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Sommario/riassunto	With upwards of 4.5 million deaths worldwide each year, and more than one tenth of these occurring in those with no previously documented heart disease, sudden arrhythmic death (SAD) is both a major public health burden and a highly emotive issue for society at large. Recent years have witnessed a marked expansion in our knowledge of the physiology underlying SAD, both in the context of hereditary and acquired cardiac disorders. Thanks largely to work in genetically modified animals, the growth in our understanding of mechanisms underlying arrhythmia in the hereditary channelopathies has been particularly marked. Our growing knowledge of the fundamental mechanisms underlying SAD has so far failed to spur substantial developments in clinical practice. Despite a large body of work in both humans and animals, it remains impossible to confidently identify those at high risk of SAD, making pre-emptive therapy a challenge. What is more, with the thankful exception of the implantable cardioverter-defibrillators and pharmacological agents in very specific situations, there has been depressingly little progress in finding new

and effective therapies. This Research Topic aims to go some way towards bridging the gap between advances in basic science and the development and delivery of new therapies. It brings together original research contributions and review articles from key opinion leaders in the field, focusing on the direct clinical implications of the basic science research now and in the future.