POST ECLAMPTIC ANURIA COMPLICATED BY HAEMORRHAGE INTO A DUODENAL CYST

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The following case history is presented because it demonstrates several of the pitfalls which may be encountered during the treatment of anuria complicated by continued uncontrolled losses of body fluids and also because of the unusual nature of the duodenal obstruction and the surgical measures taken to deal with it.

Case Report

A woman, aged 28 years, in the 39th week of her sixth pregnancy, was admitted to the University College Hospital of the West Indies at 1.30 p.m. on September 8, 1954, well advanced in labour; she had had no antenatal care. She had noticed slight swelling of her legs for some weeks before admission but had suffered from no other illness during or before this pregnancy. The blood pressure was 180/120 mm. of mercury before delivery, which took place at 2.45 p.m., 75 minutes after her admission to hospital. The child was a healthy boy weighing 7 lb. 10 oz. Some urine had been passed during labour before admission but no information as to its volume or appearance had been recorded. Fifteen hours after delivery, despite sedation with morphine and paraldehyde, which had reached the blood pressure to 150/100, she had two eclamptic fits. Immediately after these, her blood pressure fell to 100/80 but rose gradually to 180/120 within a few hours; 5 ml. of paraldehyde statim and morphine at intervals to a total of 1½ gr. were therefore given intramuscularly and no more fits occurred.

First day of puerperium. Next morning, 18 hours after delivery, no urine had been passed. Catheterization produced 465 ml. of dark brown urine which gave a strongly positive benzidine test although microscopically there were no red cells, pus cells or casts.

On examination the patient was slightly restless. Her blood pressure was 135/90 and the haemoglobin estimation was 10 g./100 ml. She was not jaundiced. The temperature was 101.8° F., pulse rate 120 per minute. Penicillin 'Seclopen,' 800,000 units, and streptomycin, 1 g. per day, were started to combat pulmonary and urinary infection. A tentative diagnosis of acute tubular necrosis was made and the Hammersmith regimen was started. The latter comprises the daily administration through a polythene or a Ryle's tube passed via the nose into the stomach of the following emulsion: Glucose, 400 g.; peanut oil, 100 g.; water, to 1,200 ml.; gum acacia to emulsify; concentrated vitamins A, B, C and D. This provides a protein 'sparing' diet of 2,500 calories, thus reducing protein breakdown to a minimum (Bull *et al.*, 1949). This was well tolerated at first without nausea or vomiting. Only 77 ml. of dark brown urine were passed during the day.

Second day. Vomiting began early in the morning and persisted. The abdomen was distended but soft. There was pain and tenderness in the epigastrium and right hypochondrium which was thought to be hepatic in origin but the liver was not palpable. During the day only 91 ml. of heavily blood stained urine containing much albumin and many granular casts were passed. The severe vomiting resulted in a fluid balance deficit of 2,700 ml.

Third day. Vomiting persisted; the upper abdomen was still distended and tender although less painful. A diagnosis of acute dilatation of the stomach was made. The patient was therefore propped up in bed, the intragastric drip discontinued and hourly gastric aspiration instituted; 3,030 ml. of 12 per cent. dextrose and a litre of isotonic sodium chloride were given intravenously. At the end of the day 2,257 ml. of dark brown fluid had been aspirated from the stomach and only 56 ml. of fairly clear urine still heavily laden with albumin had been passed (Fig. 1).

Fourth day. The patient's general condition had improved. She was much more comfortable and the vomiting had ceased; the blood pressure was 130/90 mm.; 91 ml. of urine containing granular

TABLE 1.—INTAKE AND OUTPUT

	INTAKE							OUTPUT						
Day	I.V. Fluid (ml.)	Dextrose (g.)	I.V. Sodium Chloride (g.)	I.V. Potassium chloride (g.)	Oral sodium bicarbonate (g.)	Pancreatin (g.)	Blood (ml.)	Oral fluid (ml.)	Total fluid	Urine (ml.)	Vomitus (ml.)	Stomach aspiration (ml.)	Fluid faeces (ml.)	Drainage of fistula (ml.)	Total fluid Loss (g.)
1		100						1415	1415	77					77
2		478						1195	1195	90	2600				2690
3	3030	245	9.0					65	3095	50	200	2260			2510
4	2080	104						90	2170	90		440			530
5	640	312						730	1370	170		60			230
6	1400	360					500	500	2400	210	880	860			1950
7	2800	351							2800	160		960			1120
8	1700	135	4.5					30	1730	210		1430			1640
9	2000	118	26.1						2000	300		560			860
10	1600	150	21.6						1600	750		760			1510
11	2500	250	18.0						2500	1560		100			1660
12	3450	500	7.7					140	3590	1900		510			2410
13	3350	465	7.7					80	3430	2220		200			2420
14	3000	217	7.7					420	3420	2480	980				3460
15		100						870	870	3080	1480				4560
16	2000	234	1.8					1140	3140	2520	180	1490			4190
17	4600	538	16.7					480	5000	2510		3690			7200
18	7490	898	23.0					435	7925	3290		1530			4820
19	3500	420	7.7				500	600	4100	2890	220	1440			4550
20	5550	660	14.4	6.0			500	280	5830	3850		1340			5190
21	5000	600	15.3	18.0				680	5680	3340		1940			5280
22	7000	840	15.3	2.0				570	7570	3530	240	1190			4960
23	4000	480	15.3	6.0				30	4030	2080	880	800			3760
24	3430	310	18.0	4.0			540		3970	1840		660			2500
25	2700	320	9.0					970	3670	1820		1460			3280
26	4400	530	18.0					630	5030	2800	100	330			3230
27	2600	240	9.0					780	2780	1590	230			460	2280
28	4000	480	18.0					810	4810	2670				850	3520
29	2000	240	9.0					1175	3175	2430				700	3130
30	Intravenous Therapy Discontinued							1405	1405	980				1300	2280
31								2192	2192	1530				500	2030
32								2100	2100	990				250	1240
33								2280	2280	1590				240	1830
34								2730	2730	1900				110	2010
35								2560	2560	1890				390	2280
36					4.0			3140	3140	2110			150	70	2330
37					4.0	0.6		1830	1830	1790			360	70	2220
38					4.0	0.9		2180	2180	1780			100	120	2000
39					4.0	0.9		3360	3360	1710			60	90	1860
40					4.0	0.9		2730	2730	1200			630	50	1880
41					4.0	1.2		2210	2210	1820			180	30	2030
42					4.0	1.4		2720	2720	2270				20	2290
43					4.0	1.4		2610	2610	3170				30	3200
44						1.4		2500	2500	3200				40	3240
45						1.4		2490	2490	2500				50	2550
46						1.4		2370	2370	2180				30	2210
47						1.4		2020	1400	1340				60	1400
48						1.2		2340	2340	2180				100	2280
49						1.1		2240	2240	2170				60	2230
50						0.9		2020	2020	2320				50	2370
51						0.8		1230	1230	1990				50	2040
52						0.5		1530	1530	2100				40	2140
53						0.5		1860	1860	1070				50	1120
54						0.3		1380	1380	1880				60	1940
55						0.5		2230	2230	1630				20	1650
										Pancreatic Fistula Closed					

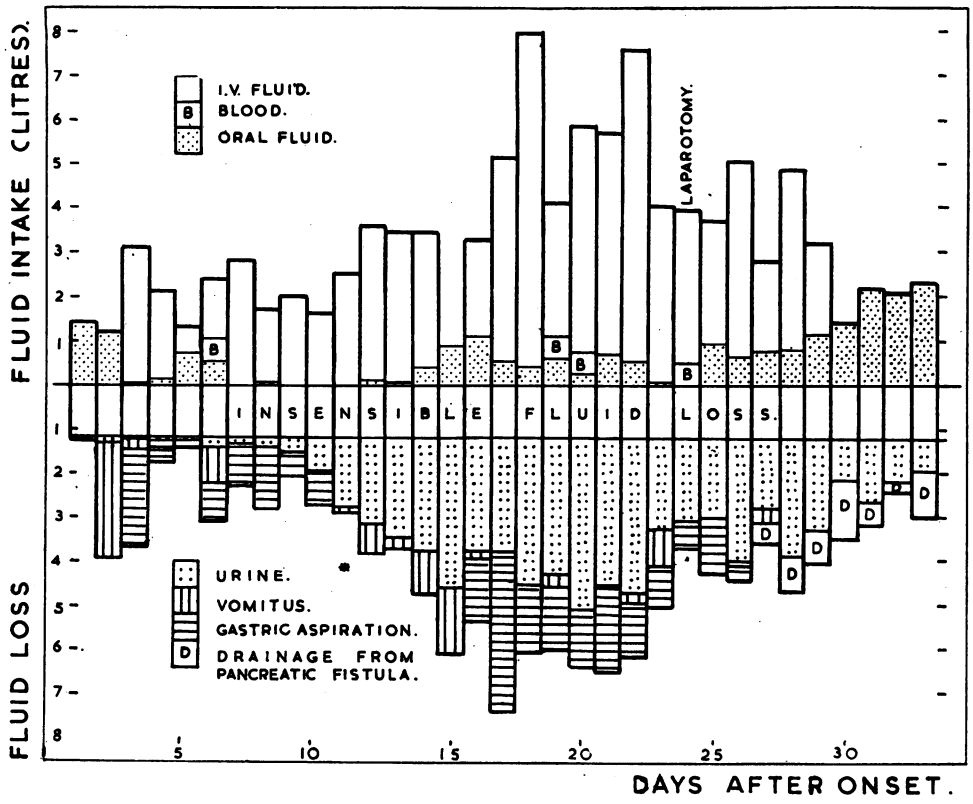


FIG. 1.—Fluid Balance Chart. The asterisk indicates the onset of the primary diuretic phase on the eleventh day. The insensible fluid loss is estimated at 1,200 ml. for a patient at rest with the atmospheric temperature about 80°. (Gamble 1947.)

casts and red blood cells were passed. During the night 445 ml. of fluid were aspirated from the stomach.

Fifth day. Because of the reduced accumulation of fluid in the stomach the intragastric drip was started again. The total urinary output rose to 129 ml. The patient was slightly jaundiced.

Sixth day. To our great disappointment the vomiting recurred. The haemoglobin had fallen to 7.0 g./100 ml. and 1,000 ml. of whole blood were given. Despite this intravenous fluid the day ended with a negative balance of 750 ml. However, the urinary output continued to rise steadily and 210 ml. were passed. The patient was noted to be rather drowsy but quite coherent.

Seventh day. The intragastric drip was not started again but 12 per cent. dextrose in water was continued intravenously. Large quantities of fluid were still being aspirated from the stomach and there was a marked fall in the serum sodium and chloride (Fig. 2 (b)). Replacement therapy was commenced adding 1.8 per cent. saline to the intravenous fluid.

In this way 4.5 g., 26.1 g. and 21.6 g. of sodium chloride were given on the eighth, ninth and tenth days respectively. Thereafter an average of 11.6 g. per day were required to maintain normal serum levels of these electrolytes (Fig. 2). The blood urea had now risen to 154 mg./100 ml. and the serum potassium was 7.67 mEq/l., but it was hoped that as the daily volume of urine was rising steadily an adequate output would soon be established. The drowsiness had increased and her mental reactions were slower.

Ninth day. Early in the morning at 4.45 a.m. a convulsion occurred followed by a second at 7 a.m. and a third at 8.30 a.m. The blood urea was 344 mg./100 ml. and the serum potassium 9.7 mEq/l. The blood pressure was 150/110 mm. Between and after the fits, which were controlled by 5 ml. of intramuscular paraldehyde, she was very drowsy but became restless when disturbed.

The convulsions did not recur but for the next 48 hours there was occasional twitching of the facial and abdominal muscles. Her mental condition could not be assessed because of the paraldehyde sedation.

Eleventh day. The serum sodium and chloride

TABLE 2—BLOOD BIOCHEMISTRY

Day after onset	Blood Sodium mEq/l.	Potassium mEq/l.	Chloride mEq/l.	Bicarbonate mEq/L.	pH	Urea, mg./100 ml.	Bilirubin 1 mn. (direct) mg./100 ml.	Bilirubin Total (Indirect) mg./100 ml.	Icteric index (units)	Haemoglobin g./100 ml.	Packed cell vol. per cent.	Total protein g./100 ml.	Albumin g./100 ml.	Globulin g./100 ml.
1						40				10.0				
2	143.5	6.39	100	15	7.3	82	1.2	2.0		8.8	26	5.7	2.0	3.7
3	147.8	6.39	92	23		175								
4						216				6.1				
5						208	2.1	3.1			18	5.8	2.0	3.8
6							2.1	4.6	20		23	6.2	2.9	3.3
7	139	7.67	75	24	7.5	154	1.6	3.3				6.2	2.8	3.4
8	130.4	6.9	68	28	7.5	222	1.3	2.1	13.5		29	6.1	2.8	3.3
9	128.3	9.7	62	29	7.6	344	0.9	2.6	11.7	10.4	33	6.1	2.8	3.3
10	134.8	9.7	74	25	7.55	365	0.9	2.6	9.0			6.0	2.8	3.2
12	163	7.67	100	25	7.4	368			9.0	8.4	25	5.4	2.5	2.9
13	152	6.64	100	24	7.5	336						6.0	2.1	3.9
14	147	5.62	97	24	7.4	320				6.7		6.2	2.1	4.1
15	147.8	5.1	110	25	7.5	224	1.1		10.5		21	6.1	2	4.1
16	147.8	4.6	97	28	7.5	230								
17	156.5	4.09	108	25		248								
19	143.5	3.2	102	25	7.3	158	0.5		6	5.4	18	5.6	2	3.6
20						128								
21		3.07		28		84								
22	139.1	4.09	105	25										
23	143.5	3.7	102	29		42			7.6			5.8	1.9	3.9
24			100	25		41				9.4	28			
26	139.1	5.1	100	26	7.35	24								
27						36	0.9	1.6				5.7	1.6	4.1
28		4.85	103	25						10	31			
29	141.8	5.1	105	25	7.4									
30	139	5.1	108	17	7.4	19	0.7		1.4	13.1				
33	130.4	6.4	103	19		40	0.6		1.4	8.8				
36	130.4	6.64	100	18		41	0.2		1	10.5				
40	139	5.37	98	30		28	0.4		0.9	8.2				
43	139	5.37	94	29	7.4	18	0.1		0.5	6				
126	143.5	4.6	101	26	7.4	13			3.3	10.9	37	7.3	3	4.3

were now almost normal and the renal function was improving rapidly; 750 ml. of urine had been passed the previous day. It was hoped, therefore, that the pylorospasm which was thought to be present, due to or aggravated by the low serum sodium chloride, would disappear. This hope was fostered by a fall in the aspirate to 104 ml. The urinary output reached 1,556 ml. and the patient was regarded as well on the way to recovery of adequate renal function. The paraldehyde was discontinued as there had been no muscle twitching for 24 hours. She was less drowsy but restless and unco-operative. The slight jaundice had disappeared.

Twelfth day. She pulled up the Ryle's tube and it was not replaced. Intravenous glucose saline was continued and once more oral fluids were cautiously begun; vomiting occurred immediately. The tenderness in the hypogastrium was still present but was greatly diminished. The bowel sounds were present and normal. The serum potassium had reached a peak of 9.7 mEq. per litre on the ninth and tenth days and was now gradually falling as a result of diuresis which reached 1,901 ml. that day. The electrocardiograph, however, still showed the high peak T waves characteristic of hyperkalaemia (Fig. 3).

Fifthteenth day. The diuresis continued and the serum electrolytes were now nearly normal. The blood urea was 224 mg./100 ml. She was alert and talkative but rather unco-operative. Four days previously the patient had passed a normally-formed stool and an enema now produced another normal stool and flatus. The bowel sounds were still normal and there was only a little soft distension of the upper abdomen. On repeated questioning no history of any dyspepsia or vomiting before delivery could be elicited. Neverthe-

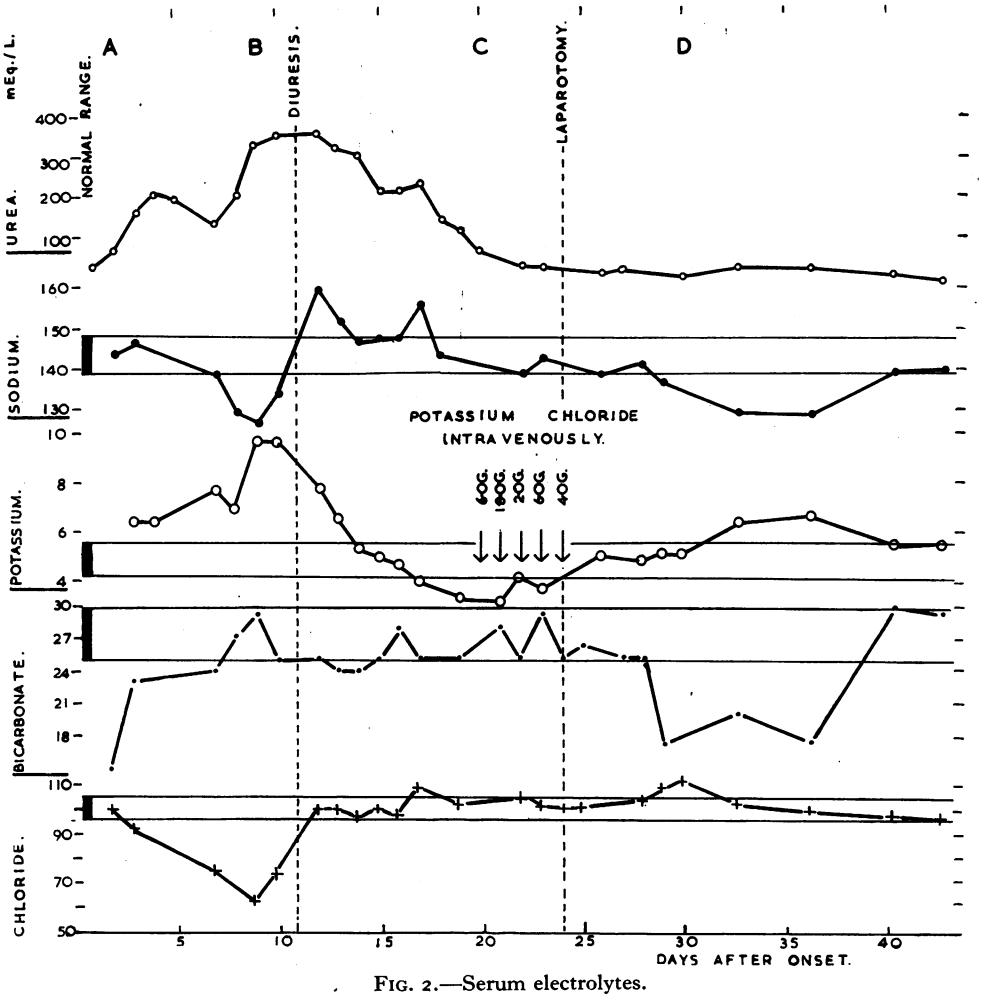


FIG. 2.—Serum electrolytes.

less, we were slowly being forced to the conclusion that there must be a partial high intestinal obstruction of some sort, but its nature remained obscure.

Sixteenth day. Visible gastric peristalsis was seen for the first time. We were still thinking in terms of a severe pylorospasm and it was decided to try the effect of the anti-spasmodic methyl atropine nitrate (' Eumydrin ').

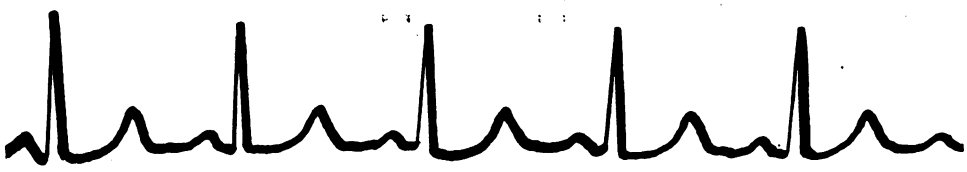
The Ryle's tube was replaced with great difficulty as the patient was quite unco-operative and appeared to have no appreciation of the gravity of her condition. Thick green gastric contents were aspirated. The following regimen was therefore instituted: The patient was kept under constant immediate supervision to prevent the tube being pulled up; she was given oral fluids, 3 oz., two-hourly; gastric aspiration was performed alternating with the feeds; gastric lavage with 10 oz. of saline was performed eight-hourly; ' Eumydrin,' 10 minims, was given three times a day through the tube; intravenous fluids were continued as before.

There was no response to this regimen; large volumes of fluid continued to be aspirated.

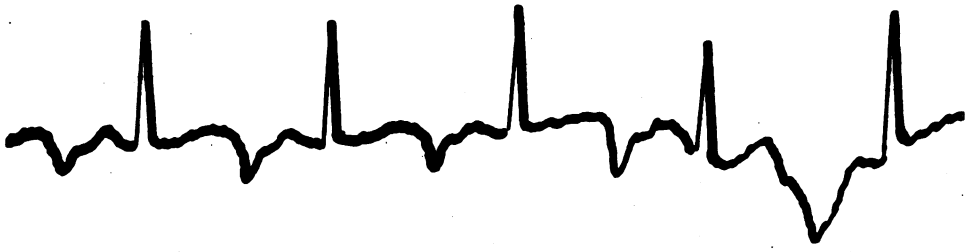
Nineteenth day. The gastric retention was unchanged. A new complication had arisen; as a result of the marked diuresis and the continued loss of potassium in the aspirated fluid the serum potassium had fallen to 4 mEq/l. (normal range 4.1 to 5.6 mEq/l.) on the 17th day. Attempts to raise it by giving potassium citrate by mouth had failed and the patient was becoming weaker and more drowsy. The haemoglobin had fallen to 5.4 g. and a transfusion of 1,000 ml. of whole blood was given during the next 24 hours.

Twentieth day. The serum potassium continued to fall and was now well below 3.0 mEq/l.; the weakness and drowsiness had increased; there was

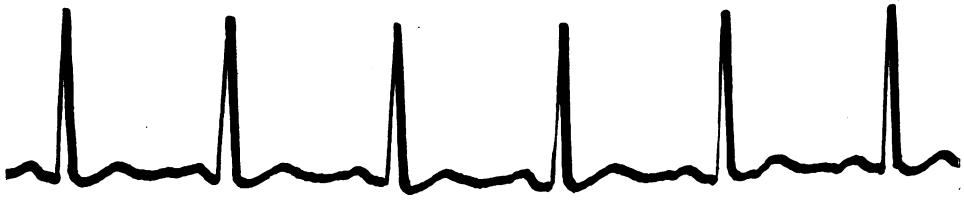
FIG. 3.—Electrocardiograph changes.



Twelfth day after onset, hyperkalaemia. Note the high-peaked T-waves (Lead II).



Twentieth day after onset, 2.45 p.m., hypokalaemia. Note the prolonged QT interval, the sharp T-wave inversion and the ventricular extrasystole (Lead II). Potassium chloride was commenced intravenously immediately after this E.C.G. was taken.



Twentieth day after onset, 4.15 p.m., normal complexes. After the administration of 1.3 g. of potassium chloride intravenously the serum potassium was still low but the E.C.G. was normal (Lead II).

dependent oedema, an irregular pulse and abnormal electrocardiograph tracings (Fig. 3). Replacement of potassium was clearly imperative; potassium chloride was therefore given intravenously; 6 g. of 'Analar' chemically pure potassium chloride were dissolved in 100 ml. of pyrogen-free doubly-distilled water in a volumetric flask and autoclaved at 15 lb. pressure for 30 minutes; the sterile solution so obtained was added to 1,900 ml. of sterile 5 per cent. dextrose solution so that each litre of solution contained 3 g., i.e. 40 mEq. of potassium chloride. This was given intravenously by slow drip over a period of ten hours so that a rate of 10 mEq. of potassium chloride per hour was not exceeded. This is regarded as a safe rate of administration where the urinary output is satisfactory. The electrocardiograph returned to normal after 1.3 g. of potassium chloride had been given (Fig. 3), but the serum potassium remained low, 3.1 mEq/l. Next day 18 g. of potassium chloride were given at the same rate; thereafter the serum potassium rose slowly to within normal limits and was maintained over the next three days by 2, 6 and 4 g. of potassium chloride respectively until operation.

Twenty-third day. The serum electrolytes were now normal once more but there was no diminution in the intestinal obstruction as judged by the volume of gastric aspirate, which averaged about a litre a day. It was now obvious that conservative measures had no further place in the treatment and that the patient's only chance of survival lay in the relief of the intestinal obstruction by operation. A barium meal was given; the appearances suggested a partial duodenal obstruction probably due to some extrinsic mass constricting the duodenal loop. The 18-hour follow-through film showed that most of the barium was still in the stomach although a little had passed through to the ascending colon.

Twenty-fourth day. Operation. Laparotomy was performed under general anaesthesia through a supraumbilical midline incision. The stomach was grossly distended, the small and large intes-

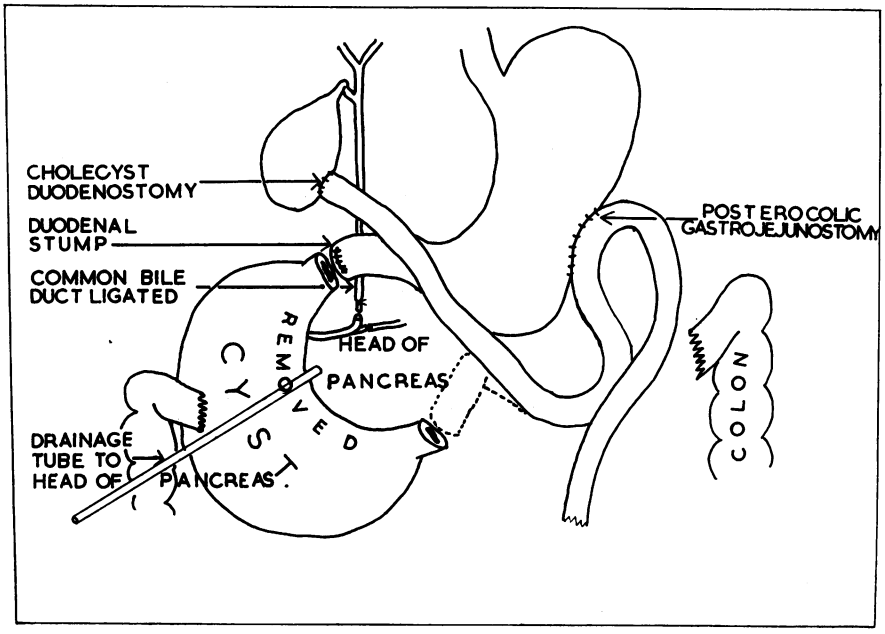
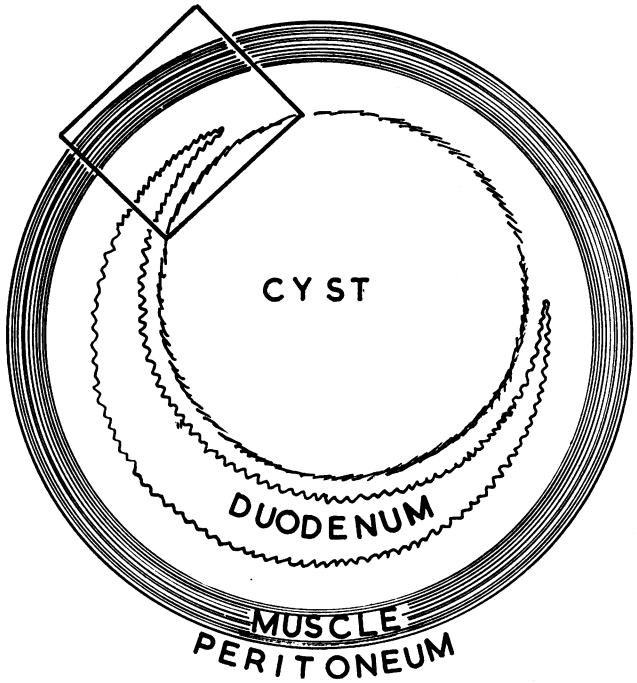


FIG. 4.—Diagram illustrating operative procedure.

FIG. 5.—Photograph of the resected duodenum and cyst. The specimen has been bisected to show the contents of the cyst and the ampulla of Vater into which a probe has been passed.



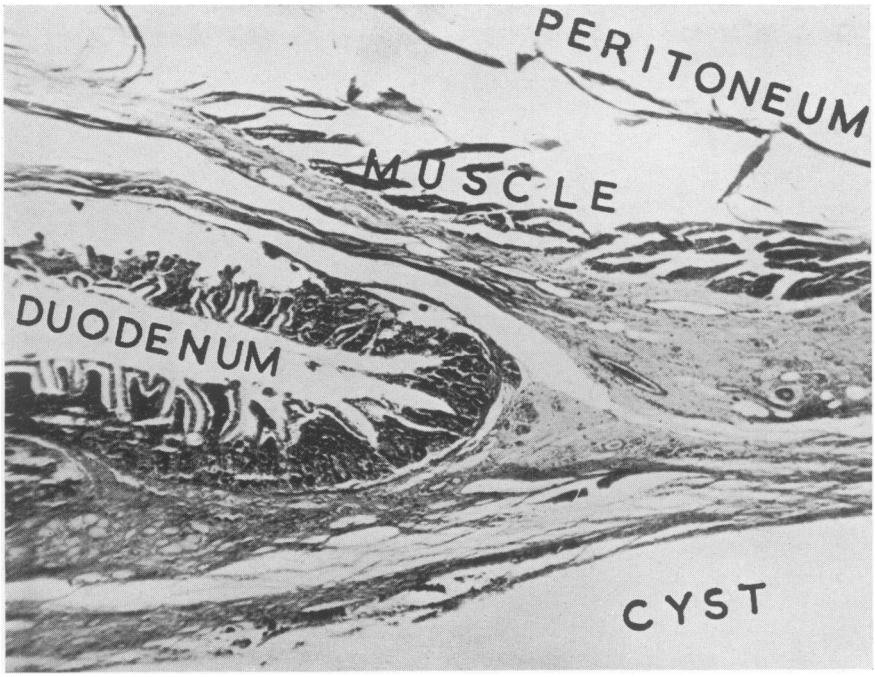


FIG. 6.—(a) Diagram of the anatomy of the duodenal cyst. (b) Photomicrograph of the area inset.

tines were collapsed, the gall bladder was distended but could be emptied on pressure. Just below the hepatic flexure of the colon there was a large cystic retroperitoneal tumour which extended across the midline to the root of the mesentery (Fig. 4). The right kidney felt normal.

The peritoneum was incised over the tumour and easily dissected off it. The tumour was intimately related to the second and third parts of the duodenum and the head of the pancreas. The duodenum was divided on either side of the tumour and removed with it; it was realized that the common bile duct and the pancreatic duct had been divided.

The proximal end of the duodenum was closed. The fourth part of the duodenum was mobilized. Anterocolic end-to-side cholecyst-duodenostomy and posterocolic gastrojejunostomy were performed. The pancreatic duct could not be located in the head of the pancreas. The peritoneum of the posterior abdominal wall was closed around a tube drain passing to the head of the pancreas and brought through a stab incision in the right loin (Fig. 4). The incision was closed in one layer with steel wire.

Pathologist's report. The specimen consists of a portion of duodenum measuring 130 x 70 x 50 mm. showing a cystic swelling in the wall. The cyst is lined by necrotic tissue and blood elements. The wall adjacent to the duodenal lumen consists of fibrous tissue and duodenal mucosa, while adjacent to the peritoneal surface it consists of fibrous tissue and duodenal muscle (Fig. 6).

Twenty-fifth day (first post-operative). The patient's condition was satisfactory; there was only slight abdominal distension and the bowel sounds were normal. The serum electrolytes were within normal limits. A Ryle's tube was in position and the stomach was aspirated half-hourly. During the day 917 ml. of fluid were given through the tube and 1,457 ml. of bile-stained fluid were aspirated.

Twenty-sixth day (second post-operative). Of 630 ml. of fluids given orally only 430 ml. were aspirated or vomited and it was assumed therefore that fluid was now passing through the anastomosis and the Ryle's tube was removed. There was very little drainage through the abdominal drainage tube.

Twenty-seventh day (third post-operative). She vomited during the early part of the day but not thereafter. There was no drainage but on applying continuous suction to the drainage tube 470 ml. of clear pancreatic juice were aspirated during the day. The abdominal wall adjacent to the fistula was protected by aluminium paste.

Twenty-ninth day (fifth post-operative). Oral fluids were now being taken satisfactorily without vomiting and the intravenous fluid, which had been given continuously for 26 days, apart from a single interval on the 15th day, was discontinued. The drainage of pancreatic fluid continued steadily averaging 600 ml. a day. The serum sodium and bicarbonate remained low; 4 g. of sodium bicarbonate were given orally each day.

Thirty-seventh day (thirteenth post-operative). The motions continued to be frequent, three to five a day, and became relaxed and pale in colour and contained undigested meat fibres. Pancreatin, 0.3 g., two hours after meals, three times a day was begun. The patient was cheerful and felt well.

Fortieth day (sixteenth post-operative). The drainage from the tube was much less, 50 ml. per day. It was purulent and coliform organisms were cultured from it. The serum electrolytes were normal and remained so thereafter. The diarrhoea was satisfactorily controlled by gradually increasing the dose of pancreatin to 1.4 g. a day.

Forty-seventh day (twenty-third post-operative). There was no diarrhoea now. Liquid neohydriol, injected into the drainage tube, entered the small intestine (Fig. 7). The tube was shortened half an inch per day and the dose of pancreatin gradually reduced to 0.3 g. twice daily.

Fifty-fifth day (thirty-first post-operative). The abdominal drainage tube was removed and no further drainage occurred. The patient was taking a normal moderately high protein diet and now weighed 102 lb.

Seventy-second day (forty-eighth post-operative). The pancreatin was discontinued on the 66th day. There was no further diarrhoea. She was discharged symptom free weighing 105 lb., blood pressure 105/65 mm. of mercury, haemoglobin 11.1 g. per 100 ml. Her renal function was still impaired as indicated by failure to concentrate urine above a specific quantity of 1,010.

Follow-up

Seven weeks later she looked well and was free of symptoms. The blood urea was 13 mg. per 100 ml.; average urea clearance (Van Slyke), 74 per cent.; urea concentration test, 0.6 to 1.7 g. per cent.; maximum urine specific gravity, 1,008; serum sodium, potassium, chloride and bicarbonate all within normal limits.

Comment

The treatment of anuria due to acute tubular necrosis consists largely in an attempt to keep the patient alive until the tubular epithelium can regenerate and begin to function. The high mortality rate for this condition in the past was mainly due to biochemical derangements which were often the result of well meaning but mis-

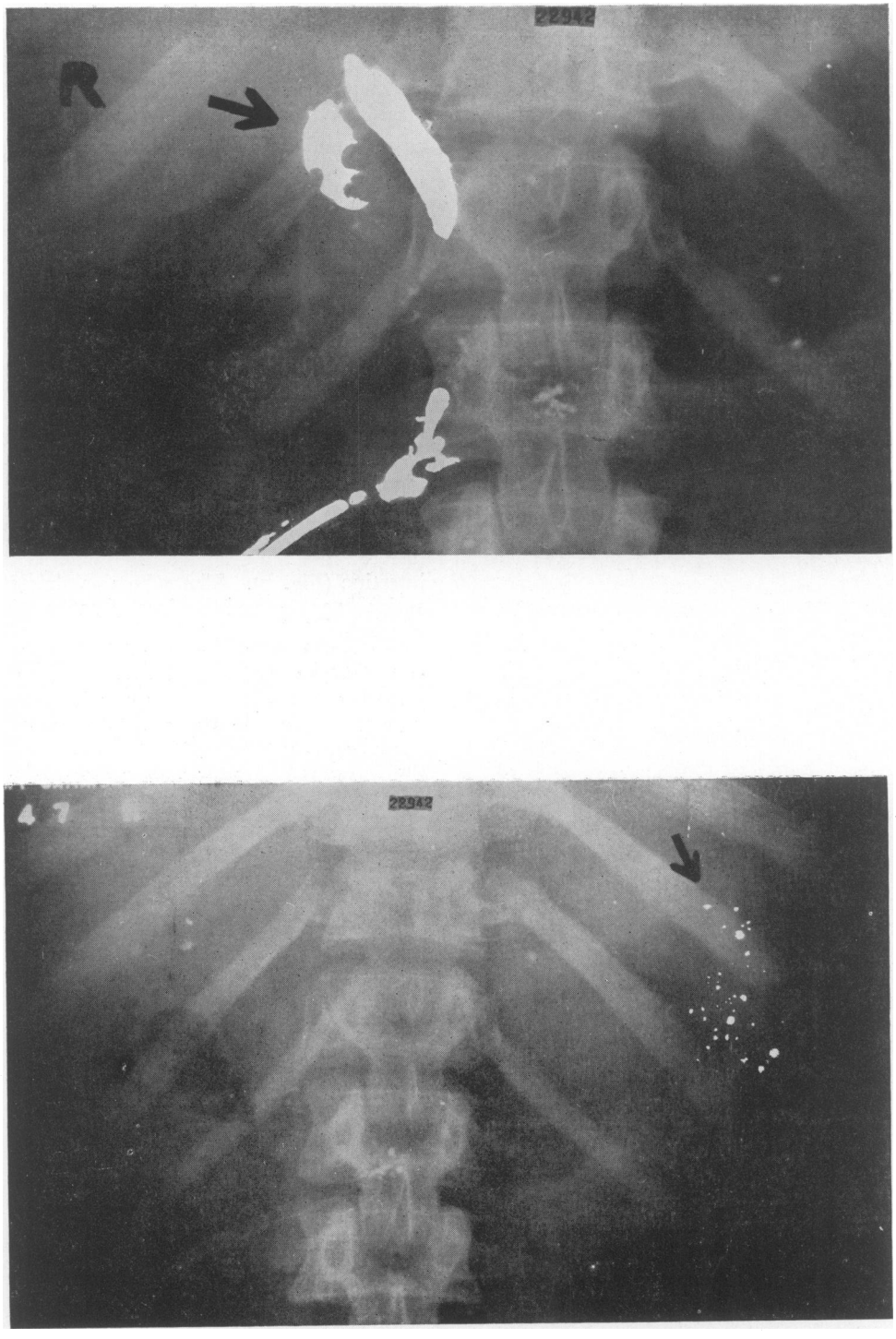


FIG. 7.—(a) Neohydriol has passed down the catheter in the fistula into the duodenum (arrowed).
(b) Twenty-four hours later there are flecks of neohydriol in the splenic flexure.

guided therapy, particularly with large quantities of intravenous water, salt and sodium sulphate, which the patient was unable to excrete. Nowadays the conservative method of treatment worked out by Bull and his colleagues at the Hammersmith Hospital, London, would appear to be the method of choice in most cases. If, however, persistent severe fluid and electrolyte loss occurs due to vomiting or diarrhoea, the patient must be sustained if possible by giving the necessary fluids and calories intravenously. The difficulties involved in maintaining normal fluid and electrolyte balance using this method are greater than with the intragastric drip technique and necessitate continuous detailed biochemical analysis of the blood plasma. In the present case there were five distinct episodes of biochemical derangement. The first became apparent on the second day (Fig. 2 (a)) when the serum bicarbonate fell to 15 mEq/l. and the serum pH to 7.3. The patient had eaten and drunk little since the onset of labour on account of the heavy sedation necessary to control the eclampsia. The ketosis disappeared rapidly with intravenous dextrose and water and was therefore presumably the result of starvation and dehydration.

The next episode (Fig. 2 (b)) was the result of replacing gastric secretion by intravenous dextrose and water only. After the seventh day the serum sodium and chloride fell rapidly (Fig. 2 (a)) and an alkalosis developed, the serum pH rising to 7.6. The sodium and chloride ions were replaced by giving 1.8 per cent. saline intravenously. Double physiological strength saline was used because it was necessary to give large amounts of salt in a relatively small volume of water as the patient was still oliguric. As a result of this treatment the serum sodium and chloride concentrations were restored to normal and remained so with an average intake of 11.6 g. of sodium chloride intravenously over the next 20 days until oral feeding was established.

Episode three, hyperkalaemia, developed concomitantly with the fall in serum sodium and chloride (Fig. 2 (b)), the serum potassium concentrations rising steadily to a maximum of 9.7 mEq/l. on the ninth and tenth days. As no exogenous potassium was being given this rise must have been the result of endogenous protein breakdown, which was inevitably large because the amount of dextrose which could be given as a 12 per cent. solution during the oliguric phase was limited and supplied an average of only 700 calories per day, while it is now generally accepted that at least 1,600 calories per day are necessary to reduce endogenous protein breakdown to a minimum. Recently 40 per cent. dextrose has been given successfully for periods of up to 21 days through a 40 mm. polythene catheter passed into the vena cava. By this means venous thrombosis, which would certainly have occurred if such a concentrated solution had been given into a superficial vein, was avoided and 1,600 calories a day could be given in a litre of fluid (Russell, 1954; Chalmers, 1955).

By the ninth day the serum potassium had reached 9.7 mEq/l. and the patient was on this account in grave danger of heart failure. The urinary output, however, had been rising steadily and on the 11th day had exceeded a litre and a half. Thereafter potassium was rapidly excreted in the urine and the serum potassium was within normal limits by the 14th day.

Large quantities of potassium were lost in the urine once the diuresis was established and this led to the fourth episode of biochemical upset, one of hypokalaemia (Fig. 2 (c)). In straightforward cases of anuria, potassium-containing foods, supplemented if necessary by potassium chloride or citrate by mouth, suffice to maintain potassium balance during the diuretic phase. Here, however, the oral route was not available and potassium had to be given intravenously. The main risk of this procedure is a sudden rise in serum potassium to levels in the neighbourhood of 10 mEq/l., when sudden heart failure may occur (Harvard and Carey, 1949; Black and Pyrah, 1954), and this is more likely if potassium is given too quickly or if there is poor renal function. In the present case the urinary output averaged over three litres a day at this stage; the potassium chloride was given slowly and a careful watch was kept on the patient's pulse and electrocardiograph tracings as well as the serum potassium concentration. Balance studies were not made but the fact that 24 g. of potassium chloride were given over 48 hours before the serum potassium level rose to normal indicates how marked the deficit had been.

An interesting feature was the rapidity with which the electrocardiograph tracings became normal after only 1.3 g. of potassium chloride had been given, while the serum potassium remained low for 48 hours until after some 24 g. of potassium chloride had been administered.

Pancreatic fistula following division of the accessory pancreatic duct (Santorini) is prone to close spontaneously when this duct drains only a small segment of the pancreas or when it communicates with the main pancreatic duct (Wirsung). Untreated partial or complete division of the main pancreatic duct produces a persistent pancreatic fistula. Warren (1951) stated that spontaneous closure of the main duct fistula was usually followed by abdominal pain, anorexia, nausea, vomiting, chills and fever. Total pancreatic fistula is usually rapidly fatal in dogs.

Newman and Eisenstein (1949) stated that the average 24-hour secretion from a complete pancreatic fistula in their patient was 1,250 cc., which compared with those of other investigators. In our patient the maximum daily secretion was 1,300 ml. on the eighth post-operative day and fell rapidly to less than 100 ml. on and after the 14th post-operative day. It was only during the first two weeks that a marked loss of sodium and bicarbonate occurred causing a well-defined fall in serum concentration of these electrolytes. This was rapidly controlled by giving sodium bicarbonate by mouth while the fistula remained open.

Principles in the management of pancreatic fistula are: (1) Skin protection. (2) Restoration and maintenance of fluid and electrolyte balance. Hildes and Ferguson (1952) stated that the sodium and potassium concentration in pancreatic fluid remained constant and approximated to the serum concentrations irrespective of the volume of secretion. With an average daily loss of 600 ml. of pancreatic fluid in this patient, the electrolyte imbalance was considerable in the absence of adequate replacement therapy (Fig. 2 (d)) and constituted the fifth and last episode of biochemical derangement. (3) Assistance in the digestion of fat and protein. Warren advised administration of 8 g. pancreatin daily in three divided doses. In the present case the maximum daily dose was 1.4 g. (4) Depression of the physiological activity of the acinar elements of the pancreas. Warren stated that atropine, ephedrine and banthine (B. diethylaminoethyl xanthine-9 carboxylate methobromide) are pancreatic depressants but Hildes and Ferguson (1952) found that banthine had no apparent effect on the daily volume of fistula juice. (5) Operative treatment if spontaneous closure does not occur after an adequate period of expectant treatment.

In this case we know from the nature of the operation that this was a complete fistula of the main pancreatic duct. The formation of this fistula had been encouraged with a view to implantation into the small intestine at a second operation. Fluid and electrolyte loss were controlled by adequate replacement therapy while the fistula persisted and digestion was aided by administration of pancreatin orally.

Spontaneous closure occurred and the patient has remained fit and well with no metabolic disturbance and on physical examination there is no evidence of cyst formation.

We postulated the formation of an abscess along the fistula which eroded into the small intestine; this was confirmed by X-ray. The probable site of the fistulo-enterostomy is in the jejunum somewhere between the cholecyst-duodenostomy and the gastro-jejunostomy stomata.

The pathogenesis of the cyst is doubtful; possible aetiologies are:

(a) Duplicative cyst of the duodenum (enterogenous cyst). The first cases of enterogenous cysts of the duodenum were described by Sanger and Klopp (1880) and Roth (1881), both in the newborn. Up to 1953, 21 cases have been reported in the literature. These cysts are characterized by the reduplication of duodenal muscle in the wall and by a mucosal lining which is usually indistinguishable from normal duodenal mucosa. Occasionally mucosa of other parts of the intestinal tract may be present. Polson and Isaac (1953) stated that lining of gastric mucosa was present in eight of the 21 cases. The mucosal lining may be partly or totally destroyed by infection or haemorrhage. These cysts cause duodenal obstruction early in life in most cases within the first four months of life and are usually diagnosed preoperatively as congenital hypertrophic pyloric stenosis. Cases have been reported in adolescent and young adults by Gardner and Hart (1935), Booher and Pack (1946), Shallow *et al.* (1947), Lorber and Machelia (1948), Gordimer and Bluestone (1950). Peple (1948) reported a case in a female aged 69, the oldest case in literature. The histology of our specimen does not fulfil the criteria of enterogenous cyst (Fig. 6).

(b) Cysts of the ampulla (choledochocoele). These are characterized by recurrent attacks of upper abdominal pain simulating cholecystitis and sometimes with recurrent jaundice. Brooks and Weinstein (1943) reported a case diagnosed as cholecystitis and peptic ulceration during acute attacks of pain. Wheeler (1940) compared these cysts with ureterocoele. In our case there was no previous attack of upper abdominal pain and the ampulla was quite distinct from the cyst wall. On the sixth day, however, the patient showed slight jaundice and the total serum bilirubin rose to a maximum of 4.6 mg./100 ml. (normal range 0.2 to 1.08 mg./100 ml.) and the icteric index to 20.0 units (normal range 0 to 10 units). This can be explained as the result of the sudden enlargement of the cyst consequent on the haemorrhage into it producing temporary obstruction of the ampulla of Vater.

(c) Cyst arising in aberrant pancreas. Norris (1946) reported a tumour of an aberrant pancreas in the duodenum and stated that these tumours occurred more often in the duodenum than elsewhere but had been found in the walls of the stomach, jejunum and ileum. Of 257 duodenal lesions at the Presbyterian Hospital, Whipple found 13 primary tumours of which three were heterotopic pancreas. Riker (1951) suggested the

possibility of necrosis and autodigestion in an aberrant pancreas producing a cyst in which no trace of the original lesion could be found.

(d) Cyst of Brunner's glands. Robertson (1941) mentioned the possibility of a retention cyst of Brunner's glands but stated that these cysts were usually only a few millimetres in diameter and were symptomless. Booher and Pack (1946) reported a case of cystic tumour of duodenum of this nature in a female aged 52.

Adenoma of Brunner's glands are rare. They present with haemorrhage or obstruction. Feyrter (1934) found three in 2,800 duodena examined. Cases have been reported by Willis and Lasersohn (1925) and Balfour and Henderson (1929). These adenomata may undergo cystic degeneration. There was no histological evidence of this in our specimen.

(e) Lymphatic cyst arising in the submucosal lymphatics. Lymphatic cysts are not uncommon in the mesentery of the small intestine. We have not been able to trace any case of submucosal lymphatic cyst in the literature.

The only similar case of duodenal cyst in the literature was reported by Riker (1951). In discussing the probable aetiology he mentioned the possibility of a duodenal cyst being the result of a tiny mucosal perforation with abscess formation and organization of haematoma from spontaneous submucosal haemorrhage.

From the histology it is evident that this cyst had been present for some time. Haemorrhage occurred into it probably at the time of the eclamptic fits. This would account for the rather marked fall in blood pressure which succeeded the convulsions, the early appearance of anaemia, the pain and tenderness in the upper abdomen which we had regarded as hepatic in origin, the slight jaundice and especially the incomplete duodenal obstruction which was such a dominant feature of the case.

Treatment of duodenal cysts is on the whole extremely unsatisfactory. One reason for this is that they usually occur in infants who, because of prolonged vomiting, are poor operative risks. Mortality in infants is as high as 70 per cent. (Gordimer and Bluestone, 1950), but it is lower in adults. In no case in the literature has duodenectomy been performed for this benign condition.

Summary

A case of post eclamptic anuria complicated by duodenal obstruction due to haemorrhage into a duodenal cyst is described. The difficulties encountered in the management of the case are discussed. A review of the literature on duodenal cysts is given.

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