EVALUATION OF SARCOMERE DYNAMICS IN ISOLATED CARDIAC MUSCLE BY LASER DIFFRACTION METHOD

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Introduction:

In order to elucidate the mechanisms of contraction and relaxation of cardiac muscle, direct measurement and control of cardiac sarcomere length, which has been technically difficult to achieve, is required. In this study we measured sarcomere length by laser diffraction method and controlled it by servo-control circuit.

Methods:

Thin papillary muscles were isolated from the right ventricles of rats, submerged in oxygenated physiological salt solution (27°C , $1.9\text{mM}\text{Ca}^{2+}$) and electrically stimulated (12/min). He-Ne laser (5mW) was illuminated to the cardiac muscle and diffracted light was converted to video-signal by photodiode array. Sarcomere length was calculated as $\lambda f/D$, where λ is wave length, f is an optical constant and D is the distance between the 0th order and the 1st order diffraction (Fig.1). Sarcomere length was controlled by the negative feed back circuit (Fig.2). The transient and steady state responses of sarcomere length to a sudden change of load were analyzed.

Result:

Isotonic velocity transients of 20-40msec duration were often

Fig.1. Principle of laser diffraction by cardiac sarcomere.

Fig.2. Schematic diagram for measurement of sarcomere length.

seen after isotonic quick re-
lease following a sudden change
of sarcomere length due to
series elasticity, when the
sarcomere was held isometric
before the isotonic quick re-
lease (Fig.3). These velocity
transients resembles those
observed in skeletal muscle.
However, the velocity transients
were not observed when the
muscle preparations were held
isometric, in which the internal

shortenings of sarcomeres were seen. The time average of the transient velocity was approximately equal to the final steady velocity, which was determined by the unique force-velocity relation. The force-velocity relation of the cardiac sarcomere for shortening looked like hyperbaric, similar to the one reported by A.V.Hill in skeletal muscle. On the other hand, the force generated by cardiac sarcomere during lengthening increased toward the constant value when the lengthening velocity increased larger than $4\mu\text{m}/\text{sec}/\text{half sarcomere}$. With the increase in time of the quick release after the stimulation, the sarcomere became less able to shorten at any relative load and progressively more resistant to lengthening.

Conclusion:

The sarcomere length of cardiac muscle was successfully measured by laser diffraction method and was successfully controlled by the negative feed back circuit. The dynamics of cardiac sarcomere had a resemblance to those of skeletal muscle, indicating the presence of basic similar mechanism of cross-bridge kinetics between the cardiac and skeletal muscle.

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