as high a concentration as this spontaneously. Nevertheless, there was no indication of increased aggression, though she was very dramatically virilised.

A wide range of ovarian tumours can secrete androgens.1 Though virilisation is often severe, serum testosterone concentrations are usually increased only up to the normal male range at the most. After resection of the tumour she showed a dramatic response that greatly exceeded everyone's expectations.

The diagnosis was delayed principally because the wrong test was carried out. This case shows that measurements of serum testosterone and androstenedione concentrations, perhaps with dehydroepiandrosterone sulphate, are very much better at excluding virilising tumours than urinary 17 ketosteroid excretion, which correlates poorly with serum androgen concentrations.2 Ketosteroid excretion generally represents the weaker adrenal androgens,3 and though her testosterone concentration 11 years previously was probably much lower than it was at the time of diagnosis it would doubtless already had been considerably increased at that time.

Comparison of proportions of children with rubella antibodies in 1985-6 and 1969 by age. Values expressed as number of children with antibody/number of children tested (as percentage)

<table>
<thead>
<tr>
<th>Age:</th>
<th>1-3 months</th>
<th>4-6 months</th>
<th>7-11 months</th>
<th>1-2 years</th>
<th>3-5 years</th>
<th>6-12 years</th>
<th>13-20 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>1985-6</td>
<td>10/12 (83)</td>
<td>1/2 (50)</td>
<td>3/5 (60)</td>
<td>6/18 (33)</td>
<td>4/8 (50)</td>
<td>6/18 (33)</td>
<td>1/2 (50)</td>
</tr>
<tr>
<td>1969*</td>
<td>14/19 (74)</td>
<td>3/18 (17)</td>
<td>0/19</td>
<td>4/47 (9)</td>
<td>10/41 (24)</td>
<td>32/40 (80)</td>
<td>42/51 (82)</td>
</tr>
</tbody>
</table>

* Figures from Brown et al.2

The table shows the distribution of rubella antibodies in the different age groups. For comparison the distribution of antibodies shown by haemagglutination inhibition in a similar study performed in Leeds in 1969 is included.4 Further analysis of the results showed that though only one (11%) of nine children aged 5 years who had acquired antibody, seven (64%) of 11 children aged 6 years were seropositive.

Comment

These findings show that passively acquired maternal antibody is usually lost by the age of 7 months and that it is rare to acquire active immunity under 2 years of age. Even in the group of children aged 3-5 years only 16% had evidence of having had rubella, and most children acquired antibody between 6 and 12 years of age. This pattern is similar to that found in 1969 and shows no evidence of having been influenced by the rubella vaccination programme. This is not surprising as the policy adopted in the United Kingdom in 1970 was a selective one aimed at protecting girls before child-bearing age and not at interrupting transmission of the virus. Although it has resulted in a decreased proportion of susceptible women of child-bearing age, a considerable number remain at risk of contracting rubella in pregnancy.14 To protect them it has been proposed that the present vaccination programme be augmented by immunising boys and girls in early childhood.3 Our findings indicate that if a high rate of uptake was achieved it would have a large impact on reducing the pool of infection and hence on preventing congenital rubella.

It is difficult to interpret the finding of rubella antibody in a child who presents with sensorineural deafness in the second or third year of life. The knowledge that only 4% of children in Leeds have acquired antibody by the age of 3 should help to clarify this difficulty.

We thank our consultant colleagues for allowing us to include their patients in the study.

   (Public Health Laboratory Service Monograph Series No 16.)

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**Fall and rise of immunity to rubella**

We report a study of the prevalence of rubella antibody in a sample of children and adolescents who attended the children's department at Leeds General Infirmary. The purposes of this study were to determine whether the age at which antibody is acquired had changed since the introduction of rubella vaccination in 1970 and to use the results as an aid in interpreting the importance of rubella antibody in young children suspected of having congenital rubella.

**Patients, methods, and results**

Blood was obtained from 245 children and adolescents who underwent venepuncture in the course of other investigations while attending the hospital between October 1985 and July 1986. All serum samples were tested for rubella antibodies by single radial haemolysis and haemagglutination inhibition tests using standard methods.1 In the microtitre haemagglutination inhibition test pigeon erythrocytes were used to indicate haemagglutination, and all serum samples were pretreated with kaolin. In all cases the presence or absence of antibody was confirmed by both the haemagglutination inhibition test and single radial haemolysis. No gross discrepancies were detected between the results of the two tests.

**Eye injuries caused by directed jets of water from a fire hose**

We report two cases of eye injuries caused by jets of water from a fire hose. Palpebral laceration and rupture of the orbital septum caused by sprinkler jets used in agricultural irrigation have been described,1 but to our knowledge severe intraocular injuries have not been reported.

**Case reports**

Two students were hit in the face by a jet of water from a fire hose at close range during their university's "frat week." The pump pressure was 10 bars (150 h/2/m2), the diameter of the hose 45 mm, and the diameter of the hose nozzle 20 mm. The length of the hose was 75 m, and the jet was directed from less than 5 m.

**CASE 1**

A student presented to the eye department immediately after the accident with reduced vision in both eyes. The visual acuity was 6/36 in the right eye, and there was perception of light in the left. Extensive lid ecchymosis and subconjunctival haemorrhages suggested bilateral retrobulbar haemorrhage. Extraocular movements were full, and there was no proptosis. Papillary reactions to light and accommodation were reduced in both eyes. Slit lamp examination of the left eye showed a 2 mm hyphaema; the pupil was dilated and the iris sphincter ruptured. The right eye showed dispersed blood in the anterior chamber with an oval pupil. The intraocular pressure in the left eye was raised at 26 mm Hg and in the right eye was normal at 18 mm Hg.

The patient was admitted to hospital for bed rest and was treated with...
in intravenous and oral acetazolamide, topical steroids, and mydriatics. Radiographic examination excluded any orbital fracture. Over the next 48 hours blood in the anterior chamber had cleared sufficiently to allow fundal views: there was vitreous haemorrhage in the left eye and bilateral commotio retinae. The visual acuity in the right eye returned to 6/5 within 72 hours, and re-examination showed no abnormality apart from residual traumatic mydriasis. Topical timolol was required to control persistently raised intraocular pressure in the left eye. After four weeks the visual acuity was still reduced at 6/18 with peripapillary subretinal haemorrhage, oedema at the posterior pole, and peripheral pigmentary retinal degeneration.

CASE 2

In this patient the visual acuity was reduced to 6/24 with a 2 mm hyphaema, rupture of the iris sphincter, an unreacting dilated pupil, and inferonasal commotio ocuæ. The right eye was not injured. The left eye was treated with topical mydriatics and steroids, and the visual acuity returned to normal within seven days. No further complications were found at the last follow up examination, but the traumatic mydriasis and associated cycloplegia persisted.

Comment

The damage inflicted by a blow to the eye depends on the kinetic energy of the blow; ocular damage is due to direct impact disrupting the anterior segment and the contra coup effect disrupting the retina. The energy on impact of solid objects such as a cricket ball or fist is dissipated over the cranial vault and facial skeleton as the blow strikes the orbital margin. A anatomical arrangement does not protect against atmospheric and immersion blast injuries or injuries due to high velocity jets of fluid; thus directed water jets can cause serious eye injuries.


(Accepted 25 November 1986)

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Pulsatility of luteinising hormone in men with chronic renal failure: abnormal rather than absent

Loss of libido, impotence, infertility, oligospermia, and low testosterone concentrations are common in men with chronic renal failure treated by intermittent haemodialysis; although the cause remains uncertain, with hypothalamic-pituitary dysfunction and primary gonadal dysfunction have been implicated. Pulsatile release of gonadotrophin releasing hormone is essential for the normal secretion of luteinising hormone. Rodger et al found that despite normal testosterone concentrations only one of six patients receiving intermittent haemodialysis or chronic ambulatory peritoneal dialysis showed luteinising hormone pulsatility between 6 am and 10 am.1

We looked for pulses of luteinising hormone in men receiving intermittent haemodialysis.

Patients, methods, and results

Ten men (mean age 38 years range 20-59) who had been stabilised on intermittent haemodialysis for at least six months were studied on the evening before their next dialysis. All were taking aluminium hydroxide, and some were taking cimetidine and β blockers. All had normal secondary sexual characteristics, and none had a history of hypogonadism before their renal failure. Six healthy men (mean age 33 years range 23-38) served as controls. To assess the pulsatility of luteinising hormone blood was taken without stasis via an indwelling venous cannula at five minute intervals for three hours and then at 15 minute intervals for four hours. A basal sample was collected for estimation of serum testosterone, prolactin, follicle stimulating hormone, and 17β oestriol concentrations.

Luteinising hormone was measured by an immunoradiometric assay (MAIACLONE Serono Diagnostics, Woking) standardised against World Health Organisation 68/40 and with an interbatch coefficient of variation of 5-9% at 7 IU/l and 5-5% at 46 IU/l. Follicle stimulating hormone, prolactin, testosterone, and 17β oestriol concentrations were measured by standard radioimmunoassays with reagents from the Chelsea Hospital for Women, St Bartholomew’s Hospital, St Thomas’s Hospital, and Storanti, St Albans, respectively. A pulse of luteinising hormone was defined as an increment in luteinising hormone concentration from nadir to peak of over 20% in two or more consecutive samples.2 Statistical analyses were performed with Student’s t test for unpaired observations.

<table>
<thead>
<tr>
<th>Mean (SEM) results of hormone assays</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>(nmol/l)</td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td>Patients receiving dialysis</td>
</tr>
<tr>
<td>Controls</td>
</tr>
<tr>
<td>Significance</td>
</tr>
<tr>
<td>Normal range</td>
</tr>
</tbody>
</table>

Seven of the 10 patients and all six controls showed pulsatility of luteinising hormone but the mean number of pulses during the seven hour study was lower in the patients (mean 1-0 (SEM 0-2) vs 3-3 (0-5) pulses, p<0.001). The mean serum testosterone concentration was lower in the patients than in the controls but not significantly so (table). Mean serum prolactin and luteinising hormone concentrations were significantly greater in the patients, but in only one was the prolactin concentration greater than 1000 mIU/l. There was no significant difference in mean serum oestriol or follicle stimulating hormone concentration between the patients and the controls. In three patients, however, serum follicle stimulating hormone concentration was greater than 20 IU/l. There was no significant difference in mean ages between the groups.

Comment

In normal men luteinising hormone is released in pulses, the largest of which have a periodicity of 90-120 minutes.1 Although loss of pulsatility results in hypogonadotrophic hypogonadism with concentrations of testosterone similar to those after castration, Rodger et al reported that five out of six patients receiving dialysis showed no pulsatility of luteinising hormone but had normal serum concentrations of testosterone.1 In contrast, in our study, with the same criteria for pulsatility but more frequent sampling and a more sensitive and precise assay, most of the patients receiving intermittent haemodialysis had pulsatile secretion of luteinising hormone. This might explain the maintenance of normal or borderline low serum total testosterone concentrations found in our study and by others.2 The large pulses were, however, less frequent than those in the normal control subjects, and this might contribute to the reported low serum free testosterone concentration and defects in spermatogenesis during intermittent haemodialysis.1 Pulses of smaller amplitude may also be present, but as these would not be detected by the conventional method of identifying pulses used in this study further investigations are in progress using spectral and periodic regression analysis.

We gratefully acknowledge the financial support of the East Anglian Health Authority.

4 Crowley WF, Filicori M, Spratt DJ, Santoro NF. The physiology of gonadotrophin releasing hormone secretion in men and women. Recent Progr Horm Res 1985;41:473-531.

(Accepted 3 December 1986)