

However, differential thermostability is the most valuable method for identifying enzyme patterns at the clinical level.

¹Dunne, J., Fennelly, J. J., and McGeeney, K. (1967). *Cancer (Philad.)*, 20, 1.

²Posen, S., Neale, F. C., Chubb, J. S. (1965). *Ann. intern. Med.*, 62, 6.

³Wilkinson, J. H. Colston Research Society—Liver Diseases, 1967—To be published. Butterworth Scientific Publications.

TREATMENT OF CHRONIC INTRAHEPATIC CHOLESTASIS WITH PHENOBARBITONE

R. P. H. THOMPSON AND ROGER WILLIAMS (*King's College Hospital, London*) Four patients with chronic intrahepatic cholestasis have been treated for one to two months with oral phenobarbitone, 90–180 mg. per day, without changing their previous treatment. Liver biopsies had shown the appearances of primary biliary cirrhosis in three and of postnecrotic cirrhosis in one. All had had constant plasma bilirubin levels for at least six months, with itching.

Plasma total and direct-reacting bilirubin levels were

measured two to three times weekly by the Michaelis method. About four days after starting treatment these levels fell by 40 to 50%, and levelled off at about the tenth day. The proportion of direct-reacting bilirubin rose. All patients noticed less itching and improved well-being, and they and their relatives commented on decreased jaundice. Side effects were limited to drowsiness, controlled by reducing the dose.

The mechanism of this reduction of plasma bilirubin levels may be related to the known induction by phenobarbitone of microsomal detoxicating enzymes in animals, in particular glucuronyl transferase. A similar reduction of serum bilirubin levels has been reported by other workers in three patients with unconjugated hyperbilirubinaemia in whom the activity of glucuronyl transferase is low. In the present patients with conjugated hyperbilirubinaemia the plasma level may fall as a result of increased uptake into the liver. The rise in the proportion of direct reacting bilirubin suggests an increased regurgitation into the blood, and the possibility of this and other mechanisms will be discussed.

CORRECTION

In Table V of the paper by H. G. Sammons *et al.* ('Modification in the xylose absorption test as an index of intestinal function', *Gut*, 8, 348) there two should be readings for cases 7 and 13, namely, the words, 'After oral iron treatment', refer to the second set of figures for case 7, and the words, 'After treatment', refer to the second set of figures for Case 13. The Table as originally published is misleading.