

持久性トレーニングによる運動時換気応答の抑制には中枢性（脳）の適応メカニズムがどの程度関与するのか？

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How Does the Central Adaptative Mechanism Contribute to the Attenuation of Exercise Hyperpnea from Strenuous Regular Exercise Training?

by

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ABSTRACT

Background: We have shown that minute ventilation [$\dot{V}E$] and end-tidal CO_2 tension [$P_{ET}CO_2$] were determined by the interaction between the properties of controller and plant. During exercise, the controller shifted to the direction of decreased $P_{ET}CO_2$, so as to compensate for the shift of plant accompanying increased metabolism. This effectively fixes $P_{ET}CO_2$ in the normal range, with the expense of exercise hyperpnea. We examined how athletes are trained to reduce this exercise hyperpnea.

Methods: In 6 trained (Tr) and 6 untrained (UT) healthy males, to characterize the controller, we induced hypercapnia by changing inspiratory CO_2 fraction and measured the linear $P_{ET}CO_2$ - $\dot{V}E$ relation ($\dot{V}E = S \cdot (P_{ET}CO_2 - B)$). To characterize the plant, we

made subjects alter $\dot{V}E$ and measured the hyperbolic $\dot{V}E$ - $P_{ET}CO_2$ relation ($P_{ET}CO_2 = A / \dot{V}E + C$). We characterized these relations both at rest and during light exercise.

Results: Physical conditioning did not affect characteristics of either controller or effector during rest. Exercise decreased B in UT, while not in Tr ($p < 0.05$). During exercise, slope S slightly increased in both groups. The hyperbolic plant property shifted right and upward during exercise as predicted by increased metabolism. Though constant C was slightly lower in Tr than that in UT, this does not contribute much to changes in $\dot{V}E$. The $\dot{V}E$ during exercise in Tr was by 22 % lower than that in UT.

Conclusion: The attenuation of exercise hyperpnea induced by regular exercise training results mainly from the adaptation of the controller, the lower sensitization of chemoreflex controller. Strenuous regular exercise training almost abolishes the exercise-induced shift of central ventilation controller.

要 旨

背景：先行研究にて，ヒト呼吸化学調節系の特性を制御部（中枢コントローラ）と被制御部（末梢プラント）に分離し定量解析することで，安静時や運動時の換気量決定機構を定量評価する方法論を開発した．本研究は，同法を用いてアスリーートの運動時換気抑制機構の詳細を明らかにすることを目的とする．方法：対象は日々持久性運動トレーニングを実施している男性アスリート6名（Tr群）とコントロール群6名（UT群）．中枢コントローラ特性（動脈血[呼吸終末] CO_2 分圧（ $P_{ET}CO_2$ ）→分時換気量（ $\dot{V}E$ ）関係）を調べるために，一定濃度の CO_2 を吸入させ， $\dot{V}E$ および $P_{ET}CO_2$ を測定した．末梢プラント特性（ $\dot{V}E$ → $P_{ET}CO_2$ 関係）を調べるために，一定の分時換気を意識的に行わせた．いずれも安静時と低強度運動時の定常状態で測定した．結果：運動時の中枢コントローラ特性（ $\dot{V}E = S \cdot (P_{ET}CO_2 - B)$ ）の傾きS値は両群間で差を認めなかったが，X軸切片B値はTr群がUT群よりも高値を示した（ $p < 0.05$ ）．運動時の末梢プラント特性（ $P_{ET}CO_2 = A / \dot{V}E + C$ ）のX軸漸近線C値はTr群がUT群より

もやや低値を示した．両サブシステムの交点（動作点）の $\dot{V}E$ 値はTr群がUT群よりも22%有意に低値を示した．両群間の換気反応の差異は中枢コントローラ特性の違いによってほぼ説明できた．結論：アスリートに見られる低強度運動時の換気抑制反応は，長期トレーニングに伴う中枢性（脳）の適応変化が主たるメカニズムであることが明らかとなった．