pteric region can be recognised[1]. In man, the more or less horizontal limb of the H is formed by the sutures between the anterior-inferior part of the parietal and the upper border of the greater wing of the sphenoid bene (Spheno-parietal pterion). In monkeys, the frontal and the temporal bones articulate at this place (Fronto-temporal pterion)[1]. The spheno-parietal type of pterion is considered typical of man, while the fronto-temporal type is characteristic of monkeys.

It has been observed while examining the crania of Rhesus monkey that, though the fronto-temporal arrangement of pterion is more common, spheno-parietal pterions may also be found. Present note records the incidence of spheno-parietal type of pterions in Rhesus monkey.

In all a total of 434 pterions were examined basing the observations on 113 male and 104 female skulls. Age has no effect on the type of articulation because a particular type of sutual arrangements in the pteric region is present at the time of birth and does not change afterwords[2]. No distinction was made for left and right as side also seems to have no effect on the type of contact[2]. The results of the observations are as under:

	Males	Females
Total No. of Pterions	226	208
Fronto-temporal (F–T)	188 (83·2%)	181 (87·0%)
Spheno-parietal (S–P)	38 (16·8%)	27 (13·0%)

This type of variation has been recorded in macaques (11·3%)[3], gorilla (1·3%)[4] and chimpanzee (11·2%)[4]. In man, orang-utan and gibbon, though the articulation is of spheno-parietal type, variation for the fronto-temporal contact has also been recorded (European, 1·9%; Orang-utan 29·0% and Gibbon 19·4%)[4]. The incidence for a few Indian populations has been recorded as 9·26% (Uttar Pradesh), 3·70% (Bihar) and 13·10% (Andhra Pradesh)[5].

Reasons for this dichotomy of form are associated with differential ossification in the bones of this region[6]. Both genetic and environmental factors can cause the differential ossification leading to this variation[6]. The manifestation of this variant in the skeleton being developmental in origin, its presence or absence forms a part of natural variation of a species[6]. It is probable that those regions which show greater differences in the arrangement of bones may be regarded as regions in which "phylogenetic stability" has not yet been achieved[7–8].

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ALLOXAN INDUCED LEUKEMOID CONDITION IN A FRESH WATER TELEOST *CLARIAS BATRACHUS*

Clarias batrachus weighing 70 to 100 g. were collected from local fresh water resources and acclimated to the laboratory conditions for 7 days before starting the experiment. A 1% (W/V) solution of alloxan (Alloxan hexahydrate) was prepared immediately prior to use in citrate-phosphate buffer at pH 4·0. The dosage of alloxan injected intramuscularly contained 50 mg/kg body weight which was repeated in 42 experimental fishes every third day. Small groups of 6 treated fishes were autopsied 24 hours after the last dosage and the blood was collected from the caudal vein after 1, 10, 20, 30 and 40 days of the initial dosage when fishes reached to comasate condition and finally died. The results were compared and confirmed with those of 14 control fishes which were given equal amounts of citrate-phosphate buffer at pH 4·0.

In many alloxan treated fishes, the skin at the site of injection became red and swlloen after 3rd dosage. The swelling increased markedly after 30 days of treatment and the skin showed wide discolouration. The swelling finally became an open wound after 30 days and the fishes reached to comasate condition during 35 to 40 days of the initial dose. However, there was no mortality recorded upto 40 days of the treatment. After 40 days the wounds at injection site became enlarged and fishes started dying. The observations were made only upto 40 days of the treatment.

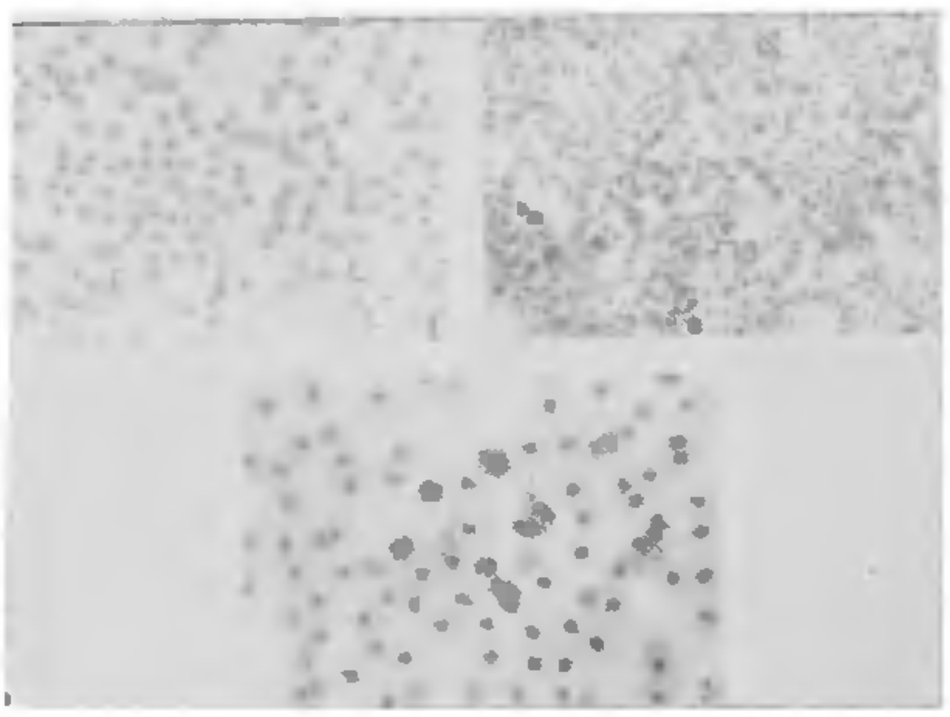
No appreciable change was recorded in the leucocyte count after 24 hours of the initial dose of alloxan. Regular increase in the number of leucocytes was observed after 10, 20, 30 and 40 days of the treatment (Table 1). No immature leucocyte could be seen in the blood smears upto 20 days of the treatment. Only a few immature leucocytes were encountered in the blood smears after 30 days of treatment while after 40 days of alloxan treatment (Fig. 2) approximately 50% of the total leucocytes were found to be immature (mainly myelocytes and few myeloblasts: recognised after Klontz *et al.*[2]). Karyokinesis was

TABLE I

Changes in the total and differential leucocyte count in Clarias batrachus *after repeated injections of alloxan at the rate of 50 mg/kg body weight (6 fishes were used in each experiment)*

Time days	Cumulative dosage mg kg	Lecuocytes thousand/cmm		Differential leucocytes (%)							
				Agranulocytes		Granulocytes		Myelocytes		Myeloblasts	
		Average	± S.E.	Average	± S.E.	Average	± S.E.	Average	± S.E.	Average	± S.E.
0	—	8·60	1·30	76·20	1·80	22·80	4·50	—	—	—	—
1	50	9·20	0·42	76·00	2·40	23·48	7·00	—	—	—	—
10	250	16·80	1·85	71·00	2·58	27·80	3·20	—	—	—	—
20	500	40·40	2·20	68·27	6·22	33·23	6·78	—	—	—	—
30	750	65·00	2·18	58·78	4·49	38·20	1·80	3·40	0·78	—	—
40	1000	88·20	3·85	16·70	7·85	33·30	6·52	45·80	4·38	4·20	1·78

also observed in some of the leucocytes at this stage (Fig. 3).



FIGS. 1–3

In normal fishes (Fig. 1), there was no immature leucocyte in circulation (as observed by the study of blood smears of control fishes). Leucocytes were counted in the control fishes in the beginning and at the end of the experiment. No significant change was recorded.

The present findings are of utmost importance since they indicate the possible leukemogenic action of alloxan, which is till now regarded as a diabetogenic agent (Falkmer[1]). The results, however, obtained so far, call for more comprehensive studies in order to permit valid conclusions on the leukemogenic role of alloxan. Further work in this respect is in progress.

Our grateful thanks are due to Dr. S. D. Loiwal, Dr. V. P. Agrawal, Dr. O. P. Saxena and P. K. Mehta for their interest in this investigation.

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NEW ADDITIONS TO FUNGI OF INDIA

DURING our mycological studies we isolated the following two fungi which are new records for India.

1. *Gliomastrix murorum* (Corda) Hughes in *Can. J. Bot.*, 1958, p. 769.

This fungus causes severe leaf spot disease of *Ficus religiosa* L. The disease appears as small, irregular brown spots which gradually increase in size and cover the whole leaf surface.

Pathogenicity tests were done and Koch's postulates were confirmed. The culture has been deposited in herb. I.M.I., Kew, No. 213284.

2. *Trichoderma longibrachiatum* Rifai aggr. in *Mycol. Pap.*, 1969, **116**, 42.

The fungus was isolated from the seeds of *Linum usitatissimum* L. (linseed) variety R-17 by "blotter test" as well as "agar method".

Linseed is a new host record for this fungus. The culture has been deposited in herb, I.M.I., Kew, No. 208085.

The authors express their thanks to Dr. A. Johnston and Dr. B. L. Brady of Commonwealth Mycological Institute, Kew, England, for help in the identification of the species.

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