Although this is apparently a simple method there was a great deal of operator variation, and further investigations into the technical factors affecting the results of multiple-puncture vaccination need to be carried out in the hope of modifying this technique to make the results comparable with those produced by intradermal vaccination.

We thank Dr. Arnold Brown, Medical Officer of Health for Cheshire County Council, for allowing this work to be carried out, and Professor Wilfrid Gaisford, Department of Child Health, Manchester University, for his encouragement and advice. The vaccine was prepared at, and supplied by, the Glaxo Laboratories, and we are grateful to Dr. J. Ungar and Dr. P. W. Muggleton for their practical help throughout the investigation. The Type A guns and the roughened needles were supplied by Messrs. Allen and Hanburys, of London, and the Type B self-firing gun was supplied for the B.T.A. pilot trial by Messrs. H. G. East, of Oxford. The photographs were taken by Mr. D. F. Boxall.

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Bowdler (1958). Zarafonitis *et al.* (1957) describe the unique sequence of pernicious anaemia, polycythaemia, and acute leukaemia occurring in the same patient.

Case Report

A married woman aged 66 was first seen by one of us (W. R.) in October, 1945. She complained of loss of weight, lethargy, and cramps in the legs. These symptoms had developed gradually over the preceding few months and her memory had deteriorated. Her previous health had been good, her husband stating that she had usually been of good facial colour.

Clinically she was a small, spare woman with slight kyphosis. She did not appear obviously anaemic. No noteworthy physical signs of disease were found apart from a sensation of fullness on palpation of the left hypochondrium. When seen in consultation by Dr. Bellingham Smith a diagnosis of carcinoma of the stomach or colon was considered. Blood examination at that time showed: Hb, 36% (5.3 g./100 ml.), R.B.C., 2,110,000/c.mm.; C.I. 0.9. The red cells showed macrocytosis, and varied in size and shape. A barium-meal examination showed no radiological abnormality of stomach or duodenum, but as the patient was unwilling to be admitted to hospital (an attitude she firmly maintained subsequently) she was treated at home with intramuscular injections of liver extract ("campolon") and oral iron. Such treatment was associated with rapid clinical and haematological improvement (see Chart) and five months later the somewhat surprising result of Hb 135% (20 g./100 ml.) and R.B.C. 7,280,000/c.mm. was reported. Clinically she had become obviously polycythaemic with red face and watering blood-shot eyes. A mass now readily palpable in the left hypochondrium was regarded as an enlarged spleen with a firm edge. The patient had lost all her old symptoms and felt remarkably well.

All treatment was discontinued and she apparently remained well until the autumn of 1948, when her original symptoms returned. In December, 1948, severe macrocytic anaemia was again present: Hb, 44% (6.5 g./100 ml.); R.B.C., 2,000,000/c.mm.; C.I., 1.1. The spleen was larger. Treatment with further parenteral liver extract alone was again followed by prompt improvement with another meteoric rise in Hb and red-cell count. Within three months of restarting liver therapy the Hb was 140% (20.8 g./100 ml.) and R.B.C. 8,500,000/c.mm. She felt well but was very plethoric. The spleen was now palpable fully a handbreadth below the left costal margin, and the liver was also palpable. There had never been any enlargement of lymphatic glands—or symptoms of glossitis, although her tongue was very red. Once again treatment was stopped, but anaemia returned. Liver extracts of various sources of manufacture were subsequently given intermittently.

In March, 1951, the patient was referred to one of us (J. N. M. C.) at St. George's Hospital, London, and more detailed studies were made. She was then aged 72 and had received an injection of refined liver extract a month before her attendance at hospital.

Clinically she did not appear anaemic—her face being somewhat florid, her conjunctivae injected, and her tongue very red although it was not smooth. While she was of small build there were no signs of wasting and there was no evidence of enlargement of superficial lymphatic glands. The abdomen was protuberant; the spleen was palpable fully a handbreadth below the left costal margin and the liver was also palpably enlarged. There was no evidence of ascites. Tachycardia associated with a collapsing pulse was present; B.P. 185/85 mm. Hg. There was no abnormality of the reflexes. Vibration sense over the shins was poorly appreciated. The urine was normal on routine testing; X-ray examination of the chest, spine, and long bones showed no abnormality apart from senile osteoporosis and osteoarthritis. A radiograph of the chest showed the lung fields to be clear, the transverse diameter of the heart being increased.

MEGALOBlastic ANAEMIA ALTERNATING WITH POLYCYTHAEMIA VERA

BY

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The development of polycythaemia vera in a patient previously proved to have megaloblastic anaemia associated with vitamin-B₁₂ deficiency is a haematological rarity, and only a small number of such cases, varying in degree of detailed study, have been reported. It is of interest to note that Sir William Osler (1903) in his classical paper on polycythaemia mentions a patient who was "as red as a beet" and suspected of having polycythaemia vera, but who had had, in fact, blood-count changes resembling pernicious anaemia. The most recent accounts of the association of pernicious anaemia and polycythaemia vera in the same patient are those of Hinz (1957) and Ellman and

Blood examination showed macrocytic anaemia: Hb, 52% (7.7 g./100 ml.); R.B.C., 1,830,000/c.mm.; P.C.V., 23%; M.C.V., 126 cubic microns; M.C.H.C., 33%; W.B.C., 10,700/c.mm. (polymorphs, many showing nuclear hypersegmentation, 76%, lymphocytes 19%, monocytes 5%). Red cells showed considerable variation in shape and size, with many macrocytes and rather oval cells. Platelets were numerous, with many large forms. Serum bilirubin was 1.3 mg./100 ml., serum urea 28 mg./100 ml., and direct Coombs test and Wassermann reaction were negative. Sternal puncture was easily performed, but only a small and unsatisfactory specimen of marrow was obtained. Smears showed megaloblasts, transitional cells, and large myelocytes and metamyelocytes—but such nucleated cells were not numerous. Masses of platelets were present but few intact megakaryocytes.

40 µg. of vitamin B₁₂ was given intramuscularly. In 17 days the Hb had risen to 89% (13.2 g./100 ml.), R.B.C. to 3,800,000/c.mm., and P.C.V. to 41%. A neutrophil leucocytosis of 23,000/c.mm. (metamyelocytes 17%, polymorphs 67%, eosinophils 4%, lymphocytes 11%, monocytes 1%) had occurred. E.S.R. (Wintrobe) was 2 mm. in one hour. There was associated clinical improvement. After a further single injection of 20 µg. of vitamin B₁₂ the Hb rose to 109% (16.1 g./100 ml.), the R.B.C. to 4,900,000/c.mm., P.C.V. 52%. A neutrophil leucocytosis of 18,000/c.mm. persisted: occasional myelocytes were found, but no cells more primitive than this.

Even with intramuscular doses of vitamin B₁₂ as small as 20 µg. every three weeks, the patient had become poly-

cythaemic again when tested two months later (see Table and Chart). A test meal at this stage—which upset the patient and caused collapse—showed histamine-fast achlorhydria; Hb was 118% (17.6 g./100 ml.), R.B.C. 7,250,000/c.mm., P.C.V. 63%. Macrocytosis had disappeared (M.C.V. 86 cubic microns) and the red cells were now hypochromic (M.C.H.C., 27.5%). White cells remained elevated at 23,600/c.mm., with 6% metamyelocytes and increase in polymorphs. Nuclear hypersegmentation of these latter cells had virtually disappeared. Platelets numbered 440,000/c.mm.

The patient was now feeling well, although she remained hypertensive and was rather light-headed at times. As she had faith in the injections which were being given to her, placebo injections of sterile saline were given as from July, 1951. As shown in the Chart, there was a fall in Hb, R.B.C., P.C.V., over the subsequent five months. Macrocytosis returned (M.C.V. 113 cubic microns), the leucocyte count was then 11,800/c.mm., and once again nuclear hypersegmentation of the polymorphs was obvious. Platelets were 350,000/c.mm. Reticulocytes, independent of therapy for five months, were 6%; bilirubin 0.75 mg./100 ml. and serum uric acid 5.3 mg./100 ml. Empirical liver-function tests were not noteworthy.

Three injections of 20 µg. of vitamin B₁₂ at fortnightly intervals were again followed by polycythaemia (P.C.V. 55%) and a leucocytosis of 25,000/c.mm. (myelocytes 2%, metamyelocytes 11%, polymorphs 68%, eosinophils 6%, lymphocytes 10%, monocytes 3%). Reduction of dosage of vitamin B₁₂ to 20 µg. every six weeks was insufficient to keep her Hb and P.C.V. within normal range and the leucocyte count fell to 11,000/c.mm. Injections of 20 µg. every month, however, kept values in fair balance between polycythaemia and anaemia during 1953, but on the same dose she became polycythaemic once again in June, 1954. She was becoming more uncomfortable owing to the greatly enlarged spleen extending into the pelvis. The liver was palpable 2 in. (5 cm.) below the right costal margin. All treatment was stopped for six months, and at the end of this time a repeat bone-marrow examination showed megaloblastic change—although once again there was difficulty in obtaining the specimen. A serum vitamin-B₁₂ estimation by the *Euglena gracilis* technique gave a result of 80 µµg./ml.

As shown in the Chart, adjustment of intramuscular vitamin B₁₂ to a dosage of 20 µg. every three to four weeks

	Before Therapy	After 140 µg. Vitamin B ₁₂ I.M. Spread Over 3 Months	After Blank Injections Over 5 Months
Hb	52% (7.7 g./100 ml.)	118% (17.6 g./100 ml.)	78% (11.4 g./100 ml.)
R.B.C.	1,830,000/c.mm.	7,250,000/c.mm.	3,080,000/c.mm.
P.C.V.	23%	63%	35%
M.C.V.	126 µ ³	86 µ ³	113 µ ³
W.B.C.	10,700/c.mm. occasional metamyelocytes	23,600/c.mm. some myelocytes and more frequent metamyelocytes	11,800/c.mm. occasional metamyelocytes
Platelets	Numerous	440,000/c.mm.	350,000/c.mm.

kept the Hb, red cells, and P.C.V. at a reasonable level during 1955 and 1956. Clinically, the gross splenomegaly rendered life most uncomfortable and she developed rectal prolapse, which was treated conservatively. During this time repeated leucocyte counts ranged from 15,000 to 20,000/c.mm. with polymorph increase—occasional myelocytes, metamyelocytes, and, rarely, normoblasts being found in blood films, but no white cells of more primitive type. The platelet count was 269,000/c.mm. She developed congestive cardiac failure, and treatment with mersalyl was given. The patient's condition deteriorated and she died on September 22, 1956.

Post-mortem Findings

Necropsy, four hours after death, was carried out by Dr. W. S. Killpack at Ealing. The body was wasted and showed oedema of the legs. The heart was dilated and the muscle showed brown atrophy. The right ventricle was hypertrophied. Coronary arteries and valves were healthy and there was little aortic atheroma. There were small effusions in both pleural sacs. The lungs were bulky and oedematous, the cut lower lobes being firm and brown, suggesting haemosiderosis.

Mouth, tongue, oesophagus, stomach, and intestines appeared healthy. No specimen of stomach was obtained from histology.

The liver, enlarged and firm, weighed 2,500 g., the cut surface being dark red. Gall-bladder and pancreas appeared healthy. The spleen was grossly enlarged and weighed 1,360 g. There were no splenic adhesions or infarcts. The cut surface was firm and dark red. The para-aortic lymph nodes showed slight enlargement. The bone-marrow of sternum and lumbar vertebrae was dark red and semi-diffuent. A long bone was not examined. No abnormality was found in brain meninges, suprarenals, or thyroid.

Histology.—Sections of the lungs showed evidence of bronchopneumonia with haemosiderin-laden macrophages in the alveolar spaces. Kidney sections showed signs of early glomerulonephritis. The liver showed a periportal exudate of lymphocytes but no other abnormality, apart from iron pigment in the Kupffer cells. Spleen sections showed evidence of autolysis and a small amount of iron pigment was found. On prolonged search an occasional cell resembling a megakaryocyte could be found but no evidence of extramedullary haemopoiesis. Malpighian bodies were more widely spaced than normal, but showed no structural abnormality. Sections of the sternal and vertebral bone-marrow showed a general marked hyperplasia with numerous prominent megakaryocytes and abundant normoblasts and granulocytes. Reticulin was present in moderate amounts. No recognizable megaloblasts could be found. Sections of the abdominal lymphatic glands showed nothing of pathological significance. Death was attributed to pulmonary oedema associated with polycythaemia vera.

Discussion

Red-cell counts above the normal range after liver therapy for pernicious anaemia were noted on occasion by Minot and Murphy (1926) and are also referred to by Koessler and Maurer (1927). However, as is stated by Ellman and Bowdler (1958), this appears only rarely as a mild non-progressive erythrocytosis, and it is widely held that generous doses of liver or vitamin B₁₂ do not raise blood-count values of normal subjects or patients with pernicious anaemia above physiological limits. Exceptionally, as occurred in our patient, vitamin-B₁₂ deficiency may develop in an individual with a propensity towards myeloproliferative disease, but the number of reported instances of this combination of blood disorders is small. The occurrence of polycythaemia vera and pernicious anaemia in the same patient has been reported by Birnie (1936), Ferrary

(1942), Galt *et al.* (1952), Skouby (1952), Hinz (1957), and Ellman and Bowdler (1958). Other likely instances of this phenomenon are reported by Avery (1930) and by Emile Weil and Boic (1932). The case described by Zarafonitis *et al.* (1957) showed the sequence of pernicious anaemia, polycythaemia, and acute myeloblastic leukaemia; and Baikie *et al.* (1958) report a case of pernicious anaemia, in which polycythaemia, leucocytosis, and thrombocythaemia developed after treatment with vitamin B₁₂—the platelet count rising to 1,750,000/c.mm. Wellington and Whitcomb (1960) describe a similar case. Croft (1956) studied a remarkable case in which myelosclerosis, polycythaemia, myeloid leukaemia, and vitamin-B₁₂ deficiency were all demonstrated at various times over a period of 13 years. Thus it is apparent that on rare occasions patients prone to a myeloproliferative syndrome may also develop vitamin-B₁₂ deficiency. The myeloproliferation may show emphasis on red-cell production or on granulocytes, platelets, or fibroblasts, and the degrees of such vary from time to time. It is masked or modified should there be a deficiency of vitamin B₁₂. Cases of uncomplicated polycythaemia vera show a normal or high serum vitamin-B₁₂ value (Mollin and Ross, 1955), and instances of myelosclerosis being associated with a rapid folic-acid clearance test have been reported by Chanarin *et al.* (1958). However, we know of no instance of a case of polycythaemia vera being complicated by megaloblastic anaemia proved to be due to folic-acid deficiency.

In the past it has been suggested that polycythaemia vera was the antithesis of pernicious anaemia and that this condition was due to an excessive secretion of gastric haemopoietic factor (see Wintrobe, 1956). However, the occurrence of both diseases in the one patient renders this an untenable concept; in the case reported by Ellman and Bowdler (1958) it was proved that added intrinsic factor was needed to increase absorption of orally administered radioactive vitamin B₁₂. As our patient was unwilling to be admitted to hospital it was impossible to use this technique or to carry out fat-balance studies. There was no obvious clinical evidence to suggest steatorrhoea; the serum vitamin-B₁₂ value was low after a period of no therapy; there was histamine-fast achlorhydria; and the haematological response to small doses of parenteral vitamin B₁₂ was dramatic. A diagnosis of pernicious anaemia developing into polycythaemia vera when treated with either liver or vitamin B₁₂ would appear almost indisputable, and this occurrence a coincidence. In 1951, in spite of recent injections of refined liver extract, megaloblastic anaemia was present, but the patient promptly showed haematological improvement with small intramuscular doses of vitamin B₁₂. It must be concluded that the batch of refined liver extract used at that time must have contained very little vitamin B₁₂ (see Adams and Timbury, 1959).

As was observed in several of the other cases which have been reported, our patient usually showed a higher granulocyte count and relatively higher platelet count values when treated with liver or vitamin B₁₂ than when in the stage of megaloblastic anaemia. In all reported cases splenomegaly has been a feature, although this was not always noted when the patients were first seen. Most cases have presented in the first place in an anaemic phase—polycythaemia developing subsequently. Wellington and Whitcomb (1960), however, reported the case of a patient with poly-

cythaemia vera who, after venesections, developed an anaemia resembling pernicious anaemia and who responded dramatically to liver therapy.

Wellington and Whitcomb (1960), however, reported the case of a patient with polycythaemia vera who, after venesections, developed an anaemia resembling pernicious anaemia and who responded dramatically to liver therapy. This patient died from pyelonephritis following prostatectomy. At necropsy normal red-blood-cell maturation was found in the bone marrow, as was myeloid metaplasia in the spleen. The case reported by Hinz (1957) showed cerebral haemorrhage and necrosis at necropsy, but otherwise the post-mortem findings were generally similar to those in the case we have described.

Summary

A case is described of a woman showing megaloblastic anaemia, who developed features of polycythaemia vera after treatment with liver or vitamin B₁₂. Anaemia recurred on discontinuation of treatment.

Clinical and haematological data over a period of 11 years are recorded. The patient was ultimately kept in satisfactory haematological balance with infrequent small doses of parenteral vitamin B₁₂.

The necropsy findings are summarized.

The occurrence of vitamin-B₁₂ deficiency developing in a patient prone to myeloproliferative disorder is regarded as a coincidence.

Thanks are due to Dr. W. S. Killpack, of the King Edward Memorial Hospital, Ealing, for the early laboratory reports and for performing the necropsy; to Miss C. M. Humphreys and Mr. Kenneth Rogers for technical assistance; and to Mr. T. F. Dee, who kindly prepared the photograph of the chart.

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Ageing, its Changes and its Promise is the report of the Tenth National Conference on the Care of the Elderly which was held in Blackpool in the spring of 1960. The conference covered the housing of the elderly; social policies: residential care for the physically and mentally frail; development in hospital care and hospital services; and a forward view of the next two years. There were discussion groups on visiting; clubs; preparation for retirement; training and recruitment; housing; and day centres and workshops. The report costs 5s. from the National Old People's Welfare Council, 26 Bedford Square, London W.C.1.

MEGALOBlastic ANAEMIA IN CONGENITAL SPHEROCYTOSIS

BY

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The purpose of this paper is to report two cases of congenital spherocytic haemolytic anaemia complicated by megaloblastic anaemia. The first case occurred in association with pregnancy. So far as we are aware, the second is unique in that, apart from the congenital spherocytosis, no cause for megaloblastic erythropoiesis could be discovered.

Case 1

The patient became pregnant at the age of 28 while in Germany. She first attended the antenatal clinic when 16 weeks pregnant and was found to have a haemoglobin level of 11.4 g./100 ml. At 22 weeks she began to complain of breathlessness and swelling of the ankles, and was noted to have a definite yellow tint to her skin. The dietary history was satisfactory and there was no abnormality of bowel habit.

She was admitted for investigation and the blood count showed: haemoglobin, 6.5 g./100 ml.; red cells, 1,790,000/c.mm.; white cells, 7,200/c.mm.; packed cell volume, 18%; mean corpuscular haemoglobin concentration, 36%; mean corpuscular volume, 101 cubic microns; reticulocytes, 7%. The red blood cells showed anisocytosis and macrocytosis with some polychromasia and an occasional normoblast. Some of the polymorph leucocytes were noted to be hypersegmented, but the platelets were normal. The bone-marrow showed erythroid hyperplasia and erythropoiesis was megaloblastic; leucopoiesis was normal. Free acid was present in the stomach. The serum bilirubin level was 1 mg./100 ml.

A diagnosis of megaloblastic anaemia of pregnancy was made and the patient was treated with folic acid in a dose of 10 mg. a day. A reticulocyte peak of 20% was reached on about the eighth day, but the haemoglobin level and red-cell count showed little change, and 19 days after her admission the haemoglobin was still 6.2 g./100 ml., and the red-cell count 1,420,000/c.mm. She was transfused with 3 pints (1.7 l.) of compatible blood, which raised her haemoglobin level to 9 g./100 ml. Despite continued folic acid therapy the haemoglobin level six weeks later had fallen to 6.5 g./100 ml. She was transfused with a further 4 pints (2.3 l.) of packed cells and three weeks later was delivered of a baby girl. Her haemoglobin level at this time was 10.4 g./100 ml. After the puerperium folic acid therapy was stopped, and five months after the delivery her haemoglobin level was 11.8 g./100 ml., red-cell count 4,770,000/c.mm., colour index 0.83, and white-cell count 9,200/c.mm.

Second Pregnancy.—The patient returned to this country and reported to the Haematology Clinic of the Royal Infirmary, Edinburgh, in the 14th week of her second pregnancy. She was complaining of severe lethargy, but was otherwise well and physical examination was negative.

Investigations.—The blood counts were as follows: haemoglobin, 9.9 g./100 ml.; red-cell count, 3,630,000/c.mm.; colour index, 0.92; white-cell count, 5,800/c.mm. The blood film showed some anisocytosis of the red blood cells, with the presence of both macrocytes and microspherocytes. The serum vitamin B₁₂ level was 80 µg./100