

disease progresses could represent regression of testicular androgen synthesis to the fetal type.

## REFERENCES

Hamilton, W., Grant, J. C., and Wilson, R. R. (1970). In vitro enzymic activity of human undescended testes. In *7th Annual Symposium of the Society for the Study of Inborn Errors of Metabolism*. Livingstone, Edinburgh.

**Congenital Malformations and Neoplasia.** Jean Keeling (introduced by Colin Berry). Published in full—Berry, C. L., Keeling, J., and Hilton, C. (1970). *Archives of Disease in Childhood*, **45**, 229. Coincidence of congenital malformation and embryonic tumours of childhood.

**Diminished Activity of Platelet Monoamine Oxidase in Down's Syndrome.** Philip F. Benson and Jennifer Southgate (*Paediatric Research Unit, Guy's Hospital, London S.E.1*). Subjects with primary trisomic Down's syndrome have low concentrations of blood serotonin (5-hydroxytryptamine) (5-HT). The reason for this is unknown. In Down's syndrome the binding of 5-HT to platelets has been found to be normal. In theory low blood 5-HT may be due either to a diminished rate of synthesis or to an accelerated breakdown.

We report a study on platelet monoamine oxidase (MAO) activities in subjects with Down's syndrome and in controls. While the plasma enzyme is practically inactive with 5-HT, platelet MAO appears to be very similar to the liver mitochondrial enzyme which is the main reservoir of MAO in the body.

Platelet MAO activity (measured fluorimetrically using kynuramine as substrate and expressed as  $\mu\text{g}$ . 4-hydroxyquinoline/mg. protein/30 minutes) was significantly lower in children with Down's syndrome (22 subjects, mean 0.81 SD 0.38) than in controls matched for age, sex, and domicile (22 subjects, mean 1.24, SD 0.70)  $p = 0.01 - 0.02$ . This does not support the view that low concentration of blood 5-HT is due to an accelerated rate of 5-HT breakdown. Moreover, a decrease in MAO activity is compatible with the observation that urinary excretion of 5-hydroxyindoleacetic acid (5-HIAA) is lower in subjects with Down's syndrome than in controls. If accelerated degradation of 5-HT cannot be incriminated as the cause of low blood 5-HT in Down's syndrome, it is reasonable to suggest that there may be a diminished rate of 5-HT synthesis. This might be due to inefficient intestinal absorption of tryptophan or to diminished activity of enzymes involved in 5-HT synthesis. Theoretically the trisomic chromosome 21 might carry a regulatory gene for MAO, leading to increased synthesis of repressor and therefore to a decreased rate of MAO synthesis.

**Effect of Cytotoxic Drugs on Immature Rat's Gonads and Subsequent Reproductive Performance.** Colin Berry (*Institute of Child Health, London W.C.1*). (To be published in full elsewhere.)

**Comparison of Effect of Isoprenaline and Salbutamol Aerosols on Airways Obstruction and Pulse Rate of Children with Asthma.** G. Hambleton (introduced by Malcolm Segall) (*Department of Paediatrics, St. Thomas's Hospital S.E.1*). (To be published in full in this journal.)

**Total Faecal Bile Acid Excretion in Children.** Clive Leyland (introduced by June Lloyd) (*Institute of Child Health, London W.C.1*). Few studies have been made on total faecal bile acid excretion and no data are available for children.

Using gas-liquid chromatography (Grundy, Ahrens, and Miettinen, 1965) faecal bile acids have been estimated on pooled 3-day stool collections. In 16 children (aged 2 months to 14 years) without evidence of fat malabsorption, the mean daily excretion varied between 10 and 85 mg. and appeared to correlate with body weight.

Very low amounts ( $< 3 \text{ mg./day}$ ) were found in 5 children who had biliary atresia. Increased excretion was found in a child with familial hypercholesterolaemia receiving cholestyramine and in 2 children with resection of the terminal ileum.

Raised excretion was found in 2 children with cystic fibrosis who had steatorrhoea, whereas in 2 other children with cystic fibrosis, in whom steatorrhoea was controlled by a low fat diet, faecal bile acid excretion was normal.

In 5 children with other diseases causing fat malabsorption steatorrhoea had been controlled by dietary fat reduction. 4 had also received supplementary medium-chain triglyceride. In all these children faecal bile acid excretion was normal.

## REFERENCE

Grundy, S. M., Ahrens, E. H., Jr., and Miettinen, T. A. (1965). Quantitative isolation and gas-liquid chromatographic analysis of total fecal bile acids. *Journal of Lipid Research*, **6**, 379.

**Sugar Absorption in Rats with Intestinal Blind Loops.** Michael Gracey, Valerie Burke, and Ademole Oshin (introduced by Graham Chance) (*Institute of Child Health, University of Birmingham*). Temporary monosaccharide malabsorption in young babies may be associated with abnormal small intestinal flora and deconjugated bile salts (Gracey, Burke, and Anderson, 1969). To investigate this association we have studied the uptake of an actively transported, non-metabolized analogue of glucose (Arbutin) in rats with intestinal blind loops.

Blind loops, 8–10 cm. in length, were constructed in the jejunum of adult Wistar rats. One to two months after operation, 5-day stool collections demonstrated steatorrhoea in all operated animals. In 6 animals, using a recently described *in vitro* technique (Semenza, 1969), and using randomized segments of intestine, we have shown inhibition of Arbutin uptake in the blind loops and in the intestine adjacent to the loops, both proximally and distally. Inhibition was most obvious in the blind loops and was least severe in the proximal segments. In 3 animals the degree of inhibition was