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## Cosmetic Plumbism

SIR,—We wish to draw your attention to further examples of plumbism among Indian children attributable to the application of lead-containing cosmetic preparations to the eyelids. This practice begins early in life and has been observed in the newborn nursery in this hospital. It seems to be particularly common among patients of Punjabi origin.

Over the last six months we have investigated 12 children from five families and found that ten had blood lead levels of more than 36  $\mu\text{g}/100\text{ ml}$  (the upper limit of normal for paediatric patients). Three patients (aged 2-3 years) had lead levels ranging from 61-69  $\mu\text{g}/100\text{ ml}$  and required rapid treatment. None of the above had a history of pica, and the homes were checked in some instances for potential sources of lead with no results. Samples of the cosmetic were obtained in three instances and were found to contain from 80 to 85% lead sulphide w/v. The cosmetic was in the form of a grey powder, and the small containers seemed unusually heavy. In all three families the substance had been brought into the country by a relative as a gift from India. One sample with a trademark MAV had an English inscription on the packet "Useful for eye disorders." Apart from the obvious risk of contaminating the fingers with transferral to the mouth, it is frequently the custom to instil this substance into the conjunctival sac.

There are two basic preparations in use. The first (Surma) is a powder applied to the eyelids with a needle or orange stick and is most likely to contain lead. The second (Kajal) is an ointment prepared by burning vegetable fat and adding charcoal to the

residue. Occasionally lead is added to this preparation also.

These preparations were the subject of a Ministry circular in 1968,<sup>1</sup> but remain a health hazard. Known importers of the materials were asked to voluntarily restrict supplies at that time. Constituting neither a food nor a drug, there is no legal restriction on the manufacture, import, or sale of such lead-containing "cosmetics."

We feel that it is time this situation was remedied by the appropriate legislation, and that in addition the hazard should be widely publicized among the immigrant population now at risk.—We are, etc.,

G. J. A. I. SNODGRASS  
DAVID A. ZIDERMAN  
V. GULATI  
JEAN RICHARDS

The London Hospital,  
London E.1

<sup>1</sup> *Lead Poison Warning*. Home Office Press Notice, 20 September, 1968.

## Spontaneous Abortion and Neural Tube Defects

SIR,—Dr. C. J. Roberts and Mrs. Setsuko Lloyd (6 October, p. 20) are to be congratulated on drawing attention to the possible effect of spontaneous abortions on the prevalence of neural tube defects at birth. Their hypothesis that differences in prevalence may be due to differential prenatal loss has important implications at the present time, when their prenatal diagnosis is becoming a feasible proposition.

It does seem that the incidence of such

malformations is higher in spontaneously aborted fetuses than in viable births. In a series of unselected spontaneous abortions we are currently collecting in London the prevalence amongst fetuses of 5 cm or longer is 20 per 1,000. However, of the 10 previous viable infants born to the 18 mothers of fetuses with neural defects, one had been anencephalic, suggesting that it may be the mothers also at risk of bearing viable affected infants who are contributing to the excess. This would be in agreement with the findings of Record and McKeown,<sup>1</sup> McDonald,<sup>2</sup> and others, who found that fraternities including affected infants also included an unduly high proportion of spontaneous abortions. This would not support the hypothesis put forward by Dr. Roberts and Mrs. Lloyd, though it is possible that the relationship between abortions and prevalence at birth may vary in different geographical areas.

It would be important to resolve the following questions. Does the spontaneous loss of fetuses with neural tube malformations occur primarily because of fetal defect? Or is the loss primarily due to maternal factors, in the sense that certain mothers are more efficient than others at "perceiving" and disposing of abnormal pregnancies? If the former proposition were true, the loss of a fetus with a neural tube defect should count equally with the birth of an affected infant as far as calculating the risk to a future pregnancy is concerned. The presumption would be that the viability depends somehow on the extent of the malformations, which might vary unpredictably between affected sibs. If the latter proposition were true, the loss of an affected fetus might have little or no bearing on the risk of recurrence