Heart cells clear up their cytosolic calcium somewhat dys-synchronously, say Hohendanner et al, and this dysynchrony gets worse in failing hearts.

Calcium is the major intracellular regulator of cardiomyocyte contraction. It is released from its intracellular storage site, the sarcoplasmic reticulum, into the cytoplasm where it then activates the contractile machinery. But after each contraction, the cell must relax—a process that requires the prompt and efficient removal of calcium from the cytosol. This combined release and clear up of calcium is known as a calcium transient. It has been shown that the release of calcium across the cell is synchronized to ensure coordinated control of the contractile machinery, but less is known about the clean-up, or decay phase. Hohendanner and colleagues now show that calcium removal occurs at different microdomains throughout the cell and that the speed of decay varies from domain to domain. Furthermore, the rate of relaxation at adjacent contractile machinery varies in concordance with the differing decay rates of the domains. They also found that the degree of dysynchrony is vastly exaggerated in failing human hearts as well as in mice with hypertrophy and pigs with chronic myocardial ischemia. This, increased dysynchrony slows down the overall rate of relaxation and, the authors suggest that it might contribute to contractile dysfunction associated with cardiac hypertrophy and heart failure.

Meanwhile, mice that lacked IKK2 in their VSMCs showed a decreased hypertensive response to three different vasoconstrictors. Moreover, mice that lacked IKK2 in their VSMCs showed a decreased hypertensive response to three different vasoconstrictors. These results suggest that IKK2 inhibition could be a novel approach for decreasing hypertension.
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