and the responses are objective. It has been used in school screening programmes in the United Kingdom and the results reported by Brooks.[2] Extensive use in Scandinavia, and my own experience, confirms the value of the instrument, the use of which obviates the need for a diagnostic, as opposed to a therapeutic, myringotomy.

The advice to prescribe a hearing-aid in cases where repeated myringotomy is required is good only as long as the patient is carefully followed. In the presence of chronic glue ear permanent damage may occur to the tympanic membrane, particularly in its posterior segment, which becomes thin, loses its middle layer, and becomes flabby. This may eventually be indriven into the sinus tympani and to the mastoid air cells, forming a cholesteatoma. Indeed, this is one of the commoner pathogenetic mechanisms of a cholesteatoma at present. Only a small proportion of children are so involved, but any child with an abnormally thin tympanic membrane should be vigorously treated and observed until complete resolution occurs. Even though this may take several years, it is not safe to discharge these children with a hearing-aid.—I am, etc.,

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1 Eagles, E. L., Wishik, S. M., and Doerfler, L. G., *Hearing Sensitivity and Related Factors in Children*, St. Louis, Mo., The Laryngoscope, 1963.
2 Brooks, D. N., *International Audiology*, 1968, **7**, 280.
3 Thomas, R., *Journal of Laryngoloy and Otology*, 1967, **81**, 1071.

SIR,—Having read your leading article on "Glue Ear" (6 December, p. 578), I am frankly amazed at the last paragraph, which must be absolute medical heresy. I am quite sure that every otolaryngologist would continue to insert Teflon tubes as long as they are required. If this is not done, a grave risk of chronic adhesive otitis is incurred, with possible or probable permanent deafness. To even suggest a hearing-aid for a form of deafness that is remediable by a simple surgical procedure is absolutely absurd, apart from the just-mentioned possible sequelae.

The exact cause of chronic seromucinous otitis is certainly not known, but many of these cases are certainly cured by an adequate adenoidectomy. I would certainly question your statement "nor has the removal of the adenoids been shown to prevent or mitigate glue ear." I think the possible allergic aetiology of this condition has been somewhat overemphasized, but a possible focus of infection in the paranasal sinuses should always be sought. I agree with your statement that the tendency to this type of otitis seems to decrease as these children grow older, and the long-term outlook for their hearing, in my experience, is very good as long as the middle ears are kept normal by artificially aerating them with some form of Teflon tube. It is in the adult patient that permanent malfunction of the Eustachian tube can be a problem, and in these cases I have no hesitation in keeping these Teflon tubes in place permanently. This is a small price to pay for normal hearing, and the patient can lead a perfectly normal life apart from his inability to swim. It has to be admitted, however, that these Teflon tubes are not curing the basic malfunction of the Eustachian tube, but neither does an artificial stapes have any effect on the underlying pathology of otosclerosis. I really feel that the final paragraph of your leading article is appalling, and I am very worried that some general practitioners and paediatricians might accept your advice and fail to refer these patients to an otolaryngologist for the appropriate therapy.

One final point—a pneumatic otoscope is essential for the diagnosis of these cases, and any general practitioner can be taught to use it in a matter of minutes.—I am, etc.,

JOHN KEOHANE.

Edmonton,

SIR,—I read Mr. F. Bauer's letter on this subject (10 January, p. 111) with great interest, and I agree with him that there is some value in using mucolytics and considerable value in attempting to inflate the middle ear by Valsalva's procedure or by others methods. There is indeed uncertainty about all forms of non-inflammatory otitis media because they are often difficult conditions to treat, but, having investigated Eustachian tubal function in a large number of children and adults during the past five years, I have found that one of the most constant factors is that they all show some degree of tubal malfunction.

The Eustachian tube is not a simple pipe; the membranous tube is a physiological mechanism with muscular control which must open actively to allow air to pass up to the middle ear. Its anatomy changes throughout childhood, becoming more efficient with age. The cartilage changes in shape and position, the two important muscles alter in their relationship to the tube, and the mucous glands diminish in number and size during childhood. These anatomical facts account in part for the age incidence of glue ear and serous otitis, conditions which I have found not to be so distinct as has been implied. I am sure that tubal malfunction is only one factor in their development, but in my opinion your leading article (6 December, p. 578) understated the importance of the tube rather than the reverse.

In many cases the adenoids are only of moderate size. I do not think that size is very significant; small infected adenoids may cause lymphatic obstruction leading to swelling in the peritubal tissues, thus making it more difficult for the relatively inefficient childhood tube to open. I have found that adenoidectomy in these cases is of real value. If we look on the Eustachian tube as a passive pipe in good order, provided that air can be forced through and provided that adenoids are not actually blocking the nasopharyngeal opening like a cork in a bottle, then treatment with grommets may seem unreasonable. If, however, we accept that tubal malfunction is an important cause of these conditions, then grommets are a logical and relatively harmless method of allowing the equalization of middle-ear air pressure acting as temporary artificial Eustachian tubes and allowing time for the real tube to resume its normal activity.

Like Mr. Bauer, I hope that very few of these children would have to be considered for hearing-aids.—I am, etc.,

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Treating Preinfected Wounds

SIR,—With reference to the letter of Mr. J. W. S. Rickett and Mr. B. T. Jackson, (3 January, p. 48) we would like to ensure that no misunderstanding arises from the quotation of our paper on the same subject.

We agree completely with the criticism in your correspondence (15 November, p. 428, 22 November, p. 493) of their double blind trial using lactose as a placebo which was associated with an infection rate of 26% (5 cases from 19) where the appendix removed was normal. In our trial in the cases where the appendix removed was normal there was no wound infection (judged on the same criteria for sepsis), whether ampicillin was instilled into the wound or not. Because the presence of lactose in a pre-infected wound may cause an increase in the incidence of infection, we decided at the outset against a double blind procedure, and these results appear to justify our decision.

Although our results were analyzed in rather more detail than the above mentioned paper, our conclusions were broadly similar if a little more specific. Our paper is to be published in the *British Journal of Clinical Practice* in the near future.—We are, etc.,

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Vagotomy for Peptic Ulcer

SIR,—Vagotomy for peptic ulcer is becoming increasingly popular but there is an appallingly high incidence of incomplete vagotomy,[1 2] which means failure to cure the ulcer diathesis. In his contribution to this problem. Lee[3] has developed a stain (leucomethylene blue) which is claimed to aid identification of smaller vagal fibres by staining them selectively at operation. We have been using this stain recently and shall publish full details later. Meanwhile, our findings are disappointing. We feel it is important to report the main results because the stain is conceptually attractive but it does not seem to work.

After dividing the obvious nerve trunks at operation, we applied the stain and removed stained structures for histological examination. In a consecutive series of 30 structures from 10 patients, 21 contained no nerve tissue at all, one contained an appreciable amount of nerve tissue, and the remainder contained connective tissue with an occasional isolated nerve fibre so minute as to be invisible to the naked eye.

We therefore found that the stain did not pick out nerve fibres selectively. In these