completely normal. A chest X-ray confirmed slight cardiomegaly and the lung markings were rather prominent. An electrocardiogram was normal. There was slight wedging of the seventh thoracic vertebral body consistent with Scheuermann's disease. Skull X-ray was normal apart from revealing a bony defect in the hard palate. No chromosomal abnormality was detected in cultured leucocytes. Skin and muscle biopsy specimens were normal. Extensive dental investigations suggested that the lack of permanent dentition was a developmental defect.

At the age of 17½ years the patient had an attack of infectious hepatitis. At 19 years he developed a tuberculous pyothorax presenting as a cold abscess in the right loin, and investigations of his liver function at that time revealed chronic active hepatitis.

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L.S.D., INTRAUTERINE AMPUTATIONS, AND AMNIOTIC-BAND SYNDROME

SIR,—The striking photograph of the hand of a child whose mother took L.S.D.[1] prompted us to review the anomalies found in 6 malformed infants born to mothers using L.S.D.[1-6] 3 patients presented with three very different and known developmental anomalies (fibular aplasia, trisomy 13, and spondylothoracic dysplasia). The deformities reported in the 3 other infants [1-3] are quite similar and present the features of lesions produced by "amniotic bands". We have seen 15 cases of the amniotic-band syndrome in newborn infants since 1958, all documented by complete placental examination.[7] In addition, we are following 5 suggestive cases in the orthopædic clinic. To our knowledge, none of the mothers took L.S.D.

1. Assemany, S. R., Neu, R. L., Gardner, L. I. *Lancet*, 1970, i, 1290.
2. Carakushansky, G., Neu, R. L., Gardner, L. I. *ibid.* 1969, i, 150.
3. Hecht, F., Beals, R. K., Lees, M. H., Jolly, H., Roberts, P. *ibid.* 1968, ii, 1087.
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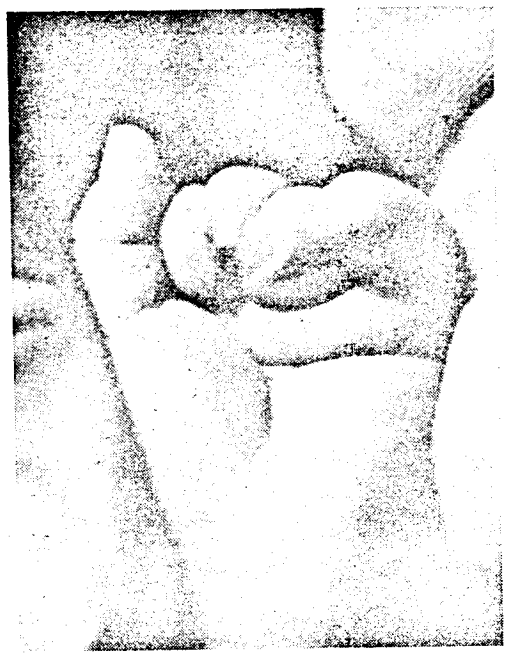

Fig. 1—Distal syndactyly and amputation in a proven case of amniotic-band syndrome.

Notice the thin fibrous band extending from the point of syndactyly of the middle three fingers.

Fig. 2—Surface of placenta from the baby whose hand is illustrated in fig. 1.

The amnion is completely absent from the fetal surface of the placenta except for a small ring around the insertion of the umbilical cord. The arrow points to a fibrous band extending from the surface of the placenta.

The evidence supporting the existence of amniotic bands and their probable pathogenesis has been reviewed in the comprehensive monograph by Tordin.[8] His observations and ours suggest that the amnion occasionally becomes separated, completely or in part, from the chorion. The free amnion or its mesodermal connections with the chorion will then assume various shapes (threads, bands, pockets), depending on the type and timing of rupture(s) and on the stresses to which it is subjected. The entanglement of fetal parts in the loose amnion or mesodermal bands may lead to adhesions of various parts of the fetal surface to the placenta, to constrictive banding and scarring with distal œdema, decrease in vascular supply, and atrophy or even complete amputation of the affected parts. Constriction rings and distal syndactyly are frequently encountered. The associated transient or permanent oligohydramnios is thought to be responsible for positional deformities such as clubfeet.

The constriction rings and amputations in the report by Assemany et al.,[1] the amputations and distal syndactyly in the report by Carakarshansky,[2] and the amputation in the report by Hecht et al.[3] are phenotypically similar to those seen in some of our proven cases of amniotic-band syndrome (figs. 1 and 2).

The association of exposure to L.S.D. and amniotic band syndrome is striking in this small group of patients. Its significance cannot be evaluated in the absence of precise data on the incidence of L.S.D. ingestion in large populations of pregnant women. The amniotic-band syndrome has an

7. Blanc, W. A. *Am. J. Path.* 1970, **59**, 27a.
8. Tordin, R. C. Fetal Malformations Caused by Amnion Rupture During Gestation. Springfield, Illinois, 1968.

incidence estimated at 1 per 10,000 to 1 per 5000 pregnancies; therefore one could expect 1 case per year in any large centre.

The examination of the placenta and fetal membranes in further cases of anomalies associated with L.S.D. ingestion will show how frequently this pathogenic mechanism can be incriminated. Possibly other factors could lead to direct lesions of embryonic or fetal parts phenotypically similar to those produced by amniotic bands.[9,10] L.S.D. might produce a predisposition to early rupture or separation of the membranes; it seems reasonable, however, to suspect that severe trauma incurred during a bad " trip " might also be a factor.

If more cases are detected, then L.S.D. may be considered teratogenic [11] through the indirect production of fetal damage.

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COITAL RATE, SEX RATIO, AND SEASON OF BIRTH

SIR,—Correspondents [12, 13] have discussed the possibility of testing my hypothesis [14] (that sex ratio is partially dependent on coital rate) by analysing seasonal variations in sex ratio.

Given that there is a seasonal variation in birth-rate, we do not know how much this is due to seasonal variations in coital rate and how much is due to other factors such as differences in fetal-wastage rate, anovulatory cycles or some other physiological component of fertility, or to changes in the size of the sexually active population (caused, for example, by men in seasonal work returning home at Christmas and for the family's summer holidays).

Nevertheless, let us suppose that *all* the variation in birth-rate is caused by variation in coital rate. What amount of seasonal variation in sex ratio would then be predicted by my theory ?

The lowest monthly birth-rate in England and Wales is about 90% of the highest monthly birth-rate. So let us suppose that the lowest monthly coital rate is about 90% of the highest. The coital rate at age twenty is about double that at age forty.[15] I have estimated, using official data for England and Wales, July, 1938, to December, 1968, that the probabilities of a male birth (live or still) in legitimate confinements to women aged twenty and forty are, respectively, 0·5157 and 0·5133. These values differ by 0·0024.

Making the assumptions above, we wish to estimate the seasonal range of sex ratio associated with a seasonal range of mean coital rate that is not more (and probably less) than a fifth of that reported above. It seems reasonable to suppose that it would be less than a fifth of the magnitude of the range of sex ratio reported above—i.e., less than 0·0005. If we want even a 50% chance of detecting at the 5% level of significance such a difference in sex ratio in two numerically equal samples, each would have to number about 8 million: for a 95% chance, we would need two samples each of 26 million.[16] To accumulate, for example, 8 million January births for England and Wales would take roughly a hundred and twenty years, so I do not feel that my hypothesis can be refuted by failure to detect seasonal variations in sex ratio in comparatively small samples.

Considerably less data would be required to detect a correlation between monthly sex ratios and monthly birth-rates. However, in England and Wales, the conception-rate waxes and wanes seasonally in reasonably close agreement with the temperature, except for a secondary peak in conceptions around Christmas and the New Year. Since it seems rather doubtful that this secondary peak is primarily due to a rise in coital rate (as opposed to a rise in the active population) it would be inappropriate to correlate monthly birth-rates against monthly sex ratios to test my hypothesis.

The U.S. sex ratio for 1915–36 and 1942–48 was highest for births between May and July and lowest for births from October to March.[17] This tendency has persisted. However, the months with the highest number of marriages in the U.S. seem to be June to September,[18] there being nearly 2½ times as many marriages in June as in January. It is virtually certain (since there is so striking a seasonal variation in marriages) that there is a considerable seasonal variation in births of first children conceived within one month of marriage. These children seem to have higher sex ratios than children conceived later in marriage [19] (perhaps, as I suggested,[14] because of the high coital rates during the first month of marriage, but perhaps for other reasons). In short, though the observed seasonality in sex ratio in the U.S. is compatible with my hypothesis, nevertheless it would be rash to construe it as more than weak evidence.

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THREE HERNIAL SACS

SIR,—A man aged 45 years was admitted as an emergency with a strangulated right inguinal hernia. On examinaiton, the mass was found to be differentiated into three parts. It was tender and irreducible; taxis under pethidine (meperidine), 100 mg. intravenously, was unsuccessful. One hour later the mass was explored in the operating-theatre. A transverse incision was made and reflexion of the skin-flaps showed three swellings, the upper two of which were separated from the lower one by the inguinal ligament. The inguinal canal was opened, and the lateral mass was found to be an indirect inguinal hernia sac containing omentum; this was reduced, and the sac excised. The medial mass was a big thick-walled sac—a direct inguinal hernia. The sac was opened, and was found to contain part of the anterior bladder wall. The sac was excised, and the inguinal canal repaired. The third (lower-medial) mass was the sac of a femoral hernia. This was opened, and contained a loop of terminal ileum with a subserosal hæmatoma. The loop was reduced; the sac excised; and the wound closed in layers.

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