tion of the other authors[1,2]. However, hypercapnia (65 torr) restored synchronous firing in both phrenic nerves. With more rostral extensions of the incision, only severe hypercapnia combined with hypoxia (85 and 30 torr, respectively) restored the phrenic nerve activity, and the 'desynchronization' of respiratory rhythms was never observed. Splitting the brainstem did not obviously affect the blood pressure or heart rate.

We conclude that the respiratory centers in monkeys and rabbits are organized in 2 symmetrical and relatively independent networks of respiratory neurones whose synchronized activity (but not the activity itself!) depends upon neural connections crossing in the medulla. In these 2 species fibers from respiratory neurones to phrenic motoneurones appear to descend predominantly ipsilaterally.

On the other hand, breathing in cats seems to be controlled by a more integrated brainstem structure in which the decussating connections play a vital part. It is not clear why the cat should differ to such an extent from 2 species that are so remote from each other as are monkeys and rabbits. Species differences in, e.g., the strength and even pattern of vagal respiratory reflexes are not uncommon[6] but - as far as we know - qualitative differences in the central organization of respiratory centers were never suspected.

Since the cat has recently become the most frequently used laboratory animal, one of the implications of our results is that one can hardly accept the universal validity of physiological concepts and models unless they are confirmed in more than one species of experimental animal.

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Effect of starvation on serum acid phosphatase levels of freshwater catfish *Clarias batrachus*

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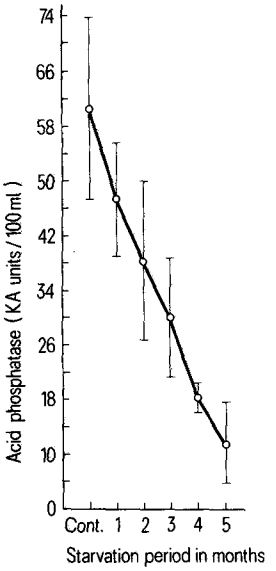
Summary. Starvation resulted in a regular and continuous decrease in the serum acid phosphatase levels of the freshwater catfish *Clarias batrachus*. The levels were found to be inversely proportional to the length of starvation. A maximal loss of 80.6% was noted at the end of the 5th month of starvation.

Starvation is one of the numerous adverse situations which fish have to face during some parts of the year. Acid phosphatase is one of several phosphatases; it catalyses the liberation of inorganic phosphate from monophosphate esters in acidic medium. Starvation causes a marked degree of change in various energy-yielding and other metabolic processes in the fish body. The effects of experimental starvation have been studied on various body and blood constituents of fishes by several investigators[2-7] in different parts of the world, but such studies on Indian freshwater fishes, particularly on blood enzymes, are scanty. This paper deals with the results obtained from the freshwater catfish *Clarias batrachus* following experimental starvation lasting up to 5 months.

Materials and methods. The methods for collection, transportation and maintenance of fish have been described earlier[2,8]. Only healthy fishes in the weight range 250-300 g were selected for the experiment. They were treated with $KMnO_4$ (2 mg/l) solution to remove external parasites, developing bacteria and fungi etc. The aquaria in which fish were kept under starvation were practically devoid of any fauna and flora, whereas the control fish were given minced goat-liver, snails and earthworms alternately, besides the naturally-growing fauna and flora. At the end of every month 4 starved and 4 control fish were taken out for the experiment. Blood was collected in a clean vial by severance of the caudal end, allowed to clot at room temperature for 10 min, then centrifuged, and the clear serum was decanted into another tube. The procedure of King and Wootton[9] was followed to determine serum acid phosphatase levels, using a Bausch and Lomb spectronic-20 colorimeter at 650 nm.

Results. The results obtained on serum acid phosphatase levels of the control and starved fishes are shown in the figure. Starvation resulted in a regular and continuous decrease in the enzyme level. After the 1 month of starvation the enzyme level fell to 47.4 ± 8.3 KA units/100 ml from the control value of 60.5 ± 13.2 KA units/100 ml, i.e. a loss of 21.7%. It decreased by another 37.0% after 2 months and 50.4% after 3 months. At the ends of the 4th and 5th months of starvation, the enzyme levels were 30.3% and 19.4% of control, respectively. Thus, acid phosphatase level was found to be inversely proportional to the starvation



Effect of starvation on serum acid phosphatase levels of *Clarias batrachus*.

period in *C. batrachus*. Statistically, the decrease in serum acid phosphatase level obtained after the 1st month of starvation was not significant ($p < 0.01$), but the values noted at the ends of the 2nd, 3rd, 4th and 5th months were significant ($p < 0.01$).

Discussion. The survival potential of fishes during the period of starvation differs from fish to fish. Metabolic cycles involve complex sequences of reactions mediated by enzymes. Love[10] noted that if in the 1st event, the arrival of metabolites in the blood stream from the gut is stopped, some reaction sequences will be arrested altogether and the enzyme will not be required. Thus, it is logical to expect that acid phosphatase, like other enzymes, will decline during a period of starvation. In the present study a linear and continuous decrease occurred in the serum acid phosphatase levels of the fish *C. batrachus*. Noda[6] studied various phosphatases of the rainbow trout, *Salmo gairdnerii*, during a period of starvation, and noted marked fluctuations in liver, intestine, pyloric caeca and spleen. He further noted that about 40% of the initial activity was lost in the course of 52 days of starvation. The trend of the decrease in the enzyme level of starved *C. batrachus* can be compared with the observations of Noda[4]. Vellas and Creach[5] studied ureogenesis in starving carp for 12 months and noted the essential role of the liver and the small participation of the kidney in it. Joshi[6] reported a fall of 49.3% and 47.3% in blood glucose and nonprotein nitrogen levels respectively by the end of the 150th day of starvation in *C. batrachus*. During the starvation experiments, the fish appeared to be gradually growing weak and sluggish, which can be correlated with a reduced metabolic activity. This feature has been noted also by Van Dam[11] and Smith[7] in eels and lungfish respectively. A regular decrease in blood urea in the catfish clearly revealed a declining metabolic rate with an increasing starvation period[2].

The gradual falling-off of serum acid phosphatase levels of *C. batrachus* with an increasing period of starvation clearly indicated that due to the sudden cut-off of its food-supply, the fish tried its best to fullfil its requirement for energy by metabolizing the reserve food stored in the liver and other parts of the body. However, with the gradual exhaustion of the reserve food, metabolic activities also ceased to function, and this resulted in a linear and continuous decrease in the level of acid phosphatase, which is known to take part in carbohydrate metabolism, transportation and absorption.

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Inhibitory effects of 1-iodo-3-aminomethyl-5,6,7,8-tetrahydro-2-naphthol (ONO-3122) and prostaglandin H_2 on vasopressin-induced osmotic water flow in toad bladder

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Summary. Both 1-iodo-3-aminomethyl-5,6,7,8-tetrahydro-2-naphthol (ONO-3122), which increases endogenous PGH_2, and PGH_2 itself, significantly depressed vasopressin-induced osmotic water flow in the toad bladder. These results suggest that ONO-3122 increases endogenous PGH_2 synthesis, and that PGH_2 and/or its metabolites inhibit vasopressin-induced water flow.

In many biological systems, prostaglandins activate adenylate cyclase and thus increase cellular levels of cyclic AMP. In some other systems, prostaglandins inhibit adenylate cyclase after having been activated by agonists such as vasoppdssin. We found that PGE_1 inhibited vasopressin-mediated adenylate cyclase activity of the hamster kidney[2], as reported previously. Beck et al.[3] also found inhibitory effects of PGE_1 on vasopressin action of the rat kidney.

In a physiological study, Orloff et al.[4] reported that PGE_1 inhibited vasopressin-induced osmotic water flow across the toad bladder membrane. They suggested that the inhibitory effect of PGE_1 was associated with the inhibition of adenylate cyclase activity.

In the present study, we examined the effect of an increased production of endogenous PGH_2 on the exogenous vasopressin-induced increment of the osmotic water flow across the toad bladder membrane.

Methods and materials. The urinary bladder of the toad, *Bufo bufo japonicus*, was used. After double pithing of the toads, the bladders were excised and immediately placed into Ringer's solution. The composition of the Ringer's solution was as follows: NaCl, 111 mM; KCl, 3.5 mM; $CaCl_2$, 0.9 mM; $MgCl_2$, 1.5 mM; NaH_2PO_4, 1.9 mM; Na_2HPO_4, 8.1 mM. The osmolality was 232 mOsm/l and the pH was 7.4. The osmotic water flow was measured as previously described[5].

1-Iodo-3-aminomethyl-5,6,7,8-tetrahydro-2-naphthol (ONO-3122) and prostaglandin H_2 (PGH_2), which were supplied by Ono Pharmaceutical Co. Ltd, and arginine vasopressin (Sigma Chem. Co., St. Louis) were used for the present study. ONO-3122 was dissolved in 0.5 N NaOH and then adjusted to pH 8.0 by using 0.1 N HCl. Acetone, a solvent of PGH_2, was evaporated by N_2 gas, and then PGH_2 was dissolved in the Ringer's solution. All procedures were performed on ice immediately before the addition to the chamber. Statistical analysis was performed by the paired t-test.

Results and discussion. Vasopressin and ONO-3122 were