Rebuttal from Sanjiv M. Narayan and José Jalife

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Rather than disputing our position that ‘rotors have been demonstrated to drive AF’ (atrial fibrillation), Allessie & de Groot (2014) actually argue in its favour, with caveats that we will address. They now accept the unquestionable fact that a single rapid rotor can serve as a driver of AF. One small quibble with their welcome acceptance is the statement that the demonstration of rotors is limited to the sheep atria perfused with acetylcholine. Certainly, rotors have been demonstrated in AF in the absence of acetylcholine (Yamazaki et al. 2012) and have also been shown to drive AF in both paroxysmal and persistent AF (Filgueiras-Rama et al. 2012). In actual fact, rotors have been shown to maintain fibrillation in the atria and ventricles of a wide variety of species, including man (Noujaim et al. 2007).

Of more concern, however, is that Allessie & de Groot (2014) overlook a fundamental tenet of scientific enquiry – experiments in which interventions to eliminate a proposed mechanism successfully eliminate the disease should rise above descriptive mapping. Several groups around the world now show that intervention at electrical rotors can eliminate AF (Narayan et al. 2012; Haissaguerre et al. 2013; Lin et al. 2013; Miller et al. 2013). We welcome debate on how to extend these results to more patients, or whether differences in rotor stability reflect technical or other factors. However, we criticize discussions that essentially neglect a plethora of evidence that AF can be sustained by localized rather than spatially diffuse mechanisms. For instance, Allessie & de Groot (2014) fail to point out that several of the studies they cite as showing ‘highly complex’ activation varying ‘from beat to beat’ actually emphasized stable high-frequency sites consistent with drivers, and reproducible vectors consistent with activation from a driver in AF (Gerstenfeld et al. 1992; Sahadevan et al. 2004). We are surprised that Allessie & de Groot (2014) still claim that we ‘represent human AF by just a single map’, and we therefore point to multiple maps and online videos of AF rotors in the CONFIRM trial (Narayan et al. 2012) and other studies. Their expectation of regular signals near a rotor core or ‘anti-phase’ signals at opposite equatorial points of a circular trajectory is a gross oversimplification of AF dynamics, in which rotors precess to produce variable electrograms (Zlochiver et al. 2008). This may explain difficulties in mapping AF by approaches designed for spatially coherent arrhythmias, including attempts to define timing sequences of complex electrograms. We accept that higher spatial resolution would be welcome, and are working to obtain this.

In conclusion, rotors have been demonstrated to drive atrial fibrillation in many patients and model systems. Future studies should define how remodelling contributes to rotor maintenance and fibrillatory breakdown.

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References

Allessie M & de Groot N (2014). CrossTalk opposing view: Rotors have not been demonstrated to be the drivers of atrial fibrillation. J Physiol 592, 3167–3170.


Additional information

Competing interests

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