CASE REPORTS

Features of a successful therapeutic fast of 382 days' duration

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Summary
A 27-year-old male patient fasted under supervision for 382 days and has subsequently maintained his normal weight. Blood glucose concentrations around 30 mg/100 ml were recorded consistently during the last 8 months, although the patient was ambulant and attending as an out-patient. Responses to glucose and tolbutamide tolerance tests remained normal. The hyperglycaemic response to glucagon was reduced and latterly absent, but promptly returned to normal during carbohydrate refeeding. After an initial decrease was corrected, plasma potassium levels remained normal without supplementation. A temporary period of hypercalcaemia occurred towards the end of the fast. Decreased plasma magnesium concentrations were a consistent feature from the first month onwards. After 100 days of fasting there was a marked and persistent increase in the excretion of urinary cations and inorganic phosphate, which until then had been minimal. These increases may be due to dissolution of excessive soft tissue and skeletal mass. Prolonged fasting in this patient had no ill-effects.

Introduction
Current opinion on fasting therapy for the obese is perhaps best summarized by the view that fasting for relatively short periods is beneficial, whereas longer term fasting (i.e. longer than 40 days) has an element of risk attached (Lawlor & Wells, 1971). It is generally agreed that the long-term outlook for the achievement and maintenance of ideal body weight is poor (MacCuish, Munro & Duncan, 1968; Lawlor & Wells 1971) unless a weight close to the ideal is achieved during the supervised phase (Munro *et al.*, 1970), a process which in the majority of cases would involve a prolonged rather than a short-term fast.

Several years ago a grossly obese young man presented himself for treatment. Initially there was no intention of making his fast a protracted one, but

since he adapted so well and was eager to reach his 'ideal' weight, his fast was continued into what is presently the longest recorded fast (*Guinness Book of Records*, 1971). This report describes some of the features which emerged during the 382 days of his fast.

Methods
Patient treatment
Patient A.B. aged 27 years, weighed on admission 456 lb (207 kg). During the 382 days of his fast, vitamin supplements were given daily as 'Multivite' (BDH), vitamin C and yeast for the first 10 months and as 'Paladac' (Parke Davis), for the last 3 months. Non-caloric fluids were allowed *ad libitum*. From Day 93 to Day 162 only, he was given potassium supplements (two effervescent potassium tablets BPC supplying 13 mEq daily) and from Day 345 to Day 355 only he was given sodium supplements (2·5 g sodium chloride daily). No other drug treatment was given. Initially, the patient was treated in hospital but for the greater part of the time he was allowed home, attending regularly as an out-patient for check-up. Twenty-four hour urine collections were made periodically throughout the fast. His mean urinary creatinine excretion was 1541 mg/24 hr, with a ±25% variation, which indicates reasonable collections. No faecal collections were made, but evacuation was in fact infrequent, there being 37–48 days between stools latterly. Venous blood specimens were obtained approximately once a fortnight and tests of carbohydrate metabolism (intravenous glucose tolerance, tolbutamide and glucagon tests) (Marks & Rose, 1965; Oakley, Pyke & Taylor, 1968) were undertaken on nine occasions during the fast. All three tests were carried out consecutively on the same day, following a standard procedure whereby the tolbutamide was given 1½ hr after the glucose infusion, when blood glucose concentrations had returned to normal, and the glucagon was given 1½ hr after the tolbutamide.

Estimations

Plasma and urinary electrolytes, phosphate, uric acid and creatinine were measured by routine methods (available on request). Blood glucose was estimated by glucose oxidase. Magnesium in both plasma and urine was measured by atomic absorption spectrophotometry (Fleming & Stewart, 1966).

Results

Body weight loss

During the 382 days of the fast, the patient's weight decreased from 456 to 180 lb. Five years after undertaking the fast, Mr A.B.'s weight remains around 196 lb.

Blood glucose levels

Blood glucose concentrations decreased systematically (Fig. 1) and remained around 30 mg/100 ml from the fourth month onwards. Values below 20 mg/100 ml were occasionally seen towards the end of the fast. Despite the hypoglycaemia the patient remained symptom-free, felt well and walked about normally.

Tests of glucose tolerance showed unimpaired capacity for glucose uptake (Table 1). Apart from Day 7, the glucose assimilation coefficient remained between 1·0 and 1·6 (low normal) throughout the fast, one low value of 0·8 being associated with an unduly slow infusion of the dextrose test load. Likewise, apart from Day 7, the 10 min increment in blood glucose remained between 62 and 75 mg/100 ml until refeeding, when it became greater than 100 mg/100 ml. Responses to the intravenous tolbutamide test also remained normal until Day 355,
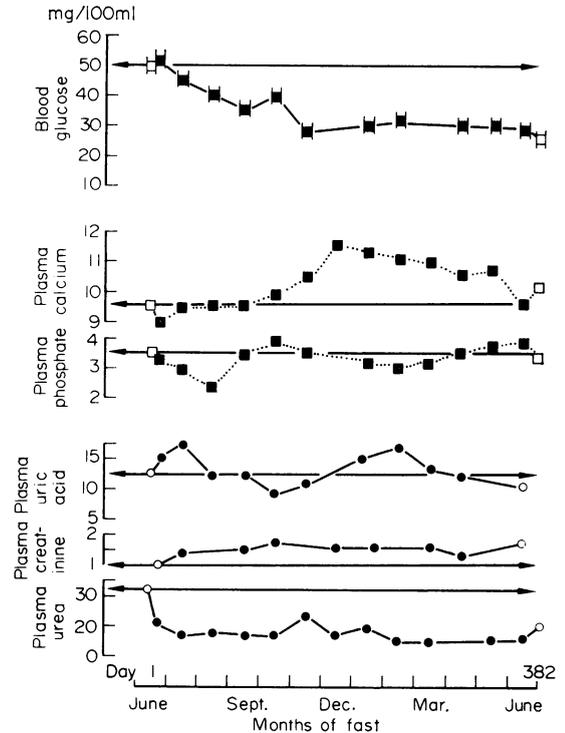

FIG. 1. Blood glucose and plasma concentration changes. Mean monthly concentrations during fasting. Open symbols, first and last day.

when unexpectedly there was no demonstrable decrease in blood glucose, this coinciding with a

TABLE 1. Tests of carbohydrate metabolism

| Day of fast | Intravenous glucose tolerance test | | | | Tolbutamide test | | Glucagon test |
	Pretest blood glucose (mg/100 ml)	Peak* blood glucose (mg/100 ml)	Peak increment (mg/100 ml)	Glucose assimilation coeff.	Blood glucose 20 min after tolbutamide (mg/100 ml)	% of control value	Increment in blood glucose 20 min after glucagon (mg/100 ml)
1	50	112	62	1·2	37	80	—
7	41	90	49	0·9	27	68	10†
30	35	110	75	1·2	35	73	8
68	34	—	—	1·6	32	73	10
101	35	110	75	1·1	38	75	8
269	37	98‡	61‡	0·8	40	69	3
355	17	90	73	1·5	50§	111§	0
R7*	41	155	114	1·2	28	62	45
R55†	25	135	110	1·0	40	73	—
Normal	—	—	—	1·0–2·5 ¶	—	<80	30–90

* Sample taken 8–10 min after beginning injection; time taken for injection 4–6 min.
† Test done Day 10 not Day 7.
‡ Sample taken 16 min after beginning injection; time taken for injection 11 min.
§ Patient fainted, recovered after ∼3 min.
¶ Marks & Rose (1965).
R7, Day 7 of refeeding, on 1000 kcal carbohydrate only.
R55, Day 55 of refeeding, on 1000 kcal diet.

TABLE 2. Mean monthly plasma electrolyte concentrations, compared with the values on the first and last day of the fast

Date	Days of fast	Na (mEq/1)	K (mEq/1)	Cl (mEq/1)	HCO$_3$ (mEq/1)
1965					
June	1 = beginning	144	4·3	103	30·0
June	2–17	141	4·1	98*	28·2*
July	18–48	143	3·7	96*	30·2*
Aug.	49–79	143	3·4	91*	30·8†
Sept	80–109	141	3·1	96	28·8
Oct.‡	110–140	145	4·4	95	24·5
Nov.‡	141–170	142	4·0	111	22·0
Dec.	171–201	142	4·3	100	24·8
1966					
Jan.	202–232	141	3·9	98	24·5
Feb.	233–260	141	4·0	105	23·0
Mar.	261–291	141	5·0	101	22·0
Apr.	292–321	—	—	—	—
May	322–352	143	3·9	103	23·3
June	353–381	143	3·6	97	22·5
30 June	382 = end	142	3·4	97	24·5

* Values decreasing throughout month.
† Values increasing throughout month.
‡ On potassium supplements for 10 weeks.

brief episode of fainting immediately after the intravenous injection.

Responses to intravenous glucagon were reduced from the early days of the fast onwards and by Day 269 and subsequently, there was no responsive increment in blood glucose at all. A normal increase was seen by Day 7 of the refeeding period, at which time the only sustenance had been entirely carbohydrate in nature (1000 kcal daily as modified liquid glucose BPC, 'Hycal'—Beechams).

Plasma concentrations (Table 2 and Fig. 1)

Over the first 4 months there was a systematic decrease in plasma potassium concentrations but the small daily supplement of 13 mEq potassium, which was then given, increased the potassium level. After a further 10 weeks, no more potassium was given, and plasma potassium remained normal. During refeeding the lowest concentration seen was 2·9 mEq/l on Day 6. For 6 months towards the end of the fast there was a limited period of hypercalcaemia (10·8–11·6 mEq/l) but this remitted spontaneously during the final month.

As expected, plasma urea decreased during the first 2 weeks of fasting and thereafter remained steady between 15 and 20 mg/100 ml. Plasma uric acid, which was high at 12·6 mg/100 ml prior to the fast (possibly reflecting pre-admission attempts at fasting), remained generally at this level throughout with one or two brief increases to 17 mg/100 ml which were not sustained. Symptomatic gout did not occur. Cholesterol concentrations remained around 230 mg/ 100 ml until 300 days of fasting, but increased to 370 mg/100 ml during refeeding.
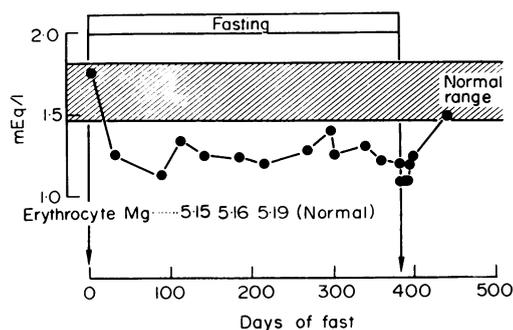

FIG. 2. Plasma magnesium concentrations.

Plasma magnesium levels

Plasma magnesium concentrations (Fig. 2) decreased over the first few weeks and thereafter stabilized between 1·2 and 1·4 mEq/l. There was a further temporary decrease to around 1·1 mEq/l during the first few days of refeeding (carbohydrate only) followed by an increase back to normal levels over the subsequent 6 weeks. Erythrocyte magnesium concentrations remained unchanged and normal.

Urinary electrolyte excretion (Fig. 3)

The excretion of sodium, potassium, calcium and inorganic phosphate decreased to low levels throughout the first 100 days, but thereafter the excretion of all four urinary constituents, as well as of magnesium, began to increase. During the subsequent 200 days sodium excretion, previously between 2 and 20 mEq daily, reached over 80 mEq/24 hr, potassium excretion increased to 30–40 mEq daily and calcium
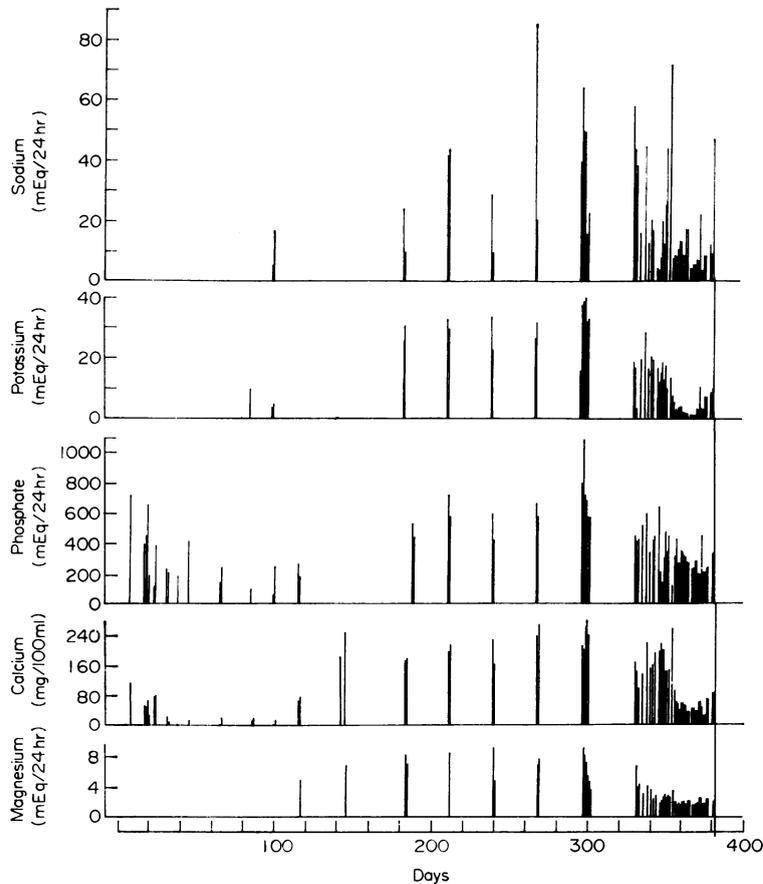
FIG. 3. Increases in urinary excretion patterns during fasting.

excretion increased from 10–30 mg/24 hr to 250–280 mg/24 hr. Magnesium excretion (which was not measured during the first 100 days) reached 10 mEq/24 hr between Days 200–300. Phosphate excretion, which had decreased to under 200 mg/24 hr, also increased to around 800 mg/24 hr, even exceeding 1000 mg/24 hr on occasion. Peak excretions of all these constituents were seen around Day 300, after which there was a marginal decrease, but excretion remained high. There was no indication in the characteristics of the weight-loss response, which was steady and sustained, or in the persistently low blood glucose or urea levels, which would have suggested that he had been surreptitiously eating. Plasma ketones were detectable throughout this period. At no time throughout his fast were any of the reactive changes seen which occurred during refeeding, with a small carbohydrate intake.

Discussion

Body weight loss and length of fast

The amount of weight lost and the rate of loss were not strikingly different from that of an earlier patient (Stewart, Fleming & Robertson, 1966) who reduced his weight from 432 to 235 lb during 350 days of intermittent starvation.

The amount of weight lost by Mr A.B. was 276 lb, with an average rate of loss of 0·72 lb/day, comparing favourably with the rates of weight loss in other long-term fasts (> 200 days) which have ranged from 0·41 lb/day (Thomson, Runcie & Miller, 1966) to 0·67 lb/day (Runcie & Thomson, 1970). The nearest comparable fasts are one of 256 days (Collison, 1967, 1971), those of 249 and 236 days (Thomson *et al.*, 1966) and two of 210 days (Garnett *et al.*, 1969; Runcie & Thomson, 1970).

There have been reports of five fatalities coinciding with the treatment of obesity by total starvation (Cubberley, Polster & Schulman, 1965; Spencer, 1968; Garnett *et al.*, 1969; Runcie & Thomson, 1970). One was attributed to lactic acidosis during the refeeding period following a 3 week fast (Cubberley *et al.*, 1965). Two were considered to be due to ventricular failure, occurring during the fast, at 3 and 8 weeks respectively in patients who had shown evidence of heart failure before beginning the fast (Spencer, 1968). One patient (Runcie & Thomson, 1970) died on the thirteenth day of his fast from small bowel obstruction. Only one of the five 'fasting' deaths has been associated with a fast of more than 200 days' duration. It occurred during the refeeding period after a fast of 210 days in an apparently well young woman (Garnett *et al.*, 1969). Following this particular report doubt has been cast on the safety of the treatment of obesity by total fasting (Garnett *et al.*, 1969; Rooth & Carlström, 1970). However, the allopurinol which had been given may have had unfavourable effects on nucleotide metabolism (Stewart & Fleming, 1969).

Blood glucose changes

Low blood glucose values, without symptoms of hypoglycaemia, have been reported by others during therapeutic fasting or starvation (Chakrabarty, 1948; Drenick, Hunt & Swenseid, 1964; Jackson, McKiddie & Buchanan, 1968; Wildenhoff, Dalsager & Schwartz Sørenson, 1969). Others (Drenick *et al.*, 1964; Spencer, 1968) have shown no blood glucose decrease at all. Some investigators (Rapoport, From & Husdan, 1965) have found only a temporary decrease during the first week.

Decreased (Jackson *et al.*, 1968; Runcie & Thomson, 1970), increased (Schless & Duncan, 1966) and normal (Anderson, Herman & Newcomer, 1969) glucose tolerance have all been described as occurring during fasting. The categorization of obese patients into 'diabetic' and 'non-diabetic' groups, even although none was overtly diabetic prior to fasting, is according to some a prerequisite in the evaluation of glucose tolerance during fasting, with the 'non-diabetic' showing decreased tolerance and the 'diabetic' showing unaltered or improved tolerance (Schless & Duncan, 1966; Jackson *et al.*, 1968). 'Improvement' has been described on the basis of lower blood glucose levels at all comparable times, even although the rate of decrease following the peak blood glucose was actually slower (Anderson *et al.*, 1969) or unchanged (Jackson *et al.*, 1969). When assessed by the rate of decrease in glucose concentrations following intravenous glucose administration as recommended, our 'non-diabetic' patient showed no systematic change in glucose tolerance throughout the fast or afterwards.

It would appear that there is considerable individual variation, both in the level of blood glucose maintained during prolonged fasting and in the response to a glucose load. However, it is obvious that even patients such as ours, who do become markedly hypoglycaemic, can tolerate their fasts as well as those who maintain more normal sugar levels.

Plasma electrolyte changes

Progressive hypokalaemia was not a feature in our patient following the cessation of potassium supplementation. Two of twenty-five patients have developed hypokalaemia, despite potassium supplements (Munro *et al.*, 1970). It may be that the release of potassium from cellular tissue during fasting of the obese can be sufficient in some cases to maintain plasma levels despite continued urinary loss. The temporary period of hypercalcaemia is difficult to explain. It was unassociated with any other evidence of parathyroid hyperactivity and coincided with a slight increase in plasma phosphate concentration, within the normal range. The coincidence between the hypercalcaemia and the occurrence of markedly increased excretion of both calcium and phosphate is impressive. It may be that the hypercalcaemia was associated with resorption of some bone salt which was no longer required in the skeleton to support the former excess weight. Hypercalcaemia, to our knowledge, has not been remarked in any of the other long-term therapeutic fasts.

Plasma magnesium changes

Decreased plasma magnesium concentrations have not previously been reported during prolonged fasting. On the contrary normal plasma magnesium concentrations, despite magnesium 'depletion' in muscle tissue, have been described (Drenick *et al.*, 1969) during short-term fasting (1–3 months). The only other relevant report is a remark (Runcie & Thomson, 1970) that one patient who fasted 71 days had a normal plasma magnesium level of 2·2 mEq/l at the time when she developed latent tetany. The decrease in the plasma magnesium concentration of our patient was systematic and persistent.

Urinary excretion patterns

A breakdown in the renal control of electrolyte excretion has been reported to occur in some patients after 40 or 100 days during prolonged fasting (Runcie & Thomson, 1970). The increased urinary excretion of not only sodium and potassium but also calcium, magnesium and phosphate, noted in our patient, may be of similar origin. The concurrent development of these losses represents a definite change in the pattern of renal excretion after about 100 days of fasting. A similar increase in the urinary excretion pattern of sodium, potassium, calcium,

phosphate and magnesium was noted during protracted intermittent starvation in a previous patient (Stewart *et al.,* 1966). It is conceivable that some specific deficiency, perhaps of potassium or magnesium, may have brought about this 'nephropathy' but its real nature is at present conjectural.

The concentrations of plasma monovalent cations as well as phosphate, appeared to be essentially stable. Once the long-term fasting levels were established, plasma magnesium, glucose and urea levels also remained stable. Apart from the plasma calcium and bicarbonate concentrations, there were no other alterations in plasma constituents coincidental with the change in urinary excretion. The overall stability of the plasma constituents studied therefore contrasts vividly with the concurrently developing increase in the urinary losses of both monovalent and divalent cations, together with inorganic phosphate which occurred after about 100 days of fasting.

Such symptom-free developments are possibly more compatible with the release of surplus structural or other tissue components than with disadvantageous decompensatory loss of electrolytes from the extracellular fluid occurring as a result of some kind of nephropathy. It has been shown (Naeye, 1969) that hyperplasia and hypertrophy, not only of adipose tissue, but also of heart, kidneys, pancreas, liver and spleen occurs in obese subjects. It is suggested that the increased excretions described may have originated in dissolution of this soft tissue and associated skeletal excess. Accordingly, the electrolyte losses described are not regarded as representing a necessarily disadvantageous development during fasting.

Conclusions

Short-term fasts, although demonstrating to the obese patient his ability to lose weight, have a poor long-term outlook with respect to subsequent weight gain (MacCuish *et al.,* 1968). We have found, like Munro and colleagues (1970), that prolonged supervised therapeutic starvation of the obese patient can be a safe therapy, which is also effective if the ideal weight is reached. There is, however, likely to be occasionally a risk in some individuals, attributable to failures in different aspects of the adaptive response to fasting. Until the characteristics of these variations in response are identified, and shown to be capable of detection in their prodromal stages, extended starvation therapy must be used cautiously. In our view, unless unusual hypokalaemia is seen, potassium supplements are not mandatory. Xanthine oxidase inhibitors (or uricosuric agents) are not always necessary and could even be potentially harmful (*British Medical Journal,* 1971) perhaps particularly in the long-term fasting situation.

In most obese patients, there exists a weight loss 'barrier' representing a balance between the desire to lose weight on the one hand and the desire to eat on the other. When the first desire continues to overcome the second, the prolongation of therapeutic fasting should not be ruled out. The various biochemical features which developed in our patient are not considered to be expressions of a failure in adaptation. Starvation therapy can be completely successful, as in the present instance.

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Tumours of the sigmoid colon following ureterosigmoidostomy

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Summary
A case of bilateral adenomatous polyps and a case of adenocarcinoma of the sigmoid colon following ureterosigmoidostomy are described. The recent literature is reviewed. The possible causes and the mucosal changes of the large bowel after ureterosigmoidostomy are briefly discussed.

NEOPLASIA complicating ureterosigmoidostomy is an uncommon but well recognized entity. The number of cases reported in individual series has been small. In 1967, Kille & Glick reported two cases from this hospital, those being the eighteenth and nineteenth cases documented in the world literature. Two further cases from this unit are now reported.

Case 1
A 25-year-old female was born with ectopia vesicae. Ureterosigmoidostomy and total cystectomy were carried out at the age of 1½ years. She was well clinically, radiologically and biochemically until 1967 when she developed left renal pain. An IVP showed some dilatation of the left renal tract. As she was taking contraceptive pills, it was considered that this could be a hormonal effect similar to that described by Marshall, Lyon & Minkler (1966). She was advised to stop the 'pill' and an IVP was repeated 6 months later, but by this time considerable bilateral hydronephrosis had developed. In September 1968, she was admitted for re-implantation of the ureters. *At operation* two groups of polyps, each measuring 3·5 × 2·5 cm were found, one at the site of each ureterocolic anastomosis. These were
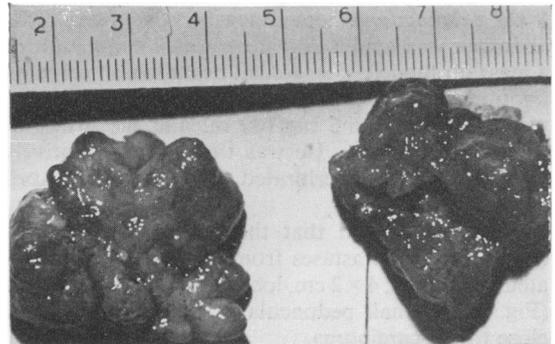

FIG. 1. Two groups of adenomatous polyps excised from Case 1. Scale in cm.

excised (Fig. 1). The ureters were re-implanted into the rectum to form a rectal bladder and a terminal iliac colostomy was fashioned.

Histology showed that the polyps appeared to be simple adenomata but the possibility of mucoid carcinoma could not be excluded. She did extremely well. The appearance on her IVP returned to normal except for a small area of pyelonephritic scarring at the lower pole of the left kidney. She was delivered of a healthy infant by Caesarean section 2 years later. Her urine is sterile and biochemistry normal.

Case 2
A 33-year-old man sustained a comminuted fracture of the pelvis, with extraperitoneal rupture of the bladder and urethra in 1941, at the age of 4 years. Almost moribund on admission to another hospital,