmic events in patients with asymptomatic pre-excitation for the 2015 ACC/AHA/HRS guidelines on SVT, it was concluded that the existing evidence suggests risk stratification with an EPS of patients with asymptomatic pre-excitation may be beneficial, along with consideration of accessory-pathway ablation in those deemed to be at high risk of future arrhythmias.[45] However, given the clear limitations of the existing data, there is need for well-designed and well-conducted studies.

The more recent EHRA guidelines on SVT state that EPS for risk stratification may be considered in individuals with asymptomatic pre-excitation.[46] We would add that this may be strongly considered in those that are professional athletes or have an occupation risk such as pilots or heavy machinery operators. It may also be taken in to account that the presence of a delta wave might exclude individuals, including school children, from important activities such as exercise and sports. Catheter ablation may be considered in asymptomatic individuals with high-risk features, (antegrade ERP of the accessory pathway <240 ms, inducible AVRT triggering pre-excited AF and multiple accessory pathways). Observation without treatment may be reasonable in those with asymptomatic pre-excitation who are low risk either due to intermittent delta wave or an EPS not demonstrating high-risk features.

Consensus statements	Symbol	References
Clinical follow-up without ablation may be reasonable in subjects with asymptomatic pre-excitation who are low risk either due to intermittent delta wave or an electrophysiology study not demonstrating high-risk features.		45
Electrophysiology study for risk stratification may be considered in individuals with asymptomatic pre-excitation. Catheter ablation may be considered in asymptomatic individuals with high-risk features, (antegrade ERP of the accessory pathway <240 ms, inducible AVRT triggering pre-excited AF and multiple accessory pathways).		46
Catheter ablation should be considered in individuals who participate in high intensity or professional sports and those with an occupational risk.		46
There should be a detailed discussion with the patient and their family regarding the individual's personal preference and willingness to accept risk, whether from an ablation or from an untreated asymptomatic WPW.		33

AF, atrial fibrillation; AVRT, atrioventricular tachycardia; ERP, effective refractory period; WPW, Wolff–Parkinson–White.

Atrial fibrillation and flutter

Asymptomatic AF usually refers to AF that is incidentally discovered during routine clinical examination or detected by screening and recorded for ≥30 s via surface ECG method(s)[5,6,47] (*Table 3*). These patients with asymptomatic AF detected by surface ECG are usually thought to have a higher arrhythmia burden, sufficient to be detected by single-point or intermittent recording using ECG/Holter/loop recorders, compared with subclinical AF detected by implanted devices providing continuous monitoring.[48,49]

The true prevalence of asymptomatic AF is unknown.[5,6] Reported rates vary from 10% to 40%, depending on the risk profile of the evaluated cohort, monitoring intensity and follow-up duration, but a greater likelihood of asymptomatic AF has been consistently observed among the elderly, males and those with non-paroxysmal AF.[2,50–60] Symptomatic patients (especially those managed using a rhythm-control strategy) may also have episodes of silent AF, particularly after AF catheter ablation.[61] Indeed, in patients implanted with a cardiac monitor before ablation, a post-ablation setting was the strongest independent predictor of asymptomatic AF episodes.[62] Since the absence of symptoms may be misleading, a solely symptom-based assessment of AF burden or ablation success is usually inaccurate. On the other hand, ablation of AF is usually performed to treat excessive or recurrent symptoms, in which case absence of symptoms may be more than welcome by the patient.

Although the presence of AF symptoms may not be driven only by concomitant cardiac and non-cardiac conditions but also by patient-related psychological and somatic factors,[63,64] available data suggest that asymptomatic AF could portend a less favourable prognosis, with greater morbidity and mortality than symptomatic AF (*Table 4*), possibly due to a later referral for thrombo-embolic risk stratification and therapeutic intervention.

Management of asymptomatic AF patients generally should be based on the same principles as symptomatic patients.[47,66–68] An integrated approach such as the ABC pathway—Avoid stroke with Anticoagulation (optimize stroke prevention), Better symptom management (patient-centred symptom directed use of rate or rhythm-control strategies), and Cardiovascular and comorbidity risk factor management (*Figure 1*) summarizes key components of AF management and can help align AF management among healthcare specialties.[70] While symptom management may not be immediately relevant in asymptomatic individuals with AF, steps to try to prevent longstanding AF or decrease the risk of developing tachycardia-induced cardiomyopathy (TICMP) are important. A trial of rhythm control in asymptomatic persistent AF patients may help discern true asymptomatic AF from symptomatic AF. There are no randomized data on treatment effects specifically in asymptomatic AF, but benefits at least similar to those seen in symptomatic AF can be assumed, at least concerning anticoagulation and rate control strategies.[5,47,68]

Table 3 Detection of asymptomatic AF: clinical setting, screening methods, and screening tools

Detection of asymptomatic AF					
Clinically detected		**Screen-detected**			
Clinical setting	• Clinical visit for other reasons (e.g. acute illness, cardiovascular risk factor management, and regular follow-up visit) • Preparation for surgery or an invasive intervention • Self-detected by home BP measurement or pulse checking	Screening methods	• Pulse check • Opportunistic screening • Screening of a pre-defined population at increased risk of AF (e.g. the elderly, post-stroke patients) • Community screening of all subjects living in a specific area • Systematic screening of the population	Screening tools	• Clinical (patient history, risk scores, pulse checking, and BP measurement) • Single-lead ECG (electrical stick, monitor, monitoring patch, and watch-like recorder) • Multi-lead ECG (Holter monitoring, and multielectrode belt) • Loop recoder
Detection of subclinical AF and AHREs					
Clinical setting	• Patients implanted with a CIED (e.g. anti-bradycardia PM, and ICD) for other reasons • Patients implanted with a cardiac monitoring device due to symptoms suggestive of an arrhythmia, post-syncope, etc.	Screening methods	• Opportunistic screening in patients implanted with a CIED for other reasons • Targeted screening for AF in patients at increased risk of AF (e.g. post an embolic stroke—ESUS)	Screening tools	• Pacemaker • ICD • Implantable loop monitor • Telemetry of an ICM

Asymptomatic AF refers to AF diagnosed by conventional means while subclinical AF is used to denote AF diagnosed by implantable devises only.
AF, atrial fibrillation; AHRE, atrial high-rate episodes; BP, blood pressure; CIED, cardiac implantable electronic device; ECG, electrocardiography; ESUS, embolic stroke of undetermined aetiology; ICD, implantable cardioverter-defibrillator; ICM, implantable cardiac monitor; PM, pacemaker.

Table 4 Baseline characteristics and outcomes in asymptomatic AF patients: *post hoc* analyses of RCTs and observational studies

Study/*post hoc* analysis (publication date) Study type	AFFIRM (2005)[53] RCT, *post hoc*	RACE (2014)[56] RCT, *post hoc*	Olmsted County (2011)[60] Retrospective	Belgrade AF (2013)[55] Single-centre, first-onset AF	UK-CPRD[a] (2014)[65] Administrative dataset	EORP-AF Pilot (2015)[58] International registry	ORBIT-AF (2016)[57] International registry	Olmsted County (2016)[2] Retrospective	Fushimi AF Registry (2017)[59] Community-based survey
Cohort size (*n*)	4060	522	4618	1100	30 260	3119	10 087	476	3749
Asymptomatic AF (%)	12	30	25	13.3	18.4[a]	39.7	38.2	33.8	52.6
Follow-up (mean) (years)	3.5	2.3 ± 0.6		9.9±6.1	≤3	1	Median 1.8	Median 6.0	3.0
Baseline characteristics of patients with asymptomatic AF									
Male predominance									
Older age									
Non-paroxysmal AF									
Slower heart rate									
More comorbidity									
Higher stroke risk									
Treatment differences									
Rate control									
Rhythm control									
OAC									
Outcomes (asymptomatic AF vs. comparator[b])									
AF progression				1.6 (1.1-2.2)					
Stroke		6% vs. 7%		2.1 (1.2–3.9)	19.4 vs. 8.4[a]	23.8% vs. 29.7%	1.13 (0.87–1.46)	2.6 (1.1–6.1)	1.28 (0.82–2.01)
Mortality	1.07 (0.79–1.46)	5% vs. 8%		0.8 (0.4–1.9)	40.1 vs. 20.9[a]	9.4% vs. 4.2%	1.00 (0.86–1.16)	4.0 (2.3–6.9)	1.71 (1.31–2.29)
MI					9.0 vs. 6.5[a]		1.05 (0.72–1.53)		
Heart failure		0% vs. 6%		0.7 (0.4–1.1)					0.96 (0.65–1.44)
Dementia									
Major bleeding		4% vs. 4%			7.7 vs. 4.0[a]		1.21 (1.02–1.45)		1.18 (0.74–1.90)

more common in asymptomatic AF;
no difference;
less common in asymptomatic AF;
greater risk of worse prognosis;
not reported.

[a]Patients incidentally diagnosed with AF were compared to matched non-AF controls; otherwise, the comparator was symptomatic AF.
[b]Outcomes presented as crude incidence rates per 1000 patient-years; otherwise, hazard ratios (95% confidence interval) or event rate, where reported.
AF, atrial fibrillation; AFFIRM, Atrial Fibrillation Follow-up Investigation of Rhythm Management; RACE, Rate Control vs. Electrical cardioversion for persistent atrial; RCT, Randomized Clinical Trial.

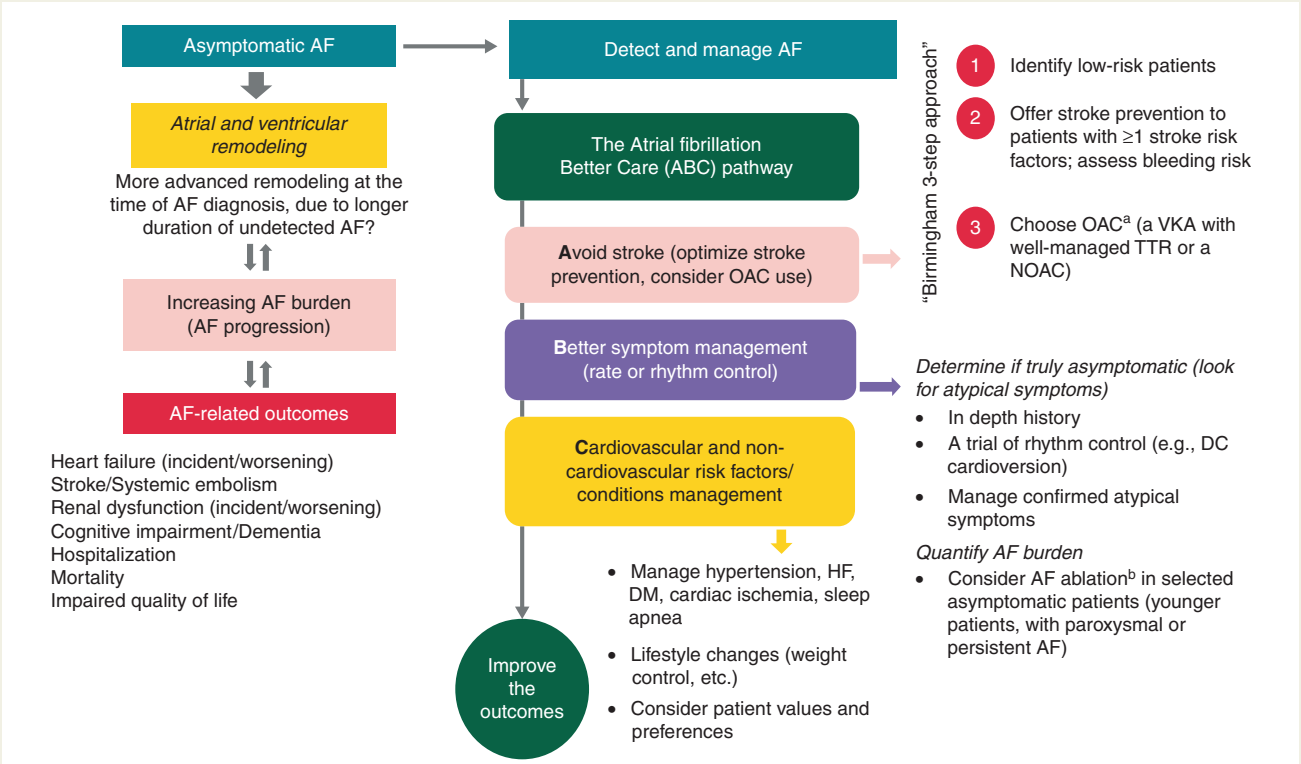


Figure 1 The Atrial fibrillation Better Care (ABC) pathway depicting some key components of AF management. [a]To aid the choice between VKAs and NOACs, the SAMeTT2R$_2$ score, assigning 1 point each to female sex, age of <60 years, history of two or more co-morbidities (i.e. hypertension, diabetes mellitus, coronary artery disease/myocardial infarction, peripheral arterial disease, congestive heart failure, previous stroke, pulmonary disease, and hepatic or renal disease) and treatment with drugs interacting with VKAs (e.g. amiodarone) and 2 points each for current or recent tobacco use and non-Caucasian ethnicity, can be used. A score of >2 is predictive of poor TTR, all-cause mortality and composite endpoint of thromboembolism, major bleeding, and mortality.[69] bSeveral ongoing randomized studies are investigating the effects of rhythm control using AF ablation on AF-related outcomes: The CABANA (Catheter Ablation versus Anti-arrhythmic Drug Therapy for Atrial Fibrillation) trial is testing the hypothesis that AF ablation is superior to rate or rhythm control drug therapy for reducing the incidence of the composite endpoint of all-cause mortality, disabling stroke, serious bleeding or cardiac arrest (NCT009911508); The EAST (Early Treatment of Atrial Fibrillation for Stroke Prevention) trial is comparing an early, structured rhythm control strategy based on antiarrhythmic drugs and catheter ablation versus usual care for the prevention of AF-related complications (NCT1288352); in the OAT (Oral Anticoagulation Therapy) Study, AF patients with a CHADS$_2$ score of ≥2 or a CHA$_2$DS$_2$-VASc of ≥3 are randomized to OAC or no OAC at 3 months after successful AF ablation (NCT01959425). AF, atrial fibrillation; DC, direct current; NOAC, non-vitamin K antagonist oral anticoagulants; OAC, oral anticoagulant therapy; TTR, time in therapeutic range; VKA, vitamin K antagonist.

Oral anticoagulant therapy (OAC) using either vitamin K antagonists (VKAs) or the non-vitamin K antagonist oral anticoagulants (NOACs), dabigatran, rivaroxaban, apixaban, or edoxaban effectively reduces stroke, systemic embolism, and mortality in AF patients at increased risk of stroke.[71,72] Compared to VKAs, NOACs exert broadly similar efficacy, but are safer with less intracranial bleeding, and more convenient for long-term use.[72,73] The decision to use OAC for thromboprophylaxis in AF patients depends on the presence of CHA$_2$DS$_2$-VASc stroke risk factors, but not on arrhythmia-related symptoms.[47,68,70] In observational studies of asymptomatic AF, OAC use vs. no therapy has been associated with a significant reduction in stroke and mortality,[65] and residual stroke risk was similar among anticoagulated AF patients and matched non-AF controls.[74] Good long-term adherence to OACs[75] may be particularly challenging in asymptomatic patients but in a study of screening detected AF, the 5-year adherence to OAC was 88% and stroke rates significantly declined.[76]

Whether OAC can be stopped after an AF ablation procedure is uncertain, especially since AF recurrences are common, and may be asymptomatic. Hence current guidelines recommend continuation of OAC in the presence of stroke risk factors, irrespective of the apparent success of rhythm-control interventions.[77] The OCEAN trial is an ongoing multicentre randomized controlled trial evaluating two antithrombotic treatment strategies (rivaroxaban vs. aspirin) for patients with risk factors for stroke after apparently successful AF ablation.[78]

With the greater availability of screening tools, asymptomatic individuals will be increasingly diagnosed with paroxysmal AF that would be missed by a routine ECG/Holter recording.[5,6,76] The incremental burden of AF, reflected by the clinical types of AF (from paroxysmal, to persistent and to permanent AF), has been associated with increasing risk of stroke in *post hoc* analyses of randomized clinical trials,[79–87] AF registries,[88–91] and a meta-analysis of 12 studies.[92] Although generally lower in paroxysmal AF compared to non-

paroxysmal AF (*Figure 2A and B*), the annual stroke rates among non-anticoagulated patients with paroxysmal AF and ≥1 CHA_2DS_2-VASc stroke risk score[79–81] (*Figure 2A*) is sufficiently high to merit OAC use.[47,68] Of note, major bleeding rates among anticoagulated AF patients were broadly similar across AF types[82–85,92] (*Figure 2C*). Incremental AF burden has been also associated with increased risk of TICMP,[93,94] heart failure (HF),[55,95–98] cognitive impairment/dementia,[99,100] and mortality.[82–85,92]

Asymptomatic AF has been independently associated with greater risk of progression than symptomatic arrhythmia (HR 1.6, 95% CI 1.1–2.2).[55] Five and 10 years after detection, paroxysmal/persistent asymptomatic AF progressed to permanent AF in 25% and 50% of patients, respectively.[55,76] Increasing evidence shows that comprehensive risk factor management (e.g. blood pressure lowering,[101–103] weight reduction,[104–106] glucose control,[107] and treatment of obstructive sleep apnoea[108]) and lifestyle modification (e.g. physical exercise and cardiorespiratory fitness,[109–112] stress management[113,114]) could decrease the burden of AF.[115–117] These interventions have not been investigated specifically in asymptomatic AF patients, but their benefits would likely be similar to those seen in symptomatic patients.[118]

A decrease in AF burden after successful AF catheter ablation could reduce the risk of major AF-related outcomes including HF, stroke, and mortality, as suggested by many observational studies evaluating mostly highly symptomatic patients.[119–133] However, observational data have numerous limitations,[134] and undertaking AF ablation to abolish the need for long-term OAC is presently not recommended.[47,68,77] The effects of rhythm control using AF ablation are currently being explored in several ongoing randomized outcome studies [e.g. CABANA (NCT00911508), EAST (NCT01288352), and OAT (NCT01959425)].

In the CASTLE-AF randomized study, AF ablation yielded a 47% mortality rate reduction compared with conventional rhythm control among symptomatic anticoagulated AF patients with HF treated with an implantable defibrillator (predominantly middle-aged males with moderate LV dysfunction) over a median 3-year follow-up.[135] In the CABANA trial,[136] there was no significant difference between catheter ablation compared to medical therapy for the primary outcome of composite of death, disabling stroke, serious bleeding, or cardiac arrest, although symptoms were improved in the ablation arm. The results of the CABANA trial, although presented, have yet to be published.

Whether (and how) asymptomatic AF patients could benefit from AF ablation still needs to be established. Incidental diagnosis of AF may trigger symptoms in susceptible patients as they become aware of a heart condition,[137] and even failed AF ablation may have a placebo effect in such patients.[138] A challenge with rhythm control (e.g. using electrical cardioversion) could identify apparently asymptomatic patients who had subconsciously adapted to AF by restricting their lifestyle or have atypical symptoms.[77,137] In many cases, a trial with an antiarrhythmic drug might be considered after cardioversion before considering ablation. Whereas these patients would likely experience symptomatic improvement after successful AF ablation,[138] a failed procedure may turn truly asymptomatic patients into symptomatic due to post-procedural atrial tachyarrhythmias (such a scenario has been reported in 24–34% of patients).[139,140]

The decision to perform an AF ablation in asymptomatic patients should be a shared informed process that considers not only potential benefits (pending further evidence from randomized studies), but also the risk of serious procedure-related complications (≤4%) and patient's values and preferences for treatment and outcomes[63] (*Figure 1*). Notwithstanding that the exact AF duration before diagnosis is difficult to establish in asymptomatic patients, AF ablation in such patients may be considered in selected younger patients with paroxysmal or persistent (but not long-term persistent) AF (Class IIb, Level of evidence C).[77]

Most of the management principles for AF also apply to atrial flutter. While the risk of thromboembolism is sometimes reported to be slightly less, the same indications exist for anticoagulation. Atrial flutter has, however, been studied to a lesser extent than AF. Very little is known specifically about asymptomatic atrial flutter but treatment options would include initial rate control and consideration of cardioversion and/or ablation.

Consensus statements	Symbol	References
Patients with asymptomatic AF should be anticoagulated, according to their calculated risk of stroke, equal to patients with overt AF.		47,66–68
Consideration should be given to screening high-risk individuals e.g. patients with a CHA_2DS_2-VASc score ≥ 2 for AF.		Expert opinion
Lifestyle changes should be advised in patients with asymptomatic AF, as in patients with overt AF.		47,66–68
Cardioversion of persistent AF in asymptomatic patients may be advised to differentiate between truly asymptomatic patients or those adapted to AF-related symptoms.		77,137
Rate control drugs should be prescribed to patients with asymptomatic AF with fast AV conduction in order to attempt to decrease risk of tachycardia-induced cardiomyopathy.		93,94
Ablation might be proposed to selected patients with asymptomatic AF, based on patient's preferences, after detailed informed consent.		77, Expert opinion

AF, atrial fibrillation; AV, atrioventricular.

Atrial high-rate episodes

Atrial high-rate episodes (AHREs), also sometimes termed subclinical AF, are different from symptomatic or asymptomatic AF essentially by the way they are noted. The definitions of AHREs do vary slightly

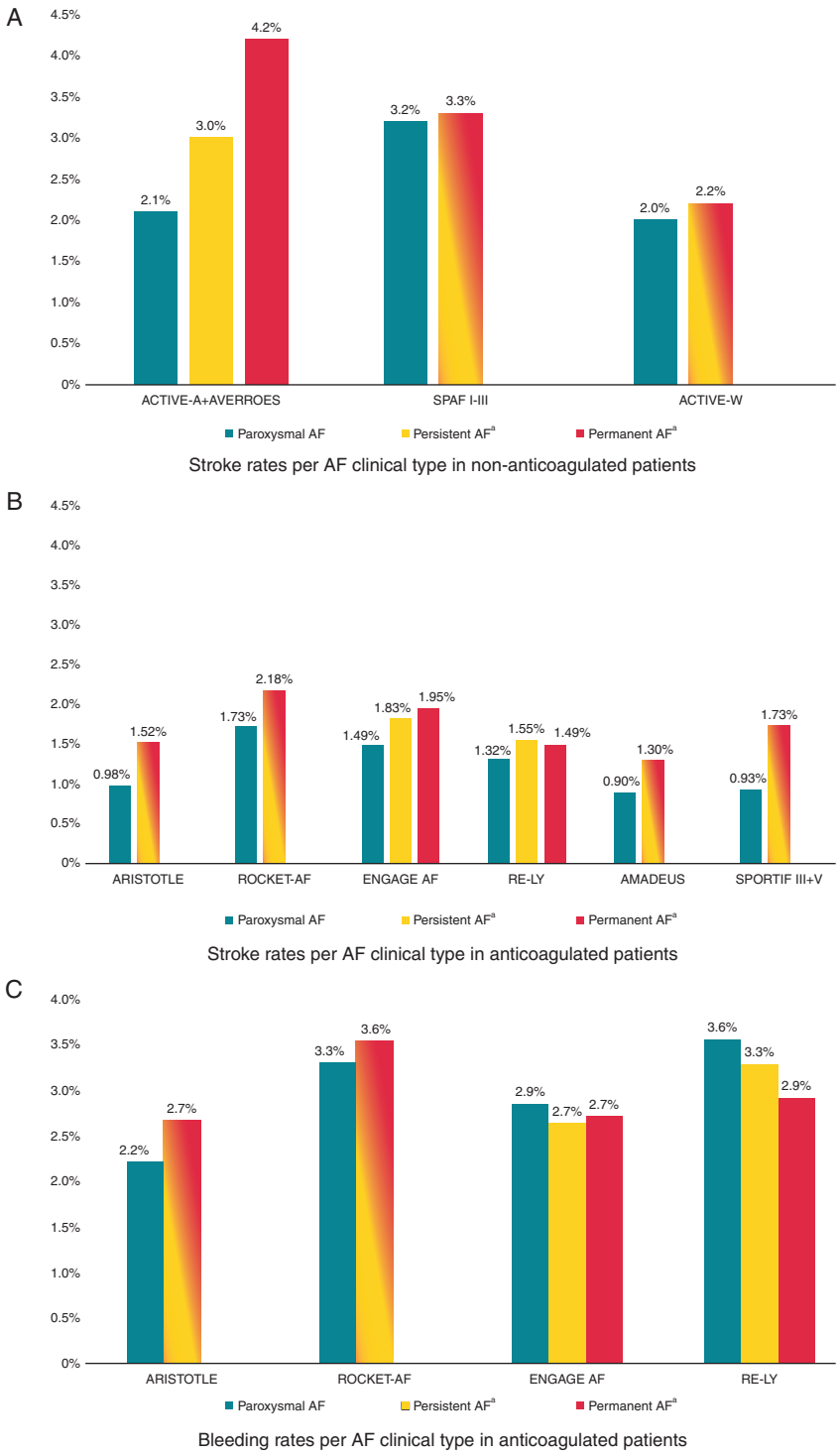


Figure 2 Annual stroke rates across the clinical AF types in non-anticoagulated (*A*) and anticoagulated (*B*) patients with AF and major bleeding rates per AF clinical type in patients taking OAC (*C*). (*A*) Stroke rates per AF clinical type in non-anticoagulated patients. (*B*) Stroke rates per AF clinical type in anticoagulated patients. (*C*) Bleeding rates per AF clinical type in anticoagulated patients. [a]Event rates are reported jointly for persistent and permanent AF (also shown as gradient bar). Dotted line shows the threshold for (N)OAC use. Active-A/W, Atrial Fibrillation Clopidogrel trial With irbesartan for Prevention of Vascular Events Aspirin/Warfarin; AVERROES, Apixaban Versus Acetylsalicylic Acid to Prevent Stroke in Atrial Fibrillation Patients Who Have Failed or Are Unsuitable for Vitamin K Antagonist Treatment; SPAF, Stroke Prevention in Atrial Fibrillation; ARISTOTLE, Apixaban for Reduction in Stroke and Other Thromboembolic Events in Atrial Fibrillation; ROCKET-AF, Rivaroxaban Once Daily Oral Direct Factor Xa Inhibition Compared With Vitamin K Antagonism for Prevention of Stroke and Embolism Trial in Atrial Fibrillation; ENGAGE AF, Effective Anticoagulation With Factor Xa Next Generation in Atrial Fibrillation; RE-LY, Randomized Evaluation of Long-Term Anticoagulation Therapy; AMADEUS, Evaluating the Use of SR34006 Compared to Warfarin or Acenocoumarol in Patients With Atrial Fibrillation; SPORTIF, Stroke Prevention using Oral Thrombin Inhibitor in Atrial Fibrillation.

from study to study.[6,47,141] In the ASSERT study, AHREs were defined as episodes of at least 5 min of atrial rate >180 b.p.m., detected by the continuous monitoring by cardiac implantable electronic devices (CIEDs),[6] while AF was defined by an episode lasting at least 30 s on an ECG with irregular RR intervals with no discernible, distinct P waves.[47] This can be either in the presence (overt AF) or absence (asymptomatic AF) of symptoms typically associated with AF (i.e. palpitations, shortness of breath, light-headedness, chest pain, pre-syncope, or syncope).

Both AHREs and AF can thus be asymptomatic. Contrary to asymptomatic AF, which can be diagnosed in any patients using any kind of diagnostic tool [ECG, Holter monitoring, event recorder, and implantable loop recorder (ILR)], AHREs are only diagnosed in patients with CIEDs. There are different algorithms from different manufacturers to detect these AHREs, albeit with variable accuracy. Not all implanted devices are able to document AHREs by providing intracardiac electrograms. The positive predictive value of various atrial rate and episode length was tested in the ASSERT population.[142] Inappropriate detection of AHREs by review of device-based arrhythmia detection counters, and by comparison to stored electrograms, was only seen in 10–17% of the >6 min episodes and 1–2% of the >24 h episodes. To what extent AHREs can be considered as an early stage of AF is not known.

The prevalence of AHREs in patients with CIEDs has been reported to range between 30% and 60%.[143] The TRENDS study showed a doubling of risk of thrombo-embolic events in the presence of >5.5 h AHREs over a 30-day period.[144] The ASSERT trial detected AHREs in 10.1% of patients over the first 3 months after pacemaker implantation.[141] In these patients with AHREs, the risk of developing overt AF was 5.6 times higher (95% CI 3.78–8.17, $P < 0.001$) and the embolic risk was 2.5 times higher (95% CI 1.28–4.85, $P = 0.007$) over a follow-up period of 2.8 years. This increased stroke risk, stratified according to the $CHADS_2$ score, was however smaller than expected in patients with comparable $CHADS_2$ scores with overt AF: an annual stroke risk of 0.6% for AHREs vs. 2.8% for AF in $CHADS_2 = 1$ patients, 1.29% for AHREs vs. 4.0% for AF in $CHADS_2 = 2$ patients, and 3.8% for AHREs vs. >5.9% for AF in $CHADS_2 \geq 3$ patients.[145] As mentioned previously, there was no temporal relationship between stroke and AHREs[146] which begs the question of whether AHREs are causal or perhaps only a risk marker of an atrial cardiomyopathy as discussed in the section on PACs. Longer episodes of AHREs (>24 h) were associated with the highest risk of ischaemic stroke or systemic embolism.[141]

The only published study assessing anticoagulation of patients diagnosed with AHREs was the IMPACT trial, in patients with remote monitoring of implantable cardioverter-defibrillators (ICDs) or cardiac resynchronization therapy (CRT), without history of stroke or documented AF.[147] There was no difference in primary outcomes (stroke, systemic embolism, major haemorrhage, and mortality) between the intervention and control arms when oral anticoagulation was decided according to the $CHADS_2$ score and to the recurrence or not of AHRE episodes between two control visits. Therefore, it should be emphasized that there are no data yet to indicate whether anticoagulation for short AHREs is beneficial or not. The ARTESIA and NOAH-AFNET 6 trials are currently ongoing to test the effect of NOACs in CIED patients with 6 min to 24 h of AHRE and with additional risk factors, but without documented AF.[148,149] The results of these studies are expected in 2021. No studies to date have suggested any benefit of any rhythm-control strategy, including any antiarrhythmic drug or ablation, in these asymptomatic patients.

Consensus statements	Symbol	References
Patients with AHRE should be considered as having a higher stroke risk compared with patients without AHRE.		141,144
The benefit of anticoagulating patients with AHREs and additional stroke risk factors, but without clinical AF, is still unclear. Patients with AHRE should be referred for further detailed evaluation unless AF is already fully confirmed by stored electrograms.		141,148,149, Expert consensus
Anticoagulation might be considered on an individual basis in some patients with AHREs and a CHA_2DS_2-VASc score ≥2.		Expert consensus
AHREs *per se* do not require antiarrhythmic treatment.		Expert consensus

AF, atrial fibrillation; AHRE, atrial high-rate episode.

Premature ventricular contractions

Isolated and sparse PVCs are a normal occurrence in most individuals. A few to multiple PVCs can be seen on most 24-h Holter monitors, including those from healthy young individuals.[150] These PVCs usually originate from different locations in both right and left ventricles. They may result from focal activity or, less likely, be due to a (micro) re-entrant mechanism. In some individuals, however, a higher number of PVCs may be present. Frequent PVCs may be a marker for underlying abnormal cardiac substrate. This may be the result of underlying electrical, ischaemic, or structural alterations, leading to enhanced automaticity (e.g. in chronically ischaemic tissue), triggered activity [e.g. in long QT syndrome (LQTS), or by drugs such as digoxin], or re-entrant mechanisms (e.g. in post-infarction patients).

Underlying cardiac disease is a prognostically unfavourable marker in asymptomatic patients with PVCs and requires a specialist approach to address the potential prognostic impact. Premature ventricular contraction characteristics, such as a high burden, a more complex presentation (e.g. couplets, triplets, or non-sustained runs), multifocal origin, and/or increasing PVC frequency with exercise, should all alert to potential underlying electrical, ischaemic, or

Table 5 Factors that may point to worse prognosis in patients with PVCs

- Underlying structural, ischemic or electrical disease
- More than 2000 PVC/24 h
- Complex PVCs (couplets, triplets, and non-sustained VT)
- Increasing number of morphologies
- Increasing number PVCs with exercise
- Non-outflow tract PVC (usually monomorphic or only slightly divergent morphologies)
- Short coupling interval of the PVCs ('R-on-T')
- PVCs with broader QRS complexes (more frequently related to cardiomyopathy)

These factors may suggest a poorer prognosis in individudals with PVCs and need a thorough investigation to rule out underlying structural, ischaemic, or electrical disease. The additional evaluations should be individually tailored, in analogy with the flowchart in *Figure 3*.
PVC, premature ventricular contractions; VT, ventricular tachycardia.

structural alterations that may be associated with the undesired outcome of major ventricular arrhythmias or sudden death (*Table 5*).[151,152] There is no absolute threshold of the number of PVCs that can be used as a cut-off for underlying disease and hence should trigger further investigations. A study in apparently healthy athletes has shown that in those with >2000 PVC per day, there was a 30% risk of finding underlying heart disease.[153] Even in the absence of demonstrable underlying disease, a moderate to high burden of PVCs are a marker for all-cause and cardiovascular mortality, indicating that continued follow-up may be warranted.[154]

The morphology of the PVCs can provide important additional information in this diagnostic conundrum, since some predilection sites of benign PVC ectopy are well recognized. The most prevalent entity in this respect are PVCs originating from the ventricular outflow tract regions, showing a clear inferior axis with high voltages in the inferior limb leads (usually with a combined amplitude of >4.5 mV of the QRS complexes in leads II, III, and aVF). Most frequently, these PVCs originate from the right ventricular outflow tract (RVOT), in which case they have a left bundle branch morphology in V1 (i.e. a dominant negative QRS complex), with transition between V3 and V4. Earlier transition, and certainly when V1 shows a right bundle branch morphology, suggests a left-sided origin, which may be within the coronary cusps of the aorta, or in the endocardium or epicardium of the left ventricular outflow tract (LVOT).[155] These PVCs are thought to be the result of triggered activity, i.e. a local cellular cause which in most cases has no serious prognostic implications. Therefore, the PVCs are usually strictly unifocal, but slight morphological changes are often seen, attributed to different exit points of the ectopic activity. Although these RVOT/LVOT arrhythmias usually occur in structurally normal hearts, they may rarely be an atypical expression of arrhythmogenic (right) ventricular cardiomyopathy.[156]

The absence of imaging abnormalities on an echocardiogram and cardiac magnetic resonance imaging (MRI) can help to rule out structural heart disease in such patients. The demonstration of PVCs of differing morphologies from the right ventricle (RV) in patients with normal LV function should prompt investigations to rule out arrhythmogenic cardiomyopathy with right ventricular dominance or sarcoidosis.[157] Similarly, multifocal PVCs of LV origin should trigger investigations for non-ischaemic cardiomyopathy.

Other less common locations of focal PVC are around the mitral or tricuspid annulus. These PVCs are strictly unifocal again but have a superior axis with LBBB or RBBB morphology. Locations away from the annulus are usually related to PVC originating from the His-Purkinje system. Finally, intramyocardial foci may occur, often related to the papillary muscles or the moderator band.[158] Some of these foci may present with a pattern of parasystole, indicating poor electrical coupling with the surrounding tissue, or generate NSVT. A strict unifocal presentation in the absence of demonstrable structural heart disease points to a benign automatic focus in such situations.

Very rarely, otherwise 'benign' PVCs may give rise to polymorphic VT or ventricular fibrillation (VF) due to their short coupling interval.[152] Short-coupled PVCs impinging on the T wave may induce polymorphic VT/VF in a setting of ischaemia, electrolyte abnormalities, underlying LQTS, or early repolarization syndrome. This may also rarely occur in a 'normal' heart, sometimes called 'short-coupled form of torsades de pointes'.[153] Often, such PVCs arise from the Purkinje network, but also other foci have been described. In such patients, the malignant electrical presentation mandates aggressive treatment, possibly by ablation. In some, an implantable defibrillator may be indicated.

Frequent PVCs (usually defined as >10–15% of the total number of beats per 24 h) can impair LV function (PVC-induced cardiomyopathy) which may be reversible with medical treatment or catheter ablation of the extrasystoles and standard HF therapy.[159,160] However, it is well recognized that not all patients with frequent PVCs will develop LV dysfunction. Factors associated with the development of LV dysfunction include: longer PVC QRS duration, epicardial PVCs, retrograde atrial activation of PVCs, and interpolation of PVCs.[161–163] The PVC burden remains one of the strongest predictors for the development of a PVC-induced cardiomyopathy, although the burden associated with cardiomyopathy varies between studies. Most studies are limited by a strong referral bias in enrolling patients who are symptomatic and referred for catheter ablation. The prevalence of LV dysfunction in these studies ranged from 7% to 52% (*Table 6*).

Asymptomatic patients with frequent PVCs were underrepresented in these studies. With these inherent limitations, there appears to be an increase in risk in the development of PVC-induced cardiomyopathy with a PVC burden >10%. However, PVC-induced cardiomyopathy has been reported in individuals with a PVC burden <10%.[166,168] A recent study measuring subtle degrees of LV impairment by using speckle tracking echocardiography (measuring LV global longitudinal strain and mechanical dispersion) showed mild impairment of myocardial function with a PVC burden >8%.[169] The wide range reported in studies may be partly explained by the single-day Holter monitoring used. A wide daily variation in PVC burden is well recognized. Longer monitoring has been shown to double the identification of patients with a PVC burden of >10%.[171] It can be difficult to determine if the PVCs are the cause or the consequence of LV dysfunction.

Table 6 Summary of studies relating PVC burden with LV dysfunction

	No. of patients with PVCs	No. of patients (asymptomatic)	No. of patients with LV dysfunction (definition)	PVC burden (no LV dysfunction)	PVC burden (LV dysfunction)	PVC burden predictive for LV dysfunction	Lowest PVC burden with LV dysfunction
Baman *et al.*[164]	174	17	57 (LVEF < 50%)	13 ± 12%	33 ± 13%	24% (sensitivity 79%, specificity 78%)	10%
Hasdemir *et al.*[165]	249	26	17 (LVEF < 50%)	8.1 ± 7.4	29 ± 9.2%	16% (sensitivity 100%, specificity 87%)	–
Munoz *et al.*[166]	70	–	17 (LVEF < 50%)	16.7 ± 13.7	29.3 ± 14.6%	15/17 had PVC burden >10%	2/17 had PVC burden <10%
Ban *et al.*[167]	127	7	28 (LVEF < 50%)	22 ± 10%	31 ± 11%	26% (sensitivity 70%, specificity 78%)	–
Blaye-Felice *et al.*[168]	186	–	96 (LVEF < 50%)	17 ± 12%	26 ± 12%	–	10/96 had PVC burden <10%
Lie *et al.*[169]	52	–	15 (GLS worse than -18%)	5%	22%	>8%	–
Park *et al.*[170]	180	36	52 (LVEF <50%)	28 ± 11.6%	30.7 ± 10%	26% (sensitivity 63%, specificity 87%)	–

LV, left ventricular; LVEF, left ventricular ejection fraction; PVC, premature ventricular contraction.

Two studies have reported on the natural history of PVCs. Niwano *et al.*,[172] followed 239 consecutive patients (mostly asymptomatic) with a PVC burden >1%, with normal baseline LV function, over a mean follow-up of 5.6 years. Patients were grouped into those with a high, moderate, and low PVC burden. Forty-six patients had a high PVC burden (>20%), 105 had a moderate burden (5–20%), and 88 patients had a low PVC burden (1–5%). Thirteen patients (5%) developed LV dysfunction (defined as a fall in LVEF by at least 6%) at follow-up. Patients with a high PVC burden >20% were more likely to develop LV dysfunction but this change in LVEF occurred very slowly over several years with no reported major adverse cardiac events. Dukes *et al.*,[173] followed 1139 elderly (>65 years) patients from the Cardiovascular Health Study who had Holter monitoring and were followed up with an echocardiogram 5 years later and for incident HF. They reported that a PVC burden in the upper quartile (roughly equivalent to >100 PVC/24 h) had a three-fold increase in the risk of incident HF compared to the lowest quartile. The population attributable risk of developing HF due to PVCs was 8.1%.

The risk of developing a PVC-induced cardiomyopathy rises with increasing PVC burden. Subtle degrees of LV dysfunction can be seen with a PVC burden as low as 8%. In studies where patients were referred for management of PVCs, usually because of symptoms, the predictive PVC burden to cause a PVC-induced cardiomyopathy is >10%, usually >20%. However, the vast majority of patients with frequent PVCs >10% will not go on to develop a cardiomyopathy. Studies from Niwano *et al.* and Hasdemir *et al.*, suggest a prevalence of 5–7% for patients with a PVC burden >10%.[165,172] Recently, the study by Dukes *et al.*,[173] suggested the prevalence of HF due to PVCs in the elderly population may be higher than previously reported.

A proposed scheme to evaluate patients with more than expected PVC is shown in *Figure 3*. Although Dukes *et al.*,[173] had described a cut-off of ±100 PVCs/24 h, we would suggest setting the bar at ≥500 PVCs to trigger an extensive workup for underlying disease, given the findings in athletes with a 2000 PVCs/24 h cut-off.[153] Since excluding underlying disease is a cumbersome and sometimes complex task, there is no defined set of 'minimal investigations', but conceptually three axes of evaluation need to be explored (imaging, electrical, and genetic) based on what may be clinically indicated. If the evaluation is negative in an asymptomatic subject, treatment is not required but it seems reasonable to perform serial measurements of LV function (yearly) in patients with a PVC burden >10%. In patients who develop symptoms, or who have or develop LV dysfunction, medical therapy (beta-blockers, calcium blockers with or without antiarrhythmic drug therapy), and/or catheter ablation is indicated.

There are a number of considerations that need to taken into account when deciding whether to treat asymptomatic PVCs. In the CAST study, suppression of PVCs by flecainide and encainide after myocardial infarction (MI) was found to be harmful.[174] In cases where patients have frequent PVCs but in the background of longstanding remodelled cardiomyopathy (eccentric with thinning) or with large, dense STEMI scars, the likelihood of benefit of intervention to improve LV function may be low.

Consensus statements	Symbol	References
Asymptomatic patients with frequent PVCs (>500 per 24 h) should be referred to a specialist for further evaluation to rule out any underlying structural, ischaemic, or electrical heart disease.		151,152
Very frequent PVCs (burden > 20%) are a marker of all-cause and cardiovascular mortality and may justify intensified follow-up.		154
PVCs should be treated in patients with suspected PVC-mediated cardiomyopathy.		Expert consensus
Treatment of patients with asymptomatic PVCs should focus on the underlying heart disease in order to improve prognosis.		Expert consensus

PVC, premature ventricular contraction.

Ventricular tachycardia

The definition of NSVT constitutes three or more consecutive ventricular beats at a rate of greater than 100 b.p.m. with a duration of less than 30 s. The prevalence of asymptomatic NSVT varies from 0.7% (healthy army population) to 10% (in a geriatric population) in patients without known heart disease.[175–177] On the other hand, it is common in ischaemic heart disease (30–80% of patients) during long-term ECG monitoring where it is usually asymptomatic.[178]

The mode of discovery of VT may vary, but a 12-lead ECG during the arrhythmia should be obtained whenever possible. Whereas NSVT may be asymptomatic, sustained VT is much more often symptomatic. Slow VT, generally slower than 150 b.p.m., may however be asymptomatic. When lasting for hours/days individuals with slow VT may, however, become symptomatic because of HF symptoms.

Definitions of different sub-types of VT are summarized in *Table 7*. Among ventricular arrhythmias, two distinct entities require specific mention; bidirectional VT and torsades de pointes VT. Bidirectional VT may be asymptomatic particularly in Andersen–Tawil syndrome. The classic causes are digitalis toxicity or channelopathies such as catecholaminergic polymorphic ventricular tachycardia (CPVT) or Andersen–Tawil syndrome. Torsades de pointes VT is a type of VT seen exclusively in the setting of prolonged QT interval whether it is acquired or congenital.

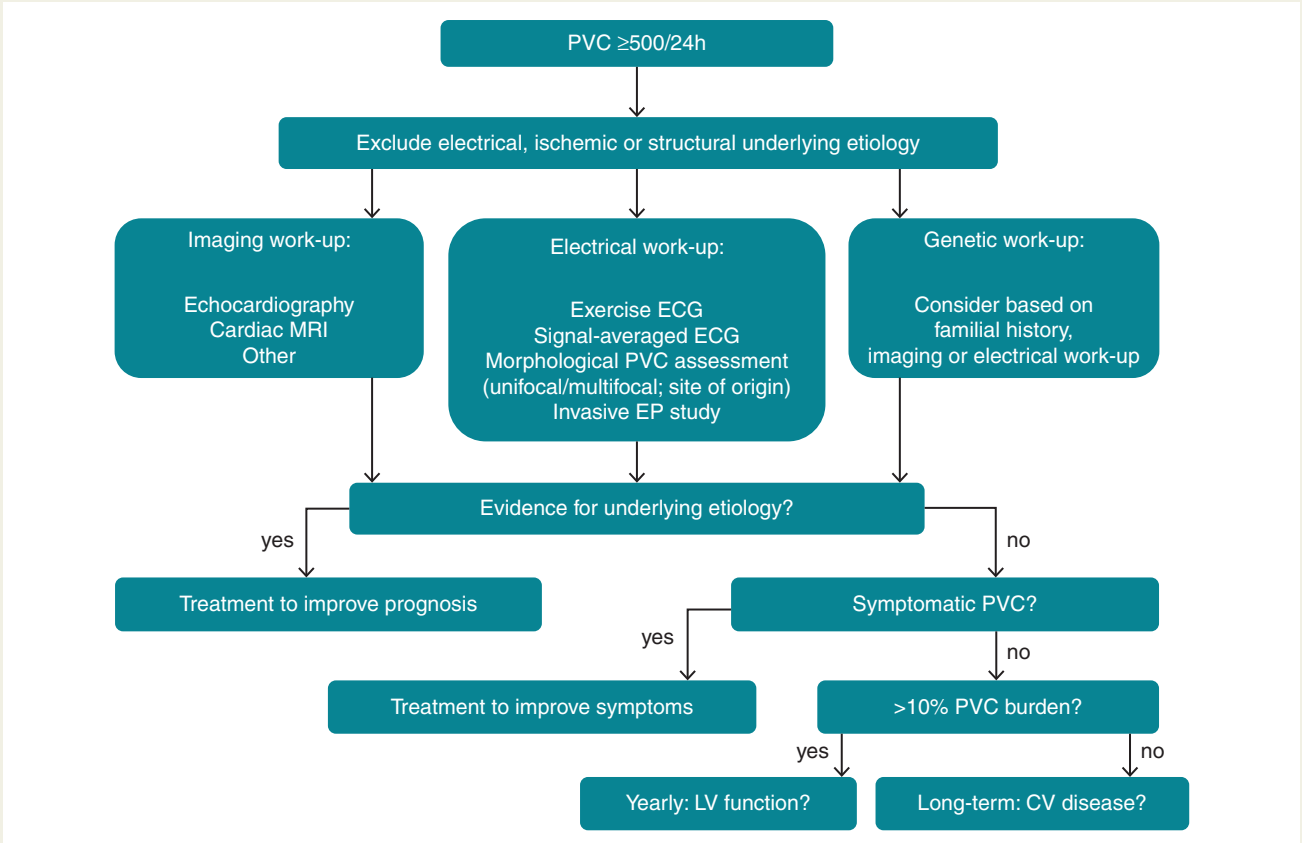


Figure 3 Evaluation of patients with >500 premature ventricular contractions per 24 h. There is no defined set of 'minimal investigations', but conceptually three axes of evaluation need to be explored (imaging, electrical, and genetic) and investigations considered on an a case to case basis. CV, cardiovascular; ECG, electrocardiography; EP, electrophysiology; LV, left ventricular; MRI, magnetic resonance imaging PVC, premature ventricular contraction.

Table 7 Definitions of different sub-types of ventricular tachycardia

Type of ventricular arrhythmia	Definition
Non-sustained VT	Three or more consecutive ventricular beats terminating spontaneously in less than 30 s with a cycle length of <600 ms (>100 b.p.m.)
• Non-sustained monomorphic VT	NSVT with a single QRS morphology
• Non-sustained polymorphic VT	NSVT with a changing QRS morphology and a cycle length between 600 and 180 ms
Monomorphic sustained VT	VT greater than 30 s in duration or terminated by external intervention with a stable QRS morphology
Bidirectional VT	VT with a beat to beat alternans in the frontal plane axis often associated with digitalis toxicity or channelopathies such as CPVT or Andersen-Tawil syndrome
Torsades de pointes	Polymorphic VT characterized by twisting of the peaks of the QRS complexes around the isoelectric line often associated with long QT. • Typical: initiation following a long/short/long coupling interval • Atypical: short coupled variant initiated by R on T PVCs
Accelerated idioventricular rhythm	Ventricular rhythm slower than 100 bpm

CPVT, catecholaminergic polymorphic ventricular tachycardia; ms, milliseconds; NSVT, non-sustained ventricular tachycardia; PVC, premature ventricular contractions; VT, ventricular tachycardia.

The goal of the evaluation is to try to identify an electrical cardiac disorder and rule out underlying heart disease, primarily coronary artery stenosis. Suggested first- and second-line evaluations are presented in *Table 8*. First-line investigations include clinical evaluation associated with a 12 lead ECG, rhythm monitoring (e.g. Holter monitor), echocardiogram, laboratory testing with or without an exercise stress test depending on the situation. Second-line investigations may include coronary artery angiography or cardiac MRI/CT scan to rule out subtle heart disease such as focal cardiomyopathy. Pharmacological testing may be considered in the absence of structural heart disease to evaluate for an inherited arrhythmic disorder. This may include ajmaline and if not available flecainide testing to uncover Brugada syndrome.[179] Other types of pharmacological testing such as epinephrine for diagnosing LQTS[180] and isoproterenol for the diagnosis of arrhythmias in ARVC have been described.[181] However, due to an associated risk of inducing life-threatening ventricular arrhythmias these pharmacologic tests should only be done by experts under optimal circumstances. Genetic testing may also be proposed but should be performed on careful clinical indications and in dedicated centres with the experience to interpret results and with the ability to provide genetic counselling. The use of signal-averaged ECG has decreased but might be considered in special circumstances, such as to search for concealed underlying structural heart disease (e.g. ARVC).

Management of asymptomatic ventricular arrhythmias largely depends on whether structural heart disease is present or not. In individuals without structural heart disease, non-sustained and sometimes repetitive idiopathic VT are usually adenosine-sensitive, based on cAMP mediated triggered activity, often aggravated by exercise or emotional stress. They mainly originate from the right or LVOT, although there are exceptions.[182–184] Rarely, verapamil-sensitive fascicular VT presents as non-sustained salvos.[185–188]

Prognosis is usually considered benign in those that have no clear heart disease,[176,189] although some cases of sudden death have been described, possibly reflecting an undetected cardiomyopathy or channelopathy.[190] However, when asymptomatic, some patients with very frequent or incessant NSVT may develop TICMP over time.[191] Pending symptoms or alteration in ventricular function, observation with no specific therapy is perfectly acceptable, although follow-up is mandatory.

Polymorphic NSVT in the absence of heart disease or channelopathy is unusual but requires detailed evaluation and in most cases treatment. Malignant polymorphic VT is extremely rare in asymptomatic patients, with premature beats triggering polymorphic VT usually arising from the RVOT or the Purkinje network[192] with, but not always, short coupling intervals.[193,194] In those patients who are asymptomatic, the possibility of quinidine, ablation, and/or ICD should be discussed with expert electrophysiologists after elimination of reversible causes.

In patients with structural heart disease, the presence of asymptomatic arrhythmias usually is a more ominous sign. No antiarrhythmic drugs, except beta-blockers, have been shown to decrease mortality in patients with asymptomatic ventricular arrhythmia and structural heart disease. Optimal medical therapy including beta-blocker, angiotensin converting enzyme (ACE) inhibitors ± mineralocorticoid receptor antagonist is the first step in individuals with impaired LV systolic function. After ruling out acute coronary artery stenosis, an ICD is indicated for sustained VT without a reversible cause in those with LVEF <35%. However, in case of mild structural heart disease with LVEF >40% and well-tolerated VT, VT ablation alone has sometimes been proposed in ischaemic cardiomyopathy[195] and ARVC.[196] This, however, needs to be determined on a case-by-case basis.

Table 9 summarizes the treatment for asymptomatic patients with NSVT depending on the underlying substrate. Non-sustained ventricular tachycardia in an asymptomatic patient with a LVEF ≥ 40% does usually not require specific antiarrhythmic therapy, but optimization of the treatment of the underlying heart disease.[202] However, the prognostic value of an EP study in patients with ischaemic cardiomyopathy and a LVEF >40% is currently being investigated.[210] Despite

Table 8 Evaluation of patients with asymptomatic sustained or non-sustained VT

First line evaluation	
History	Prior cardiovascular disease, hypertension, syncope or near-syncope, relation of VT to exercise.
Family history	SCD, inherited arrhythmia syndromes, coronary artery disease, cardiomyopathy
Medications	QT prolonging drugs, sodium channel blockers, drug interactions
Physical examination	Sign of structural heart disease or heart failure
Twelve-lead ECG	Q-waves, ischaemic changes, prolonged or fractionated QRS, QT prolongation or shortening, J point elevation and coved-type ST elevation V1–V3, early repolarization, epsilon waves, or T-wave inversion anteriorly, laterally or inferiorly
Prolonged rhythm monitoring (Holter-ECG)	Day/night/effort appearance. Frequency and duration of episodes
Echocardiography	Signs of structural heart disease
Laboratory	Serum electrolytes, renal function, thyroid function and BNP
Stress test	Suspicion of coronary artery disease, exercise-related symptoms, borderline QT interval. VT provocation by exertion
Second line evaluation	
Non-invasive evaluation of coronary artery	Low suspicion of coronary artery disease
Coronary arteriography	High suspicion of coronary artery disease
Cardiac MRI	Suspicion of structural heart disease such as ARVC, HCM, cardiac sarcoidosis, congenital abnormalities
Electrophysiological study	In case of NSVT, coronary artery disease and moderate LV dysfunction (EF<40%), syncope
Pharmacological testing • Ajmaline test • Flecainide test	To unmask suspected Brugada syndrome
Genetic testing	In case of inherited arrhythmic disorders or in the setting of familial screening when a mutation is identified in the family.

ARVC, arrhythmogenic right ventricular cardiomyopathy; BNP, brain natriuretic peptide; ECG, electrocardiography; HCM, hypertrophic cardiomyopathy; LV, left ventricular; NSVT, non-sustained ventricular tachycardia; SCD, sudden cardiac death; VT, ventricular tachycardia.

the high rate of sudden death after MI among patients with a low ejection fraction, ICDs are generally not indicated until 40–90 days after MI. Somewhat surprisingly, the results of the recent VEST trial showed that among patients with a recent MI and an ejection fraction of 35% or less, the wearable cardioverter-defibrillator did not lead to a significantly lower rate of the primary outcome of arrhythmic death than control.[211]

In patients with LV assist devices, VT is common[212] and may be well-tolerated because of the preserved cardiac output from the device. However, VT episodes seem linked to higher mortality[212] and may alter right ventricular function. Therefore, ablation may be considered in case of frequent VT episodes under these circumstances.[213]

Detailed management of asymptomatic patients with channelopathies is a topic that exceeds the purpose of this article and can be found in dedicated consensus documents.[7,214] A brief summary is found in *Table 10* but a few points warrant mention. When the QTc interval is prolonged, an assessment of why this has occurred is necessary. Medications are relatively frequent causes of QT prolongation. If drug induced QT prolongation and electrolyte disturbances such as hypokalaemia have been ruled out, consideration should be given to genetic testing for further diagnosis.

Asymptomatic non-sustained polymorphic VT in patients with Brugada syndrome or early repolarization should be considered a potentially malignant event and be managed accordingly.[179] However monomorphic NSVT (particularly originating from the RVOT) may sometimes be recorded and may not convey an increased risk. The management of such patients should, if possible, be discussed with an electrophysiologist, who is an expert in Brugada syndrome.

Currently, it is unknown if the few asymptomatic patients with multifocal ectopic Purkinje-related PVCs with preserved LV function may benefit from quinidine,[215] with SCD only being described in patients with altered LVEF. Patients with Andersen–Tawil syndrome are often asymptomatic despite presenting with frequent salvos of VT, often bidirectional, and may be incessant.[216] The rate of malignant events seem to be low on beta-blockers even if they are not directly effective in decreasing the VT burden. Flecainide seems effective against VT, especially when combined with beta-blockers. Catheter ablation does not seem to be an option in these patients.[216]

The occurrence of VT in athletes, even when asymptomatic, should lead to a thorough evaluation to eliminate the possibility of structural heart disease or the use of illegal and/or performance enhancing substances. An echocardiogram, cardiac MRI, and exercise test should all be considered. Once these possibilities have eliminated, it is well recognized that intense physical activity may not only induce VT[217] but also exercise-induced arrhythmogenic RV remodelling.[218] Sports-induced PVCs and VTs were not associated with adverse events in athletes without structural heart disease.[217] Interruption of physical activity lead to long-term resolution of exercise-induced VT, but athletes with persistent VT may be considered candidates for ablation to permit return to competitive sports.[217]

Table 9 Treatment of asymptomatic patients with NSVT depending on underlying structural heart disease

Clinical setting	Risk of sudden cardiac death	Prognostic evaluation	Treatment	References
Acute STEMI <48 h	Not increased	Coronary artery disease	• Optimal medical therapy including beta-blocker • Revascularization	
Acute STEMI >48 h	Increased risk	Waiting for 6 weeks post-MI	• Optimal medical therapy (ACE inhibitors, beta-blockers, and mineralocorticoid receptor antagonist)	197,198
Previous MI and LVEF 36–40%	Increased risk	EPS	ACEI, beta-blocker ± ICD depending on EPS	199,200
Previous MI and LVEF ≤ 35%	Increased risk	Careful evaluation of LVEF	ACE, beta-blocker, mineralocorticoid receptor antagonist ICD	200,201
Non-ischaemic dilated CMP	Uncertain	Uncertain • Cardiac MRI to identify an underlying substrate • EPS is controversial	• Optimal medical therapy (ACE inhibitors, beta-blockers, and mineralocorticoid receptor antagonist) • ICD if LVEF <30% See relevant guidelines	7,202–204
Myocarditis sequelae	Uncertain	Uncertain • Cardiac MRI to identify an underlying substrate • EPS may be considered • Exercise test	• Beta-blockers • ICD when LVEF<30% and acute phase of myocarditis ruled out	7
Mitral valve prolapse CMP	Possible increased risk	Uncertain • Cardiac MRI to identify myocardial scar	• Benefits of beta-blocker unclear • ICD may be discussed in selected cases	205–207
HCM	• Increased risk • NSVT defined as ≥3 consecutive ventricular beats at ≥120 b.p.m. lasting <30 s	Determine other criteria for risk stratification:TTE, cardiac MRI, stress test or stress echo, and genetic testing	ICD or nothing depending on risk stratification (see relevant guidelines)	208
ARVC	Probably increased risk	• Evaluation of RV and LV function • Consider EPS	• Beta-blocker • ICD should be discussed according to risk stratification • Consider catheter ablation in carefully selected cases	196,209
Left ventricular non-compaction	Uncertain	None	Same criteria than for non-ischaemic dilated CMP	7
Cardiac amyloidosis	Uncertain	None	• Specific treatment of amyloidosis • No ICD indication for primary prevention at present time	7

ACE, angiotensin converting enzyme; Acute STEMI, acute ST elevation myocardial infarction; ARVC, arrhythmogenic right ventricular cardiomyopathy; b.p.m., beats per minute; CMP, cardiomyopathy; EPS, electrophysiology study; HCM, hypertrophic cardiomyopathy; ICD, implantable cardioverter-defibrillator; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NSVT, non-sustained ventricular tachycardia; RV, right ventricle; TTE, Transthoracic echocardiography.

However, a word of caution is warranted in this situation as individuals with CPVT, who usually have structurally normal hearts, may present with exercise-induced VT which could be the first sign of high risk for sudden death. Similarly, patients with asymptomatic myocarditis would not always qualify for structural heart disease and they are known to die suddenly during exercise. This underscores the importance of a careful evaluation in these individuals. Ventricular tachycardia associated with an isolated sub-epicardial RVOT scar can be found in high-level endurance athletes without any evidence of ARVC, and this can be successfully treated by ablation with excellent outcomes.[218]

Table 10 A brief summary of treatment of patients with asymptomatic ventricular arrhythmias in the setting of channelopathies

Asymptomatic ventricular arrhythmia	Treatment options
Monomorphic sustained or non-sustained VT	If evaluation for structural heart disease with echo, cardiac MRI is normal, no therapy. Need follow-up and monitoring of LV function
Polymorphic VT	• Culprit PVC ablation • Discuss ICD and/or quinidine
MEPPC	Discuss quinidine
Andersen–Tawil	Beta-blockers ± flecainide or calcium channel blocker
CPVT on beta-blockers	• Ascertain the intake of beta-blocker • Add flecainide and/or left cardiac sympathetic denervation • Discuss ICD as last option
Long QT syndrome	• Correction of hypokalaemia if present • Careful consideration of QT prolonging drug withdrawal. Consider genetic testing and beta-blockers if no reversible cause found
Brugada syndrome and early repolarization syndrome	• Quinidine • Discuss ICD with expert in Brugada syndrome

CPVT, catecholaminergic polymorphic ventricular tachycardia, ICD, implantable cardioverter-defibrillator; LV, left ventricular; MEPPC, multifocal ectopic Purkinje-related premature contractions; PVC, premature ventricular contractions; VT, ventricular tachycardia.

While PVCs are common during pregnancy, VT and SCD are exceptionally rare.[219] Asymptomatic VT in pregnant women with a healthy heart may in most cases be monitored without specific therapy. In cases of pregnancy induced cardiomyopathy and asymptomatic VTs a temporary use of a life vest might be considered.

In infants without any cardiac abnormality, asymptomatic ventricular arrhythmias are rare and often resolve during the first year. Left ventricular dysfunction may be due to asymptomatic VT or frequent PVCs and is reversible when the burden is decreased.[220] No benefit of any antiarrhythmic drug has been shown[221] in this situation in asymptomatic children with a normal heart.

Individuals with congenital heart disease may have a variety of arrhythmias, both symptomatic and asymptomatic. In patients with tetralogy of Fallot (ToF), the risk related to NSVT is debated.[222,223] An EP study may be proposed in case of NSVT.[202] An ICD is a Class IIa indication in ToF patients presenting with other risk factors, and a Class IIb indication in patients with advanced single or systemic right ventricular dysfunction in association with other risk factors, according to the 2015 ESC Guidelines on Ventricular Arrhythmias and Prevention of Sudden Cardiac Death.[7]

Non-sustained ventricular tachycardia are common in patients undergoing chronic dialysis but mostly unrelated to SCD in this population;[224,225] thus, no therapy is recommended. Chemotherapeutic agents (mainly anthracyclines, but also other drugs such as melphalan) may acutely promote VT or torsades de pointes by different mechanisms, even in those without identifiable underlying heart disease.[226,227] Due to the risk of SCD, chemotherapy should be postponed in case of asymptomatic VT, until necessary decisions have been made about cessation or continuation of the drug together with adapted preventive therapy.

Ventricular arrhythmias are sometimes evidenced by the rhythm monitoring during a general anaesthesia. In this particular situation, it is very important to obtain a tracing of the arrhythmia. Ventricular tachycardia under these circumstances should lead to complete evaluation as mentioned above and in *Table 8*. If normal, there is usually no need for treatment.

Asymptomatic accelerated idioventricular rhythm may be observed in adults and children without structural heart disease. It does not convey any increased risk of SCD and therefore, no therapy or monitoring is needed.[228,229]

Consensus statements	Symbol	References
Patients with asymptomatic NSVT should be referred for careful evaluation to detect any underlying structural, ischaemic, or electrical heart disease.		Expert consensus
After ruling out acute coronary artery stenosis, an ICD is indicated for sustained VT without a reversible cause in those with LVEF < 35%.		Expert consensus
NSVT in an asymptomatic patient with a LVEF ≥ 40% does not usually require specific antiarrhythmic therapy, but optimization of the treatment of the underlying heart disease.		[202]

ICD, implantable cardioverter-defibrillator; LVEF, left ventricular ejection fraction; NSVT, non-sustained ventricular tachycardia; VT, ventricular tachycardia.

Tachycardia-induced cardiomyopathy

Both supraventricular and ventricular arrhythmias can lead to TICMP (*Table 11*). Tachycardia-induced cardiomyopathy may be divided into two types: (i) pure, where tachycardia is the sole mechanism of worsening of LV function; and (ii) mixed, or impure as it was originally termed, where tachycardia worsens a pre-existing cardiomyopathy due to a different cause.[191] However, the fact that pure TICMP may develop with variable incidence and severity in different patients with a similar fast heart rate over a similar duration raises the question that a latent cardiomyopathy or an underlying myocardial susceptibility could play a role in the development of TICMP.

Several studies in both animal models and in humans have suggested possible pathophysiological mechanisms for the development

of TICMP (*Figure 4*), although the understanding of this entity remains in many aspects unclear. Overlap of the mechanisms leading to TICMP may vary in the different types of arrhythmias and their presentations. In patients with AF, the restoration of sinus rhythm results in significant improvements in ventricular function, particularly in the absence of ventricular fibrosis on cardiac MRI.[230]

Table 11 Types of arrhythmias that can lead to tachycardia-mediated cardiomyopathy

Supraventricular tachycardia
• Atrial fibrillation
• Atrial flutter
• Atrial tachycardia
• Permanent junctional reciprocating tachycardia
• AV nodal re-entrant tachycardia
• AV re-entrant tachycardia
• Inappropriate sinus tachycardia (rare)
Ventricular tachycardia
• Any type of ventricular tachycardia
Premature contractions
• High burden of premature ventricular contractions
Pacing
• High-rate atrial pacing
• Persistent rapid ventricular pacing
• Permanent pacing with right ventricular stimulation

AV, atrioventricular.

There are no firm diagnostic criteria for TICMP. In patients presenting with new-onset LV dysfunction and a chronic or recurrent tachycardia with a heart rate >100 b.p.m., the diagnosis of TICMP may be suggested by the elements listed in *Table 12*.[231] Due to the retrospective nature of the diagnosis, it is often difficult to confirm a diagnosis of TICMP which can also be made by default after exclusion of other causes of worsening ventricular dysfunction. The dilemma in clinical practice is to differentiate TICMP from other forms of dilated cardiomyopathy that may be associated with atrial or ventricular arrhythmias.

Assessment of HF patients with a suspected tachycardia aetiology should include ECG, to evaluate the cardiac rhythm and look for signs of myocardial ischaemia, while an echocardiogram should be conducted to determine LV structure, functional characteristics, and to exclude valvular and pericardial abnormalities. A Holter monitor should be considered in the event of the tachyarrhythmia being paroxysmal. Evaluation of coronary arteries (by non-invasive methods or invasive coronary angiography) is necessary to rule out a potential ischaemic aetiology of the ventricular dysfunction. Cardiac MRI imaging can rule out a ventricular scar, shed light on myocarditis and some specific aetiologies of cardiomyopathy, and myocardial biopsy is now rarely used in this setting.[231–233]

Atrial fibrillation, the most prevalent sustained cardiac rhythm disorder, is considered as the most common cause of TICMP[234,235] in adults whilst permanent junctional reciprocating tachycardia is the most common arrhythmia associated with TICMP in children.[236] The incidence of TICMP is variable depending upon the type of tachycardia. In a study of 625 patients referred for radiofrequency ablation of various tachyarrhythmias, TICMP was found in 17 (2.7%; 1.3% with

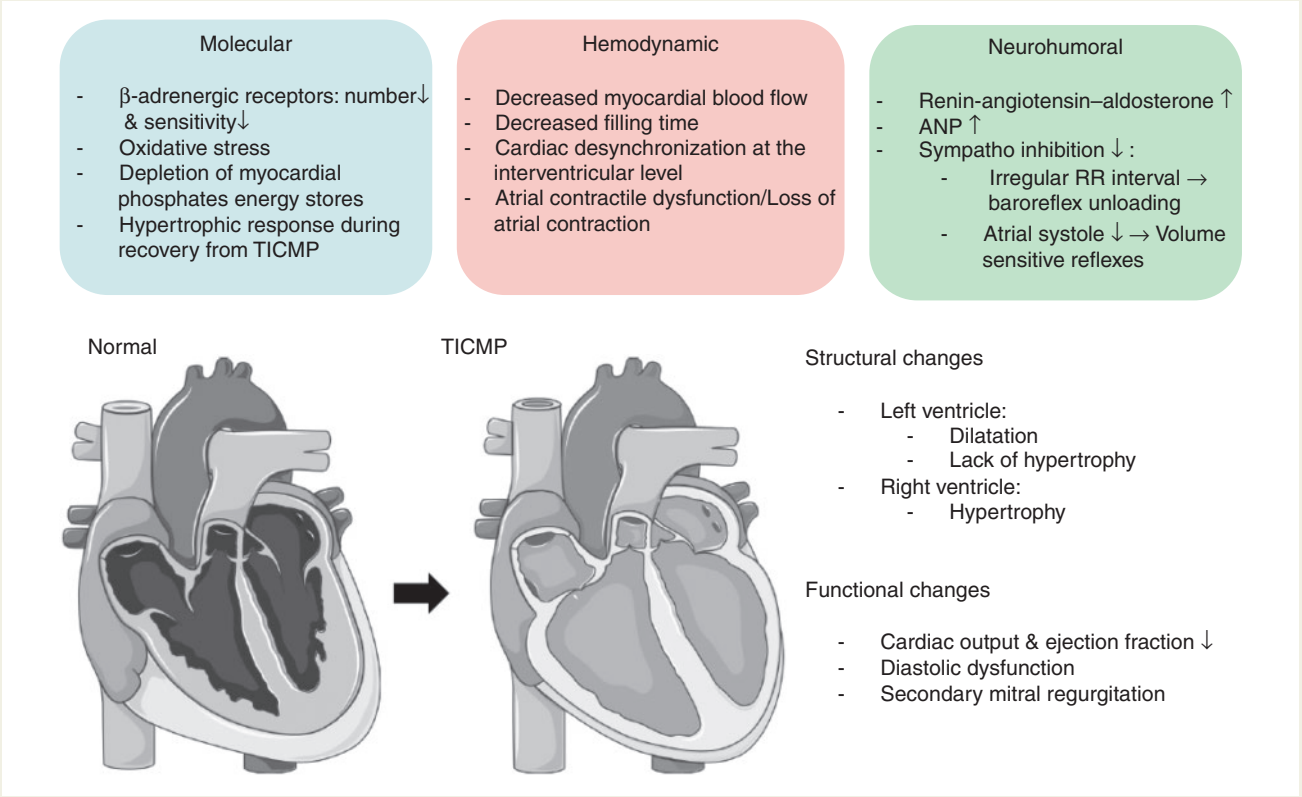


Figure 4 Possible pathophysiological mechanisms leading to tachycardia-induced cardiomyopathy (TICMP). ANP, atrial natriuretic peptide.

Table 12 Elements for the diagnosis of TICMP

(1)	No other cause of cardiomyopathy (myocardial infarction, valve disease, hypertension, alcohol or drug use, stress etc.)
(2)	Absence of left ventricular hypertrophy
(3)	No major increase in LV dimensions (LV end-diastolic dimension <6.5 cm)
(4)	Recovery of LV function after control of tachycardia (by rate control, cardioversion, or radiofrequency ablation) within a time frame of 1–6 months.
(5)	Rapid decline in LVEF following recurrence of tachycardia in a patient with recovered LV function after previous control of tachycardia.

LV, left ventricular; LVEF, left ventricular ejection fraction; TICMP, tachycardia-induced cardiomyopathy.

Table 13 Possible predictors or elements associated with development of TICMP

TICMP induced by supraventricular tachycardia, including AF
- Younger age
- Male sex
- Slower tachycardia (with less symptoms before heart failure is present)
- Incessant arrhythmia
- Irregularity of R-R interval
- Lack of symptoms in AF of atrial flutter

TICMP induced by premature ventricular contractions (PVC)
- PVC burden (from >10 000/ 24 h to >24% of total beats; threshold may be lower for right as compared to left ventricular PVCs)
- Wider PVCs
- PVCs of epicardial origin
- Presence of interpolated PVCs
- Presence of retrograde P waves
- PVCs that are asymptomatic

AF, atrial fibrillation; PVC, premature ventricular contraction; TICMP, tachycardia-induced cardiomyopathy.

AF or flutter, 0.5% with other SVT, and 1% with PVCs) patients.[235] The incidence for specific arrhythmias has been described as ranging from 10% in patients with focal atrial tachycardia to 25% in patients with permanent atrial flutter and 20–50% in patients with paroxysmal AVRT.[231,237] Patients with rapid paroxysmal tachycardia are more likely to be symptomatic and be diagnosed sooner than those with slower, but incessant tachycardias. Possible predictors of TICMP in patients with frequent supraventricular arrhythmia or PVCs are listed in *Table 13*.[162,164,166,167,238]

Tachycardia-induced cardiomyopathy usually resolves with treatment of the arrhythmia. The time course of improvement in LVEF is variable due to possible persistent ultrastructural changes and is also influenced by the duration of the arrhythmia. Some patients with TICMP may be at increased risk for SCD after apparent improvement, which could be due to persistent myocardial fibrosis.[239] Treatment goals for patients with TICMP are to slow the heart rate or reduce extrasystoles, relieve symptoms, prevent hospitalization, and improve survival. Management of TICMP comprises evidence-based treatment for HF with reduced LVEF, including angiotensin converting enzyme inhibitors and beta-blockers and aldosterone receptor antagonists, which are fundamentally important in modifying the course of systolic HF.[240] Diuretics may be used to relieve congestive symptoms.

Treatment of TICMP due to AF involves control of the ventricular response with rate-controlling drugs, use of antiarrhythmic drugs, direct current cardioversion, or catheter ablation of the tachyarrhythmia (*Figure 5*), in addition to anticoagulation. Atrial fibrillation management also aims to reduce symptoms and prevent systemic thromboembolism. Randomized trials comparing outcomes of rhythm vs. rate control in AF almost two decades ago found no differences in morbidity or mortality between these approaches. However, patients were included in these trials because the two strategies were considered possible options at baseline, which does not appropriately reflect the clinical setting of patients with TICMP where resolution of the arrhythmia is a main therapeutic target. In addition, ablation was not widely available at that time.

Rate control therapy commonly includes beta-blockers and/or digitalis. Non-dihydropyridine calcium channel antagonists should be avoided in the context of systolic HF associated with TICMP. Amiodarone can be used in patients otherwise refractory to rate control. Amiodarone is also the most frequently used drug to control the cardiac rhythm in this setting.[47] The decision to control the rate or rhythm should be individually tailored.[241] Catheter ablation has been used in the setting of AF with HF with the consistent demonstration of an improvement in LVEF, reduction in symptoms, and improvement in quality of life.[242,243] Building on these observational series, two recent randomized studies (AATAC and CASTLE-AF) have demonstrated superiority in reducing AF burden, reduced hospitalization, and reduced mortality with catheter ablation in systolic HF when compared to drug therapy.[135,243] The CAMERA-MRI study provides further mechanistic insights using cardiac MRI in patients with AF and HF, demonstrating the greatest improvement in LV function in individuals with a lower ventricular fibrosis burden.[244] It is important to recognize that these studies enrolled patients with a rather narrow clinical profile and were undertaken in highly specialized units, i.e. they are not fully generalizable to other patient populations.

While these studies included 'symptomatic' patients, it is important to recognize that in the setting of HF, it is not always possible to discern the symptoms related to AF from those related to HF. Whether these findings can be extrapolated to the truly 'asymptomatic' patient is not known. For now, consideration of ablation as a strategy in the asymptomatic patient will need to be individualized, taking in to account patient preference, the experience at each centre and the disease state. Atrial flutter is more difficult to rate control compared to AF. Given the high success rate and low risk of complications with catheter ablation of typical right sided atrial flutter, ablation to eliminate atrial flutter is recommended when TICMP is suspected.[245] For atypical atrial flutter, this should be decided on an individual basis as indicated before.

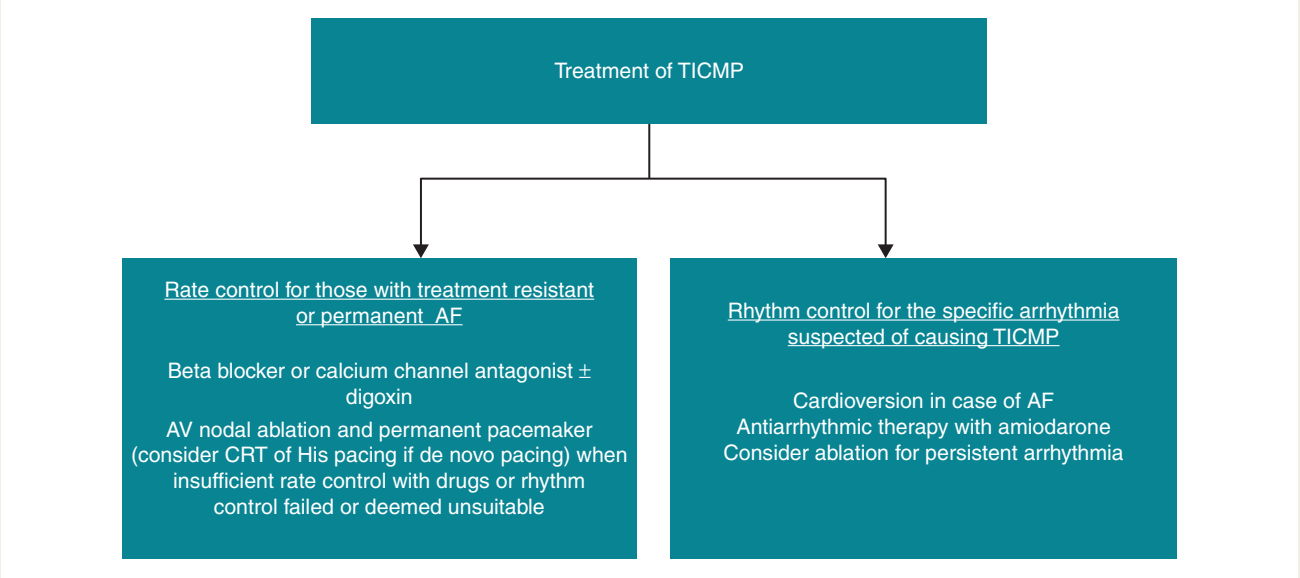


Figure 5 Potential management strategy for patients with tachycardia-induced cardiomyopathy (TICMP). AF, atrial fibrillation; AV, atrioventricular; CRT, cardiac resynchronization therapy.

A radical form of rate control strategy is AV nodal ablation with implantation of a permanent pacemaker programmed to VVIR mode.[47] This procedure may be associated with a better prognosis and seems more relevant for older patients with significant co-morbidities.[246] Since continuous right ventricular pacing may be deleterious for LV systolic function due to LV dyssynchrony, CRT should be considered as *de novo* pacing in those already with LV dysfunction. His bundle pacing holds promise as an attractive mode to achieve more physiological pacing.[247] Atrioventricular nodal ablation is associated with a substantial reduction in all-cause mortality, cardiovascular mortality, and rates of hospitalization for HF, with improvements in New York Heart Association functional class when compared with medical therapy in AF patients receiving CRT in observational and randomized studies,[248,249] and this may apply to patients with AF-related TICMP. Atrioventricular nodal ablation might be a feasible early strategy in individuals with TICMP and a pre-existing pacemaker or CRT device.

Premature ventricular contractions leading to TICMP can either be suppressed by use of antiarrhythmic agents or eliminated by use of radiofrequency ablation (*Figure 4*) as discussed in Atrial fibrillation and flutter section. A therapeutic trial with drugs for at least 3 months or catheter ablation may be considered for patients with presumed PVC-induced TICMP. Beta-blockers, amiodarone, and dofetilide (in some countries) can all suppress PVCs and can be safely used in patients with LV dysfunction.[250–252] Flecainide is not recommended in this setting. Catheter ablation is a very efficient and recognized option for these patients as the safety and efficacy profiles of the procedure have improved. Several studies have documented an improvement in LVEF following PVC ablation in nearly all patients along with significant reductions in LV end-diastolic dimensions, mitral regurgitation, and New York Heart Association functional class.[253–255] Short-term ablation success rates of between 70% and 90% have been reported.[256,257] Early improvement in LVEF after ablation may help to predict complete recovery of LV systolic function.[257] Although these strategies with antiarrhythmic drugs or ablation differ in their ability to suppress PVCs and in LV dysfunction improvement, their effect has not translated into improvement in survival so far. There is no clear data to support safe withdrawal of standard HF treatment after improvement of LV function.

Consensus statements	Symbol	References
Other causes of cardiomyopathy (myocardial infarction, valve disease, hypertension, alcohol or drug use, stress, etc.) should be eliminated before considering a diagnosis of tachycardia-induced cardiomyopathy (TICMP).		191,232
Management of TICMP should involve drug treatment for heart failure, rate control in the case of atrial fibrillation (AF) when rhythm control is not feasible and rhythm control for the specific arrhythmia (including AF) causing TICMP.		191,230–232
Ablation may be preferred for rhythm control of persistent or repetitive atrial or ventricular arrhythmia, even when asymptomatic, in suspected TICMP cases.		231,232

Asymptomatic bradycardia

Asymptomatic bradyarrhythmias, including sinus node dysfunction (SND) and AV conduction disturbances can be noted during routine evaluation or diagnostic workup of an individual who is symptomatic from another cardiac or extracardiac disorder. In this situation, it is important to differentiate between those who are truly asymptomatic from individuals who, frequently due to slow progression of the disease, have yet to notice subtle symptoms. For further evaluation, a 24–48 h Holter may give additional information. Also, functional tests such as a treadmill exercise test or a stationary bicycle exercise test can be useful to evaluate whether there is an appropriate chronotropic response to exercise and to potentially unmask symptoms. A pacemaker implantation is only indicated for symptomatic bradycardia with very few exceptions.

Few studies have examined the prognostic value of asymptomatic bradycardia in the general population. From an outpatient database, the long-term outcome of 470 patients aged >60 years with asymptomatic sinus bradycardia (i.e. heart rate <55 b.p.m.) were compared with 2090 patients without bradycardia.[258] During a mean follow-up of 7.2 years, patients with bradycardia had a very low rate of subsequent pacemaker implantation (<1% per year) and asymptomatic bradycardia had no adverse impact on all-cause mortality and may even have been protective. Molgard *et al.*,[259] performed repeated 24-h Holter monitoring in 183 healthy individuals aged 40–85 years. Pauses were documented in 16–31% of the recordings. Subjects with pauses had a significantly lower average heart rate. Pauses >1.5 s occurred in 6–6.5% of the subjects whereas pauses ≥2 s were rare in non-athletes, occurring in 1–1.6% of the subjects. The majority of the pauses were due to SND, mainly sinus arrest in older patients. In another study of 26 elderly (>70 years) subjects studied by Holter monitoring, the longest sinus pauses were observed during sleep and ranged from 0.8 s to 2.5 s, and were not associated with symptoms.[260]

Certain bradyarrhythmias (first-degree and second-degree Mobitz type I AV block) are common in younger individuals in a resting state and also in competitive athletes. They are generally deemed to be of little concern in the absence of underlying structural heart disease. Recently, a systematic review on cardiac pauses in competitive athletes was performed.[261] The study population comprised 194 individuals with cardiac pauses of 1.35–3.0 s. When specific records for pause durations were provided, 106 athletes had pauses ≤3 s, of whom 92 were asymptomatic and 14 had pauses >3 s, of whom 9 were asymptomatic. Few subjects were deemed to require medical intervention at the time of diagnosis, and there were no deaths during 7.5 ± 5.1 years of follow-up. It was concluded that the accepted 3 s pause threshold does not adequately discriminate between potentially asymptomatic and symptomatic competitive athletes, and in isolation should not be used as a determining factor to exclude potential competitors. Further, the 3 s pause threshold does not appear to warrant either exercise restriction or early therapeutic intervention.

With the increasing availability of prolonged monitoring techniques, it is not unusual to document even long asymptomatic pauses in patients that have experienced syncope. In the absence of a cause–effect relationship, the meaning of asymptomatic pauses in patients with a diagnosis of unexplained syncope is uncertain. This issue is of practical importance, since a good correlation with the index syncope would allow the use of non-syncopal documented events as surrogate endpoints. Few studies have found a good intra-patient correlation between non-syncopal and syncopal episodes. In a study of 60 patients with unexplained syncope, asymptomatic severe bradyarrhythmias, including >5 s pauses, >10 s 3° AV block and heart rate <30 b.p.m. for >10 s while awake, was observed in 7 patients and led to pacemaker implantation.[262]

In a sub-study of the ISSUE2 study, Moya *et al.*,[263] correlated the ECG findings saved by an ILR during non-syncopal episodes, either pre-syncope or non-specific symptoms, with those recorded during syncope in order to evaluate their possible role in predicting the mechanism of syncope. Nine patients had an automatic activation of their ILR, i.e. were asymptomatic, and nine had non-specific symptoms. In these 18 patients, the documentation of an arrhythmia showed a high probability as a diagnostic finding, making it unnecessary to wait for syncope to be documented and allowing, in those cases in which it is considered indicated, to initiate therapy earlier.

There are limited data regarding the beneficial effect of pacing in patients with a history of syncope but with asymptomatic intermittent bradycardia without extended pauses during monitoring. The length of the documented pause is of importance for the decision to implant a pacemaker. While ventricular pauses of 3 s or longer are uncommon, these pauses usually do not cause symptoms, and the presence of these pauses does not necessarily portend a poor prognosis or the need for pacing in asymptomatic patients.[264] The position of the patients when the pause occurs may have an impact on how long a pause is needed to impact consciousness. In patients with a clinical diagnosis of neurocardiogenic syncope and asymptomatic pause(s) >6 s, there is weak evidence that cardiac pacing may be effective and useful for the reduction of syncopal recurrences. The rationale behind the 6 s cut-off value includes data showing that loss of consciousness may take up to 7 s in case of circulatory arrest.[11]

In patients presenting with asymptomatic intermittent night-time bradycardia (sinus bradycardia or AV block), sleep apnoea should be considered as a possible cause. It was estimated that episodes of heart block occur in approximately 20% of patients with severe sleep apnoea, i.e. apnoea–hypopnoea index >60/h) and approximately 7.5% of an unselected group of patients with obstructive sleep apnoea.[265] Rapid eye movement sleep and excessive vagal activation due to hypoxia and apnoea seem to be important mechanisms leading to bradycardia. Treatment with continuous positive airway pressure should be attempted first, since it has been shown to lead to complete prevention of heart block in 80–90% of these patients.

It is not uncommon to record asymptomatic episodes of AV block on prolonged ECG monitoring. Asymptomatic bradycardia is not uncommon and interpretation of this should be made in the clinical context of the patient. In healthy subjects, pauses >2.5 s are infrequent but this alone does not necessarily define a clinical disorder. Asymptomatic bradycardia is common in athletes, and the accepted 3 s pause threshold neither warrants exercise restriction or early therapeutic intervention.[261] Pauses in AF patients between 3 and 5 s are frequently seen and may be a normal occurrence. Treatment is not required except in case of symptoms.

In the case of AV block, it is crucial to distinguish nodal from infra-nodal localizations, the latter usually requiring pacemaker implantation. Key for this distinction are the type of AV block, the presence or absence of wide QRS complexes and the heart rate changes at the time of AV block. Progressive PR prolongation prior to the non-conducted P wave is typical for Type I second-degree AV block. True Type II second-degree AV block, with no PR prolongation prior to the non-conducted P wave, should be recognized through careful analyses of the tracings since it denotes a severe disease of the conduction system and may in most cases warrant pacemaker implantation. In the case of a single asymptomatic non-conducted P wave, a decision should be made on a case-by-case basis. When wide QRS complexes are present, an infra-Hisian localization should be considered, even in case of Type I second-degree AV block and may prompt an electrophysiological study. Slowing of the heart rate at the time of AV block is diagnostic of AV nodal localization while block in the His-Purkinje system can be tachycardia-dependent. In addition, careful analysis of the tracings should rule out concealed His bundle extrasystoles which may mimic both Types I and/or II AV block. Exercise testing may be beneficial to identify infra-Hisian block. There is some data indicating that pacing of Type 1 second-degree AV block may be of benefit in selected asymptomatic elderly individuals.[266] However, this area clearly needs further investigation before any conclusive recommendations can be made.

There is consensus that third-degree AV block in the absence of correctable causes and Type II second-degree AV block (Mobitz II) should be treated with a pacemaker even in the absence of symptoms given the potential severity of these findings.[267]

Consensus statements	Symbol	References
In individuals with syncope, the presence of asymptomatic severe bradycardia or pauses >6 s should be considered a diagnostic finding and lead to treatment.		263
Completely asymptomatic bradycardia does in itself not require treatment.		267,268

Patient perspective

As previously stated in this document, cardiac arrhythmias can be episodic or persistent, and in many individuals, may be asymptomatic. However, the absence of symptoms in arrhythmic conditions should not necessarily imply that the patient does not require treatment or free from risk of adverse outcomes. Indeed, asymptomatic cardiac arrhythmias may be associated with worse outcomes as often the detection of the arrhythmia is made following a first presentation to hospital for a serious arrhythmia-related event, such as stroke (in AF), cardiac arrest (e.g. ventricular arrhythmias), or TICMP.

Even in the absence of symptoms, once an arrhythmia has been diagnosed, patients may still experience significant distress and worries about the arrhythmia. This may extend to the therapeutic options with their possible side effects (e.g. bleeding with oral anticoagulant therapy, side effects of AADs and other medications, ICD implantation, etc.) and the potential consequences of the arrhythmia (death, stroke, HF, etc.).[269–272] Absence of symptoms may also affect the choices that the individual makes in regard to treatment pathway/options, as the patient may perceive that they have 'less severe' disease due to lack of symptoms. This may in turn lessen their appreciation of the seriousness of the consequences of the arrhythmia and the necessity of treatment. Lack of symptoms may also affect the treatment options offered to patients, for example in AF rhythm-control strategies (cardioversion and ablation) are usually targeted at symptomatic patients, as the goal of such therapy is symptom reduction or resolution. Lifestyle restrictions and/or modifications frequently accompany a cardiac arrhythmia diagnosis, either directly as result of the arrhythmia (e.g. inherited arrhythmia disorders, WPW, etc.) or due to the treatment options required to manage the arrhythmia (e.g. ICD). In those with an inherited arrhythmia or WPW, the diagnosis typically occurs when the patient is relatively young and otherwise fit and well and such a diagnosis may permanently alter the quality of life and may lead to profound psychological distress.[270,273]

It is essential that, regardless of the type of arrhythmia, patients are fully informed about the trajectory of their condition; the available treatment options (risks and benefits and side effects, particularly for ICD implantation); lifestyle changes that are required to modify their risk factors and reduce their risk of adverse outcomes; likelihood of treatment success and what can be achieved so that people can form realistic expectations of treatment and make informed decisions about the treatment options that are right for them.[46,47,63,274,275] Patient education is a fundamental part of arrhythmia management,[63,274,276] irrespective of symptoms. It is also crucial to acknowledge patients' concerns and assess and monitor the psychological impact of the arrhythmia and its treatment on the patient and their family, and to formulate a plan to manage distress,[63] since psychological distress can influence patient adherence and persistence with treatment and drastically reduces patients' quality of life and that of their families,[277–282] irrespective of the presence or absence of symptoms.

A 2015 EHRA consensus document summarizes the current literature on cardiac arrhythmias and patients values and preferences for their management[63] and also provides important topics for physician–patient discussions concerning their arrhythmia and disease course, treatment options and goals, and outcomes, and helpful resources to elicit these conversations.

Shared decision-making should be the approach adopted to accomplish this target; incorporating both the patient and the physician/healthcare professional, mutual shared information, bilateral (patient and physician) deliberation about preferences, options, and reaching a shared treatment decision, including no treatment as a possibility.

Consensus statements	Symbol	References
Education is an essential component of the management of cardiac arrhythmias to enable patients (and their carers/family members) to understand their condition, the available treatments, disease trajectory, and possible outcomes, regardless of symptoms.		63
All patients should receive individually tailored disease- and treatment-specific information from their healthcare team which is reiterated over time and when new management strategies are discussed.		63
Patients' preferences for treatment should be discussed, documented, and incorporated into management decisions.		63

Areas of future concern

During the process of writing this consensus document on asymptomatic arrhythmias, it has become increasingly evident that there is a real paucity of data from studies adequately representing asymptomatic patients. Many of the sections in this consensus document have included data which have been extrapolated from studies on predominantly symptomatic arrhythmias and some even from highly selected subgroups. Therefore, the drafting of consensus statements for asymptomatic arrhythmias has been in some ways a complex task. Also, asymptomatic arrhythmias vary considerably in regard their risk to cause adverse effects and the need for intervention. In cases where restraining from treatment might lead to potentially severe consequences, such as stroke in AF and subsequent cardiac arrest in individuals with sustained VT, it would be irresponsible to withhold treatment despite lack of symptoms from the arrhythmia.

In the future, strong consideration should be given to improved and structured systematic assessments and survey instruments for symptoms in arrhythmia studies. Additionally, the relationship between symptoms and arrhythmia burden requires better exploration. The reasons for the lack of symptoms in some individuals are still poorly understood. While treatments aiming to decrease the perception of benign symptomatic arrhythmias are common, technology aiming to enhance patients and care providers awareness of serious arrhythmias could also be useful. Such alerts as an example already exist in some CIEDs and serve the purpose to notify a patient having an asymptomatic but potentially malignant ventricular arrhythmia.

The increased availability of medical devices and apps to consumers will likely lead to a significant increase the diagnostic yield and observed prevalence of asymptomatic tachycardia, bradycardia, and AF in the very near future. A number of companies including Fitbit, Garmin, Apple, and Samsung, all have devices on the market with heart rate alert features. Smartwatch and fitness band wearable consumer electronics can passively measure pulse rate from the wrist using photoplethysmography.

Identification of pulse irregularity or heart rhythm variability from these data has the potential to identify AF. The rapidly expanding use of these devices will allow for detection of undiagnosed AF in a novel manner. Companies, including AliveCor and Withings also have ECG watches or watch accessories. Apple has an irregular rhythm notification now that is being prospectively evaluated in a clinical trial which will include more than 400 000 individuals.[283] While the opportunities created by these possibilities are exciting, they will undoubtedly emphasize the need for clearer guidance on how and when to intervene in those patients with asymptomatic heart rhythm abnormalities detected by play of chance.

Although this document focuses mostly on asymptomatic arrhythmias, patients should be warned of symptoms that might develop or that have been dismissed in the past, such as syncope and near syncope. It is also important to remember that in the past medical misassessments have included diagnosis of anxiety and/or hypoglycaemia in patients that have arrhythmias.

Finally, in many different asymptomatic heart rhythm irregularities, the distinction and cut-offs between a significant and non-significant burden of an arrhythmia remain unclear. Furthermore, some arrhythmias might have been diagnosed during either direct or indirect screening, or because of the increased use of detection devices, such as smartphones apps or special watches. These individuals may represent a group having a lower burden of arrhythmias, and to the same extent as subclinical AF discovered through CIEDs, using continuous monitoring, may represent potentially less harmful arrhythmias. Further studies are needed to evaluate the seriousness and net clinical benefit of treatment in these patients.

Supplementary material

Supplementary material is available at Europace online.

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