BJR

Received: 21 December 2013 | Revised: 3 April 2014 | Accepted: 14 April 2014

doi: 10.1259/bjr.20140004

Cite this article as:
Granata A, Zanoli L, Clementi S, Fatuzzo P, Di Nicolò P, Fiorini F. Resistive intrarenal index: myth or reality?. Br J Radiol 2014;87:20140004.

REVIEW ARTICLE

Resistive intrarenal index: myth or reality?

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ABSTRACT

In renal diagnosis, the B-mode ultrasound is used to provide an accurate study of the renal morphology, whereas the colour and power Doppler are of strategic importance in providing qualitative and quantitative information about the renal vasculature, which can also be obtained through the assessment of the resistive index (RI). To date, this is one of the most sensitive parameters in the study of kidney diseases and allows us to quantify the changes in renal plasma flow. If a proper Doppler ultrasound examination is carried out and a critical analysis of the values obtained is performed, the RI measurement at the interlobar artery level has been suggested in the differential diagnosis between nephropathies. The aim of this review is to highlight the pathological conditions in which the study of intrarenal RI provides useful information about the pathophysiology of renal diseases in both the native and the transplanted kidneys.

Renal ultrasonography has acquired a strategic importance in the early detection of several renal diseases thanks to its non-invasivity, low cost, reliability and high sensitivity. The B-mode ultrasound is a widely used technique for the study of kidney morphology, including renal pelvis, to provide information on parenchymal echogenicity and to detect space-occupying lesions.

The characteristic ultrasonographic pattern in chronic kidney disease (small kidneys, reduced parenchymal thickness and detection of cysts) allows a simple and accurate diagnosis of this pathological condition. On the other hand, the diagnostic validity of the B-mode ultrasound in the detection of acute renal disease is still under debate because of the lack of sensitivity and specificity of the commonly used parameters such as the increase of renal size and the reduction of the parenchymal echogenicity.

The advantage of using Doppler ultrasound (DUS) lies in its ability in detecting not only renal morphological abnormalities but also functional ones; colour Doppler, power DUS and spectral analysis provide qualitative and quantitative haemodynamic information about the intrarenal and extrarenal vasculature highlighting changes in the renal blood flow.

The measure of renal resistive index (RI) or Pourcelot index is one of the most sensitive parameters in the study of disease-derived alterations of renal plasma flow.

The aim of this review is to evaluate the significance of the renal RI as a non-invasive marker of renal histological damage in several pathological conditions (Table 1).

EXAMINATION TECHNIQUE

To perform a correct measurement of RI, a standardized study protocol is required.

The first phase is to detect the correct B-mode acoustic window with a precise regulation of focus and gain; subsequently, the colour box is opened, trying to contain its extension both to facilitate the analysis of Doppler and to improve the sensitivity and the frame rate.

The colour Doppler functions are set for a study focused on interlobar arteries, that is, the highest gains possible, the use of the lowest filters and a low pulse repetition frequency (PRF) of 1–1.5 kHz that must be preferred while always limiting the aliasing phenomenon.

With the activation of the pulsed wave Doppler module, the sample volume is placed in the lumen of the vessel and the speed–time curve is recorded.

Table 1. Intraparenchymal renal resistive index (RI) and possible clinical meanings described in the literature

Clinical setting		RI	Proposed clinical value
All nephropathies		>0.75	Indicator of tubulointerstitial nephropathy[1]
AKI		>0.75	Useful in discriminating between ATN and pre-renal form[2]
Chronic renal failure		>0.80	Indicator of irreversible damage
		>0.70	Independent risk factor for worsening function[3–6]
Renal colic		>0.70	Signs of complete ureteral obstruction[7,8]
		ΔRI > 0.08–0.10	
Kidney transplantation		>0.80	In SKT graft, unfavourable prognostic factor[9]
		>0.80	Association with recipient survival[10]
		>0.75	Long-term RF for NODAT[11]
Diabetes	Type 1—children 7–15 years old	>0.64	Risk factor for diabetic nephropathy[12]
	Type 2	>0.70	Indicator of advanced glomerular lesions and/or arteriosclerotic lesions[13]
		>0.73	Predictor of DN and its progression[14]
Renal artery stenosis		>0.80	Poor renal improvement after PTA[15]
Cirrhosis		>0.78	Risk factor for HRS[12]

ΔRI, difference in resistive index; AKI, acute kidney injury; ATN, acute tubular necrosis; DN, diabetic nephropathy; HRS, hepatorenal syndrome; NODAT, new-onset diabetes after transplantation; PTA, percutanous transluminal angioplasty; SKT, single kidney transplantation.

The size of the sample volume must be set for interlobar arteries (approximately 1–2 mm) in order to avoid artefacts due to under or over sampling.

As for the colour Doppler, a careful adjustment of pulsed wave Doppler functions (PRF 1.5–3 kHz, gain, depth, filter wall and Doppler frequency) is crucial to obtain a correct speed–time curve. Through the measure of the peak systolic velocity (VPS) and the telediastolic velocity (VTD), and according to the RI = VPS − VTD/VPS formula, the renal RI is calculated.

Multiple Doppler sampling in three different areas of the kidney (*i.e.* upper, mid or lower pole) at the interlobar or arcuate arteries level has been shown to be more effective than a single sampling: by increasing the number of samples, by minimizing the intra- and interoperator variability and considering the RI to be a highly reproducible test.

The correct value of the RI is the arithmetic average obtained from the measurements.

In adults an RI value <0.70 is considered normal, while in newborn babies and children, up to 6 months old, an RI >0.70 should not necessarily be considered pathological.[16]

A correct measurement of renal RI can be very difficult in various clinical conditions or in the presence of several confounding factors such as severe hypotension, heart rhythm disorders including tachy–brady arrhythmias, renal compression for perirenal or subcapsular fluid collections, various extrarenal causes of impaired vascular elasticity and during the Valsalva manoeuvre.[17]

PARENCHYMAL NEPHROPATHIES

Several studies have shown that DUS parameters, particularly the RI measured at the level of the interlobar arteries, are correlated with biopsy parameters such as tubulointerstitial and vascular lesions, and many authors have reported an increased RI in tubulointerstitial nephropathies in comparison with nephropathies characterized by purely glomerular involvement.[3]

Although the mechanisms by which tubulointerstitial damage can increase RI remain unknown, interstitial fibrosis acting on post-glomerular vessels could increase resistance to renal cortical blood flow and reduce glomerular perfusion. Considering that tubulointerstitial damage is the best histological parameter that correlates with renal function, DUS can be an excellent predictor of long-term renal prognosis to detect tubulointerstitial lesions and reveal the increase in RI that correlates closely with the chronicity index, the progression of chronic renal disease and the onset of proteinuria and hypertension.[1,4,18,19]

To date, few studies have been carried out on patients affected by systemic vasculitis to evaluate the association between the anatomopathological findings and DUS parameters. Interestingly, the morphological changes of the Doppler waves found in these patients are similar to those of patients affected by renal microangiopathy related to haemolytic–uraemic syndrome. In this case, a reduction of the diastolic rather than the systolic pattern has been widely reported.[20] In patients affected by lupus glomerulonephritis, the detection with DUS of an RI value >0.70 is associated with an increased risk of development of chronic renal failure.[18] High–normal RI values (0.65–0.7) are related to an excellent response to steroid therapy in different glomerulopathies, whereas higher RI values (>0.7) are not

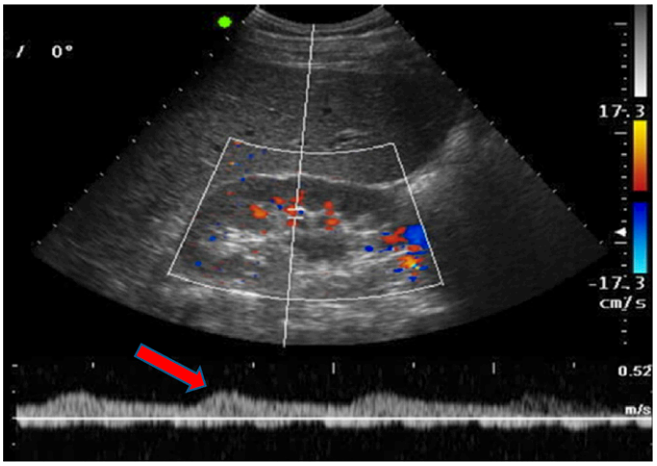
associated with significant improvements in renal survival, suggesting poor responsiveness.[4]

The remodelling of the arterial wall (especially in small vessels) plays an important role in the progression of hypertension by reducing vascular compliance, which causes an increase in vascular stiffness and increases peripheral resistance and pulse wave velocity.

Increased intrarenal RI is considered a marker of intrarenal arterial stiffness and is associated with the worsening of renal function and tubulointerstitial damage related to the inflammatory status of the essential hypertension: in humans, high-sensitivity C-reactive protein (hs-CRP) serum levels correlate with urinary markers of tubulointerstitial damage, and in hypertensive patients, the hs-CRP value directly correlates with the RI.[21]

The most common cause of renovascular hypertension is the renal artery stenosis (RAS) owing to atherosclerotic disease. In these patients, renal intraparenchymal RI has been proven to be higher than in the cases of RAS induced by Takayasu's arteritis or fibromuscular dysplasia, because, in atherosclerotic patients, arteriosclerosis, intrarenal arteriolosclerosis and glomerular sclerosis are associated with an increase in vascular stiffness, a reduction in arterial compliance and inward remodelling of the RA. When the main RA cannot be directly studied, an indirect evaluation of the stenosis can be made in more accessible arterial segments with DUS (hilar or interlobar arteries): the presence of RAS >80% proximal to these districts is commonly associated with the Doppler wave pattern of the "tardus" (slow)—"parvus" (little) pulsus in which a low systolic acceleration and a slow velocity of the waveform reveal a dampened flow[22] (Figure 1); the presence of a significant difference in the RI value ($\Delta RI > 0.06–0.08$) between the two kidneys can also be used to suggest the presence of an RAS, but this condition can be seen only for RAS in >80% of the vascular lumen.

Figure 1. The tardus et parvus waveform in a patient with renal artery stenosis. Note that the systolic acceleration is slow, and the systolic peak is dampened (arrow). The waveform has a rounded appearance.



Several studies have been performed in order to evaluate the therapeutic efficacy of percutaneous transluminal angioplasty (PTA) in cases of RAS. Recent trials have reported no benefit in blood pressure control and improvement of renal function or reduction of left-ventricular mass.[23] However, the benefits of RA stenting are still under debate. Our group and other authors have suggested that the lack of benefits of renal PTA could be owing to ineffective selection criteria used in these trials.[23,24] The monitoring of RI after PTA could be useful to predict the worsening of renal function: an RI value ≤0.75 before renal angioplasty and stenting is associated with good clinical outcomes,[25] whereas RI ≥0.80 obtained in segmental renal arteries is associated with no improvement in blood pressure, renal function and kidney survival.[15] Moreover, recent studies have shown that the longitudinal diameter of the kidney, the renal volume and RI are predictive of the therapeutic response after PTA.[26]

ACUTE KIDNEY INJURY

The intrarenal RI plays a crucial role in the differential diagnosis between the two most common types of acute kidney injury (AKI): the functional (pre-renal) AKI and the organic (renal) AKI. The former AKI is characterized by a reduction in renal perfusion and is rapidly reversible if promptly treated [otherwise, it can evolve into the organic form associated with an acute tubular necrosis (ATN)], whereas the latter AKI is caused by direct damage of the renal parenchyma, and the renal dysfunction tends to be persistent.

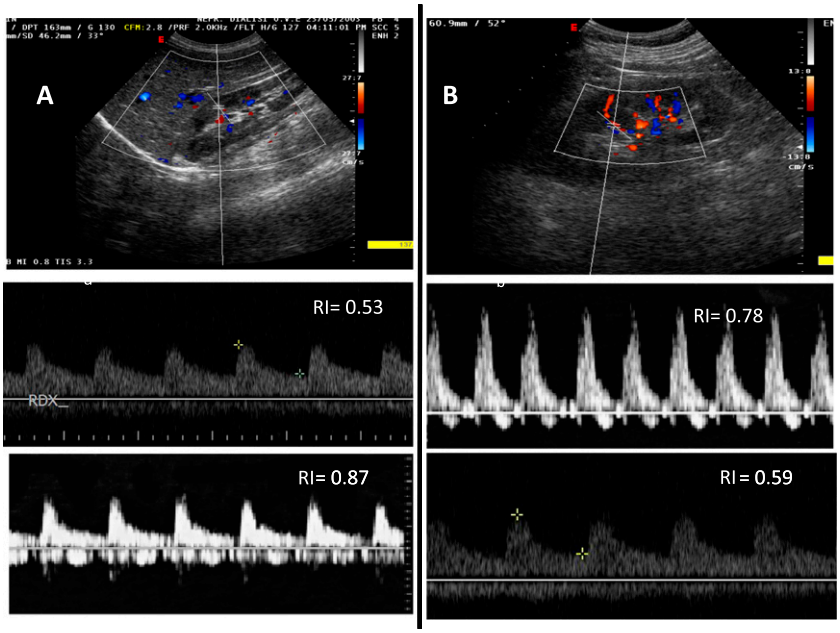
Although ATN is always characterized by a significant increase in RI values, the use of DUS is not conclusive in establishing the causes of AKI because increased intrarenal RI values are found in several pathological conditions such as hypovolaemia, rhabdomyolysis, sepsis, nephrotoxic substances and multiple organ failure.[27]

In a study by Platt et al,[2] B-mode ultrasound allowed the detection of morphological changes in only 11% of patients with AKI, while the DUS was a valid diagnostic tool: increased intraparenchymal RI (≥0.75) occurred in 91% of patients with ATN against only 20% of patients with pre-renal azotemia, and the mean RI of the ATN group was significantly higher than that of patients with pre-renal AKI (0.85 ± 0.06 against 0.67 ± 0.09, respectively).

RI is also a good predictor of the onset of AKI in the early postoperative period of patients undergoing cardiac surgery, and high values of RI have been used as an indicator of occult bleeding in patients with polytrauma.[28,29]

Thanks to its high specificity and sensitivity (85% and 92%, respectively), an RI >0.80 is a more reliable indicator of persistent AKI than that of common urinary markers, and as it does not require blood or urine samples, it is unaffected by changes in Na or Cr in urine or serum after diuretics or haemodialysis and can be performed at the bedside; it could be a promising tool to predict the reversibility of AKI in critically ill patients[30] (Figure 2).

Figure 2. Two cases of acute kidney injury (AKI). Left side of the image "Case A": a patient treated for transient AKI showed a clinical worsening to persistent AKI despite medical therapy with concomitant progressive elevation of resistive index (RI; 0.53–0.87). Right side of the image "Case B": an oliguric patient with a remarkable urea increase (RI, 0.78); the great response to therapy with clinical resolution of renal failure was anticipated by RI reduction (0.59).



CHRONIC RENAL FAILURE

RI is considered a marker of progression of renal damage, and an RI value >0.80 is an important indicator of irreversible damage in patients with chronic renal failure. In cases of RAS, the increase in RI is associated with a reduced likelihood of improved renal function after PTA and is a predictor of poor allograft survival after kidney transplantation.[9] Sugiura and Wada[5] have demonstrated that a value of RI >0.70 was an independent risk factor for worsening renal function in patients affected by chronic renal failure. An independent correlation has been reported between RI >0.70 and the percentage of serum creatinine variation, regardless of the initial value of the glomerular filtration rate (GFR).[31] In particular, subjects with RI ≥0.70 at baseline developed a rapid worsening of renal function with a reduction of GFR >50% after 6 years of follow-up.[6]

OBSTRUCTIVE UROLITHIASIS

Although helical CT is considered the gold standard imaging test for the detection of obstructive uropathy, the use of ultrasound is increasing steadily because 5% of all urinary calculi are radiolucent, and radiopaque calculi in the pelvic ureter may be confused with phleboliths.

Ultrasound is very sensitive in the detection of dilatation of the collecting system, but up to 50% of patients with acute urinary obstruction are not detected by B-mode ultrasound examinations because of the absence of dilatation.

Haemodynamic changes developing after a urinary obstruction could be owing to the increase of pelvic pressure, and the consequent increase in vascular resistances can be revealed by the RI shift; an RI value >0.70 and a ΔRI of 0.08–0.10 between obstructed patients and unobstructed ones were proven to be highly specific and sensitive parameters for acute complete ureteral obstruction.[7]

Other authors[32] have not confirmed these hypotheses perhaps because vascular reaction to the obstruction (vasodilatation followed by vasoconstriction) can be quite heterogeneous for the use of vasodilating drugs for pain treatment (*e.g.* non-steroidal anti-inflammatory drugs) or in the presence of intermittent obstruction.

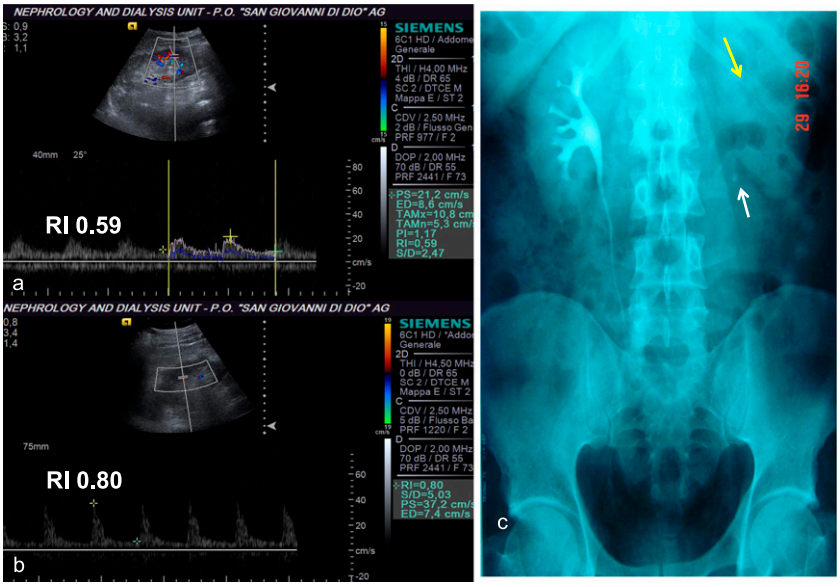
In fact, the increase of RI and ΔRI is related to the degree of ureteral obstruction, which is possibly because ureteral pressure has to exceed a threshold value to determine the increase of vascular resistances,[8] and high ΔRI is, indeed, significantly related to the presence of a functionally excluded kidney[33] (Figure 3).

Moreover, RI is probably a time-dependent index, with a powerful diagnostic value in acute renal obstruction between 6 and 48 h after the onset of symptoms.[32] Taking these limits into account, DUS is a sensitive and specific imaging test in the diagnosis of total obstruction and represents the first-line imaging test in cases of renal dysfunction, in pregnant women or in patients who are allergic to the contrast media.[8,33]

TRANSPLANTED KIDNEY

B-mode ultrasound and colour Doppler are the most widely used imaging tests in the study and follow-up of a transplanted kidney thanks to the kidney's easily assessable position: the two methods allow us to identify surgical, urological and vascular complications both in the early post-operative period and in the long term. Significant evidence suggests that DUS is not useful in differentiating between several medical complications of the

Figure 3. A patient with left flank pain radiating in the groin and microscopic haematuria. No hydronephrosis was found on standard ultrasound examination. With the Doppler ultrasound study, a difference between the (b) left kidney resistive index (RI) (0.80) and (a) right kidney RI (0.59) emerged. (c) On urography, the left kidney was functionally excluded (upper arrow) because of a calculus (lower arrow).



renal allograft, such as ATN, chronic vascular rejection and nephrotoxicity of drugs, as these pathological conditions are all characterized by an increase in RI values. For these reasons, causal diagnosis of graft dysfunction should be performed only by biopsy.[34] Conversely, high RI of the renal allograft is a prognostic factor in the early post-operatory period, since DUS is useful in evaluating the antirejection treatment by promptly detecting the rise of RI, a situation associated with poor prognosis of the graft.[9] RI >0.80 represents an unfavourable prognostic factor for the survival of both the graft and the patient.[9]

Recent studies have demonstrated the role of the pulse pressure and urinary albumin excretion as risk factors in the development of post-transplant diabetes (PT-DM), suggesting the involvement of microcirculation in the pathogenesis of this disease: a study performed on 4908 patients (mean follow-up 5.7 years) has shown that high values of RI, assessed in the immediate post-transplant period, are a risk factor for the long-term onset of PT-DM.[11]

In a recent study by Naesens et al[10] involving 321 renal allograft recipients, the RI routinely measured at pre-defined time points after transplantation was not associated with renal allograft histological features but with overall graft survival. This fact appeared uniquely attributable to the consistent association between the RI and recipient death (*i.e.*, loss of a functioning graft because of patient's death) leading to the conclusion that RI reflected characteristics of the recipient but not those of the graft. In other words, the RI indeed acted as a recipient survival indicator and not as a graft survival one.

DIABETIC NEPHROPATHY

Diabetic nephropathy (DN) is defined as persistent proteinuria >500 mg per 24 h or albuminuria >300 mg per 24 h. Increasing evidence suggests that the increase of RI is a sensitive marker of renal damage, including any ischaemic pathological conditions owing to the endothelial dysfunction, and that DUS can significantly contribute, more than the other clinical parameters (proteinuria, hypertension and diabetic retinopathy), to the identification of underlying nephropathy in Type 2 diabetes mellitus (T2-DM) subjects.[35] Diabetic patients without microalbuminuria present significantly higher baseline RI and a lower response to the vasodilatory effect of nitroglycerin than those of hypertensive or healthy subjects, confirming the role of RI in the early detection of vascular disease in diabetic patients.[36]

In T2-DM subjects with normal renal function, an RI value >0.70 has been closely associated with advanced arterioscleorotic lesions[13] and with the future development of proteinuria. Consequently, DUS may be helpful in selecting those patients at increased risk of developing DN and the worsening of the renal function in an early stage, even before the onset of microalbuminuria.[14]

In our experience, it seems that the detection of RI values >0.72 suggests the diagnosis of diabetic nephropathy, reducing the indications to renal biopsy only in the presence of values <0.72.[37]

Although, over the past decades, the incidence of Type 1 diabetes mellitus (T1-DM) has constantly increased, <1% of these subjects have developed a DN. Several studies[19] have reported an early involvement of the glomerulus in patients with T1-DM even before the onset of clinical manifestations. Youssef et al[12] have reported a positive correlation between the increase of RI and age, duration of disease, and levels of glycated haemoglobin and GFR in children with T1-DM.

Thus, the increase in RI is considered an early marker of diabetic nephropathy in children affected by T1-DM.[38]

CIRRHOSIS AND HEPATORENAL SYNDROME

The hepatorenal syndrome (HRS) is the most severe renal complication in end-stage liver diseases, and it should be suspected in the case of an increase in urea, sodium retention and oliguria without any specific and detectable cause of kidney damage. Although a precise pathophysiological mechanism has not yet been clarified, it is assumed that the accumulation of vasoactive amines and the consequent imbalance between vasoconstrictors and vasodilators may cause the haemodynamic changes responsible for the hypoperfusion of the kidney.

The increase in serum creatinine is not helpful in providing an early diagnosis of HRS, as it occurs at a late stage, whereas the increase in renal vascular resistance and the reduction in renal plasma flow is detectable early and develops progressively. This pathological condition is associated with peripheral systemic arterial vasodilatation combined with renal intense vasoconstriction and is responsible for the development of renal failure within weeks or months. Therefore, the rise in RI related to increased renal vascular resistance provides us with an early assessment of these haemodynamic variations before clinically overt disease occurs.[39]

Cirrhotic patients with ascites and/or oesophageal varices presented significantly higher RI values than those of compensated cirrhotic patients without ascites and/or oesophageal varices and healthy subjects. In this regard, almost half of patients with RI >0.70 have ascites, and the degree of renal vasoconstriction varies according to the severity of ascites. Studies[40] carried out on hepatopathic patients have reported that RI is lower in subjects with porto systemic shunt than in those without. To date, liver transplantation is considered the only effective treatment for these subjects. The renal RI values have been shown to rapidly reduce after transplantation despite serum creatinine levels remaining almost constant. Many authors have demonstrated a positive correlation between high pre-transplant RI (>0.70), post-transplant morbidity and a poor outcome.[41] In conclusion, high RI (≥ 0.78) is an extremely sensitive and specific parameter for the early identification of high-risk patients of HRS, and DUS is a useful HRS tool.

CONCLUSIONS

By providing information about intraparenchymal vascularization and, in particular, through the measurement of renal RI and its changes over time, the DUS is a very useful tool in several nephrological diseases.

The RI measure at the interlobar artery level can help in identifying patients at risk of progressive kidney disease, in predicting the worsening of renal function in specific clinical settings and in obtaining a proper nephrologic diagnosis for both transplanted and native kidneys.

In order to obtain reliable and repeatable data, considerable competence in measuring RI and the operator's extensive experience in using the DUS module are mandatory.

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