who, on the basis of observations on pigs, postulated that selenium and/or vitamin E deficiency might cause sudden death in human infants. In addition, there is the notion that breast-feeding may protect against this syndrome, which would be reasonable in the light of Money’s hypothesis and the requirement of vitamin E in human milk (7-8 IU/L). However, we have previously shown that the blood selenium levels of infants dying suddenly and inexplicably are essentially identical to those of normal controls;1 their plasma vitamin E levels appeared to be slightly lower than normal, but our initial studies were performed on only a small number of samples and thus did not permit a strong conclusion regarding this point.

We now have obtained additional data on the plasma vitamin E levels of infants dying suddenly and controls. Plasma samples from 18 victims of the sudden infant death syndrome, confirmed by necropsy, were obtained through the courtesy of Mr. Robert Creason, Coroner, San Diego County. Plasma samples from 17 normal control infants were obtained from Dr. Victor H. Lipp, Department of Pediatrics, School of Medicine, University of California, San Diego. Fresh cord blood samples1 from 18 normal neonates were obtained from the Blood Bank, University Hospital, San Diego. Total vitamin E levels in plasma were determined spectrophotometrically, after recovery of vitamin E and using a standard recovery curve.4,5 The observed vitamin E levels of neonatal plasma (0.28 ± 0.11 mg/100 ml), while lower than those noted by Nitowsky et al.,6 were similar to more recently published values.6-11 None had low plasma vitamin E levels than either the infants dying suddenly (0.49 ± 0.45 mg/100 ml) or control infants (0.79 ± 0.26 mg/100 ml; P = 0.01). The infants who died suddenly (age at death 2.33 ± 2.85 months), though younger than the controls (4.28 ± 1.97 months), did not have significantly lower vitamin E levels than the controls (P = 0.05). Amongst the infants who died suddenly but were unlikely to have been dying suddenly are seriously deficient in vitamin E during any period of their life.

To determine the effect of breast-feeding on the incidence of the sudden infant death syndrome, we studied dietary histories of 46 infants dying suddenly and 38 control infants in San Diego County. The two groups were similar in respect of parental social class, age, occupation, education, race, and income, date and hospital of birth, sex, race, birth order, and birth weight (P = 0.05).

In these samples similar proportions of infants dying suddenly (39%) and control infants (27%) were breast-fed during early infancy. This is comparable with the results of other studies.10 While the average age at death of the 27 infants dying suddenly who had been wholly formula-fed was 3.6 ± 2.79 months, the 19 totally or partially breast-fed infants died at 1.78 ± 0.70 months (P = 0.02); thus breast-feeding does not appear to protect against sudden death.

All the infants who died suddenly appeared to have received an adequate amount of dietary vitamin E, since the plasma vitamin E levels were part of the state of the newborn.1,2 In addition, both groups were part of the same population of infants, and the diet of breast-fed infants was the same as that of formula-fed infants.5,6

In conclusion, there was no significant difference in the plasma vitamin E levels of infants dying suddenly and controls. Though the significance of the latter observation still remains to be explained, no other differences between the dietary habits of the two groups were found. In agreement with earlier studies,11 we also noted a slightly higher incidence of maternal smoking during pregnancy in the sudden infant death group (one-tail, P = 0.05); other maternal factors were not significantly different. Though we found that breast-feeding has no protective effect,2 —We are, etc.,

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Abortion in 1972

Sir,—Because of my interest in and concern about abortion deaths I should like to comment on some of the statistics in the Chief Medical Officer’s annual report for 1972.1 (1) The number of haemorrhages in general are 2.9 per 1000 pregnancies when terminated before 13 weeks, and 6.1 for sepsis and 5.1 for haemorrhage at or after 13 weeks, these being rates per 1000 notified abortions in each group. In other words the risks are roughly doubled after the first trimester.

(2) The report states that the pregnancy was terminated at or after 13 weeks in 23% of abortions in 1971 compared with 79% in 1970. However the actual numbers of abortions performed after 13 weeks has increased from about 24,000 in 1970 to 29,000 in 1971.

(3) Of the 7,534 haemorrhages, 2,986 were performed before 13 weeks. It may be that this was to allow sterilization, but vaginal termination followed by laparoscopic sterilization or tubal ligation would surely be safer.

(4) A total of 6,532 vaginal terminations, either by dilation and curettage or by vacuum aspiration, were performed at 15-23 weeks and 63 after 24 weeks. I distrust the complication rates for these methods, chiefly because of the high incidence of 0-1 day in abortions performed in "approved places," and I personally regard vaginal termination after 15 weeks as a hazardous procedure.

(5) The sepsis rates when uterine paste is used, whether before (48-5 per 1000) or after 13 weeks (32-1 per 1000), should surely condemn this method.

(6) Though there has been an increase in abortions performed on women from abroad (50,179 in 1972) it should be noted that just over 50,000 women resident in England and Wales had abortions in "approved places," compared with 55,826 in N.H.S. hospitals.

The annual total of abortions continues to rise, but abortions must be regarded as the least satisfactory and most dangerous method of birth control. I think it is unlikely that we shall copy the Chinese in rejecting promiscuity, but the only alternative is to persuade participants to use the more efficient methods of contraception which have been developed. Meanwhile abortions after the first trimester should be done only if there is a danger to life or a serious risk of fetal abnormality.—I am, etc.,

HUMPHREY ARTHUR

London W.1


Rubella Arthritis

Sir,—Your leading article (27 October, p. 186) prompts me to report a possible associated complication of rubella.

After rubella a 35-year-old married woman developed polyarthritis which, on a weekly basis, responded to phenylbutazone. Ten days later she developed an extremely acute right carpal tunnel syndrome with lesser symptoms in the left hand. As she was not relieved by pethidine or by local injection of hydrocortisone, after three sleepless nights complete relief was obtained by division of the flexor retinaculum under a general anaesthetic. The flexor synovial sheath was seen to be pale yellow, glistening, and swollen. Examination of a section (Dr. R.T. Cooke) showed delicate infiltration with polymorphonuclear leucocytes. The less severe pain in the left hand persisted and was relieved recently by operation.

There were no general signs of fluid retention, so I presume that the wrist synovial swellings were part of a viral synovitis rather than a side effect of phenylbutazone.—I am, etc.,

WAY ELLIS

Stockton-on-Tees

Influence of Digitalis on Labour

Sir,—The conclusions reached by Drs. Judith B. Weaver and J. F. Pearson (9 September, p. 519) concerning the apparent