NEW TRENDS IN TREATMENT OF URINARY TRACT STONES

ESSAY

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bY

AYMAN ABD-ALLAH ABD-RABU

M. B., B. Ch.

SUPERVISORS

PROF. DR. ABD-ALLAH EL-FIKY

PROF. OF GENERAL SURGERY
FACULTY OF MEDICINE
AIN SHAMS UNIVERSITY

DR. SAYED M. RASHAD

LECTURER OF GENERAL SURGERY
FACULTY OF MEDICINE
AIN SHAMS UNIVERSITY

FACULTY OF MEDICIENE

AIN SHAMS UNIVERSITY

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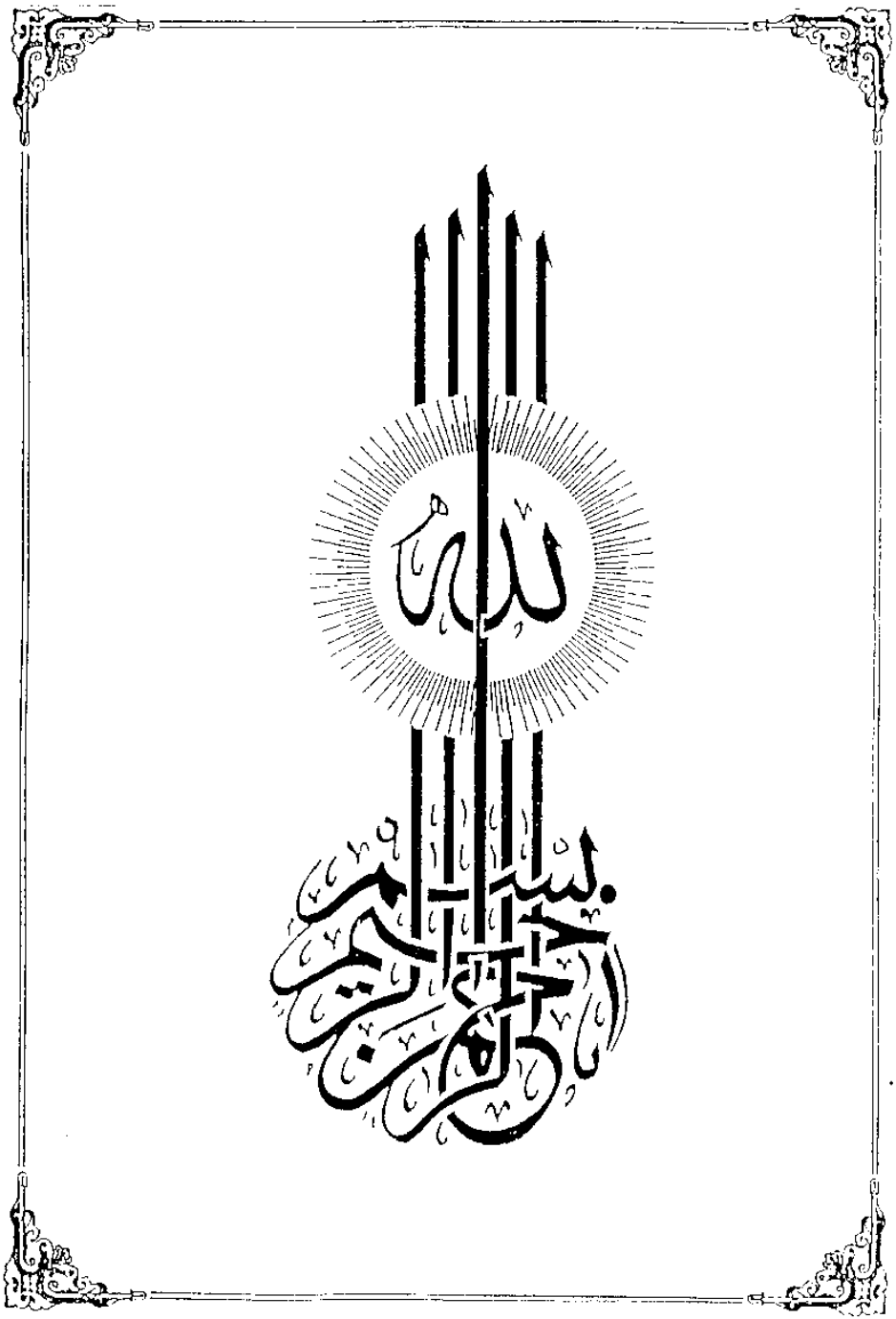

C O N T E N T S

INTRODUCTION

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Upper urinary tract stone disease is a long life diesease. Recent epidemiological studies indicate that renal stone disease is a rapidly increasing problem already affecting more than 2-3 percent up to 10 percent of the adult population and the prevelance is continuing to rise at the present time [Boyce et al., 1956].

Complete removal ofthe stone does nothing to affect the underlying cause of stone formation and subsequent recurrence occurs.

One third of the stones fail to pass spontaneously or under medical treatment and have to be treated surgically . Long term follow up service demonstrate recurrence rate as high as 60-80 percent [Williams, 1974] reflecting the ineffectiveness of the available preventive measures.

Unfortunately it was found that one of the most traumatic operations in surgery is the removal of stone from kidney. conventional incision causes considerable tissue damage in terms of muscular disruption and significant morbidity in terms of pain, prolonged post operative discomfort and prolonged hospital stay.

Actually, not only is the surgery of stone disease on the point of full evaluation, but the whole area of renal surgical disease is coming under rapid reappraisal in an attempt that in the near future conventional renal surgery through the grossly traumatic loin incision would be something of the past.

PHYSIOLOGY OF URINE FORMATION

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* Introduction

The kidney recieve 20 percent of the cardiac output while constituting only one-half of 1 percent of total body mass. The 180 liters of glomerular filtrate produced each day are finely processed to maintain the internal milieu with exquisite precision by mechanisms that continue to intrigue physiologists, nephrologists and urologists a like.

* Renal Haemodynamics

Functional organization of the renal circulation:

The renal circulation is designed to simultaneously accomplish bulk filtration & reabsorption and precise selective regulation of the constituents of normal urine. From an enormous bloodflow of about 1 litre per minute only 1 c.c. of urine per minute is formed. The energy requirement is about 10 percent of basal oxygen consumption [Bëeuwkes et al., 1981], yet the efficiency of the kidney is reflected in its low arteriovenous oxygen difference.

The kidney is not composed of a single homogenous circulation but of several distinct microvascular networks. These include the glomerular microcirculation, the cortical peritubular microcirculation, and microcirculations that

nourish and drain the inner and outer medulla [Beeuwkes, 1981].

About 20 afferent glomerular arterioles supply one or more of the 1.5 million glomeruli of the human kidney. The vascular pathways in the glomerulus change under different physiologic conditions. Hence, there is intermittent flow within glomeruli, which may play a role in regulation of glomerular filtration rate. The discovery that the glomerular mesangium contains contractile elements that respond to angiotensin II and other vaso active substances supports this hypothesis [Brenner et al., 1980]. Beyond the glomerulus the efferent arterioles either form dense peritubular capillary plexuses that nourish the proximal or distal convoluted tubules situated in the cortex or pass into the medulla [Especially from juxtamedullary glomeruli] and divide into bundles of vasarecta that parallel medullary ray[Book stein and Clark, 1980]. Microdissection and injection studies recently have shown that except for the initial portion of the peritubular capillaries in the outercortex, the efferent peritubular capillary net work and the nephron arising from each glomerulus are dissociated [Beeuwkes, 1979]. There are distinct outer and inner medullary capillary net works. In the inner medulla, the degree of organization of vascular-tubular relations correlates with concentrating ability [Kaissling et al., 1975].

Total R. B. F. estinated by para Amino Hippuric clearance [C_{PAH}] technique is 1200 ml/min/1.$7_3$$m^2$. In infants up to 1 year of age R. B. F. is about one half of the adult flow; it reaches the adult level at about 3 years of age [McCrory, 1972].

The phenomenon of auto-regulation of R. B. F. was described as early as 1947. Over a wide range of perfusion pressure from 80 to 180 mmHg, there is less than 10 percent change in R. B. F. or G. F. R. [Forster and Maes, 1947].

* *Glomerular Filtration Rate [G. F. R.]*

The elaboration of urine begins at glomerulus with the formation of a nearly protein free ultra filtrate of plasma, which enters Bowman's space. As the filtrate passes through the tubules, substances may be removed "Reabsorption" or added "Secretion ". Out of 170 liters of ultra filtrate filtered by glomeruli daily, only 1.5 litres of urine are excreted daily [Tucker and Blantz, 1977].

About 700 ml of plasma pass through the kidney per minute of which about 125 ml are filtered per minute [Brenner and Humes, 1977].

Factors affecting Glomerular filtration :

Micropuncture studies of individual nephrons have permitted direct measurement of the factors that determine single nephron glomerular filtration rate [Osgood et al., 1982].

The principal driving force for the glomerular filtration is the hydrostatic pressure at the glomerular capillary. It is a consequence of the forces that maintain systemic blood pressure, cardiac output and systemic vascular resistance. The hydrostatic pressure in glomerular capillary which favours ultrafiltration, is opposed by the hydrostatic pressure in Bowman's space of the renal tubule.

Complementing these hydrostatic forces are the osmotic pressures exerted by plasma proteins known as colloid osmotic pressure or oncotic pressure. The oncotic pressure of glomerular capillary plasma tends to oppose trancapillary fluid movement; the oncotic pressure of tubular fluid tends to favour it.

Changes in any of the foregoing variables in health or disease will have predictable effects on the single nephron glomerular filtration rate [Dworkin et al., 1983].

Autoregulation of Glomerular filtration Rate [G.F.R.]

Autoregulation of G.F.R. is believed to occur mainly through variations in afferent arteriolar resistance. In response to changes in arterial pressure, this results in parallel regulation of glomerular filtration rate and renal blood flow. Only at very low arterial perfusion pressure does an increase in efferent arteriolar resistance contribute to the maintainance of the hydrostatic pressure of glomerular capillary, sustaining single nephron glomerular filtration rate at reduced renal blood flow [Blythe, 1983].

The mechanism of autoregulation remains incompletely defined. Evidence from micropuncture studies supports the hypothesis that changes in the rate of fluid flow in the distal tubule elicit changes in glomerular arteriolar resistance. This phenomenon is known as distal tubule-glomerular feed back [Wrigth and Briggs, 1977].

An alternative theary explains autoregulation as a consequence of variations in afferent arteriolar tone that occur as a direct result of changes in arterial blood pressure [Fried and Stein, 1983].

Glomerular permeability

The fluid entering Bowman's space is nearly free of albumin and larger molecules. Restriction to glomerular filtration of certain molecules is known as glomerular permselectivity. The determinants of glomerular permselectivity include effective glomerular "pore" size and the electrostatic charge on the glomerular filtration barrier.

The filtration of molecules larger than inulin [molecular radius = 14 A^{o}] is progressively restricted, approaching zero at molecular radii of about40 A^{o} . [Brenner et al., 1977].

**** Sodium and Water***

Sodium and its associated anions [mostly chloride and bicarbonate] are confined to the extracellular fluid compartement and are the principle determinants of extracellular fluid osmolality. Because water moves freely across cell membrances, and because the osmolality of extracellular fluid is kept constant, it follows that the volume of extracellular fluid is directly related to the total body content of sodium. Renal tubular reabsorption of sodium and water preserves extracellular fluid volume despite glomerular filtration of large volumes of plasma.

Changes in tubular sodium reabsorption defend extracellular fluidvolume against changes in the filtered sodium load produced by changes in glamerular filtration rate. In addition, changes in sodium excretion maintain sodium balance at varying levels of sodium intake.

The reabsorption of Na ion takes place againt electrical and chemical gradients and requires expenditure of metabolic energy. such a process is descriped as active transport. The energy for the bulk of sodium reabsorption derives from aerobic metabolism. There is a direct, linear relationship between the rate of sodium reabsorption and oxygen consumption by the kidney.

The exact mechanisms of sodium transport throughout the nephron continue to be investigated. In the proximal tubule, sodium in the tubular lumen travels down its concentration gradient, across the luminal [apical] membrane, into the tubular epithelial cell. Within the cell, the sodium concentration is kept low by pumps in the basal and lateral membranes that extrude sodium into the peritubular space, from which it can enter the peritubular capillaries. These pumps, involving the enzyme Na,K-ATPase, represent the active component of sodium transport. For the most part, chloride reabsorption follows sodium as a consequence of the negative luminal potential created by out ward sodium movement.