in many instances observed for comparatively short periods, and that three out of only eight cases had survived for five to six years.

Professor McMichael began his series in 1951 and had seen 73 cases, of whom 11 died before treatment was started and 10 others were too advanced to respond (blood urea over 100 mg. per 100 ml.). The *estimated* 5-year-survival rate of the remaining 52 cases with an initial blood urea under 60 mg. per 100 ml. was 50%; this was calculated from a survivorship table expressing the experience of the group to that date, but does not mean every member of the group had been followed for five years. In an earlier report[3] the survival curve of the first 32 cases to the 3-year stage was shown, and came down to about 35% at three years. Combining these two reports, 20 cases had been followed for less than two years, but the early mortality of these had been sufficiently low to raise the 5-year expectancy of the whole group to 50%. The number actually observed for the full five years was probably fairly small.

Most people, I think, now accept that in the best hands ganglion-blocking agents are capable of yielding better results than sympathectomy, and this is reflected in the small number of sympathectomies, less than 10 per year, carried out by Mr. H. S. Shucksmith since 1953. However, it should also be pointed out that these results are not easily obtained, even if "confidently expected" by Dr. Stewart. Both Smirk and McMichael describe elaborate schemes of treatment and careful follow-up arrangements, and their results are undoubtedly due to meticulous attention to detail. In this respect, the comments of Smirk on his grade 3 cases are illuminating. For some years rather less detailed attention was given to this group than to group 4, and he found results were no better than in the malignant grade; it was only when group 3 was given equal attention that these rather more favourable cases experienced a lower mortality rate. Evidently success with ganglion-blocking agents is closely related to detailed supervision by the physician and complete co-operation by the patient. When co-operation is less than perfect, when the patient lives too far away from detailed supervision, or when side-effects of the drugs are prohibitive, sympathectomy offers better prospects than imperfect or ineffective medical treatment. Such cases occur somewhat more frequently than Dr. Stewart's backward adolescent with malignant hypertension.—I am, etc.,

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[1] *Hypotensive Drugs*, edited by M. Harington, 1956, p. 109. Pergamon Press, London.
[2] Smirk, F. H., *High Arterial Pressure*, 1957. Blackwell, Oxford.
[3] McMichael, J., and Murphy, E. A., *J. chron. Dis.*, 1955, **1**, 527.

Anaemia and Polycythaemia in Uniovular Twins

SIR,—The paper by Dr. Margaret M. Kerr (*Journal*, April 4, p. 902) is of great practical importance, and she is to be congratulated on publishing it. It is not only of importance to paediatricians but should be brought to the notice of obstetricians and midwives.

Six weeks before the publication of her paper I was asked to see one of female twins at Highbury Hospital, Nottingham, on account of obvious clinical anaemia.

The mother was Rhesus-positive and this baby (twin 1) was pale, haemoglobin 70%; the other baby seen for comparison (twin 2) can only be described as "red"—the haemoglobin was not estimated. No explanation such as blood loss could be found to explain the anaemia. The anaemia was treated with intramuscular iron.

It was only after beginning to read through Dr. Kerr's paper that the explanation of a distinct clinical diagnostic puzzle became clear. Subsequent retracing of the history of the confinement showed that the anaemic twin was born in a shocked state and was very pale. There was a single large placenta, but unfortunately there are no further details.

Inquiry amongst several experienced midwifery sisters has disclosed the fact that they can recall quite a number of occasions on which they have observed clinical pallor in one twin. I must confess that I have up to now not noticed this, and have probably overlooked it as of no importance. It would therefore appear likely that Dr. Kerr's statement that "The syndrome, especially in a mild form, is probably more common than is realized . . ." is correct, especially from the observations of midwives.

Dr. Kathleen Laing, paediatric registrar, when the matter was being fully discussed, then recalled almost certainly another example of the syndrome seen in the Paediatric Baby Unit of the City Hospital, Nottingham, in October, 1958.

Mother group O, Rhesus-positive. Severe toxaemia hydramnios and accidental haemorrhage. *Twin 1.*—Vertex. Birth weight—3 lb. 5 oz. (1.5 kg.). Cried and then collapsed with apnoea for 20 minutes. Cyanosed. Haemoglobin 18 g./100 ml. Developed a cerebral syndrome and died on 13th day. Necropsy: intracerebral haemorrhage. *Twin 2.*—Breech. Birth weight 3 lb. 8¼ oz. (1.6 kg.). Baby cried soon after birth but noticed to be very pale. Haemoglobin 5.1 g./100 ml. Immediate transfusion of 60 ml. packed cells into saphenous vein. Baby survived.

Perusal of the maternity notes of the mother disclosed that the placenta was uniovular and very large and had two joined portions, one very congested and small and the other large, pale, and oedematous. The description of the placenta is similar to Cases 1 and 2 described by Dr. Kerr.—I am, etc.,

Nottingham Children's Hospital, Nottingham. A. P. M. PAGE.

Hereditary Haemorrhagic Diseases

SIR,—In his paper on the hereditary haemorrhagic diseases (*Journal*, April 25, p. 1059) Professor Armand J. Quick states with reference to Christmas disease (haemophilia B) . . . "The possibility is presented that a condition may perhaps occur in which lack of activation rather than a true deficiency of Christmas factor is the basic defect." We believe that we have in fact found such a case in a lady of 74 who has had a long-standing haemorrhagic thrombocythaemia. The Christmas defect was picked up by the thromboplastin generation test[1] and confirmed by a failure of mutual correction with known Christmas deficient sera.

The important feature is that the patient's $Al(OH)_3$-treated plasma, in the presence of patient's platelets, normal platelets, and phospholipid platelet substitute[2] respectively, partially corrected her serum defect in the thromboplastin generation test, in contradistinction to normal $Al(OH)_3$-treated plasma, suggesting that the presumptive inactive Christmas factor is resistant to absorption by inorganic gels.

After treatment with ^{32}P, the patient's platelets fell from 2,000,000 to 260,000 per c.mm. and the Christmas factor level rose from 30% to 137%. These Christmas assays were kindly carried out for us by Dr. F. G. Bolton. (The lowest limit of normal is 68%.[3]) At the time of these investigations the patient's one-stage prothrombin time, thrombin generation, and prothrombin consumption tests