polyps were resected. One child with a bleeding juvenile polyp was also found to have ileal nodular lymphoid hyperplasia, and two children with ulcerative colitis in remission bled from juvenile polyps in the rectum and sigmoid colon.

No complications arising from polypectomy were encountered. New polyps (three in the rectum and one in the sigmoid colon) occurred in three children after a minimum of eight months).

Discussion

This study shows that colorectal polyps may be responsible for a surprisingly large proportion of cases of rectal bleeding in childhood, particularly where the incidence of inflammatory bowel disease is not high.¹⁻⁶ It also confirms the value of total colonoscopy in investigating children with lower bowel bleeding; digital examination, single contrast barium enema, and proctoscopy alone are insufficient. We have abandoned the use of large bowel radiology in our hospital because of the superiority of colonoscopy in detecting polyps. We were unable, however, to compare the diagnostic values of these methods in children.

Bleeding from the lower gastrointestinal tract may have two simultaneous causes in one child: ileal nodular or follicular lymphoid hyperplasia. Colorectal polyps were seen in two patients with quiescent ulcerative colitis. Failure to detect the

polyps could have led to a wrong diagnosis that the inflammatory bowel disease was in relapse.

Histology of resected polyps showed that juvenile polyps predominated, as has been found in previous studies.¹⁻⁶ The finding of a large bowel adenoma in a 2 year old boy is particularly important as such lesions can be premalignant. Thus the removal of all large bowel polyps in children as well as in adults is essential, even in the case of histologically confirmed juvenile polyps.

I thank Professor A S McNeish, Dr I W Booth, and Mr A Picken for constructive help with the manuscript.

References

- 1 Silverman A, Roy CC. *Pediatric clinical gastroenterology*. St Louis: C V Mosby, 1983:453-93.
- 2 Grybosky J, Walker WA. *Gastrointestinal problems in the infant*. Philadelphia: W B Saunders, 1983:86-121.
- 3 Williams CB, Laage NJ, Campbell CA, et al. Total colonoscopy in children. *Arch Dis Child* 1982;57:49-53.
- 4 Mougnot JF, Vargas J. La coloscopie chez l'enfant. *Arch Fr Pediatr* 1983;40:189-96.
- 5 Douglas JR, Campbell CA, Salisbury DM, et al. Colonoscopic polypectomy in children. *Br Med J* 1980;280:1386-7.
- 6 Howdle PD, Littwood JM, Firth J, et al. Routine colonoscopy service. *Arch Dis Child* 1984;59:790-3.

Correspondence to Dr V N Perisic, Institute for Child and Mother Care, Radoja Dakica 6-8, 11070 Novi Beograd, Yugoslavia.

Received 25 September 1986

Hearing loss due to mumps

R HALL AND H RICHARDS

Clwyd Health Authority, Mold and Wrexham, Clwyd, Wales

SUMMARY The possibility of mumps was considered in 33 children with profound unilateral sensorineural hearing loss of unknown origin. Fifteen gave a history of mumps, of whom 12 contracted the infection between the last normal and first abnormal hearing tests. Hearing should be tested after mumps infection.

The epidemiology of mumps in England and Wales is reported to be changing from an epidemic disease of young adults and older children to a more endemic disease affecting younger age groups, with an associated change in the age distribution of general practitioner consultations.¹ Although the

incidence of recognised clinical disease may be falling, mild and symptomless mumps infections are possibly more common. Complications of mumps may arise, irrespective of the severity of infection, and can occur in the absence of parotitis.²

Deafness is a widely recognised complication of mumps, characteristically unilateral and sensorineural in nature, and is thought to result from endolymphatic labyrinthitis.² The hearing loss is usually acquired suddenly, four to five days after the onset of illness, and results in permanent profound hearing impairment. The incidence of this complication has been regarded as rare, of the order of 0.05/1000 cases,³ although unrecognised mumps can cause severe deafness.⁴

This study reports an investigation undertaken in

South Clwyd (with a population of roughly 55 000 aged 16 years or under⁵) to identify children who may have acquired profound unilateral sensorineural hearing loss as a result of mumps, to assess whether audiological assessment is indicated routinely after this infection.

Methods

Over a 12 month period a search was conducted of the audiological records of children aged 16 years or under in South Clwyd to identify known cases of severe (70 db or greater, across the range 250 to 8000 Hz) unilateral sensorineural hearing loss.

Using a prepared proforma, further information was obtained about individual cases from child health and school health records. Dates of the last normal hearing test and the first abnormal test, the degree of impairment, and the opinion of the consultant ear, nose, and throat (ENT) specialist concerning the aetiology of the defect were recorded. Information was sought about the birth and medical history and family history of deafness and if necessary clarified by contacting either the parents or general practitioner.

Results

Thirty six cases of profound unilateral sensorineural hearing loss were identified for whom normal hearing test results had previously been recorded. Three of these cases were excluded from further consideration, as hearing impairment was acquired gradually in one and essential information could not be obtained about the other two. Thirty three cases (19 boys and 14 girls) therefore formed the basis of this study, with acquired hearing loss of profound degree on first ascertainment.

Records confirmed that each child had attended a consultant ENT specialist after the discovery of the hearing defect, previously normal hearing test results being available at that time. The causes of hearing loss proposed in the consultant's letters written subsequently were mumps (three cases), meningitis (three), congenital (11), and inconclusive (16).

The interval between the last normal hearing test recorded and the first abnormal hearing test ranged from four months to six years two months, with a mean interval of two years five months. In 12 cases (36%) more than three years had elapsed between the consecutive normal and abnormal hearing tests. A positive history of mumps was given by 15 of the 33 children, at ages varying from 2 to 10 years (mean 5 years 1 month and median 4 years 3 months), of whom 10 were boys. The date of mumps infection in

Figure Timing of mumps infection in relation to normal and abnormal hearing test results in 15 cases with a positive history of mumps.

Table Proposed aetiology of hearing loss given by the consultant ear, nose, and throat specialist for cases 1 to 15

Case No	Aetiology of hearing loss	Comment
1-3	Mumps	Severe mumps infections reported
4	Meningitis	Profound hearing loss after meningitis
5-7	Congenital	Diagnoses of congenital hearing loss were made despite normal hearing tests previously
8-15	Inconclusive	No definite cause proposed

each of these 15 cases is shown in the Figure in relation to the last date when normal hearing was recorded and the first date when the unilateral hearing loss became apparent. The Table indicates the opinion of the consultant ENT specialist concerning the cause of hearing impairment in cases 1-15.

The temporal relation between mumps infection and loss of hearing suggested mumps as a possible aetiological agent in cases 1-12, although clinically meningitis was the most likely cause in case 4. Mumps was an unlikely cause in cases 13, 14, and 15.

Discussion

Unilateral hearing loss may be overlooked in young children and pass unrecognised without formal hearing testing. It is important to preserve optimum hearing in the unaffected ear and to advise parents to seek early medical advice if ear conditions arise. Conductive hearing defects, which are common in this age group, may result in considerable functional impairment if superimposed on established sensorineural loss.

Screening services for hearing vary considerably between districts. In South Clwyd routine screening

is offered using distraction tests at 7 months and audiometric assessments at school entry and in alternate academic years until school leaving age. This would seem to give comprehensive coverage, but in practice variations arise: some tests are postponed due to illness or absenteeism and children screened at the beginning of one academic year may not be recalled until the end of the academic year when further testing is scheduled, an interval approaching three years. A period of this duration could be of long term consequence for a young child with unrecognised unilateral hearing loss.

Our investigation indicated difficulty in attributing a cause for sensorineural hearing loss in many of the cases identified. In 11 the defect was described as congenital despite available evidence of previously normal hearing and in 16 no conclusion was reached. Delay in ascertainment of hearing impairment, which may have been unsuspected by parents, and the extent of credence given to hearing testing in the community by hospital staff are both factors that might hamper diagnosis.

Temporal evidence suggests that mumps could be considered as a cause in a third of the cases of hearing loss studied. Subclinical or unrecognised mumps may have occurred in other instances. Seven cases contracted mumps between 1 April 1981 and 31 March 1982, in association with a nationally observed periodic rise in incidence.⁶ The possibility that particular strains of mumps virus might be more prone to cause auditory damage requires consideration.

The incidence of hearing loss due to mumps is

unclear as few studies are available, particularly in relation to young children. This investigation concludes that mumps could be considered more widely as an explanation for some cases of unilateral hearing loss and that a formal prospective study with serology is indicated. Meanwhile, the changing epidemiology of mumps to a milder illness of younger children, the fact that permanent hearing impairment can result from subclinical or unrecognised infections, and the importance of unilateral hearing loss in this age group are factors that confirm the need for regular screening of hearing. In addition, routine testing of hearing should be considered after a history of mumps, particularly in districts where a comprehensive screening programme is not available.

References

- Galbraith NS, Pusey JJ, Young SEJ, Crombie DL, Sparks JP. Mumps surveillance in England and Wales 1962-1981. *Lancet* 1984;i:91-4.
- Lindsay JR, Davey PR, Ward PH. Inner ear pathology in deafness due to mumps. *Annals of Otolaryngology* 1960;69:918.
- Vuori M, Lahikainen EA, Pettonen T. Perceptive deafness in connection with mumps. *Acta Otolaryngol (Stockh)* 1962; 55:231-6.
- Fowler ER. Deafness in mumps. *Pediatrics* 1960;77:243-5.
- Jones DT. *Annual statistical report for the year ended 31/12/84*. Mold, Clwyd, Wales: Clwyd Health Authority, 1985.
- Communicable Disease Surveillance Centre. Mumps surveillance 1969-83. *Communicable Disease Report* 1984;No 51.

Correspondence to Dr R Hall, Clwyd Health Authority, Preswylfa, Hendy Road, Mold, Clwyd CH7 1PZ, Wales.

Received 9 September 1986

Meningitis presenting as hypertension

K WATERS AND J GILLIS

Intensive Care Unit, Royal Alexandra Hospital for Children, Sydney, Australia

SUMMARY A 21 month old girl who presented with what seemed to be hypertensive encephalopathy is described. Although her encephalopathy resolved with antihypertensive treatment, subsequent investigations revealed haemophilus meningitis.

We report a case of a child with otherwise uncomplicated meningitis who presented with fever, systemic hypertension, and encephalopathy. The possible pathogenic mechanisms are discussed and the need to consider meningitis in the differential diagnosis of acute hypertension is emphasised.

Case report

A 21 month old girl was referred because of acute systemic hypertension associated with fever and drowsiness. There was no important history. A maternal aunt had had unspecified renal disease. The present admission had been preceded by three days of coryzal symptoms with no systemic illness. On the morning of admission she developed fever associated with deterioration in conscious state. She vomited a single dose of oral antibiotic but had not received antipyretics.

On examination at this time she was drowsy but rousable, with a temperature of 37.5°C (from