and was having haematemesis and melaena. The temperature rose intermittently to 102° F. (38.9° C.), and the pulse to 160.

As death appeared imminent from blood loss, 600 ml. of Group O blood was transfused, with marked clinical improvement, on July 23. The same day urethane, 1½ gr. (0.1 g.) thrice daily orally, was started.

The succeeding blood counts are shown in the accompanying Table and Chart. The changes in the blood picture were associ-

Record of Blood Counts

Date	Hb %	W.B.C. per c.mm.	Polymorphs, %	Metamyelocytes, %	Lymphoblasts, %	Lymphocytes and "Smudge" Cells, %	Mono-cytes, %
21/7/47	42	27,700	2	—	—	98	—
23/7/47	28	5,500	4	0.5	12	83.5	—
1/8/47	58	11,900	37	—	2.5	58.5	2
13/8/47	66	5,700	60	0.5	2	32	5.5
22/8/47	60	4,000	61	—	—	35	4
1/9/47	110	5,400	42	—	—	57	1
9/9/47	102	5,200	67.5	—	—	30	2.5
23/9/47	100	18,150	76.5	2.5	—	18	3

ated with slow but definite clinical changes from a moribund condition to that of a child behaving normally for its age. She was now afebrile, and although the cervical lymph glands could still be palpated they were considerably smaller. On Aug. 22 the leucocyte count had fallen to 4,000 per c.mm., with 61% polymorphs. Urethane was discontinued, a total of 51 gr. (3.25 g.) having been given over 34 days. A further transfusion of 600 ml. of Group O blood was given to combat leucopenia.

The apparent recovery of the child called for a reconsideration of the diagnosis. A Paul-Bunnell test for glandular fever proved negative. When sent home on Sept. 28 the child appeared to be quite well.

Unfortunately she was not seen as an out-patient because she lived 50 miles away, but in a reply to a letter her private doctor stated that "she went downhill rapidly and died on Oct. 27."

Discussion

Watkins (1947) states that in acute leukaemia of children little or no benefit is obtained from urethane, and that there is not sufficient change to warrant its use other than experimentally. Undoubtedly in this case blood transfusion was a life-saving measure at first, but the dramatic clinical and haematological improvement was far greater than could have been due to transfusion alone. Spontaneous remissions are not uncommon after transfusion in acute leukaemia of children, but a remission rarely lasts more than a week, and the disease is usually fatal within two months of the onset. In the present case the improvement after transfusion was maintained, and would appear to have been enhanced and prolonged by urethane treatment.

The prognosis was exceedingly grave in view of the child's age and of the fall in haemoglobin level to 28%. Paterson *et al.* (1946) attach importance to the initial haemoglobin level, for in three out of four of their cases of lymphatic leukaemia in which the haemoglobin was below 55% the patient died despite adequate blood transfusions.

It is unfortunate that no blood count was done when the final deterioration began, as it is now impossible to say whether this was due to an exacerbation of the original disease or to the toxic effect of urethane on the myeloid cells. This was foreshadowed in the blood count of Sept. 23, when of 18,150 white cells 2.5% were metamyelocytes and 76.5% were young band forms of polymorphonuclear leucocytes.

The use of urethane in this case profoundly modified the disease process, and therefore seems to have been justified.

Summary

A case is described of acute lymphoblastic leukaemia which responded initially to urethane therapy.

The palliative effect in this case would seem to justify further trials of urethane in acute leukaemia.

It is uncertain whether the fatal outcome in the case described was due to the disease or to the toxic effect of urethane on cells of the myeloid series.

Whilst it is probable that the large volume of blood given to this child contributed largely to the remission in the leukaemic state, it seems reasonable to suppose that the duration of this remission was related to the use of the drug.

My thanks are due to Dr. E. D. Scott and Dr. M. A. Leslie-Smith for permission to publish the case, and to Dr. S. Instone for her encouragement and advice.

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Medical Memoranda

Malignant Tumour of the Small Intestine

Malignant tumours of the small intestine are relatively uncommon, although reports on more than 500 cases are now available. Carcinoma is said to occur more often than sarcoma in the proportion of 6 to 4 (Mayo, 1940), carcinoma being found most commonly in the jejunum and sarcoma in the ileum. The age incidence in carcinoma is accepted to be higher than in sarcoma, and the sex incidence is in favour of males in the ratio of 2 to 1.

The following report of a case of adenocarcinoma of the ileum differs from those recorded by Rankin and Mayo (1930) in that no loss of weight occurred and no occult blood was found.

CASE REPORT

A publican aged 59 was admitted on Jan. 6, 1948, complaining of generalized abdominal pain and vomiting. His symptoms first began 11 months previously, and consisted of gripping pains and vomiting in attacks lasting 10 to 14 days. He suffered some half-dozen such attacks, with intervals of about a month's freedom, before consulting his doctor. Two days before admission he developed generalized abdominal pain with vomiting and for the first time he had absolute constipation.

During the 1914-18 war he contracted malaria and sandfly fever while serving in Palestine. Since that time he had always had two or three loose motions daily, but at no time did he notice blood or mucus in the stools. He suffered for many years from winter bronchitis.

On examination he was seen to be a well-built man with no obvious loss of weight. He was in considerable pain and was vomiting at fairly frequent intervals. The abdomen showed a generalized distension, with some diffuse tenderness on palpation, but no mass was felt. He had shifting dullness in the flanks, and loud borborygmi were heard. Rectal examination showed no abnormality. He was given an enema, which produced a faecal result in the form of a foul-smelling, pale, toothpaste-like ribbon. A further enema one hour later was returned unchanged, and a diagnosis of intestinal obstruction was made, the cause of which was not obvious.

Operation.—The abdomen was opened through a lower right paramedian incision and a quantity of straw-coloured fluid escaped from the peritoneal cavity. There was marked distension of the small intestine down to a point 30 in. (76 cm.) from the ileo-caecal valve.

A hard mass about the size of a tangerine orange involved the gut at this point and occluded the lumen of the bowel. A resection of 8 in. (20 cm.) of the bowel including the tumour, together with a wedge of mesentery containing enlarged glands, was carried out, continuity being restored by lateral anastomosis. The anastomosis was performed first, as the patient's condition had deteriorated, but as it subsequently improved resection became possible. The abdomen was closed and the patient returned to bed with an intravenous glucose-saline drip and a gastric aspiration in operation.

Pathology of the Tumour-bearing Intestine.—"The loop of small intestine is involved by a stenosing carcinoma of scirrhus appearance. The tumour is about 1 in. (2.5 cm.) in length and encircles the whole wall. No gross ulceration of the mucosa is observed and no polypi are seen. Careful search fails to reveal any associated lymph glands involved by metastases. Histological examination showed an adenocarcinoma which is deeply infiltrating the bowel wall, with an accompanying scirrhus reaction. The lesion is not of carcinoid origin, the tumour consisting of acini of varying size, showing irregularity in cell type and nuclear configuration."

Convalescence was lengthened by a post-operative pneumonia but was otherwise uneventful, and the patient was discharged on Jan. 23. When last seen two months later he was well and leading a normal life.

I am indebted to Mr. G. F. Mitchell, under whose care the patient was admitted, for permission to publish this case, and to Dr. R. L. Bishton and Dr. R. C. Hill for the pathological report on the specimen.

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A Case of Inherited Spina Bifida

The aetiology of foetal deformity is still uncertain. Such diverse factors as heredity, multiparity, diabetes, placenta praevia, and maternal rubella have variously been incriminated. The inheritance of minor deformities along Mendelian lines is not uncommon; while the occurrence of a major deformity in siblings increases the chance of such deformity reappearing, though Gibberd (1947) considers this to be only a fourfold increase above the normal probability. Direct inheritance of major deformity from the mother is naturally uncommon, and the following example is worthy of record.

CASE HISTORY

A primigravida aged 21, in her 37th week of gestation, was admitted to hospital on May 7, 1947, with diagnosis of toxæmia and disproportion. The toxæmia proved to be minimal, but examination disclosed a fluctuant swelling the size of a coconut in the midline of the lumbar region, extending from the second lumbar to the first sacral vertebra. There was exaggeration of the lumbar lordosis, but no scoliosis; the swelling was compressible and had a well-marked cough impulse. Both feet showed pes cavus (right more than left). There was wasting of the thigh and calf muscles on the right, and $\frac{1}{4}$ in. (1.9 cm.) shortening of the right lower extremity. A diagnosis was made of meningocele, and the spinal deformity was confirmed by radiography at a later date.

Obstetrically, the case was one of brim disproportion, with a floating head and some slight degree of hydramnios. X-ray examinations were intended, but labour started spontaneously the night after admission, and vaginal examination soon showed that the disproportion was caused by a hydrocephalic head; this was perforated at half dilatation of the cervix, and a female foetus weighing 6 lb. 1 oz. (2.75 kg.) was delivered uneventfully. It showed a moderate hydrocephalus, with a gross dorsi-lumbar spina bifida of the open myelocele type.

COMMENT

No reference to the inheritance of spina bifida has been found in the available literature, and Ford (1946) does not include it in an exhaustive list of inherited disorders. Despite the difference in degree of the foetal and maternal condition it seems reasonable to suppose that some genetic factor had been transmitted and that further pregnancies would run a very considerable risk of being similarly affected. It is impossible to assess the relative importance of heredity and environment in relation to the developmental errors of the young embryo; the majority of gross abnormalities are lost as abortions, while of those which are born alive only a very small percentage reach reproductive life and have the opportunity of passing on to the next generation whatever genetic

factor may be involved. There is a growing tendency to find environmental factors such as dietetic deficiency (Warkany, 1944), rhesus iso-immunization (Wiener, 1946), and maternal rubella (Gregg, 1941) to account for foetal deformity, but it seems probable that many cases of gross defect are genetic in origin.

My thanks are due to Mr. Samuel Davidson for permission to publish this note.

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Occurrence of Convulsions During Treatment with Calciferol

The value of high dosage of calciferol in the treatment of lupus is well established, and in view of the success of the method its use is likely to be further extended until the therapeutic effect has been evaluated in other tuberculous infections. Calciferol has been recognized as a potentially toxic drug, and anorexia, nausea, vomiting, and polyuria are well known to follow its use. So far, however, I have seen no record of the occurrence of convulsions after calciferol and therefore consider that the following case should be put on record.

CASE HISTORY

A boy aged 10 was admitted to the Surrey County Hospital with a history of malaise for one month and an unexplained pyrexia of one week's duration. On admission the temperature was 101° F. (38.3° C.). The abdomen was full and slight tenderness was present on deep palpation in the R.I.F. No definite mass was felt, but on bimanual examination with a finger in the rectum a large gland was discovered low in the right side of the abdomen. White cells numbered 7,500 per c.mm. (P. 70%, L. 20%, M. 9%, E. 1%). The Mantoux test was positive 1 in 10,000. X-ray examination of the chest revealed nothing abnormal. A diagnosis of tuberculous mesenteric adenitis was made, and treatment with calciferol 50,000 units twice daily was started. The temperature settled rapidly, with an occasional evening rise to 99° F. (37.2° C.). On Nov. 21, 1947, the boy was allowed home at his parents' request, to rest in bed and continue calciferol, 50,000 units twice daily. There had been no symptoms of intolerance during his stay in hospital, but the parents were warned of the possible occurrence of toxic symptoms. On Dec. 15 he was readmitted to hospital suffering from generalized convulsions. It appeared that, in addition to the 100,000 units of calciferol prescribed, an ampoule of strogyl containing 600,000 units had been given by mouth a week previously. Soon afterwards nausea, occasional vomiting, and obstinate constipation had developed; he became increasingly drowsy and could not be roused for some hours before the first convulsion began.

On admission very frequent generalized convulsions were occurring; no focal signs were present on examination of the central nervous system except for an extensor plantar response on the right side, which became flexor 48 hours later. Treatment included 0.25 g. of soluble hexobarbitone intravenously, followed by $1\frac{1}{2}$ gr. (0.1 g.) of soluble phenobarbitone intramuscularly two hours later. Apart from a further slight convulsion and slight vomiting recovery was uninterrupted, being complete in 48 hours. The blood calcium was 18 mg. per 100 ml. on the day of admission, falling to 14.8 on Dec. 22 and 12.9 on Dec. 29. The urinary output was well maintained and the urine contained no albumin or casts. The blood urea was not estimated.

There was no family history of fits, nor had the boy suffered previously from convulsions.

COMMENT

In view of the fact that increasing concentrations of calcium are known to decrease the frequency of impulses discharged from nerve cells stimulated with acetylcholine it seems likely that the fits were due to a direct toxic effect of calciferol rather than to the associated hypercalcaemia. Overdosage with calciferol is known to produce a rise in the blood urea, but in the absence of oliguria or albuminuria it seems unlikely that the convulsions were uraemic in origin.

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