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DIURNAL PROFILES OF PLASMA CORTISOL,
ALDOSTERONE, RENIN, ANGIOTENSINOGEN
AND ANGIOTENSINASES IN NORMAL SUBJECTS

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The synthesis of aldosterone is regulated, among other factors, by ACTH and renin (angiotensin II). Similar to plasma cortisol (ACTH), plasma aldosterone and renin follow a circadian rhythm which is interrupted by irregular episodic changes. The present study was performed to examine diurnal variations of aldosterone in comparison with those of cortisol, renin, angiotensinogen and angiotensinases.

Plasma cortisol and renin were estimated in 1 h intervals, plasma aldosterone, angiotensinogen and angiotensinases in 3 h intervals over a period of 24 h in 6 normal male volunteers (age 20—26) under control conditions and under subsequent suppression of ACTH release by dexamethasone.

Under control conditions, the highest cortisol levels were found at 7 a.m. (21.5 ± 3.7 (SD) and 14.8 ± 4.3 µg/100 ml), minimum levels between 9 p.m. and 1 a.m. Between 10 a.m. and 3 a.m. mean cortisol concentrations varied from 1.4 ± 1.9 to 7.8 ± 4.8 µg/100 ml. Dexamethasone reduced cortisol to mean values below 1 µg/100 ml throughout the 24 h period.

Aldosterone was highest at 4 a.m. under control conditions (165 ± 51 pg/ml) and under dexamethasone (190 ± 63 pg/ml), and showed the lowest concentrations between 4 a.m. and 10 p.m. There were no significant differences between mean aldosterone concentrations at corresponding time points within the control and the dexamethasone period. However, the 24 h mean value was significantly increased under dexamethasone in 3 of the 6 subjects.

Similar to aldosterone, renin showed peak values at 4 a.m. (12.1 ± 3.4 (control) and 14.2 ± 3.5 (dexam.) ng angio I/ml-h). Under the influence of dexamethasone, all values between 7 a.m. and 11 p.m. were significantly increased, and the 24 h mean value was significantly higher in all subjects.

No evidence could be found for the existence of circadian rhythms of angiotensinogen and angiotensinases. There were no significant differences between the mean values of these parameters at corresponding time points within the control and the dexamethasone period.

The data suggest that circadian rhythms of aldosterone and renin are similar to each other but not exactly synchronized to that of cortisol. When ACTH release is inhibited by dexamethasone, renin increases to maintain normal or slightly elevated aldosterone levels. Angiotensinogen and angiotensinases, parameters which may influence the active concentration of angiotensin II, do not seem to be involved in the regulation of aldosterone.