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Review

Catheter ablation of ventricular tachycardia associated with structural heart disease: Current status and perspectives

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ABSTRACT

Catheter ablation is an effective and safe treatment for ventricular tachycardia attributable to structural heart disease, reducing the risk of recurrent arrhythmias and defibrillator shock therapy. Advances in medical technology and an accumulation of data have led to the development of detailed guidelines. Successful ablation requires accurate identification of the arrhythmia substrate and effective delivery of radiofrequency energy to the target tissue. Modern practice requires use of traditional electrophysiological mapping processes such as entrainment mapping and three-dimensional activation sequence mapping in combination with newer functional mapping techniques for which there is growing support. Thorough non-invasive preoperative assessment is also necessary before an invasive procedure is undertaken. In this review, we summarize contemporary practice and recent randomized controlled trials underpinning the latest developments in mapping and ablation and discuss potential future developments in this field.

Introduction

Ventricular tachycardia (VT) is a significant cause of morbidity and mortality in patients with either ischemic cardiomyopathy (ICM) or non-ischemic cardiomyopathy (NICM) [1]. Although prognosis has improved with guideline-directed medical therapy, use of implantable cardioverter defibrillators (ICDs), and use of cardiac resynchronization therapy devices, patients continue to experience recurrent VT with appropriate ICD shocks, cardiac hospitalizations, and VT-associated morbidity and mortality [1–3]. Antiarrhythmic drug (AAD) therapy is commonly used for recurrent ventricular arrhythmias. However, AADs are not without significant side effects, and patients can suffer pulmonary or hepatic toxicity (an effect of amiodarone) [4], aggravation of the arrhythmia, or bradyarrhythmia, which may increase the need for pacing, potentially aggravating the ventricular dysfunction. Further, limitations in AAD-based control of arrhythmia over the long-term have been recognized [5]. Radiofrequency (RF) catheter ablation is an established adjunctive treatment option for VT in patients with cardiomyopathy, particularly those with a ventricular arrhythmia refractory to AAD therapy and those who cannot tolerate AADs. There have been numerous reports providing evidence that RF ablation, in addition to use of advanced technologies, can be used to improve clinical outcomes, even life expectancy, of patients with VT associated with structural heart disease.

Herein, we review and discuss the reported evidence that RF ablation, used in addition to advanced technologies, can improve clinical outcomes, even life expectancy, of patients with VT associated with structural heart disease.

Indications

For VT associated with structural heart disease, expert consensus guidelines currently recommend catheter ablation of the VT after the failure of AAD therapy and for patients in whom AAD therapy is either not tolerated or not desired (class I indication) [6]. The Japanese guideline recommendations for catheter ablation of sustained monomorphic VT in patients with structural heart disease are shown in Table 1 (JCS/JHRS 2019 Guideline) [7].

Whether catheter ablation is indicated for sustained VT should be determined under careful consideration of the risks and benefits with respect to the patient's general condition and the physician's level of experience. In cases of sustained VT associated with a history of myocardial infarction or structural heart disease such as cardiomyopathy, ICD implantation is the mainstay for prevention of sudden death [8,9]. However, frequent VT attacks and ICD-delivered shocks have been reported to worsen patients' quality of life and to increase the risk of all-cause death [10–13]. In cases of VT or ventricular fibrillation associated with hemodynamic compromise, the patient is more likely to experience

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Table 1
Indications for catheter ablation of sustained monomorphic VT in patients with structural heart disease (modified from the JCS/JHRS 2019 guideline).

Class I indication
In patients with structural heart disease and incessant monomorphic VT or VT storm, for whom AADs are ineffective or not tolerated, catheter ablation is recommended.
In patients with ICM and symptomatic sustained monomorphic VT for whom AADs are ineffective or not tolerated, catheter ablation is recommended.
In patients with ICM, sustained monomorphic VT, and an ICD who experience frequent ICD therapies, catheter ablation is recommended.
In patients with ICM who experience recurrent monomorphic VT despite chronic amiodarone therapy, catheter ablation is recommended in preference to escalating AAD therapy.
In patients with bundle branch reentrant VT, catheter ablation is recommended for reducing the risk of recurrent VT.
Class IIa indication
In patients with ICM and sustained monomorphic VT who undergo an ICD implantation, perioperative catheter ablation should be considered to reduce the risk of recurrent VT or ICD therapies.
In patients with non-ischemic cardiomyopathy and sustained monomorphic VT for whom AADs are ineffective or not tolerated, catheter ablation should be considered.

AAD, antiarrhythmic drug; ICD, implantable cardioverter-defibrillator; ICM, ischemic cardiomyopathy; VT, ventricular tachycardia.

syncope even if the ICD is activated, and in patients in the conscious state, DC shocks can cause fear and discomfort [12,13]. Concomitant use of AADs reduces the frequency of ICD shocks, but it is not curative, and there are potential side effects [14]. Catheter ablation of sustained VT due to structural heart disease is a complicated procedure requiring technical proficiency, and the arrhythmia often recurs. However, such technological advances as 3D mapping systems [15,16] and irrigated tip ablation catheters [14,17] used in combination with cardiac magnetic resonance imaging (MRI), have significantly improved outcomes [18,19].

Several randomized controlled trials (RCTs) on ablation before or after ICD implantation for sustained VT associated with a prior myocardial infarction have been conducted [20]. The SMASH-VT [21] and VTACH [22] trials showed significantly fewer ICD interventions in ablation group patients than in non-ablation group patients. However, in a CALYPSO pilot study [20] that compared ablation treatment against AAD treatment, non-superiority of the ablation treatment over the AAD treatment was found. The VANISH trial [4], which compared ablation and AAD escalation in patients with post-myocardial infarction VT, showed significantly fewer ICD shocks in the ablation therapy arm. Thus, sustained VT with underlying ICM is now considered a class I indication for ablation (Table 1).

In comparative studies of ICM and NICM [NICM is a broad term that includes dilated cardiomyopathy (DCM), hypertrophic cardiomyopathy (HCM), cardiac sarcoidosis, arrhythmogenic right ventricular cardiomyopathy (ARVC), and lamin A/C-related cardiomyopathy] as the underlying diseases, the VT-free rates after ablation were lower among NICM patients than among ICM patients, and VT inducibility was found to be a prognostic factor [23,24]. Accordingly, sustained monomorphic VT in patients with NICM is now considered a Class IIa indication in the Japanese guidelines [7]; the substrate for reentry is often located in the mid-myocardium or epicardium, making the ablation procedure more difficult.

RF catheter ablation of ventricular arrhythmias

Effective catheter ablation of scar-related VT is of two critical components. The first is substrate identification, i.e. identification of the critical substrates involved in the initiation and/or maintenance of the arrhythmia. Substrate identification requires assimilation of all available data, including detailed analysis of the patient's electrocardiogram (ECG), pre-procedure investigations, successful induction and mapping of specific VT circuits, and information obtained from invasive mapping performed during baseline rhythm. The second is the ablation itself, i.e. delivery of sufficient energy to the target tissue to ensure permanent destruction and prevent further ventricular arrhythmia. The following is a typical VT substrate identification method.

Entrainment mapping

Entrainment mapping has been used for >30 years to discriminate VT circuits. It does not require advanced mapping systems and can be performed simply with the use of ECG recordings and a stimulation device (Fig. 1A). Ideally, induction of VT must be reproducible, the QRS morphology must be uniform from beat to beat, and the VT must be sustained and hemodynamically stable. These conditions are often not met in patients with structural heart disease. In cases of scar-related VT (e.g. ICM), finding a protected region of diastolic activity used as an essential part of the reentrant circuit is desirable because ablation at this site is likely to eliminate the tachycardia. As a result of significant changes in electrophysiology resulting from the previous injury (e.g. from infarction or myopathy), many sites in the ventricle may show diastolic activation but may not be related to the sustained VT. These "bystander sites" make activation mapping more difficult. Pacing techniques such as entrainment can determine whether a site is part of the circuit or a bystander. When pacing is stopped and the tachycardia resumes, the time of the first compound relative to the last paced beat indicates how close the pacing site is to part of the VT circuit (Fig. 1A). During entrainment, the paced wavefront activates a part of the ventricle, and part is forced to leave the circuit earlier than normal, resulting in a fusion complex on the ECG. Pacing from within a critical portion of the circuit produces a QRS complex that matches that of the VT; fusion occurs only within the circuit and is "concealed," Sites with low-amplitude, isolated mid-diastolic potentials that cannot be separated from the tachycardia by pacing perturbations and where entrainment with concealed fusion can be demonstrated are very likely to be sites for successful ablation (Fig. 1B).

Activation mapping during VT

Activation mapping determines the overall pattern of ventricular myocardial activation from the timing of the regional ECG and in reference to a particular time point (e.g. start of the QRS complex). This process is mainly automated with use of electroanatomical mapping software.

In scar-related reentrant VT, the activation map requires a different interpretation. This is because activation has no focal point; there exists, rather, a continuous circuit of electrical activity (Fig. 1C): the local ECG signal just before the QRS represents the point at which the wavefront exits the protected canal into the myocardium, and the presence of a mid-diastolic potential identifies electrical activity through the critical isthmus. However, slow passive conduction is common within scar tissue, and the mid-diastolic activation alone is insufficient to determine any active involvement in the reentrant circuit. Therefore, the essential components of the re-entry circuit can be accurately identified only by entrainment mapping. Basically, entrainment pacing is performed on the presumed site of the critical isthmus of the VT delineated on the electroanatomic map (EAM) to confirm that it is the optimal site for treatment.

Substrate mapping during the baseline rhythm

In a significant proportion of patients with VT and structural heart disease, activation mapping and entrainment cannot be performed

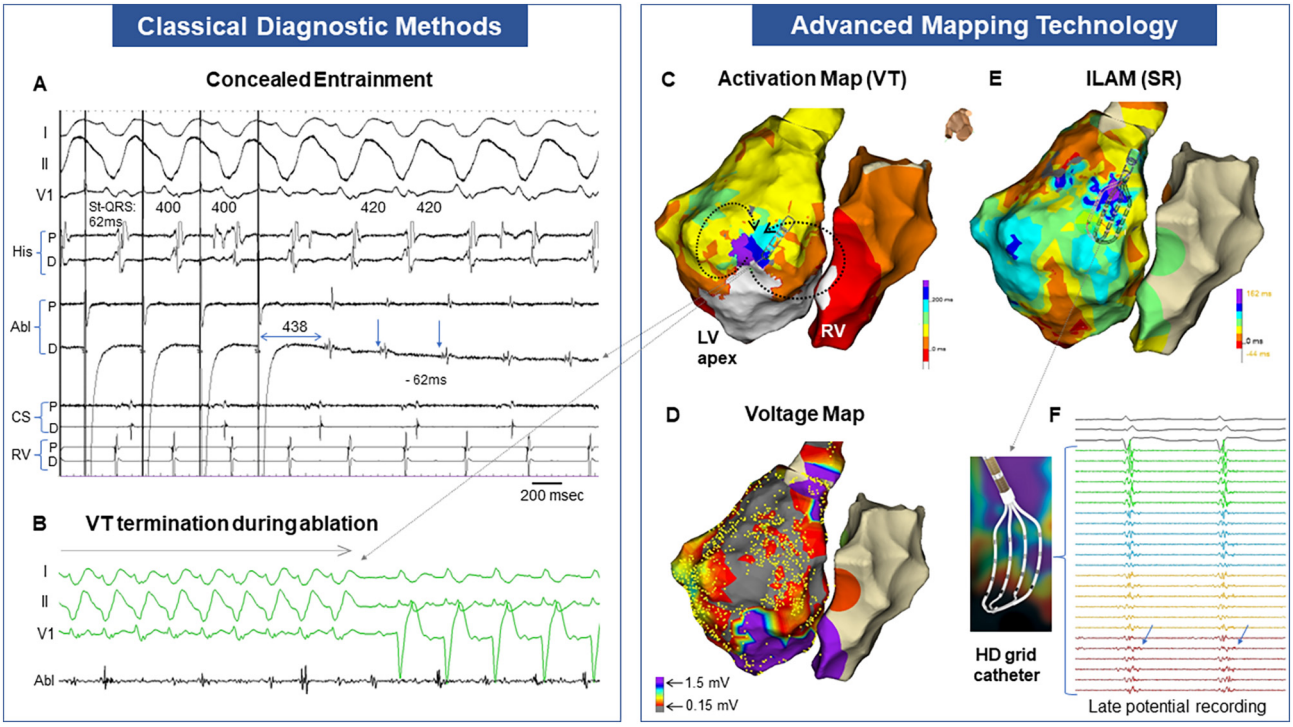

Fig. 1. Identification of the ventricular tachycardia (VT) circuit by classic diagnostic methods and by means of electroanatomical mapping. (A) "Concealed" entrainment of post-infarction VT. The three complexes on the left represent pacing during VT, with a stimulus (St)-QRS interval of 62 ms. Once pacing ends, the VT resumes. The electrogram (arrow) in Abl D (distal electrode pair of the ablation catheter) is 62 ms before QRS onset. The paced and VT QRS complexes are almost identical. (B) Ablation at this site terminated the VT. (C) Activation map displayed during the tachycardia in the same case as on the left, showing the VT to be of a figure-of-eight reentry pattern with eight time components: white, red, orange, yellow, light green, light blue, blue, and purple. Notably, most red components bear right ventricular (RV) activation, suggesting a complex three-dimensional VT circuit rather than a two-dimensional circuit. The site of tachycardia cessation shown in A, i.e. the isthmus (narrow corridor) of the VT, is indicated by an arrow. (D) Voltage map obtained during sinus rhythm in the same case. (E) Isochronal late activation map (ILAM) obtained during sinus rhythm in the same case. As in panel C, it is shown with eight time components. The transmission velocity is heterogeneous due to damaged cardiac tissue. The shortest distance between time phases is the deceleration zone (DZ). The time phase immediately before the most delayed time phase (purple) is the DZ, which can be seen to be in spatial proximity to the tachycardia termination site (panel C) and also the late potential recording site (panel E) during sinus rhythm. (F) Late potential recording obtained with an Advisor HD Grid Mapping Catheter with 16 electrodes. Late potentials are recorded extensively around the DZ shown in panel D.

due to a patient's low hemodynamic tolerance during arrhythmia or to failure to initiate sustained tachycardia during the electrophysiological study. In these circumstances, an additional method, substrate mapping, can be used. Substrate mapping involves both voltage amplitude-mapping and activation mapping during sinus or baseline rhythm.

The local ECG amplitudes can distinguish between normal and scar tissue, and automated bipolar and unipolar voltage maps are generated and displayed on a three-dimensional EAM. Normal bipolar voltage of the endocardium, measured between two adjacent electrodes on the endocardial surface, is generally considered to be above 1.5 mV, whereas bipolar voltage below 0.5 mV identifies dense scar [25] (Fig. 1D). The multipolar catheters allow for estimated localization of channels supporting VT on the high-density voltage map. However, to accurately identify the VT channels with relatively high voltages (within areas of low-voltage scarring), voltage-limiting adjustments may be necessary [26]. Tung et al. created separate voltage maps using different activation wavefronts (right ventricular pacing, left ventricular pacing, and intrinsic conduction) and re-entry in which approximately 18 % of the critical areas of the VT were found in regions where the voltage was low in one activation wavefront and normal (>1.5 mV) in another activation wavefront [27]. Further, a recent trial on post-myocardial infarction remodeling suggested a move away from the standard range for accurately identifying substrates [28].

Isochronal late activation maps (ILAMs), created during sinus rhythm or ventricular pacing, have been proposed as complementary materials for identifying critical ablation targets. An example ILAM is shown in Fig. 1E. ILAMs are easily created with electroanatomic mapping software. Local ECGs are timed according to the latest bipolar component, meaning the completion of local activation, and are displayed across isochrones that are equally distributed. Where the conduction velocity slows, isochrones appear densely packed, allowing the "deceleration zone" to be identified [29]. VT ablation guided by creation of an ILAM has also been performed prospectively, and in 95 % of cases, the site of termination coincided with the deceleration zone, with high rates of freedom from VT at 12 months [29].

Late potentials are seen as local ECGs that occur after the terminal portion of the surface QRS, either because they are entirely isolated from other local activity or because of continuous fractionation. They are found in most patients who have suffered myocardial infarction and can be easily identified by mapping during sinus rhythm. An example late potential recording is given in Fig. 1F. Removal of all late potential sites is a possible endpoint of a substrate-based ablation strategy. In a large study of patients with ischemic cardiomyopathy, a combined endpoint of non-induction and removal of all late potentials was employed, and a low incidence of recurrent VT and a significant reduction in cardiac deaths were observed [30]. The study, however, was not an RCT.

Pace mapping technique

Pace mapping serves as a corroborative method for localizing the VT circuit. It can be used to identify the presumptive exit or isthmus region of the VT circuit but is not sufficiently specific or sensitive to be the sole guide for ablation. Pace mapping in normal sinus rhythm after VT termination is attempted at potential isthmus sites (as identified by activation and entrainment mapping during VT). The resulting 12-lead ECG

morphology is compared with that of the VT. Automated pace map matching is now available in some recording and electroanatomic mapping systems. The greater the degree of concordance between the morphology during pacing and the tachycardia, the closer the catheter is to the exit zone of the VT isthmus. Evaluation of the S-QRS interval is also of value. Sites at which pace mapping produces the same QRS morphology as that of the initial isthmus site but with different S-QRS delays are identified to trace the course of the VT isthmus.

Recently, pace mapping was also used to unmask the VT isthmus in patients with re-entrant VT after myocardial infarction [31]. In that study, as expected, the highest pace map ratio was found at the exit of the isthmus, and the lowest was found near the entrance site of the isthmus. Therefore, on high-density 3D pace maps, abrupt changes in pace map match ratios are associated with the central isthmus, consistent with the location of the wavefronts identified by activation mapping.

It is now standard practice at many centers to search for the VT substrate by means of the methods described above and to deliver adequate RF energy to the target tissue to remove the substrate.

Latest trends in catheter ablation of VT

New ECG definitions and 3D maps that represent functional substrates of VT, such as local abnormal ventricular activity and decremental evoked potentials, have been reported to indicate optimal treatment sites [32,33]. Efforts to incorporate ventricular premature stimulation during mapping, rather than fixed rhythms, such as sinus rhythm or a pacing rhythm, to find decremental properties that characterize substrates and target tissues for VT have been actively pursued in recent years. Tung et al., using high-density detailed endocardial and epicardial mapping, reported that most myocardial reentries are characterized by complex three-dimensional (3D) activation patterns and heterogeneous transmuralities, with rare two-dimensional planar configurations (see the activation map in Fig. 1C) [34]. It is no exaggeration to say that state-of-the-art methodologies and technologies have unraveled the complex VT circuitry that results from the extensive and heterogeneous damage to the 3D myocardial architecture caused by structural heart disease.

Careful pre-procedure inspection seems to be essential to finding the decremental conduction properties within the complex substrate in the limited time available during the ablation procedure. Thus, thorough preoperative substrate localization based on surface 12-lead ECG, noninvasive ECG imaging, and contrast-enhanced MRI [18,19,35], as well as sensitive and systematic mapping and ablation, will contribute to improved outcomes.

New ablation and mapping technologies

Omnipolar technology

Omnipolar ECGs are being used increasingly to create ventricular EAMs. An actual example created with use of the Advisor HD Grid Mapping Catheter (Abbott, St Paul, MN, USA) is shown in Fig. 1F. This new approach to voltage mapping makes use of the directionality of the electric field of the wavefront traveling over the myocardial surface. Preliminary studies have shown that omnipolar ECGs are directionally independent and virtual bipolar ECGs aligned along the direction of the wavefront [36]. Omnipolar ECG-based ventricular mapping has been achieved in animal models, but whether this technology is clinically applicable to ablation of VT remains unclear.

Retrograde coronary venous ethanol

RF ablation of VT may fail due to lack of access to the critical substrate. For example, in left ventricular summit VT, the critical substrate may be identified only by mapping the coronary sinus and its tributaries. In multicenter prospective trials, retrograde coronary venous ethanol was shown to be safe and effective and to provide long-term control of drug- and RF-refractory ventricular arrhythmias. In an earlier study, use of single-agent or adjunctive retrograde coronary ethanol was successful in 98 % of patients ($n = 56$), 77 % of whom remained relapse-free at 12 months [37].

Stereotactic body radiotherapy

Radiotherapy is a long-established treatment modality that delivers high-energy X-, gamma-, and photon-rays to abnormal tissue (mainly cancer cells). In recent years, animal models have shown that myocardial irradiation induces percutaneous fibrosis similar to catheter ablation, and radiotherapy has gained attention as treatment for arrhythmias [38]. In a series of 5 patients with refractory VT, high-density surface mapping achieved with a 252-electrode ECG vest was used to characterize the VT and combined with chest CT to define the substrate in a completely non-invasive manner [39]. After treatment, 99.9 % reduction in the total VT burden was observed. However, the evidence supporting non-invasive stereotactic radiotherapy is limited to small case reports and RCTs are needed.

Recent RCTs and future directions

VT is associated with severe cardiac disease, and the occurrence of VT itself reduces cardiac function. The occurrence of VT, particularly occurrence of an ICD shock for VT, predicts subsequent hospitalizations or death from heart failure [40]. Therefore, we can argue, on a biological basis, that preventing VT by catheter ablation reduces heart failure and improves the survival.

The VANISH trial showed a treatment benefit when catheter ablation was added to ICD implantation in patients with ICM but no difference in mortality [4]. Drug-resistant VT is found in patients with relatively severe structural heart disease. The hypothesis that early VT ablation improves mortality and risk of hospital admission seems reasonable, but previous trials have not been sufficiently powered to assess the effect of catheter ablation on mortality [21,22]. Results of two recent RCTs have supported catheter ablation for prevention of sustained monomorphic VT in patients with structural heart disease [41,42]. Importantly, these two trials included patients with NICM in addition to patients with ICM.

The PAUSE-SCD trial [41] aimed to determine whether catheter ablation reduces the composite endpoint of recurrent VT, cardiovascular hospitalization, and death among patients who received an ICD following spontaneous or inducible VT. A total of 133 patients were randomly allocated to ablation or standard (control) treatment. Ablation was performed within 2 days before the ICD implantation. Over a median of 31 months, the primary outcome was reduced to 45 % in the ablation group and 59 % in the control group [hazard ratio, 0.58 (95 % CI, 0.35–0.96), $p = 0.035$]. This difference was driven by a reduction in VT recurrence [31.7 % vs. 50.8 %, hazard ratio, 0.51 (95 % CI, 0.29–0.90)]. In contrast to the smaller PARTITA trial discussed below, there were no significant differences in mortality (8.3 % vs 6.6 %) or cardiovascular hospitalizations (28.3 % vs. 32.8 %). The trial was conducted in Asia (four centers in Japan participated), with relatively few ICM (34.7 %), NICM (30.6 %), and ARVC (34.7 %) cases. Notably, subgroup analysis revealed that, in terms of the primary endpoint, outcomes of ablation were better (though not significantly so) for patients with ARVC or ICM than for those with NICM (64.7 % vs. 50.0 %).

The PARTITA trial [42] evaluated the effect of VT ablation on the composite endpoint of all-cause mortality or hospitalization for worsening heart failure in patients for whom therapy had not failed and were given an ICD. Patients receiving an appropriate shock after ICD implantation were allocated to ablation ($n = 23$) or standard treatment ($n = 24$), and the trial was stopped when the first interim analysis showed that the primary endpoint occurred in 1 patient in the ablation group and 10 in the standard treatment group [hazard ratio, 0.11

(95 % CI, 0.01–0.85); $p = 0.034$]. There were 0 deaths in the ablation group and 8 in the standard treatment group. This difference is striking and noteworthy, but the relatively low number of events requires careful consideration. Ablation significantly reduced the incidence of ICD shocks for recurrent VT (8.7 % vs. 41.7 %, $p = 0.39$). Although it would be tempting to assume that the reduction in arrhythmic events led to the reduction in mortality, only 3 of the 8 deaths were cardiogenic [worsening heart failure ($n = 2$) and cardiac arrest ($n = 1$)]. Therefore, the mechanism by which ablation contributed to the mortality is unclear.

These two trials showed that prompt catheter ablation at approximately the same time as the ICD implantation or after an ICD shock reduced the recurrence of VT. If VT recurs after ICD implantation, it is reasonable to consider immediate ablation to prevent further VT in patients with ICM or ARVC if they have received shock treatment. NICM seems to be more technically challenging, and careful decision-making regarding early intervention by ablation may be warranted. Further studies are needed to clarify whether reducing VT events with ablation reduces hospitalizations and improves the survival.

Declaration of competing interest

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