nebulised compared with aerosolised or dry powder β agonists. There was a tendency evident in this study to provide patients with persistent asthma with nebulisers rather than take the time and effort needed to educate the patient adequately about effective management, proper technique of using metered dose inhalers, and self adjustment of oral corticosteroid regimens.

Grant's view that the overuse of domiciliary compressor driven nebulisers to deliver β agonist without supplementary oxygen may have been a main reason for the excess mortality from asthma in New Zealand⁴ was widely publicised. Supervised use of nebulisers in patients who are also treated with other appropriate drugs can often produce substantial improvement,^{14,15} though some patients may obtain equal benefit from use of cheaper and more portable aerosol spacers and chambers. Severe nocturnal asthma and "morning dips" can best be relieved by β agonist. We believe that there is a place for the use of β agonist by nebuliser in the chronic and emergency management of asthma but caution that some patients may regard these devices as more effective than they are in the management of lifethreatening attacks. Patients must be clearly instructed about action to be taken when use of the nebuliser does not bring appreciable relief confirmed with a peak flow meter reading. Such instruction may best be achieved by a written "crisis plan" which includes instructions about when to start or increase corticosteroids as well as when to seek medical help. Asthma may be rapidly fatal and undue delays must be avoided if mortality is to be reduced.

In summary, of 75 patients prescribed domiciliary nebulisers who subsequently died of asthma, at least 16% and possibly up to 30% delayed seeking help because of undue reliance on high dose β agonists. While this accounted for at most only 8% of all deaths from asthma in a two year period and does not of itself explain the high mortality in New Zealand, adequate precautions in prescribing and supervising use of nebulisers should reduce these figures. Prompt

medical attention and use of systemic corticosteroids when bronchodilator treatment is ineffective are essential to the management of severe asthma.

We thank the general practitioners and hospital medical staff and particularly the relatives of patients for their cooperation. The dedicated work of the interviewers who obtained so much detailed information about each patient is gratefully acknowledged. The study was supported by the Medical Research Council of New Zealand, the Maurice and Phyllis Paykel Trust, the New Zealand Asthma Society, and regional asthma societies.

References

- 1 Stainforth JN, Lewis RA, Tattersfield AE. Dosage and delivery of nebulised beta agonists in hospital. *Thorax* 1983;38:751-4.
- 2 Anonymous. The nebuliser epidemic [Editorial]. *Lancet* 1984;ii:789-90.
- 3 Laroche CM, Harries AVK, Newton RCF, Britten MG. Domiciliary nebulisers in asthma: a district survey. *Br Med J* 1985;290:1161-3.
- 4 Grant JWB. Asthma in New Zealand. *Br Med J* 1983;286:374-7.
- 5 Stolley PD, Schinnar R. Association between asthma mortality and isoproterenol aerosols: a review. *Prev Med* 1978;7:519-38.
- 6 Jackson RT, Beaglehole R, Rea HH, Sutherland DC. Mortality from asthma: a new epidemic in New Zealand. *Br Med J* 1982;285:771-4.
- 7 Wilson JD, Sutherland DC, Thomas AC. Has the change to beta-agonists combined with oral theophylline increased cases of fatal asthma? *Lancet* 1981;ii:1235-7.
- 8 Sears MR, Rea HH, Beaglehole R, et al. Asthma mortality in New Zealand: a two year national study. *NZ Med J* 1985;98:271-5.
- 9 Sears MR, Rea HH, de Boer G, et al. Accuracy of certification of deaths due to asthma: a national study. *Am J Epidemiol* 1986;124:1004-11.
- 10 McDonald JB, Seaton A, Williams DA. Asthma deaths in Cardiff 1963-74; 90 deaths outside hospital. *Br Med J* 1976;ii:1493-5.
- 11 Ormerod LP, Stableforth DE. Asthma mortality in Birmingham 1975-7: 53 deaths. *Br Med J* 1980;280:687-90.
- 12 British Thoracic Association. Deaths from asthma in two regions in England. *Br Med J* 1982;285:1251-5.
- 13 Sears MR, Rea HH, Rothwell RPG, et al. Asthma mortality: comparison between New Zealand and England. *Br Med J* 1986;293:1342-5.
- 14 McGivern DV, Ward M, Revill S, Sechiari A, Macfarlane J, Davies D. Home nebulisers in severe chronic asthma. *Br J Dis Chest* 1984;78:376-82.
- 15 Cochrane GM, Prior JG, Rees PJ. Home nebulisers for airflow limitation. *Br Med J* 1985;290:1608-9.

(Accepted 28 November 1986)

SHORT REPORTS

Exceptional virilisation due to ovarian tumour and reversal after resection

We report on a woman who had a virilising tumour and an exceptionally high testosterone concentration whose diagnosis was delayed by 11 years because of inappropriate biochemical tests.

Case report

A woman aged 35 presented with hirsutism, deepening voice, and amenorrhoea one year after the birth of her third child. She was referred to the local hospital, where the concentrations of her urinary 17 ketosteroids were measured. These were normal in most samples and marginally increased in others. An intravenous pyelogram was normal. Air insufflation studies to visualise the adrenal glands were technically unsatisfactory because of her obesity (127 kg). She was then lost to follow up.

Eleven years later she was referred again as her condition had worsened and she had become a recluse. The hirsutism was now so severe that she was shaving daily. She had considerable frontal balding, clitoromegaly, a deep voice, and an appreciable increase in muscle bulk. She was also obese. Serum biochemical tests showed: testosterone 310 nmol/l (normal female range 0.5-2 nmol/l, normal male range 10-30 nmol/l), androstenedione 98 (normal 2-10) nmol/l, dehydroepiandrosterone sulphate 19 (2.2-9.8) μ mol/l, sex hormone binding globulin 31 (16-119) nmol/l, and oestradiol (E2) 1385 (110-180 early follicular phase) pmol/l. Serum gonadotrophins were undetectable, and urinary free cortisol concentrations were normal. Computed axial tomography showed normal adrenal glands but a large mass in the left ovary, which was extending suprapubically but difficult to palpate on abdominal examination because of her considerable obesity. She was made to follow a strict diet and lost 16.8 kg. She then had a laparotomy and was found to have a solid benign ovarian tumour 8 cm in diameter, which was resected completely. The other ovary and the uterus were removed. Histological examination showed a mixed adrenal-Leydig cell tumour with no evidence of malignancy. Ovarian blood taken before operation showed concentrations of testosterone of 2700 nmol/l, androstenedione 1200 nmol/l, dehydroepiandrosterone sulphate 10.5 μ mol/l, and oestradiol 3670 pmol/l.

Her postoperative recovery was dramatic. Within a few days she had started to

shed vast quantities of hair into the bedclothes. Her serum testosterone concentration decreased during two days to 1 nmol/l, and gonadotrophin concentrations increased progressively to the normal postmenopausal range. Two years after surgery she no longer needed to shave, and her scalp hair had grown dramatically (fig). She felt physically and mentally better and regained an interest in knitting and sewing. Her voice rose considerably to its previous pitch and timbre. She noticed no change in sex drive either before or after surgery. She received hormone replacement therapy with yearly implants of 50 mg oestradiol.

Comment

This patient had an unusually high concentration of testosterone for either sex, and we have been unable to find any record of a man or woman achieving

Appearance of patient before and six months after removal of virilising tumour.

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 have been considerably increased at that time.

- 1 Ireland K, Woodruff JD. Masculinising ovarian tumours. *Obstet Gynecol Surv* 1976;31:83-111.
- 2 Farber M, Millan VG, Turksoy RN, Mitchell JR. Diagnostic evaluation of hirsutism in woman by selective bilateral adrenal and ovarian venous catheterisation. *Fertil Steril* 1978;30:283-8.
- 3 Judd HL, McPherson RA, Rakoff JS, Yes SSC. Correlation of the effects of dexamethasone administration on urinary 17 ketosteroid and serum androgen levels in patients with hirsutism. *Am J Obstet Gynecol* 1977;128:408.

(Accepted 9 October 1986)

Departments of Medicine and Dermatology, University of Manchester

H M BUCKLER, BMEDSCI, MRCP, tutor in medicine
D M MITCHELL, MB, MRCP, registrar in dermatology
M H BECK, MB, MRCP, consultant dermatologist
D C ANDERSON, MD, FRCP, professor of endocrinology

Correspondence to: Professor D C Anderson, Department of Medicine, Clinical Sciences Building, Hope Hospital, Salford M6 8HD.

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.

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 (and percentage)

Age:	1-3 months	4-6 months	7-11 months	1-2 years	3-5 years	6-12 years	13-20 years
1985-6	10/12 (83)	1/12 (8)	0/21	3/54 (6)	6/38 (16)	64/84 (76)	18/24 (75)
1969*	14/19 (74)	3/18 (17)	0/19	4/47 (9)	10/41 (24)	32/40 (80)	42/51 (82)

* 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.² Further analysis of the results showed that though only one (11%) of nine children aged 5 years 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 childbearing age and not at interrupting transmission of the virus. Although it has resulted in a decreased proportion of susceptible women of childbearing age, a considerable number remain at risk of contracting rubella in pregnancy.^{3,4} To protect them it has been proposed that the present vaccination programme be augmented by immunising boys and girls in early childhood.⁵ 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.

- 1 Pattison JR, ed. *Laboratory investigation of rubella*. London: HMSO, 1982. (Public Health Laboratory Service Monograph Series No 16.)
- 2 Brown T, Hambling MH, Ansari BM. Rubella-neutralising and haemagglutinin-inhibiting antibodies in children of different ages. *Br Med J* 1969;iv:263-5.
- 3 Hambling MH. Changes in the distribution of rubella antibodies in women of childbearing age during the first eight years of a rubella vaccination programme. *J Infect* 1980;2:341-6.
- 4 Wild NJ, Sheppard S, Smithells RW. The consequences of antenatal rubella testing. *Health Trends* 1986;18:9-10.
- 5 Miller CL, Miller E. Rubella vaccination in the UK: time for a complete strategy. *Lancet* 1985;iii:732.

(Accepted 6 October 1986)

Department of Paediatrics and Child Health, Leeds General Infirmary, Leeds LS2 9NS

N D MUNRO, MRCP, tutor
N J WILD, MRCP, lecturer
S SHEPPARD, MB, CHB, senior research fellow
R W SMITHELLS, MD, FRCP, professor of paediatrics

Department of Virology, Public Health Laboratory, Leeds LS15 7TR

M H HAMBLING, MD, FRCPATH, consultant virologist

Correspondence to: Dr Munro.

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 "rag week." The pump pressure was 10 bars (150 lb/in²), 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. Pupillary 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 28 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