Cleft palate and glue ear

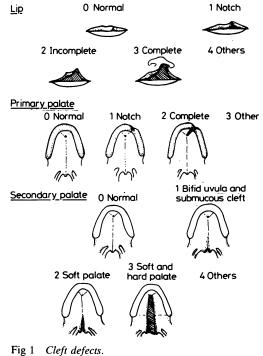
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SUMMARY The invariable presence of otitis media with effusion in children with cleft palate aged from 2 to 20 months is confirmed in a prospective trial, and the diagnosis was made in each case by myringotomy. Treatment planning should take account of this, and long term ventilation of at least one ear seems to be mandatory in order to correct bilateral congenital deafness. Close cooperation between otologist and plastic surgeon is essential for diagnosis and treatment of otitis media in these patients.

The association between hearing loss and a cleft palate is well established.¹⁻⁴ The hearing loss observed in adults with cleft palate is usually conductive and related to scarring, adhesions, and perforations of the tympanic membrane. It is suggested that these symptoms are late sequelae of untreated or unresolved middle ear effusions.⁵⁶ Much recent research centres on otological problems in children and infants with cleft palate. Various incidences of otitis media (OME) have been recorded,^{7 8} however, the precise incidence is not established. This is largely due to methodological differences in the studies concerned, particularly the use of different techniques to assess middle ear function. The effects of OME on hearing, speech, and intellectual development have also been the subject of clinical research.⁹ ¹⁰ Thus at the present time it seems important to establish the incidence of OME in children with cleft palate, and it is important to decide whether early and active treatment of middle ear effusions will prevent or minimise the long term otological and developmental sequelae.

In 1984 a multicentre prospective trial was established at four hospitals with both plastic surgical and ear, nose, and throat units: University College Hospital, London, The Hospitals for Sick Children (Great Ormond Street, and Queen Elizabeth Hospital, Hackney), and the Queen Victoria Hospital, East Grinstead. In this trial myringotomy was performed for all children with cleft palate irrespective of previous otological findings. When OME was confirmed one ear only was ventilated, thus each child provided a ventilated and a non-ventilated (control) ear. The main aim of the trial was to answer these questions: (1) What is the incidence of OME in cleft palate children? (2) What is the effect of palate repair on subsequent eustachian tube function? (3) Are there any advantages in early long term ventilation of the middle ear? This report concentrates on the first of these questions only.



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Patients and methods

Cooperative otological and plastic surgery was performed at each participating hospital to minimise admissions and anaesthetics. Investigations included a formal assessment of cleft defect at presentation (fig 1), otoscopic inspection of tympanic membranes and impedance measurement, otoscopic examination with a microscope, and bilateral myringotomy under anaesthetic. All observations were documented and a database established using a dBASE 11 data management system and an Apple 11E computer.

With parental consent children with cleft palate who were treated in the plastic surgery departments of the above hospitals were registered and entered into the trial, some at birth, others just before plastic surgery.

The child attended the ear, nose, and throat outpatient clinic the day before surgery, for otoscopy and tympanometry. (The type of impedance meter varied: at the Queen Victoria Hospital an American AR85 was available, and at the other three hospitals a Graystad GS1 28 was used.) The next day, under general anaesthetic, both ears were inspected with an operating microscope, and myringotomy and aspiration of middle ear contents by microsuction was performed. With OME confirmed by aspiration a long term ventilation tube (the protocol recommended a Goode 'T' tube) was inserted in one ear at random. For children readmitted for a second examination with a microscope the above procedure was repeated for the non-ventilated (control) ear, and patency of the in situ ventilation tube confirmed.

Otological follow up was maintained to assess the patency of the ventilation tube, the state of the ear undergoing myringotomy alone, and to monitor hearing. Early liaison with a speech therapist ensured subsequent speech and language assessment. The trial protocol is outlined in fig 2.

Results

By July 1986, 112 children had been registered in the study. Fifty five children had undergone examination with a microscope and bilateral myringotomy: 15 at the time of lip repair, 34 at palate repair, and six as a separate procedure (see fig 2). Six children had a second examination with myringotomy of the

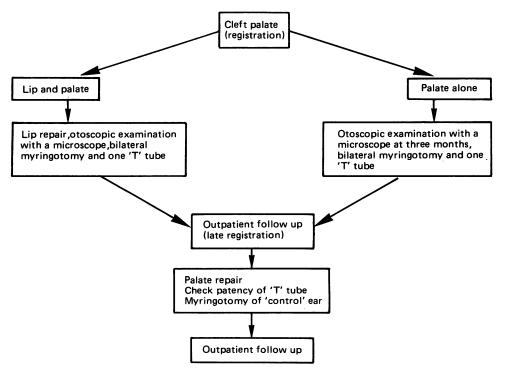


Fig 2 Trial protocol. *With late registration bilateral myringotomy and ventilation of one ear were performed at palate repair.

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Table	Age a	at myringotomy:	61	operations	in	55	children	
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Age in months Nos of children	_	-	5		-		 	 	 	 	

'control' (non-ventilated) ear. Thus observations were recorded for 116 ears (bilaterally at 55 operations and unilaterally at six operations). The table shows the age at myringotomy. Age at surgical repair varied between hospitals and surgeons, but on average lip repair was at 3.3 months (range 2–4 months) and palate repair at 13 months (range 7–17 months).

The diagnostic rate of tympanometry in 51 ears (55 children) was: normal peaked curve, four (7.8%) and flat (type B) curve, 47 (92.2%). Wax occluded the external auditory canal in 42 out the 116 ears, which prevented reliable observation. The small number of ears effectively tested by impedance measurement reflects practical difficulties associated with this technique. Tympanometry accurately predicted findings at myringotomy for 46 out of 51 ears.

On examination with an operating microscope otoscopic findings on 116 ears in 55 children (two observations were made in six ears) were: normal tympanic membrane, five ears (4.3%) and diagnostic appearance of OME, 107 ears (92.2%). Some children with apparently normal tympanic membranes had effusions at myringotomy; for two patients (four ears) the appearance of the tympanic membranes was not documented. When confirmed by myringotomy there was a high incidence of OME in 113 of the 116 ears (97.4%). Three middle ears contained no fluid.

The type of fluid aspirated at myringotomy in the 113 ears with OME was: serous, in 11 ears (9.7%); mucoid, in 23 ears (20.4%); 'glue', in 57 ears (50.4%); and it was not recorded in 22 ears (19.5%). The distribution was similar to that seen in children without a cleft palate who have OME.

Discussion

In this typical sample of children with cleft palate myringotomies were performed irrespective of previous otological findings, and were not limited to those children with symptoms. The age of objective diagnosis of OME by myringotomy, and the 97% incidence reported here, merit careful consideration. Furthermore, had any of the three 'dry' ears contained serous fluid capable of displacement under general anaesthetic the incidence of OME would have been even higher.

Too-Chung has suggested the middle ear is

aerated from birth until 4 months of age in children with cleft palate.¹¹ This claim is not supported by our evidence. In this study no myringotomies were performed before 2 months of age, and therefore the state of the middle ear at birth cannot be inferred. OME was confirmed, however, by myringotomy from 2 months onwards; this supports earlier findings that OME is present in very young children with cleft palate, and as it occurs throughout the observed age range spontaneous resolution seems unlikely.⁸ In addition, Too-Chung's observations relied on tympanometry, which may not be the most appropriate technique for assessment of middle ear function in very small children.

In this study the observation rate for tympanometry was disappointing. For 55 children with cleft palate, only 51 ears were effectively tested with an impedance meter. In our experience wax occluding the external auditory canal, a narrow pliable ear canal, or an uncooperative child all mitigate against successful assessment. In addition impedance meters and technical help were not always available at short notice. Thus despite a 90% diagnostic reliability many children simply could not be tested. By contrast the observation rate for examination with an operating microscope was 100%.

Many authorities have placed great emphasis on impedance testing to confirm or exclude OME in children with cleft palate and a wide range of incidences is reported. Unfortunately many of these series are retrospective and the reliability of this technique cannot be confirmed. Although a dedicated impedance meter and technical help in each plastic surgery unit would have improved the observation rate achieved in this study, for the reasons listed above, we conclude that accurate diagnosis of OME must rely on otoscopy followed by myringotomy in all children with cleft palate.

The advantages and disadvantages of inserting grommets to treat OME in normal children is currently a matter of heated debate.¹² ¹³ The precise aetiology of OME remains uncertain although eustachian tube function is strongly implicated in children with cleft palate.¹⁴ Medical treatment of persisting OME is disappointing. In consequence reliable restoration of hearing by surgical intervention and insertion of grommets is favoured and accounts for this procedure being the most common paediatric operation performed in both the United Kingdom and North America.

In children with a cleft palate the clinical picture of OME from birth or shortly before is very different from that found in children without a cleft palate where the peak incidence is between 3 and 6 years of age. For the former group the onset of OME and associated hearing loss preceeds language acquisition and early speech development. Furthermore the condition seems to persist throughout infancy and early childhood. Thus in addition to risks of long term otological sequelae of OME, the untreated child with cleft palate may also suffer language, intellectual, and emotional disability as a consequence of both early hearing loss and original deformity.⁹ Adenoidectomy can play no part in treatment of children with cleft palate for fear of further impairing velopharyngeal competence or exaggerating rhinolalia aperta, and it is also hazardous in very young children. Active treatment of deafness must therefore include ventilation of the middle ear on one or both sides, but should logically only follow accurate diagnosis.

If parents accept the need for surgical diagnosis and treatment of OME this should not require separate anaesthesia because it can be linked to surgical repair of the lip or palate, or both, if close cooperation between surgeons is achieved. Symptomatic OME necessitating myringotomy before or after plastic surgery may need a further anaesthetic, but in our experience the number of children requiring this will be small.

The Shepard grommet may not be the most suitable device for maintaining middle ear aeration in children with cleft palate because it will be extruded on average at 6.6 months,¹⁵ and recurrent OME will necessitate repeated anaesthetics. The Goode T tube, on the other hand, has been shown to remain in situ for substantially longer periods without causing additional tympanic membrane or middle ear complications.¹⁶ Long term ventilation with this type of tube should reduce the incidence of the late sequelae of OME including adhesive otitis, ossicular damage, and cholesteatoma, all of which are particularly evident in untreated children with cleft palate.³ It is hoped that long term follow up in this trial will yield additional information on this matter.

The members of the Cleft Palate Study are: plastic surgeons: JE Bowen, IW Broomhead, TD Cochrane, BM Jones, BD Morgan, CC Walker; otologists: CM Bailey, JM Graham, CM Milton, RJ Sergeant; and an audiologist: SC Bellman. The authors kindly thank them for their enthusiastic participation, and their advice in preparing this report. Figures were prepared by the Department of Clinical Illustration, Queen Victoria Hospital, East Grinstead.

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