Influence of aldosterone on aerobic metabolism during increasing bicycle ergometer exercise

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Introduction
In earlier studies we found that the application of aldosterone induced an increase of the maximal oxygen uptake (VO₂max) by about 10% [1]. In the present study we investigated in which range of submaximal exercise an increased level of aldosterone would elevate the aerobic metabolism.

Methods
Two sets of experiments were performed with 10 male subjects. In aldosterone experiments (AE), 0.5 mg Aldocorten was injected subcutaneously 5 and 3 hours before starting exercise, while in control experiments (CE), a placebo (NaCl) was applied. Work load was increased stepwise by 40W every 3 min until the level of exhaustion was reached. The ventilatory parameters were determined using breath by breath computerized analysis. Half of subjects performed CE 7 days before AE, the second half vice versa.

Results
VO₂ during exercise increased significantly more in AE. Differences amounted to about 100ml/min in the biginning of work load and increased during the final steps. VO₂max was augmented in AE by about 400ml/min or 10.8±12.4% (p<0.005) (Fig. 1). In AE period of last exercise intensity was prolonged by approximately 0.5min resulting in an elevation of total exercise by 8.1±15.6kJ (p<0.05). Acid base values of blood pressure before and after exercise did not differ between AE and CE equivalent to corresponding experiments with swimming exercise [2].

Discussion/Conclusion
By augmentation of the aerobic part of exercise during AE resulting in a lower degree of anaerobic metabolism, subjects had been able to extend their working time until acid base values reached values of CE. Combined with the augmented VO₂ in AE, especially during heavy exercise, a significant increase of oxygen pulse can be concluded. A suppression of lactate production caused by the augmented VO₂ in AE is demonstrated in our second experiments with constant defined exercise intensity [3]. In the present study the heart rate influenced by lactate concentration increased slower during AE up to the final phase with same values as in CE. (Fig.2). Starting in the range of endurance capacity, aldosterone enhances VO₂ in relation to the same level of heart rate. Respiratory values are giving no indication for an augmented oxygen extraction of blood. By this, an inotropic effect of aldosterone [2], [4] must be considered as the second main point of our results.

References