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Mechanotransduction in Cardiac Myocytes

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It is well known that myocardial stretch causes changes in electrical signalling and contractility of the heart. For example, mechanical stretch depolarises the membrane potential of cardiac cells and alters the shape of action potentials. As a result, these effects either accelerate the frequency of heart rate or induce arrhythmias of the heart. Mechanical stress also causes molecular transformations: stretching of cultured neonatal cardiomyocytes increases gene expression, for example, pro-oncogene expression and protein synthesis (hypertrophy). In addition, atrial natriuretic peptide (ANP), which is known to be synthesised and stored in the atrium, is secreted in response to local atrial wall stretch. Several intracellular signalling mechanisms have been shown to be activated by mechanical stretch, including phospholipases, tyrosine kinases, mitogen-activated protein kinases, etc. However, little is known about how mechanical stretch is sensed and transduced into intracellular signals, to bring about its effects. In the present study, we applied direct stretch to rat atrial myocytes using two patch pipettes attached to either end of the cell. We found that stretch of cells resulted in depolarisation of the membrane potential, an activation of stretch-activated cation currents (SAC) and the secretion of ANP. It is concluded that the SAC can be the sensing mechanism of mechanical stretch in atrial myocytes.