personal NO_2 exposure with extrapolated levels from central monitoring stations, and concluded that ambient NO_2 concentrations should be used ''with caution'' in assessing individual exposure—rightly pointing out that a major source of NO_2 is gas cooking. Second, the association between NO_2 and SIDS may be confounded by correlated pollutants such as inhaled particles,[8] or an unrecognised social variable with a high spatial correlation with outdoor NO_2. Indeed, concomitant emissions of NO_2 and CO from vehicle exhausts[9] may account for the association between CO and SIDS. Third there is no biological explanation for a mechanism of interaction between NO_2 and SIDS, although in the past uncertainty about mechanisms has not been a barrier to successful SIDS reduction interventions.[10] One possible explanation is that NO_2 alters the pulmonary immunological response to trivial viral infections—an interaction that has been reported for asthmatic children.[11] Nevertheless, Klonoff-Cohen and colleagues' study,[4] whose findings are compatible with a recent Canadian report which found a significant association between daily rates of SIDS and increased NO_2 (and SO_2) on the previous day,[12] should help to refocus researchers' attention on gaseous pollutants, and young children as an important vulnerable age group. The methodological issues of research in this age group are challenging,[12] but newly developed computer models which calculate gaseous emissions and their dispersion at the spatial level of individual households,[13] may allow reanalysis of pre-existing birth cohort datasets. Until more data become available, no specific recommendations can be given to parents who are concerned about reducing the risk of SIDS. Wide variations in NO_2 occur within small spatial areas, and both avoiding exposure and living a normal life is virtually impossible. It may well be that regulators concerned about the potential health impact of NO_2 on young infants should not concentrate on this single pollutant, but aim to reduce all combustion products emitted within suburban areas. However, when developing exposure reduction policies, data on the association between NO_2 and SIDS will be important in any health impact analysis.

Arch Dis Child 2005;**90**:662–663.
doi: 10.1136/adc.2004.068379

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Competing interests: none

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Urology

Time to review the value of imaging after urinary tract infection in infants

K Verrier Jones

Commentary on the paper by Moorthy *et al* (see page 733)

Early descriptions of childhood urinary tract infection (UTI) focused on findings at postmortem examination or children referred to hospital because of chronic or recurrent infection often persisting for months or years.[1] Many of these children had gross vesicoureteric reflux (VUR), chronic pyelonephritis, and sometimes other serious underlying anomalies such as neurogenic bladder.[2] Further investigation revealed proteinuria, hypertension, anaemia, complicated pregnancies, and impaired renal function. Long term follow up studies have supported this impression, and in a significant proportion of children and adults, end stage renal failure is thought to be due to chronic pyelonephritis.[3] Such cases were often collected over many years and brought together for the purpose of describing the constellation of symptoms to other health professionals, with a view to identifying diseases and syndromes and starting to understand their causes and prevention. These early studies were not generally epidemiological studies but highly selected groups who showed the most severe or persistent symptoms.

The natural history of UTIs probably started to change in the 1950s with the advent of antibiotics and development of paediatric services. The radiological anomalies associated with recurrent UTIs, particularly vesicoureteric reflux and renal scarring, were described by Hodson and Edwards.[4] The high rate of detection of vesicoureteric reflux and renal scarring in children investigated following UTI prompted a call for routine imaging tests in all children following UTI in an attempt to detect high risk cases early and thus prevent avoidable renal scarring. This strategy assumed that renal scarring was both acquired and preventable, that vesicoureteric reflux and infection combined was the cause of renal damage, and that high risk cases could be clearly identified at an early stage through imaging tests such as intravenous urography and micturating cystography.

In the past two decades many of these assumptions have been challenged. Some children with vesicoureteric reflux and small or scarred kidneys have congenital renal defects that cannot be prevented by ureteric reimplantation or

prophylactic antibiotics.[5] Three important studies comparing reimplantation with prophylactic antibiotics failed to show benefit from ureteric reimplantation, and there are no controlled studies comparing prophylaxis with intermittent short course treatment for UTI.[6] Even the value of prophylaxis in preventing UTIs has now been challenged and there have never been studies to test the effectiveness of prophylaxis in the prevention of scarring.[7]

Over the past three decades there have been several reports of the non-specific symptoms of UTI in infants, and it has become clear that many cases have been missed, some in hospital and more in primary care.[8] This situation has changed gradually and sick children and infants with fever, vomiting, or failure to thrive are now usually tested for urine infection if they attend hospital and sometimes in primary care. Parents have increased expectations for referral to a hospital or paediatrician as an emergency if their child is unwell so that relatively few children are left untreated for long periods with symptomatic UTIs.

Children are often offered imaging and prophylactic antibiotics after the first UTI, based on the assumption that a third will have VUR, in line with the published guidelines of the Royal College of Physicians.[9] This is based on the premise that they are at increased risk of recurrent UTIs and that scarring in these children will be prevented by prophylaxis. However these assumptions are unproven and the potential value of imaging and prophylaxis in this group may well be different from the groups described in earlier studies.

Symptomatic UTI in infancy and childhood is now recognised as a common problem among healthy children affecting around 6–7% of girls and 2–3% of boys.[10] Since the publication of the guidelines in 1991, huge resources have been expended on referring young children to paediatricians and on to radiologists for imaging, which for children in the first year includes DMSA scanning and cystography. This latter test is particularly distressing, time consuming, expensive, invasive, and involves radiation.[11] VUR may be missed in up to 15% of cases, and there is a significant risk of introducing bacteria and causing UTI. To justify these risks to the patient and use of resources there should be clear benefits from this test and the subsequent interventions.

In this issue, Moorthy *et al* describe the outcome of cystography in 108 children after the first UTI in the presence of a normal ultrasound examination.[12] Although VUR was detected in 12% of renal units we are not told how many patients were affected. Abnormal DMSA scans were found in 4/25 (16%) refluxing renal units and 8/216 (4%) non-refluxing renal units. They used simple statistical tests to show that in the population studied, the presence of VUR is not a useful way of identifying children at high risk of renal scarring. These results are different from the historical reports on which current practice is based. It is useful to consider possible reasons for these differences.

The children described by Moorthy *et al* are all under 12 months and many will have been referred following the first UTI. They are younger and probably healthier than children described in the early studies. We are not told how urine was collected or what culture methods were used in the laboratory; however, unless invasive samples are collected by catheter or suprapubic puncture it is likely that there were some false positive samples. Although from a purely scientific view point this might be seen as a weakness, this represents the situation in many children's units in the UK. This could explain the relatively low incidence of VUR in this study. Similarly this could have contributed to the low prevalence of renal scarring detected. All children with anomalies of the urinary tract including single kidneys and urinary tract dilatation were excluded prior to the analysis.

In conclusion, a number of factors have been identified that may explain the difference between the results of the study by Moorthy *et al* and the results from historical observational studies. These factors include improved health care such as greater awareness of UTI in infancy, better diagnosis and earlier treatment of UTI, the widespread availability and use of antibiotics, and better child health surveillance. Differences between the populations described in terms of age, number of previous UTIs, presence of congenital anomalies detectable on ultrasound, and available health care can account for significant differences in prevalence of additional abnormalities detected at cystography and DMSA scans. Common sense dictates that it is inappropriate to use high volume high cost resources on invasive tests on healthy children after recovery from relatively trivial illness in the absence of evidence of benefit. A change in practice with greater emphasis on earlier detection and treatment of UTIs in the first year of life and less emphasis on imaging after the event is more likely to be effective in preventing renal damage as well as minimising the adverse effects of acute illness. This point has been made by the York Centre for Reviews and Dissemination in their recent publication on diagnosing urinary tract infection following a Health Technology Assessment.[13]

Arch Dis Child 2005;**90**:663–664.
doi: 10.1136/adc.2004.067330

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Competing interests: none declared

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