MODERN TRENDS

IN

UROLOGY

(SECOND SERIES)

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FOREWORD

SOME YEARS have elapsed since the first Series of *Modern Trends in Urology* was published. Since then enormous advances have been made in our knowledge of this specialty. Sir Eric Riches has shown wisdom in presenting us with an entirely new second series of this book with the aid of a number of distinguished contributors.

The biochemical aspects of renal disease are given a chapter to themselves, which indicates the importance of this branch of medicine in relation to urinary diseases. The use of the artificial kidney is still, in some respects, in the experimental stage but already it has been the means of saving many lives. The chapter on this subject is written by one whose experience with it is probably the most extensive in Great Britain. Another piece of apparatus which is revolutionizing our knowledge of the physiology of the urinary tract is the image intensifier. But it has done more than this. It has, among other things, demonstrated to the surgeon that the operative approach to the treatment of hydronephrosis need no longer be empirical. Here again Sir Eric Riches has shown a sound judgment in his choice of contributors. He, himself, has written three chapters. In one of them, with the aid of a very experienced radiotherapist, he reviews the results and complications of the treatment of 91 cases of bladder growths by supervoltage therapy.

A full account is given in Chapter 9 of hypertension due to renal artery disease. This is a fascinating story of the advance of surgery of the arterial system. The importance of paediatric urology is revealed in the account of the medical and surgical treatment of mega-ureter and the rehabilitation of children suffering from congenital deformities.

Four different authors, one of whom is an American, have described the uses of the small and large intestines in urology. The survey is a comprehensive one.

There has been a tendency in the past to ignore the subject of infertility. An excellent account of this is given by a urologist who has made a special study of it.

Pathology has not been forgotten. One leading authority describes renal pelvic growths, another testicular neoplasms and yet another granulomatous prostatitis. All have added to the practical understanding of these lesions.

Incontinence of urine due to various causes is fully discussed, together with its general management, in the last chapter. Such cases cause grave anxiety both to patient and doctor. It has been wise to give prominence to their treatment.

A short time ago at an international meeting on the Continent of Europe, the writer was asked by a British ambassador if he considered that the urologists of English-speaking countries were making as important contributions to our knowledge of genito-urinary diseases as those in other parts of the world. The answer is in this volume of *Modern Trends in Urology*, edited by Sir Eric Riches, a book which discloses that its contributors have advanced our knowledge of pathology, diagnosis and treatment in this branch of medicine and surgery.

A. CLIFFORD MORSON

London, 1960

INTRODUCTION

THE kind reception accorded to the first series of *Modern Trends in Urology* has prompted the preparation of a completely new second series. Urology is a progressive science and in the intervening years great advances in both diagnosis and treatment have been made; many of them are now described. Some are established as standard urological procedures but others must still be regarded as "trends" before they can justify inclusion in a text-book.

The increasing need for close collaboration between surgeons, physicians and laboratory workers is indicated by the larger number of chapters which at first sight appear to be mainly of medical interest. The advances in pathology and biochemistry and a knowledge of the influence of hormones and of nuclear sexing are all subjects with which the modern urologist must be familiar; he must seek the aid of an experimental surgeon in the search for the solution of the immunological problems which limit the full development of renal transplantation.

Although each chapter expresses the opinion of its individual author an effort has been made to avoid any views which are in direct conflict, but rather to attempt a correlation which will link up the different facets of the same problem. For example, the conditions of mega-ureteric obstruction are shown to be closely connected with physiological movements of the ureter; the success of its replacement by small intestine depends on the reproduction of these movements as far as possible. The inclusion of small or large intestine in the urinary tract as a conductor or a reservoir is now sufficiently established to merit detailed descriptions of the operative techniques.

The number of new contributors is an indication of the expanding interest in urology throughout Great Britain. To all, both old and new, we tender our thanks for their keen co-operation. The publishers have done their work quickly and well. Most of the illustrations are new and have been specially drawn for the book by the skilled medical artists to whom we are all indebted. A few blocks have been borrowed and we are grateful to the Editors and publishers of the *British Journal of Urology*, the *Annals of Internal Medicine* and the *Journal of Urology*, and to the Genito-Urinary Manufacturing Co., Ltd., for their loan.

ERIC RICHES

London, 1960.

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CHAPTER 1

METABOLIC DISORDERS IN RENAL DISEASE

G. A. SMART

ACUTE RENAL FAILURE

ACUTE renal failure, the result of damage to the kidneys, is associated with a diminished rate of urinary secretion. In the extreme case complete anuria occurs, but more often there is oliguria.

Definition of oliguria

A normal person with a low water intake may have a very low rate of urinary secretion, but under these circumstances the urine will be concentrated and have a high specific gravity. Greater difficulty may arise in the case of, for example, a post-operative subject who has not been taking any protein-containing foods. In general, such a person with normal kidneys will pass relatively small volumes of urine, but the specific gravity will not be fixed between the range 1008–1014 which is characteristic of renal failure—it will tend to be higher than this. Theoretically, if the intake of fluid solute and nitrogen were suitably arranged, normal subjects would pass small quantities of urine (less than about 700 ml. per 24 hours) with a specific gravity in the range 1008–1014. This was put to experimental test by Joekes, Mowbray and Dormandy (1957) who showed that this could in fact occur. However, the urine of such subjects contained less than 20 milliequivalents (mEq.) per litre of sodium whereas that of patients with renal failure may contain 60 mEq. per L. or more. Thus, one can define the oliguria of renal failure as being a urinary output of less than about 700 ml. per 24 hours with a specific gravity of between 1008 and 1014 and with a sodium content higher than 40 mEq. per L. One can understand that such a situation may be entirely missed unless accurate measurements of urinary output are being made and unless the specific gravity and, if necessary, the sodium concentration of the urine are determined.

Classification of metabolic disorders

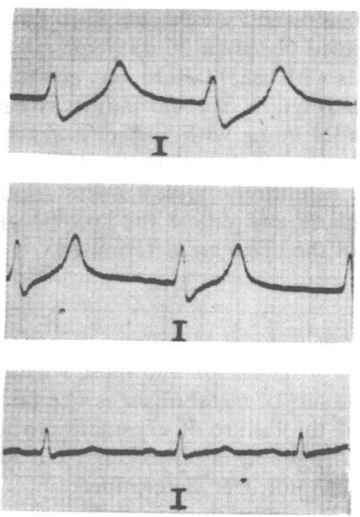
The metabolic disorders resulting from acute renal failure fall into two categories, namely those which derive from failure of regulation and those which are the result of the failure to eliminate the end products of metabolism. The failure in regulation affects water and electrolytes and the failure of elimination affects urea and other, largely unknown, products of nitrogenous metabolism, fixed acids and other substances, the significance of which has not been determined.

Failure of regulation

One of the main functions of the kidney is to act as an organ by means of which the body can regulate not only the concentrations of sodium, potassium, chloride, phosphorus, bicarbonate and hydrogen ion in the extracellular fluid, but also to

some extent the total amounts of these substances and of water in the body. Given time, defects in all of the above types of regulation may occur and in addition secondary effects, such as alteration in the serum calcium concentration, may ensue. When the failure is acute, however, these secondary effects are relatively small and the initial abnormalities are of prime importance. It is obvious that, with failure of renal regulation, the particular abnormality which may first become of paramount importance will depend largely upon the intake of nutrients and of water. If excessive amounts of water are given the patient may develop evidence of water intoxication, too great an intake of sodium will result in a high concentration of this ion in the extracellular fluid, and if both water and sodium are ingested in too great amounts, oedema will ensue; the giving of excessive amounts of water and sodium chloride is one of the commonest forms of mismanagement and stems from the failure to distinguish oliguria due to renal damage from that due to sodium and water deprivation or loss. If a diet is taken in which there is a large amount of potassium, or which leaves an acid residue when ashed, then the results will be respectively to produce high levels of potassium in the extracellular fluid or an acidaemia. In practice any or all of these abnormalities may be the first of a serious nature to be encountered in acute renal failure.

However strict the control of intake, metabolic processes are bound to continue which result in the release into the extracellular fluid of the end products of metabolism—most notably potassium, fixed acids and urea (as an index of nitrogenous end-products). Of these, increased potassium concentration is the most likely to be the first to give rise to serious effects (Fig. 1). Minor, but nevertheless important, effects on general cellular metabolism can sometimes be achieved by therapy with protein anabolic substances such as norethandrolone (McCracken and Parsons,



SERUM. K. 8·5 mEq./L.

SERUM. K. 7·0 mEq./L.

SERUM. K. 3·0 mEq./L.

FIG. 1.—Electrocardiogram showing the high T wave and splaying out of the ventricular complex characteristic of high serum potassium levels. The second and third tracings were taken after intravenous insulin and glucose, and after dialysis respectively.

1958) but, when it is realized that a major factor in prolonging the viability of a patient with acute anuria lies in a careful control of the intake, one cannot help but be shocked by the ineptitude with which such cases are sometimes treated. The dangerous practices of giving intravenous sodium sulphate, of "pushing fluids" in the hope of increasing urine flow, of giving large amounts of intravenous fluids or of giving potassium citrate when urinary infection is suspected, have still not entirely died out.

Failure of elimination

The most obvious result of failure of excretion is the rise in urea concentration in the body fluids. The urea itself, so far as one can tell, rarely causes trouble and then only when the concentration is very high so that crystals of the substance may appear upon the skin or irritation and ulceration of the intestine may result from its breakdown by micro-organisms with the release of ammonia. However, the concentration of urea in the blood is a useful index of the overall failure of excretion of metabolic end products and when the level exceeds about 400 mg. per 100 ml. serious effects, such as spontaneous haemorrhage, may occur.

Principles of treatment

The principles of treating patients with acute renal failure have many times been carefully enunciated (Borst, 1948; Bull, Joekes and Lowe, 1949; Bull, 1955), and since this was first done only minor modifications have been found to be necessary.

It is necessary in the first place to determine the cause of the anuria or oliguria, as it is most important to distinguish oliguria resulting from dehydration from that due to inherent renal damage. The total clinical situation is of the greatest help in making this differentiation and, as outlined above, simple measurement of urinary specific gravity may be of the first importance, since water lack will be associated with urine of high specific gravity, whereas urine secreted by damaged kidneys will generally have a specific gravity between 1008 and 1014.

Once it has been determined that the renal failure is due to potentially reversible renal damage, careful conservative management is essential. Water intake should equal the insensible loss together with the volume lost from other sources, minus the water produced by metabolism. It may sometimes be difficult to measure this, for the patient may be incontinent, or there may be seepage from burned areas. In such cases daily weighing, with an allowance of about 300–400 g. for tissue breakdown, is the most satisfactory way of estimating total water loss.

From balance studies on patients with acute renal failure Bluemle, Potter and Elkinton (1956) showed that a total of about 500 ml. (or 330 ml. per square metre of body surface) of exogenous water in excess of measured water loss were required to keep a constant ratio of total body water to total body solids in the oliguric stage.

Sodium should be given only in quantities sufficient to replace that lost, but the extracellular potassium concentration may increase to dangerous levels if negligible quantities of potassium are lost from the body, even if none is given. This is due to the release of potassium from glycogen and as a result of tissue breakdown. Concentration of about 8·5 mEq. per L. or more may prove fatal and emergency measures should be taken to deal with the situation. Intravenous glucose and insulin can be given and this is usually a temporarily effective expedient, since some

potassium as a result moves into cells. Sodium charged cation-exchange resin if given by mouth or by rectum will remove considerable amounts of potassium from the body and may alone be effective, but occasionally it may be necessary to correct hyperkalaemia with an artificial kidney or by some other form of dialysis (*see* Chapter 4).

Tissue breakdown is reduced to a minimum by giving adequate calories in the form of glucose or fat emulsion, but many have now discarded the latter preparations because of their tendency to cause diarrhoea. It is difficult to give sufficient glucose, because of the small daily allowance of water in which it may be dissolved. It tends to be nauseating when given by mouth, and intravenously the strong solution causes venous thrombosis with the risk of emboli. It may well be that in units possessing an artificial kidney or other means of dialysis a relatively normal diet, low in protein and salt, may be much more satisfactory.

Strict precautions must be taken to prevent, if possible, the patient acquiring any infection. This increases the metabolic rate and thus increases the speed at which serious metabolic disturbances may arise.

During the recovery phase a diuresis sometimes occurs with the loss of large amounts of water, sodium, potassium and chloride; in spite of this there may still be retention of nitrogen, though this is unusual with outputs of more than 1000 ml. per 24 hours. Unless careful assessment of water and electrolyte loss is continued during this phase the patient may become depleted of water, sodium and potassium, or the balance of these substances may be disturbed by inappropriate therapy.

CHRONIC RENAL FAILURE

In chronic renal failure there occurs a partial failure of regulation and of excretion. It seems likely that nephrons which have survived the chronic disease process are hypertrophied and that each, individually, has a high glomerular filtration rate. The *total* glomerular filtration rate is, however, very low since the majority of the nephrons have been destroyed. Owing to the high rate of glomerular filtration in each surviving nephron its tubule is presented with a very large load and thus, although glucose and amino-acid reabsorption can usually be carried out satisfactorily, the reabsorption of sodium and the normal regulatory functions of the distal tubule, which may entail the exchange of sodium for hydrogen ions, is deficient. The situation is somewhat similar to that obtaining with an osmotic diuresis. Because the dietary intake usually contains an excess of fixed acid over fixed base there is characteristically a metabolic acidosis.

Sodium metabolism

It can be seen from what has been said that there is a tendency for the chronic nephritic to lose a considerable quantity of sodium (and of chloride) in the urine. The intake is not regulated automatically so that, unless it happens to be equivalent to the almost fixed output, the total quantity of sodium in the body will become abnormal. Usually the urinary output is higher than the intake and therefore depletion occurs. The concentration of sodium in extracellular fluid tends, however, to be maintained at a normal level by an equivalent water deficiency and this results in an inadequate plasma volume with a further increase in renal failure. Still further depletion may occur as a result of vomiting and a patient entering this