the present patient the daily output of potassium in the faeces represented one-half of the total sensible loss of potassium from the body.

Summary

In a patient with primary hyperaldosteronism the rates of net transport and of unidirectional fluxes of sodium, potassium, and water in the intact colon were measured before and after removal of the adrenocortical tumour, by perfusing the colon with an isotopically labelled test solution introduced into the caecum through a tube passed by mouth. The results in this patient were compared with those in eight control subjects. Before removal of the aldosterone-producing tumour the colon of the patient secreted potassium at four to five times the rate in control subjects. The unidirectional flux of potassium into the colonic lumen was greatly enhanced and the daily loss of potassium in the faeces increased. The rates of potassium transport returned to within the range observed in control subjects after the removal of the tumour.

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Preliminary Communications

Direct Evidence for Presence of Ph^1 Chromosome in Erythroid Cells

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Strong but indirect evidence (Tough *et al.*, 1963; Trujillo and Ohno, 1963; Whang *et al.*, 1963) has suggested that in chronic granulocytic leukaemia erythroid as well as myeloid precursors contain the Ph^1 chromosome. Whang *et al.* (1963), for instance, found a 90–100% incidence of the Ph^1 chromosome in bone marrow cells of 13 patients in drug-induced clinical remission known to have a moderately high proportion of dividing normoblasts. Recently, Clein and Flemans (1966) obtained more direct evidence by combining standard cytogenetic techniques with Perls's Prussian blue stain for iron. They demonstrated siderotic granules in the cytoplasm surrounding some Ph^1-positive metaphase plates derived from a patient with blastic crisis of chronic granulocytic leukaemia in whose marrow a prominent sideroblastic element was also present.

To obtain further direct evidence for the occurrence of the Ph^1 chromosome in erythroblasts, a bone marrow aspirate from a patient with chronic granulocytic leukaemia in drug-induced clinical remission was cultured with ^{59}Fe and ^{55}Fe (Suit *et al.*, 1957) and the Ph^1 chromosome was found in all erythroid metaphase plates examined.

Materials and Methods

The patient was a 56-year-old man who presented with chronic granulocytic leukaemia in January 1967. He was subsequently treated with busulphan, and was in good clinical remission at the time of this study in July 1967, when a sternal bone marrow aspirate contained 178 normoblasts per 1,000 nucleated cells as determined by May–Grünwald–Giemsa staining.

Materials.—The culture medium consisted of 12 ml. of Medium 199, an additional 0.1 ml. of Solution D-G-P for Medium 199 (Commonwealth Serum Laboratories, Melbourne); 0.01 ml. of heparin, 0.02 μg. of demecolcine, and 8 ml. of AB serum. The isotopes used were ^{59}Fe and ^{55}Fe, obtained as ferric chloride in 0.01 N HCl from the Radiochemical Centre, Amersham. Kodak AR-10 stripping film was used for the autoradiography.

Methods.—The bone marrow aspirate was added to the culture medium and the resulting cell suspension divided into 10-ml. aliquots, to which were added 200 μCi of ^{59}Fe or 200 μCi of ^{55}Fe. Cultures were incubated at 37° C. for 12 hours, which was found to be the optimal time for the uptake of isotope by actively dividing normoblasts. Both samples were then treated as follows. The cells were washed with Hanks's saline, and smears made to determine ^{59}Fe and ^{55}Fe uptake by the normoblasts. The remainder of each sample was then incubated for 10 minutes at 37° C. with 0.075M KCl hypotonic solution, after which the cells were fixed with a freshly prepared formaldehyde–acetic-acid–methanol fixative for 10 minutes. They were then resuspended in fresh fixative and chromosome preparations were made immediately by the "flaming" technique.

Autoradiograms of the smear and chromosome preparations were exposed for 21 days at 4° C., and developed for five minutes at 20° C. with Kodak D-19 developer. The smears were stained with May–Grünwald–Giemsa, and the chromo-

some preparations with Giemsa. After photography of the labelled metaphase plates with the focus on the labelling pattern the silver grains were bleached with a 10% solution of potassium ferricyanide and the metaphases rephotographed.

RESULTS

Examination of the smears showed that only the erythroid cell series was labelled with isotope under the conditions of the experiment. There was no labelling of any cells of the myeloid, lymphoid, or megakaryocytic cell types as determined by careful examination of six smears. In order to preserve cytoplasmic labelling in the chromosome preparations the fixation time was kept short. This resulted in less than optimal fixation, and a relatively high proportion of metaphase plates were unsuitable for detailed study. However, a large number of both labelled and unlabelled metaphases were well spread, and all contained the Ph^1 chromosome. This chromosome was clearly determined in 92 labelled metaphase plates.

Examples of labelled metaphases before and after grain bleaching are given in Figs. 1 and 2.

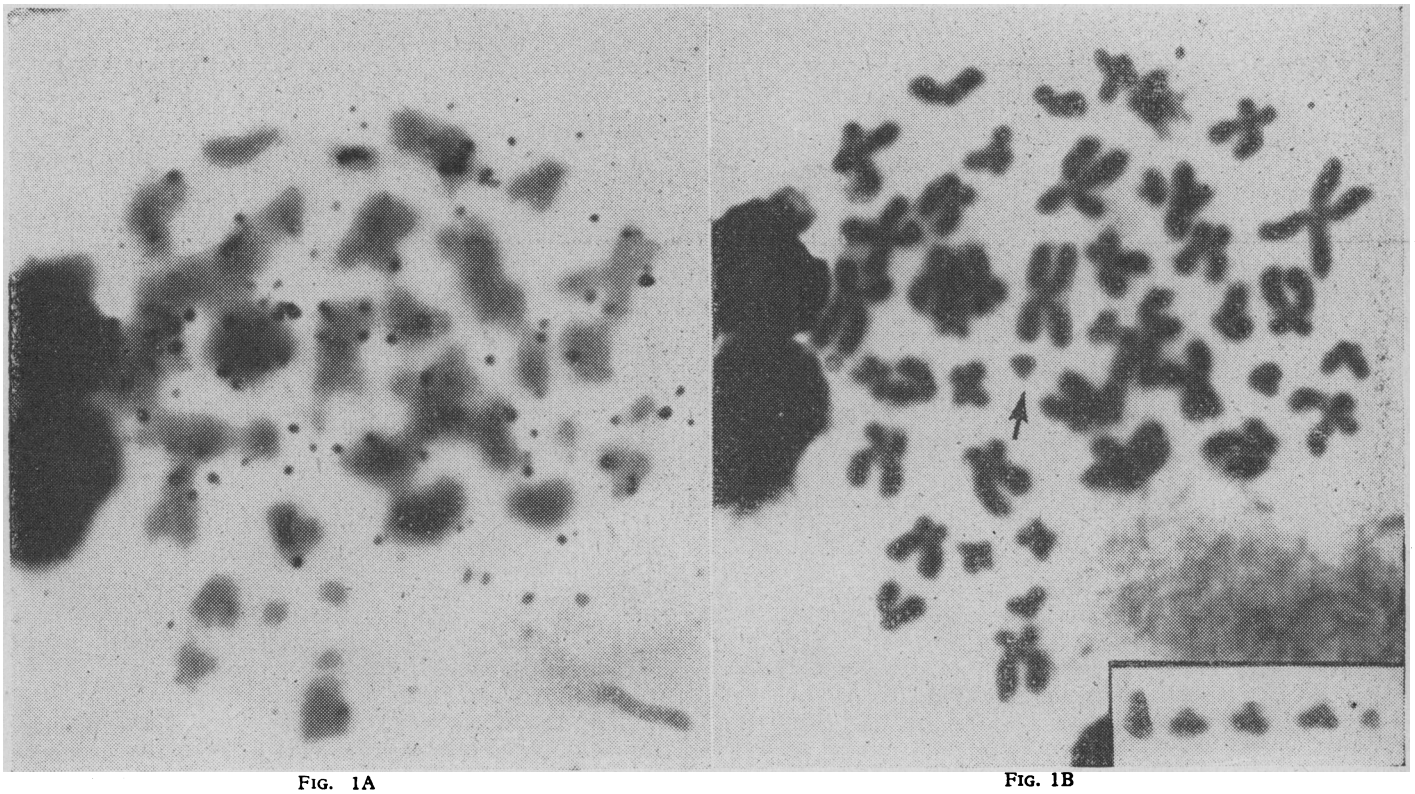

FIG. 1A FIG. 1B

FIG. 1.—A, Metaphase labelled with ^{59}Fe, showing the labelling pattern. B, Bleached metaphase. The Ph^1 chromosome is indicated by an arrow. Inset: chromosomes of the G group, and the Y chromosome.

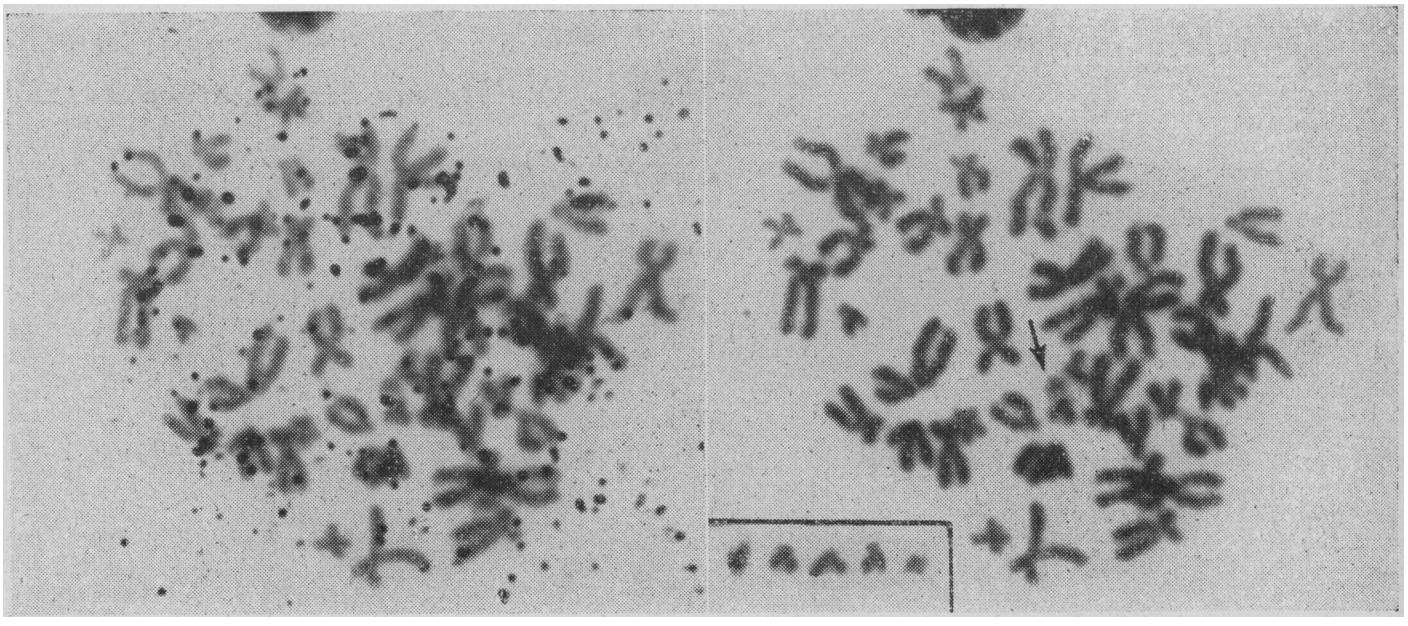

FIG. 2A FIG. 2B

FIG. 2.—A, Metaphase labelled with ^{59}Fe, showing the labelling pattern. B, Bleached metaphase. The Ph^1 chromosome is indicated by an arrow. Inset: chromosomes of the G group, and the Y chromosome.

DISCUSSION

The above work is believed to present direct evidence that the Ph[1] chromosome is present in the erythroid as well as in the myeloid cell series. Several workers (Tough *et al.*, 1963 ; Trujillo and Ohno, 1963 ; Whang *et al.*, 1963) have, on the basis of good circumstantial evidence, postulated the probable involvement of megakaryoblasts.

It therefore seems reasonable to suggest, on the basis of evidence to date, that the erythroid, myeloid, and possibly the megakaryocytic cell series are derived from a stem cell common to all three types.

Two further patients with chronic granulocytic leukaemia are at present being studied, and a more detailed report is in preparation.

SUMMARY

Chromosome preparations of the bone marrow of a patient with chronic granulocytic leukaemia were made after the cells had been cultured with ^{59}Fe or ^{55}Fe. The autoradiograms of these preparations give direct evidence that the Ph[1] chromosome is present in the erythroid cell series.

JOAN M. RASTRICK, B.SC.
P. H. FITZGERALD, PH.D.
F. W. GUNZ, M.D., PH.D.

Cytogenetics Unit,
Cancer Society of New Zealand,
Christchurch Hospital,
Christchurch, New Zealand.

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Medical Memoranda

Partial Hydronephrosis Due to Pressure from Normal Renal Arteries

[WITH SPECIAL PLATE BETWEEN PAGES 82 AND 83]

Brit. med. J., 1968, **1**, 98–99

The appearance of arterial impressions in a pyelogram is now well known (Kreel and Pyle, 1962). It is less widely recognized that normal renal arteries may, rarely, compress the infundibulum and cause a localized hydronephrosis.

CASE REPORT

A 3-year-old boy was admitted to hospital under the care of Mr. David Levi with a history of intermittent haematuria for two and a half months. There had been no other complaints and no abnormalities were discovered on physical examination. Numerous urine samples were normal microscopically and on culture, both for pyogens and tubercle bacilli. Examination of the blood was also normal.

An intravenous pyelogram on 8 October 1965 showed early but definite dilatation of the upper calicine group in the left kidney ; no other abnormalities were demonstrated in the urinary tract (Special Plate, Fig. 1). A left selective renal arteriogram was then carried out. Under general anaesthesia a small nylon catheter with a small preformed curve was introduced through the right femoral artery, a modified Seldinger technique being used. The catheter passed quite readily into the left renal artery, and serial angiograms were obtained after the injection of 1.5 ml. of 45% Hypaque (Special Plate, Fig. 2). Comparison of the angiogram with the pyelogram showed that a filling defect in the infundibulum corresponded accurately with the point at which it was crossed by a curving artery, close to a second arterial branch. It was thought that the infundibulum was trapped between the two branches of the renal artery (Special Plate, Fig. 3).

On 25 January 1966 the left kidney was explored by Mr. Levi. The infundibulum in the upper pole was identified and was found to have been caught in a fork in the artery to the upper pole, one branch passing in front and the other behind the infundibulum. There was also a little fibrous tissue in the area. This and the posterior branch were divided. There was no evidence of ischaemia following division of the artery.

The patient made an uninterrupted recovery after the operation and was discharged on the twelfth day. He has remained well since then, and there has been no recurrence of haematuria. When last seen, 10 months after the operation, there was no evidence of hypertension and an intravenous pyelogram was normal (Special Plate, Fig. 4).

COMMENT

There has been considerable controversy regarding the role of abnormal renal arteries in the pathogenesis of hydronephrosis. It is probable that such arteries with their accompanying fibrous tissue play a significant part in producing a hydronephrosis in some patients, but not in others. This would account for the still unresolved disagreement on this point. We had not encountered, at that time, a partial hydronephrosis caused by normal intrarenal arterial branches. Since then a similar series has been described by Fraley (1966), who records the case histories of four adult patients with right-sided loin pain and pyelographic appearances similar to those in our patient. All were cured by relief of the infundibular obstruction or partial nephrectomy. In two of these patients the obstruction was caused by pressure exerted by an artery and a vein. We have recently seen another boy with a partial hydronephrosis which corresponded accurately in its distribution with an aberrant renal artery arising from the aorta ; at operation, however, obstruction was found to have been due to a vein accompanying the artery and not to the artery itself. Another case of a partial right hydronephrosis due to normal renal arteries is described by Frimann-Dahl (1966). Arterial impressions appear particularly common on the right upper group of calices, and this presumably correlates with the fact that five out of the six cases mentioned here have been on the right, the present case being the sole exception.

The evidence for an arterial cause of this patient's hydronephrosis would seem very strong, though the fibrous tissue encountered round the artery may also have played a part. Why the child developed haematuria as the presenting symptom is, however, far from clear. There was never any evidence of a urinary infection as a possible explanation. A further, as yet unresolved, problem is the possibility of the later development of ischaemic hypertension. It seems reasonable to assume that if the kidney had been made ischaemic to the right degree, hypertension would have developed immediately. The child is being followed with this in mind.

Partial hydronephrosis could be caused by stenosis of the infundibulum due to infection (particularly tuberculous) or as