A major controversy in cardiac electrophysiology is whether ventricular fibrillation (VF) is maintained by wandering wavelets or mother rotors. On the basis of a computer model of atrial fibrillation by Moe developed in 1962, fibrillation was hypothesized to be maintained by multiple small, wandering wavelets, in which the activation pattern is constantly changing. According to the wandering wavelet hypothesis, fibrillation is maintained by constantly changing reentrant circuits formed by some of the wandering wavelets. Because this random reentry can occur anywhere within the cardiac muscle, this hypothesis suggests that all parts of the ventricular myocardium are equally important for the maintenance of VF.

The mother rotor hypothesis was put forward for the maintenance of atrial fibrillation first by Lewis and later by Gurvich. Recently, this hypothesis has been applied to VF by Jalife’s group. This group reported that in isolated guinea pig hearts and in isolated right ventricular slabs of sheep, a fractionation occurred. Supporting this explanation was the wavefronts that gave rise to the disorganized pattern in the ECG. The region containing the mother rotor had a shorter refractory period than the remainder of the myocardium. As opposed to the mother rotor, these daughter wavefronts did not have regular, repeating activation sequences. Instead, activation of the daughter wavefronts was less organized, with changing activation sequences, conduction block, and fractionation. It was these unstable daughter wavefronts that gave rise to the disorganized pattern in the ECG. The region containing the mother rotor had a shorter refractory period than the remainder of the myocardium. Jalife’s group postulated that the reason activation was less organized in the regions outside of the mother rotor was that the longer refractory periods in these regions did not allow them to keep up with the mother rotor, whose cycle length was shorter than their refractory period, so that block and fractionation occurred. Supporting this explanation was the observation that the activation rate in the region containing the mother rotor was faster than in all of the other regions of the myocardium. According to the mother rotor hypothesis, all regions are not equally important for VF maintenance; rather, the region containing the mother rotor is most important. In the guinea pig, this region was found to be in the anterior left ventricle.

Not all studies support the mother rotor hypothesis. For example, Salama’s group did not find evidence for stable, high-frequency regions characteristic of a mother rotor in the isolated guinea pig heart. Ideker’s group mapped VF in in situ pig hearts and found that although rotors lasting for seconds were sometimes observed, they did not produce a net outflow of wavefronts and did not appear to be responsible for sustaining the arrhythmia. Rogers et al performed panoramic optical mapping of almost the entire ventricular epicardium in isolated pig hearts. Again, rotors lasting for several seconds were frequently present on the epicardium; all but one eventually broke down, however, causing that group to conclude that, instead of a persistent mother rotor, continual formation of new rotors is necessary for VF maintenance. Chen’s group has reported that 2 types of VF can occur, with type I consisting of wandering wavelets and type II consisting of a mother rotor with daughter wavelets. Chen’s and Olgin’s groups have provided evidence that the type of VF is influenced by drugs, heart disease, and the duration of VF; therefore, the same heart can exhibit different types of VF at different times, and the same heart can even exhibit different types of VF in different regions at the same time.

All of these studies were performed in animals in which the heart is smaller than the human heart. For example, the guinea pig heart is <2% of the mass of a human heart. There have been only a few previous studies in which activation sequences were mapped in humans during VF. One of these studies recorded from a 14-cm line of electrodes on a catheter within the left ventricle, whereas the 2 other studies recorded from a plaque containing ≈500 electrodes 1.6 to 2.0 mm apart to map activation sequences over a 12- to 20-cm² area of epicardium, which is less than 10% of the ventricular epicardium. All 3 studies found that the wavefronts of VF in humans are large, suggesting that only a few wavefronts are present at any one time and that these wavefronts frequently follow similar activation pathways for several consecutive cycles. The 2 studies that recorded from the epicardium both detected reentry. Reentry was not present within the mapped region the majority of the time, however, and when it was present, it lasted for no more than a few cycles and did not always occur in the same region. Therefore, these studies did not detect a mother rotor during human VF. They mapped from such limited regions, however, that a

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Human Ventricular Fibrillation
Wandering Wavelets, Mother Rotors, or Both?

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mother rotor could have been present (but undetected) elsewhere during VF.

The article by Nash et al in this issue of Circulation provides important new information, because recordings were made from almost the entire ventricular epicardium for 20 to 40 seconds during VF in 10 patients undergoing cardiac surgery. Nash and colleagues found that human VF was characterized by periods in which large coherent wavefronts swept large parts of the epicardium. These wavefronts were generated by a handful of reentrant sources, and at times, only 1 source. This reentry was frequently long lasting; the study by Nash et al illustrates VF episodes in which a rotor was present continuously for 20 to 30 cycles lasting 5 seconds each. The periods of organized activity were punctuated by more complex patterns consistent with wandering wavelet activation.

On the basis of these findings, the authors conclude that both mother rotor and wandering wavelet mechanisms can be responsible for the maintenance of VF in humans. One could argue that the reentrant circuits observed when large, coherent wavefronts were present do not meet the definition of a mother rotor, which is a permanent rotor that is always fixed in location. Although in some cases the rotors were stable in location, in others they drifted across the epicardium. The rotors appeared in different regions in different hearts and even appeared in different regions in the same heart as VF progressed. The authors do not report any instances in which a rotor was continuously present for the entire time that they mapped. The article does not discuss whether the large wavefronts present over most of the epicardium when long-lived reentry was present gave rise to smaller, fractionated wavefronts consistent with daughter wavelets.

Whether or not the definition of a mother rotor is precisely met, the study by Nash and colleagues presents the important finding that VF in humans is frequently highly organized, with large coherent wavefronts arising from 1 or a few reentrant circuits. However, the mapping methods used may have made the wavefronts appear more organized than they actually were. Recordings were made with 256 electrodes dispersed over almost the entire ventricular epicardium, and an average of 171 of these electrodes produced usable signals. Thus, the average distance between electrodes was \( \approx 1 \text{ cm} \). This relatively coarse spatial resolution could have a smoothing effect on VF patterns by bridging gaps between some wavefronts and missing very small wavefronts entirely. The overall picture of VF in the study by Nash et al is largely consistent with the study by the Kay et al., in which a similar proportion of the swine epicardium was mapped, but with much higher resolution \( \approx 1.6 \text{ mm} \). In that study, relatively long-lived epicardial reentrant circuits were also common, with many lasting for seconds. As in the study by Nash et al., these circuits were generally not permanent. Despite this similarity, overall activation patterns were much more complex in pigs than in humans. This difference may be at least partly explained by the difference in spatial resolution.

In addition, Nash and coauthors did not use the usual method for electrical mapping of identifying times of activation when the derivative of the recorded potential reached an activation threshold. Instead, they used a method normally used for optical mapping, in which phase loops were created. The net effect of this approach is that an activation time was always assigned each VF cycle at every electrode except at the few electrodes where a phase singularity was present, even if the signals did not meet an activation threshold because they were over infarct scars, blood vessels, or epicardial fat, which is frequently present in patients undergoing cardiac surgery. Thus, this approach could also have made the wavefronts appear more organized than they actually were.

If we assume that the overall findings of Nash et al are broadly correct, however, they could have important clinical consequences. The findings indicate that VF in humans is not usually maintained by a large number of simultaneous reentrant circuits. Instead, only a few reentrant cycles, and sometimes only 1, appear to be present at any given time. This finding may explain why VF frequently halts spontaneously after a few seconds when it is induced during implantation of a cardioverter-defibrillator and why the spontaneous termination of VF is occasionally observed in Holter recordings. If only 1 or 2 moderately unstable rotors are present during VF, the odds of their spontaneously halting before another rotor appears is much greater than if a large number of rotors are present simultaneously. This finding also raises the hope that pharmacological or low-energy electrical means can be found to increase the probability that these 1 or 2 rotors will halt spontaneously, thus terminating VF.

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