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 scirrhous 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 scirrhous 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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REFERENCES

Mayo, C. W. (1940). *Coll. Pap. Mayo Clin.*, **32**, 312.
Rankin, F. W., and Mayo, C. (1930). *Surg. Gynec. Obstet.*, **50**, 939.

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 toxaemia and disproportion. The toxaemia 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 ¾ in. (1.9 cm.) shortening of the right lower extremity. A diagnosis was made of meningomyelocele, 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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REFERENCES

Ford, E. B. (1946). *Genetics for Medical Students.* London.
Gibberd, G. F. (1947). *Short Textbook of Midwifery.* London.
Gregg, N. M. (1941). *Trans. ophthal. Soc. Aust.*, **3**, 35.
Warkany, J. (1944). *J. Pediat.*, **25**, 476.
Wiener, A. S. (1946). *Amer. J. clin. Path.*, **16**, 319.

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 sterogyl 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 1½ 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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