Electromechanical Coupling I. Introduction

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In skeletal muscle fibres contraction is initiated by the action potential which travels inward along the transverse tubular system and causes in some way the release of Ca from the terminal cisternae of the sarcoplasmic reticulum (SR). Ca-release is probably controlled by a potential-sensor (intramembrane charge movement) in the wall of the T-system which in a triad is about 10 – 20 nm apart from the membrane of cisternae. Little is known about the (chemical?) processes which ultimately bridge this gap and which might be associated with the so-called feet or pillars observed between the two systems. Ca ions which enter the fibre during the course of an action potential are not a prerequisite for contraction activation but modify the mechanical response by improving the coupling efficiency and retarding inhibition of Ca-release. During a tetanus or a depolarization the increase in internal Ca^{2+} as observed with the aequorin-method shows a complicated time course with usually two peaks. This suggests that activation occurs in two steps. The latter might probably be related to the phenomenon called Ca induced regenerative Ca-release.

Considering some structural similarities between skeletal and heart muscle cells it appears that EC coupling follows the same principle in both muscle types and that different responses upon activation are rather due to modifications of the same principle and not an expression of completely different processes in EC coupling. The situation may be different in other muscle types.

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