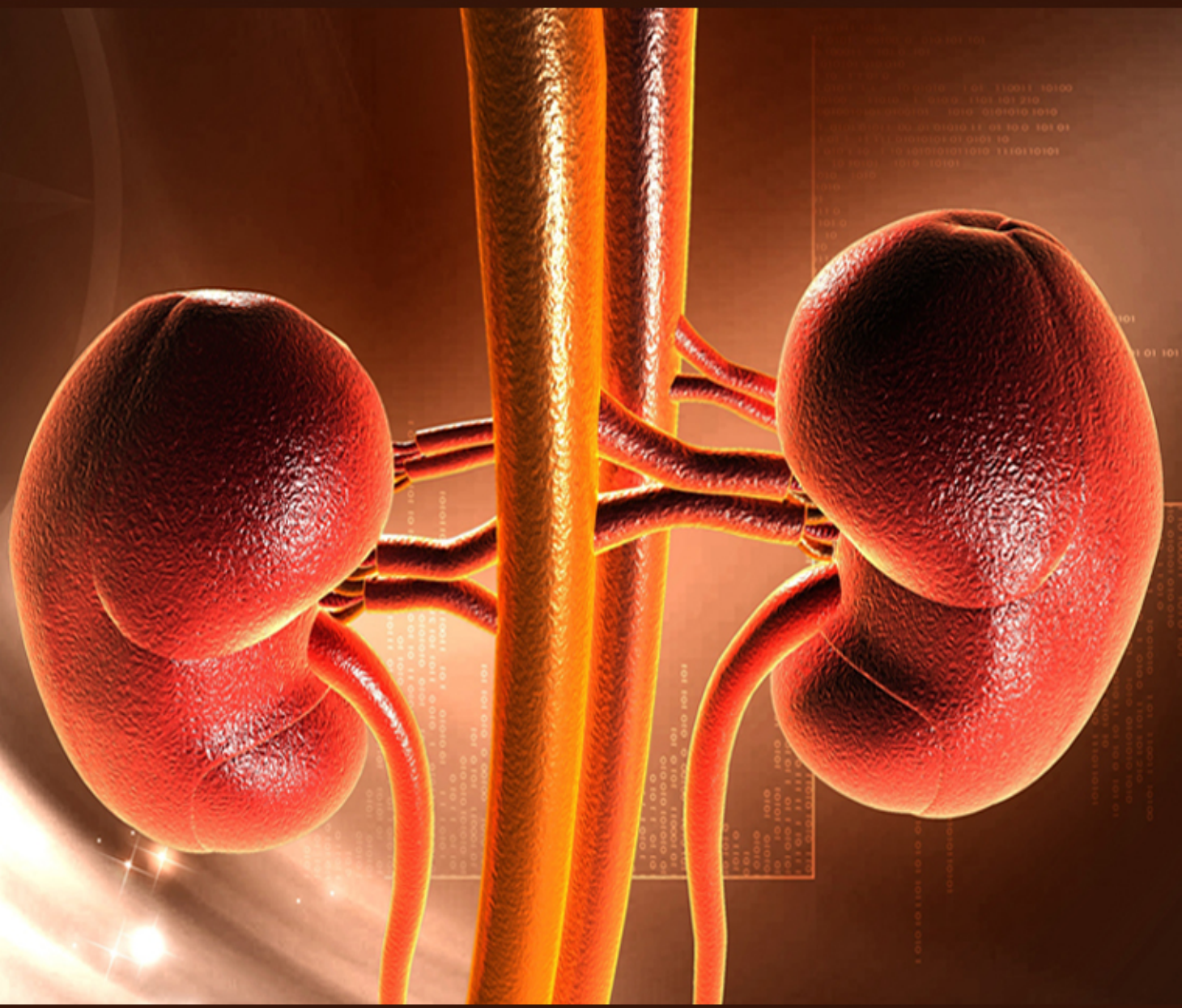
Fluid Management in the Intensive Care Unit after End of Renal Transplantation Surgery and Effect of Hot Weather in Sudan



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Fluid Management in the Intensive Care Unit after End of Renal Transplantation Surgery and Effect of Hot Weather in Sudan

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Dedication

To my parents, my wife, and my children

Abbreviations

ISP: International Standard Protocol

CVP: Central Venous Pressure

BP: Blood Pressure

Temp: Temperature

E): End of Operation

(A): After 24hrs from end of Operation

ATPase: Adenosine Triphosphatase

ADH: Antidiuretic Hormone

ACE: Angiotensin Converting Enzyme

GFR: Glomerular Filtration Rate

RBF: Renal Blood Flow

UN: Blood Urea Nitrogen

Abstract

This study was designed to determine the outcome of renal transplantation program in Sudan, specifically in the fluid management in the intensive care unit after end of renal transplantation surgery, keeping in mind the hot weather in Sudan. The non interventional, retrospective study which was conducted at Ahmed Gasim Hospital - intensive care unit-in the period from November 2005 to May 2006, where the ICU admission sheets for 60 patients, were studied to determine the effect of giving international standard protocol of fluid therapy which was modified by Sudanese physicians from 30m1 to 50m1 + urine output which is replaced by normal saline on central venous pressure, blood pressure, temperature and puls.

The study showed that were different levels of significant changes range from slight to strong changes in pulse, blood pressure, central venous pressure and temperature. The results showed that the application of international standard protocol of fluid management (ISP) has a great value to regulate the vital signs in recipient patient in Sudan.

Chapter 1

Introduction

Introduction

In the last decade, the availability of care for patients with kidney failure grew rapidly throughout the world. Transplantation of kidneys leads to several physiologic changes in the recipients who, during their anephertic state on chronic haemodialysis, have increased total body water and several electrolyte imbalances. Several abnormal parameters are observed in the physiology of the renal recipient.

The central venous pressure (CVP) and non invasive blood pressure in recipient invariably declines despite vigorous fluid resuscitation. Sixty Kidneys transplants were studied retrospectively, in which observation to a significant decline in central venous pressure (CVP), blood pressure, pulse and temperature in the immediate post transplant period up to 24 hours from declamping of the ureter. This phenomenon occurred despite aggressive fluid management and positive fluid balances averaging nearly 10 liters of normal saline. This study aims at exploring this area in Sudanese recipient patients during first twenty four hours from operation.

Literature review

Anatomy

Kidneys: The kidneys are two reddish - brown - organs situated high up on the posterior abdominal wall, one on each side of the vertebral column. The left kidney lies slightly higher than the right, each kidney gives rise to a ureter that runs vertically downward to the urinary bladder, located within the pelvis. The urine leaves the body through the urethra (‘).

Hilum of kidney: On the medial concave border of each kidney is a vertical slit that is bounded by thick lips of renal substance called hilum, which extends into a large cavity called renal sinus. The hilum transmits from front to backward, the renal vein, two branches of the renal artery and the ureter. Lymph vessels and sympathetic fibers also pass through the hilum (‘).

Coverings: The kidneys have the following coverings from inner side to the outer:

a) Fibrous capsule

b) Perirenal fat

c) Renal fascia

d) Para renal fat

All these structures support the kidneys and hold them in position on the posterior abdominal wall (‘).

Renal structure: Each kidney has a dark brown outer cortex and a light brown inner medulla. The medulla is composed of about a dozen renal pyramids, each having its base oriented toward the cortex and its apex.

Relationships:

Right kidney: Anteriorly lined by supra renal gland, the liver, the second part of duodenum, and the right colic flexure. Posteriorly lined by the diaphragm, the twelfth rip, and psoas, transverses Abdominis muscles [1].

Left kidney: Anteriorly lined by supra renal gland, the spleen, the stomach, the pancreas, the left colic flexure, and coils of jejunum. Posteriorly lined by the diaphragm, eleventh and twelfth ribs, and the psoas, transverses abodminis muscles [1].

Blood supply:

Arteries: The renal artery arises from the aorta at the level of the second lumbar vertebra. Each renal artery usually divided into 5 segmental arteries that enter the hilum of the kidney.

Veins: The renal vein emerges from the hilum in front of the renal artery and drains into the inferior vena cava.

Lymphatic: Drainage into lateral aortic lymph nodes around the origin of the renal artery.

Nerve supply: Through renal sympathetic plexus, the afferent fibers that travel through the renal plexus enter the spinal cord in the tenth, eleventh, and twelfth thoracic nerves(‘). Opposed by both plasma oncotic pressure (about 25mm Hg) and renal interstitial pressure (about 1Omm Hg). Both afferent and efferent arteriolar tones are important determinants of filtration pressure: Pressure is directly proportional to efferent arteriolar tone, but inversely proportional to afferent arteriolar tone. Approximately 20% of plasma is normally filtered as blood passes through the glomerulus [2].

The proxoimal tubule: Sixty-five to 75 percent of the ultrafiltrate formed in Bowman’s capsule is normally reabsorbed isotonically (proportional amounts of water and sodium) in the proximal renal tubules. To be reabsorbed, most substances must first transverse the tubular (apical) side of the cell membrane, and then cross the basolateral cell membrane into the renal interstitial before entering peritubular capillaries. The major function of the proximal tubule is Na^+ reabsorption. Sodium is actively transported out of proximal tubular cells at their capillary side by membrane-bound Na^+-K^+ adenosine triphosphateas (ATPase). The resulting low intracellular concentration of Na+ allows passive movement of Na^+ down its gradient from tubular fluid into epithelial cells. Angiotensin II and norepinephrine enhance Na^+ reabsorption in the early proximal tubule [2,3].

Sodium reabsorption is coupled with the reabsorption of other solutes and the secretion of H. Specific carrier proteins use the low concentration of Na^+ - K^+ ATPase activity (exchanging $3Na^+$ for $2K^+$), favors the absorption of other cations (K^+, Ca^{2+}, and Mg^{2+}). Thus the Na^+ - K^+.

Renal physiology: The kidneys play a vital role in regulating the volume and composition of body fluids, eliminating toxins, and elaborating hormones such as rennin, erythropoietin, and the active form of vitamin D. Surgery and anesthesia can have important effects on renal function. Failure to take these effects into consideration could result in serious errors in patient management. Fluid overload, hypovolemia, and postoperative renal failure are major causes of postoperative morbidity and mortality.

The Nephron: Each kidney is made up of approximately 1 million functional units called nephron. Anatomically, a nephron consists of six major anatomic and functional divisions including the glomerular capillaries, the proximal convoluted tubule, the loop of Henle, the distal renal tubule, the collecting tubules and the juxtaglomerular apparatus. An ultra filtrate of blood is formed which passes through the nephron, its volume and composition are modified by both the reabsorption and the secretion of solutes. The end product is eliminated as urine [2].

The Glomerular capillaries: The glomerulus is composed of tufts of capillaries that jut into Bowman's capsule, providing a large surface area for the filtration of blood. Blood flow is provided by a single afferent arteriole and is drained by a single efferent arteriole. Glomerular filtration pressure (about 60mm Hg) is normally about 60% of mean arterial pressure and is ATPase at the basolateral side of the renal cells provides the energy for the reabsorption of most solutes. Sodium reabsorption at the luminal membrane is also coupled with counter transport (secretion) of H^+. The latte mechanism is responsible for reabsorption of 90% of the filtered bicarbonate ions. Unlike other solutes, chloride can traverse the tight junctions between adjacent tubular epithelial cells. As a result, chloride reabsorption is generally passive and follows its concentration gradient. Active chloride reabsorption may also take place as a result of a K^+ - Cl^- co transporter that extrudes both ions at the capillary side of the cell membrane. Water moves passively out the proximal tubule along osmotic gradients.

The proximal tubules are capable of secreting organic cations and anions. Organic cations such as creatinine, cimetidine, and quinidine may share the same pump mechanism and thus can interfere with the excretion of one another. Organic anions such as urate, ketoacids, penicillins, cephalosporin's, diuretics, salicylates, and most x-ray dyes also appear to share common secretary mechanisms. Both pumps probably play a major role in the elimination of many circulating toxins. Low molecular weight proteins, which are filtered by glomeruli, are normally reabsorbed by proximal tubular cells but are metabolized intracellularly [2,3].

The Loop of henle: The loop of Henle consist of descending and ascending portions. The thin descending segment is a continuation of the proximal tubule and descends from the renal cortex into the renal medulla. In the medulla, the descending portion acutely turns back upon itself and rises back up toward the cortex as the ascending portion. The ascending portion consists of a functionally distinct, thin ascending limb, a medullary thick ascending limb, and a cortical thick ascending limb. Cortical nephrons (30-40%) have relatively short loops of Henle, while those near the medulla (juxtamedullary nephrons, 10%) loop deeply into the medulla. the loop of Henle is responsible for maintaining a hypertonic medullary interstitium and indirectly provides the collecting tubules with the ability to concentrate urine. Only 25-35% of the ultrafiltrate formed in Bowman's capsule normally reaches the loop of Henle. This part of the nephron usually reabsorbs 15-20% of the filtered sodium load. With the notable exception of the ascending thick segments, solute and water reabsorption in the loop of Henle is passive and follows concentration and osmotic gradients, respectively. Active Na^+ reabsorption still results from Na^+-K^+ ATPase activity on the capillary side of epithelial cells [2,3].

The Distal tubule: The distal tubule receives hypotonic fluid from the loop of Henle and is normally responsible for only minor modifications of tubular fluid. In contrast to more proximal portions, the distal nephron has very tight junctions between tubular cells and is relatively impermeable to water and sodium. It can therefore maintain the gradients generated by the loop of Henle. Sodium reabsorption in the distal tubule normally accounts for only about 5% of the filtered sodium load. As in other parts of the nephron, the energy is derived from Na^+ -$1K^+$ ATPase activity on the capillary side. The distal tubule is the major site of parathyroid hormone- and vitamin D- mediated calcium reabsorption [2,3].

The collecting tubule: This tubule can be divided into cortical and medullary portions. Together, they normally account for the reabsorption of 6-7% of the filtered sodium load.

A. Cortical Collecting Tubules: This part of the nephron consists of two cell types:

a) priciplal cells (P cells), which primarily secrete potassium and participate in aldosterone-mediated Na^+ reabsortpion, and

b) Intercalated cells (1 cells), which are responsible for acid-base regulation [2].

B. Medullary Collecting Tubule: The medullary collecting tubule courses down from the cortex through the hypertonic medulla before joining collecting tubules from other nephrons to form a single ureter in each kidney. This part of the collecting tubule is the principal site of action for antidiuretic hormone (ADH), also called arginine vasopressin (AVP); Dehydration increases ADH secretion, rendering the luminal membrane permeable to water. As a result, water is osmotically drawn out of the tubular fluid passing through the medulla, and a concentrated urine (up to 1400 mOsm/L) is produced. Conversely, adequate hydration suppresses ADH secretion; the fluid in the collecting tubules therefore passes through the medulla unchanged and remains hypotonic. Moreover, this part of the nephron is responsible for acidifying urine; the hydrogen ions secreted are excited [2,3].

The Juxtaglomerular apparatus: This small organ within each nephron consists of a specialized segment of the afferent arterent arteriole, containing juxtaglomerular cells within its wall, and the end of the thick. Ascending cortical segment of the loop of Henle, the macula densa. Juxtaglomerular cells contain the enzyme rennin and are innervated by the sympathetic nervous system. Release of rennin depends on S1-adrenergic sympathetic stimulation, changes in afferent arteriolar wall pressure, and changes in chloride flow past the macula densa. Rennin released into the bloodstream acts on angiotensinogen, a protein synthesized by the liver, to form angiotensin I. This inert decapeptide is then rapidly converted, primarily in the lungs, by angiotensin-converting enzyme (ACE) to form the octapeptide angiotensin II. Angiotensin II plays a major role in blood pressure regulation [2].

The Renal circulation: Renal function is intimately related to renal blood flow. In fact, the kidneys are the only organs for which oxygen consumption is determined by blood flow; the reverse is true in other organs. The combined blood flow through both kidneys normally accounts for 20-25% of total cardiac output. In most person, each kidney is supplied by a single renal artery arising from the aorta. The renal artery then divides at the renal pelvis into interlobar atteries, which in turn give rise to arcuate arteries at the junction between renal cortex and medulla. Arcuate arteries further divide into interlobular branches that eventually supply each nephron via a single afferent arteriole. Blood from each glomerulus is drained via a single efferent arteriole and then travels alongside adjacent renal tubules in a second (peritubular) system of capillaries. In contrast to the glomerular capillaries, which favor filtration, peritubular capillaries are primarily “reabsorptive.” Venules draining the second capillary plexus finally return blood to the inferior vena cava via a single renal vein on each side [1-3].

Glomerular filtration rate: The GFR is normally about 20% of RPF. Normal values for GFR are about 120 ± 25mL/min in men and 95 ± 20 mL/min in women [2,3].

Control mechanisms: Regulation of renal blood flow represents a complex interplay between intrinsic autoregulation, tubuloglomerular balance, and hormonal and neuronal influences.

Intrinsic regulation: Autoregulation of RBF normally occurs between mean arterial blood pressures of 80 and 180mm Hg. Blood flow is generally decreased at mean arterial pressures less than 70mm Hg. Although the exact mechanism is not known, it is thought to be an intrinsic myogenic response of the afferent arterioles to changes in blood pressure. Within these limits, RBF (and GFR) can be kept relatively constant by afferent arteriolar vasoconstriction or vasodilation. Outside the autoregulation limits, RBF becomes pressure-dependent. Glomerular filtration generally ceases when mean systemic arterial is less than 40-50mmg Hg [2,3].

Tubuloglomerular balance and feedback: Changes in renal tubular flow rates affect GFR; increases in tubular flow tend to reduce GFR, while decreases in flow tend to favor increases in GFR. Tubuloglomerular feedback probably palys an important role in maintaining GFR constant over a wide range of perfusion pressures [2,3].

Hormonal regulation: Increases in afferent arteriolar pressure stimulate rennin release and formation of angiotensin II. Angiotensin II causes generalized arterial vasoconstriction and secondarily reduces RBF. Both afferent and efferent arteriole is smaller, its resitance becomes greater than that of the afferent arteriole; GFR therefore tends to be relatively preserved. Very high levels of angiotensin II constrict both arterioles and can markedly decrease GFR. Adrenal catecholamines (epinephrine and norepinephlar tone) directly and preferentially increase afferent arteriolar tone, but marked decreases in GFR are minimized indirectly through activation of rennin release and angiotensin II formation. Relative preservation of GFR during increased aldosterone or catecholamine secretion appears to be at least partly mediated by angiotenin-induced prostaglandin synthesis and is blocked by inhibitors of prostaglandin synthesis (nonsteroidal anti-inflammatory drugs). Renal synthesis of vasodilating prostaglandins (PGD2, PGE2, and PGI2) is an important protective mechanism during periods of systemic hypotension and renal ischemia [2].

Neuronal regulation: Sympathetic outflow from the spinal cord at T4 - L1 reaches the kidneys via the celiac and renal plexuses. Sympathetic nerves innervate the juxtaglomerular apparatus (f3i) as well as the renal vasculature (al). This innervationis probably responsible for stress-induced reductions in RBF. ai-Adrenergic receptors enhance sodium reabsorption in proximal tubules while al receptors decrease such reabsorption and promote water excretion.

Effects of anesthesia on renal function: Clinical studies attempting to define the effects of anesthetic agents on renal function are complicated by difficulties in differentiating between direct and indirect effects and often fail to control many important variables. These variables include the type of surgical procedure, fluid administration, and pre-existing cardiac and renal function [4].

a. Reversible decreases in RBF, GFR, urinary flow, and sodium excretion occur during both regional and general anesthesia.

b. Changes are generally less marked during regional aneasthesia.

c. Most of these changes are indirect and are mediated by autonomic and hormonal influences.

d. These effects can be at least partially overcome by maintenance of an adequate intravascular volume and a normal blood pressure.

e. Only a few anesthetics (methoxyflurane and, theoretically, enflurane and sevolurane) in high doses can cause specific renal toxicity.

Indirect effects:

Cardiovascular effects: Most inhalational and intravenous anesthetics cause some degree of cardiac depression or vasodilation and therefore are capable of decreasing arterial blood pressure. The sympathetic blockade associated with regional anesthesia (spinal or epidural) can similarly cause hypotension as a result of increased venous capacitance and arterial vasodilation. Decreases in blood pressure below the limits of autoregulation can therefore be expected to reduce RBF, GFR, urinary flow, and sodium excretion. Intravenous fluid administration often at least partially reverses the hypotension and ameliorates its effects on renal function [4,5].

Neural effects: Sympathetic activation commonly occurs in the perioperative period as a result of light anesthesia, intense surgical stimulation, tissue trauma, or anesthetic-induced circulatory depression. Sympathetic overactivity increases renal vascular resistance and activates various hormonal systems. Both effects tend to reduce RBF, GFR, and urinary output [4,5].

Endocrine effects: Endocrine changes during anesthesia generally reflect a stress response that may be induced by surgical stimulation, circulatory depression, hypoxia, or acidosis. Increases in catecholamines (epinephrine and norepinephrine), rennin, angiotensin II, aldosterone, ADH, adrenocorticotropic hormone, and cortisol are common. Catecholamines, ADH, and angiotensin II all reduce RBF by inducing renal arterial constriction. Aldosterone enhances sodium reabsorption in the distal tubule and collecting tubule, resulting in sodium retention and expansion of the extracellular fluid compartment. Nonosmotic release of ADH also favours water retention and, if marked, may result in hyponatremia. The endocrine response to surgery and anesthesia is probably at least partly responsible for the transient postoperative fluid retention that is seen in many patients [4,5].

Direct anesthetic effects: The direct effects of aneasthetics on renal function are minor compared with the secondary effects described above.

Volatile agents: Halothane, enflurane, and isoflurane have been reported to decrease renal vascular resistance. Studies of their effect on autoregulatin have had conflicting results. In some animal studies, halothane appears to depress sodium reabsorption. Methoxyflurane has been associated with a syndrome of polyuric renal failure. Its nephrotoxicity is dose-related and is the result of release of fluoride concentrations greater than 50 1.1mol/L have been associated with renal toxicity that is characterized by a defect in urinary concentrating ability. Methoxyflurane doses greater than 1 minimum alveolar concentration for 2 hours are associated with a high incidence of renal impairment. Fluoride production is negligible during halothane, desflurane, and isoflurane anesthesia but can become significant following the prolonged administration of enflurane and possibly sevoflurane. High plasma fluoride concentrations following prolonged enflurane anesthesia may also occur in obese patients and those receiving isoniazid therapy, but an increased incidence of renal dysfunction has not been reported. Compound A, breakdown product of sevoflurane that is formed at low flows, can cause renal damage in laboratory animals. Clinical studies have not detected significant renal injury in humans during sevoflurane anesthesia. Nonetheless, most authorities recommend fresh gas flow of at least 2 L/min with sevoflurane to prevent significant production of compound A [4,6].

Interavenous agents: Studies on opioids and barbiturates generally show minor effects when they are used alone. In the presence of nitrous oxide, these agents can produce effects similar to those observed with volatile agents. Ketamine is reported to minimally affect renal function and to preserve renal function during hemorrhagic hypovolemia.

Other drugs: Many drugs and dyes that are used in the perioperative period can adversely affect renal function, especially in the setting of preexisting renal dysfunction. These include antibiotics (eg, aminoglycosides and amphotericin B), immunosuppressive agents (eg, cyclosporine and tacrolimus), and radiocontrast dyes. Mechanisms of injury include renal arterial vasospasm, direct cytotoxic properties, and renal microvascular or tubular obstruction [4,6].

Diuretics: Diuretics increase urinary output by decreasing the reabsorption of Na^+ and water. They are most commonly classified according to their mechanism of action. Unfortunately, many diuretics have more than one such mechanism, so the classification system is imperfect; only major mechanisms will be reviewed here. The majority of diuretics exert their action on the luminal cell membrane from within the renal tubules. Because nearly all diuretics are highly protein-bound, relatively little of the free drug enters the tubules by filtration. Most diuretics must therefore be secreted by the proximal tubule (usually via the organic anion pump) to exert their action. Impaired delivery into the renal tubules accounts for resistance to diuretics in patients with impaired renal function [4-6].

Osmotic diuretics (mannitol): Osmotically active diuretics are filtered at the glomerulus and undergo limited or no reabsorption in the proximal tubule. Their presence in the proximal tubule limits the passive water reabsorption that normally follows active sodium reabsorption. Although their major effect is to increase water excretion, in large doses, osmotically active diuretics also increase electrolyte (sodium and potassium) excretion. The same mechanism also impairs water and solute reabsorption in the loop of Henle. Mannitol is the most commonly used osmotic diuretic. It is a six-carbon sugar that normally undergoes

little or no reabsorption. In addition to its diuretic effect, mannitol appears to increase RBF. The latter can wash out some of the medullary hypertonicity and interfere with renal concentrating ability. Mannitol appears to activate the intrarenal synthesis of vasodilating prostaglandine. It also appears to be a free radical scavenger [4,5].

A. Uses

i. Prophylaxix against acute renal failure in high-risk patients: This group of patients includes those with massive trauma, major hemolytic reactions, rhabdomyolysis, and severe jaundice as well as those undergoing cardiac or aortic operations. The efficacy of prophylaxis in these instances may be related to dilution of nephrotoxic substances within the renal tubules, prevention of sludging and obstruction within the tubules, maintenance of RBF, and perhaps reduction of cellular swelling and preservation of cellular architecture.

ii. Evalation of acute oliguria: Mannitol in the presence of hypovolemia will augment urinary output. In contrst, it will have little effect in the presence of severe glomerular or tubular injury.

iii. Conversion of olgiuric renal failure to nonoliguric renal failure: Althouth this indication is controversial, the lower mortality rate associated with nonoliguric renal failure still prompts many clinicans to use mannitol in that setting.

iv. Acute reduction of intracranial pressure and cerebral edema:

v. Acute reduction of intraocular pressure in the perioperative period:

   a. Intravenous dosage: Mannitol, 0.25- lg/kg.

   b. Side effect: Mannitol solutions are hypertonic and acutely raise plasma and extracellular osmolality. A rapid intracellular to extracellular shift of water can transiently increase intravascular volume and precipitate cardiac decompensation and pulmonary edema in patients with limited cardiac reserve. Transient hyponatremia and reductions in hemoglobin concentration are also common and represent acute hemodilution resulting from rapid movement of water out of cells; a modest, transient increase in plasma potassium concentration may also be observed. It is also important to note that the initial hyponatremia does not represent hyposmolality but reflects the presence of mannitol. If fluid and electrolyte losses are not replaced following diuresis, mannitol can result in hypovolemia, hypokalemia, and hypernatremia. The hypernatremia occurs because water is lost in excess of sodium [4-6].

Loop diuretics: The loop diuretics include furosemide (Lasix), bumetanide (Bumex), ethaccrynic acid, and torsemide. All loop diuretics inhibit Na^+-K^+-$2Cl^-$ luminal carrier protein be occupied. Loop diuretics compete with Cl^- for its binding site on the carrier protein. With a maximal effect, they can lead to excretion of 15-20% of the filtered sodium load. Both urinary concentrating and urinary diluting capacities are impaired. The large amounts of Na^+ and Cl^- presented to the distal nephron are limited the reabsorptive capability. The resulting urine remains hypotonic. The reason for the latter is not clear but may related to rapid urinary flow rates that prevent equilibration with the hypertonic renal medulla or interference with the action of ADH on the collecting tubules. A marked increase in diuresis may occur when loop diuretics are combined with thiazides, especially metolazone. Some studies suggest that furosemide increase RBF and can reverse the redistribution of blood flow from the cortex to the medulla. Loop diuretics increase urinary calcium and magnesium excretion [4,6].

A. Uses

i. Edematous states (sodium overload): These disorders include heart failure, cirrhosis, the nephritic syndrome, and renal insufficiency. When given intravenously, these agents can rapidly reverse cardiac and pulmonary manifestations.

ii. Hypertension: Loop diuretics may be used as adjacent to other hypotensive agents, particularly when thiazides are ineffective.

iii. Evaluation of acute oliguria: The response to a small dose (10-20mg) of furosemide may be useful in differentiating between oliguria resulting from hypovolemia and oliguria that results from redistribution of RBF to juxtamedullary nephrons. Little or no response is seen with hypovolemia, whereas resumption of normal urinary output occurs with the latter.

iv. Conversion of Oliguric Renal Failure To Nonoliguric Renal Failure: Use of these drugs in this setting is as controversial as with mannitol. Moreover, mannitol may be more effective.

v. Treatment of Hypercalcemia:

vi. Rapid Correction of Hyponatermia:

   a) Intravenous dosages: Forosemide, 20-100mg; bumetanide, 0. 5-1mg; ethacrynic acid, 50-100mg; torsemide 10-100mg.

   b) Side effects: Increased delivery of Na^+ to the distal and collecting rubules increases K^+ and H^+ secretion at those sites and can result in hypokalemia and metabolic alkalosis. Marked Na^+ losses will also lead to hypovolemia and prerenal azotemia. Secondary hyperaldosteronism often accelerate the hypokalemia and metabolic alkalosis. Hypercalciuria can result in stone formation and occasionally hypocalcemia. Hypomagnesemia may be seen in patients receiving long-term therapy. Hyperuricemia is

thought to result from increased urate reabsorption and competitive inhibition of urate secretion in the proximal tubule. Reversible hearing loss has been reported with both furosemide and ethacrynic acid but may more common with ethacrynic acid [5,6].

Timazede-type diuretics: This group of agents includes thiazides, chlorthalidone (Thalitone), quinethazone (Hydromox), metolazone (Zaroxolyn), and indapamide (Lozol). These diuretics act at the distal tubule, including the connecting segment. Inhibition of sodium reabsorption at this site impairs urinary diluting but not concentrating ability. The thiazide diuretics compete for the Cl^- site on the luminal Na^+ - Cl^- carrier protein. When given alone, thiazide-type diuretics increase Na^+ excretion to only 3-5% of the filtered load because of enhanced compensatory Na^+ reabsorption in the collecting tubules. In contrast to their effects on sodium excretion, thiazide-type diuretics augment Ca^{2+} reabsorption in the distal tubule.

A. Uses

i. E type Y tenstion: Thiazides are often selected as first-line agents in the treatment of hypertension.

ii. Edematous Dinoder ("sodium °veilload): These agents are exclusively used as oral agents for mild to moderate sodium overload.

iii. Co Hypereaeimia: Thiazide diuretics are often uses to decrease calcium excretion in patients with hypercalciuria who form renal stones.

iv. Nephrogernic diabetes insipidus: The efficacy of these agents in this disorder reflects their ability to impair diluting capacity and increase urine osmolality.

a) Intravenous dosages: These agents only given orally.

b) Side effects: Although thiazide-type diuretics deliver less sodium to the collecting tubules than do loop diuretics, the increase in sodium excertion is enough to enhance K+ secretion and frequently results in hypokalemia. Enhanced Fl^+ secretion can also occur, enogh to result in metabolic alkalosis. Impairment of renal diluting capacity may produce hyponatremia in some patients. Hyperuricemia, hyperglycemia, hypercalcemia, and hyperlipidemia may also be seen.

Potassium sparing diuretms: These weak agents characteristically do not increase potassium excretion. Potassium-sparing diuretics inhibit Na+ reabsorption in the collecting tubules and therefore can maximally excrete only 1-2% of the filtered Na^+ load. They area usally used in conjunction with more potent diuretics for their potassium-sparing effect.

Aldosterone antagonists (Spironolactone): Spironolactone (Aldactone) is a direct aldasterone receptor antagonist in collecting tubules. It acts to inhibit aldosterone-meiared Na^+ reabsorption and K^+ secretion. As a result, spironolactone is only effective in patients with hyperaldosteronism. This agent also has some antiandrogenic properties.

A. Uses

i. Primary and secodnary hyperaldosteronism:

Spironolactone is usually used as an adjuvant in the treatment of refractory edematous states associated with secondary hyperaldosteronism. It is especially effective in patients with advanced liver disease.

a. Intravenous dosage: Spironolactone is only given orally.

b. Side effect: Spironolactone can result in hyperkalemia in patients with high potassium intake or renal insufficiency and in those receiving S-blockers or ACE inhibitors. Metabolic acidosis may also be seen. Other side effects include diarrhea, lethargy, ataxia, gynecomastia, and sexual dysfunction [4-6].

Carbonic anhydrase inhibitors: Carbonic anhydrase inhibitors such as acetazolamide (Diamox) interfere with Na+ reabsortion and H secretion in proximal tubules. They are weak diuretics because the former effect is limited by the reabsorptive capacities of more distal segments of nephrons. Nonetheless, these agents significantly interfere with H± secretion in the proximal tubules and impair HCO3 reabsorption [5].

A. Uses

i. Correction of metabolic alkalosis in edematous patients: Carbonic anhydrase inhibitors often potentiate the effects of other diuretics.

ii. Alakalinization of urine: Alkalinization enhances urinary excretion of weakly acidic compounds such as uric acid.

Other "Diuretics": These agents may increase GFR by elevating cardiac output or arterial blood pressure. Drugs in this category are not primarily classified as diuretics because of their other major actions. These agents is include methylxanthines (theophylline), cardiac glycosides (digitalis), inotropes, and saline infustions.

Anesthesia for patients with renal disease: Diseases affecting the kidneys are often grouped into syndromes based on common clinical and laboratory findings: nephritic syndrome, acute renal failure, chronic renal failure, nephritis, nephrolithiasis, and urinary tract obstruction and infection. The anesthetic care of patients with these syndromes is facilitated by grouping patients according to the status of their preoperative renal function rather than by syndrome [7].

Evaluating renal function: Accurate assessment of renal function relies heavily on laboratory determinations. Renal

impairment can be due to glomerular dysfunction, tubular dysfunction, or obstruction of the urinary tract. Because abnormalities of glomerular function cause the greatest derangements and are mostly readily detectable, the most useful laborartory tests are those related to the glomerular filtration rate [7].

Blood urea nitrogen: The primary source of urea in the body is the liver. During protein catabolism, ammonia is produced from the deamination of amino acids. Hepatic conversion of ammonia to urea prevents the build-up of toxic ammonia levels:

$2NH_3 + CO_2 - + H_2N - CO - NH_2 + H_2O$

BUN is therefore directly related to protein catabolism and inversely related to glomerular filtration. As a result, BUN is not a reliable indicator of the GFR unless protein catabolism is normal and constant. Moreover, 40-50% of the filtrate is normally reabsorbed passively by the renal tubules; hypovolemia increases this fraction. The normal BUN concentration is 10-20mg/dL. Lower values can be seen with starvation or liver disease; elevations usually result from decreases in GFR or increases in protein catabolism. The latter may be due to a high catabolic state (trauma or sepsis), degradation of blood either in the gastrointestinal tract or in a large hematoma, or a high-protein diet. BUN concentrations greater than 50mg/dL are generally associated with renal impairment [7].

Serum creatinine: Creatinine is a product of muscle metabolism that is nonezymatically converted to creatinine. Creatinine production in most persons is relatively constant and related to muscle mass, averaging 20-25mg/kg in men and 15-20mg/kg for women. Creatinine is then filtered (and to a minor extent secreted) but not reabsorbed in the kidneys. Serum creartinine concentration is therefore directly related to body muscle mass but inversely related to glomerular filtration. Because body muscle mass is usually failry constant, serum creatinine measurements are generally reliable indices of GFR. The normal serum creatinine concentration is 0.8-1.3mg/dL in men and 0.6-1mg/dL in women each doubling of the serum creatinine represents a 50% reduction in GFR. GFR declines with increasing age in most persons (5% per decade after age 20), but because muscle mass also declines, the serum creatinine remains relatively normal; creatinine production may decrease to 10mg/kg. Thus, in elderly patients, small increases in serum creatinine may represent large changes in GFR [7].

Urinalysis: Urinalysis continues to be the most common test routinely performed for evaluating renal function. Although its utility for that purpose is justifiably questionable, urinalysis can be helpful in identifying some disorders of renal tubular dysfunction as well as some nonrenal disturbances. A routine utinalysis typically includes ph, specific gravity, detection and quantification of glucose, protein, and bilirubin content, and microscopic examination of the urinary sediment. Urinary pH is helpful only when arterial pH is also known. A urinary pH greater than 7.0 in the presence of systemic acidosis is suggestive of renal tubular acidosis. Specific gravity is related to urinary osmolality; 1.010 usually corresponds to 290 mmol/kg. A specific gravity greater than 1.018 after an overnight fast is indicative of adequate renal concentrating ability. A lower specific gravity in the presence of hyperosmolaility in plasma is consistent with diabetes insipidus. Glycosuria is the result either of a low tubular threshold for glucose (normally 180mg/dL) or of hyperglycemia. Proteinuria detected by routine urinalysis should be evalutated by means of 24-hour urine collection. Urinary protein excretions greater than 150mg/d are significant. Elevated levels of bilirubin in the urine are seen with biliary obstruction. Microscopic analysis of the urinary sediment detects the presence of red or white blood cells, bacteria, casts, and crystals. Red cells may be indicative of bleeding due to tumor, stones, infection, coagulopathy, or trauma. White cells and bacteria are generally associated with infection. Disease processes at the level of the nephron produce tubular casts. Crystals may be indicative of abnormalities in oxalic, uric acid, or cystine metabolism [7].

Altered renal function & the effects of anesthetic agents: Most drugs commonly employed during aneasthesia are at least partly dependent on renal excretion for elimination. In the presence of renal impairment, dosage modifications may be required to prevent accumulation of the drug or active metabolites. Moreover, the systemic effects of azotemia can potentiate the pharmacologic actions of many of the agents. This latter observation may be the result of decreased protein binding of the drug, greater brain penetration due to some breach of the blood-brain, or a synergistic effect with the toxins retained in renal failure [7].

Intravenous agents:

Barbiturates: Patients with renal disease often exhibit increased sensitivity to barbiturates during induction, even though pharmacokinetic profiles appear to be unchanged. The mechanism appears to be an increase in free circulating barbiturate as a result of decreased protein binding Acidosis may also favor a more rapid entry of these agents into the brain by increasing the noniovized fraction of the drug [6].

Ketamine: Ketamine pharmacokinetics are minimally altered by renal disease. Some active hepatic metabolites are dependent on renal excretion and can potentially accumulate in renal failure. Ketamine's secondary hypertensive effect may be undesirable in hypertensive renal patients [6].

Propofol & Etomoidate: The pharmacokinetics of both propofol and etomidate are not significantly affected by impaired renal function. Decreased protein binding of etomidate in patients with hypoalbuminemia may enhance its pharmacologic effects [6].

Benzodiazepines: These drugs undergo hepatic metabolism and conjugation prior to elimination in urine.

Because most are highly protein-bound, increased sensitivity may be seen in patients with hypoalbuminemia. Diazepam should be used cautiously in the presence of renal impairment because of a potential for the accumulation of active metabolites [6].

Opioids: Most opioids currently in use in anesthetic management (morphine, meperidine, fentanyl, sufentanil, and alfentanil) are inactivated by the liver; some of these metabolites are then excreted in urine. With the exception of morphine and meperidine, significant accumulation of active metabolites generallyh does not occur with these agents. The accumulation of morphine (morphine-6-glucuronide) and meperidine metabolites has been reported to prolong respiratory depression in some patients with renal failure. Increased levels of normeperidine, a meperidine metabolite, have been associated with seizures. The pharmacokinetics of the most commonly used opioid agonist-antagonists (butorphanol, nalbuphine, and buprenorphine) are unaffected by renal failure [6].

Anticholinergic agents: In doses used for premedication, atropine and glycopyrrolate can generally be used safely in patients with renal impairment. Because up to 50% of these drugs and their active metabolites are normally excreted in urine, however, the potential for accumulation exists following repeated doses. Scopolamine is less dependent on renal excretion, but its central nervous system effects can be enhanced by azotemia [6].

Phenothiazines, H2 Blockers, & Related Agents: Most phenothiazines, such as promethazine, are metabolized to inactive compounds by the liver. Although pharmacokinetic profiles are not appreciably altered by renal impairment, potentiatin of their central depressant effects by azotemia can also occur. Their antiemetic actions are particularly useful in the setting of preoperative nausea. Droperidol may be partly dependent on the kidneys for excretion. Although accumulation may be seen following large doses in patients with renal impairment, relatively small doses of droperidol (< 2.5mg) are usually used clinically. All H2 receptor blockers are very dependent on renal excretion. Meoclopramide is partly excreted unchanged in urine and will also accumulate in renal failure.

Inhalational agents:

Volatile Agents: Volatile anesthetic agents are nearly ideal for patients with renal dysfunction because of their lack of dependence on the kidneys for elimination, their ability to control blood pressure, and generally minimal direct effects on renal blood flow. Although patients with mild to moderate renal impairment do not exhibit altered uptake or distribution, accelerated induction and emergency may be seen in severely anemic patients (hemoglobin < 5g/dL) with chronic renal failure; this observation may be ecxplained by a decrease in the blood:gas partition coefficient or a decrease in cause of its potential nephrotoxicity. Because of its potential nephrotoxicity, methoxyflurance is the only volatile agent that should not be used. Enflurane and sevoflurane may disease undergoing long procedures because of a similar potential for fluoride accumulation.

Nitrous Oxide: Many clinicians omit or limit the use of nitrous oxide to 50% in patients with renal failure in an attempt to increase arterial oxygen content in the presence of anemia. This rationale may only be justified in severely anemic patients (haemoglobin < 7 g/L), in whom even a small increase in the dissolved oxygen content may represent a significant percentage of the arterial to venous oxygen difference [6,8].

Muscle relaxants:

Succinylcholine: Succinylcholine can be safely used in the presence of renal failure, provided the serum potassium concentration is known to be less than SmgEq/L at the time of induction. When the serum potassium is higher or is in doubt, a nondepolarizing muscle relaxant should be used instead. Although decreased pseudocholinesterase levels have been reported in a few uremic patients following dialysis, significant prolongation of neuromuscular blockade is rarely seen [6,8].

Cisatracurium, Atracurium, & Mivacurium: Mivacurium is minimally dependent on the kidneys for elimination. Minor prolongation of effect may be observed due to reduced plasma pseudocholinesterase. Cisatraccurium and atracurium are degraded in plasma by enzymatic ester hydrolysis and nonezymatic Hofmann elimination. These agents may be the drugs of choice for muscle relaxation in patients with renal failure [6,8].

Vecuronium & Rocuronium: The elimination of vecuronium is parimarily hepatic, but up to 20% of the drug is elimainated in urine. The effects of large doses of vecuronium (> 0.1mg/kg) are only modestly prolonged in patients with renal insufficiency. Rocuronium primarily undergoes hepatic elimination, but prolongation by severe renal disese has been reported [6,8].

Curare: Elimination of curare is dependent on both renal and biliary excreted in urine. Increasingly prolonged effects are observed following repeated doses in patients with significant renal impairment. Smaller doses and longer dosing intervals are therefore required for maintenance of optimal muscle relaxation.

Pancuronium, Pipecuronium, alcuronium & Doxacurium: All these agents are primarily dependent on renal excretion (60-90%). Although pancuronium is metablolized by the liver into less active intermediates, its elimination half-life is still primarily dependent on renal excretion (60-80%). Neuromuscular function should be closely monitored if these agents are used in patients with abnormal renal function [6,8].

Metocurine, Gallamaine, & Decamethonium: All these agents are almost entirely dependent on renal excretion for elimination and should generally be avoided in patients with impaired renal function.

Reversai Agents: Renal excrection is the principal route of elimination for edrophonium, neostigmine, and pyridostigmine. The half-lives of these agents in patients with renal impairment are therefore prolonged at least as much as any of the mentioned above relaxants.

Anesthesia for patients with renal failrue

Preoperative considerations

Acute Renal Failure: This syndrome is a rapid deterioration in renal function that results in retention of nitrogenous waste products (azotemia). These substances many of which behave as toxins, are by-products of protein and amino acid metabolism. They include urea, guanidine compounds (including creating and creatinine), urates, aliphatic amines, and various peptides and metabolism of circulating proteins and peptides may also contribute to widespread organ dysfunction. Azotemia can be divided into prerenal, renal, and posternal types depending on its causes. Prerenal azotemia results from an acute decrease in renal perfusion. Renal azotemia is usually due to intrinsic renal disease, renal ischemia, or nephrotoxins. Postrenal azotemia is usually due to intrinsic renal disease, renal ischemia, or nephrotoxins. Posternal azotemia is the result of urinary tract obstruction or disruption. Both prerenal and postrenal azotemia are readily reversible in their initial stages but with time progress to renal azotemia. Most adult patients with renal failure develop oliguria. Nonoliguric patients (those with urinary outputs > 400mL/d) continue to form urine that is qualitatively poor; these patients tend to have greater preservation of GFR. Although glomerular filtration and tubular function are impaired in both cases, abnormalities tend to be less severe in nonoliguric renal failure. The course of acute renal failure varies widely, but the oliguria typically lasts for 2 weeks and is followed by a diuretic phase marked by a progressive increase in urinary output. This diuretic phase often results in very large urinary output and usually absent in nonoliguric renal failure. Urinary function improves over the course of several weeks but may not return to normal for up to 1 year [9].

Chronic Renal Failure: This syndrome is characterized by a progressive and irreversible decline in renal function over the course of at least 3-6 months. The most common causes are hypertensive nephrosclerosis, diabetic nephropathy, chronic glomerulonephritis, and polycystic renal disease. The full mainifestations of this syndrome - often referred to as uremia-are seen only after the GFR decreses below 25mL/min. patients with clearances below 10mL/min (often said to have end-stage renal disease) are dependent on dialysis for survival until they receive a successful transplant. Dialysis may take the form of intermittent hemodialysis through an arteriovenous fistula or continuous peritoneal dialysis via an implanted catheter. The generalized effects of uremia can usually be controlled by dialysis. Unfortunately, with time some uremic complications can become refractory. Moreover, some complications are directly related to the dialysis itself. Hypotension, neutropenia, hypoxemia, and the disequilibrium syndrome are generally transient and resolve within hours after dialysis. Factors contributing to hypotension during dialysis include the vasodilating effects of acetate dialysate solutions, autonomic neuropathy, and rapid removal of fluid. The intraction of white cells with cellophane-derived dialysis membranes can result in neutropenia and leukocytemediated pulmonary dysfunction leading to hypoxemia [9].

Disequilibrium syndrome: Is characterized by transient neurologic symptoms that appear be related to a more rapid lowering of extracellular osmolaity than intracellular osmolality.

Preoperative Evaluation: The generalized effects of azotemia mandate a thorugh evaluation of patients in renal failure. Most patients with acute renal failure requiring surgery are critically ill. Their renal failure is frequently associated with a postoperative complication or trauma. Patients with acute renal failure also tend to have accelerated protein breakdown. Optimal perioperative management is dependent on preoperative dialysis. Hemodialysis is more effective than peritoneal dialysis and can be readily accomplished via a temporary internal jugular, subclavian or femoral dialysis catheter. The need for dialysis in nonoliguric patients should be assessed on an individual basis. Patients with chronic renal failure most commonly present to the operating room for creation or revision of an arteriovenous fistula local or regional anesthesia. Regardless of the procedure or the anesthetic employed, complete evaluation is required to make certain that they are in optimal medical condition; all reversible manifestations of uremia should be controlled. Preoperative dialysis on the day of surgery or on the previous day is usually necessary. Physical and laboratory evaluation should focus on both cardiac and respiratory functions. Signs of fluid overload or hypovolemia should be sought. Intravascular volume depletion often results from overzealous dialysis. A comparison of the patient's current weight with previous predialysis and postdialysis weights may be helpful. Hemodynamic data, if available, and a chest film are invaluable in confirming clinical impressions. Arterial blood gas analysis is also useful in detecting hypoxemia and evaluating acid-base status. The electrocardiogram should be examined carefully for signs of hyperkalemia or hypocalcemia as well as ischemia, conduction blocks, and ventricular hypertrophy. Echocardiography can be invaluable for evaluating cardiac function in patients undergoing major surgical procedures because it can evaluate the ventricular ejection fraction, as well as detect and quantitate hypertrophy, wall motion abnormalities, and auscultation in patients with a pericardial effusion. Preoperative red blood cell transfusions should generally be given only to severely anemic patients (hemoglobin < 6-7g/dL) or when significant intraoperative blood loss is expected. A bleeding time and coagulation studies are advisable, especially if regional anesthesia is being considered. Serum electrolyte, BUN, and creatinine measurements can asses the adequacy of dialysis. Glucose

measurements are helpful in evaluating the potential need for preoperative insulin therapy. Preoperative drug therapy should be carefully reviewed for drugs with significant renal elimination. Dosage adjustments and measurements of blood levels (when available) are necessary to prevent drug toxicity (‘°).

Premedication: Alert patients who are relatively stable can be given reduced doses of an opioid or a benzodiazepine. Promethazine, 12.5-25mg in tramuscularly, is a useful adjunct for additional sedation and for its antiemetic properties. Aspiration prohpylaxis with an H2 blocker may be indicated in patients with nausea, vomiting, or gastrointestinal bleeding. Metoclopramide, 10mg orally or slowly intravenously, may also be useful in accelerating gastric emptying, preventing nausea, and decreasing the risk of aspiration. Preoperative medications-especially antihypertensive agents-should be continued until the time of surgery [10].

Intraoperative considerations:

Monitoring: The surgical procedure as well as the patient’s general medical condition dictate monitoring requirements. Because of the danger of occlusion, blood pressure should not be measured by a cuff in an arm with an arteriovenous fistula. Intra-arterial, central venous, and pulmonary artery monitoring are often indicated, especially for patients undergoing procedures associated with major fluid shifts. intravascular volume is often difficult to assess based on clinical signs alone. Direct intra-arterial blood controlled hypertensive monitoring may be indicated especially in diabetic patients with advanced renal disease undergoing major surgery; this group of patients may have up to 10 times the perioperative morbidity of diabetics without renal disease. The latter probably reflects the high incidence of advanced cardiovascular complications in the first group [8,10].

Induction: Patients with nausea, vomiting or gastrointestinal bleeding should undergo rapid-sequence induction with cricoid pressure. The dose of the induction agent should be reduced in debilitated or critically ill patients. Thiopental 2-3mg/kg or propofol 1-2mg/kg is often used. Etomidate, 0.2-0.4mg/kg may be preferable in hemodynamically unstable patients. An opioid, S-blocker (esmolol), or lidocaine may be used to blunt the hypertensive response to intubation. Succinylcholine, 1.5mg/kg can be used for endotracheal intubation if the serum potassium is less than 5mEq/L rocuronium (0.6mg/kg), cisatracurium (0.15mg/kg) atracurium (0.4mg/kg) or mivacurium (0.15mg/kg) should be used for intubating patients with hyperkalemia. Atracurium in this dosage generally causes little histamine release. Vecuronium, 0.1mg/kg, may be a suitable alternative, but some prolongation of its effects should be expected [4,10].

Maintenance: The ideal maintenance technique should be able to control hypertension with minimal effects on cardiac output, because increase in cardiac output is the principal compensatory mechanism for anemia. Volatile anesthetics, nitrous oxide, propofol, fentanyl, sufentanil, alfentanil, remifentanil, hydromorphone, and morphine are generally regarded as satisfactory maintenance agents. Isoflurane and desfurane may be the preferred volatile agents because they have the least effect on cardiac output. Nitrous oxide should be used cautiously in patients with poor ventricular function and should probably not be used in patients with very low hemoglobin concentrations (< 7g/dL) to allow the administration of 100% oxygen. Meperidine may not be a good choice because of the accumulation of normeperidine. Morphine may be used, but some prolongation of its effects should be expected. Controlled ventilation is safest for patients with renal failure. Spontaneous ventilation under anesthesia can result in respiratory acidosis that may exacerbate preexisting academia, leading to potentially severe circulatory depression and dangerous increases in serum potassium concentration. Respiratory alkalosis may also be detrimental because it shifts the hemoglobin dissociation curve to the left, can exacerbate preexisting hypocalcemia, and may reduce cerebral blood flow [4,10].

Fluid Therapy: Superficial operations involving minimal tissue trauma require replacement of only insensible fluid losses with 5% dextrose in water. Procedures associated with major fluid losses of shifts require isotonic crystalloids, colloids, or both. Lactated Ringer’s injection is best avoided in hyperkalemic patients when large volumes of fluid may be required, because it contains potassium (4 mEq/L); normal saline may be used instead. Glucose-free solutions should generally be used because of the glucose intolerance associated with uremia. Blood loss should generally be replaced with packed red blood cells. Blood transfustion either has no effect or may be benefical for patients in renal failure who are renal transplant candidates; such transfusion may decrease the likelihood of rejection following renal transplantation in some patients [7,11].

Objectives

General objective: To determine the effects of application of international protocol of fluid management in Sudanese recipient patients.

Specific objectives: To determine the effect of given (50m1 + urine output) replaced by normal saline to recipient patients in the following vital signs:

a) CVP

b) BP

c) Pulse

d) Temperature

Chapter 2

Methodology

Methodology

Study area

The study area was done at Ahmed Gasim Hospital in the period from November 2005 to April 2006.

Study design:

This is a non interventional, retrospective study in which search for the effect of application of international standard protocol of fluid management in haemodynamic status (i.e. CVP, BP, pulse & temp.) in Sudanese recipient Pt. in the first 24 hours.

Study sample, data collection: Sample was collected sixty kidney transplanted pt. (recipient) from sheets taken from postoperative recovery room, according to excluding criteria.

Excluding criteria:

a) Acute graft rejection.

b) Any case with injured ureter, obstructed or kincking.

c) Any case of delay graft function.

Data analysis: The data collected was digitally analyzed using a master sheet and computer program SPSS. The data were than presented in figures and table. Hypotheses were tested and 0.05 probability level was predetermined as the level of significance.

Determinant of good graft function:

i. Variables name, age, sex, wt.

ii. BP immediately after end of operation.

iii. Hourly BP for 24 hours.

iv. C.V.P immediately after end of operation.

v. Hourly CVP for 24 hours.

vi. Hourly pulse for 24 hours.

vii. Hourly temperature for 24 hours.

viii. Hourly Control of volume, and type of fluid.

By application of international standard protocol (hourly urine output + 30m1 of N/S - modified by Sudanese physician to 50m1 because of hot weather in Sudan.

Chapter 3

Results

Results

In this study, in renal transplanted Patients (recipient) were monitored for 24houres post operatively, monitoring included vital signs such as BP, pulse, C.V.P, temp. , total fluid intake and urine output according to modified international standard protocol for fluid replacement (hourly urine output +50m1) of normal saline .

1.1. Test for systolic BP after the end of operation has a mean (Table 1) equal to 147.9 and after 24 hours is 130.8 which is significant according to value of (sig) less than 0.05 (Figure 1) resulting, the systolic BP is reducing after 24 hours.

1.2. Test for diastolic BP after end of operation has a mean (Table 2) equal to 90.18 and after 24 hours is 80.17 which is significant according to value of (sig) less than 0.05 (Figure 2) resulting, the diastolic BP is reducing after 24 hours.

1.3. Test for pulse (Table 3) reveal that there is obvious different between end of operation and 24 hours later because of the level of (sig) less than 0.05.

1.4. Test for C.V.P reveal that there is a different between end of operation and 24 hours later because the level of (sig) (Table 4) less than 0.05.

1.5. Test for Temp. revealed a significant changes after 24 hours because (sig) (Table 5) less than 0.05.

1.6. They were deferent in demographic variable. Male patients (Table 6) more than female patients (Figure 3). Aged group 24-37 years (Table 7) was the most frequent group (Figure 4). Weight between 50 to 70 kg (Figure 5) had a high frequency (Table 8).

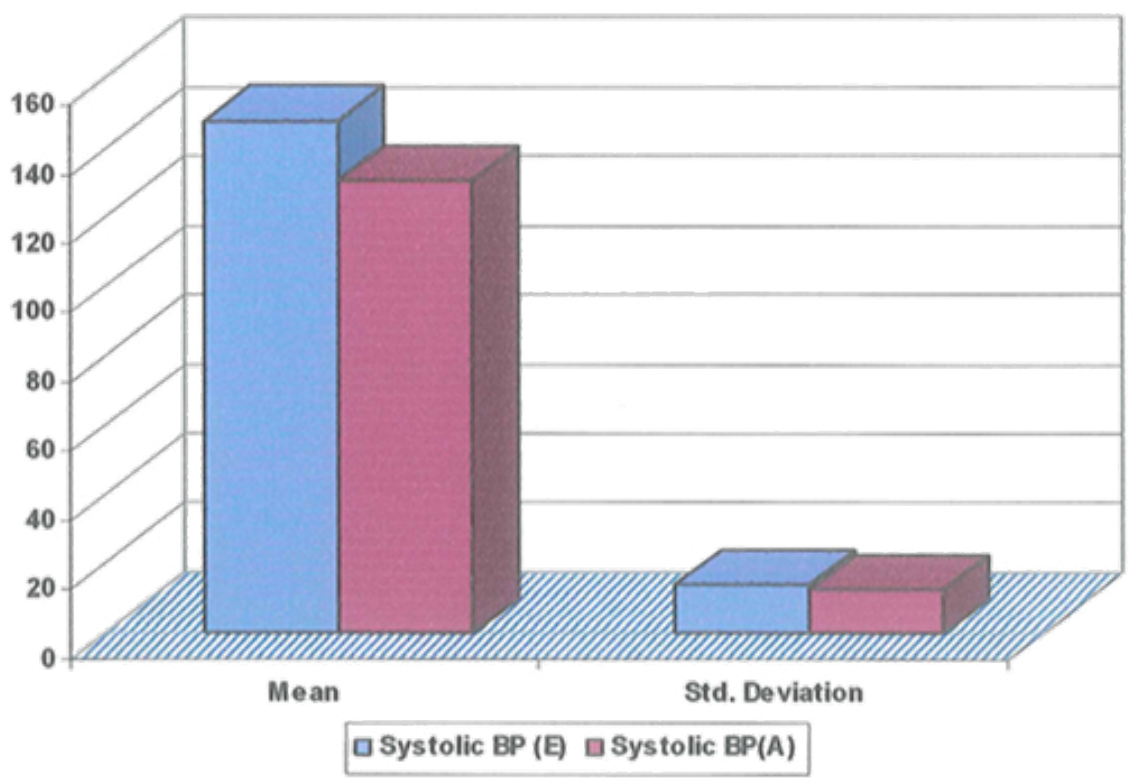

Figure 1: Systolic BP (E) and (A).

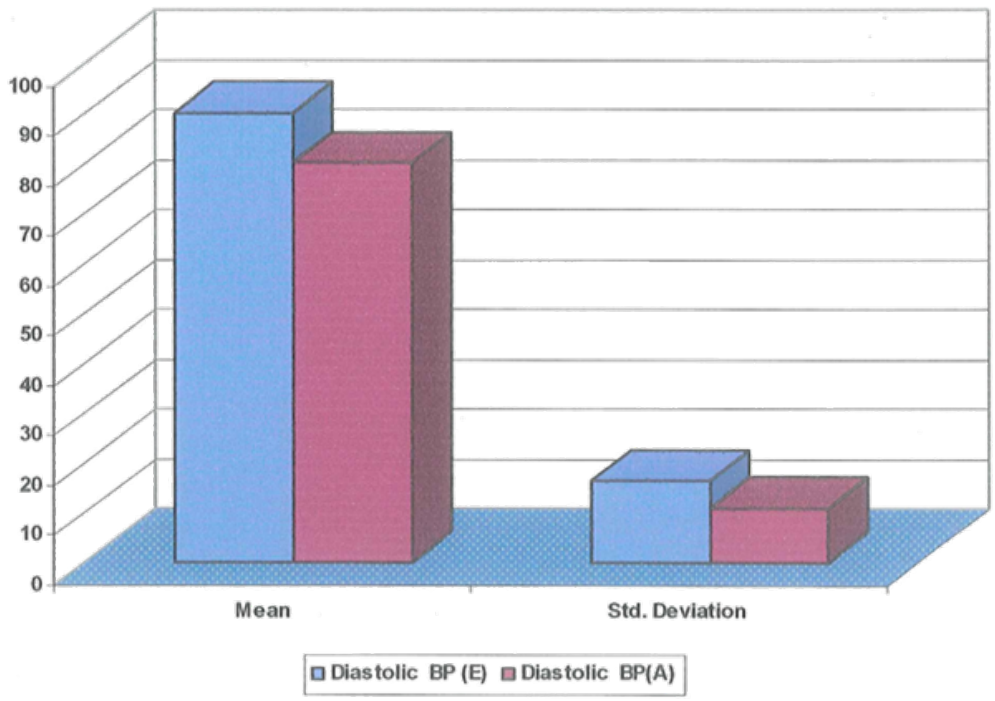

Figure 2: Diastolic BP (E) and (A).

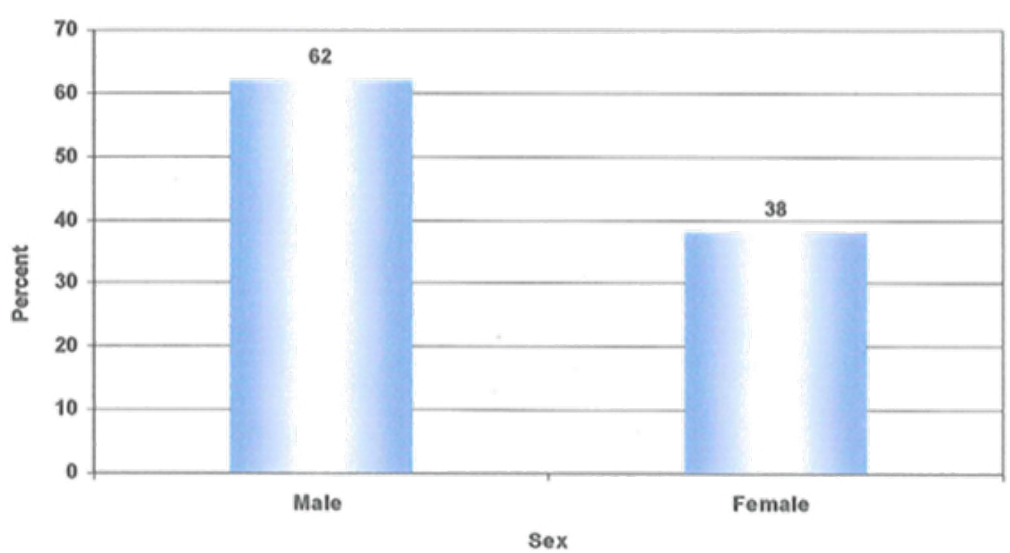

Figure 3: Gender distribution among the study group.

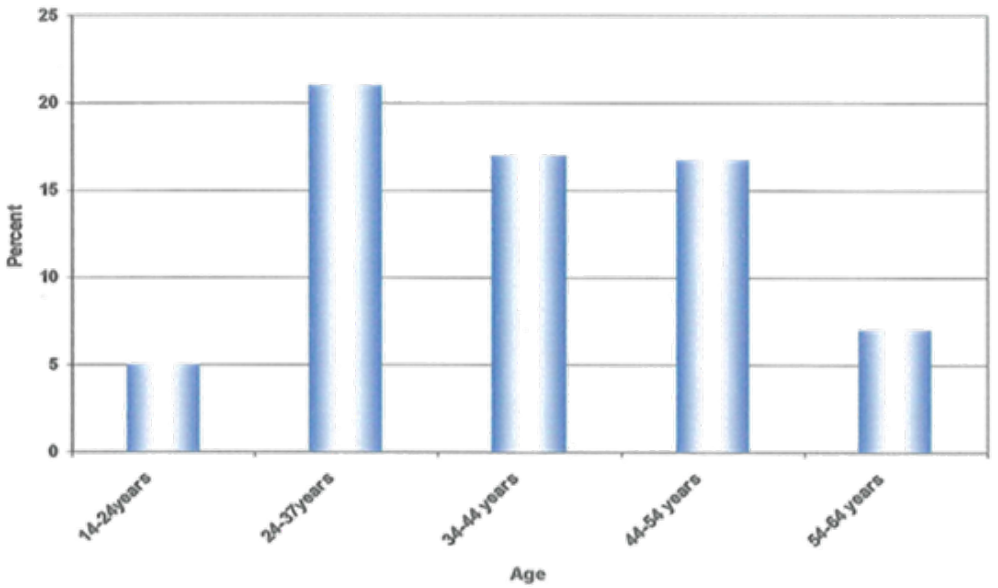

Figure 4: Age distribution among the study group.

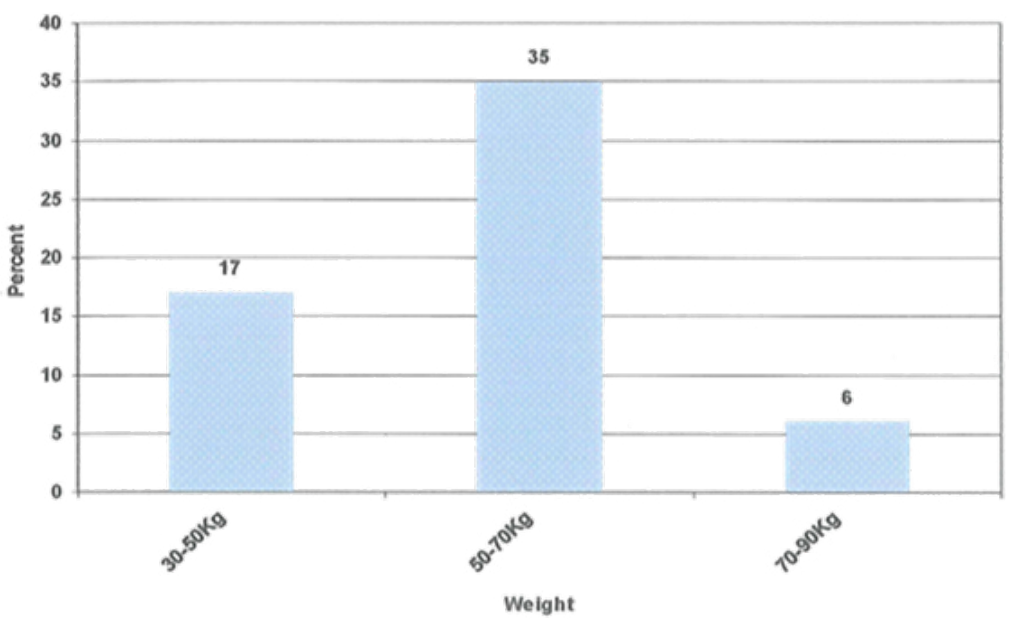

Figure 5: Weight distribution among the study group.

Table 1: Systolic BP (E) and (A).

	Mean	No	Std. Deviation	Correlation	Sig.
Systolic BP (E)	147.9	60	14.01	0.183	0
Systolic BP(A)	130.8	60	12.53	0.183	0

Table 2: Diastolic BP (E) and (A).

	Mean	No	Std. Deviation	Correlation	Sig.
Diastolic BP (E)	90.18	60	16.54	0.594	0
Diastolic BP(A)	80.17	60	11.03	0.594	0

Table 3: Pulse (E) and (A).

	Mean	No	Std. Deviation	Correlation	Sig.
Pulse (E)	89.74	60	16.99	0.02	0.047
Pulse (A)	84.17	60	11.32	0.02	0.047

Table 4: CVP (E) and (A).

Pulse (E)	89.74	60	16.99	0.02	0.047
CVP (A)	9.57	60	2.18	0.34	0.008

Table 5: Temperature (E) and (A).

	Mean	No	Std. Deviation	Correlation	Sig.
Temp. (E)	34.85	60	0.86	0.133	0
Temp. (A)	36.78	60	0.426	0.133	0

Table 6: Gender.

Gender	Frequency	Percent
Male	37	61.7
Female	23	38.3
Total	60	100

Table 7: Age.

Age	Frequency	Percent
14-24years	5	8.3
24-37years	21	35
34-44 years	17	28.2
44-54 years	16.7	16.7
54-64 years	7	11.7
Total	60	100

Table 8: Gender.

Weight	Frequency	Percent
30-50Kg	17	28.3
50-70Kg	35	58.3
70-90Kg	6	10
Total	58	96.7

Table 9: Fluid intake and urine output.

	Mean	No	Std. Deviation	Sig.
Fluid intake	1129.8	60	4002	0
Urine output	9194.3	60	9194.3	0

Chapter 4

Discussion

Discussion

The incidence of chronic renal failure is increasing throughout the world. The function of many organ system of great interest to anaesthetists especially the renal system. Chronic renal failure and end stage renal disease are functional diagnosis characterized by progressive decrease in glomerular filtration rate (GFR). For most patients with kidney failure, kidney transplantation has the greatest potential for restoring a healthy, productive life. Post operative hemodynamic evaluation is critical for several reasons for routine postoperative management; to optimize graft function; to assess the significance of urine output and to undertake prompt regulation of vital sings. In this study, a large concentration given to the first 24 hours period, from end of the surgery, in intensive care unit. Application of international standard protocol of fluid therapy in this period, given of (30mL + urine output) replaced by normal saline which modified by Sudanese physicians to 50mL due to hot weather in Sudan.

A much better reading of blood pressure was recorded, most of patients had a high BP at the end of operation but started to decrease to normal after 24 hours (table 4,5), because the initial blood flow to the graft is primarily dependent on mean systemic blood pressure, there is intrinsic autoregulation of graft vascular reactivity which lead to kept renal blood flow and glomerular filtration rate relatively constant or increased also changes toward the normal range (8 - 10) mmHg of the central venous pressure occurring during the first 24 hours (Table 4). Also this study determine the relationship between central venous pressure and blood pressure in critically ill patients, results showed that the mean difference between CVP and BP has narrow different according to 95% confidence interval of the difference.

Overall, the direction of change in blood pressure (rise or drop) predicted a same direction of change in CVP with high accuracy. However, as changes in BP parallel, in direction with changes in CVP, serial measurements of blood pressure may have a value in determining volume status and guiding fluid therapy in recipient patients. There is obvious different in pulse between end of operation and 24 hours later (Table 3). There is a significant reversal to normal temperature after 24 hours (Table 5). Urine output has potent relationship with fluid intake i.e. receiving large amount of fluid leads to production of large amount of urine which is an indicator of good graft function (Table 9), the patient should be kept mildly hypervolemic to counteract with the great urine output due to increase GFR. In demographic variable, found that males are more than females in renal transplantation surgery. Most of them aged between 24 to 37 years and weight between 50 to 70 kg.

Conclusion

International standard protocol of fluid management has a main role in maintenance of vital signs such as central venous pressure, blood pressure, pulse, and temperature as the study results mentioned before. Blood pressure decreased to normal level despite the large amount of normal saline which given to patients. Central venous pressure also come near to its normal level in most of patients. And both CVP and BP has same direction of change. There is a significant changes in pulse in most patients. Temperature has a significant change towards the normal in all patients.

Recommendations

Application of international standard protocol of fluid management in Sudanese patients has a good outcome graft function. So it is suitable for Sudanese recipient patients. Giving of large fluid volume(normal saline) has a great effect in maintenance of vital signs.

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