PERIOPERATIVE OLIGURIA

E A S S A Y

Submitted in Partial Fulfilment
of the Master Degree in
ANAESTHESIOLOGY

BY

NAGLAA MOHAMED ALI
M.B., B. Ch.

SUPERVISED BY

Prof. Dr. MOHAMED HAMED SHAKER
Professor of Anaesthesia
Faculty of Medicine
Ain Shams University

Dr. MEGAHED MOHAMED ABDEL FATAH
Lecturer of Anaesthesia
Faculty of Medicine
Ain Shams University

AIN SHAMS UNIVERSITY
Faculty of Medicine
(1989)

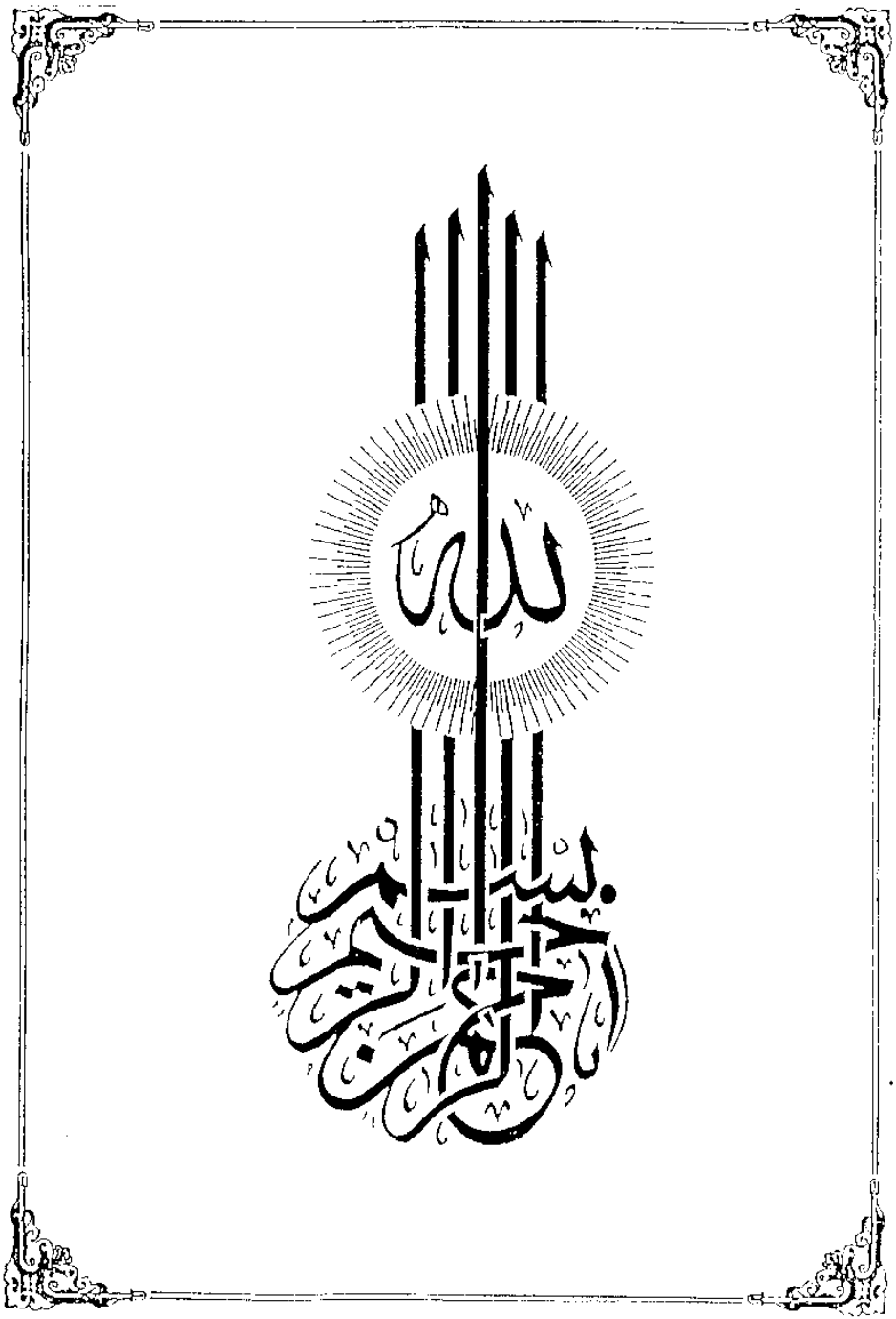
ACKNOWLEDGEMENT

First and foremost, I thank God, the Beneficent and the Merciful.

I may greatly incapable to express my great appreciation to ***Prof. Dr. MOHAMED HAMED SHAKER,*** *Professor of Anaesthesiology, Faculty of Medicine, Ain Shams University for his eminent guidance, constat support and also for his kind help in this work.*

I should also express may sincerest apperiation to ***Dr. MEGAHED MOHAMED ABDEL FATAH,*** *Lecturer of Anaesthesiology, Faculty of Medicine, Ain Shams University, for his fruitful advice and supervision throughout the preparation and fullfilment of this work.*

Lastly, I would like to express my deepest gratitude to all the members of the Anaesthesiology Department and to my colleagues, for their outstanding assistance.



CONTENTS

INTRODUCTION

INTRODUCTION

One of the most ominous signs in clinical practice is marked cessation or reduction of urinary output in a surgical patient during or after operation. Such a decrease in urine output can occur suddenly or over a period of several hours or days. This decrease in urine output is known as oliguria.

Oliguria by definition is the state in which the urine output is less than 400 ml. per day.

The rationale for calling attention to this urine volume is that there is an obligation to excrete an osmotic load of at least 400-500 m. Osm. daily, which represents the products of normal metabolism [urea, creatinine, uric acid and ammonium]. As the maximum urine osmolality achievable by the normal human is approximately 1200 m. Osm. per kg, a urine volume of at least 400 ml is necessary to excrete the daily osmotic load.

Hence, when the urine volume is less than 400 ml, normal needs are no longer being met and there is accumulation of nitrogenous wastes; such a patient is said to be oliguric (Goldstein, 1983).

Intraoperative oliguria cannot be diagnosed unless there is a means of monitoring urine production. During operation, urinary output should be charted on the anaesthesia record, at least hourly. A review of vital signs, blood loss, third space sequestration and the intake and output usually will reveal the cause of a low measured urinary volume.

When oliguria occurs acutely and perioperatively, it must be quickly and expertly managed. This is because, if the processes which initiate oliguria are sufficiently severe and sufficiently prolonged, acute renal failure will result (Abreo et al., 1986).

The mortality rate of these cases still exceeds 50%.

Perioperative renal failure accounts for approximately half of the acute haemodialysis instituted. Therefore, the anaesthesiologist has an unique opportunity to reduce the incidence of this severe complication by appropriately applying physiologic and pharmacologic principles to management.

PHYSIOLOGY OF URINE FORMATION

2. PHYSIOLOGY OF URINE FORMATION

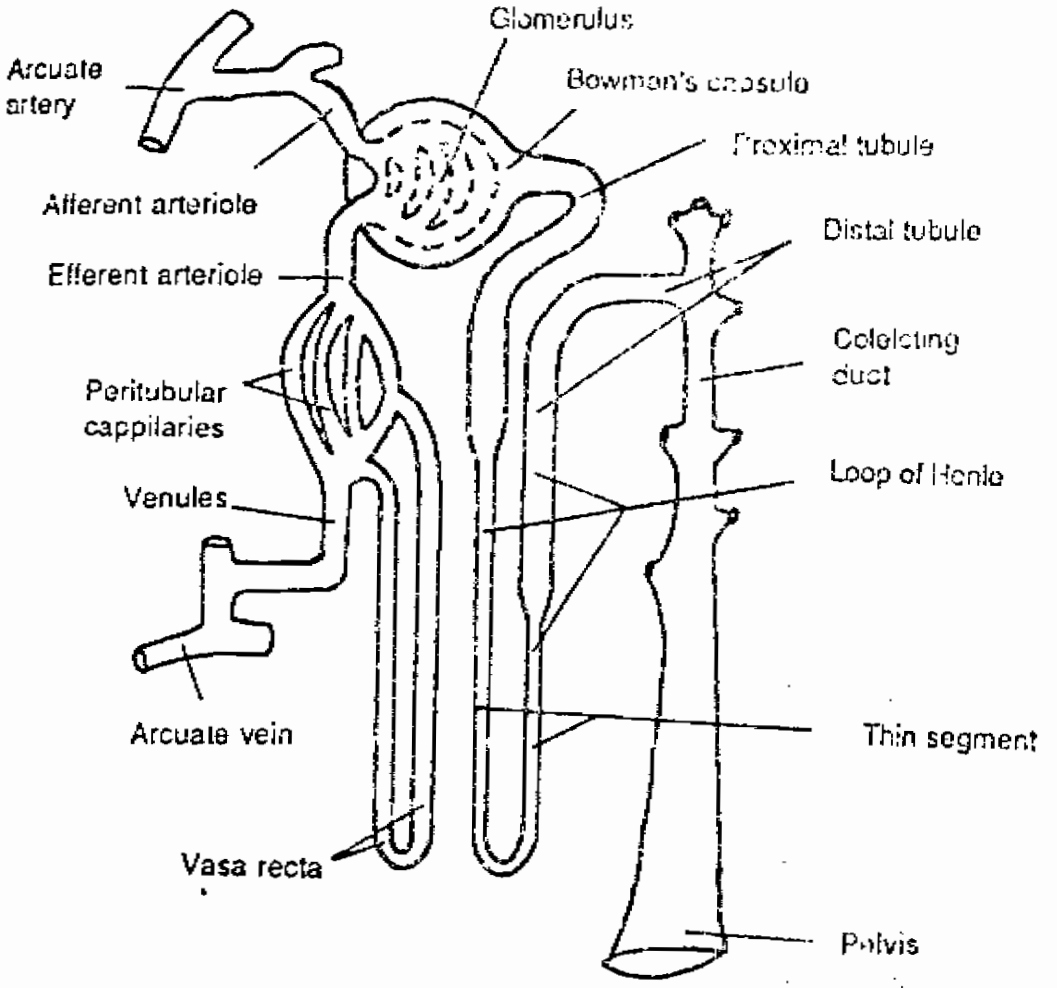
In the kidneys, a fluid that resembles plasma is filtered through the glomerular capillaries into the renal tubules [glomerular filtration].

As this glomerular filtrate passes down the tubules, its volume is reduced and its composition altered by the processes of tubular reabsorption and tubular secretion to form urine.

A comparison of the composition of the plasma and an average urine specimen illustrates the magnitude of some of these changes and emphasizes the manner in which wastes are eliminated while water and important electrolytes and metabolites are conserved.

Urine and Plasma concentration of important substances

Substance	Concentration in	
	Urine	Plasma
Glucose (mg/dl	0	100
Na (mEq/L)	150	150
Urea (mg/dl)	900	15
Creatinine(mg/dl)	150	1



The functional nephron

Furthermore, the composition of the urine can be varied, and many homeostatic regulatory mechanisms minimize or prevent changes in the composition of the ECF by changing the amount of water and various specific solutes in the urine i.e. role of kidney in pH regulation (Ganong, 1979).

1. Glomerular Filtration:

Approximately 180L of glomerular fluid is produced each day by the process of filtration through the glomerular capillary membrane.

This process does not involve expenditure of metabolic energy; rather filtration depends on hydrostatic pressure produced by contraction of the heart.

In the glomerulus, hydrostatic pressure in the afferent arteriole is approximately 76 mmHg. This is opposed by a plasma oncotic pressure of approximately 30 mmHg and an intraglomerular pressure in Bowman's capsule of 10 mmHg.

Factors affecting the glomerular filtration rate (GFR) :

1. Changes in glomerular capillary hydrostatic pressure.
2. Changes in renal blood flow.
3. Changes in hydrostatic pressure in Bowman's capsule.
4. Changes in concentration of plasma proteins.

The normal GFR in an average-sized normal man is approximately 125 ml/min. which equals 180 L/day, whereas, the normal urine volume is about 1 L/day.

Thus, 99% or more of the filtrate is normally reabsorbed. The process by which some substances are filtered and others are not is still not completely understood.

Recent theories include:

1. Pore theory:

This theory suggests that the glomerular capillary membrane, which has approximately 1 m^2 of total filtration area contains pores that are negatively charged.

These pores are freely permeable to water, ions and small negatively charged molecules less than 30 to 40 A° in diameter. Molecules of 40 to 80 A° are variably filtered, the extent of filtration being dependent on molecular charge. Thus albumin, which is negatively charged and has a diameter of about 60 A°, usually is not filtered, whereas, neutral dextran of the same diameter has a filtration index of about 0.5.

Molecules greater than 80 A° in diameter will not pass through the membrane at all. This results in complete filtration of unbound substances of molecular weight 15,000 or less, reduced filtration of substances of molecular weight in the range of 35,000, and insignificant filtration of substances with a molecular weight of 70,000 or more.

2. Hydrated Gel Theory:

This theory proposes that the glomerular membrane acts as a hydrated gel that permits certain substances to pass through while preventing others from filtration.

3. Aqueous channels theory:

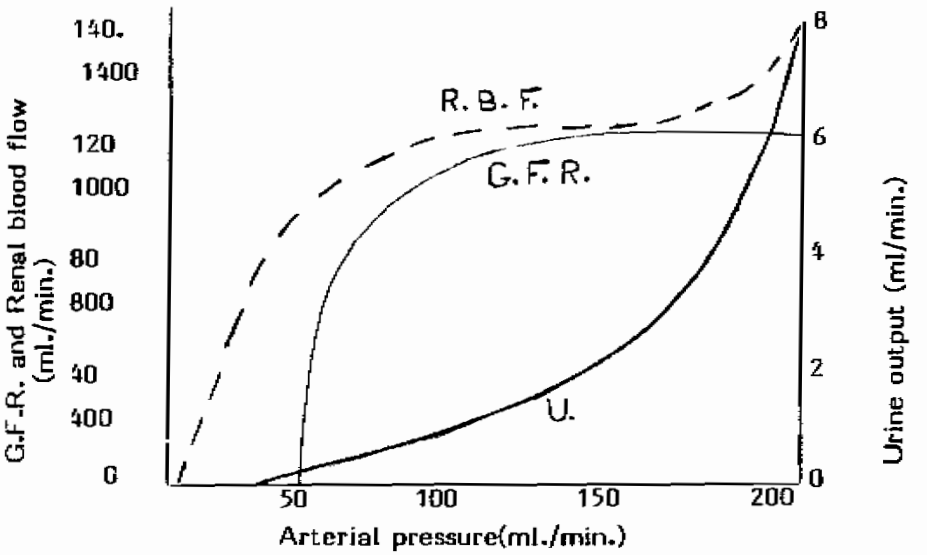
This theory postulates that the membrane is not a fixed structure but contains aqueous channels between loosely bonded protein and lipid components.

It permits filtration, by the process of diffusion, of water and low molecular-weight solutes. [(Brenner and Humes, 1977); (Brenner and Hostetter, 1978);[Marsh, 1983)].

Autoregulation of Glomerular Filtration Rate:

A change in arterial pressure causes a marked change in urinary output. Arterial pressure can change from as low as 75 mm.Hg, to as high as 160 mm.Hg while causing very little change in G.F.R., the mechanism by which the autoregulation is done is through two main feedback systems:

1. Afferent arteriolar vasodilatation feedback mechanism , and
2. The efferent arteriolar vasoconstrictor feedback mechanism. The combination of these two feedback mechanisms. is called the tubulo-glomerular feedback.



Autoregulation of glomerular filtration (G.F.R.) and renal blood flow (R.B.F.) when the arterial pressure is increased but there is lack of autoregulation of urine.

1. **The afferent arteriolar feedback mechanism.** The mechanism by which the afferent arteriolar feedback takes place is summarized in the following 4 steps:

   a. The little flow of glomerular filtrate into the tubule causes decreased chloride ion concentration at the macula densa.

   b. The decreased chloride concentration causes afferent arteriolar dilatation.

   c. This in turn increases the rate of blood flow into the glomerulus and increases the glomerular pressure.

   d. The increased glomerular pressure increases the glomerular filtration back to the required level.

2. **The efferent arteriolar feedback mechanism.** The mechanism by which the efferent arteriolar feedback takes place is as following:

   a. A too low glomerular filtrate causes excess reabsorption of chloride ions reducing its concentration at the macula densa.

   b. The low chloride concentration at the macula causes the juxtaglomerular cells to release renin from their granules.

   c. Renin causes formation of angiotensin II.

   d. The angiotensin II constricts the efferent arterioles, which causes the pressure in the glomerulus to rise.

e. The increased pressure then causes the glomerular filtration to return back to normal (Brenner, 1978).

2. Tubular transport:

The renal tubules function not only to reduce the large quantity of glomerular filtrate from 180 L to approximately 1 L/day, but also to alter the composition of the tubular fluid by the processes of reabsorption and secretion.

Tubular transport processes are of two types, passive and active. Passive transport is attributed to physical forces, commonly due to differences in concentration or electrical potential that create gradients across membranes.

Active transport is defined as the net movement of a particle against an electro-chemical potential gradient at the cost of metabolic energy. Inhibition of active transport is commonly seen if a system is cooled, deprived of oxygen or exposed to specific metabolic inhibitors.

a. Tubular reabsorption :

Water and many solutes are reabsorbed from the tubular lumen into the peritubular interstitial fluid and then into the blood. Tubular reabsorption permits the conservation of substances essential to normal function.

Some substances, such as water and sodium, are reabsorbed throughout the nephron, whereas others, such as glucose, amino-acids, and bicarbonate, are reabsorbed in one area (the proximal tubule).