

Secretory otitis media and grommets

Secretory otitis media is seen mainly in children, varying in prevalence with season, geographical location, and the age of the patient. Much time and effort are spent in recognising and treating the complaint, and especially in inserting countless grommets into eardrums. In one study covering a group of 278 2-year-old children,¹ secretory otitis media was suspected in nearly 11% of cases on the basis of the otoscopic findings and the flat compliance graph of tympanometry. This high figure is probably realistic in view of the close correlation between the results of tympanometry and the subsequent finding of fluid in the middle ear at myringotomy.²⁻⁴

Loss of hearing due to secretory otitis media may be responsible for educational retardation and psychological and social problems; the disorder may also lead to more serious chronic inflammatory middle ear disease,⁵ and some authors believe that it may cause permanent sensorineural hearing loss.⁶ The desirability of treating the condition has been emphasised by all; notwithstanding the observation of spontaneous resolution in some cases,⁷ almost all ear, nose, and throat surgeons advise active treatment, and in the main this has meant the insertion of grommets into the eardrum.

Without doubt, the placing of grommets (otherwise known as ventilation or tympanostomy tubes) is effective in restoring hearing. In a study which included an eight-year follow-up, the threshold for speech reception was 20 decibels or less in 97% of patients fitted with grommets.⁸ The same study showed, however, that some patients still had some abnormal findings on tympanometric assessment in spite of their improved hearing, and complications included recurrent secretory otitis media, perforation of the drum, and cholesteatoma. The proportion of patients having unsatisfactory hearing after insertion of grommets has been as high as 21% in some series,⁹ with complication rates including adhesive otitis (11%) and cholesteatoma (6%).

Tympanosclerotic plaques are commonly seen in the drum after the use of grommets,⁵ and a thin lax atrophic membrane is found in some ears.⁸ Secretory otitis media often recurs,¹⁰ and all too often several insertions of grommets may be needed. A further complication is acute otitis media, which will cause the escape of a purulent discharge through the grommet into the auditory canal, sometimes making the removal of the grommet essential.^{11 12}

With all these drawbacks, how convincing is the case for insertion of grommets as a first-line treatment? A recent

investigation¹³ has challenged the concept of their use as a definitive method of treatment for glue ear. In a group of 55 patients suffering from bilateral secretory otitis media, one ear was treated with a grommet and the other managed conservatively. Within the first six months the gain in hearing was better on the side of the grommet, but after six months there was little to choose between the two sides. Secretory otitis may be self-limiting—a view that has been argued before.¹⁴⁻¹⁶

More recently¹⁷ a double-blind trial compared the effects of co-trimoxazole given over three weeks with an anti-histamine/decongestive preparation. Resolution occurred in 60% of the patients treated with co-trimoxazole compared with 30% in the other group. Such a result lends weight to the argument that secretory otitis media may be partly infective in aetiology, despite the negative results of bacteriological cultures in most patients.¹⁸

Certainly a more conservative approach may be warranted on recent evidence. Probably co-trimoxazole offers a reasonable compromise as a first-line treatment (though it may not prevent recurrences). Those patients who do not improve after treatment with antibacterial drugs may then have grommets inserted.

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⁶ Ganzer U, Arnold W. Das innenohrhörvermögen beim chronischen seromukotympanon. *Laryngol Rhinol Otol (Stuttg)* 1977;**56**:850-9.

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⁸ Tos M, Poulsen G. Secretory otitis media. Late results of treatment with grommets. *Arch Otolaryngol* 1976;**102**:672-5.

⁹ Gundersen T, Tonning F-M. Ventilating tubes in the middle ear; long term observations. *Arch Otolaryngol* 1976;**102**:198-9.

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¹¹ Birck HG, Mravec JJ. Myringostomy for middle ear effusions. Results of a two-year study. *Ann Otol Rhinol Laryngol* 1976;**85**, suppl 25: 263-7.

¹² Reck R. A rare complication of use of the middle ear ventilation tube (PVC). *HNO* 1977;**25**:114-5.

¹³ Brown MJKM, Richards SH, Ambegaokar AG. Grommets and glue ear: a five-year follow up of a controlled trial. *J R Soc Med* 1978;**71**:353-6.

- ¹⁴ Lildholdt T. Unilateral grommet insertion and adenoidectomy in bilateral secretory otitis media: preliminary report of the results in 91 children. *Clin Otolaryngol* 1979;**4**:87-93.
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- ¹⁶ Willis R. The natural history of secretory otitis media. *Journal of the Otolaryngological Society of Australia* 1977;**4**:123-6.
- ¹⁷ Marks NJ, Mills RP, Shaheen OH. A controlled trial of co-trimoxazole therapy in serous otitis media. (In press.)
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Management of alcohol withdrawal symptoms

The current epidemic of alcoholism shows no sign of abating, and with 20-30% of hospital patients currently thought to be excessive drinkers^{1 2} doctors are likely to have to deal increasingly with the alcohol withdrawal syndrome.

Dependent drinkers react in different ways to the sudden withdrawal of alcohol, so that even if an individual's drinking habits are known his response may be difficult to predict. Some people seem to be scarcely affected, while at the other end of the scale a few, put at less than 5% of the total, develop the dramatic features of delirium tremens, especially if they are also stressed by infection, injury, or operation. Most display some features of anxiety, agitation, tremor, and sweating, accompanied by fever, nausea, and retching. The general hyperactivity is often associated with varying degrees of insomnia, itching, cramps, hyperacusis and tinnitus, and perceptual disturbances; and both tachycardia and hypertension are common. Confusion, paranoia, visual hallucinations, and convulsions occur in the more severely affected patients. Gross and his colleagues³ have devised a method of grading withdrawal symptoms by a series of scales, which can be used for comparative studies and for assessing different treatments.

Symptoms begin six to eight hours after the last drink (which explains why the chronic alcoholic steadies the nerves with the early morning drink), reach a peak at about 48 hours, and then subside over the course of a week. Their cause is not known. Increased secretion of sympathomimetic⁴ and other amines has been suggested to explain the hyperactivity, and though the adrenal cortex is overactive (as shown by raised plasma concentrations of cortisol) the response to stress seems to be inadequate.⁵ The dehydration and oliguria found in the early phase of withdrawal possibly result from the chronic diuresis caused by alcohol or from increased secretion of antidiuretic hormone. Hypokalaemia may be prominent in severer forms of withdrawal.⁶

Management requires sympathetic handling and careful observation. If possible patients should be treated in a general medical ward, even though they tend to be disruptive. The cause of the syndrome should be established, infections such as pneumonia being the most common, and treated vigorously. If necessary an intravenous drip should be set up to combat dehydration and to facilitate the giving of sedatives. For the first day or two additional supplements of 50-100 mmol potassium should be given in divided doses, and injections of a high-potency vitamin preparation are said to be beneficial. Over 100 drugs have been tried in the treatment of withdrawal symptoms,⁷ and the choice is often dictated by personal preference. The current favourite in Britain is chlormethiazole⁸ in a dose of 500-1500 g every six hours initially; the aim should

be to reduce the dose as symptoms disappear and to stop the drug as soon as possible to avoid dependence. Chlordiazepoxide is a useful alternative and may need to be given in large doses at first—for example, 25-50 mg every six hours; cumulative effects can be avoided by reducing the total amount by a quarter each day. Both drugs will suppress anxiety and prevent convulsions. If convulsions do occur chlordiazepoxide is the drug of choice; phenytoin does not act quickly enough. Beta-blocking agents, such as propranolol 40 mg six-hourly, have been used to treat the hyperactivity, but they will not prevent convulsions and do not augment the action of sedatives.⁹

Since the symptoms arise because of the sudden withdrawal of alcohol, treatment is sometimes advocated with small quantities of alcohol. The equivalent of about 8 g absolute alcohol, (half a pint of beer or a glass of spirits) can be given every four to six hours and gradually tailed off; it has the advantage of being safer than drugs and allows the latter to be used in smaller quantities. Alcohol is also a valuable preventive agent in known alcoholics who are undergoing an operation, for example; and it can be tried as a first step in the patient who unexpectedly becomes confused or delirious while in hospital.

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Multiple sclerosis in the Orkney and Shetland Islands and in north-east Scotland

Sutherland reported that multiple sclerosis in 1954 appeared to be more common in the Orkney and Shetland Islands and in Caithness in the north-east of Scotland than in the west of Scotland, and postulated that there might be a disadvantageous genetic factor responsible for this high prevalence.¹ Since then three further studies of the prevalence of multiple sclerosis, based on prevalence days in 1962, 1970, and 1974, have been undertaken in the Orkney and Shetland Islands, the last two by Poskanzer and his colleagues.²⁻⁹ The population of each group of islands had varied slightly between 17 000 and 20 000 people; and the prevalence of ascertained cases of probable multiple sclerosis increased during 1954-74 from 82 to 258 per 100 000 (from 17 to 45 patients) in the Orkney Islands and from 118 to 152 per 100 000 (from 22 to 28 patients) in the Shetland Islands. Other studies have also shown that repeated