Renin-angiotensin-aldosterone system during acute hypoxemia

To the Editor: I read with great interest the article "Effect of hypoxemia on the renin-angiotensin-aldosterone system in humans" by Colice and Ramirez (2). They describe the phenomenon of decreased plasma aldosterone concentration despite no change in plasma renin activity or serum angiotensin-converting enzyme (ACE) activity during acute hypoxemia. In addition, they contribute further to our understanding by showing that the decrease in aldosterone was not due to inhibition of adrenocortical sensitivity to exogenous adrenocorticotropic hormone (ACTH) or to an increase in hepatic blood flow (i.e., steroid metabolism). They conclude that the mechanism for the decrease in aldosterone is still not understood.

These conclusions are in agreement with some previous studies in animals that failed to show a decrease in ACE assessed either by [3H]benzoyl-l-phenylalanyl-l-alamyl-l-proline metabolism (4) or by correlation of measured angiotensin II and plasma renin activity (5). What is disconcerting about their study is the lack of measurement or discussion of plasma potassium. Plasma potassium is a very important and potent stimulus to various studies in animals that failed to show a decrease in plasma potassium. Plasma potassium change during acute hypoxemia may be responsible for the observed decrease in aldosterone during acute hypoxemia despite no change in plasma renin activity, serum ACE, or adrenal sensitivity to pharmacological levels of ACTH. One could argue that if plasma potassium was decreased, the aldosterone response to exogenous ACTH should have been attenuated. However, pharmacological levels of ACTH can probably overwhelm this subtle physiological interaction. Even if a decrease in plasma potassium is not the mechanism, plasma electrolytes should be measured in studies examining the physiological control of aldosterone secretion.

REFERENCES


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REPLY

To the Editor: We agree with H. Raff’s position that potassium levels are an important controlling mechanism of aldosterone secretion. However, we do not feel that changes in potassium levels explain the fall in plasma aldosterone concentrations (PAC) observed in our subjects. Claybaugh and associates (1) found that 2 h of hypoxemia caused an increased serum potassium but a decreased PAC, whereas Heyes et al. (4) noted that both serum potassium and PAC fell after 1 h of hypoxemia. This work indicates to us that acute hypoxemia causes significant falls in PAC without a consistent change in serum potassium levels. We also feel that this conclusion is consistent with the results of more prolonged studies at altitude. Serum potassium levels have been reported to increase following decreases in PAC at high altitude (3, 5). Furthermore, potassium retention (increased plasma potassium levels and decreased urinary potassium excretion) has been found to occur despite respiratory alkalosis at high altitude (6). These effects on potassium with ascent to high altitude, which coincide with decreases in PAC, indicate that hypoxemia-mediated falls in aldosterone determine potassium levels and not that alkalosis-induced decreases in potassium cause the decrement in PAC. However, the latter studies were conducted over several days, rather than the 1- to 2-h experiments we performed. Information on the relationship between alkalosis, potassium levels, and PAC during 1 to 2 h of hypoxemia is sparse.

REFERENCES


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Hemodynamic and metabolic effects of exercise in Crotalaria-induced pulmonary hypertension

To the Editor: In their paper (J. Appl. Physiol. 57: 1829-1833, 1984), L. J. McNabb and K. M. Baldwin state in the first paragraph of their discussion that monocrotaline has been shown to produce pulmonary hypertension with right ventricular hypertrophy in several animal species including humans. In support of this statement they quote the paper “Experimental chronic pulmonary hypertension” published by Herget and Palacké (Int. Rev. Exp. Pathol. 18: 347-406, 1978. These authors refer to monocrotaline producing pulmonary hypertension only in rats and monkeys. There is, of course, no mention of this alkaloid producing pulmonary hypertension in human subjects.

One of the intriguing things about the pyrrolizidine alkaloids is the wide species variation in their affects on the lung and pulmonary vasculature. I am not aware of any reports of pyrrolizidine alkaloids producing pulmonary hypertension in human subjects. Some years ago we examined the lungs of West Indian patients who had died of hepatic venoocclusive disease following the ingestion of pyrrolizidine alkaloids in bush tea. Their pulmonary blood vessels were normal.

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REPLY

To the Editor: J. M. Kay is correct in stating that pyrrolizidine alkaloids have not been shown to produce pulmonary hypertension in human subjects.

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