

INTRODUCTION

Kidney Failure (KF) occurs when both kidneys fail to function as desired and are not able to get rid off the harmful toxins and excessive fluid. Haemodialysis (HD) is a method for removing waste products such as creatinine and urea as well as free water from blood when kidneys are in fails (*Crawford & Ierma, 2008*).

In Egypt, chronic renal failure (CRF), the incidence is about 225 per million populations, from them 1700 children suffers of CRF. The HD remains the only alternative treatment over the last decade. There has been a significant improvement in HD technique due to improvement in technology of dialysis machine (*Wheatheral, 2007; Ministry of Health and Population [MOH], 2009*).

Dialysis nurses provide extensive care for children who suffer from renal failure (RF) or chronic kidney condition. The evolution of dialysis technologies requires nurses who know how to operate the equipment (*Kopppe & Shina, 2007*). Dialysis nurses' responsibilities vary and depend upon educational and work experience (*Borah & Catch, 2006*).

The HD pediatric patient is cared by highly skilled nurse in haemodialysis unit (HDU) where nursing goals are formulated and implemented to orders each nursing diagnosis identified for HD pediatric patient. Successful implementation of the nursing

care plan helps for improving health state of the pediatrics' patient and improving the quality of life [QOL] (*Cheigh et al., 2006*).

The process of evaluation of care provided to the HD patient requires variety of skills. The skills depend on knowledge of standard care, normal respond to care provided, and the ability to monitor the effectiveness of nursing intervention (*Kobrin, 2007*).

Significance of the Study:

Chronic renal failure is a major health problem and is considered as the most common disease of childhood. Worldwide, slightly more than 30 people in every 100,000 develop KF each year, and the annual rate of children who develop CRF is 1 or 2 new cases in every 100,000 children. The risk increases steadily with age (*National Kidney and Urologic Diseases Information Clearinghouse [NKUDICH], 2010*). In Egypt, the CRF incidence is about 225 per million populations; from them 1700 children suffer of CRF. Nurses are the key members in helping with uremia by providing care for children with CRF undergoing HD (*Harambat et al., 2012*). The HD is the therapy used most often among pediatric patients with end-stage renal disease [ESRD] (*Hafeez, 2002*)

AIM OF THE STUDY

The aim of the study was to assess nurses' knowledge versus their performance towards care of children undergoing HD procedures in PDUs in Cairo through:

- Assessing nurses' knowledge during and after connecting the child to HD machine.
- Assessing nurses' performances regarding care given to the child before, during and after connecting to the HD machine.
- Comparing between nurses' knowledge and practices to judge if nurse practices are based on scientifically sound knowledge.
- Assessing children satisfactory level with nurses' care.

Research Questions

- 1- Do nurses have acceptable level of performance (competent)?
- 2- Do the competent nurses have sound scientifically knowledge?
- 3- Do nurses' socio-demographic characteristics have influence on their performance?
- 4- Are children satisfied with the care given to them?

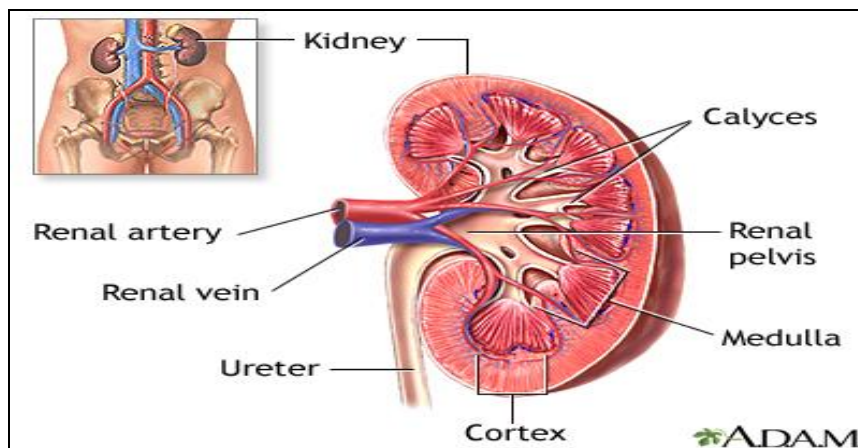
REVIEW OF LITERATURE

Anatomy of Kidney

The kidneys lie in the retroperitoneal space slightly above the level of the umbilicus. They range in length and weight, from approximately 6 cm and 24 g in a full-term newborn to 12 cm or more and 150 g in an adult (*Price & Finney, 2010*).

The cortex contains the glomeruli, proximal and distal convoluted tubules, and collecting ducts, and an inner layer, the medulla, which contains the straight portions of the tubules, the loops of Henle, the vasa recta, and the terminal collecting ducts (*Arant, 2009*) (Figure 1).

Figure (1): Kidney Anatomy

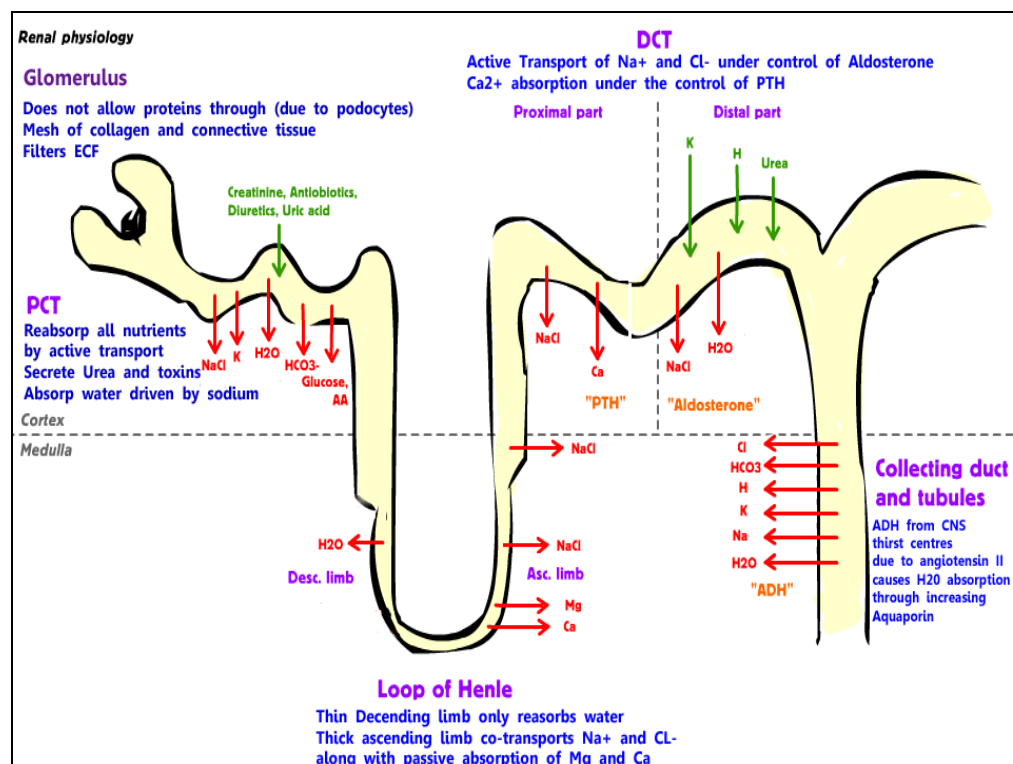


Dugdale, DC. (2011): Kidney anatomy. Medline Plus. Available: at <http://www.nlm.nih.gov/medlineplus/ency/imagepages/1101>.

Physiology of the Renal System

Renal physiology (Latin *rēnēs*, "kidneys") (Fig. 2) is the study of the physiology of the kidney. This encompasses all functions of the kidney, including re-absorption of glucose, amino acids, and other small molecules; regulation of sodium, potassium, and other electrolytes; regulation of fluid balance and blood pressure; maintenance of acid-base balance; the production of various hormones including erythropoietin, and the activation of vitamin D (*Ferri, 2014; Encyclopedia, 2014a*).

Figure (2): Physiology of Renal System



Encyclopedia, (2014a): Renal physiology. Available at: http://en.Wikipedia.org/wiki/Renal_physiology

Renal function, in nephrology, is an indication of the state of the kidney and its role in renal physiology. Glomerular filtration rate (GFR) describes the flow rate of filtered fluid through the kidney. Creatinine clearance rate (Ccr or CcCl) is the volume of blood plasma that is cleared of creatinine per unit time and is a useful measure for approximating the GFR. The Ccr exceeds GFR due to creatinine secretion, which can be blocked by cimetidine (*Stevens et al., 2006; Guyton & Hall, 2012*).

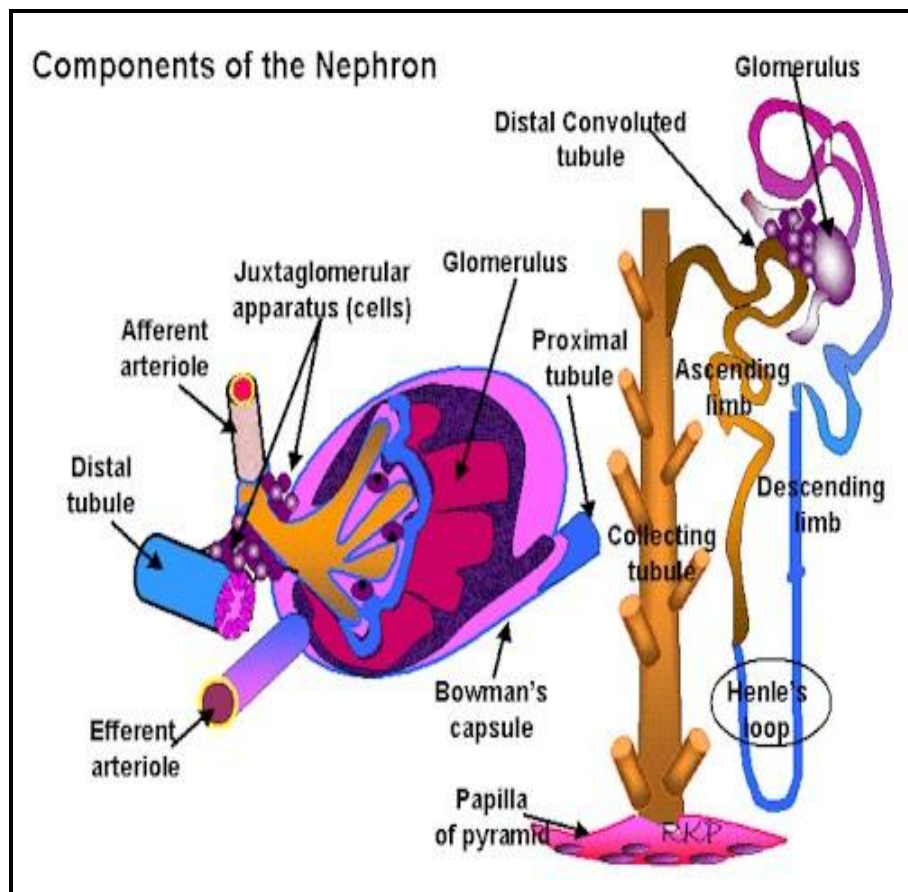
According to *Schwartz et al., (2008)* creatinine clearance (Ccr) is calculated from the creatinine concentration in the collected urine concentration (Ucr), urine flow rate, and the plasma concentration (PCr). Since the product of urine concentration and urine flow rate yields creatinine excretion rate, which is the rate of removal from the blood, creatinine clearance is calculated as removal rate per min ($Ucr \times V$) divided by the plasma creatinine concentration. This is commonly represented mathematically as:

$$C_{Cr} = \frac{U_{Cr} \times V}{P_{Cr}}$$

The blood supply to each kidney usually consists of a main renal artery that arises from the aorta; multiple renal arteries may occur. The main artery divides into segmental branches within the medulla and these into inter lobar arteries that pass through the medulla to the junction of the cortex and medulla (*Schwartz et al., 2008*).

Each kidney contains approximately 1 million nephrons (glomeruli and associated tubules). In humans, formation of nephrons (Fig. 3) is complete at birth. But functional maturation with tubular growth and elongation continues, because new nephrons cannot be formed after birth, progressive loss of nephrons may lead to renal insufficiency (*Yared & Ichikawa, 2009*).

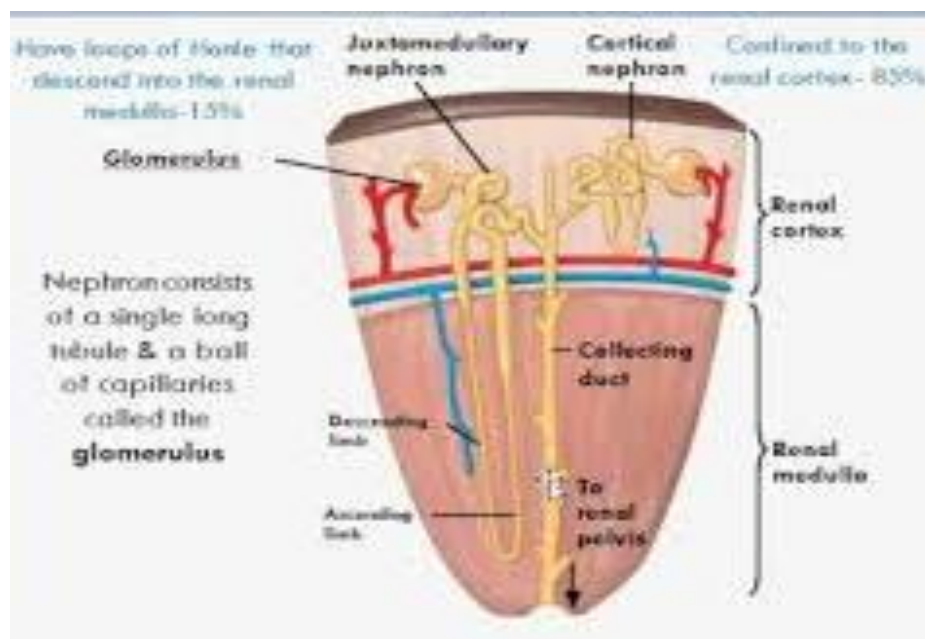
Figure (3): Components of the Nephron.



Schmitt, CP. & Mehls, O. (2011): Mineral and bone disorders in children with chronic kidney disease. Nat. Rev Nephrol; 7: 624- 634.

As the blood passes through the glomerular capillaries, the plasma is filtered through the glomerular capillary walls. The ultra-filtrate, which is cell free, contains all the substances in the plasma (electrolytes, glucose, phosphate, urea, creatinine, peptides, low molecular weight proteins) except proteins (like albumin and the globulins) having a molecular weight of 68,000 or more. The filtrate is collected in Bowman space and enters the tubules, where its composition is modified by solute and fluid secretion and absorption in accordance with tightly regulated homeostatic mechanisms until it leaves the kidney as urine (Fig. 4) (*Schwartz et al., 2008*).

Figure (4): Nephron Functional Unit.



Mikulov, F. (2010): The Kidney: Physiology and Pathophysiology, 3rd ed. New York: Lippincott Williams & Wilkins. pp. 2597–2644.

Preterm infants of 30 weeks' gestational age (GA) have a cc of less than 10 ml/min/1.73 m² within the first 24 to 40 hours of birth (*Veille et al., 2009*), whereas cc is higher in full term infants and ranges between 10 and 40 ml/min/1.73 m² (*Chevalier, 2010*).

Glomular filtration rate is seen over the first 2 weeks of life in term infants and reaches adult values by 2 years of age (*Bueva & Guignard, 2008*). Fetal GFR correlates well with both GA and body weight (*Kleinman & Lubbe, 2010*). Blood enters the kidneys via the renal arteries which are branches of aorta. Each kidney receives approximately 625 ml/min of blood; this constituted a total 25% of the cardiac output (*Lewis et al., 2010*).

Glomerular filtration is the process by which fluids and salts moved from the vascular system into the tubular system of the nephron, from an area of relatively high pressure to an area of low pressure. The GFR is the best indicator of how well kidneys are working. GFR measures the plasma volume that frame. In a person with normal renal function, the GRF is about 180 L/day. The GRF can be used as an indicator of adequacy of renal function (*Christensen & Kockrow, 2005; Schwartz et al., 2008*).

Overview of Chronic Kidney Disease

Chronic kidney disease (CKD) is defined by a presence of kidney damage (for example, any structural or functional

abnormality involving pathological, laboratory or imaging findings) for ≥ 3 months or a $\text{GFR} < 60 \text{ ml/min/1.73 m}^2$ for ≥ 3 months (*Crawford & Lerma, 2008*).

Chronic kidney disease and RF have been recognized as significant medical problems for most of the last 2 centuries and, until relatively recently, were uniformly fatal. Scientific and technologic improvements during the second half of the 20th century provided renal replacement therapy (RRT) as a life-sustaining option for many individuals who otherwise may have died. The impact of these medical advancements has been remarkable (*Gulati, 2012*).

Chronic kidney disease is characterized by an irreversible deterioration of renal function that gradually progresses to (ESRD). The CKD has emerged as a serious public health problem. Data from the United States Renal Data System (USRDS) show that incidence of KF is rising among adults and is commonly associated with poor outcomes and high cost. Moreover, in the past 2 decades, the incidence of the CKD in children has steadily increased, with poor and ethnic minority children disproportionately affected (*USRDS, 2011*).

The major health consequences of CKD include not only progression to KF but also an increased risk of cardiovascular disease (CVD). Evidence-based clinical practice guidelines (EBCPG) support early recognition and treatment of CKD-related complications to improve growth

and development and, ultimately, the QOL in children with this chronic condition. Appropriate pediatric care may reduce the prevalence of this complex and expensive condition (*National Kidney Foundation [NKF], 2002*).

Chronic kidney disease (CKD)—or chronic renal failure (CRF), as it was historically termed—is a term that encompasses all degrees of decreased renal function, from damaged-at risk through mild, moderate, and severe CKD. CKD is a worldwide public health problem. In the United States (US), there is a rising incidence and prevalence of KF, with poor outcomes and high cost. The Kidney Disease Outcomes Quality Initiative (KDOQI) of the NKF established a definition and classification of CKD (*Levey et al., 2003; Arora, 2014*).

The definition and classification of CRD may help identify affected individuals, possibly resulting in the early institution of effective therapy. To achieve this goal, the Kidney Disease Outcomes Quality Initiative (KDOQI) working group of the KF defined CKD as "evidence of structural or functional kidney abnormalities (Abnormal urinalysis, imaging studies, or histology) that persist for at least 3 months, with or without a decreased GFR, as defined by a GFR of less than 60 ml/min per 1.73 m²" (*Kopple, 2001; NKF, 2002; KDOQI, 2009*).

Chronic kidney disease also was known as a progressive loss in renal function over a period of months or years. The symptoms of worsening kidney function are non-specific, and might include feeling generally unwell and experiencing a reduced appetite. Often, CKD is diagnosed as a result of screening of people known to be at risk of kidney problems, such as those with high blood pressure (BP) or diabetes and those with a blood relative with CKD. The CKD may also be identified when it leads to one of its recognized complications, such as CVD, anemia or pericarditis (*NKF, 2002; Sarnak et al., 2003; Amaresan, 2005; Thakar et al., 2011*).

Kidney failure occurs when both the kidney fails to function as desired and is not able to get rid off both harmful toxins and excessive fluid. The HD is a method for removing waste products such as creatinine and urea as well as free water from blood when kidney is in fail (*Crawford & Lerma, 2008*).

Chronic renal disease (CRD) may be the result of congenital, acquired, inherited, or metabolic renal disease (MRD), and the underlying cause correlates closely with the age of the pediatric patients at the time when the CRF is first detected. The CRF in children younger than 5 years is most commonly a result of congenital abnormalities such as renal hypoplasia (RHP), dysplasia, and/or obstructive uropathy (OUP) (*Vogt & Avner, 2011*).

Etiology and Pathophysiology

The main causes of CKD in children include hypoplastic (HP) or dysplastic kidneys (DPK), reflux nephropathy (RNP), focal segmental glomerulosclerosis (FSGS) as a variant of childhood nephritic syndrome (NS), polycystic kidney disease (PCKD), autosomal-recessive and autosomal-dominant varieties (*Gulati, 2012*). The three most common causes of CKD are diabetes mellitus (DM), hypertension, and glomerulonephritis (*USRDS, 2011*).

Despite the diverse etiologies, once CKD develops, the subsequent response of the failing kidney is similar. The kidney initially adapts to damage by increasing the filtration rate in the remaining normal nephrons, a process called adaptive hyperfiltration. As a result, pediatric patients with mild CKD often have a normal or near-normal serum creatinine concentration. Additional homeostatic mechanisms permit the serum concentrations of sodium, potassium, calcium, and phosphorous and total body water to also remain within the reference range, particularly among those with mild to moderate stages of CKD (*Gulati, 2012*).

Adaptive hyperfiltration, although initially beneficial, appears to result in long-term damage to the glomeruli of the

remaining nephrons, which is manifested by pathologic proteinuria and progressive kidney insufficiency (PKI). This irreversibility appears to be responsible for the development of end stage renal failure (ESRF) among persons in whom the original illness is either inactive or cured (*Gulati, 2012*).

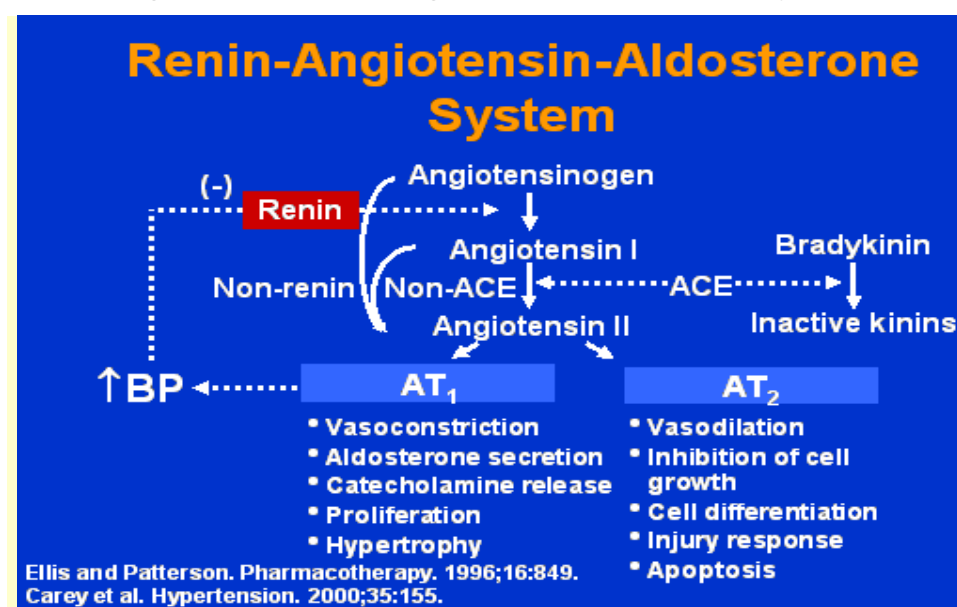
Although the underlying problem that initiated CKD often cannot be treated primarily, extensive studies in experimental animals and preliminary studies in humans suggest that progression in CRD may be largely due to secondary factors that are unrelated to the activity of the initial disease. These include anemia, osteodystrophy, systemic and intraglomerular hypertension, glomerular hypertrophy, proteinuria, metabolic acidosis, hyperlipidemia, tubulointerstitial disease (TID), systemic inflammation, and altered prostanoid metabolism. This common sequence of events in diverse types of CKD is the basis for the common management plan for children with CKD, irrespective of the etiology (*Gulati, 2012*).

The classification system describes the stages according to level of estimated GFR, not serum creatinine levels; mild chronic renal insufficiency (CRI): GFR 50-75 mL/min/1.73/m² moderate chronic renal insufficiency: GFR 25-50 mL/min/1.73m². CRF: GFR 10-25 mL/min/1.73m² ESRD: GFR < 10 mL/min/1.73m² (*Kliegman et al., 2011*)

According to the *National Kidney Foundation [NKF]*, (2002), CKD is initially without specific symptoms and is generally only detected as an increase in serum creatinine or protein in the urine. As the kidneys' function decreases:

- Blood pressure (BP) is increased due to fluid overload and production of vasoactive hormones created by the kidney via the renin-angiotensin system (RAS) (Fig. 5) increasing one's risk of developing hypertension and/or suffering from congestive heart failure (CHF).

Figure (5): Renin-angiotensin-Aldosterone System



Ellis, ML. & Patterson, JH. (2000): A new class of antihypertensive therapy: angiotensin II receptor antagonists. Pharmacotherapy; 16(5): 849- 860.

Carey, RM.; Wang, ZQ. & Siragy, HM. (2000): Role of the angiotensin type 2 receptor in the regulation of blood pressure and renal function. Hypertension; 35 (1 Pt 2): 155- 163.