each twin. Because of the presence of foreign leucocytes it seems more appropriate to call these people blood chimeras than blood-group chimeras. We have found no evidence of hormonal effects; Mrs. W. is a normal healthy woman with three healthy children and therefore not a freemartin.

There are two pairs of twins among first cousins, but one of each pair died in infancy. Attempts have been made to investigate the survivors, but so far without success.

Summary

A pair of binovular twins is described, each of whom has blood which is a mixture of the same two distinct types differing in ABO, MN, and Rhesus groups. Each twin has, however, only a single Lewis group, and the Lewis groups of the twins are different, each corresponding to that expected from the secretor status. A family study of the blood groups has been made. Nuclear sexing of neutrophils shows that foreign leucocytes are present in the blood of the male twin and probably present in the female twin.

It is considered that these are chimeras similar to that reported by Dunsford *et al.* The Lewis group findings confirm the demonstration by Sneath and Sneath that the Lewis antigen belongs to the tissues and is only secondarily attached to the red cells. Results in the Lewis group are comparable with those in the J group of bovine chimeras.

We are indebted to Mrs. W. and all her family for their co-operation in this investigation; to Dr. Dorothy Parkin, of the Medical Research Council Blood Group Reference Laboratory, for some of the preliminary investigations of the twins, and also for first demonstrating that Mr. G. is also a chimera; to Dr. R. R. Race, Dr. Ruth Sanger, and Miss Phyllis Moores, of the Medical Research Council Blood Group Research Unit, for checking our results, determining the Kidd and S groups, and examining A cells separated by their technique. We are also grateful to Dr. Race for much valuable advice and to Dr. W. M. Davidson, of King's College Hospital Medical School, for undertaking the nuclear sexing.

REFERENCES

- Anderson, D., Billingham, R. E., Lampkin, G. H., and Medawar, P. B. (1951). *Heredity*, 5, 379.
Booth, P. B., Plaut, G., James, J. D., Ikin, E. W., Moores, P., Sanger, R., and Race, R. R. (1957). *British Medical Journal*, 1, 1456.
Davidson, W. M., and Smith, D. R. (1954). *Ibid.*, 2, 6.
Dunsford, I., Bowley, C. C., Hutchison, A. M., Thompson, J. S., Sanger, R., and Race, R. R. (1953). *Ibid.*, 2, 81.
Owen, R. D. (1945). *Science*, 102, 400.
Race, R. R., and Sanger, Ruth (1954). *Blood Groups in Man*, 2nd ed. Blackwell Scientific Publ., Oxford.
Sneath, J. S., and Sneath, P. H. A. (1955). *Nature (Lond.)*, 176, 172.
Stormont, C. (1949). *Proc. nat. Acad. Sci. (Wash.)*, 35, 232.

Medical Memorandum

Leukaemia and Oestrogen Therapy

Oestrogens have been shown by many workers to produce neoplasms in experimental animals. The risk of producing or accelerating neoplastic growth in human therapy is usually thought to be entirely hypothetical, provided that the dosage is within the usual therapeutic limits, which is much less than that used in experimental work.

Kirschbaum (1951), Burrows and Horning (1952), and Gardner (1953) have reviewed the literature on the effects of oestrogens on the incidence of leukaemia in mice. It is clear that, experimentally, oestrogens do increase the incidence of leukaemia in animals and that this is of the lymphatic type.

No record has been found of human lymphatic leukaemia associated with oestrogen therapy. A case is recorded in which lymphatic leukaemia occurred during stilboestrol therapy for carcinoma of the prostate.

CASE REPORT

A man aged 70 was seen as an out-patient in March, 1953, with symptoms of frequency and dysuria, said to be of six weeks' duration. The prostate was not enlarged, but was hard and nodular. The other findings relative to our story were that the liver was not palpable and that no abdominal mass was felt, indicating that the spleen was not palpable. Some posterior cervical glands were noted. Radiological examination revealed no metastases in spine, pelvis, and skull. The blood count on April 1 showed: haemoglobin, 86% (12.6 g./100 ml.); R.B.C., 4,410,000 per c.mm. A leucocyte count was not made. The formaldehyde stable acid phosphatase was 35 units per 100 ml. A diagnosis of prostatic carcinoma was made, and stilboestrol therapy was begun. He was treated as an out-patient. About the middle of April 10 mg. thrice daily was prescribed, and during the next few months he had 4,000 mg.

On September 19 his general practitioner observed marked oedema of the legs and an enlarged spleen. He was referred to the out-patient department, and the blood count on September 29 was: haemoglobin, 55% (8.1 g./100 ml.); R.B.C., 2,800,000 per c.mm.; formaldehyde stable acid phosphatase, 0.8 unit per 100 ml. A leucocyte count was not done.

On October 31 he was admitted to hospital with severe symptoms of anaemia. Blood count on November 1 showed haemoglobin, 28% (4 g./100 ml.); R.B.C., 1,620,000 per c.mm. Leucocytes 44,000 per c.mm. (lymphocytes and smear cells 95%, polymorphs 5%). Spleen was enlarged to the level of the umbilicus. Blood transfusions were given, and the degree of anaemia fluctuated accordingly. The total leucocyte count increased to 70,000 on November 12 and fell to 38,200 on the 16th, with no change in the type of cell present. Marrow aspirated from the ilium on November 10 showed the appearances typical of lymphocytic leukaemia. Death occurred on November 21.

At post-mortem examination enlarged glands were found in the neck, mediastinum, mesentery, and around the aorta and common iliac arteries. The spleen was grossly enlarged (1,400 g.), and had some infarcts. The prostate was enlarged, but not hard; it contained a few small firm masses. The marrow of the lower lumbar vertebrae was leukaemic. In the prostate was a moderately well differentiated carcinoma. The spleen and glands showed typical lymphatic leukaemia.

COMMENT

There is no proof that this patient did not have leukaemia before receiving stilboestrol therapy. The leucocyte count was not performed, but, if normal, lymphatic leukaemia would not have been excluded, as experience shows that when all cases of anaemia are exhaustively investigated the aleukaemic variety is relatively common. The haemoglobin and red-cell count were almost normal at the beginning of therapy; no splenic enlargement was noted, but posterior cervical glands were present. If leukaemia were already present before stilboestrol therapy, progress would at this age be slow, and the cell type was well differentiated.

Thus there are three alternatives: the stilboestrol induced the leukaemia; the stilboestrol accelerated the leukaemia; the stilboestrol did not affect the course of the leukaemia. But a lymphocytic leukaemia of well-differentiated cell type in an elderly person presented as a rapidly progressive and fatal anaemia. Therefore I regard this last alternative as improbable.

Co-operation from Mr. F. I. Evans, Dr. Blair Macaulay, and Dr. N. C. W. Owen is gratefully acknowledged.

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REFERENCES

- Burrows, H., and Horning, E. S. (1952). *Oestrogens and Neoplasia*, p. 80. Blackwell, Oxford.
Gardner, W. U. (1953). *Advances in Cancer Research*, 1, p. 204. Academic Press, New York.
Kirschbaum, A. (1951). *Cancer Res.*, 11, 742.