Cardiac Excitation – Contraction Coupling

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Calcium signalling is pivotal for cardiac function, but the complex interaction between cell structure, protein expression and function is far from clear. While the discovery of calcium sparks now forms a cornerstone for our understanding of cardiac excitation-contraction coupling, the problem of calcium spark termination has been resistant to clarification. Using detailed computer models, we now have a robust explanation of calcium spark termination that depends on the detailed microanatomy of the cardiac cell. Furthermore, we have found that disrupted cell anatomy, in the form of de-tabulation, is very closely linked to the loss of contractile performance seen in heart failure. Loss of t-tubules will reduce the efficiency of excitation-contraction coupling but also promote "late calcium sparks" which prolong the calcium transient and would be pro-arrhythmic. These late calcium signalling events are likely to become a new area for intensive study as we attempt to link deranged calcium signalling to arrhythmias and sudden cardiac death.

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