Effect of starvation on total RBC and WBC counts, Hb content and PCV of *Ophiocephalus punctatus* (Bloch) during different time periods

Constituents	Control	Starvation time 15 days	21 days	27 days	33 days
Total RBC count (10^6/mm^3)	3.31 ± 0.05 (10)	2.88 ± 0.16 (6)	2.47 ± 0.10 (5)	2.40 ± 0.14 (4)	0.68 ± 0.07 (4)
Total WBC count (per mm^3)	13000 ± 176.62 (10)	7900 ± 278.95 (6)	7600 ± 256.06 (5)	6400 ± 147.42 (4)	5800 ± 138.26 (4)
Haemoglobin content (g %)	15.0 ± 0.34 (10)	13.8 ± 0.33 (6)	10.8 ± 0.05 (5)	10.6 ± 0.69 (4)	8.0 ± 0.22 (4)
Packed cell volume (%)	49.23 ± 1.50 (10)	47.38 ± 0.12 (6)	37.36 ± 0.46 (5)	36.66 ± 1.24 (4)	30.00 ± 0.09 (4)

All values are means ± SEM. Number of fish in parentheses.

Smirnova[11] reported that white blood cells (WBC) were reduced in number during starvation of a fresh water fish, *Lota lota*. A decline in red cell number was observed in the beginning, but later on, due to prolonged starvation, the number of RBC increased. This he accounted for by the decreased volume of blood, which was a result of prolonged starvation. The restoration of feeding in this case led to a further decline in red cell count, since blood volume increased on the availability of food.

Changes in red, white and immature blood cell numbers during starvation have also been reported in *Salmo gairdnerii* by Kawatsu[12]. His results also reveal that the decline in red cell count was preceded by an increase. So it may also be suggested that with prolonged fasting the volume of the blood decreases resulting in the increase of the number of blood cells in a unit area. But after prolonged fasting when food is given it again increases the blood volume which makes the number of blood cells less in a unit area. Constant availability of normal food probably accelerates the division of blood cells in the bone marrow, which results in the increase of blood cells to the normal level. This normal condition is not possible during the starved (ill health) condition of the fish.

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Altered circadian rhythm of catecholamines in patients with apallic syndrome[1]

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Summary. Circadian rhythms of catecholamines were investigated in 4 healthy subjects and in 6 patients suffering from an apallic syndrome. The clinical picture of this syndrome is characterized by disturbed consciousness (coma vigile), by suspension of the sleeping and waking rhythm, by lack of emotional reactions and by appearance of primitive motor patterns. 5 of the 6 apallic patients showed an abolished rhythmicity compared with the control group. These results were interpreted as an indication that endogenous, centrally controlled processes are the cause of circadian rhythms.

Circadian rhythms are the expression of endogenous processes, which have the characteristics of self-sustained oscillations. They are influenced by exogenous periodic factors (= Zeitgeber) such as light-dark cycles, environmental temperature and social cues[3–5]. These factors cause the period of the biological rhythm - which is about 24 h (= circadian) after the elimination of all Zeitgebers - to adapt to the period of the environmental periodicity which is exactly 24 h[6]. In patients suffering from an apallic syndrome, the functions of the brain stem are retained. Traumatic, hypoxic or inflammatory processes lead to a disintegration of the cerebrum functions to the level of the mesencephalon[7]. The clinical symptoms of the apallic syndrome are: disturbed consciousness, suspension of the sleeping and waking rhythm with irregularly distributed waking and sleeping phases over the 24-h day, lack of emotional reactions, stretched position of torso and extremities and appearance of primitive motor patterns. An investigation of such patients with regard to circadian rhythms is of interest in view of the possibilities that either the influence of exogenous factors has been abolished or that the center responsible for the rhythms has been damaged. Another possibility might be the impairment of the afferent tracts, which lead to the suprachiasmatic nuclei as putative circadian oscillators[8].

4 healthy male subjects (24–29 years of age) and 6 patients

(14–83 years of age) suffering from an apallic syndrome were investigated over a period of 48 h. All participants in the study were hospitalized. Meals were given at 7.30, 11.30 and 17.00 h, sleep time was between 22.00 h and 06.00 h. Blood was withdrawn with the aid of an indwelling venous cathether every 2 h from 09.00 h of the 1st day to 09.00 h of the 3rd day.

2-h urine samples were collected for 48 h, beginning at 0.8.00 h. Urine from the patients was collected with the aid of a bladder catheter. The patients were fed by tube 5 times a day and by i.v. infusions. Respiration, heart function and body temperature were normal. Besides antiepileptics – and in the case of 1 patient additionally dexamethasone – the patients were given no further drugs. In each case more than 10 days had elapsed between the incidence of the full stage of the apallic syndrome and the taking of the samples. For 4 patients there existed computer tomograms of the brain, taken shortly after the accidents. No conclusions could be drawn from these tomograms as to the different patterns in circadian rhythms. At the time of writing 2 of the 6 apallic patients had died.

The determination of the catecholamines in plasma and urine was performed by radioenzymatic assay[9]. The intra-assay variability for norepinephrine, epinephrine and dopamine in plasma was 4,6%, 5,2% and 5,5%, respectively and in urine 4,5%, 4,6% and 3,3%, respectively. Statistical analyses were carried out with analysis of variance for paired data (Friedman-test)[10]. Significant differences between the sampling times indicate the existence of a circadian rhythmicity.

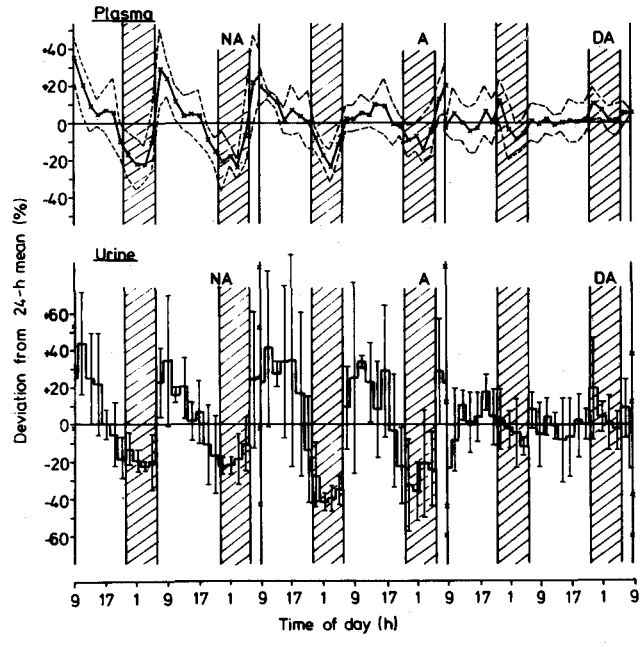

Figure 1. Circadian rhythms of norepinephrine (NA), epinephrine (A) and dopamine (DA) in 4 healthy volunteers. Values shown are means ± SD measured in 2-h intervals over 48 h. Shaded bars represent sleep time.

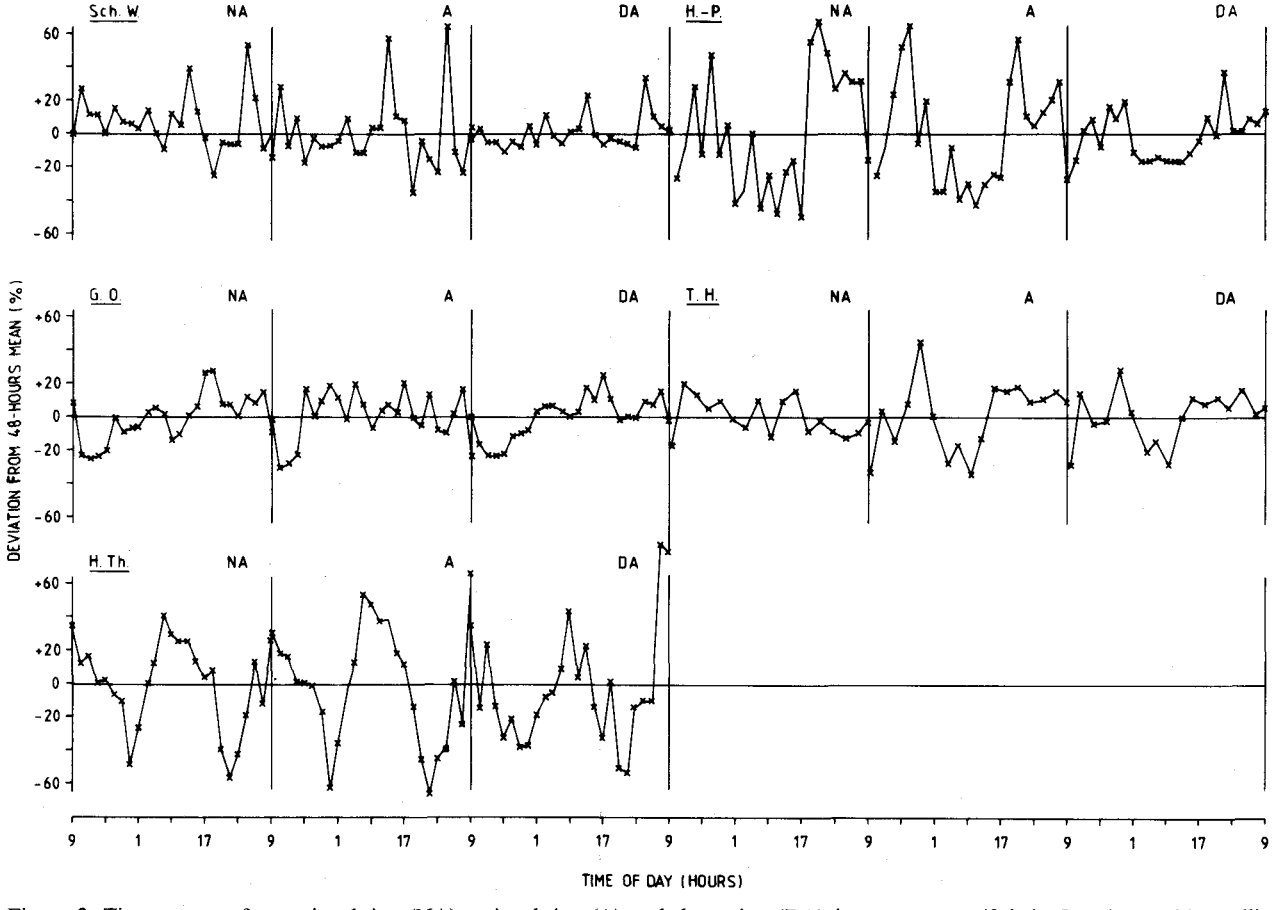

Figure 2. Time course of norepinephrine (NA), epinephrine (A) and dopamine (DA) in serum over 48 h in 5 patients with apallic syndrome.

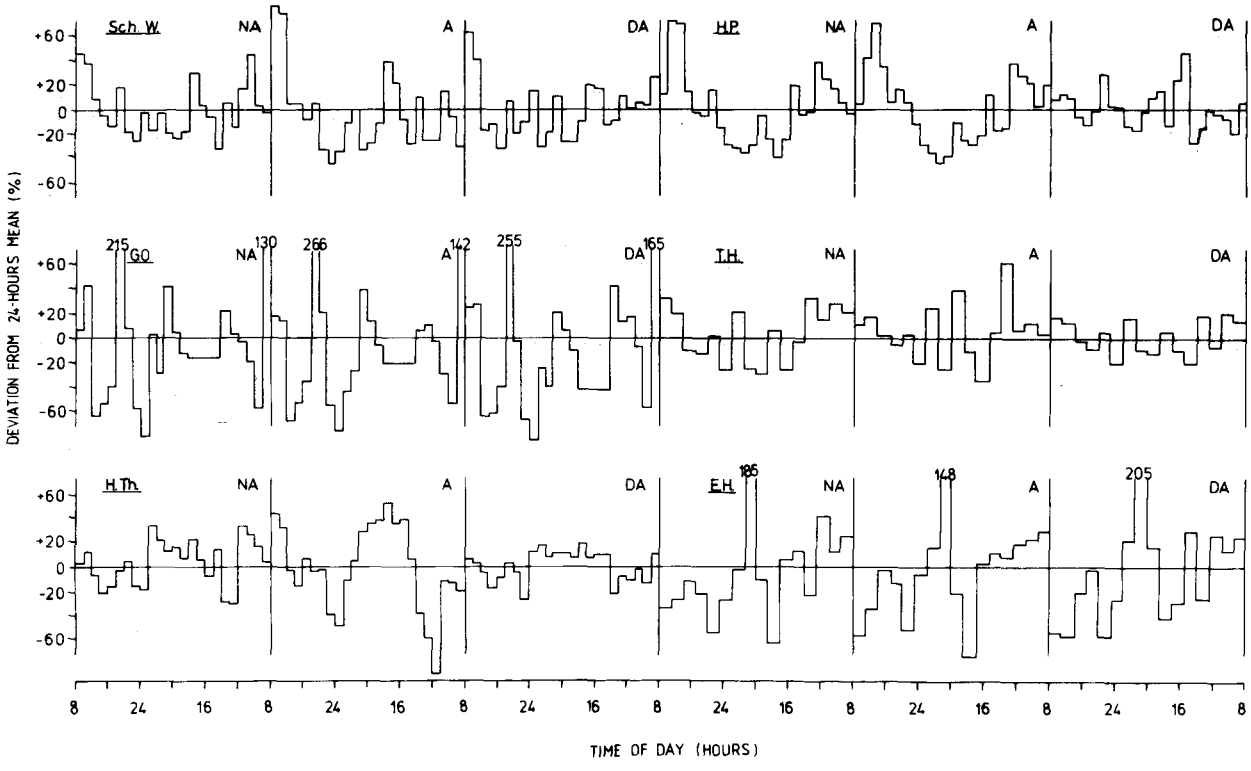

Figure 3. Urinary excretion of catecholamines in 2-h intervals in a 48-h period in 6 apallic patients.

The results from control subjects are partly summarized in figure 1. The absolute values of plasma concentrations as well as urinary excretions were converted into percental deviations from the daily mean. In the control group a circadian rhythm of plasma concentrations and urinary excretions of norepinephrine and epinephrine could be observed. The statistical data confirm these statements (Friedman-test: plasma NA and A $p < 0.001$; urinary A: $p < 0.001$, urinary NA: $p = 0.038$). Dopamine in plasma and urine showed no recognizable rhythmicity ($p = 0.466$ and $p = 0.659$). The smaller amplitudes of plasma levels compared with urinary excretions might be the result of the awakening of the subjects during the nocturnal withdrawals of blood.

Figure 2 shows the timecourse of catecholamines in serum, figure 3 the urinary excretion of catecholamines in each apallic patient.

Only in 1 of the 6 apallic patients (H.Th.) was a circadian rhythm of catecholamines found. No circadian rhythmicity of catecholamines could be observed for the other 5 apallic patients ($p = 0.368$, $p = 0.09$, $p = 0.494$ for plasma NA, A and DA; $p = 0.986$, $p = 0.993$, $p = 0.405$ for urinary NA, A and DA).

In the apallic patients the mean urinary NA and A excretions were higher than in the control group (59 ± 26 µg/24 h, cf. 33 ± 5; 30 ± 11 µg/24 h, cf. 11 ± 3) and DA was lower (226 ± 40 µg/24 h, cf. 334 ± 61). For plasma values the range was much greater in the apallic patients even though the means for NA and A were similar. Plasma DA was lower (55 ± 16 ng/l, cf. 75 ± 10). These measurements can be interpreted as follows: in 5 of the 6 patients the circadian rhythm of catecholamine secretion was abolished since the brain area responsible for the circadian rhythmicity had been reversibly or irreversibly damaged. In 1 patient (H.Th.) there was a clearly pronounced diurnal rhythm of catecholamines. The findings of the computer tomogram of this patient revealed no evidence for differently located brain damage compared to the other patients. In this patient the central regulation of circadian rhythms must be functioning normally. Therefore the disturbed sleep-wake cycle cannot be responsible for the abolished rhythm of catecholamines in the other patients. In comparison with the control subjects this patient shows an enhanced rhythm of catecholamines, especially noticeable in the case of dopamine. This suggests that the influence of exogenous or endogenous factors tending to obscure the biorhythms is reduced by the disease process. We also measured plasma prolactin and cortisol levels as well as urinary cortisol excretions. The circadian rhythmicity of prolactin was abolished in all patients whereas cortisol showed the same behaviour as the catecholamines. The number of patients in this study is too low to give a statement on the value of rhythm as a prognostic parameter in apallic patients. Our findings lend significant support to the concept that circadian rhythms are controlled centrally.

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