concept proposes that the substrate of persistent AF is the result of progressive endo-epicardial dissociation, transforming the atria into an electrical double layer of dissociated waves that constantly ‘feed’ each other. Figure 1 gives a diagrammatic presentation of the double layer hypothesis. In Figure 1A and B, a rotor is compared with an epicardial breakthrough. Whereas a rotor at best can maintain itself, endo-epicardial breakthroughs act as multiplication sites of fibrillation waves. Figure 1C illustrates how endo-epicardial (and epi-endocardial) breakthroughs can perpetuate AF. We calculated that in patients with long-standing AF, endo-epicardial breakthroughs can generate > 400 fibrillation waves per second. Also in the present study of Lee et al., > 35% of the fibrillation waves arose from a focal point on the epicardium, distributed over the entire atrial surface. Simultaneous endo-epicardial mapping is required to answer the question of whether long-standing persistent AF is due to a rotor or to endo-epicardial dissociation.

Conflict of interest: none declared.

References
The list of references is available in the online version of this paper.

CARDIOVASCULAR FLASHLIGHT

Figure 1

Positional ventricular tachycardia in left ventricular assist device: a new frontier in ventricular tachycardia ablation

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A left ventricular assist device (LVAD) was implanted in a 54-year-old male patient with severe acute heart failure stemming from dilated cardiomyopathy. Immediately after implant, his condition improved and he could be weaned from mechanical ventilation. However, mobilization was not possible because with any shift away from the supine position, ventricular tachycardia (VT) occurred (Panel A). Treatment with several antiarrhythmic drugs including mexiletine and amiodarone was not successful.

During electrophysiological testing, no VTs could be induced. Substrate mapping of the LV showed no endocardial low-voltage areas < 1.5 mV (Panel B, Carto 3 bipolar voltage map). On fluoroscopy, we detected an unusual position of the LVAD inflow cannula that was more oriented towards the septum than the mitral inflow (Panel C). We inferred that with positional changes, the septum was sucked towards the LVAD inflow cannula leading to mechanical induction of VT. We decided to ablate the myocardium around the LVAD inflow cannula (Panel B) to prevent this phenomenon. During recovery, the patient was successfully mobilized without any further VT occurrences and was discharged 3 weeks later in an ambulatory condition.

In our patient, surgical repositioning of the LVAD cannula seemed inevitable. However, by means of the described catheter ablation we were able to avoid exposing the patient to the risks of a corrective surgery. To the best of our knowledge, this approach of circumferential ablation around the LVAD cannula to treat positional VT has not been previously described.

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