Therapeutic Response of Idiopathic Edema to Captopril

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Sir, References

I have read with interest the report by Docci et al. [1] concerning the therapeutic response of idiopathic edema to captopril. The place of the renin-angiotensin-aldosterone (RAA) system in the pathogenesis of idiopathic edema is not clear. There is a hypothesis about an exaggerated aldosterone response to the upright posture [2]. It seems likely that in most, otherwise healthy women, there is a stimulation of the RAA system by diuretics [4] or/and sudden increase in sodium and carbohydrate intake after periods of starvation and sodium deprivation [3,4]. After stopping diuretics PRA fell in all patients to normal or below [5], but that the edema and weight gain can persist, suggests that sodium continued to be retained in these patients by some additional mechanism like a peripheral resistance to the action of the natriuretic factor. So in this view the use of captopril appears to be an inadequate pathophysiological approach.

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