of the antidote if necessary, and would be helpful also if the tablets were substandard.

Earlier this year [6] you wrote that all containers of medicine should be labelled routinely when dispensed. This is an essential first step, and the advantages are that all types of medicine (liquids, solids, and ointments) are included, whereas identity marks can only appear on solids. Patients, however, frequently change their containers and even mix different tablets in one bottle. If there were identifying marks as well there would be a double safeguard in the case of tablets. The Standing Pharmaceutical Advisory Committee mentioned the technical difficulties with these markings. Hastings [7] (who is a technical director in the industry) disposed of most of the difficulties in relation to cost and apparatus. It is agreed that new equipment would be required to mark pan-coated tablets but surely this is hardly an excuse for not marking the many uncoated tablets as a start. A well-known peppermint with a central hole is clearly identifiable and is marketed at 12 for 2½d. Hardly a costly procedure I should have thought.

The Committee considers the question of cost in detail and the difficulties with imported and exported tablets. I accept difficulties for the imported tablets but surely we should export readily identifiable tablets to merit the slogan "Buy British for better identifiable drugs". We could lead the world in this respect. "It has always been considered bad pharmaceutical practice to rely on colour or surface markings for the identification of tablets," says the report. I suppose it is a good "pharmaceutical practice" not to be able to identify a tablet at all when a patient's life may be in danger.

The identification of drugs is an international problem which could be more fully considered by W.H.O. There is, I regret, a lot of prejudice in the medical and pharmaceutical profession against identifying drugs at all, which I can only assume harks back to the old days when the treatments were not so effective as today, when it is essential to have available information quickly.

London W.12. J. D. W. WHITNEY.

LACTIC DEHYDROGENASE ISOENZYMES IN PREGNANCY

SIR,—We have lately completed a survey of serum-enzyme levels throughout normal pregnancy, during labour, and in the early postpartum period.

Sera from 250 pregnant women, together with cord-blood samples from some 30 of their babies, have been examined for lactic dehydrogenase, isocitric-acid dehydrogenase, 2-hydroxybutyric dehydrogenase, glutamic-oxaloacetic and glutamic-pyruvate transaminases, alkaline-phosphatase, and pseudocholinesterase activity.

In view of the interest shown by Dr. Hawkins and Mr. Whyley (May 26) in lactic dehydrogenase (L.D.H.) enzyme and isoenzyme activity in pregnancy, we wish to mention our experience with this enzyme. Our results show an increase in maternal L.D.H. activity commencing in the last trimester, increasing during labour and the immediate postpartum period, and falling a few days after delivery. Cord-blood samples show L.D.H. activities twice or thrice the corresponding maternal levels.

L.D.H. isoenzyme studies have been carried out on sera from pregnant women near term and in labour, on cord blood, and on human tissue extracts. The samples were separated by electrophoresis in starch gel and on cellulose acetate, and the isoenzyme fractions were demonstrated by an elegant enzyme-staining technique developed by Dr. Hannah Barnett of Queen Mary's Hospital, Roehampton. Our results were as follows (using L.D.H. "1" to refer to the fast moving isoenzyme fraction):

Sera	*L.D.H. isoenzyme fraction number* 1	2	3	4	5
Normal women ..	+	+ +	± or −	−	−
Pregnant women near term and in labour	+	+ +	+	± or −	−
Cord blood	+	+ +	+ + or +	±	−
Placental homogenates	+	+ +	+ + or +	+ or ±	−

These results show an increase in L.D.H. isoenzyme fractions 3 and 4 in maternal, cord, and placental samples. This pattern was not obtained with normal serum or red-cell, heart, kidney, liver, or muscle extracts.

We therefore suggest that the increased L.D.H. activity demonstrated in the sera of pregnant women and in cord blood is placental in origin.

A full account of this work together with details of alkaline-phosphatase isoenzymes during pregnancy is being prepared for publication.

Department of Chemical Pathology, Group Laboratory, Kingston upon Thames. B. W. MEADE S. B. ROSALKI.

SEGREGATION RATIOS AND LINKAGE STUDIES IN A FAMILY WITH SIX TRANSLOCATION MONGOLS

SIR,—In view of the interest in the segregation behaviour of translocation chromosomes in mongol families, I should like to summarise findings in a kindred where six mongols have appeared in four related sibships. This family carries a translocation involving no. 21 and a chromosome of the 13-14-15 group. Altogether, 32 individuals in ten segregating sibships have been karyotyped with the following results:

Translocation-carrier parent	*No. of carriers*	*Offspring* 45T	46N	45T or 46N*	46M	*Total*
Progenitor (sex unknown)	1	3†	1	2	0	6
Male	3	2	4	9	0	15
Female	6	9	10	5	6‡	30
Total	10	14	15	16	6	51

T = translocation carrier. N = normal. M = translocation mongol.
* Not tested, but normal phenotype.
† Includes one deceased individual who is the parent of two mongols, grandparent of one mongol, and sib of two known translocation carriers.
‡ Includes two deceased mongols.

It is apparent that the translocation carriers (45T) and normals (46N) are segregating in the expected one-to-one fashion.

In contrast, Hamerton et al.[1] reported chromosome studies of 13 phenotypically normal individuals in three segregating sibships, and found all 13 to bear the translocation. 10 of these segregants arose from 2 male carrier parents. Their findings deserve careful consideration, because it is possible that there are basic biological differences to account for the lack of agreement between families. Such discrepancies also emphasise the difficulties in interpreting pooled data from several pedigrees.

Hamerton et al.[1] suggested preferential segregation and selective fertilisation as possible mechanisms to explain their observations. A "segregation distorter" (S.D.) gene, analogous to that described by Sandler and Hiraizumi [2] in wild populations of drosophila is another possible explanation. In the male fly, this gene acts during synapsis to cause a defect in replication of the normal homologue (formally equivalent to a chromosome break), thus allowing preferential recovery of the S.D. allele. The similarities of heterochromatin, intimate association with the centromere, and chromosomal aberration in the S.D. region of the drosophila chromosome and the human acrocentric chromosomes cannot be ignored.

Finally, I should like to comment on our serological data, testing the possibility of genetic linkage between seven different blood-group loci and the centromere of the translocation chromosome. The MNSs, Rh, P, and Duffy loci are probably not closely linked. No conclusions could be drawn regarding the Kell and Kidd loci because of the lack of critical matings. Independent assortment of the O allele of the ABO locus and the centromere of the translocation chromosome was not observed in this family, suggesting the possibility that this important autosomal marker is located on one of the acrocentric chromosomes.

Full details of this family and two other mongol families are in preparation.

Department of Human Genetics, University of Michigan Medical School, Ann Arbor, Michigan. MARGERY W. SHAW.

6. See *Lancet*, March 3, 1962, p. 470.
7. Hastings, J. J. H. *Pharm. J.* 1960, **184**, 45.

1. Hamerton, J. L., Cowie, V., Giannelli, F., Briggs, S., Polani, P. E. *Lancet*, 1961, ii, 956.
2. Sandler, L., Hiraizumi, Y. *Genetics*, 1960, **45**, 1671.