not told what the ultrasound diagnoses in the other 21 was, presumably dilatation of unknown cause. Cystic renal dysplasia can be accurately diagnosed by ultrasound in most cases if the strict criteria of non-communicating cystic spaces, which vary in size and number, is adhered to. It is well recognised that in a few cases it may be difficult to differentiate from hydropnephrosis but this is readily achieved either by cyst puncture or radionuclide scintigraphy. It is, however, good practice in all cases to perform a dimercapto-succinic acid (DMSA) scintigram to confirm total absence of function.

The place of postnatal renal tract ultrasound is quite clear. It should act as a guide to the next examination, although in many cases the specific diagnosis will be apparent. Tudor and Whitaker have recently described a protocol for the detection and management of the dilated fetal urinary tract, which is to be commended.3 The ultrasound scans should be performed by individuals skilled in paediatric ultrasound. In a tertiary referral centre one would expect all the scans to be repeated by such an individual before the initiation of treatment or further investigation.

References

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Gallstones in homozygous sickle cell disease

Sir,

We recently communicated detailed findings on the prevalence and risk factors for gall stones in children with homozygous sickle cell disease.1 The study has been repeated to include other sickle genotypes for which there are few data,2 and this has enabled comparison of gall stone prevalence between genotypes. It has also enabled us to examine the role of haemolytic rate and mean serum bilirubin concentration, which is the prime determinant of gall stone prevalence in homozygous sickle cell disease.1

The children who participated in the Jamaican cohort study of sickle cell disease were aged between 6 and 14 years at the date of the study. Gall stone prevalence was determined by real time ultrasound in a cross sectional study with informed consent in every case. Details of the number of children with each genotype, the total examined, and the prevalence of gall stones are given in the table. The mean serum total bilirubin concentration for each genotype was calculated from steady state values obtained at each child’s birthday after four years; this is because of the rapid change in haematological indices before this age.3

<table>
<thead>
<tr>
<th>Genotype</th>
<th>No of children in cohort study</th>
<th>No of children screened</th>
<th>No (%) with gall stones</th>
<th>Mean (range) serum bilirubin (μmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SS</td>
<td>242</td>
<td>218</td>
<td>30 (14)</td>
<td>35 (27–38)</td>
</tr>
<tr>
<td>SB</td>
<td>11</td>
<td>9</td>
<td>2 (22)</td>
<td>27 (20–35)</td>
</tr>
<tr>
<td>SC</td>
<td>152</td>
<td>138</td>
<td>4 (3)</td>
<td>19 (16–22)</td>
</tr>
<tr>
<td>SB°</td>
<td>33</td>
<td>25</td>
<td>0</td>
<td>15 (12–17)</td>
</tr>
</tbody>
</table>

Gall stones were more common in homozygous sickle cell (SS) than sickle cell/haemoglobin C (SC) disease (χ²=9.7, p<0.01), but other differences in prevalence were not significant. Gall stone prevalence was unreliable estimated in sickle cellβ (SB) thalassaemia due to small patient numbers. Mean serum bilirubin concentrations varied by genotype in a similar manner to gall stone prevalence, and data from the cohort study have shown differences by genotype in mean haemoglobin and reticulocyte count consistent with variation in haemolytic rate.4 Our data indicate that haemolysis, mean bilirubin, and gall stone prevalence vary by sickle genotype, and that the risk of gall stones is low in children with SC disease and SB° thalassaemia.

References

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Haemophilus influenzae type b disease in the Oxford region

Sir,

Tudor-Williams et al report an incidence of systemic Haemophilus influenzae type b disease in children less than 5 years of age of 33.4/100 000.1 This was determined from a detailed laboratory based study in the Oxford region and they urge others to set up similar prospective studies.

I have examined Hospital Activity Analysis (HAA) data for all deaths and discharges for H influenzae meningitis (ICD Code 320-0) and acute epiglottitis (ICD Code 464-3) in the South Western region from 1976–86 and 1979–86 respectively. These two groups accounted for 80% of all
invasive *H. influenzae* type b disease in those under 5 in the Oxford study. A total of 268 cases of meningitis and 118 cases of epiglottitis were identified. The annual incidence rate for *H. influenzae* meningitis in children less than 5 years of age was 14-1/100 000 (compared with 23-6/100 000 in Oxford) and 8-6/100 000 for epiglottitis, similar to that found in Oxford. Over the last four years, however, the incidence rate for *H. influenzae* has been 18-1/100 000, which may represent an improvement in data collection. If immunisation will prevent 85% of systemic *H. influenzae* type b disease the impact of such a programme will be detectable by monitoring HAA data. The Communicable Disease Centre at Colindale already collect details of invasive *H. influenzae* disease. Local surveillance could also include scrutiny of statutory notifications and death registrations. It would seem that information systems already in place should be able to monitor the effectiveness of introducing immunisation for *H. influenzae*.

References


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Effect of clonidine on serum gonadotrophin concentrations

Sir,

Clonidine, an alpha_2_ adrenergic agonist and a potent stimulator of pituitary growth hormone release, is being used in the treatment of short children in some centres.

Clonidine also causes release of luteinising hormone in rats, however, and restoration of suppressed luteinising hormone pulsatility. This observation could have important implications for the treatment of short children because clonidine might induce puberty. We report the effects of clonidine on serum gonadotrophins in normal subjects.

Clonidine (0-15 mg/m^2_ orally) was given at 09.00 (time 0) to eight healthy adult men volunteers and placebo to one subject. Twelve blood samples to measure luteinising hormone and follicle stimulating hormone were drawn at 15 minute intervals between −45 and 120 minutes through an indwelling venous cannula. Blood pressure was determined every 15 minutes throughout the test. The blood samples were centrifuged and the serum stored at −20°C until assay. Luteinising hormone and follicle stimulating hormone assays were performed by immunoradiometric assay kits from the North East Thames Regional Immunoassay Scheme in duplicates and the concentrations were measured as IU/l.

<table>
<thead>
<tr>
<th>Study group (n=8)</th>
<th>Luteinising hormone (IU/l)</th>
<th>Follicle stimulating hormone (IU/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal</td>
<td>4-2 (1-1)</td>
<td>4-4 (1-7)</td>
</tr>
<tr>
<td>Peak after clonidine</td>
<td>5-1 (1-5)</td>
<td>4-3 (1-7)</td>
</tr>
<tr>
<td>Control (n=1)</td>
<td>2-6 (0-7)</td>
<td>2-5 (0-6)</td>
</tr>
<tr>
<td>Basal</td>
<td>2-4 (0-5)</td>
<td>2-6 (0-5)</td>
</tr>
</tbody>
</table>

No significant decrease in blood pressure was observed. The results shown in the table clearly show that clonidine does not have any effect on gonadotrophin concentrations. Alpha_2_ adrenergic blockers reduce the frequency of luteinising hormone pulses and suppress pulsatile release of luteinising hormone in animals, implying that the major effect of the alpha-adrenergic system is to stimulate gonadotrophin releasing hormone release. When clonidine was administered to rats with abolished luteinising hormone pulsatility, it re-established luteinising hormone pulsatility and increased the secretion of luteinising hormone.

The reason why clonidine affected luteinising hormone pulsatility in the rats was that it was given in large doses. The small doses used in this study were effective on the alpha_2_ receptors with no apparent effect on luteinising hormone pulsatility.

References


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Arch Dis Child 1989 64: 1342-1343
doi: 10.1136/adc.64.9.1342-a

Updated information and services can be found at: http://adc.bmj.com/content/64/9/1342.2.citation

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