ANALYSIS OF DAILY RHYTHMS OF ADRENAL FUNCTION IN MEN WITH QUADRIPLEGIA DUE TO SPINAL CORD SECTION[1]

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INTRODUCTION

THE reviews of Nichols and Tyler (1967) and Halberg and Reinberg (1967) summarise 25 years of studies on circadian rhythms of physiological functions in man. Little information is available on the possible alteration of these rhythms by lesions of the spinal cord that cause interruption of afferent pathways.

We have studied adrenal function in patients paralysed by a section of the spinal cord at C4, C5, or C6 (hereafter called quadriplegics) in order to learn if isolation of the spinal cord sympathetic chain diminishes feedback information to the part of the brain that controls hypothalamic secretion, resulting in deletion of established daily rhythms of adrenal cortical and medullary function. This hypothesis was suggested by the results obtained by Vallbona, Lipscomb and Carter (1966), who found that adrenal responses elicited by a massive fall in blood pressure in intact subjects are absent in quadriplegics. The study was also done to gain information on the role of the central nervous system in the modulation of aldosterone rhythms.

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METHOD

Subjects. Eleven male quadriplegic patients were studied. All had suffered spinal cord injury and from a clinical standpoint had a complete transection of the cervical spinal cord. One of the patients who subsequently showed signs of voluntary motor recovery was deleted from the study. Age, level of injury, time elapsed between injury and study and the analyses performed on plasma and urine are listed in Table I. The patients were divided into acute and chronic, taking eight months of quadriplegia as the criterion for the classification.

The initial experimental design called for studies during weekends to avoid the possible effect of physical and occupational therapy upon daily rhythms.

No relationship could be detected between the nursing notes regarding the patient's physical activity and emotional functioning and the values of corticosteroids excreted. Thus, the other patients were studied for six consecutive days starting on Monday regardless of activity schedule. The study on patient C.1 was interrupted because of the interference of medication on the measurement of 17-hydroxycorticosteroids (17-OHCS). Serial blood sampling was discontinued because most patients had hypersensitive antecubital areas, became very upset by withdrawal of blood, and refused to continue the experiment when large quantities of blood were needed. Only methylhydroxymandelic acid (MHMA) and 17-OHCS were measured in patients B.1, B.2, B.3 and C.1.

The patients' intake of electrolytes was contained in their meals, taken at regular hours. There was a mean of $170 \pm 13{\cdot}6$ mEq. of sodium and $89 \pm 2{\cdot}3$ mEq. of potassium ingested daily that was regularly distributed within each week (range: Na, 158 to 178 mEq.; K, 79–95 mEq.).

Endocrine and Metabolic Studies. The chemical analyses were performed on all the urines collected over periods of six hours, ending at 2 p.m., 8 p.m., 2 a.m. and 8 a.m. The night samples are those collected from 8 p.m. to 2 a.m. and from 2 a.m. to 8 a.m. The analyses included measurement of: (1) urinary metabolites of catecholamines (methylhydroxymandelic acid (MHMA) by the method of Pisano and Crout (1962); (2) urinary metabolites of cortisol (17-OHCS) by the method of Glenn and Nelson (1953), modified by replacement of the silica column by paper chromatography in three solvent systems; (3) urinary acidlabile conjugate of aldosterone by a single-isotope labelling method; (4) sodium and potassium by flame photometry.

Fluid balance was obtained by measuring the differences between volumes of fluid intake and urine output during each period. The amounts of special solutions used to irrigate the indwelling catheters were subtracted from the output.

Blood was collected at 8 a.m., 2 p.m., 8 p.m. and 2 a.m. and analysed for 17-OHCS by the method of Nelson and Samuels (1952) and subsequently by the formation of an isotopic derivative. The new methods were developed to obtain a greater specificity in the measurement of the steroids or group of steroids in order to permit the study even when the patient received anabolic steroids and many other drugs. The method for plasma cortisol uses only one millilitre of plasma.

In these methods, the steps for steroid isolation are: (1) hydrolysis, either acid for the urinary acidlabile aldosterone conjugate and enzymatic hydrolysis

TABLE I

Subjects Studied and Tests Obtained[a]

Group	Name	TIRR No.	Age at injury (years)	Duration[b]	Level of cord section	Plasma 17-OHCS	Urine Na	Urine K	Urine Fluid bal.	Urine MHMA	Urine 17-OHCS	Urine Aldosterone
Acute B.1	R. C.	17490	17	2 mo.	C4	2	—	—	—	15	15	—
B.2	R. W.	17448	31	1 mo.	C6	1	—	—	—	10	15	—
B.3	S. W.	17277	15	1 mo.	C4, C5	2	—	—	—	15	15	—
B.4	A. F.	17762	23	3 mo.	C6	—	6	6	6	6	6	6
B.5	P. W.	17857	18	2 mo.	C5, C6	—	6	6	6	6	6	6
B.6	D. S.	17957	16	2 mo.	C5	—	6	6	6	6	6	6
Chronic C.1	R. H.	15488	34	3 yr.	C6	1	—	—	—	8	8	—
C.2	B. J. E.	16942	19	1 yr.	C3, C4	—	5	5	5	5	5	5
C.3	L. F. E.	17846	44	9 mo.	C6	—	6	6	6	6	6	6
C.4	B. M.	15522	19	13 yr.	C5, C6	—	6	6	6	6	6	6

[a] = The figures in the table identify the number of days during which four samples of urine or blood were analysed.
[b] = Time elapsed between the injury and the start of the study.

for the urinary 17-hydroxycorticosteroids (17-OHCS); (2) organic solvent extraction of the corticosteroids and; (3) alkaline, acid and aqueous washing of the organic extracts of steroids.

The crude extracts are purified by three or four sequential partitions using the paper chromatographic systems described by Zaffaroni (1953). After three chromatographic separations, the 17-OHCS are still near the origin and the 4th solvent system will separate these from each other. For the measurement of 17-OHCS, the mixed steroids are eluted after the 3rd separation. Half of each eluate is analysed by the Porter-Silber reaction, the other half is used as a blank to obtain the optical density of the phenylhydrazones by spectrophotometry. For the measurement of aldosterone or cortisol, aldosterone (mixed with cortisone) or cortisol (mixed with tetrahydrocortisone) is eluted after the 4th paper chromatographic separation and acetylated with 100 μg. (2 microcuries) of ^{14}C-acetic anhydride (specific activity, 2 mC./millimole). The labelled acetates are extracted by liquid partition and isolated by paper chromatography after addition of stable carriers. The beta emissions of the isotopic derivatives are counted by liquid scintillation. Concentrations are calculated from data obtained by acetylation and chromatographic separation of pure steroid standards and suitable blanks (Claus-Walker, 1968).

Analysis of Data. The raw data were analysed by computer to obtain ratio of variance 'F' between periods and total variance, between patients and total variance, and between patients or groups of patients and periods. The significance of the 'F' values was established at three levels: 0·001, identified by three asterisks; 0·01, by two asterisks; and 0·05, by one asterisk.

The same statistical analysis was repeated using the data transformed to percentages of daily output. This was done to offset individual variation in daily excretion that might mask daily rhythms.

RESULTS

In the results calculated from raw data there are differences for some of the values obtained in samples from different periods, but there are also large variations due to the patients, especially for 17-OHCS and for aldosterone. There is also interaction of periods and patients for Na and K in the chronic group. Transformation of the data to percentages of daily output eliminates the variations due to the patients and can thus be used to evaluate the differences due to sampling periods. The tables illustrate the mean values of the raw data, the transformed data, and their variance ratio.

1. *Data on excretion of electrolytes, aldosterone and water in three chronic* (C) *and three acute* (B) *quadriplegics during six consecutive days.*

The data on *fluid balance* show an increased fluid retention from 8 a.m. to 2 p.m. This retention is of greater magnitude and significance in the acute group than in the chronic (370 ml. vs 330 ml. 'F' = 12·451*** vs 5·123**) (table II).

The maximum *sodium* (Na) excretion is between 8 a.m. and 8 p.m. It is significant in the chronic only (63 per cent., 'F' = 11·833***) (tables III and IV). Groups C and B are different.

The maximum *potassium* (K) excretion occurs from 8 a.m. to 8 p.m. It is

significant in the chronic (C) and acute (B) patients (62 per cent., 'F' = 6·006*** and 57 per cent., 'F' = 4·752**) (tables III and IV). Groups C and B are different.

Excretion of *aldosterone* shows no significant rhythms and no group differences (tables III and IV). The daily excretion was larger in the acute group.

TABLE II

Fluid Balance in three Chronic (C) and three Acute (B) Quadriplegics

Raw Data (ml.)

Group	Days	Sampling periods			
		8 a.m. to 2 p.m.	2 p.m. to 8 p.m.	8 p.m. to 2 a.m.	2 a.m. to 8 a.m.
C	17	330	70	6	—282
B	18	370	123	—102	—212
		Ratio of variance			
		Within groups (Periods/Total)		Between groups C and B (Periods/Total)	
C	17	5·123**		—	
B	18	12·451***		14·164***	

* Significant at 0·05 level. ** Significant at 0·01 level.
*** Significant at 0·001 level.

2. *Data on excretion of 17-OHCS and MHMA in four chronic and six acute quadriplegics.*

Analysis of variance from raw data shows very large variations due to the patients and no variation due to the sampling periods (table V) whereas the same analysis performed on transformed data discloses the presence of 17-*OHCS* rhythms with significant maximum excretion between 2 p.m. and 8 p.m. in the chronic group (28 per cent., 'F' = 9·220***) and 8 a.m. to 8 p.m. in the acute group (62 per cent., 'F' = 2·643*). The analysis of the transformed data also shows a significant decrease in the excretion of *MHMA* between 2 a.m. and 8 a.m. in the chronic quadriplegics (23 per cent., 'F' = 2·861*) (table VI).

Table VII shows the concentration of cortisol in the plasma of three acute and one chronic quadriplegics and six healthy men during bedrest. The data for the control study were obtained previously at the Texas Institute for Rehabilitation and Research (Cardus *et al.*, 1965). Results on quadriplegics show a much larger standard error from the mean than those on controls. Analysis of variance shows that plasma cortisol levels at the four sampling times are significantly different in the control group only.

TABLE III

Electrolytes and Aldosterone Excretion in three Chronic (C) and three Acute (B) Quadriplegics

Raw Data

Metabolite	Group	Days	Sampling periods: 8 a.m. to 2 p.m.	2 p.m. to 8 p.m.	8 p.m. to 2 a.m.	2 a.m. to 8 a.m.
Sodium (mEq.)	C	17	40***	37**	21	20
	B	18	40	41	41	33
Potassium (mEq.)	C	17	14**	12**	7	8
	B	18	13	11	11	9
Aldosterone (μg.)	C	17	2·13	2·53	2·44	1·34
	B	18	5·20	6·75	8·03	6·48

Ratio of Variance within Groups

Metabolite	Group	Periods/Total	Patients/Total	Patients/Periods
Sodium	C	10·894***	9·875***	3·004**
	B	0·697	3·008	0·974
Potassium	C	5·626**	9·110***	2·445**
	B	1·674	0·387	0·754
Aldosterone	C	1·187	0·972	0·769
	B	0·353	5·125**	0·291

Ratio of Variance between Groups C and B

Metabolite	Periods/Total	Groups/Total	Periods/Groups
Na	4·383**	9·307**	2·157
K	4·989**	0·679	1·211
Aldosterone	0·465	17·985***	0·275

TABLE IV

Electrolyte and Aldosterone Excretion in three Chronic (C) and three Acute (B) Quadriplegics. Results expressed as Percentages of Daily Output[a]

Raw Data

Metabolite	Group	Days	Sampling periods: 8 a.m. to 2 p.m.	2 p.m. to 8 p.m.	8 p.m. to 2 a.m.	2 a.m. to 8 a.m.
Sodium (mEq.)	C	17	31***	32***	19	18
	B	18	27	27	25	21
Potassium (mEq.)	C	17	32***	30***	19	19
	B	18	31**	26**	23	20
Aldosterone (μg.)	C	17	24	28	29	28
	B	18	27	20	25	28

Ratio of Variance

Metabolite	Group	Days	Within groups (Periods/Total)	Between groups (Periods/Total)
Sodium	C	17	11·833***	—
	B	18	1·308	10·125***
Potassium	C	17	6·006***	—
	B	18	4·752**	11·682***
Aldosterone	C	17	1·217	—
	B	18	0·881	0·419

[a] =These values are brought to the next decimal for fractions greater than 0·5.

DISCUSSION

The fluid balance studies could not be compared to other studies because normally the night urine accumulates in the bladder and is voided in the morning, whereas in the quadriplegics with catheters it is collected as soon as formed and truly reflects the urinary excretion. Both chronic and acute patients retained fluid during daytime (when intake is maximum) and have a negative balance during the night. The rhythm is stronger in the acute than in the chronic patients. This may be due to their life schedule: immediately after onset of quadriplegia due to spinal cord injury, the immobilised patients are passively turned and tilted slightly

TABLE V

Excretion of 17-OHCS and MHMA in four Chronic (C) and six Acute (B) Quadriplegics

Raw Data

Metabolite	Group	Days	Sampling periods: 8 a.m. to 2 p.m.	2 p.m. to 8 p.m.	8 p.m. to 2 a.m.	2 a.m. to 8 a.m.
17-OHCS (mg.)	C	25	2·05	2·38	1·75	1·76
	B	58	0·82	0·93	0·76	0·77
MHMA (μg.)	C	25	0·85	0·92	0·93	0·84
	B	58	1·07	0·93	0·81	1·04

Ratio of Variance within Groups

Metabolite	Group	Periods/Total	Patients/Total	Patients/Periods
17-OHCS	C	1·571	78·617***	0·230
	B	1·073	10·290***	0·549
MHMA	C	1·927	4·367**	1·266
	B	0·573	7·817***	1·215

Ratio of Variance between Groups

Metabolite	Periods/Total	Groups/Total	Periods/Groups
17-OHCS	1·042	56·265***	0·491
MHMA	0·081	1·561	1·855

(Spencer *et al.*, 1966). Later on they are subjected to a more active programme of rehabilitation therapy and resume a new rhythm of activity. It is of significance that we 'impose' a regular 24-hour rhythm in these quadriplegics by the very nature of our rhythmic hospital treatment routine. Changes in blood distribution brought about by postural changes stimulate baroreceptors in the atria and the carotid arteries. These reflexes travel through the vagus (Share *et al.*, 1966) and modulate the tonic inhibition of antidiuretic hormone (ADH, vasopressin). Upright (tilt) or sitting position increases the release of ADH as reflected by a positive fluid balance.

The excretion of sodium has no rhythm in the group of acute quadriplegics and has a flattened normal rhythm in the chronic quadriplegics. As neither group

TABLE VI

Excretion of 17-OHCS and MHMA in four Chronic and six Acute Quadriplegics. Results expressed as Percentages of Daily Output.

Raw Data

Metabolite	Group	Days	Sampling periods: 8 a.m. to 2 p.m.	2 p.m. to 8 p.m.	8 p.m. to 2 a.m.	2 a.m. to 8 a.m.
17-OHCS	C	25	26	28***	21	25
	B	58	30*	32*	19	19
MHMA	C	25	25	26	27	23*
	B	58	28	24	21	27

Ratio of Variance

Metabolites	Group	Within groups (Periods/Total)	Between groups (Periods/Total)
17-OHCS	C	9·220***	—
	B	2·643*	7·432***
MHMA	C	2·861*	—
	B	2·110	0·433

shows periodicity in aldosterone excretion that would coincide with sodium retention, it is likely that modulation of sodium excretion is also under control from CNS via nervous afferent pathways that are disrupted in the early stages after spinal cord injury and reappear after rehabilitation. This suggests the important role played by the CNS in the conservation of sodium (Anderson *et al.*, 1957; Wolf, 1967).

The excretion of potassium has a flattened pattern in both groups, but the daily peak is higher in the chronic patients.

Because of quadriplegia, the patients are more immobilised than normal subjects, but very early after the onset of paralysis they are submitted to physical therapy, occupational therapy; they are tilted and they spend part of the day in wheelchairs. Thus one would expect the excretion of aldosterone to increase during the day. There is no rhythmical pattern of aldosterone excretion in either group. Regulation of aldosterone is in part controlled by catecholamines secreted following postural activation of the vasomotor centre or by sodium depletion: autonomic insufficiency depresses both (Gordon *et al.*, 1967). Autonomic insufficiency is severe in the quadriplegics. In health, there is an intrinsic pattern

TABLE VII

Concentration of Cortisol in the Plasma of three Acute (B) and one Chronic (C) Quadriplegics and in the Plasma of six Healthy Men during Bedrest (Cardus *et al.*, 1965) during a 24-hour Period (Results in micrograms in 100 ml. plasma)

Subject	Duration of illness at test	Sampling periods				
		8 a.m.	2 p.m.	8 p.m.	2 a.m.	
B1	66 days	16·90	20·00	7·70	—	
	142 days	5·53	3·57	1·78	0·71	
B2	17 days	5·06	8·22	9·73	6·33	
	6 months	6·84	5·00	1·73	0·00	
B3	40 days	14·90	9·80	—	20·00	
	49 days	14·60	14·30	8·90	11·40	
C1	3 years	12·40	16·00	—	20	
		8·70	20·00	25·70	7·20	
Mean and SEM .		10·49 ± 1·68	11·98 ± 2·29	8·51 ± 2·71	8·57 ± 2·81	F = 0·25

	Sampling periods				
	8 a.m.	4 p.m.	8 p.m.	12 midnight	
Six healthy subjects after three days of bedrest	13·4	7·7	6·1	1·9	
	10·9	9·7	6·3	2·3	
	13·1	7·1	9·4	15·9	
	7·0	6·0	6·5	3·7	
	11·6	5·0	6·2	3·4	
	10·8	8·0	4·5	3·8	F = 13·6*
Mean and SEM .	11·10 ± 0·95	7·25 ± 0·67	6·50 ± 0·65	5·17 ± 2·18	

of daily variation in renin and thus probably in aldosterone secretion (Gordon *et al.*, 1966). If this variation were not mediated through the sympathetic nervous system, it would have been observed in the quadriplegics who have postural hypotension due to sympathetic denervation. The results of our study suggest that the postural modulation of aldosterone secretion is dependent upon the integrity of both central and peripheral nervous systems.

The daily output of aldosterone is high in some of the acute patients (26·4 ± 7 μg.) who also have increased daily sodium excretion (155 ± 18 mEq.). This suggests that the effect of sodium upon aldosterone secretion can occur without connection between the spinal cord and the central nervous system. This

should be confirmed by a systematic study of the effects of Na loading and Na restriction upon excretion of aldosterone in men with a C5 spinal cord section.

The plasma levels and the urine concentration of 17-OHCS in the acute group have very little rhythm: there is a weak morning peak and a higher afternoon peak in the urine. Statistically, the morning and afternoon urinary peaks are significant (0·05) in the acute, but the afternoon peak emerges strongly (0·001) in the chronic quadriplegics. This suggests that in the acute period, daily rhythms of cortisol secretion acquired during childhood (Franks, 1967) are disappearing. During the period of rehabilitation, information to the hypothalamus toward acquisition of new daily rhythms of cortisol secretion must be carried by stimulation of afferent pathways entering the CNS above the spinal injury. Afferent pathways in the spinal cord for the release of corticotrophin following chemical or electrical stimulation were described in rodents (Redgate, 1962; Makara *et al.*, 1967). Immobilisation alone does not alter diurnal rhythms of plasma cortisol (Eik-Nes *et al.*, 1958; Cardus *et al.*, 1965). The very regular schedule of activities of the patients start later in the morning than previously and may account for the shift of the peak in urinary corticosteroids from morning to afternoon.

The excretion of MHMA does not show rhythmicity in the acute quadriplegics. In the chronic sample, there is a small, significant (0·05) decrease in the early morning instead of during the whole night (Sunderman *et al.*, 1960). The displacement of MHMA excretion may be related to the displacement of the 17-OHCS excretion from morning to afternoon. Measurement of individual catecholamines and evaluation of cholinergic activity are needed to correlate neural and hormonal activities (Guillemin, 1955; Krieger *et al.*, 1967; Naumenko, 1967; Hedge, 1968).

Illnesses that involve damage to a component of an endocrine feedback system: damage to the hypothalamus, the pituitary gland (Krieger *et al.*, 1966) or to the adrenal gland (Knapp *et al.*, 1966) suppress or modify the secretory rhythms of 17-OHCS. Diseases of the central nervous system other than the hypothalamic or pituitary areas alter the circadian patterns of 17-OHCS secretion (Eik-Nes *et al.*, 1958; Krieger *et al.*, 1966). Conditions that alter metabolism cause a flattening of daily rhythms: impairment of hepatic degradation of the hormone and severe malnutrition in children affect the diurnal secretion pattern of 17-OHCS (Tucci *et al.*, 1966; Alleyne *et al.*, 1967); ageing leads to some deterioration of diurnal rhythms of excretion of potassium (Lobban *et al.*, 1966). Diseases of the cardiovascular system that involve the autonomic nervous system lead to abnormal circadian patterns of plasma 17-OHCS (Gann, 1966; Knapp *et al.*, 1966; Cade *et al.*, 1967) and lead to a decrease in aldosterone excretion (Slaton *et al.*, 1967).

The common factor of all conditions described above is an impairment in part of the neuroendocrine system by disease or faulty metabolism. Patients paralysed by a section of the spinal cord at C5 or C6 have the spinal cord and the sympathetic chain isolated from the whole brain, including the medulla and the reticular formation. Afferent and efferent nervous pathways are interrupted in the somatic and the sympathetic systems. Reflexes across the spinal cord below the injury are present but not regulated by central integration and inhibition. During the period described as spinal shock and for several months after, it is likely that disorganised stimulation occurs above and below the injury, obliterating or reinforcing stimulation of severed pre-existing nervous pathways.

The results obtained confirm part of the hypothesis stating that isolation of

the spinal cord and sympathetic chain results in deletion of established daily rhythms of adrenal function. They refute the hypothesis that isolation of the spinal cord and sympathetic chain results in lack of subsequent information to the hypothalamus that controls excretion of 17-OHCS. The information gained on the role of the central nervous system upon aldosterone secretion is that section of the spinal cord and interruption of the sympathetic chain definitely eliminated the postural but not the sodium regulatory mechanisms.

Summary

Circadian rhythms of adrenal function were studied in men with an isolated spinal cord and sympathetic chain due to traumatic cord injury at C4, C5 or C6. Six patients were studied shortly after the onset of paralysis, and four eight months or more after the injury. The purpose of the study was to gather information on afferent pathways to the central nervous system which affect physiological rhythmicity.

The results obtained show that section of the spinal cord at C4, C5 or C6 dampens the established daily rhythms of 17-hydroxycorticosteroids excretion and suggests that, during and after rehabilitation, new patterns of information lead to the establishment of different rhythms. The results also show that section of the spinal cord and the sympathetic chain eliminates the effect of posture on the excretion of aldosterone and methylhydroxymandelic acid. The lack of postural control of aldosterone rhythms did not unmask a pattern related to an intrinsic secretory rhythm. The results suggest that afferent sensory stimuli from above the level of the injury modulate the central nervous system control of secretion of corticotrophin (ACTH), thus affecting the secretion of cortisol and the excretion of 17-OHCS. On the other hand, the connection of the central with the peripheral nervous system is necessary at all times to modulate secretion of aldosterone, epinephrine and norepinephrine.

Résumé

Etude du rythme quotidien de la fonction adrenale chez l'homme paralysé par une section de la moelle épiniere cervicale.

Le rythme quotidien de la fonction adrénale a été étudie sur des hommes dont la moelle epinière et la chaine sympathique avaient été isolées du reste du système nerveux central. Cet isolement étant du à un traumatisme accidentel au niveau de la quatrième, cinquième ou sixième vertèbre cervicale. Six malades ont été étudiés durant les 8 premier mois suivant l'accident; quatre autres après un délai dépassant 8 mois.

Le but de cette étude était de se renseigner sur le trajet des voies afférentes au système nerveux central, qui influencent certain rythmes physiologiques.

Les résultats obtenus montrent que les rythmes journaliers d'excrétion des 17 hydroxysteroides sont moins marqués après la section de la moelle au niveau des vertèbres cervicales 4, 5 ou 6. Pendant et après la réhabilitation des malades, il s'établit des rythmes differents des rythmes preexistants. Il semble aussi que la section de la moelle cervicale et l'isolation de la chaine sympathique eliminent l'effet de la posture sur l'excrétion de l'acide méthylhydroxymandelique et de l'aldosterone. L'absence d'un rythme posturel de l'aldostérone n'a pas permis de déceler un rythme sécretoire particulier.

Ces resultats conduisent à penser que les stimulations sensorielles afférentes, entrant la moelle epinière audessus des vertèbres cervicales 4, 5 ou 6, modulent le contrôle de la sécretion d'ACTH par le systeme nerveux central, affectant la sécretion de cortisol et finalement l'excrétion des 17 hydroxysteroides. D'autre part, il semble que la connection entre les systemes nerveux central et peripherique est indispensable pour que soit modulée la sécretion d'aldosterone, d'épinephrine et de norepinéphrine, affectant l'excretion.

ZUSAMMENFASSUNG

Die Circadian Rhythmen der adrenalen Funktion wurden studiert in Patienten mit Unterbrechung des Rückenmarks und der sympathischen Kette als Folge von Rückenmarksverletzung in Höhe von C4, C5 oder C6. 6 Patienten wurden bald nach Beginn der Lähmung und 4 acht Monate oder länger nach dem Unfall untersucht. Der Zweck der Studie war, Information über die afferenten Leitungsbahnen zum Zentralnervensystem zu sammeln, welche die physiologische Rhythmik beeinflussen.

Die Ergebnisse zeigen, dass eine Querschnittslähmung in der Höhe von C4-C6 den Rhythmus der 17-Hydroxykortikosteroid Ausscheidung herabsetzt und sie weisen darauf hin, dass während und nach der Rehabilitation neue Informationen zur Entwicklung anderer Rhythmen führen. Die Resultate zeigen fernerhin, dass eine Durchschneidung des Rückenmarks den Einfluss der Körperhaltung auf die Ausscheidung von Aldosteron und Methylhydroxomandelic Säure eliminiert. Die Resultate weisen darauf hin, dass afferente sensible Reize oberhalb der Verletzung die Kontrolle des Zentralnervensystems auf die Kortikotrophin (ACTH) Aussecheidung modulieren und folglich auch die Sekretion von Kortisol und die Ausscheidung von 17-OHCS beeinflussen. Andrerseits ist die Verbindung des zentralen mit dem peripheren Nervensystem immer notwendig, um die Sekretion von Aldosteron, Epinephrin und Norepinephrine zu modulieren.

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