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Renal Doppler Sonography for Assessment of Renal Injury in the Asphyxiated Newborn Infant

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Abstract

Background: Asphyxia is an important cause of acute kidney injury (AKI) and transient kidney impairment, there is a high incidence of AKI among the asphyxiated infants (50 – 72%). The normal renal ultrasound appearance in a neonate typically shows higher cortical echogenicity than in older child. Normally the parenchymal echogenicity is equal to or greater than that of liver and spleen. Doppler examination of the renal artery includes complete evaluation of the kidneys. Left and right decubitus patient positions are preferred for the kidney examination (left decubitus for the right kidney and vice versa). Both kidneys are examined carefully with respect to size, echogenicity and smoothness of outline, together with assessment of the corticomedullary differentiation.There are two approaches are used in Doppler examination, anterior abdominal approach and flank approach.

Keywords: Perinatal Asphyxia, Renal Doppler Sonography.

Pediatric Renal Doppler Sonography:

Septicemia is a potential clinical status which is a result of irresistible sickness or a inflammatory Babies and small children are examined without special preparation, but may be given juice or milk during the examination in order to calm them, to increase hydration, and to provide an acoustic window through the fluid – filled stomach. Sedation is very rarely necessary. The aorta and main renal arteries are examined by means of an anterior left paramedian as well as a left axillary approach with longitudinal and transverse views. The color mode is used to trace the renal arteries which are then examined with serial pulsed Doppler samples, especially in areas of high – velocity flow. Even if the entire renal artery cannot be outlined because of overlying intestinal gas, the retrocaval portion of the right renal artery and the hilar arteries can usually be analyzed. A segmental or interlobar artery in each third of the kidney (upper, middle and lower) is then studied by pulsed Doppler and the resistivity index (of Pourcelot) (RI) or pulsatility index is calculated **(1).**

The renal arterial bed normally has low resistance, and there is a constant flow into the kidney throughout the cardiac cycle. The resistivity index estimates impedance to arterial inflow. Normally the RI is < 0.7, but there is a wide range of normal measurements, depending in part on the age of the patient. In the neonatal period, probably concurrent with the physiologic low GFR, the resistance of the renal arterial bed is somewhat higher: RI = 0.7 to 0.8 and it decreases during childhood to reach 0.55-0.6 in adolescents. The normal RI in adults is estimated at 0.65 ± 0.10.

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Because there is a range of normal values of the RI, serial tracings are more reliable in detecting an abnormality, especially if the Doppler findings are correlated with the clinical state of the patient **(1).**

Any increase in intrarenal arterial pressure results in decreased flow. Diastolic flow occurs at the lowest pressure during the cardiac cycle, so it will decreaseor disappear before systolic Doppler flow curves are affected appreciably. The causes of increased intrarenal resistance to flow can be classified as intravascular, perivascular and perirenal **(1).**

Technique of Renal Duplex

I. Principle of Doppler Examination :-

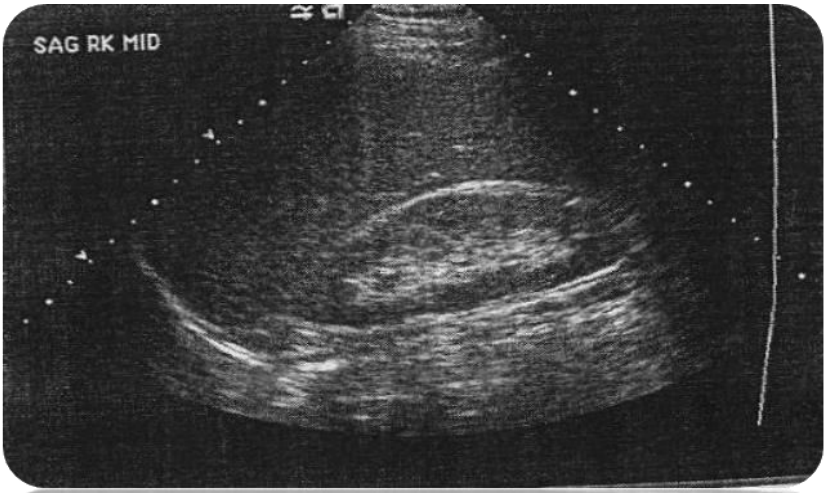
Duplex ultrasound evaluation of renal vessels is a technically difficult examination. Most sonographers are initially frustrated by difficulties with locating and following the renal arteries and with obtaining Doppler signals from vessels. However, with a little patience a sonographer can become adept at this study and perform the examination in a reasonable period of time. Literature reports indicate that as many as 95% of main renal arteries can be adequately examined in adult patients. The key to the renal Doppler examination is accurate demonstration of the vascular anatomy. This requires an understanding of renal vascular anatomy as well as the ability to recognize normal and abnormal Doppler waveforms **(2).** Throughoutthe course of examination of the renal artery, color Doppler is frequently switched on to confirm the nature and direction of flow. Proper color Doppler adjustment allows the examiner to "screen" the vessel quickly and then place the Doppler sample volume to determine the flow velocity. Pulsed Doppler spectral analysis must be used in conjunction with color flow imaging as it provides quantitive information through the measurement of blood flow velocity **(2).**

II. Patient Preperation

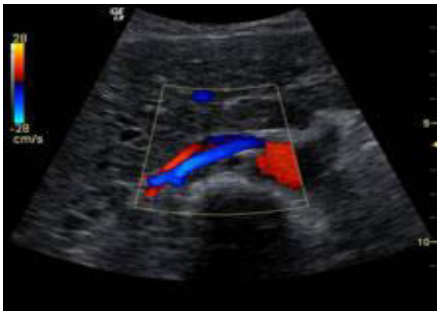
For successful abdominal examination, adequate patient preparation is important. All patients should fast prior to abdominal vascular scanning as fasting tends to reduce the amount of bowel gas, which produces scatter and attenuates the ultrasound beam **(2).**

III. Renal Ultrasound

Doppler examination of the renal artery includes complete evaluation of the kidneys. Left and right decubitus patient positions are preferred for the kidney examination (left decubitus for the right kidney and vice versa). Both kidneys are examined carefully with respect to size, echogenicity and smoothness of outline, together with assessment of the corticomedullary differentiation. The adrenal areas should also be carefully examined to exclude any obvious mass. The time taken to perform a complete vascular examination of the kidney is wasted if the initial examination has missed a renal mass, an adrenal tumor, or a small or absent kidney **(3).**



(Fig. 1): Longitudinal images of normal right kidney **(4)**



Scanning Technique

A 3.5-5 MHz probe is typically used to scan the kidney (9-11 MHz linear probe in neonates).

For the right kidney,have the patient lie supine and place the probe in the right lower intercostal space in the mid axillary line. Use the liver as your "acoustic window" and aim the probe slightly posteriorly (toward the kidney). Gently rock the probe (up and down or side to side) to scan the entire kidney. If needed, you can have the patient inspire or exhale, which allows for subtle movement of the kidney. Obtain longitudinal (long axis), and transverse (short axis) views **(5).**

For the left kidney,have the patient lie supine or in the right lateral decubitus position. Place the probe in the lower intercostal space on the posterior axillary line. The placement will be more cephalad and posterior than when visualizing the right kidney. Again gently rock the probe to scan the entire kidney. Obtain longitudinal and transverse views **(5).**

Renal Doppler:

Patient Positioning & scanning orientation

There are two approaches are used in Doppler examination, anterior abdominal approach and flank approach **(6).**

In anterior abdominal approach, the patient is supine and with a transverse scan just to the left of the midline, the aorta is localized, and superior mesenteric artery identified. Approximately 1 cm below the origin of the superior mesenteric artery, both renal arteries can be identified arising from the lateral surface of the aorta. On moving the probe slightly to the left of midline and angling it toward the patient's right side. The right renal artery is seen to turn posteriorly and laterally underneath the IVC, as it is the only major vessel posterior to the IVC **(6).**

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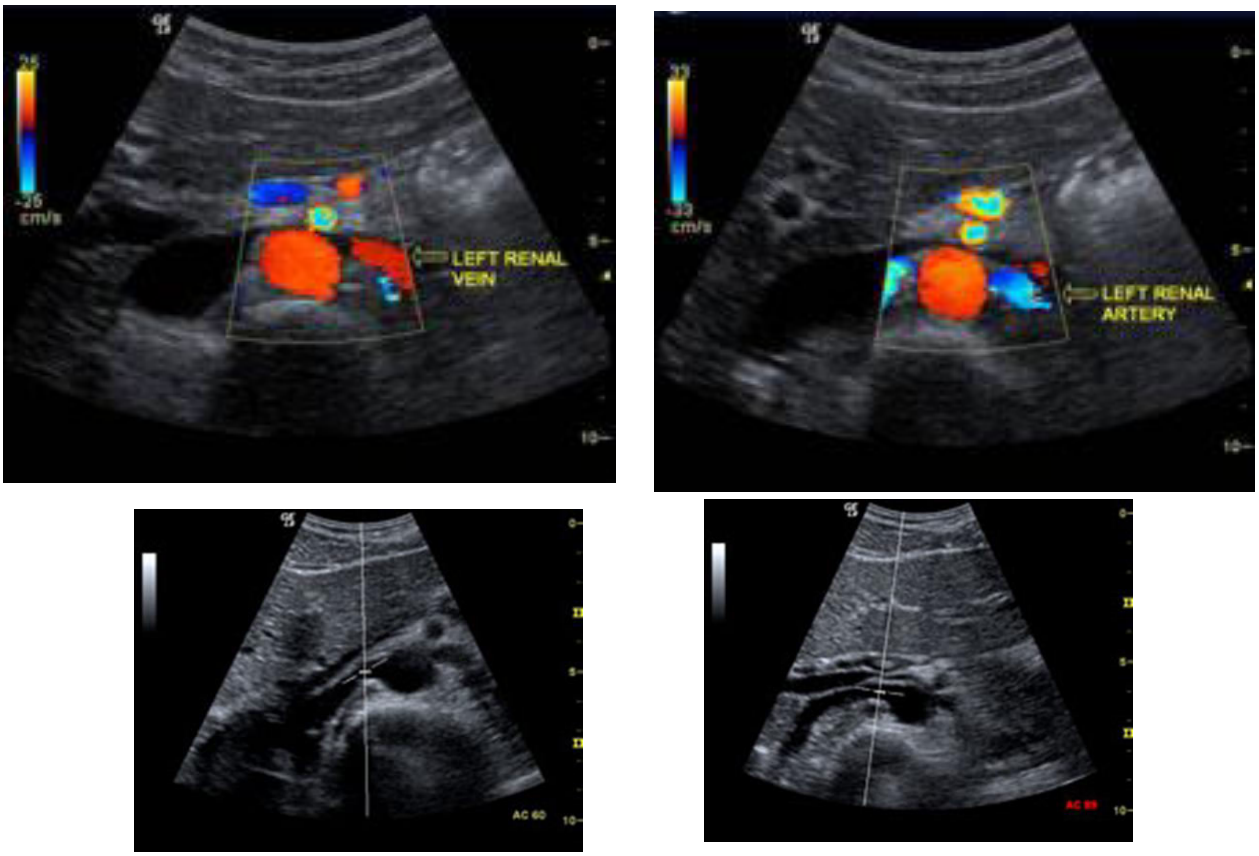

(Fig. 2): Transverse B-mode view of the abdominal aorta and right renal artery from an anterior approach. The ultrasound probe is oriented at midline and the Doppler cursor placed in the proximal right renal artery (A). The angle of incidence of the Doppler beam to the flow is unacceptable at approximately 89 degrees. By moving the probe to the left of midline and angling toward the patient's right, an acceptable Doppler angle of 60 degrees is achieved (B) **(7).**

It is sometimes difficult to distinguish the border between the IVC and renal artery, but color Doppler will help. Setting the color PRF (velocity scale) high enough so that aliasing is minimized improves the boundary definition. When the PRF is low, the flow signal is aliased making it difficult to determine flow direction, detect turbulence or even to differentiate the vessel border between the IVC and renal artery. In slim patients, it is occasionally possible to follow the renal artery and vein into the hilum of the kidney by applying compression with the transducer and slight angulation **(2).**

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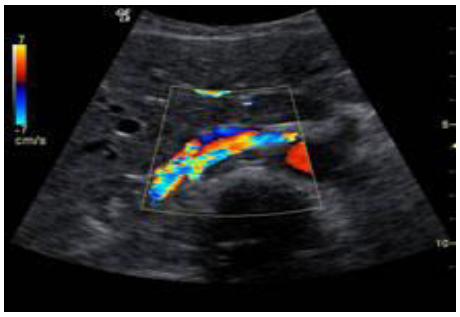

(Fig. 3): transverse color Doppler images of the right renal artery passing underneath the IVC. It is difficult to recognize the right renal artery or distinguish the boundary between IVC and right renal artery when using a low PRF. This is because color Doppler aliasing tends to hide the boundary between the two vessels (A). By increasing the PRF, flow direction becomes more apparent, and the right renal artery is readily identified separate from the IVC (B) **(7)**.

By slight displacement of the probe to the right of the midline and angulation to the left, the first short segment of the left renal artery will be seen posterior and slightly inferior to the renal vein. On grayscale alone, the left renal artery is usually difficult to be seen from an anterior approach, but once color is activated the proximal portion is often well visualized **(3).**

The left renal vein is an excellent landmark for locating the renal artery as it is frequently detected before the artery and appears as a large vessel **(6).**

In anterior approach, the mid to distal renal artery is not often imaged adequately and for this reason it is better for the examiner to turn into flank approach. The flank approach is usually the most successful view for imaging the entire length of both renal arteries as well as the intrarenal arteries. To evaluate the right renal artery, the patient is rolled into a left decubitus position. The patient is asked to relax the abdominal muscles as much as possible. The probe is placed in a sagittal view in the soft part of the abdomen below the rib cage **(6).**

The probe is manipulated slightly until the aorta and IVC are seen in long axis and by slightly varying the probe angle; both renal arteries can be seen arising from the aorta. The right renal artery will course toward the probe and the left will course away**(3).**

This is an excellent view for obtaining a Doppler signal from each renal artery origin as well as the abdominal aorta to calculate RAR. Next, the probe is oriented into a transverse plane and positioned at the renal hilum to image the mid and distal portion of the right renal artery **(6).**

To evaluate the left renal artery, the patient is rolled into a right decubitus position. The probe is positioned in a sagittal plane over the left kidney. It will be necessary to vary the probe angle until both the abdominal aorta and left kidney are visible in the image. Imaging of the renal artery can be performed though either a sagittal or transverse orientation of the kidney **(3).**

Evaluation of the intrarenal arteries is best done through a flank approach with the patient rolled into a decubitus position. The probe is placed along a lateral or slightly posterior approach and by the help of color Doppler, intrarenal branches can be visualized**(6).**

The intrarenal Doppler waveforms must be obtained at angles less than 30 degrees or the early systolic peak may not be visualized. Color Doppler helps to estimate the best angle of approach

for Doppler. Typically, the probe is rotated more posteriorly to improve the Doppler angle for the upper pole intrarenal arteries. For the mid kidney, the probe is centered in a coronal plane. The best Doppler angle for the lower pole intrarenal arteries is usually obtained by rotating the probe slightly anterior to the mid coronal line **(6).**

Renal color duplex examination should include screening of the abdominal aorta from the celiac artery to the iliac bifurcation to evaluate the amount of atherosclerotic plaque, this is done with both gray-scale and color flow Doppler. Gray-scale evaluation is important to assess for irregular plaque and ostial lesions, which may be obscured by color flow blooming. The presence of significant atherosclerotic plaque should increase the suspicion for possible ostial renal artery disease, particularly in elderly or diabetic patients **(2).**

Each renal artery should be examined with color flow imaging from its origin to the hilum of the kidney. Look for areas of stenosis, indicated by color shifts or aliasing. Interrogate these areas with spectral Doppler analysis. If there are no areas of abnormal flow, obtain PSV from the origin, proximal, mid and distal segments of each renal artery. Finally, waveforms are also obtained from the segmental arteries in the upper, mid and lower poles of each kidney. Thus, at least seven waveforms are captured from each side **(2).**

Renal injury in the asphyxiated newborn infant

Introduction:
During asphyxia, ischemia and hypoxia play a role in the impairment of kidney function. Perinatal asphyxia causes renal damage by 2 mechanisms: directly via hypoxia and indirectly via decreased RBF due to diversion of blood away from the kidneys and towards the brain, heart and adrenals **(8).**

Perinatal asphyxia is a main cause of renal hypoperfusion which is a major prerenal cause of ARF in the newborn infants**).** The term vasomotor nephropathy (VMNP) is often used as an imprecise alternative for prerenal ARF. It indicates renal dysfunction, with or without parenchymal damage due to reduced renal perfusion. Continued VMNP can lead to irreversible renal damage **(9).**

Pathophysiology of Ischemic Renal Injury:
Initially, studies on the pathophysiology of ARF were descriptive and involved the development of experimental models. Contemporary studies have been concerned with the pathophysiology of cell injury, particularly related to renal tubule function and metabolism. Work during the past several years has produced new insights into many aspects of cell biology of ischemic injury to the tubulointerstitium. **(10).**
The major processes are conveniently considered in terms of ‘early events’, the prelethal and lethal cell injuries that occur during the period of ischemia and in the first hours of reperfusion, and ‘late events’, the proliferation and fibrosis which repair and restructure the damaged tissue **(10).**

In considering the pathophysiology and cell biology of ischemic renal injury, the relationships between ischemia, hypoxia and anoxia must be kept in mind. ‘Ischemia’ involves cessation of circulation to a tissue so that metabolic substrates are not delivered and metabolic products are not

removed. 'Hypoxia' indicates a state in which oxygenation is reduced below the critical PO_2, the level of oxygen required for complete oxidation of cytochrome C. This value for proximal tubules is 10-17 mmHg **(11).**

Gradients of oxygenation which determine degrees of hypoxia are commonly observed both within tissues, as delineated in recent studies of perfusion and hypoxic injury to the outer medulla, and within individual cells.'Anoxia' indicates complete absence of oxygen, a condition which should be verified by documentation that mitochondrial cytochromes are fully reduced **(12).**

A. Renal Hemodynamics:

The GFR of the healthy newborn is low, which explains the vulnerability of renal (glomerular) function in early extrauterine life. In order to minimize this vulnerability and to assure that the low precarious effective filtration pressure is maintained under most pathophysiological circumstances, a delicate balance of intrarenal vasoconstrictive and vasodilator forces is essential **(13).**

Renal hemodynamic factors play an important role in initiating VMNP (prerenal ARF). Renal vasoconstriction is a well – documented factor in initiating VMNP. The hypothesis was that an insult to renal tubular epithelium resulted in release of vasoactive compounds that increased cortical vascular resistance, thereby decreasing RBF and perpetuating injury to the tubule. Release of vasoconstrictive compounds diminished GFR by constricting afferent and efferent arterioles, which led to a diminished urine output or oliguria. Therefore, emphasis has been placed on identifying vasoactive compounds that are stimulated or induced by hypoxic- ischemic insult **(14).**

Vasoconstriction:

The main humoral candidates for mediating renal vasoconstriction in VMNP are angiotensin II (AII), adenosine, endothelin (ET) and thromboxane A_2 (TXA_2) **(9).**The renin-angiotensin-aldosterone system (RAAS) is very active during perinatal period. In the human newborn and in developing animals, plasma renin activity, renal renin gene and renal AII: receptor expression are higher than in adults.The RAAS functions in a manner consistent with the renal vasoconstriction hypothesis. An injury to the more proximal portion of the nephron results in stimulation of intrarenal release of renin with subsequent generation of AII which is a potent renal vasoconstrictor. However, the role of RAAS as the modulator of renal injury is uncertain **(14).**

Clinical and Laboratory Affection of the Kidneys in Perinatal Asphyxia:

The spectrum of renal damage following hypoxic- ischemic injury extends from mild tubular dysfunction to ATN or irreversible cortical necrosis **(15).**

Hypoxic / ischemic ATN is one of the commonest causes of ARF. Clinical manifestations of all forms of renal necrosis may be similar to those of ARF including oliguria or anuria and varying degrees of hematuria and proteinuria **(16).**

Acute retention of urine may also occur in full–term asphyxiated infants which may require bladder catheterization. ARF is a recognized complication of birth asphyxia, it carries a poor immediate prognosis and may result in permanent renal damage in up to 40% of survivors **(17).**

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ARF is defined as a sudden and severe reduction in GFR. This will result in a rise in serum creatinine, and eventually in further metabolic disturbances. However, the serum creatinine in newborns is very variable and an absolute cut - off value is not helpful in making diagnosis of ARF. It is more useful to suspect renal impairment if serum creatinine rises or fails to show the normal postnatal fall. Normally, serum creatinine levels fall quickly from 0.8 mg/ dL at birth to 0.5 mg/ dL at 5 to 7 days and reach a stable level of 0.3 to 0.4 mg/ dL by nine days. The rate of decrease in serum creatinine in the first few weeks is slower in decreasing gestational age**. Waikar et al (17)** definedARF as serum creatinine > 1.5 mg/ dL (130 umol/L) for at least 2 consecutive days, **Durkan and Alexander (18) defined** neonatal ARF as serum creatinine values > 1.5 mg / dL (133 umol/ L) for at least 24 hr.

Table 1: Causes of renal failure in the newborn(19).

Preneral	Intrinsic	Obstructive
Hypovolemia - Severe congenital heart disease - Severe PDA* with large left – right - shunt - Septicemia - Intraventricular hemorrhage -Dehydration (diarrhoea/ phototherapy) **. Perinatal hypoxaemia / asphyxia** - RDS* -Intrauterine obstruction of umbilical cord - Obstetric hazards - Traumatic delivery - Severe bleeding (placenta ablation / twin – twin blood transfusion) - CNS* damage / intraventricular hemorrhage - Severe cyanotic	**. Congenital abnormalities** **. Maternal Drugs** ACE inhibitors Cyclo-oxygenase inhibitors **. Acute tubular necrosis** **. Acute cortical necrosis** **. Hemoglobinuria** **. Myoglobinuria** **. Renal vascular thrombosis** **. DIC** **. Pyelonephritis** **. Nephrotoxins**	**. Congenital malformations** Ureterocele Posterior urethral values Vesicoureteric reflux Megacystis – megaureter PUJ* obstruction Prune belly syndrome **. Extrinsic compression** Sacrococcygealtumour Hematocolpos **. Intrinsic obstruction** Fungal ball **. Neurogenic bladder** Asphyxia Spina bifida

heart disease **. Septicemia/ multiorgan failure** **. Hypothermia** **. Iatrogenic** Mechanical ventilation Renal artery and/ or vein catheterization Drug therapy ACE* inhibition – tolazoline Prostaglandin inhibitors – nephrotoxic agents		

PDA: Patent ductus arteriosus ACE: Angiotensin converting enzyme
DIC: Disseminated intravascular coagulation RDS: Respiratory distress syndrome
CNS: Central nervous systemPUJ: Pelvi – ureteric junction

Clinical Assessment and Investigations:

The management of the asphyxiated infant should include anticipation of the possibility of renal failure. Since asphyxiated infants are at risk for ATN and SIADH, and to avoid water overload, the infant should be given only the volume of fluid necessary for adequate hydration, and a reasonable starting point is restricting to 20% less than the normal maintenance rate for the first 48 hours. This should be associated with regular assessment of hydration, serum electrolytes, urine output and specific gravity or osmolality **(15).**

Special attention should be given to urine output. Careful monitoring of urine output should commence immediately on admission. Failure to monitor urine output accurately may delay recognition and increase the risk of prerenal impairment progressing to intrinsic renal failure. Urine output is measured with urine collection bag. Bladder catheterization increases the risk of ascending urinary tract infection, with the associated increase in morbidity and mortality. Bladder emptying should be confirmed by cautious supra- public pressure, ultrasonographic examination of the bladder, and, if necessary, by intermittent sterile bladder catheterization **(9).**

About 30% of healthy newborns void during delivery or soon thereafter, 92% within 24 hours and 99% within 48 hours **(16).**Therefore, if the urine flow rate falls abruptly in a previously stable infant, or drops below 1 mL/ kg/ hr in the first few days after birth, immediate investigation is mandatory.

Oliguria (< 1 mL/kg/ hr) or anuria is often the first sign of incipient ARF. Additionally, a significant association between clinical signs of HIE and long-term neurologic deficits with

persistent oliguria (present for at least 36 hours) was reported by **Doi et al. (20).**

Moreover, the presence of persistent oliguria was associated with the occurrence of HIE, abnormal findings of cranial ultrasound and poor short-term outcome in comparison with outcome of infants with normal urine output (particularly in the preterm infants).
On the other hand, renal failure with normal urine output- so- called nonoliguric ARF has been found in about one- third of term newborns with ARF and in 60% of those with ARF in the course of severe asphyxia **(21).** Therefore, unless plasma creatinine (Pcr) levels are monitored, nonoliguric ARF can be missed.

The plasma creatinine level is a much better measure of a renal glomerular disturbance than the plasma urea concentration, the latter being affected by extrarenal factors, particularly oral protein intake and total body protein metabolism **(16).**

However, in the first days of life Pcr is high reflecting maternal Pcr concentrations. The normal high Pcr levels may even rise after birth, with a slow decrease during the following 2-3 weeks. The more premature the infant, the higher the Pcr. This is due to temporary tubular reabsorption of creatinine (back- diffusion) through leaky immature tubules**.** Although any rise in Pcr levels should suggest ARF, these other factors should be taken into account. The rapidity of rise in Pcr is also of importance: the faster the increase the higher the possibility of VMNP / ARF **(9).**

Uric acid shows the same pattern as creatinine**.** Perinatal and postnatal asphyxia increase plasma uric acid levels, which poses the danger of uric acid nephropathy and ARF**. Hemachandar and Boopathy (22)** studied the possible role of uric acid in hyperechogenicity in neonatal hypoxia. They found transient insufficiency of renal function among asphyxiated cases developing hyperuricemia and hyperuricosuria. Ultrasonographic examinations showed hyperechogenicity of the renal pyramids which appeared within 24 hours and disappeared in a short time. They concluded that the neonatal kidney is very sensitive to hypoxia and that hypoxic renal failure is accompanied by hyperechogenicity of the kidneys and that uric acid is a possible cause of renal hyperechogenicity.

Several laboratory tests may be helpful in differentiating prerenal from established (intrinsic) renal failure in oliguric neonate.

Table 2: Urine:plasma diagnostic indices in neonates with acute oliguria(23)

	Acute Prerenal Failure	**Acute Intrinsic Renal Failure**
Uosm (mOsm/ kg/ H_2O)	>400	<400
UNa (mmol/L)	<40	>40
U: Purea	>20	<10
U: Posm	<2.0	<1.0
FeNa*	<2	>3
RFI*	<1.5	>6

* FeNa: Fractional excretion of sodium (%) = (U/P) sodium x (P/U creatinine) x 100.

* RFI: Renal failure index = U_{Na} x P/U creatinine.

It has been suggested that the best indicator to distinguish prerenal from established renal failure in the oliguric neonate is the FeNa. FeNa has also been commonly used to estimate renal tubular function in neonates. FeNa is based on both tubular sodium reabsorption and glomerular filtration, both of which are decreased in renalfailure.

If tubular function is intact and sodium reabsorption continues, the infant is in oliguric prerenal failure and FeNa will be less than 3%. Once tubular necrosis has occurred the FeNa is usually above 10%. Prerenal oliguria demands urgent attention to renal perfusion, whereas a high FeNa (usually > 3%) suggest established renal failure and the equally urgent need for restriction of fluid intake**(15).**

Unfortunately, the FeNa, and other indices such as RFI, have poor sensitivity and specificity. Caution should be taken when applying these indices to premature infants who may under normal conditions have FeNa> 3%. Falsely high FeNa values can be obtained if a large sodium load is administered or if potent diuretics or volume expanders are used prior to thedetermination of these indices **(18).**

Several studies reported the occurrence of detectable tubular dysfunction even with no glomerular disturbance in sick newborns including asphyxiated infants.These infants undoubtedly have a degree of tubular dysfunction that is clinically unsuspected, but which can be shown with sensitive tests **(17).**
The proximal tubular function, being themost susceptible to hypoxic – ischemic insult, can be assessed through the estimation of electrolytes in serum and urine as previously discussed or through the measurement of various tubular markers thatproved useful in the diagnosis and surveillance of a number of renal disorders**.** Low molecular weight proteins, such as β_2-microglobulin and retinol binding protein (RBP), are freely filtered at the glomerulus and almost completely reabsorbed by the proximal tubular cells in health, so that when tubular dysfunction is present, increased quantities appear in urine **(17).**

Prerenal ARF, treated promptly, in most cases reverses quickly. If ATN ensues, one can expect a prolonged period of anuria or oliguria (10 to 21 days), eventually with complete clinical recovery. The renal function returns to normal but the length of time before recovery is quite variable. Recovery of renal function may be accompanied by a diuretic phase with voluminous urine output at a time when the tubules are beginning to recover but have not recovered sufficiently to reabsorb solute and water appropriately, posing the risk of dehydration and hypoelectrolytemia. Except in very severe insults when the vasculature is involved in microthrombi formation leading to the development of cortical necrosis, the prognosis of ATN is good and depends on the underlying events that precipitated the ischemic / hypoxic insult **(1).**

The prognosis for nonoliguric ARF is regarded to be better as compared with oligoanuric ARF. One common explanation of the better prognosis of nonoliguric ARF is that the insult is less severe. This is the rationale given for the association of poor neurologic outcome with persistent oliguria; any injury to the renal cortex is likely to have injured the brain as well. Lesser insults may result in tubular damage but not oliguria or significant neurologic residual (**20).**

Despite tremendous advancements in medicine in recent years, treatment of ARF has remained essentially supportive. Unfortunately, overcoming the acute crisis does not promise a full recovery of kidney function. On the contrary, several studies have already demonstrated that up to 40-50% of neonates recovering from ARF are being left with residual renal damage manifested by structural, glomerular or tubular abnormalities, or hypertension **(24).**

Renal hemodynamic alterations by duplex ultrasound:

Nowadays Doppler ultrasound is rapidly gaining ground as a screening tool in critically ill patients. The performance of cardiac, lung and abdominal ultrasound in patients after cardiac arrest, major operations and during shock has become standard policy. However renal ultrasound, which could be easily incorporated in this screening, is not commonly performed. Renal vasoconstriction is an early manifestation of AKI **(25).**

Renal Doppler ultrasound can measure the renal resistive index (RRI), a sonographic index that reflects alterations in blood flow profile of the intrarenal arcuate or interlobar arteries. It reflects the relation between the decline in speed loss of flow ("flow velocity") between the peak of systole and the end of diastole in (renal) blood vessels: RRI = (peak systolic velocity—end diastolic velocity)/(peak systolic velocity). Previous studies have shown that elevated RRI is related to hemodynamic parameters such as systolic- and diastolic blood pressure, pulse pressure and pulse wave velocity, which is a measure for arterial stiffness **(26).**

Renal ultrasound imaging and Doppler technique could offer the possibility to fill the gap at least in part. B-mode allows to point out variations in kidney parenchyma brightness and in longitudinal length that are easy reproducible, rapid and non-invasive that may identify renal preclinical dysfunction or vascular damages, to prevent the onset of clinical renal dysfunction.

Both (color) Doppler sonography and amplitude-coded color Doppler investigations add functional imaging to the anatomic-morphologic description of US and are mandatory in neonatal oligo anuria and renal failure. Reduced renal systolic flow and diastolic flow velocities as well as increased resistive index in asphyxiated neonates has been reported to sensitive for subsequent development of RF. These and further findings can influence therapy significantly **(27)**.

Only a few studies describing the application of Doppler sonography in the evaluation of intrarenal hemodynamic abnormalities have been published so far, and most of these were performed in adults (**26).**

ConflictofInterest: Noconflictofinterest.

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