The prenatal detection of urinary tract anomalies is changing paediatric practice but in many areas the impact on clinical outcome remains difficult to quantify. However it is already apparent that termination of pregnancy has reduced the numbers of infants with lethal pulmonary hypoplasia and renal dysplasia who would previously have been liveborn but destined to succumb as neonates. Similarly, referrals of major non-lethal abnormalities such as bladder extrophy are declining as parents increasingly opt for termination.

Fetuses at greatest risk of early onset postnatal renal failure can now be identified with considerable accuracy on prenatal ultrasound. Termination, prompted by quality of life considerations, could result in reduced numbers of infants and young children requiring end stage renal failure treatment in the first few years of life. Prenatal detection of anomalies such as PUJ obstruction and reflux undoubtedly provides an opportunity to avert functional deterioration and minimise urinary infection. But the proportion of children who genuinely benefit has proved difficult to assess. The prenatal detection of mild dilatation is of doubtful benefit in all but a minority of cases. Clinically significant underlying pathology is rare yet this common prenatal finding often generates disproportionate parental anxiety.

**INTRODUCTION: EPIDEMIOLOGY AND METHODOLOGY**

The introduction of routine prenatal ultrasonography has had a profound impact on obstetric practice, and although the benefits have sometimes been questioned, it is unrealistic to contemplate returning to the pre-ultrasound era. Anomalies of the fetal urinary tract are well visualised by ultrasound and, in the two decades since the first reported cases of prenatally detected uropathy appeared in the literature, prenatal diagnosis has rapidly become an established feature of paediatric urological practice throughout the Western world. It is important to note, however, that the prenatal diagnosis of urological disorders has largely emerged as a product of obstetric practice rather than as a goal specifically sought by paediatric clinicians.

Whilst prenatal ultrasound has unquestionably altered the pattern and timing of the diagnosis of urological anomalies, its contribution to management and clinical outcome is more difficult to assess. Indeed any analysis of the effects of prenatal diagnosis on long-term outcome is beset by problems of interpretation and methodology. Many of the disorders now being routinely detected in utero, such as pelvi-ureteric junction (PUJ) obstruction, vesico-ureteric reflux (VUR) and multicystic dysplastic kidney (MCDK) are clinically undetectable at birth. Despite the extensive experience of prenatal diagnosis acquired over the last two decades it remains unclear what proportion would have been destined to present clinically, and what proportion would have remained clinically ‘silent’ and possibly unrecognised throughout the affected individual’s lifetime. Furthermore, any historical analysis of the impact of prenatal diagnosis on clinical outcome is limited by the paucity of reliable population-based information dating from the pre-ultrasound era. Finally, even where reasonably reliable historical data are available, as for posterior urethral valves (PUV), it is difficult to establish to what extent reductions in mortality and medium-term morbidity are specifically attributable to prenatal diagnosis – as opposed to broader advances in neonatal and nephrological care over the last two decades.

From this preamble it will be apparent that any assessment of the capacity of prenatal diagnosis to alter clinical outcome is inevitably a somewhat arbitrary and imprecise exercise.

**CATEGORIES OF UROPATHY**

Congenital anomalies of the urogenital tract span a broad pathological spectrum and for this reason are best considered according to their severity as follows:

1. Lethal uropathy
2. Non-lethal uropathy of a severe nature resulting in early onset renal failure
3. Non-lethal uropathy carrying a low risk of renal failure
4. Mild dilatation (otherwise termed ‘pelvicaliectasis’ or ‘pyelocaliectasis’).

**LETHAL UROPATHY**

Routine fetal anomaly scans performed in the second trimester currently constitute the only formal screening for urological abnormalities in the general, low-risk
fetal population. In a 3-year prospective study of approximately 175,000 pregnancies in the Yorkshire region, 369 pregnancies were terminated following the discovery of severe fetal anomalies – of which 35 were terminated for urinary tract malformations (Brand et al., 1994).

Of these, renal agenesis and MCDK (or combinations thereof) were the most common diagnoses, prompting more than 60% of terminations, whilst bladder outflow obstruction in male fetuses accounted for most of the remainder. Comparisons with Ashley and Mostofi’s (1960) historical autopsy data indicate that the frequency of bilateral renal agenesis in terminated pregnancies is broadly comparable to the incidence with which bilateral renal agenesis previously figured as a ‘renal’ cause of stillbirth or neonatal death.

Since bilateral agenesis is invariably lethal, prenatal diagnosis and termination only alter the timing of the inevitable demise rather than the outcome itself. By contrast, the potential impact of prenatal diagnosis in fetuses with bladder outflow obstruction is less easy to define, since, unlike bilateral renal agenesis, this is not an ‘all or nothing’ malformation, but a spectrum of severity extending from urethral atresia with lethal renal dysplasia to minor forms of urethral valve obstruction with little or no impairment of renal function. In practice, however, minor forms of urethral obstruction tend not to give rise to detectable dilatation as early as the second trimester and those cases that are detected by fetal anomaly scanning are usually at the severe end of the pathological spectrum. Moreover, qualitative aspects of the ultrasound appearances such as oligohydramnios, bladder distension and ‘bright’, echogenic kidneys also serve to identify severe outflow obstruction – although not always with sufficient specificity to differentiate fetuses destined to succumb from pulmonary hypoplasia from those who would survive the neonatal period and develop early-onset renal failure. Thus, prenatal diagnosis can be thought to have altered the outcome in some cases of severe urethral obstruction which resulted in termination but which nevertheless would have been compatible with survival into the neonatal period (Figure 1). In addition, the advent of prenatal diagnosis (and termination of pregnancy) has coincided with a dramatic decline in new cases of prune-belly syndrome, which, whilst sometimes associated with renal dysplasia, is in many cases accompanied by surprisingly good renal function and relatively normal quality of life.

NON-LETHAL UROPATHY – EARLY-ONSET RENAL FAILURE

Although congenital urethral obstruction (Figure 2) (due mainly to PUV) accounts for only 10% of prenatally detected uropathies, it generates a disproportionate burden of morbidity – as reflected in the fact that males account for 90% of infants and children receiving treatment for end-stage renal failure in the first 4 years of life. Correspondingly, congenital urethral obstruction and associated renal dysplasia account for almost 70% of renal transplants performed in the children aged under 5 years (Avner et al., 1995; Minoja et al., 1995).

It is now apparent from a number of studies that the

Figure 1—Early neonatal death. Urethral obstruction associated with lethal pulmonary hypoplasia. Case dating from the 1980s. Termination of pregnancy alters the timing of demise rather than the lethal outcome itself

Figure 2—Characteristic prenatal ultrasound appearances of the dilated fetal bladder and posterior urethra in posterior urethral valves (PUV)
likelihood of early-onset renal failure can be largely predicted from the ultrasound appearances of the urinary tract in the second trimester. In a study undertaken in the author's unit (Hutton et al., 1994), the medium-term functional outcome in a group of 17 affected infants whose urinary tracts had demonstrated the characteristic ultrasound features of severe urethral obstruction before 24 weeks' gestation was compared with the outcome in a second group of 14 boys whose second trimester ultrasound appearances had been normal and in whom urinary tract dilatation had only become apparent on subsequent scans. Of those diagnosed in the second trimester, 24% subsequently died in infancy from pulmonary and/or renal failure and 35% went on to develop early-onset renal failure. By contrast, where the second trimester scan had been normal and dilatation only developed after 24 weeks, none succumbed in infancy and with one exception all (93%), maintained normal renal function in early childhood. Other authors have also identified early distension of the fetal bladder as a sensitive predictor of poor functional outcome – particularly when combined with other ultrasonographic markers of outflow obstruction such as bladder wall hypertrophy and oligohydramnios. For example, Quinn and colleagues observed that dilatation of the fetal bladder before 28 weeks carried a poor functional prognosis, regardless of its aetiology (Quinn et al., 1996). These and other studies have identified ultrasonographic prognostic markers that will predict, with considerable accuracy, those fetuses at greatest risk of pulmonary hypoplasia and early-onset renal failure (Table 1).

Confronted with this information parents and clinicians have a number of options. They may decide to allow the pregnancy to run its course, in which case prenatal diagnosis will not have altered the outcome for the affected fetus, although for the parents the knowledge of the severe fetal anomaly will inevitably have a profound psychological impact on them for the duration of the pregnancy. Alternatively parents and their medical advisers have the option of utilising this information to alter the outcome of the pregnancy in a number of ways.

**Termination of pregnancy**

The treatment of chronic and end-stage renal failure in infants and young children entails prolonged hospitalisation, repeated admissions and surgical interventions. For many parents, the implications for the quality of life of their unborn child will weigh heavily in their decision to opt for termination.

**Fetal intervention (fetal surgery)**

Experimental work undertaken by Harrison and his colleagues in San Francisco provided the scientific rationale for the introduction of fetal intervention into clinical practice in the early to mid-1980s (Harrison et al., 1982). Unfortunately the benefits of intrauterine decompression of experimentally induced bladder outflow obstruction demonstrated in animal models, notably the fetal lamb, have not translated into clinical practice. Where results have been published, they have generally been disappointing and the early enthusiasm for fetal intervention waned throughout the 1980s and early 1990s. In 1986, the Fetal Surgery Register reported an overall mortality rate of 59% in a series of 73 fetuses treated in utero (Manning et al., 1986). Elder and associates (1987) reviewed the (largely unfavourable) outcome in a further 57 published cases, noting a 45% incidence of significant procedure-related complications. By the mid-1990s, fetal intervention was confined to a few centres and was being undertaken on an ad hoc and largely unreported basis. In 1999, however, Freedman and associates in Detroit reported their continuing experience with fetal intervention over the period 1987–1996 in a series of 34 fetuses treated by vesico-amniotic shunting (Freedman et al., 1999). Fourteen of the seventeen surviving infants were reassessed after 2 years of age – by which time eight (57%), had developed renal failure and five had already undergone renal transplantations. From the total of 34 treated fetuses only six (18%) were alive with adequate renal function at the time of follow-up.

Although various biochemical markers have been studied in the hope of identifying fetuses with deteriorating but potentially recoverable function (rather than those with irreversible renal dysplasia) predictive sensitivity is poor and selection for fetal intervention remains an imprecise exercise.

Proponents of fetal surgery have argued that the disappointing results reflect the fact that its use has largely been reserved for fetuses with the worst prognosis. Perhaps the indications for fetal intervention should be expanded to include virtually every fetus with evidence of bladder outflow obstruction? A controlled trial would be required to establish whether fetal intervention would be of benefit for fetal uropathy of intermediate severity, but for a variety of practical reasons it seems unlikely that hypothesis could ever be put to the test.

**Elective preterm delivery and ex utero early treatment**

Many paediatric urologists have formed the anecdotal impression that infants with PUV who have been born prematurely and treated shortly after delivery have developed better levels of renal function than would otherwise have been expected. However, this approach
has not been formally studied and the perceived benefits for renal function have to be carefully balanced against the undoubted risks associated with prematurity and increased maternal morbidity. Nevertheless, if premature labour occurs spontaneously, it may be reasonable to allow it to proceed, providing there are no major concerns regarding lung maturity.

Implications for paediatric urological and nephrological services

Paediatric urologists in many Western European countries, including Roman Catholic countries, are witnessing declining referrals of infants with major lower urinary tract malformations such as bladder extrophy and its variants (Figure 3). Whilst these anomalies do not usually pose a threat to renal function they do nevertheless demand major reconstructive surgery, entailing multiple procedures, prolonged or repeated hospitalisation and lifelong requirement for self-catheterisation. Four out of five sets of parents referred to the Hospital for Sick Children, Great Ormond Street, London, following a prenatal diagnosis of bladder extrophy, opted to terminate the pregnancy despite receiving specialist counselling (Dhillon, personal communication, 1999).

Recently the Department of Health for England and Wales announced its decision to limit recognition and funding for the treatment of bladder extrophy to two major centres whereas previously this condition was managed in every region throughout these countries. The diminishing workload of new cases (due, at least in part, to prenatal diagnosis and termination) was an important factor in this decision and it is possible that, in the future, similar considerations may drive the rationalisation of renal failure and transplant services for young children. If termination of pregnancy was extended to include most cases of severe but non-lethal fetal uropathy this could be anticipated to result in a diminishing requirement for renal replacement therapy and transplantation in children under 5 years of age since this workload consists predominately of boys with congenital urethral obstruction and renal dysplasia.

In summary, the evidence of recent years indicates that prenatal diagnosis is already altering the outcome of major uropathies and bladder malformations by prompting termination of affected pregnancies. A significant reduction in the numbers of newborn infants with these conditions would have implications for the provision and organisation of the relevant specialist paediatric services.

NON-LETHAL UROPATHY – LOW RISK OF RENAL FAILURE

Although some of the conditions in this category of severity are described in more detail by other contributors to this issue, those aspects relating specifically to the impact of prenatal diagnosis on clinical outcome are considered below.

Pelvi-ureteric junction (PUJ) obstruction

PUJ obstruction accounts for between 35% and 50% of all prenatally detected uropathies and occurs bilaterally in 10–20% of cases. Although fetal intervention (nephrostomy) was performed on an occasional basis in the 1980s, there is no evidence of benefit and it is difficult to envisage any circumstances in which this form of fetal intervention could now be justified.

Ideally, prenatal detection affords an opportunity to counsel parents on the possible requirement for postnatal surgery but, in practice, the indications for pyeloplasty in an individual case may be difficult to predict without the additional information provided by postnatal isotope imaging.

Antibiotic prophylaxis is usually prescribed for infants with prenatally detected PUJ obstruction – on the justification that before the days of prenatal diagnosis infection was the most frequent mode of presentation of PUJ obstruction in the first year of life. Whether prophylaxis is effective in reducing the risk of infection has not been proven since the relevant controlled trial has not been undertaken. However, some empirical evidence does exist to support this approach and to suggest that when infection does occur its severity and duration is reduced (because prenatal ultrasound has alerted parents and doctors to the existence of the PUJ obstruction, avoiding delayed diagnosis).

Contrary to the claims of some early reports, early pyeloplasty rarely, if ever, results in significant functional recovery in obstructed kidneys which already demonstrate impaired function at the time of initial surgery.
postnatal assessment. For example, McAleer and Kaplan (1999) found that regardless of the level of pre-operative function there was no functional recovery in 79 infants following pyeloplasty performed as early as a mean age of 6 months. The more relevant question, therefore, is whether prenatal diagnosis can alter functional outcome by identifying PUJ obstruction at a stage when progressive deterioration can be averted by pyeloplasty?

According to data published by Chertin and associates (1999) this would appear to be the case. These authors found that poor function (defined as <30% differential function) was present in only 12% of 50 infants with prenatally detected PUJ obstruction assessed at a median age of 11 months. By contrast, poor function was found in 89% of a group of 63 children whose PUJ obstruction had been diagnosed prenatally or neonatally but who had then been lost to follow-up until they presented again at around 4–5 years of age with symptomatic complications.

Whilst these findings appear to demonstrate that the outcome is favourably altered by early pyeloplasty, closer scrutiny raises questions about the methodology of this comparative study. In particular, the incidence and the severity of renal damage in their series of clinically presenting children was far greater than most other published series. For example, Capolicchio and associates (1999) reported that differential function was normal (>40%) in more than half the children with clinically presenting PUJ obstruction studied. However, these authors, in common with Chertin et al., did observe some difference in mean differential function between prenatally detected PUJ obstruction and PUJ obstruction presenting clinically in later childhood.

Unfortunately, these and similar studies provide only a ‘snapshot’ of the functional status of two populations of different ages and in whom the severity and natural history of obstruction may not be comparable. A linear study comprising prospective measurement of differential function in a defined population represents a more reliable method of assessing the true risk of functional deterioration. To date, the only study to meet these criteria has been the randomised controlled trial conducted at the Hospital for Sick Children, Great Ormond Street, London (Dhillon, 1998) in which 48 children underwent pyeloplasty whilst 52 were managed non-operatively. Of those managed conservatively, nine (17%) subsequently developed functional deterioration and underwent pyeloplasty – resulting in functional recovery in most (but not every) case. Over the period of the study, 14 conservatively managed kidneys (27%) showed evidence of resolving obstruction, whilst 29 (56%) retained stable function but remained obstructed. In a study specifically aimed at assessing the ‘recoverability’ of function, Subramaniam and associates (1999) reported that functional recovery was less likely to occur when pyeloplasty had been delayed beyond 2 years of age.

Although there is now an extensive literature on prenatally detected PUJ obstruction it remains at best confusing and at worst frankly contradictory. Insofar as there are consistent themes they are as follows:

1. Functional damage that has occurred in utero cannot be reversed by early pyeloplasty.
2. The principal rationale for pyeloplasty lies in averting further functional deterioration.
3. The overall risk of significant functional deterioration in the first few years of life is difficult to quantify but is probably of the order of 15–20%.
4. The individual risk of functional deterioration broadly correlates with the severity of dilatation – being less than 10% where the antero-posterior (AP) diameter of the renal pelvis is <20 mm, according to Dhillon and associates.
5. Careful monitoring is indicated when the pelvic AP diameter >20 mm since this carries a higher risk of functional deterioration, which may not always recover fully following pyeloplasty.
6. Increasing dilatation is a sign of impending functional deterioration and should signal the need to reassess function and drainage with a view to probable surgery.

In summary, the available evidence indicates that prenatal diagnosis is able to alter the long-term functional outcome of PUJ obstruction but the benefit is largely confined to the more severe end of the obstructive spectrum.

**Posterior urethral valves (PUV)**

Prenatal diagnosis has undoubtedly altered the presentation and medium-term outcome of PUV but it is too early to judge whether this will also be reflected in an improved long-term functional outcome and reduced risk of late-onset chronic renal failure. Prior to prenatal diagnosis, 50–60% of boys with PUV presented with infection that was often of life-threatening severity and associated with septicaemia, hypovolaemic shock and acidosis. Indeed, published series dating from the 1960s and 1970s documented an appreciable mortality from sepsis. Fortunately this clinical picture is extremely rare since more than 80% of cases are now detected prenatally, thus providing an opportunity to commence antibiotic prophylaxis and decompress the obstructed system within the first few hours or days after birth. Cases that are not detected prenatally tend to be at the milder end of the spectrum and probably carry a lower risk of overwhelming sepsis.

Although the last two decades have seen a substantial reduction in mortality from sepsis and overall medium-term morbidity it is seems unlikely that prenatal diagnosis is solely responsible since this timeframe also encompasses a period of considerable advances in paediatric nephrology and urology. Late morbidity most commonly takes the form of end-stage renal failure, which affects 15–30% of individuals over the course of 10–15 years of follow-up (Cuckow, 1998) and which may not become apparent until later childhood or adolescence. Amongst the
factors implicated in late-onset renal failure are congenital dysplasia, postnatal obstruction, high-pressure bladder dysfunction and pyelonephritis. Persuasive empirical evidence indicates that prenatal diagnosis does have the ability to ameliorate or prevent the effects of postnatal obstruction and infection. As yet, however, there is no justification for believing that prenatal diagnosis and early postnatal intervention can significantly change the prognosis for bladder dysfunction – or its impact on the upper tracts. More importantly, no form of postnatal intervention can alter the long-term consequences of renal dysplasia – which appears to play a greater role than was previously recognised. Unfortunately, despite prompt valve ablation, careful management and close follow-up, boys with prenatally detected PUV are continuing to enter end-stage renal failure in their early teens.

In summary, it can be confidently stated that prenatal diagnosis has contributed to a dramatic reduction in mortality and early morbidity but its ability to alter the long-term prognosis may be more limited. Moreover, it may be many years before any benefit can be assessed in view of the protracted natural history of renal failure in these patients.

Vesico-ureteric reflux (VUR)

Prenatally detected primary VUR, which accounts for 15–20% of clinically significant prenatally detected uropathies, is predominantly a disorder of male infants, with a male:female ratio of approximately 5:1 in most published series. This contrasts with clinically presenting reflux and reflux nephropathy in which females outnumber males by approximately 1.5:1. In addition to gender differences, the characteristics of prenatally detected reflux and its natural history indicate that infants identified by prenatal ultrasound comprise a subpopulation which is not representative of the paediatric reflux population as a whole. Urodynamic studies reported by Sillen (1998), Chandra et al. (1996), Yeung et al. (1997) and others have implicated fetal bladder dysfunction in the aetiology of high-grade primary reflux, which in some cases is thought to result from self-limiting transient urethral obstruction in utero. Prenatally detected reflux shows a strong tendency to resolve spontaneously. Elder (1998) reviewed seven published series totalling 413 prenatally detected refluxing units and noted that the overall figure for spontaneous resolution of reflux was 78% for grades I to III reflux and 36% for grades IV to V. Only 13% of children developed urinary infection during the course of medium-term follow-up on conservative management.

From our knowledge of the pathogenesis of infective renal scarring, prenatal diagnosis could be expected to alter the outcome favourably for the (undefined) proportion of boys with high-grade reflux who would otherwise have developed urinary infection in infancy – when the risk of scarring is greatest. In the absence of controlled trials this assumption remains unsubstantiated, but indirect support is provided by studies which have shown a lower incidence of renal scarring in infants with prenatally detected VUR when compared with infants presenting with infection. Further support can be invoked from sibling screening studies in which renal scarring has been shown to be more frequent in index cases whose reflux has presented clinically than in their asymptomatic siblings whose reflux had been identified during the course of screening. But whilst prenatal diagnosis is likely to confer protection against postnatal infective scarring, there is anecdotal evidence that in males with high-grade primary reflux congenital damage may also make a significant contribution to any long-term risk of renal failure. Moreover, the contribution to reducing the overall burden of reflux morbidity is likely to be very limited since prenatally detected cases account for only a small proportion of all children with reflux.

Multicystic dysplastic kidney (MCDK)

The introduction of routine prenatal ultrasound has revealed that the true prevalence of this developmental anomaly is far higher than was previously realised (Figure 4). Figures collected prospectively over a 10-year period at Northwick Park Hospital, London (Liebeschuetz and Thomas, 1997) put the prevalence at 1 in 2400 livebirths. However, the majority of prenatally detected MCDKs are small, impalpable and, in the days before prenatal ultrasound, would have remained undetected. Serial ultrasound has demonstrated that the natural history is characterised by involution, both pre- and postnatally. The arguments surrounding the role of ‘prophylactic’ nephrectomy are outside the scope of this review but centre on the perceived magnitude of the risks of hypertension and malignancy. Proponents of nephrectomy argue that removing the kidney alters the outcome favourably by abolishing these risks.

Conversely, advocates of conservative management (including the author) would argue that the bulk of available evidence points to a very low order of risk in relation to the true prevalence of the anomaly.

Figure 4—Multicystic dysplastic kidney (MCDK). The role of ‘prophylactic’ nephrectomy in prenatally detected cases remains controversial.
MILD DILATATION (‘PELVIC CALICESTASIS’)

This common sonographic finding is present in 1 in 100–200 pregnancies (Chitty et al., 1990) (Figure 5). Possible causes include low grade or ‘burnt out’ PUJ obstruction, VUR or a simple anatomical variant such as a prominent extra-renal pelvis. Numerous attempts have been made to identify a ‘cut-off’ value to differentiate between ‘pathological and physiological’ degrees of dilatation, and although an AP renal pelvic diameter of 1 cm has been widely used it has never been convincingly validated as a predictor of renal pathology. Moreover, very few studies have attempted to correct for gestational age. A notable exception is the study reported by Scott and Renwick (2001) who assessed the significance of renal pelvic diameters at different stages in gestation by correlating 813 measurements with the postnatal outcome. These authors concluded that a fetal renal pelvic diameter >7 mm at 18 weeks’ gestation is likely to denote a clinically significant degree of dilatation and they recommended follow-up and postnatal investigation in such cases. However, providing the renal pelvic diameter does not exceed 15 mm at any stage in gestation (or postnatal life) the data of Dhillon and associates point to a very low risk of clinically significant obstruction.

But whilst mild dilatation (not exceeding 15 mm in later pregnancy or on postnatal scans) can be effectively dismissed as an indicator of significant obstruction, its role as a possible marker for VUR is less well documented – particularly since ultrasound is a notoriously unreliable modality for the diagnosis of reflux at any age. Scott and Renwick (2001) found that reflux infrequently gave rise to prenatal dilatation and, when it did, this was predominantly a feature of males. Unfortunately the dilemma posed by the common finding of mild dilatation is compounded by the lack of a reliable non-invasive screening test for VUR. In practice, the conventional contrast cystogram entailing urethral catheterisation remains by far the most reliable investigation. There is little argument that an Micturating Cysto Urethrogram (MCU) is justified by the finding of calyceal or ureteric dilatation but these cases are considerably outnumbered by infants with ‘simple’ mild dilatation confined to the renal pelvis. In this situation the central question is ‘how many healthy infants with mild dilatation have to be submitted to an unnecessary MCU in order to identify one infant with VUR in whom this information is relevant to clinical outcome?’

When framed in these terms the clinical yield from routinely submitting infants to cystography appears to be low. For example, we followed 29 infants with mild dilatation for a mean of 4.2 years (representing a total of 122 child/years of follow-up). During this period, two children experienced a documented episode of urological morbidity – neither of which was related to reflux. MCUs were performed on half the children in the study. Mild reflux was identified in two boys and was managed conservatively without any evidence of infection during the period of follow-up (Thomas et al., 1994).

Nevertheless, this was a relatively small study and there remains a pressing need for a sizeable prospective study with medium- to long-term follow up to establish the sensitivity of mild dilatation as a marker for clinically significant reflux and to determine the extent, if any, to which this aspect of prenatal diagnosis alters outcome.

For clinicians concerned primarily with the implications for the affected fetus and infant it is easy to underestimate the impact on the parents of the prenatal diagnosis of a renal abnormality. Harding et al. (1999) measured anxiety scores in a questionnaire-based study designed to assess the psychological impact on parents of the prenatal diagnosis and postnatal management of ‘minimal hydronephrosis’ (defined by these authors as renal pelvic dilatation of
DOES PREGNATAL DIAGNOSIS ALTER OUTCOME?

CONCLUSIONS

It is clear that prenatal ultrasound scanning results in the detection of many renal abnormalities and has profoundly altered the spectrum of disease seen by paediatric urologists and nephrologists. It increases parental choice when faced with the diagnosis of a severe uropathy likely to result in perinatal death or significant morbidity due to early-onset renal failure. As such prenatal diagnosis has contributed to a reduction in mortality and early morbidity. The treatment of clinically silent pathology, however, remains unclear and there is a need for long-term follow-up studies to determine the natural history of many prenatally diagnosed conditions. There is some indirect evidence that prenatal diagnosis and early treatment in the neonatal period, antibiotic prophylaxis for reflux and PUJ obstruction, etc, which suggests that prenatal diagnosis may result in a decrease in long-term morbidity. However, it is likely that follow-up studies over many years will be required in order to assess the benefit in terms of later-onset renal failure.

REFERENCES


