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Azimilide

A Viewpoint by Bramah Singh

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In the evolving reorientation of arrhythmia management, there has been a recent change in therapeutic practice. Accordingly, the role of sodium channel blockers has declined and the use of agents with class III effects (such as amiodarone and sotalol) and the pure class III agents (azimilide, ibutilide and dofetilide) has increased. Azimilide is the first of the class III agents that blocks both components (I_{Kr} and I_{Ks}) of the delayed rectifier potassium current. The resulting lack of rate dependency in repolarisation and cardiac muscle refractoriness is associated with a low incidence of torsades de pointes. However, the proarryhthmic potential of azimilide has not been directly compared with that of other class III agents.

The available clinical data on the haemodynamic and electrophysiological effects of azimilide are predictable on the basis of its known electropharmacological properties. Oral azimilide is well tolerated at dosages of 75 to 150 mg/day. Maximum increments in the QT interval are between 24% and 28%, and no significant increases in the PR or QRS intervals, heart rate or blood pressure have been noted. Moreover, azimilide does not depress ventricular function. These observations suggest a lack of effect on sodium and calcium channels, and on the sympathetic and parasympathetic nervous systems.

In the current changing therapeutic landscape of arrhythmia control, azimilide may have particular value in the acute conversion of atrial flutter and fibrillation. Additionally, it has the potential to maintain sinus rhythm after pharmacological or electrical conversion of paroxysmal and persistent atrial fibrillation. Appropriate studies in patients with atrial fibrillation/flutter, and in postmyocardial infarction patients at high risk of sudden death, have either been completed or are in progress. The outcomes of these studies are likely to more clearly define the role of azimilide in arrhythmia control. ▲