detected until it had resulted in cardiac arrest. This can easily happen; I have reported[1 2] several fatalities from this cause with specialist anaesthetists.—I am, etc.,

J. G. BOURNE

Salisbury, Wilts

1 Bourne, J. G., *Anaesthesia*, 1970, **25**, 473.
2 Bourne, J. G., *Lancet*, 1973, **1**, 35.

X-Trisomy Associated with Hodgkin's Disease

SIR,—X-trisomy is not connected with any characteristic clinical syndrome. Many 47,XXX women have no clinical abnormality, gynaecolgical or otherwise, and are even fertile. Others are mentally retarded.[1-3]

We should like to report a case of X-trisomy associated with Hodgkin's disease. An 18-year-old girl (whose mother was aged 42 and father aged 45 at the time of her birth) had generalized lymph node enlargement which had appeared one month previously. Biopsy of a lymph node showed Hodgkin's disease. The patient was of medium growth and had menstruated regularly since the age of 14. Gynaecological examination showed a slightly hypoplastic uterus. Her mental development was normal (I.Q. not estimated).

The cells obtained directly from the lymph node and from culture of peripheral lymphocytes contained 47 chromosomes, the additional one being the size of a member of group C. The presence of an extra chromosome in group C in a patient without phenotype anomalies suggested that it was an X chromosome. This suggestion was supported by the occurrence of double Barr's bodies in cells from buccal mucosal smears (fig. 1). Thus the 47,XXX karyotype was

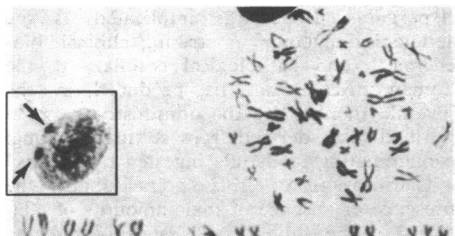

Fig. 1.—Lymph node cell with a 47,XXX karyotype. Inset—Buccal epithelial cell with double Barr's bodies.

established. Mitoses from the peripheral blood showed a short-arm deletion of a G chromosome, probably 22 p-, in 30% of the cells counted. A similar anomaly was described by Gunz and Fitzgerald in lymphatic leukaemia.[4] The fact that this anomaly was not seen in lymph node cells in our case is difficult to explain. Tetraploid cells were an almost constant feature in cases of Hodgkin's disease observed by Coutinho *et al.*,[5] though in some cases chromosomal abnormalities were not found in lymph node cells.

It is known that constitutional chromosome anomalies may predispose to neoplasia.[6] On the other hand the results of O'Riordan *et al.*[7] indicate that (with the exception of Down's syndrome) there is no increased susceptibility to cancer in general among persons who carry autosomal rearrangements or sex chromosome aneuploidy.—We are, etc.,

HALINA LECH
BARBARA POLANIECKA
LUCJAN WISNIEWSKI

Department of Clinical Genetics,
Institute of Obstetrics and Gynecology,
Białystok Medical School,
Warsaw

1 Dewhurst, C., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1971, **78**, 1058.
2 Lucas, M., Dewhurst, C. J., Hurley, R., Anderson, S., and Blunt, S., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1971, **78**, 1087.
3 MacLean, N., Court Brown, W. M., Jacobs, P. A., Mantle, D. J., and Strong, J. A., *Journal of Medical Genetics*, 1966, **5**, 165.
4 Gunz, F. W., Fitzgerald, P. H., and Adams, A., *British Medical Journal*, 1962, **2**, 1097.
5 Coutinho, V., Bottura, C., and Falcao, R. P., *British Journal of Cancer*, 1971, **25**, 789.
6 Koller, P. C., *The Role of Chromosomes in Cancer Biology* (Recent Results in Cancer Research No. 38). London, Heinemann, 1972.
7 O'Riordan, M. L., Langlands, A. O., and Harnden, D. G., *European Journal of Cancer*, 1972, **8**, 373.

Cauda Equina Syndrome in Ankylosing Spondylitis

SIR,—The association between the cauda equina syndrome in ankylosing spondylitis and posteriorly projecting diverticula from the lumbosacral canal confirmed by Dr. D. J. Thomas and others (26 January, p. 148) has been fully established. Since reporting my original cases[1] I have seen one further example and have been sent details of two other cases. Similar findings have been reported by other observers[2 3] who have commented on the widening of the canal often seen. These diverticula will not be seen if myelography is performed in the usual prone position as they do not extend posteriorly but excavate the laminae and spinous processes.

How these remarkable destructive lesions arise and how they could be responsible for the cauda equina lesion is quite unknown, but indeed very little seems to be known about the state of the meninges and the spinal canal in this disease in the absence of this neurological complication.—I am, etc.,

W. B. MATTHEWS

Department of Clinical Neurology,
Churchill Hospital,
Oxford

1 Matthews, W. B., *Journal of Neurological Sciences*, 1968, **6**, 561.
2 Rosenkranz, W., *Journal of Neurosurgery*, 1971, **34**, 241.
3 Russell, M. L., Gordon, D. A., Ogryzlo, M. A., and McPhedran, R. S., *Annals of Internal Medicine*, 1973, **78**, 551.

Histidine for Treatment of Uraemic Anaemia

SIR,—Professor C. Giordano and his colleagues in Naples have recently presented data suggesting that histidine supplements improve the anaemia associated with chronic renal failure.[1] But there are some inconsistencies in their work on this subject which make it difficult to interpret the results.

The data from 20 of the 28 patients whom they discuss in this recent paper were previously reported at the European Dialysis and Transplant Association (E.D.T.A.) meeting in Vienna in June 1973.[2] These 20 patients were then stated to have received no iron supplements, whereas according to the recent paper[1] the 28 patients were given iron and folic acid. As patients on haemodialysis are sometimes iron-deficient it is important to know whether or not the patients were given iron. The protocol of the study described in the abstract[3] of the E.D.T.A. communication was different from that of the study which was subsequently published.[1 2] In both studies patients on maintenance haemodialysis were given histidine supplements. According to the abstract[3] the reticulocyte count rose significantly after histidine, whereas in the most recent paper[1] there was a significant fall in reticulocytes by the end of two months' treatment with histidine.

The patients' diet is described in the two papers[1 2] as a "free diet" without the protein intake being specified. The protein content of the diet is of immediate relevance in a study in which it is claimed that amino-acid supplements are necessary. There is some evidence that the patients were eating a low-protein diet. Fifty per cent. of the patients received only 12-15 hours of haemodialysis per week. But in spite of being underdialysed their predialysis blood urea levels varied from only 75 to 110 mg/100 ml. This suggests that the patients were eating a low-protein diet, perhaps because of habit or advice or because of the anorexia associated with underdialysis. It is possible, therefore, that the low plasma histidine levels before histidine treatment were due in part to deficiency of protein and therefore of histidine. It would seem more reasonable to correct this deficiency by increasing the protein intake and, if necessary, the hours of dialysis than to attempt to correct with histidine supplementation what is probably a generalized amino-acid deficiency.

Professor Giordano suggests that there should be an extensive controlled trial of histidine treatment for the anaemia associated with chronic renal failure. Before this suggestion is acted upon it would be of great help if the data concerning iron supplements, reticulocyte response, and dietary protein intake could be clarified.—We are, etc.,

M. PHILLIPS
C. POLITIS-TSEGOS
HUGH DE WARDENER

Charing Cross Hospital Medical School,
London W.6

1 Giordano, C., *et al.*, *British Medical Journal*, 1973, **4**, 714.
2 Giordano, C., *et al.*, *Proceedings of the European Dialysis and Transplant Association*, 1973, **10**, 160.
3 Giordano, C., De Santo, N. G., Rinaldi, S., Esposito, R., and Acone, D., *Kidney International*, 1973, **3**, 4A.

Oral Contraceptives and Thromboembolism

SIR,—I was rather surprised to read the paper by Mrs. May A. Badaracco and Dr. M. P. Vessey (9 February, p. 215) which draws important conclusions about oral contraceptives from what appears to be slender evidence from the patients involved.

In your leading article (p. 213) you are happy to accept this evidence. You quote Drill,[1] who reported on 15 prospective studies each involving more than 1,000 woman-years of use of the contraceptive pill.