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原著

末梢神経障害による骨格筋内部膜機能変化

西 島 博 明

川崎医療福祉大学 医療技術学部 健康体育学科

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Changes of Ca-release Mechanism on Sarcoplasmic Reticulum in the Neurogenic Atrophic Skeletal Muscle

Hiroaki NISHIJIMA

Department of Health and Sports Sciences Faculty of Medical Professions Kurashiki, 701-01, Japan

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Abstract

It is known that the function of skeletal muscle is dependent on the integrity of the relevant motornerve. The contraction of skeletal muscle ~1'as caused by the Ca release from the sarcoplasmic reticulum (SR) which was induced by the depolarization of T-tubular membranes with the nerve-stimulus. Therefore, it has been speculated that the function of Ca release channels on the SR tightly contacted with the voltage dependent Ca channels in the T-tubular membranes. However, the relationship of both Ca-channels remains the key unresolved problem in excitation-contraction coupling. In this experiment, the physico-chemical aspects of Ca release channels on the SR in the atrophied skeletal muscle which was induced by the defection of the relevant motornerve were examined. While the Ca-uptake in SR decreased the dependence on the denervated periods, the Ca-release from SR by caffeine vanished rapidly. However, the vanished Ca-release from SR recovered with the innervation of the relevant nerve. When the ryanodine binding in the Ca-release channels on SR was examined, the affinity for ryanodine on the SR in the denervated muscle was reduced in height, with no change in the maximum number of binding sites. These results indicated that the changes in phvsicochemical aspects of the voltage dependent Ca-channels on T-tubular membranes worked a revolution in that of Ca-release channels as the ryanodine binding sites.

要 約

骨格筋の収縮一弛緩障害の成因は筋自体と支配神経に起因する場合が多い.末梢神経障害によって引き起こされる骨格筋の萎縮は支配神経の再修復によって回復することが知られている.一方,神経刺激は支配骨格筋筋鞘T骨膜の脱分極を導く,この情報が筋細胞内Caイオン貯留部位である小胞体膜からのCaイオン遊離を引き起こす.骨格筋の収縮は小胞体膜からのCaイオン遊離によって引き起こされる.即ち,T管膜電位依存性Ca-channelと筋小胞体膜のCa remease channelの機能は密接に連携しているとされる.　そこで本実験は骨格筋の支配神経を離断した場合に支配骨格筋の筋小胞体膜Ca release channel機能がどのような変化を受けるか,さらにT管膜電位依存性Ca-channe1との関係について検討した.その結果,脱神経骨格筋小胞体膜Ca遊離は脱神経によって消失し,神経再支配によって骨格筋小胞体膜のCa遊離機能は回復することが明らかとなった.しかし,筋小胞体膜のCa release channe1開口の指標となるryanodine結合を調べると,脱神経筋小胞体膜Ca release channelのryanodine結合のBmaxは正常小胞体膜のCa re1ease channelのryanodine結合のBmaxと同等であるにも拘わらず,Kd値は明らかな相違を示した、この結果はDihydropyridine系薬剤によってT管膜のL型Ca-channelの抑制によって引き起こされる小胞体膜Ca-channelの抑制とは異なる結果である.従って,T骨膜のL型Ca-channelの蛋白構造変化が小胞体膜のCa release channelに直接連動している可能性を示唆していると同時にT管膜のL型Ca-channelの蛋白構造変化には幾つかのプロセスが有るように考えられる.