CLINICAL MANAGEMENT OF ACUTE URINARY SUPPRESSION

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SYNOPSIS

It may occasionally be difficult to differentiate acute oliguria of pre-renal origin from the renal variety. Although the history can help, it may, occasionally be unavailable, or even unreliable or misleading in some cases. In our series of 13 patients, we find the ratio of urinary urea/blood urea to be useful. A ratio of above 8 designates pre-renal origin and a ratio below 5 designates renal origin. Intravenous injections of either Frusemide or Mannitol could further differentiate acute functional tubular insufficiency from established acute organic tubular necrosis. The importance of central venous pressure monitoring is discussed and a simplified plan of approach to acute oliguria is presented.

INTRODUCTION

Acute urinary suppression or oliguria is a common medical condition. By definition, oliguria is a state in which urinary flow is less than 20 ml. per hour or 400 ml. per 24 hours. Although oliguria is a cardinal feature in most reported cases of acute renal failure, the two terms are not necessarily synonymous since reports have appeared in the literature of non-oliguric acute renal failure (Vertel *et al*, 1967).

When confronted with acute oliguria, one must determine whether the cause is one of the following:—

1. Hypoperfusion of the kidney causing impaired renal function without parenchymatous damage. This is sometimes referred to as "pre-renal uraemia" and is usually due to shock or severe dehydration. If renal hypoperfusion is severe or long enough, acute tubular necrosis may result.
2. A variety of renal parenchymatous diseases including the acute glomerulopathies and established acute tubular necrosis. This is designated acute parenchymatous renal failure.
3. Acute obstruction of the urinary tract or post-renal oliguria.

A careful history and physical examination remains the basis of differentiation. It is generally agreed that measurement of specific gravity is unreliable on two accounts—first, because of the effect of proteinuria and second, very often these patients do not produce sufficient amount of urine for the test to be carried out. A number of authors have used urine-urea and blood-urea ratios (Perlmutter *et al*, 1959), sodium content of the urine (Handa *et* Marrin, 1967), and sodium and potassium ratio of the urine (Meroney *et* Rubini, 1959) as basis of differentiation. We have reinvestigated the various aspects of the problem and attempt to establish a simple procedure that would be applicable in the ward. Post-renal oliguria is not discussed here as it is essentially a surgical problem.

MATERIALS

13 patients were selected for this study. They were by no means all the patients with acute oliguria. All these patients have normal renal function prior to the onset of illness as far as one can ascertain. Selection for this study required the following criteria: first, the patients were categorised clinically into pre-renal and renal failure as stated in Para 2 or at necropsy; second, an in dwelling urinary catheter with closed drainage system was established and the urine obtained was estimated for urea and electrolytes. Hourly urinary output was noted. All these patients were oliguric from 24-72 hours and none of them had urine output of more than 10 ml. per hour.

METHOD

Samples of blood for urea and electrolytes were obtained from patients on admission. Catheterized urine specimens were obtained for estimation of urea and electrolytes. Urea was estimated according to method of Van Slyke *et al* (1914) and electrolytes were estimated by flame photometry using an EEL model (Varley, 1964) with minor modifications. Thereafter urinary output was monitored hourly. All patients were given intravenous fluids in an attempt to replace any

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fluid deficit that may be present. Central venous pressure was monitored in cases where doubt exists as to optimal fluid replacement. The value of this simple yet important procedure has been well established by a number of authors (Carruthers *et* Parsons, 1967; Cohn, 1967). When no response was obtained after adequate hydration, 40-80 mgm. of Frusemide was given intravenously. When this failed to establish adequate urinary output, 100 ml. of 25% mannitol were given intravenously over a period of 10 minutes. Other forms of therapy included aramine, effortil, hydrocortisone, antibiotics and peritoneal dialysis. A summary of the clinical features, biochemical data, treatment regime and response to treatment are presented in Table I(*A*) and (*B*).

RESULTS

Pre-renal Oliguria

The majority of this group belong to what is known as "shocked kidney." The urine-urea/blood-urea ratios varied between 8-20. This high ratio is in accordance with most published series (Perlmutter *et al*, 1959). The urinary sodium was low, less than 50 mEq./L. The urinary sodium/potassium ratio however was variable. Majority of these patients failed to respond to pure intravenous fluid replacement. This phenomenon is difficult to explain since by definition no parenchymatous renal failure was present at the start. We have no doubt however that the use of diuretics has at least hastened the onset of adequate urinary flow. Two patients died in this group. The mortality rate of this group is dependent on the underlying disease rather than on the renal dysfunction *per se*. Balsov and Jorgensen (1963) found that the mortality in this group rises from 3% in patients with slight or curable underlying disease to 80% in patients with severe, complicated underlying disease.

Renal Oliguria

The urine-urea/blood-urea ratios varied between 0·5-5. This again is in accordance with most published series. Almost all the patients had a high urinary sodium content (more than 60 mEq./L). and a high urinary sodium/potassium ratio. None of these patients responded to intravenous fluids or frusemide. 3 of the patients responded to mannitol infusion and may be regarded as having incipient acute tubular necrosis (Eliahou, 1964). 4 of the patients required peritoneal dialysis with two survival.

DISCUSSION AND CONCLUSION

Although our series is small, a number of conclusions can be drawn together with the experience of others. A careful history remains the best method of differentiating the various types of oliguria. Of the various indices of acute renal failure, urinary-urea/blood-urea ratio appears to be superior in our hands. This is at variance with the view of Chisolm *et al* (1966) who reported that measurements of the urine-urea/blood-urea ratio is of very limited clinical value. Mannitol infusion remains the method of choice in establishing adequate urinary flow in the incipient acute renal failure group (Barry *et* Malloy, 1962; Luke *et al*, 1965). Although it has been described as a double-edged weapon, mannitol is safe except in cases of obvious cardiac failure and hypervolaemia when pulmonary oedema can be precipitated. Damage to red cells has also been reported with mannitol (Roberts *et* Smith, 1966). It can also cause hypovolaemic shock in the presence of inadequate fluid replacement. Central venous pressure should be monitored in doubtful cases.

A SIMPLIFIED PLAN OF APPROACH

In conclusion we would like to suggest the following steps for physicians who are confronted with the problem of acute urinary suppression.

1. An adequate history and physical examination. In doubtful cases urine-urea/blood-urea ratio could help in differentiating pre-renal from renal oliguria.
2. Adequate fluid replacement. This may necessitate central venous pressure monitoring.
3. Maintenance of adequate blood pressure. This is in fact the most difficult problem facing those who deal with acute renal failure. Vaso-constrictors have more or less been abandoned since they further compromise renal blood flow. Isoprenaline and dibenzyline appear to be drugs of choice (Hardaway III, 1969).
4. A test infusion of 100 ml. of 25% mannitol given over a period of 10 minutes could differentiate between functional acute tubular insufficiency and established acute renal failure.
5. Once irreversible failure has been established, appropriate measures like peritoneal dialysis or haemodialysis should be instituted.
6. Appropriate anti-biotics are required in septic cases.
7. Hourly monitoring of urinary output by means of an indwelling catheter and close drainage system is essential.

TABLE I

DETAILS OF OLIGURIC PATIENTS DIVIDED INTO PRE-RENAL AND RENAL CAUSES

Case No.	Clinical Diagnosis	Urine Urea mgm.%	Blood Urea mgm.%	$\frac{U}{B}$ Urea	Urine Sodium mEq./L	Urine Potassium Emq./L	$U\frac{Na}{K}$	Fluid Therapy ml.	Frusemide i/v mgm.	Mannitol 25% i/v ml.	Others	Results
A	OLIGURIA OF PRE-RENAL ORIGIN											
1	Pyopneumothorax and dehydration	1,262	68	19	9	84	0·10	1,000	80	100	CVP, aramine	Brisk diuresis Improved
2	Septic shock	715	72	10	48	56	1·20	4,900	80	100	CVP, aramine, hydrocortisone, isoprenaline	No response Died
3	Nephrotic syndrome with gastro-enteritis	835	103	8	9	32	0·30	2,000	80	—	—	Brisk diuresis Improved
4	Hyperosmolar non-ketotic diabetic coma	1,468	118	12	20	68	0·30	3,500	80	—	—	Brisk diuresis Improved
5	Chest infection, vomiting and dehydration	2,700	90	30	3	40	0·07	3,000	—	—	—	Brisk diuresis Improved
6	Diabetes mellitus, gastro-enteritis, severe dehydration, shock	798	63	11	22	57	2·60	3,050	80	100	CVP, hydrocortisone, Effortil	No response Died
B	OLIGURIA OF RENAL ORIGIN											
1	Lead poisoning	300	60	5·0	90	7·3	12·0	2,400	40	100	—	Brisk diuresis Improved
2	Leptospirosis	124	254	0·5	68	15·0	4·0	700	80	100	Peritoneal dialysis	Improved
3	Cholecystitis and jaundice	332	153	2·0	84	28·0	3·0	2,500	40	100	—	Brisk diuresis Improved
4	Status epilepticus with prolonged dehydration (PM-ATN)	356	214	1·7	Urine blood stained			2,000	80	100	Peritoneal dialysis	No diuresis Died
5	Leptospirosis	183	67	3·0	22	64·0	0·3	1,000	80	100	CVP	Brisk diuresis Improved
6	Acute renal failure ? cause	395	188	2·0	125	9·2	13·0	1,000	80	100	Peritoneal dialysis	No diuresis Died
7	Falciparum malaria, acute renal failure	633	406	1·3	68	17·0	4·0	1,500	80	100	Peritoneal dialysis	Improved

All patients had urinary flow of less than 10 ml./min. for at least 24 hours.

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