these bands have been assigned to $\nu_{(z)}$ (NH), $\nu_{(z)}$ (NH), $\nu_{(t)}$ (NH), and ν (C = S) modes respectively. The configurations of the former three bands are given by Walter and Ruess[12] as Z.Z, Z.E. and Z.E. respectively.

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BIOCHEMICAL CHARACTERIZATION OF CASES OF MUCOPOLYSACCHARIDOSES DETECTED AT BANGALORE

MUCOPOLYSACCHARIDOSES (MPS) are a group of inherited disorders due to a faulty metabolism of mucopolysaccharides also called glycosaminoglycans (GAG). In this group there are at least six types, *viz.*, Hurler syndrome (MPS-I); Hunter syndrome (MPS-II); Sanfilippo syndrome (MPS-III); Morquio syndrome (MPS-IV); Scheie syndrome (MPS-V) and Maroteaux-Lamy syndrome (MPS-VI). All these types differ in clinical and biochemical characteristics[1]. The present paper reports observations made in nine cases of Hurler syndrome (MPS-I) detected at Bangalore.

Cases seen at the Mental Retardation (MR) Clinic of this institute formed the clinical material and a total of 1,480 cases of MR were available for this study. In all these cases fresh random urine samples were collected and subjected to various bio-chemical and chromatographic tests to detect metabolic defects.

Urinary mucopolysaccharides were detected by the toluidine blue and Azure-A spot tests and turbidity tests using cetylplatinyl chloride, cetyltrimethyl ammonium bromide and acid-albumin[2]. Whenever a case with excretion of mucopolysaccharides was noted, twnenty-four hour urine samples were obtained and the following confirmatory tests done[3]. Chromatography to determine the nature of MPS: ion exchange chromatography using Dowex (1 × 2; 200/400 mesh, Cl-form) and determination of uronic acid. In addition, the plasma levels of the enzymes β-galactosidase and β-N-acetyl glucosaminidase were determined using the substrates obtained from M/s. Sigma Chemicals, U.S.A.

This study led to the detection of twenty-five cases of MR who showed abnormal excretion of MPS in random urine as noted in the spot tests and also turbidity tests. It was, however, possible to obtain confirmation by repeat samples in only nine cases reported here. All nine cases showed the following salient clinical features (among others) suggestive of MPS (formerly clubbed as cases of "gargoylism"); a grotesque facies ; dwarfism; thick, protruding tongue; short; stubby fingers and mental retardation. Urines from age and sex matched normal children were also simultaneously processed (to serve as controls). The results are indicated in Table I.

TABLE I

Uronic acid in 24 hour urine sample

	Age	Uronic acid mg/24 hrs.
Cases	4±2·44	19·20±7·21*
Controls	4±2·44	5·00±1·39

(* $p < 0.01$)

In these nine cases ion-exchange chromatography showed excretion of dermatan and heparan sulfates. Also, the increased excretion of uronic acid characterised these cases as either MPS-I or MPS-II. Presence of corneal opacity suggested that these were cases of MPS-I. Our cases were found in both sexes, whereas MPS-II is an X-linked recessive trait; hence found only in males. The uronic acid determination showed that fractions containing dermatan sulfate had twice as much of uronic acid as in fractions containing heparan sulfate. D:H ratio is 2:1 in MPS-I while it is 1:1 in MPS-II. It was also noted that the former fraction showed a 30% reduction in colour when conducted without borate in the reaction mixture.

The activity of plasma enzymes are indicated in Table II.

TABLE II

Plasma enzyme activities in Cases of Hurler's syndrome

	Beta-galactosidase*	Beta-N-Acetyl-glucosaminidase*
Patients	0·433 – 0·120 (0·01 < p < 0·05)	1·398 ± 0·580 (p < 0·05)
Controls	0·262 ± 0·047	0·790 ± 0·079

* Micromoles of substrate converted per minute per litre. The values represent mean. Range is shown in parentheses.

It was noted that there was an increase of beta-galactosidase activity, a finding which needs to be confirmed on a larger number of cases.

Metabolic disorders which are rather rare in any given population provide an opportunity to understand normal pathways and their study has thus been highly rewarding. A proper identification of the biochemical basis can also help in rational therapy and prevention of similar abnormality in a subsequent offspring.

Apart from the urine examinations to detect MPS, attempts have been made with success to diagnose specific enzyme defects in each type of MPS by leukocyte studies or by using skin fibroblasts grown in tissue cultures[4]. It has also been noted that while skin fibroblasts of MPS–I or MPS–II in tissue cultures show metachromasia and accumulation of S^{35} when tested separately, this is not seen when they are grown together[1]. This indicates that these two conditions are genetically distinct. MPS–I is an autosomal recessive trait and hence to a couple who has had one child so affected, there is a 25% chance of having a similarly affected child. However, no satisfactory therapy for MPS is as yet availale. Vitamin A, Steroids, etc., are of no value. Enzyme therapy by plasmapheresis has a short action only. Prenatal diagnosis is now possible to diagnose either syndrome. The enzyme estimations[7] can be made on the amniotic fluid cells grown in culture. Selective therapeutir abortion of affected fetuses can help reduce the number of mentally retarded and allow parents to have a normal baby.

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OCCURRENCE OF TRIPLET FOETUSES IN THE SLENDER LORIS, *LORIS TARDIGRADUS LYDEKKERIANUS* CABR.

DURING our studies on the placental and foetal development in the slender loris, *Loris tardigradus lydekkerianus*, we came across a pregnant uterus containing three foetuses. It is not uncommon in these prosimians to find twins as reported by Hill[1] and Ramaswami and Anand Kumar[2]. So far as we know, this is the first report of triplet foetuses in the slender loris. In other prosimians, such as *Cheirogaleus*[3], *Tupaia*[4], and *Microcebus*[5] usually triplets are born. Zuckermann[6] reported a single instance of triplets in *Lemur macace*.

The three foetuses reported here differ in their size and weight (Table I and Fig. 1). The largest foetus

TABLE I

Weights and lengths of the triplet foetuses

	Weight in mg.	Length in mm	
		Total length	CR length
Foetus A	1210	47	35
Foetus B	810	40	32
Foetus C	340	34	25

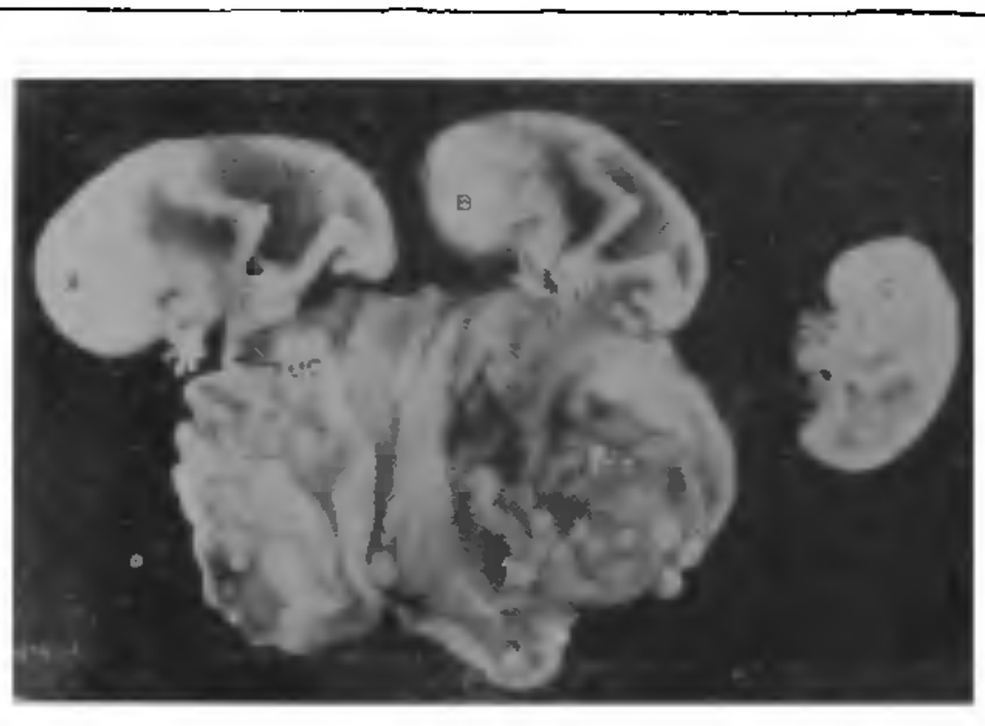

FIG. 1. Photograph of the triplet foetuses A, B and C with their placental connections. × 2 (2¾"/4⅓"). UC, Umbilical cord.