visits to the clinic and were unable to attend for a specimen to be collected and examined by routine methods. The response of established infections to chemotherapy could also be followed on an outpatient basis, slides being inoculated on several consecutive days after starting treatment.

Discussion

The dip-slide is based on Mackey and Sandys's idea of a dip-inoculum transport medium. It is made from easily available materials and is cheap. It has two further advantages. Firstly, two media are inoculated simultaneously. This helps considerably in distinguishing between pure cultures of pathogenic organisms and mixed growths due to contamination. Secondly, a larger surface area of medium is available for inoculation. This makes colony counting easier, and the transparency of the preparation is also helpful in this respect. Up to 500 colonies can be easily separated on one side of the dip-slide, the growth becoming confluent only with counts approaching 1,000 colonies. Our clinical trial shows that all specimens containing more than 10^5 organisms per ml. gave dip-slide counts of 200 or more colonies, and specimens with more than 10^4 organisms per ml. produced counts above 40 colonies. Colony counts between 40 and 200 are particularly convenient to read.

We suggest that dip-slide colony counts between 100 and 500 should be regarded as doubtful and an indication for further investigation of the patient. In our series there were 45 specimens in this range, of which eight were considered to be truly infected. It must be remembered that our patients were highly selected, and it is probable that in a random population the incidence of doubtful results would be smaller.

The dip-slide has been in clinical use for over one year. In general practice it was found to overcome difficulties in bacteriological diagnosis which may arise when there is an unavoidable delay between collection of the specimen and its delivery to the laboratory. The ease of inoculation makes it useful in screening clinics where large numbers of specimens have to be processed. With a little experience the majority of dip-slides may be assigned to significantly or non-significantly infected groups by inspection without counting colonies. The quantitative accuracy of the method is greater than that obtained by standard-size loop inoculation methods; on the other hand, the identification of bacterial species and subculturing are easier than from a pour plate. In our series, which included 60

specimens shown to be infected by conventional methods, the dip-slide yielded no false-negative results, and false-positive results were also not a problem.

We regard the dip-slide as an adjunct to conventional methods of urine examination. It does not obviate the need for microscopy of the deposit, tests for protein, or cultures for the identification of the infecting organism. We do not suggest its introduction for routine use where specimens can be delivered to the laboratory quickly after collection. It may, however, be conveniently used as the only screening method in surveys such as at antenatal clinics. Patients from whom specimens are obtained with dip-slide colony counts in the infected or doubtful range would then be recalled for further investigation by routine methods.

Summary

A dip-inoculum transport medium for the diagnosis of significant bacteriuria is described, consisting of a microscope slide coated with culture media. Laboratory and clinical testing shows the dip-slide to give consistent quantitative results which compare well with pour-plate viable counting.

The dip-slide enables ready differentiation between heavy contamination of urine specimens and genuine infection. In a series of 385 specimens no false-negative results were obtained, and there was an insignificant number of false-positive cultures.

The dip-slide is recommended whenever there is likely to be delay in delivery of urine specimens to the laboratory. It is particularly useful in general practice if specimens have to be sent by post. Owing to the ease of inoculation and reading it is also suited to the routine screening of large numbers of specimens such as at antenatal clinics.

We wish to thank Miss I. J. Barker, Miss V. A. Lane, and Miss S. E. Minns for technical assistance, and the outpatient nursing staff of Addenbrooke's Hospital for their skill in obtaining satisfactory urine specimens. One of us (D.G.) is grateful to Dr. M. H. Gleeson-White for provision of laboratory facilities.

REFERENCES

- Cattell, W. R., and Lefford, M. J. (1963). *Brit. med. J.*, 1, 97.
Guttman, D., and Stokes, E. J. (1963). *Ibid.*, 1, 1384.
Houghton, B. J., and Pears, M. A. (1957). *Ibid.*, 1, 622.
Kass, E. H. (1956). *Trans. Ass. Amer. Physns*, 69, 56.
Leigh, D. A., and Williams, J. D. (1964). *J. clin. Path.*, 17, 498.
Mackey, J. P., and Sandys, G. H. (1965). *Brit. med. J.*, 2, 1286.
— (1966). *Ibid.*, 1, 1173.
Naylor, G. R. E., and Guttman, D. (1967). *J. Hyg. (Camb.)*, 65. In press.

Sodium Uptake in Peritoneal Dialysis

J. D. SWALES,* M.B., M.R.C.P.

Brit. med. J., 1967, 3, 345-347

Peritoneal dialysis has come to occupy a prominent place in the treatment of renal disease, and has, indeed, largely replaced haemodialysis in some centres (Cameron, 1966). Pulmonary complications frequently occur, though in many cases they consist of asymptomatic segmental collapse (Berlyne *et al.*, 1966). An occasional, and more often fatal, complication is acute heart failure. In a recent series of 91 patients this complication accounted for two deaths (Stewart *et al.*, 1966).

When dialysis fluid cannot be drained from the peritoneal cavity, fluid overload and heart failure are easily accounted for in terms of the slow absorption of dialysis fluid. However,

even where the patient is not in positive fluid balance, heart failure may occur during the course of peritoneal dialysis (see Case Report below). The purpose of this paper is to present data on sodium absorption during 13 consecutive peritoneal dialyses, to suggest that it is the uptake of sodium in hyponatraemic patients which is responsible for the development of heart failure, and to suggest how this potentially fatal complication may be avoided.

Case Report

A 14-year-old schoolboy gave a history of lifelong nocturia. For five years he had had polydipsia and polyuria with recurrent dysuria. In March 1965 he was found to have a blood urea of 450 mg./

* Registrar, Medical Professorial Unit, Westminster Hospital, London S.W.1

100 ml., and serum sodium 123, potassium 6.5, and bicarbonate 12 mEq/l. Treatment by continuous bladder drainage and sodium supplements produced a marked improvement and his urea fell to 70 mg./100 ml. He continued as an outpatient, taking 2 g. of sodium chloride and 2 g. of sodium bicarbonate daily. Six months later he was readmitted with a week's history of vomiting. Apart from slight drowsiness there were no abnormal physical signs. His blood pressure was 110/70 mm. Hg. The blood urea was 404 mg./100 ml., serum sodium 125 mEq/l., and serum potassium 4.6 mEq/l. His sodium supplements were stopped, and peritoneal dialysis was started. The dialysis was technically satisfactory in that after eight exchanges he was in a negative fluid balance of 400 ml. However, after 12 hours he became slightly hypertensive (135/95 mm. Hg) and developed an apical gallop rhythm (III sound). At the beginning of the ninth exchange, 22 hours after starting dialysis, he suddenly became acutely dyspnoeic and cyanosed, with fine crepitations over both lung fields. He showed no response to aminophylline, digoxin, and oxygen therapy, and died

FIG. 1.—Sodium concentration simultaneously measured in serum and peritoneal dialysis fluid at start of peritoneal dialysis.

20 minutes later. Post-mortem examination showed severe pulmonary oedema with bilaterally shrunken pyelonephritic kidneys containing numerous small cysts.

Comment.—This patient developed a dramatic and fatal pulmonary oedema, though fluid had actually been withdrawn. It could be postulated that significant amounts of sodium were

moving along the concentration gradient between the peritoneal dialysis fluid (141 mEq/l.) and his serum (125 mEq/l.), and that this caused the acute pulmonary oedema. No quantitative study has hitherto been made of sodium movements during peritoneal dialysis in relation to the serum sodium concentration. In order to ascertain whether such an uptake of sodium occurs the following study was carried out.

Method

Peritoneal dialysis at this hospital is performed by running in a solution of dextrose (1.36 or 6.36 g./100 ml.) containing the following ions: sodium 141, calcium 3.6, magnesium 1.5, chloride 100.8, bicarbonate (as lactate) 44.6 mEq/l. Generally two such disposable plastic bags are used (Allen and Hanburys). The fluid is warmed to 37° C. and run into the peritoneal cavity through a subumbilical catheter as quickly as possible—usually about 10 minutes. After 45 minutes the fluid is drained out through the same catheter and along a side arm attached to the giving set, which leads to a Winchester bottle beneath the patient's bed. A pressure of -5 mm. of mercury is applied to this bottle by means of a Roberts pump. As much fluid as possible is removed, and then two more bags of fluid are run in. The dialysis in this series continued for 18 to 72 hours. During this period 20 to 35 exchanges were possible.

For this study 13 consecutive dialyses on seven uraemic patients were observed. All the patients were either anuric or severely oliguric and remained so during the period of study. A record was obtained of the type and volume of fluid drained. An aliquot was taken from the dialysis fluid returned from each exchange, and the sodium concentration estimated by means of a flame photometer. Daily serum sodium measurements were also made on the patients.

Results

The serum sodium concentration was low initially in most patients, but generally rose during peritoneal dialysis. The sodium concentration of the peritoneal dialysis fluid which drained from the patient was proportional to the serum sodium concentration, though the dialysis fluid sodium tended to be higher (Fig. 1). This suggests incomplete equilibration between the two fluids.

FIG. 2

FIG. 3

FIG. 4

FIG. 2.—Sodium balance in two patients from whom only small amounts of fluid were removed (0–150 ml. per exchange). FIG. 3.—Sodium balance in five patients from whom 150–300 ml. per exchange was removed. FIG. 4.—Sodium balance in four patients from whom large amounts of fluid were removed (300–450 ml. per exchange).

Four patients developed a positive sodium balance during peritoneal dialysis. In one patient this had reverted to a negative balance by the end of the dialysis. The nature of the sodium balance depended partly on the amount of fluid which was removed (Figs. 2–4) and partly on the initial serum sodium level. Where only a small amount of fluid was removed from a patient with a low serum sodium, a positive sodium balance developed. Two patients are omitted from Figs. 2–4. In one a positive fluid balance occurred because of inadequate drainage, and this was responsible for the sodium uptake of 453 mEq, and in the other fluid was removed more rapidly than in the rest of the series (450 ml. per exchange), with a rapidly developing negative sodium balance in excess of 400 mEq.

Discussion

Urea and electrolytes diffuse along the concentration gradient between fluid within the peritoneal cavity and the serum (Boen, 1961). This process continues until equilibrium is reached after 90 to 120 minutes. During this period the difference in osmolalities determines the volume of fluid absorbed (Shear *et al.*, 1965). The latter workers have shown that subsequently a phase of isotonic absorption occurs when 29.7–39.3 ml. of fluid is absorbed per hour. This latter phase is usually unimportant in peritoneal dialysis, where fluid is not allowed to remain in the peritoneal cavity longer than 45 minutes.

The present study was undertaken to quantify the sodium transfer during the initial period of equilibrium and to determine whether such movement would be physiologically important. The results suggest that significant amounts of sodium are absorbed when a hyponatraemic patient undergoes peritoneal dialysis in which only small amounts of fluid are removed. This may account for the occasional episode of pulmonary oedema during peritoneal dialysis which, as in the case described, may be fatal.

How can this complication be avoided? One answer would be to lower the sodium concentration of the fluid administered. This would necessitate the preparation of several types of dialysis fluid, containing different concentrations of sodium, making the management of peritoneal dialysis a much more complicated operation. Alternatively, one could reproduce the situation seen in the majority of cases in this series, and withdraw large volumes of fluid from the patient by means of the dialysis. The present series shows that this keeps the patient in negative sodium balance.

Hypertonic dialysis fluid may remove greater quantities of urea than isotonic fluid. This is not merely due to the increased amount of fluid withdrawn, but is also partly attributable to the phenomenon known as “solvent drag.” When a large volume of fluid passes through a porous membrane, solute tends to move with it, even against a concentration gradient. It has been postulated that solute particles (in this case urea) are entrained in the solvent stream (Henderson, 1966). In order to determine whether sodium could be withdrawn in such a way by using a hypertonic fluid, peritoneal dialysis fluid containing 6.36 g. of glucose per 100 ml. was administered intermittently and the sodium concentration after each such exchange measured. This failed to produce any increase in the sodium concentration of the dialysis fluid removed (see Table).

Effect of Varying Glucose Concentration on Sodium Concentration of Fluid Drained from a Peritoneal Dialysis

Dialysis fluid glucose (g./100 ml.)	1.36	1.36	6.36	6.36	1.35
Sodium concentration of effluent (mEq/l.)	125	126	123	122	125

Nevertheless, the present series of cases indicates that if a patient on peritoneal dialysis is kept in a negative fluid balance of several litres there is little danger of excessive sodium absorption.

Summary

In hyponatraemic patients there is a marked gradient between concentration of sodium in the peritoneal dialysis fluid and the serum. In order to establish whether this causes a positive sodium balance the sodium uptake was measured during dialysis. In 4 out of 13 dialyses there was a positive sodium balance, in one case reaching 400 mEq, despite a negative fluid balance in three of these cases. It is suggested that this may be responsible for acute heart failure during dialysis.

REFERENCES

- Berlyne, G. M., Lee, H. A., Ralston, A. J., and Woolcock, J. A. (1966). *Lancet*, 2, 75.
 Boen, S. T. (1961). *Medicine (Baltimore)*, 40, 243.
 Cameron, J. S. (1966). *Brit. med. J.*, 2, 811.
 Henderson, L. W. (1966). *J. clin. Invest.*, 45, 950.
 Shear, L., Swartz, C., Shinaberger, J. A., and Barry, K. G. (1965). *New Engl. J. Med.*, 272, 123.
 Stewart, J. H., Tuckwell, L. A., Sinnett, P. F., Edwards, K. D. G., and Whyte, H. M. (1966). *Quart. J. Med.*, 35, 407.

Thalassaemia and Hydrops Foetalis—Family Studies

DAVID TODD,* M.D., F.R.C.P.ED.; MICHAEL LAI,† B.Sc.; CAROLINA A. BRAGA,‡ M.A., M.B., B.S., M.R.C.O.G.

Brit. med. J., 1967, 3, 347–349

Stillbirth or neonatal death associated with severe hydrops foetalis, hypochromic anaemia, erythroblastosis, and large amounts of haemoglobin Bart's was first described by Lie-Injo and Jo (1960). In subsequent studies Lie-Injo (1962) and Lie-Injo, Lie, Ager, and Lehmann (1962) reported that the parents of such infants showed haematological changes consistent with α -thalassaemia trait, and they thought that the

hydropic babies were homozygous for α -thalassaemia. Reports by Diamond, Cotgrove, and Parker (1965) and Pootrakul, Wasi, and Na-Nakorn (1966) lend support to this view.

In Hong Kong haematological studies have been made on the families of seven Chinese babies with severe hydrops foetalis associated with over 75% of haemoglobin Bart's, and, while there was evidence that five were homozygous for α -thalassaemia, in two a possible interaction between α - and β -thalassaemia was suggested. The purpose of this communication is to report these studies.

* Senior Lecturer, Department of Medicine, University of Hong Kong.
 † Demonstrator, Department of Biochemistry, University of Hong Kong.
 ‡ Senior Lecturer, Department of Obstetrics and Gynaecology, University of Hong Kong