literature review was submitted and accepted prior to the publication of this study, it was regrettably not possible to include these findings.[3]

Conflict of interest: None declared.

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Estimated incidence of previously undetected atrial fibrillation on a 14-day continuous electrocardiographic monitor and associated risk of stroke: comment

This Letter to the Editor refers to the article 'Estimated incidence of previously undetected atrial fibrillation on a 14-day continuous electrocardiographic monitor and associated risk of stroke' by William F. McIntyre *et al*. https://doi.org/10.1093/europace/euab324.

We read with interest the article by McIntyre *et al*.[1] who investigated the incidence of previously undetected atrial fibrillation (AF) on a 14-day continuous electrocardiogram (ECG) monitor and the associated risk of stroke. For these patients, who were aged >65 years with hypertension and a pacemaker, the findings demonstrated that a 14-day ECG detected AF >6 min in 3.1% and highlighted an associated increase in stroke risk [2.18%/year, adjusted hazard ratio (HR) 3.02]. As the findings have important clinical implications concerning the risk stratification of stroke, we propose that the authors also investigate other significant factors.

The association between AF burden and increased stroke risk is corroborated by other recent clinical trials. Chu *et al.*[2] conducted a retrospective cohort study of 152 patients, investigating pacemaker-detected AF burden, and associated ischaemic stroke or systemic thromboembolism risk (SSE). Using multivariate Cox regression, the authors determined that AF burden >6 min was a predictor of significantly higher SSE risk (propensity-adjusted HR, 6.75; $P = 0.023$) and reported a notably higher HR of 9.33 ($P = 0.033$) for any AF episode (including ≤6 min). Consequently, it would be interesting to see whether McIntyre *et al.*'s data on AF episodes ≤6 min was associated with increased incidence of SSE, allowing us to further evaluate the designated 6-min cut-off.

In the stratification of stroke risk, CHA2DS2-VASc scoring is a clinical tool used to determine the need for oral anticoagulation in AF patients based on key risk factors. Nonetheless, in a study of 21 768 non-anticoagulated patients with pacemakers, Kaplan *et al.*[3] reported that patients with a CHA2DS2-VASc score ≥5, despite not having AF, also crossed the actionable stroke risk threshold of >1%/year. While McIntyre *et al.* have provided a hazard ratio that is adjusted for the CHA2DS2-VASc score, we would recommend a separate analysis of the relationship between the CHA2DS2-VASc score and SSE in the patients without AF burden. This could provide highly valuable insights into the utility of CHA2DS2-VASc, and subsequent oral anticoagulation, in pacemaker populations without AF.

Furthermore, the type of AF and ectopic beat burden may have bearing on the relationship with stroke risk, as observed by Cho *et al.*[4] in their observational study of 10 000 AF patients. Atrial pre-mature beats were present in higher proportions in non-paroxysmal AF as opposed to paroxysmal ($P = 0.001$) in 24-h ECG monitoring, with a significantly increased risk of stroke in non-paroxysmal AF (adjusted HR 2.08, $P = 0.001$). Henceforth, we suggest that McIntyre *et al.* could explore SSE risk by AF type and ectopic beat burden—as these findings could deepen our understanding of the insidiousness of AF characteristics with respect to stroke, assisting prophylaxis, and risk management.

Conflict of interest: None declared.

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New-onset ventricular arrhythmias after surgery for mitral valve prolapse: how to classify and manage?

This Letter to the Editor refers to 'EHRA expert consensus statement on arrhythmic mitral valve prolapse and mitral annular disjunction complex in collaboration with the ESC Council on valvular heart disease and the European Association of Cardiovascular Imaging endorsed by the Heart Rhythm Society, by the Asia Pacific Heart Rhythm Society, and by the Latin American Heart Rhythm Society', by A. Sabbag *et al*., https://doi.org/10.1093/europace/euac125.

A link between mitral valve prolapse (MVP) and malignant ventricular arrhythmias (VAs) has been reported. The recently published consensus statement summarized current literature and provided practical suggestions for risk stratification and management of patients with arrhythmic MVP.[1]

The mechanism of VAs in MVP, although not well explained, may be linked to anatomical substrates as areas of patchy myocardial fibrosis in the sub-valvular apparatus, triggered activity due to mechanical stretch of papillary muscles that leads to stretch-activated early afterdepolarizations and abnormal repolarization as a result of endocardial and mid-myocardial fibrotic changes on the papillary muscles and adjacent left ventricle.[1] Despite the common fibrosis close to the mitral annulus, detected using cardiac magnetic resonance imaging, the findings of a recent systematic review may imply that the main mechanism of ventricular arrhythmias leading to sudden cardiac death (SCD) is non-reentrant.[2] In this way, the committee does not endorse the routine use of electrophysiological study with programmed ventricular stimulation for risk stratification.

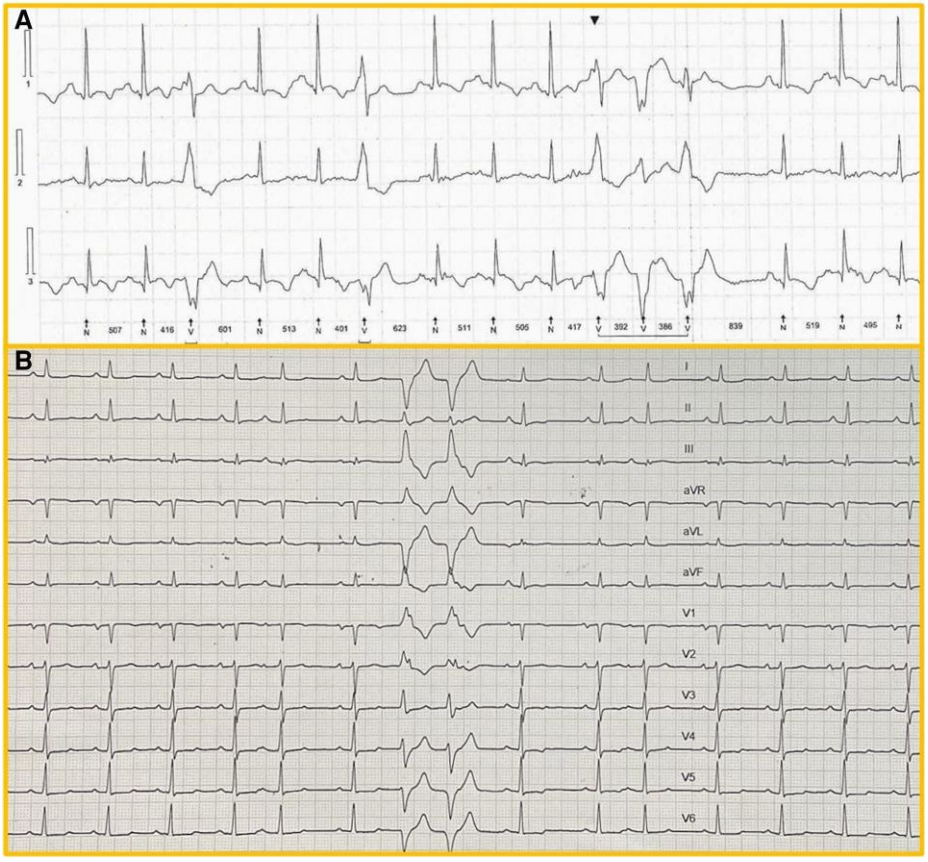

Figure 1 New-onset ventricular arrhythmias in a 23-year-old female patient early after surgical valve repair for mitral valve prolapse with severe mitral regurgitation *A.* 24 h Holter monitoring showing a high burden of new-onset ventricular ectopy and non-sustained ventricular tachycardias. *B.* 12-lead ECG with a couplet of premature ventricular extrasystoles showing a right bundle branch block pattern with a late *R/S* transition in V4 precordial lead, likely originating from the anterolateral papillary muscle.

Integrative grading of mitral valve regurgitation (MR) severity is also essential for evaluating the risk of VAs, as several studies have demonstrated that patients with severe degenerative MR carry an increased risk of SCD. Mitral valve surgery, through suppressing the progression of MVP, may have a role in preventing SCD by reducing VA burden. However, data are inconsistent and mainly derived from case series and thus, the surgical approach of MVP is not proposed in patients with high-risk VAs without severe MR.[1,3,4]

On this point, we should mention that not only VA reduction but also new-onset VAs have been described after corrective valve surgery, including mitral valve repair.[5] Importantly, most clinically significant VAs in this setting have been revealed to be due to scar-related reentry as they are inducible with programmed stimulation.[5] These arrhythmias present a similar ECG morphology that is compatible with papillary muscle or mitral annular/left ventricular basal origin impeding distinction of the underlying mechanism (*Figure 1*). *R/S* ratio and QRS duration are reliable predictors for differentiating papillary muscle VAs from fascicular arrhythmias that can also occur after valve surgery.[5] However, previously undetected arrhythmic MVP VAs of non-reentrant mechanism cannot be excluded. Interestingly, these arrhythmias can occur even years after surgery. Therefore, they could also be related to a degenerative process of MVP.

Taking into consideration the abovementioned characteristics of post-surgery VAs, we suggest that a distinct algorithm may be applied for the approach of new-onset (or presumably new-onset) VAs after surgery for MVP. Although validation from specifically designed studies is warranted, the role of electrophysiological study may be more crucial for the evaluation of these arrhythmias.

Conflict of interest: None declared.

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