

## PERITONEAL DIALYSIS

ANTHONY A. BUSUTTIL \*  
M.D.

Department of Pathology,  
Royal University of Malta.

According to S. T. Boen (1961), Wegner, in 1877, was the first to point out that the peritoneum could be considered as an inert, semi-permeable membrane permeable to water, solutes and substances of a low molecular weight. A. J. Hamburger concluded that the exchange of solutes and other diffusible products between the "rinsing fluid" in the peritoneal cavity and the blood depends upon the concentration gradients between the two fluid compartments. Experiments on healthy animals and on animals rendered uraemic went on for some time. In 1923, Gantner first published a report on the use of peritoneal lavage as a therapeutic measure in man; 1.5 litres of physiological saline were used as a dialysing solution in a case of bilaterally ureteric obstruction, due to a carcinoma of the cervix uteri, with very promising results.

Two systems of peritoneal dialysis have been devised: the "intermittent" or "short-term", and the "continuous" systems. In the intermittent system a volume of fluid is introduced into the peritoneal cavity, left in for a variable time until an equilibrium of concentrations with the blood is established, and then is drained out. In

\* in receipt of a travel grant from the Royal University of Malta.

the continuous method, the fluid is made to run through as an uninterrupted stream continuously rinsing the peritoneal cavity. The former method was used by Gantner in 1923, and later by Abbott and Shea in 1946, and by Grollman *et al.* in 1951. The continuous method was used by Wegner in 1877, and later by Rosenak and Siwon in 1926, by Wear, Sisk and Trinkle in 1939, and by Frank, Seligman and Fine (1946 and 1948); but it has largely been abandoned because of the risk of infection and because of the large amounts of irrigation fluid needed.

A modification of the intermittent method is in use in the Renal Unit of the Cardiff Royal Infirmary (CRI). The fluid, obtainable as No. 61 and No. 62 Dialaflex solutions (Allen & Hanburys Ltd.), is available in clear, collapsible, plastic, litre containers. The No. 62 Dialaflex solution has a higher dextrose concentration which doubles its osmolarity thus making the removal of water from the patient's circulation possible when indicated. Two of either such packs, warmed to body temperature, are used for each dialysis cycle. 0.5g of potassium chloride can be added to each change when indicated and 500 i.u. heparin to each of the first four changes. The composition of each pack is shown in Table 1.

TABLE 1

DIALAFLEX 61			DIALAFLEX 62			
	<i>mEq/L</i>	<i>g/L</i>	<i>mOsm</i>	<i>mEq/L</i>	<i>g/L</i>	<i>mOsm</i>
Sodium	140.4	—	140.4	140.4	—	140.4
Calcium	3.6	—	1.8	3.6	—	1.8
Magnesium	1.5	—	0.75	1.5	—	0.75
Total Chloride	100.8	—	100.8	100.8	—	100.8
Lactate	44.6	—	44.6	44.6	—	44.6
Dextrose	—	13.6	75.5	—	63.6	353.3
TOTAL	—	—	364.37	1.26	—	1.26
Sod. metabisulphite (antioxid)	0.52	—	0.52	—	—	642.91

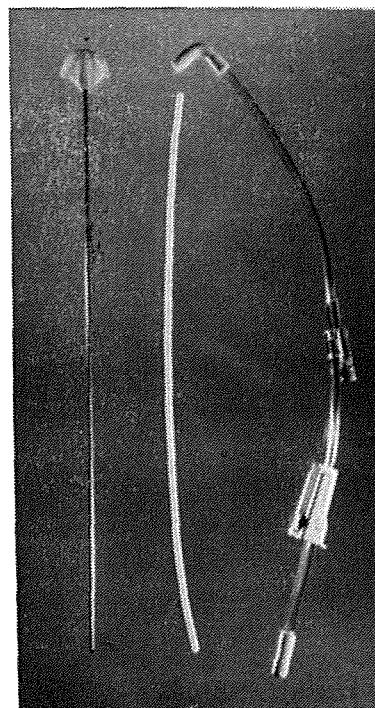
Automated peritoneal dialysis making use of the Mark 1 cycling machine produced by the Industrial Automation Division of Hawker Siddley Dynamics Ltd. of Coventry (Mallick, M. P. *et al.*, 1967), the "Cardiff" machine, is also used on some patients. The need for nursing supervision is reduced as is also the risk of infection since the machine is a closed system. The dialysing fluid used with the machine is prepared in 20 litre containers by diluting a concentrated electrolytic solution with sterile, pyrogen-free, distilled water. Higher flow rates (as much as 4 L/hr) can be achieved; thus automated peritoneal dialysis is a more rapid and efficient procedure. The machine also allows for "pauses" of the fluid inside the peritoneal cavity.

### Technique

If possible the procedure is first explained to the patient in simple terms. After sedation and catheterisation (if required), the patient is helped to lie flat in bed and the abdominal skin is shaved and cleaned carefully as before a major operation. With full aseptic and antiseptic precautions the puncture site is injected with 10cc of 2% lignocaine solution down to the level of the peritoneum. The cannula is threaded onto a stylet, and is then introduced through a small incision previously made. After piercing the peritoneum the stylet is withdrawn for 1 inch and the catheter is then advanced downwards towards the pelvic gutter.

The cannula is usually introduced in the midline below the umbilicus; at times above the umbilicus or in one of the iliac fossae. The cannula used at the CRI is the one prepared by the McGraw Laboratories of California, (Trade name: Trocath figure 1)).

Having inserted the catheter as far as it will go downwards without causing discomfort to the patient, the stylet is removed and placed in a container of antiseptic solution for possible further later use in adjusting the position of the catheter. The catheter is now connected via the connecting tube supplied with it (Fig. 1)



to the fluid bags through an administration set (the one used is Flexitron R. 61 produced by Baxter Labs. of Illinois). Sterile disposable connecting tubing is attached to the other end of the Y-tube to drain the fluid out of the peritoneal cavity into a measuring cylinder. The catheter is now trimmed to about 1 inch above the level of the skin and a purse-string suture inserted round the point of entry of the cannula. The inflow and outflow paths are now tested making use of about 200cc of dialysing fluid; if all is satisfactory the dialysing solution is then introduced into the peritoneum (fig. 2).

Some difficulties may be experienced during the introduction of the cannula. Oozing at the site of entry through the skin is usually well-controlled by pressure. In patients with multiple operative scars and in very obese patients it is relatively easy to introduce the cannula in the plane between the parietal peritoneum and the overlying adipose tissue; in such cases the entry of the stylet is facilitated if the peritoneum had been incised prior to the entry of the stylet. Perforation of the abdominal and pelvic viscera has been described in patients with a history of abdominal ope-

rations, in puerperal patients and with a subinvolved uterus, and in patients with severe ileus.

### Indications for P.D.

1. Uraemia with prominent neuromuscular, gastro-intestinal and cardiovascular changes due to acute or acute-on chronic renal insufficiency. In the 127 peritoneal dialysis performed at the CRI during the early part of 1967, 32.2% had disturbances of consciousness and 11.3% had other CNS disturbances, 52.4% had gastro-intestinal upsets, 10.5% had pericarditis, 90.0% were anaemic and 9% had a bleeding tendency prior to the commencement of the dialyses. In cases of renal insufficiency, the need to dialyse depends more on the condition of the patient, on the rate of rise of blood urea and other forms of deterioration of blood chemistry, than on any one single and specific criterion (Merrill, 1965). However, in hyperkeratobolic renal insufficiency when the blood urea rises by more than 50mgm/100ml/day peritoneal dialysis is not usually effective.

2. Fluid overload especially if associated with intractable pulmonary oedema. In such cases Dialaflex Solution 62 is needed to remove the excess of fluid in the patient's circulation. The patient should be carefully weighed several times during the period of dialysis in order to assess its effectiveness.

3. Hyperkalaemia (Maxwell *et al.*, 1959), if it cannot be effectively controlled by glucose and insulin, cation exchange resin and the other more orthodox conservative methods.

#### 4. Certain intoxications

a. acute barbiturate poisoning (Maxwell *et al.*, 1959) if conservative therapeutic mea-

sures have not proved effective.

- b. salicylate intoxication. Peritoneal dialysis has been shown to be very effective in animals poisoned by salicylates using a 5% albumen solution as the rising fluid (Eteldorf *et al.* 1960).
- c. acute methyl alcohol poisoning (Stinebaugh, 1960).

### Illustrative Cases

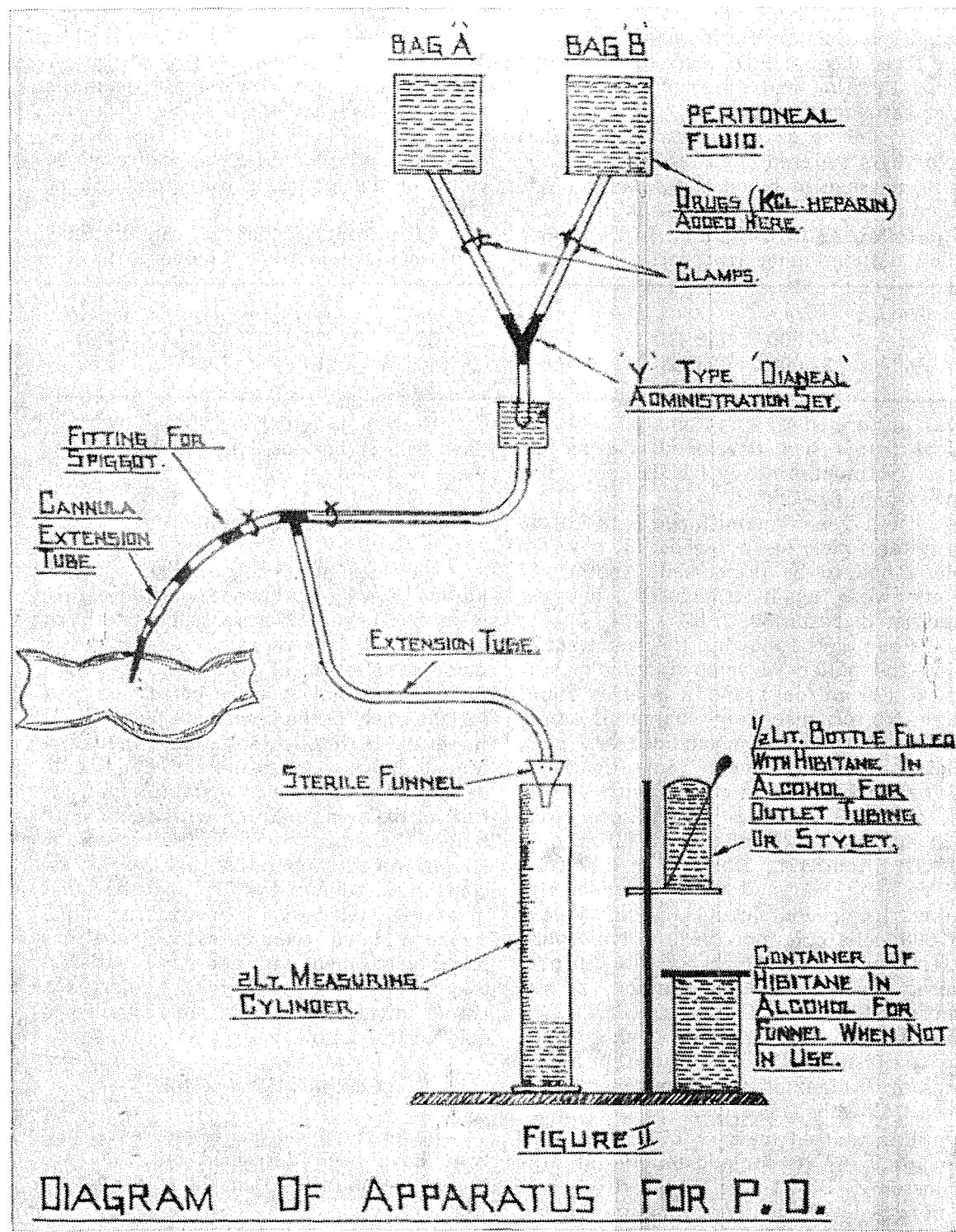
1. Mrs. P.W. 42 years, a chronic renal patient. First admission: July 1963, presenting with bilateral, staghorn, renal calculi, very severe pyelonephritis, oliguria and severe renal insufficiency. Since then, numerous hospital admissions for exacerbations of the pyelonephritis and deterioration of the renal insufficiency. Last admission: 3. 9. 67 with a ten-day history of generalised asthenia and malaise, frequency, nocturia and dysuria, no haematuria. No response to nitrofurantoin therapy. In the three days prior to admission more pronounced urinary symptoms, oliguria, severe drowsiness, confusion, heavy hissing respiration, backache, nausea and vomiting. Examination on admission: semicomatose, responding only to violent stimulation with moaning and groaning. Kussmaul respiration. P: 100/min; BP 110/70; Temp. 35°C. Investigations: Hb: 7.2g%; PCV 25; WBC: 14,300 cells /c.mm; Bl. urea: 350 mg %; Na<sup>++</sup>: 143mEq/L; K<sup>+</sup>: 6.6 mEq/L; HCO<sub>3</sub>: 5 mEq/L; pH central venous blood: 7.17; pCO<sub>2</sub>: 13.

The K<sup>+</sup> level fell following the administration of glucose and insulin.  $\frac{1}{2}$ L of 5% Sodium Bicarb. were also given i/v prior to the commencement of P.D. at 10.45 p.m. on 3. 9. 67.

After the first twelve hours of P.D.

TABLE II

Date	In lit.	Out lit.	Pre. urea	Post urea	Hrs.	Changes	Rate/Hr
3.9.67	2.100	1.720	350	200	1.25	1	1.68
4.9.67	35.700	34.680	200	111	24.00	17	1.49
5.9.67	33.600	35.650	111	95	19.50	16	1.72
	71.400	72.050	—	—	44.75	34	—



the patient's general condition was much better — less hyperpnoea and confusion, urinary output on the increase. Following the three days of P.D., state of health and renal function had returned to their former satisfactory level.

2. Mr. R.R. 60 years, for the last two years suffered from episodes of L.I.F. pain, passage of blood and slime per rectum, and pyrexia. Diagnosed as diverticulitis following investigation. In December, 1966 defunctioning transverse colostomy

patient suffering from renal insufficiency due to a polycystic disease of the kidneys. Last admission to the Renal Unit CRI on 12. 8. 67 with severe ascites, severe pitting oedema of the lower limbs, genitalia, and sacral pad, and oliguria. Investigations: blood urea: 242 mg %;  $\text{Na}^+$ : 124 mEq/L;  $\text{s.K}^+$ : 5.3 mEq/L; P.D. was indicated because of the excessive water and salt retention.

On introduction of the cannula 3.000 L of ascitic fluid were removed.

Dialysis	In	Out	Pre-urea	Post urea	Changes	Hrs.	Rate/Hr
1st	46.200	48.410	242	168	22	25.5	1.81
2nd	21.000	25.720	168	146	10	13.3	1.58

was performed as symptoms of a colovesical fistula had developed. During the last few months heavy bleeds per rectum every 5 to 6 weeks.

On 17. 6. 67 admittance to a Cardiff hospital following a bleed; 11 pints of blood had to be transfused. Another 11 pints were again transfused following another bleed on 30. 7. 67.

Four days following the last transfusion the patient developed jaundice, oliguria (20 ml/day) and a soaring blood urea. No response to i/v mannitol. Admitted to CRI: 6. 8. 67 jaundiced and completely disorientated. Investigations: Urine: dark brown in colour, with a deposit of RBCs and pus cells. Blood urea: 320 mg%;  $\text{Na}^+$  139 mEq/L;  $\text{K}^+$  4.9 mEq/L;  $\text{HCO}_3$ : 15 mEq/L;  $\text{Hb}$ : 16.7 g/l (113%); PCV: 51%; RBCs: 5.1 millions/cub mm; Retics: 4%; Platelets: 115,000; WBCs: 16,000 cells/cub mm (96% polymorphs); s. total proteins: 5.6 g/L; s. alb: 3.0 g/L; raised gamma globulin fraction; s. alk. phos: 7.9 K.A. units%; total s. bilirubin: 3.1 (direct 2.4), thymol turb: 1 unit; blood group: 0 Rh; CDE/cde Anti-Kell bodies present (saline, albumen, enzymes, iCT); Direct Coombs: negative.

P.D. started on 6. 8. 67 and stopped on 10. 9. 67 (uncomplicated despite the colostomy). Blood urea then had fallen to a normal level and renal function had returned to normal with a urinary output of more than 1 L per 24 hours.

3. Mr. H.Y. 58 years, a chronic renal

In these two dialyses spread over two days 6.93 L (besides 3.3 L of ascitic fluid) were removed.

### Contra-indications

No absolute contra-indications are known. Age is no barrier; peritoneal dialyses has been performed successfully on an infant 3 days of age. Pregnancy may give rise to technical difficulties; peritoneal dialysis can, however, be performed very satisfactorily in the immediate puerperium. In severe peritonitis, some hold that peritoneal dialyses is absolutely contra-indicated; others however maintain that the flow of dialysing solution with added antibiotics helps to clear up the infection. Recent surgical operation, as well as, recent intra-abdominal injury, may be a bar to operational dialysis. In my brief experience I have seen peritoneal dialyses being performed with its usual satisfactory results, on a patient with a colostomy, active diverticulitis and an established colo-vesical fistula.

### Complications of P.D.

Some complications of peritoneal dialysis have been described but they are the exception rather than the rule (Doolan, 1959).

1. Peritonitis with the later formation of adhesions. The risk is light — in only 17 out of the 127 CRI dialyses being

quoted did infection of the peritoneum supervene during the dialysis. Only 1 of these patients had clinical peritonitis although the cultures of the effluent fluid, routinely made, were positive. Regular bacteriological examinations of the peritoneal outflow and the appropriate use of effective antibiotics both systematically and into the dialysing solutions lowers further the incidence.

2. *Pain or abdominal discomfort.* This usually occurs after a prolonged dialysis and is ingravescent. Pain can be referred to the lower part of the chest-wall and to the shoulder-tip, and is exacerbated by moving, coughing and deep breathing. Its occurrence is related to the volume of dialysing fluid introduced into the peritoneal cavity. Pain only occurred in 13 of the dialyses and more commonly during the introduction of fluid.

3. *Pulmonary complications* (Berlyne *et al.*, 1966). The chest complications of peritoneal dialyses that have been described are pneumonia, atelactasis, purulent bronchitis and pleural effusion. These probably arise from partial collapse of the lower pulmonary lobes due to elevation of the diaphragm caused by overdistension of the peritoneal cavity by the dialysing solution. It has, however, been thought that anatomical communications between the pleural and the peritoneal cavities might allow dialysing fluid to pass into the chest. The uraemic patient is prone to chest infection because of his immunity insompetence and the likelihood of aspiration of bronchial secretion due to inhibition of coughing by pain and disturbances of consciousness. It has, however been shown (M. Poppen and U. von Sydow, 1966), that these complications are greatly reduced by prophylactic breathing exercises started concomitantly with the dialysis. At the CRI breathing exercises are encouraged by an expert physiotherapist, routinely, in all patients undergoing dialysis; the incidence of chest complications is negligible.

4. *Cardiac arrhythmias.* Such arrhythmias are probably due to movement of water and electrolytes between the cells and the extracellular fluid. Adequate con-

trol of the electrolytic status of the patient is therefore essential during peritoneal dialysis. In only 6 dialyses from the CRI series quoted did cardiac irregularities develop.

5. *Losses of protein and amino-acids.* This has proved to be the most important complication of peritoneal dialysis. It has been noted and studied by a number of workers; Frethien and Selvaag in 1948, Bcen 1961 and Burns *et al.* in 1962. Losses of amino-acids have also recently been proved by Berlyne *et al.* (1967). Figures for protein losses vary from no protein loss to losses of 0.2-8.0 g/L. The total loss of protein may be large, thus in a dialysis of 30 cycles, of one litre exchange containing 5 g of protein per litre, the loss would be that of 150 g of protein during the one dialysis. As well as albumen various plasma globulin fractions are lost, in particular gamma globulin in large amounts, leading to further deterioration in the immunological competence of the patient with renal insufficiency (Berlyne *et al.*, 1967).

6. *Overhydration and dehydration.* Regular estimations of arterial blood pressure, careful observations of the jugular venous pressure, and thorough, repeated, physical examinations of the patient should serve as an adequate safeguard against this complication. It is also important that the patient be weighed accurately at frequent intervals. The tonicity of the dialising fluid should be regulated in the light of these observations by the use of 1.36% and 6.36% concentrations of glucose in the dialysing solutions.

7. *Hyperkalaemia and Hypokalaemia.* Regular estimations of the serum potassium and electrocardiograms should help prevent these complications.

8. *Hypocalcaemia.* The irrigation solution should contain at least 3 mEq/L of calcium otherwise hypocalcaemia may result. The Dialaflex solutions used contain adequate amounts of  $\text{Ca}^{++}$ .

Briefly the success of peritoneal dialysis depends on meticulous control and monitoring by both the medical and the

nursing staff. The following are some points of special clinical importance:

1. Blood pressure reading (one hourly or two hourly B.P. Chart)
2. Pulse (note especially rate and rhythm)
3. Auscultation of the heart (arrhythmias, gallop rhythm)
4. Auscultation of the lung fields (Basal crepitations)
5. Central venous pressure observations
6. Very efficient fluid balance charts
7. Careful weighing of the patient.

### Summary

A brief history of the origin of peritoneal dialysis has been given. The variations on the "intermittent" technique of peritoneal dialysis used on the Renal Unit, Cardiff Royal Infirmary have been described. The ever-increasing importance of peritoneal dialysis as a therapeutic tool has been outlined, and illustrative cases from personal experience at the CRI quoted. Possible complications of peritoneal lavage have been discussed.

### Acknowledgements

I wish to thank Professor H. Scarborough and Dr. G. Coles for all their interest and help and Professor G. P. Xuereb for his advice and criticism.

### References

ABBOTT, A.M. and SHEA, C.L. (1946). Amer. J. Med. Sci. 312.

BERLYNE, G.M. *et al.* (1966). Lancet, *ii*, 75.

BERLYNE, G.M. *et al.* (1967). Lancet, *i*, 738.

BERLYNE, G.M. *et al.* (1967). Lancet, *i*, 1337.

BOEN, S.T. (1961). Peritoneal Dialysis in Clinical Medicine.

Doolan, P.D. (1959). Amer. J. Med. 26, 831.

ETTELDORF, J.N. *et al.* (1960). J. Ped. 56, 1.

FRANK, H.A., SELIGMAN, A.M. and FINE, J. (1946). J. Amer. med. Ass., 130, 703.

FRANK, H.A., SELIGMAN, A.M. and FINE, J. (1946). J. Clin. Investig., 25, 211.

MALLICK, M.P. *et al.* (1967). Lancet, *i*, 1303.

MAXWELL, L.H. *et al.* (1959). J. Amer. med. Ass. 170, 917.

MERRILL, J.P. (1965). The Treatment of Renal Failure, 2nd edit.

POPPEN, M. and von SYDOW, U. (1966). Lancet, *ii*, 753.

STINEBAUGH, B.J. (1960). Arch. Int. Med. 105, 613.

WEAR, S. *et al.* (1938). J. Urol., 39, 53.