

**American College of Radiology
ACR Appropriateness Criteria®**

Clinical Condition: **Renal Failure**

Variant 1: **Acute renal failure, unspecified.**

Radiologic Procedure	Rating	Comments	<u>RRL*</u>
US kidneys and bladder retroperitoneal	9	Preferably with Doppler methods.	None
Tc-99m MAG3 scan kidney	5	Global and differential function. Assess recoverability; distinguish from chronic.	Med
MRA abdomen	4	Newer techniques with gadolinium are very effective for renal artery evaluation. Rule in/out renal vein/caval thrombosis. See statement regarding contrast in text under "Anticipated Exceptions."	None
Arteriography kidney	3	Potentially helpful in trauma, evaluation for renal artery occlusion. Consider aortography with CO ² to avoid nephrotoxicity of iodinated contrast.	Med
CT abdomen without contrast	3	Potentially helpful in trauma. Noncontrast helical CT more sensitive than KUB for calculi. Evaluation of ureteral obstruction due to retroperitoneal diseases, masses, tumors (hydronephrosis on sonography, but cause undetectable).	Med
X-ray abdomen (KUB)	2	Assess for calculi; however, insensitive for 30% of calculi.	Low
X-ray voiding cystourethrography	2	VCUG may be indicated if vesicoureteral reflux is suspected as a contributing factor in ARF.	Low
MRI abdomen with or without contrast	2	Potential role in search of sonographically unclear causes of ureteral obstruction. With contrast preferred if not contraindicated.	None
<u>Rating Scale:</u> 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Clinical Condition:**Renal Failure****Variant 2:****Chronic renal failure.**

Radiologic Procedure	Rating	Comments	<u>RRL</u>*
US kidneys and bladder retroperitoneal	9	Preferably with Doppler methods.	None
MRA abdomen	6	Noninvasive evaluation of renal arteries as cause of renal failure. Preferred with contrast unless contraindicated. See statement regarding contrast in text under "Anticipated Exceptions."	None
CTA abdomen	3	Effective in detecting RAS, if nephrotoxicity is not a problem.	Med
X-ray abdomen (KUB)	3	Provides information about calcification, majority of calculi, occasionally renal size.	Low
X-ray voiding cystourethrography	3	If reflux is suspected. Particularly appropriate in children.	Low
Tc-99m MAG3 scan kidney	3	Global and differential renal function; prognosis for recovery.	Med
CT abdomen without contrast	3	Potentially helpful in trauma. Helical noncontrast CT for calculi. Search for retroperitoneal mass/adenopathy as the cause of obstruction. Surveillance of native kidneys for RCCA.	Med
Arteriography kidney	2	Problem of contrast nephrotoxicity. CO ² aortography an option. Newer MRA techniques preferred.	Med
MRI abdomen with or without contrast	2	Surveillance of native kidneys for RCCA. Preferred with contrast unless contraindicated.	None
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

RENAL FAILURE

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Summary of Literature Review

Renal failure is defined as the inability of the kidney to maintain homeostasis leading to azotemia or the accumulation of nitrogenous wastes; however, exact biochemical or clinical criteria for this diagnosis are not defined clearly. “Renal failure” is distinguished from “renal insufficiency,” where renal function is abnormal but capable of sustaining essential bodily functions [1]. Renal failure is defined as anuric when urine volume is less than 50 ml for 24 hours; oliguric when the volume is less than 500 ml for 24 hours; and nonoliguric when the volume is from 500-6,000 ml for 24 hours. Urine output above 6,000 ml is designated polyuric [2].

Causes of renal failure are conventionally separated into three categories: prerenal, intrarenal, and postrenal. Hypoperfusion is the cause of prerenal failure (fluid loss, fluid sequestration, low cardiac output, renal artery stenosis [RAS]). Causes of intrarenal failure include acute tubular necrosis (ATN) and interstitial, glomerular, or small-vessel disease. Obstruction is the usual postrenal cause of failure (also, distal renal tubular obstruction). Distinction between acute renal failure (ARF) and chronic renal failure (CRF) can often be made clinically [3]. However, many patients are first seen with markedly elevated serum creatinine of unknown duration, so that classification into ARF or CRF is not possible.

There are significant limitations in using serum creatinine as an accurate measure of renal function, including decreased muscle mass and poor nutritional status [4]. Creatinine clearance measures the ability of the glomerulus to filter creatinine from the plasma and approximates the glomerular filtration rate (GFR); there is a reasonable correlation between the 2-hour and 24-hour creatinine clearance ($r=0.85$), but the error in calculation may vary from 10%-27%. Creatinine clearance of less than 60 ml/min may be termed renal insufficiency; less than 30 ml/min is renal failure. End-stage renal disease (ESRD) implies CRF of a degree (ie, GFR <10-12 ml/min) such that life cannot be sustained long-term without dialysis. In ARF, the creatinine clearance is usually less than 25 ml/min. Unfortunately, creatinine clearance is often not helpful when the creatinine value is changing.

Acute Renal Failure

ARF can be broadly defined as a sudden decrease in renal function resulting in azotemia. It can develop in the setting of pre-existing renal insufficiency or can develop in a patient with previously normal kidneys [5]. In a patient with previously undiagnosed renal failure, initial evaluation of renal size by gray-scale ultrasonography (US) is most helpful. If the kidneys are small and echogenic, the process of long-standing evaluation by US helps to identify a correctable cause of renal failure such as obstruction. If hydronephrosis is present, retrograde or antegrade relief of the obstruction is usually undertaken. If no hydronephrosis is evident and the patient does not have hypertension or other history to suggest RAS, further workup of small, echogenic kidneys is not warranted. Conversely, if the kidneys are of normal size with or without increased echogenicity, this may represent reversible renal failure, most often ARF, and a more extensive evaluation is initiated. It should be noted that, as with all modalities and procedures, the yield of gray-scale sonography in the detection of hydronephrosis is low among patients without a high pretest probability for urinary obstruction (eg, flank pain, urolithiasis, pelvic masses, etc), and, therefore is of little value in patient management [6]. Scintigraphy with a tubular secretion agent (eg, MAG-3) can help assess the level of renal function as well as the potential reversibility of the process causing the renal failure. Therefore, in addition to the history, physical examination, and laboratory analysis of serum and urine, US and radionuclide scintigraphy are imaging tools that are used early in the evaluation of the patient with previously undiagnosed renal failure. If RAS or occlusion is suspected, magnetic resonance angiography (MRA) techniques may be used to avoid nephrotoxic iodinated contrast media.

Over 75% of patients with ARF will have either prerenal azotemia (PRA) or ATN (parenchymal, intrarenal process) as the cause [3]. Prerenal causes of ARF relate to hypoperfusion or hypovolemia. Clinical suspicion of ARF usually leads to a fluid challenge with central monitoring

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and correction of the hypovolemic state, which in turn corrects the renal failure. A common exception to this approach is the patient with heart failure or liver failure. Acute renal artery occlusion in a solitary kidney or solitary functioning kidney is an uncommon cause of lack of response to the therapeutic trial of intravascular fluid; imaging techniques are used to define the cause of hypoperfusion.

A high ratio of blood urea nitrogen (BUN) to creatinine (Cr) has long been considered a marker of PRA. In addition, a characteristic laboratory finding in PRA is avid sodium retention, with urine sodium concentration of less than 20 mEq/L [7]. Meta-analysis of various laboratory studies in an attempt to differentiate PRA from ATN reveals that most determinations (urine/plasma creatinine index, urine/plasma urea, or urinary sodium) are often nonspecific or unreliable [5]. Still, most experienced clinicians find that when urine output is less than 500 ml for 24 hours, determination of urinary fractional excretion of sodium is helpful.

Duplex Doppler sonography has been suggested to distinguish acute prerenal failure from ATN (intrarenal failure). Compared with traditional gray-scale US, which shows normal kidneys in most patients with ATN, duplex Doppler sonography shows an elevated resistive index (RI) in 96% of patients with ATN; false negatives include nephrotoxic drug-induced ATN [8]. ATN has a higher RI than prerenal ARF, but there is some overlap in that 20% of patients with prerenal ARF had resistive indices over 0.75. Hepatorenal syndrome is a distinct form of prerenal failure that is associated with an elevated RI [8]. Tubulointerstitial causes of intrarenal ARF usually have an elevated RI. Acute glomerular-based processes will often have a normal RI, whereas chronic glomerular processes typically show an elevated RI [9]. Consequently, Doppler sonography cannot replace renal biopsy. Although there is a weak linear relationship between the RI and serum Cr, the RI returns to normal before serum Cr in ARF; RI therefore may be useful in predicting the course of ARF [10].

Trauma presents a unique constellation of prerenal, intrarenal, and/or postrenal causes of ARF. In major trauma centers, body computed tomography (CT) is used increasingly for initial abdominal trauma assessment of causes of renal failure such as renal artery occlusion, severe kidney trauma, and clot obstruction occurring bilaterally or in a solitary kidney. Nephrotoxic drugs and ATN following prolonged shock with precipitation of hemoglobin and/or myoglobin in the tubules are other causes of ARF that may cause abnormal CT findings [2].

Obstruction is an uncommon cause of ARF but may occur in the oncology patient, the trauma patient, or the patient with a solitary kidney [8,9]. Gray-scale US is the most effective way to exclude subacute or chronic obstruction. Regular gray-scale US is not accurate in the minimally dilated obstructive situation, such as with retroperitoneal metastatic tumor or idiopathic retroperitoneal fibrosis, where ureter encasement interferes with peristalsis; in one

series, 4%-5% of patients with obstruction showed minimal or no upper tract dilation [11]. Duplex Doppler sonography is less effective in acute obstruction since obstruction for longer than 6 hours is necessary to show a consistently elevated RI; false negatives (ie, normal RI) occur in patients who are examined earlier than 6 hours after the onset of obstruction [12]. Furthermore, RI measurements are often normal in patients with acute intermittent obstruction. The patient with a renal transplant can present with ARF. Because Platt et al [13] found an elevated RI in 85% of transplanted kidneys with obstruction, a normal RI should argue strongly against obstruction, unless a ureteral leak is also present. In addition to obstruction, an elevated RI can also be found in rejection and ATN; therefore, RI measurements are not reliable in the differential diagnosis of these entities. If sonography cannot determine the cause of the obstruction, MRI or nonenhanced CT can be obtained (retroperitoneal mass, lymphadenopathy, fibrosis, calculi, etc).

After US excludes obstruction, it is suggested that renal scintigraphy with technetium-labeled MAG-3 be performed. Progressive parenchymal accumulation without significant excretion is suggestive of ATN. Absent uptake suggests more serious conditions such as acute cortical necrosis and acute glomerulonephritis. In ARF, GFR is more affected than renal blood flow, hence Tc 99m DTPA accumulation is decreased, and this agent is less able to distinguish acute from chronic renal disease. However, quantitative studies with the tubular agents such as Tc 99m MAG-3 can be used [14,15]. These methods assess effective renal plasma flow (ERPF) and the degree of renal function, and they also have prognostic significance. Patients with ERPF greater than 125 ml/min and good uptake usually recover completely or markedly improve. ATN, hepatorenal syndrome, and acute interstitial nephritis belong in the category of good prognosis. Patients with low uptake have a poor prognosis and eventually require dialysis or transplantation.

Clinical evaluation and volume replacement resolve the majority of prerenal causes of renal failure. US evaluates for obstruction and renal size, and it can provide a measure of renal perfusion. Some suggest that duplex Doppler sonography can supplant radionuclide scintigraphy, MRA, or contrast angiography in evaluating the renal arteries; however, these results have not been reproduced in many centers. Newer MRA techniques offer improved images of the main and segmental renal arteries [16]. Magnetic resonance imaging (MRI) can also provide direct assessment of renal blood flow [17]. Scintigraphy is useful for renal perfusion and for determining ERPF, which helps assess recoverability of function in ARF. CT is used to evaluate the trauma patient and supplement technically unsatisfactory or equivocal sonography. Excretory urography has no role in investigating ARF.

Chronic Renal Failure

CRF often presents insidiously and is characterized by a steady decrease in GFR. Causes of CRF that lead to ESRD and result in transplantation are (in order of

decreasing frequency): chronic glomerulonephritis, diabetic nephropathy, hypertensive nephropathy, polycystic renal disease, chronic pyelonephritis, and renal calculi [18].

The most common causes of CRF in children are chronic glomerulonephritis and pyelonephritis [19]. US best differentiates between obstruction and intrinsic parenchymal disease. In children with small-scarred kidneys, voiding cystourethrography (VCUG) is performed. For adults with ESRD and urinary tract infection (UTI) or calculi, evaluation with VCUG, urodynamics, and retrograde pyelography is also advised [18,20].

[Appendix 1](#) lists the five stages of chronic renal failure based on GFR calculations, as defined by the National Kidney Foundation.

Patients with ESRD on dialysis develop cysts, hemorrhage, and neoplasia. Evaluation for cystic change in these patients is done optimally with CT, which showed 60% of cysts, whereas sonography showed only 18% [21]. Although solid renal masses in these patients were shown equally well by sonography and CT, the ability of CT to detect acquired cystic disease and the need to follow for possible neoplasm warrants use of CT as screening after 3 years of dialysis [21]. Early enhanced CT is recommended [22]. In acquired cystic disease, follow-up imaging with CT seems advisable only in selected populations [23]. Alternatively, MRI and US can be used. Patients with CRF on temporary dialysis should not be given intravascular iodinated contrast media. The administration of gadolinium-based MR intravascular agents in patients with CRF is also restricted (see “Anticipated Exceptions”).

Analgesic nephropathy has been in decline worldwide over the last several years due to its known relationship to excessive use of nonsteroidal analgesics and the banning of acetaminophen, and now accounts for only about 1% of patients in the USA who are on chronic dialysis and, often, develop papillary necrosis [24]. Calcification along the papillary line and a “wavy” renal contour are the most common radiographic findings. The use of plain tomography, sonography and unenhanced CT in the search of renal calcifications and their pattern in regards to analgesic nephropathy was studied. Unenhanced CT was found to be the superior modality for the detection of the calcifications (92% sensitivity, 100% specificity) [24,25]. Noncontrast helical CT is also more sensitive and specific than plain radiography for ureteral calculi [26].

Hypertensive nephropathy is now one of the most common causes of ESRD and in one study accounted for 25% of all patients [27]. Atherosclerotic RAS presenting as CRF accounted for 14% of patients older than 50 in another study [28]. Reports on the ability of duplex Doppler sonography to detect RAS vary widely; some reports are as high as 90%, whereas others show poor results [8,28-30]. Over one-third of patients evaluated with earlier Doppler methodology had an unsatisfactory

exam [31]. With use of a posterior or posterolateral translumbar approach and analysis of intrarenal vessel waveforms, duplex Doppler sonography has been reported to detect significant (over 70%) RAS as a cause of renal failure, with a sensitivity of 95% and specificity of 97% [32-34]. Examinations were almost always technically feasible and accomplished within half an hour [33]. The study by Kliever et al [35] found it effective in evaluating RAS, but only when the RAS was 80% or greater. Usually, high-grade stenoses are associated with renal failure. A subsequent study was not able to reproduce results adequately to support the use of duplex Doppler sonography as a screening test for RAS [36]. Duplex Doppler sonography for diagnosis of RAS is very operator-dependent [37].

Multiple factors influence the management of hypertensive nephropathy secondary to RAS. Although not critical in most reviews regarding treatment options and outcomes, renal size is nevertheless important. The typical, medium to long standing RAS kidney is small and/or smaller than the contralateral kidney without RAS. Normal renal size/length has a long range that depends on age, sex, race, body habitus, and, to some extent hydration. Likewise, the thickness of the normal renal cortex is not clearly defined, and many reports in the literature prefer to depend on biochemical and clinical facts in judging patient eligibility for and outcomes of treatment (eg, the duration of symptoms, presence of nephrosclerosis, GFR or serum creatinine clearance, etc). Best results of RAS treatment are encountered among patients, whose kidneys measure >8 or 9 cm in length, whereas a thickness of 8-10 mm (as well as estimates of renal polar cortical areas) has been given for normal renal cortex. Overall renal size and cortical thickness clearly regress above the age of 70 years. Furthermore, reports in the literature indicate that for kidneys >7 cm in length, nephrectomy (nowadays laparoscopic) or renal ablation may be preferable to an attempted repair of the RAS. For obvious reasons, if one's institution or hospital abides by these numbers, it will suffice to document overall renal size, but a full Doppler examination may be useless [37-41].

Renal scintigraphy with technetium 99m DTPA and an angiotensin-converting enzyme inhibitor (ACEI) has high sensitivity and specificity in detecting RAS in patients with normal or near-normal renal function. Its utility is also reported preserved in patients with renal insufficiency [42,43]. However, it becomes less accurate in patients with renal failure because DTPA is a pure glomerular agent and there is a variable response to ACEI in patients with low baseline renal function (eg, GFR less than 15 ml/min) [44]. On the other hand, scintigraphy with technetium 99m MAG-3, because it is secreted by the tubules as well as filtered by glomeruli, is similar to iodine 131 Hippuran; it is more effective in patients with renal failure [14,15,44]. However, scintigraphy with ACEI does not indicate the presence of RAS, but only activation of the renin-angiotensin system; conversely, a negative test does not exclude RAS but only absence of

activation [45]. Global and differential renal function can be used to estimate prognosis for recovery. Whereas visualization of the kidney is nonspecific, nonvisualization of the kidneys indicates a poor prognosis.

MRA is able to demonstrate, with high sensitivity and specificity, atherosclerotic narrowing of the orifice and proximal renal artery [45-47]. Aortic or proximal renal artery disease is the usual culprit when atherosclerosis causes renal failure, making MRA a helpful imaging modality [48]. Newer ultrafast MRA techniques using intravenous gadolinium agents during breath-held imaging provide excellent images of the entire renal artery and often the segmental branches [16,49,50]. Gadolinium agents have less nephrotoxicity than conventional iodinated contrast media and, therefore, are available when contrast-enhanced imaging is necessary [51,52]. Angiography with iodinated contrast material and digital subtraction (DSA) technique remains the gold standard, but its use must be carefully considered because of the risk of contrast nephrotoxicity. Some institutions use carbon dioxide as the “contrast” agent and thereby can avoid the toxicity associated with iodinated contrast media.

Following recent advances in MDCT technology, CTA has emerged as an effective alternative to MRA and duplex sonography in the evaluation of RAS, if intravascular administration of contrast media is not contraindicated [53,54]. Both MRA and CTA, although not as sensitive as DSA, were shown to be better than duplex sonography and radionuclide captopril renography. Currently, CTA has improved spatial resolution and shorter examination times compared to MRA. It can also determine the extent of calcified atheromatous plaques not seen on MRA. Disadvantages of CTA include the radiation exposure and risk for renal damage in patients with compromised renal function [55-58].

Urinary obstruction as a cause of CRF is best evaluated by US. If azotemia is secondary to obstructive uropathy, hydronephrosis will almost always be demonstrable. US has sensitivity approaching 100% in moderate to severe hydronephrosis. There may be a false positive rate of up to 26%, caused by such entities as vesicoureteral reflux, full bladder, renal sinus cysts, and normal vessels in the renal sinus; however, vascular structures causing confusion can be resolved with duplex Doppler sonography or color duplex Doppler sonography [59]. When kidneys fail secondary to chronic obstruction, resistive indices may return to normal [11].

Newer and future techniques of determining renal function in patients with renal failure include determination of clearance of small doses (10 ml) of low osmolar contrast media (LOCM) (iohexol), dynamic MR imaging with gadolinium DTPA, and MR imaging with ultrasmall particles of iron oxide (USPIO) [49,60-65].

CRF is often due to intrinsic renal disease such as diabetes and/or hypertension. Obstruction is the most

important cause to be excluded initially, and this is done best by US. If RAS is a possible consideration, various modalities are available. Although angiography is the gold standard, it usually requires potentially nephrotoxic contrast medium. Radionuclide scintigraphy is helpful in measuring ERPF and defining renal artery compromise, though ACEI-modified renography becomes less effective in patients with renal failure. Duplex Doppler sonography, even using newer techniques, has not proved to be a reliable method to screen for RAS, but does seem to be effective in identifying high-grade stenoses; newer CTA and MRA techniques with gadolinium now rival arteriography for evaluating the renal artery; and because ischemic nephropathy is a significant contributor to renal failure, MRA is assuming a more prominent role in evaluation.

The Nephrology literature contains several reports stressing the importance of preservation of residual renal function (RRF) in patients on peritoneal dialysis (PD) or hemodialysis (HD), who may require intravascular administration of contrast media for diagnostic purposes. Apparently, preservation of RRF even after the initiation of dialysis, has been shown to result in better survival, better electrolyte and fluid balance, nutritional status, and quality of life, and has shown a decrease in morbidity and the need for fewer dialysis sessions (fewer or shorter dialysis sessions result in cost savings). The recommendation is to continue to protect the RRF in patients on PD or HD, by balancing the risks versus benefits derived from the use of intravascular contrast media [66-68]. It should be emphasized that if a patient is on temporary (on demand) dialysis, the use of such contrast media is usually withheld. The standard of practice is to administer intravascular contrast media to patients on permanent dialysis, only when indicated. This is supported by evidence that no accelerated loss of RRF was observed in PD patients undergoing diagnostic studies after administration of intravascular contrast media [69,70].

Summary

- US is the first imaging study for evaluating the patient with previously undiagnosed renal failure. It helps the clinician separate chronic ESRD from potentially reversible ARF or CRF by defining renal size, echogenicity, presence or absence of hydronephrosis, and cystic disease. Duplex Doppler sonography can define renal flow; however, the specific utility of duplex Doppler sonography in evaluating the patient with renal failure needs further investigation.
- Radionuclide scintigraphy provides an assessment of global and differential renal function and potential reversibility of renal failure.
- If the US is equivocal for obstruction or cystic disease, add CT.
- CT is of value in the trauma patient with ARF, to rule out stone disease and survey the retroperitoneum for masses in oliguric or anuric renal failure, and for

periodic evaluation of the native kidneys in patients with end stage renal disease, who are at risk of developing renal cell carcinoma. Nonenhanced CT may also be useful in the detection of stone disease in renal transplant patients, if sonography is not diagnostic. Although DSA continues to be the gold standard in the detection of RAS, MDCT CTA in patients who can receive intravascular iodinated contrast media can be an alternative, noninvasive, effective diagnostic tool.

- If the blood pressure is elevated or in the clinical setting of prominent peripheral atherosclerotic vascular disease, add MRA when duplex Doppler sonography or ACEI scintigraphy is positive or nondiagnostic in the patient with renal failure who is not a candidate for contrast angiography. MRI is also useful in the screening of native kidneys with cystic changes of end stage renal disease for the detection of suspected renal cell carcinomas.

Anticipated Exceptions

Nephrogenic systemic fibrosis (NSF) is a disorder with a scleroderma-like presentation and a spectrum of manifestations that can range from limited clinical sequelae to fatality. It appears to be related to both underlying severe renal dysfunction and the administration of gadolinium-based contrast agents. It has occurred primarily in patients on dialysis, rarely in patients with very limited glomerular filtration rate (GFR) (ie, $<30 \text{ mL/min/1.73m}^2$), and almost never in other patients. There is growing literature regarding NSF. Although some controversy and lack of clarity remain, there is a consensus that it is advisable to avoid all gadolinium-based contrast agents in dialysis-dependent patients unless the possible benefits clearly outweigh the risk, and to limit the type and amount in patients with estimated GFR rates $<30 \text{ mL/min/1.73m}^2$. For more information, please see the [ACR Manual on Contrast Media](#) [71].

Relative Radiation Level Information

Potential adverse health effects associated with radiation exposure are an important factor to consider when selecting the appropriate imaging procedure. Because there is a wide range of radiation exposures associated with different diagnostic procedures, a relative radiation level (RRL) indication has been included for each imaging examination. The RRLs are based on effective dose, which is a radiation dose quantity that is used to estimate population total radiation risk associated with an imaging procedure. Additional information regarding radiation dose assessment for imaging examinations can be found in the ACR Appropriateness Criteria® [Radiation Dose Assessment Introduction](#) document.

Relative Radiation Level Designations	
Relative Radiation Level	Effective Dose Estimate Range
None	0
Minimal	< 0.1 mSv
Low	0.1-1 mSv
Medium	1-10 mSv
High	10-100 mSv

Supporting Document(s)

- [ACR Appropriateness Criteria® Overview](#)
- Evidence table under review

References

1. Davidson AJ, Hartman DS. Radiology of the kidney and urinary tract. Philadelphia, Pa: WB Saunders Co. 1994:777-790.
2. Stene JK. Renal failure in the trauma patient. *Crit Care Clin* 1990; 6(1):111-119.
3. Rose BD. Acute renal failure-prerenal disease versus ATN. In: Rose BD, ed. *Pathophysiology of renal disease*. 2nd ed. New York, NY: McGraw-Hill; 1987:63-117.
4. Becker JA. Evaluation of renal function. *Radiology* 1991; 179(2):337-338.
5. Kellen M, Aronson S, Roizen MF, Barnard J, Thisted RA. Predictive and diagnostic tests of renal failure: a review. *Anesth Analg* 1994; 78(1):134-142.
6. Gottlieb RH, Weinberg EP, Rubens DJ, Monk RD, Grossman EB. Renal sonography: can it be used more selectively in the setting of an elevated serum creatinine level? *Am J Kidney Dis* 1997; 29(3):362-367.
7. Adeco MJ, Collins B, Zager RA. The differential diagnosis of acute renal failure. *Contemp Issues Nephrol* 1992; 25:73-117.
8. Platt JF, Rubin JM, Ellis JH. Acute renal failure: possible role of duplex Doppler US in distinction between acute prerenal failure and acute tubular necrosis. *Radiology* 1991; 179(2):419-423.
9. Platt JF. Duplex Doppler evaluation of native kidney dysfunction: obstructive and nonobstructive disease. *AJR* 1992; 158(5):1035-1042.
10. Yoon DY, Kim SH, Kim HD, et al. Doppler sonography in experimentally induced acute renal failure in rabbits. Resistive index versus serum creatinine levels. *Invest Radiol* 1995; 30(3):168-172.
11. Spital A, Valvo JR, Segal AJ. Nondilated obstructive uropathy. *Urology* 1988; 31(6):478-482.
12. Platt JF, Rubin JM, Ellis JH. Acute renal obstruction: evaluation with intrarenal duplex Doppler and conventional US. *Radiology* 1993; 186(3):685-688.
13. Platt JF, Ellis JH, Rubin JM. Renal transplant pyelocaliectasis: role of duplex Doppler US in evaluation. *Radiology* 1991; 179(2):425-428.
14. Fresco GF, DiGiorgio F, Curti GL. Simultaneous estimation of glomerular filtration rate and renal plasma flow. *J Nucl Med* 1995; 36(9):1701-1706.
15. Taylor A, Jr., Manatunga A, Morton K, et al. Multicenter trial validation of a camera-based method to measure Tc-99m mercaptoacetyl triglycine, or Tc-99m MAG3, clearance. *Radiology* 1997; 204(1):47-54.
16. Tello R, Thomson KR, Witte D, Becker GJ, Tress BM. Standard dose Gd-DTPA dynamic MR of renal arteries. *J Magn Reson Imaging* 1998; 8(2):421-426.
17. Dagher PC, Herget-Rosenthal S, Ruehm SG, et al. Newly developed techniques to study and diagnose acute renal failure. *J Am Soc Nephrol* 2003; 14(8):2188-2198.
18. Kabler RL, Cerny JC. Pre-transplant urologic investigation and treatment of end stage renal disease. *J Urol* 1983; 129(3):475-478.
19. Frankel DG, Narla D. Imaging of children with chronic renal failure. *J Pediatr* 1996; 129(2):s33-38.
20. Confer DJ, Banowsky LH. The urological evaluation and management of renal transplant donors and recipients. *J Urol* 1980; 124:305-310.

21. Taylor AJ, Cohen EP, Erickson SJ, Olson DL, Foley WD. Renal imaging in long-term dialysis patients: a comparison of CT and sonography. *AJR* 1989; 153(4):765-767.
22. Takebayashi S, Hidai H, Chiba T, Takagi H, Koike S, Matsubara S. Using helical CT to evaluate renal cell carcinoma in patients undergoing hemodialysis: value of early enhanced images. *AJR* 1999; 172(2):429-433.
23. Levine E. Acquired cystic kidney disease. In: Pollack HM, McClellan BL, eds. *Clinical Urography*. Philadelphia, Pa: WB Saunders Co; 2000:1343-1358.
24. De Broe ME, Elseviers MM. Analgesic nephropathy. *N Engl J Med* 1998; 338(7):446-452.
25. Elseviers MM, De Schepper A, Corthouts R, et al. High diagnostic performance of CT scan for analgesic nephropathy in patients with incipient to severe renal failure. *Kidney Int* 1995; 48(4):1316-1323.
26. Levine JA, Neitlich J, Verga M, Dalrymple N, Smith RC. Ureteral calculi in patients with flank pain: correlation of plain radiography with unenhanced helical CT. *Radiology* 1997; 204(1):27-31.
27. Zucchelli P, Zuccala A. The diagnostic dilemma of hypertensive nephrosclerosis: the nephrologist's view. *Am J Kidney Dis* 1993; 21(5 Suppl 2):87-91.
28. Scoble JE, Maher ER, Hamilton G, Dick R, Sweny P, Moorhead JF. Atherosclerotic renovascular disease causing renal impairment--a case for treatment. *Clin Nephrol* 1989; 31(3):119-122.
29. Stansby G, Hamilton G, Scoble J. Atherosclerotic renal artery stenosis. *Br J Hosp Med* 1993; 49(6):388-395, 398.
30. Taylor DC, Kettler MD, Moneta GL, et al. Duplex ultrasound scanning in the diagnosis of renal artery stenosis: a prospective evaluation. *J Vasc Surg* 1988; 7(2):363-369.
31. Berland LL, Koslin DB, Routh WD, Keller FS. Renal artery stenosis: prospective evaluation of diagnosis with color duplex US compared with angiography. Work in progress. *Radiology* 1990; 174(2):421-423.
32. Middleton WD. Doppler US evaluation of renal artery stenosis: past, present, and future. *Radiology* 1992; 184(2):307-308.
33. Schwerk WB, Restrepo IK, Stellwaag M, Klose KJ, Schade-Brittinger C. Renal artery stenosis: grading with image-directed Doppler US evaluation of renal resistive index. *Radiology* 1994; 190(3):785-790.
34. Stavros AT, Parker SH, Yakes WF, et al. Segmental stenosis of the renal artery: pattern recognition of tardus and parvus abnormalities with duplex sonography. *Radiology* 1992; 184(2):487-492.
35. Kliever MA, Tupler RH, Carroll BA, et al. Renal artery stenosis: analysis of Doppler waveform parameters and tardus-parvus pattern. *Radiology* 1993; 189(3):779-787.
36. Kliever MA, Tupler RH, Hertzberg BS, et al. Doppler evaluation of renal artery stenosis: interobserver agreement in the interpretation of waveform morphology. *AJR* 1994; 162(6):1371-1376.
37. Aitchison F, Page A. Diagnostic imaging of renal artery stenosis. *J Hum Hypertens* 1999; 13(9):595-603.
38. Emamian SA, Nielsen MB, Pedersen JF, Ytte L. Kidney dimensions at sonography: correlation with age, sex, and habitus in 665 adult volunteers. *AJR* 1993; 160(1):83-86.
39. Hallett JW, Jr., Fowl R, O'Brien PC, et al. Renovascular operations in patients with chronic renal insufficiency: do the benefits justify the risks? *J Vasc Surg* 1987; 5(4):622-627.
40. Mounier-Vehier C, Lions C, Devos P, et al. Cortical thickness: an early morphological marker of atherosclerotic renal disease. *Kidney Int* 2002; 61(2):591-598.
41. Reilly JM, Rubin BG, Thompson RW, et al. Revascularization of the solitary kidney: a challenging problem in a high risk population. *Surgery* 1996; 120(4):732-736; discussion 736-737.
42. Chen CC, Hoffer PB, Vahjen G, et al. Patients at high risk for renal artery stenosis: a simple method of renal scintigraphic analysis with Tc-99m DTPA and captopril. *Radiology* 1990; 176(2):365-370.
43. Setaro JF, Chen CC, Hoffer PB, Black HR. Captopril renography in the diagnosis of renal artery stenosis and the prediction of improvement with revascularization. The Yale Vascular Center experience. *Am J Hypertens* 1991; 4(12 Pt 2):698S-705S.
44. Davidson RA, Wilcox CS. Newer tests for the diagnosis of renovascular disease. *JAMA* 1992; 268(23):3353-3358.
45. Zuccala A, Zucchelli P. Ischemic nephropathy: diagnosis and treatment. *J Nephrol* 1998; 11(6):318-324.
46. Debatin JF, Spritzer CE, Grist TM, et al. Imaging of the renal arteries: value of MR angiography. *AJR* 1991; 157(5):981-990.
47. Kim D, Edelman RR, Kent KC, Porter DH, Skillman JJ. Abdominal aorta and renal artery stenosis: evaluation with MR angiography. *Radiology* 1990; 174(3 Pt 1):727-731.
48. Myers DI, Poole LJ, Imam K, Scheel PJ, Eustace JA. Renal artery stenosis by three-dimensional magnetic resonance angiography in type 2 diabetics with uncontrolled hypertension and chronic renal insufficiency: prevalence and effect on renal function. *Am J Kidney Dis* 2003; 41(2):351-359.
49. Bakker J, Beek FJ, Beutler JJ, et al. Renal artery stenosis and accessory renal arteries: accuracy of detection and visualization with gadolinium-enhanced breath-hold MR angiography. *Radiology* 1998; 207(2):497-504.
50. Christensson A. Renovascular disease and renal insufficiency--diagnosis and treatment. *Scand J Urol Nephrol* 1999; 33(6):400-405.
51. Bellin MF, Deray G, Assogba U, et al. Gd-DOTA: evaluation of its renal tolerance in patients with chronic renal failure. *Magn Reson Imaging* 1992; 10(1):115-118.
52. Prince MR, Arnoldus C, Frisoli JK. Nephrotoxicity of high-dose gadolinium compared with iodinated contrast. *J Magn Reson Imaging* 1996; 6(1):162-166.
53. Aspelin P, Aubry P, Fransson SG, Strasser R, Willenbrock R, Berg KJ. Nephrotoxic effects in high-risk patients undergoing angiography. *N Engl J Med* 2003; 348(6):491-499.
54. Jo SH, Youn TJ, Koo BK, et al. Renal toxicity evaluation and comparison between visipaque (iodixanol) and hexabrix (ioxaglate) in patients with renal insufficiency undergoing coronary angiography: the RECOVER study: a randomized controlled trial. *J Am Coll Cardiol* 2006; 48(5):924-930.
55. Glockner JF, Vrtiska TJ. Renal MR and CT angiography: current concepts. *Abdom Imaging* 2007; 32(3):407-420.
56. Leiner T, de Haan MW, Nelemans PJ, van Engelshoven JM, Vasbinder GB. Contemporary imaging techniques for the diagnosis of renal artery stenosis. *Eur Radiol* 2005; 15(11):2219-2229.
57. van Helvoort-Postulart D, Dirksen CD, Nelemans PJ, et al. Renal artery stenosis: cost-effectiveness of diagnosis and treatment. *Radiology* 2007; 244(2):505-513.
58. Vasbinder GB, Nelemans PJ, Kessels AG, et al. Accuracy of computed tomographic angiography and magnetic resonance angiography for diagnosing renal artery stenosis. *Ann Intern Med* 2004; 141(9):674-682; discussion 682.
59. Scola FH, Cronan JJ, Schepps B. Grade I hydronephrosis: pulsed Doppler US evaluation. *Radiology* 1989; 171(2):519-520.
60. Dalla-Palma L, Panzetta G, Pozzi-Mucelli RS, Galli G, Cova M, Meduri S. Dynamic magnetic resonance imaging in the assessment of chronic medical nephropathies with impaired renal function. *Eur Radiol* 2000; 10(2):280-286.
61. Furukawa A, Murata K, Morita R. [Evaluation of renal function using Gd-DTPA dynamic MR imaging]. *Nippon Igaku Hoshasen Gakkai Zasshi* 1996; 56(5):264-274.
62. Gaspari F, Perico N, Ruggenti P, et al. Plasma clearance of nonradioactive iohexol as a measure of glomerular filtration rate. *J Am Soc Nephrol* 1995; 6(2):257-263.
63. Knesplova L, Krestin GP. Magnetic resonance in the assessment of renal function. *Eur Radiol* 1998; 8(2):201-211.
64. Laissy JP, Benderbous S, Idee JM, Chillon S, Beaufile H, Schouman-Claeys E. MR assessment of iodinated contrast-medium-induced nephropathy in rats using ultrasmall particles of iron oxide. *J Magn Reson Imaging* 1997; 7(1):164-170.
65. Sterner G, Frennby B, Hultberg B, Almen T. Iohexol clearance for GFR-determination in renal failure--single or multiple plasma sampling? *Nephrol Dial Transplant* 1996; 11(3):521-525.
66. Chandna SM, Farrington K. Residual renal function: considerations on its importance and preservation in dialysis patients. *Semin Dial* 2004; 17(3):196-201.
67. Dittrich E, Puttinger H, Schillinger M, et al. Effect of radio contrast media on residual renal function in peritoneal dialysis patients--a prospective study. *Nephrol Dial Transplant* 2006; 21(5):1334-1339.
68. Sterner G, Frennby B, Mansson S, Ohlsson A, Prutz KG, Almen T. Assessing residual renal function and efficiency of hemodialysis--an application for urographic contrast media. *Nephron* 2000; 85(4):324-333.

69. Moranne O, Willoteaux S, Pagniez D, Dequiedt P, Boulanger E. Effect of iodinated contrast agents on residual renal function in PD patients. *Nephrol Dial Transplant* 2006; 21(4):1040-1045.
70. Weisbord SD, Bernardini J, Mor MK, et al. The effect of coronary angiography on residual renal function in patients on peritoneal dialysis. *Clin Cardiol* 2006; 29(11):494-497.
71. American College of Radiology. *Manual on Contrast Media*. Available at: http://www.acr.org/SecondaryMainMenuCategories/quality_safety/contrast_manual.aspx.

The ACR Committee on Appropriateness Criteria and its expert panels have developed criteria for determining appropriate imaging examinations for diagnosis and treatment of specified medical condition(s). These criteria are intended to guide radiologists, radiation oncologists and referring physicians in making decisions regarding radiologic imaging and treatment. Generally, the complexity and severity of a patient's clinical condition should dictate the selection of appropriate imaging procedures or treatments. Only those examinations generally used for evaluation of the patient's condition are ranked. Other imaging studies necessary to evaluate other co-existent diseases or other medical consequences of this condition are not considered in this document. The availability of equipment or personnel may influence the selection of appropriate imaging procedures or treatments. Imaging techniques classified as investigational by the FDA have not been considered in developing these criteria; however, study of new equipment and applications should be encouraged. The ultimate decision regarding the appropriateness of any specific radiologic examination or treatment must be made by the referring physician and radiologist in light of all the circumstances presented in an individual examination.

Appendix 1. The Five Stages of Chronic Kidney Disease

Your glomerular filtration rate (GFR) is the best indicator of how well your kidneys are working. In 2002, the National Kidney Foundation published treatment guidelines that identified five stages of chronic kidney disease (CKD) based on declining GFR measurements. The guidelines recommend different actions based on the stage of kidney disease.	
Increased risk of CKD. A GFR of 90 or above is considered normal. Even with a normal GFR, you may be at increased risk for developing CKD if you have diabetes, high blood pressure, or a family history of kidney disease. The risk increases with age: People over 65 are more than twice as likely to develop CKD as people between the ages of 45 and 65. African Americans also have a higher risk of developing CKD.	
Stage 1	Kidney damage with normal GFR (90 or above). Kidney damage may be detected before the GFR begins to decline. In this first stage of kidney disease, the goals of treatment are to slow the progression of CKD and reduce the risk of heart and blood vessel disease.
Stage 2	Kidney damage with mild decrease in GFR (60 to 89). When kidney function starts to decline, your health care provider will estimate the progression of your CKD and continue treatment to reduce the risk of other health problems.
Stage 3	Moderate decrease in GFR (30 to 59). When CKD has advanced to this stage, anemia and bone problems become more common. Work with your health care provider to prevent or treat these complications.
Stage 4	Severe reduction in GFR (15 to 29). Continue following the treatment for complications of CKD and learn as much as you can about the treatments for kidney failure. Each treatment requires preparation. If you choose hemodialysis, you will need to have a procedure to make a vein in your arm larger and stronger for repeated needle insertions. For peritoneal dialysis, you will need to have a catheter placed in your abdomen. Or you may want to ask family or friends to consider donating a kidney for transplantation.
Stage 5	Kidney failure (GFR less than 15). When the kidneys do not work well enough to maintain life, you will need dialysis or a kidney transplant.
In addition to tracking your GFR, blood tests can show when substances in your blood are out of balance. If phosphorus or potassium levels start to climb, a blood test will prompt your health care provider to address these issues before they permanently affect your health.	
From the National Kidney and Urologic Diseases Information Clearinghouse (NKUDIC) a Service of the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDKD), National Institutes of Health available at: http://kidney.niddk.nih.gov/kudiseases/pubs/yourkidneys/index.htm .	