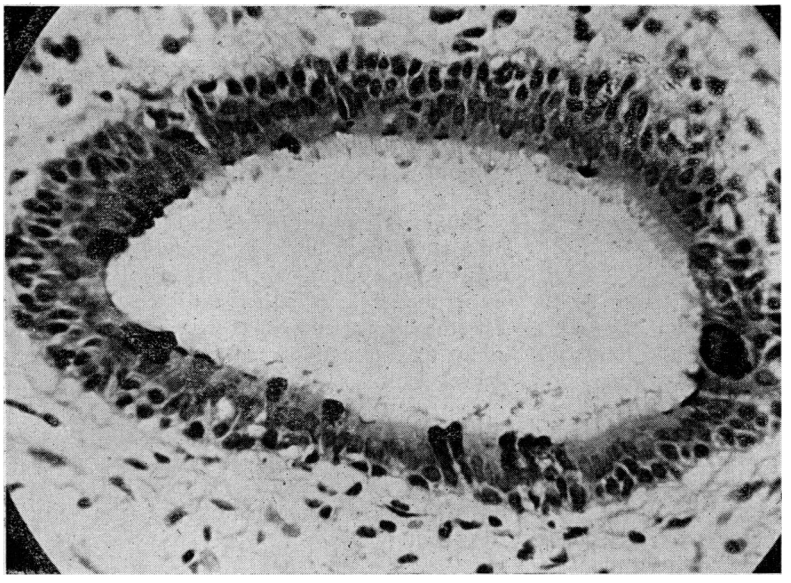

Fig 8 *Lining of mastoid cell at higher magnification showing ciliated columnar epithelium with numerous goblet cells.* ×330

discharge might, therefore, bring about a similar response in the altered mucosa of the middle ear cleft with all its consequences.

There are of course other contributory causes. Chronic otitis media may be incriminated. Moreover, cases of acute suppuratory otitis media, inadequately treated, have been adding to the increasing numbers of 'secretory (mucoid) otitis media'. The role of the secretory epithelium of the eustachian tube must not be underestimated.

Further investigations are required and our own investigations are going to be intensified to answer some of the remaining problems. It seems reasonable to conclude at this stage that the mucosal changes here described form an essential and important histopathological factor in the pathogenesis of mucoid secretory otitis media.

Acknowledgments: Thanks are due to the Surgical and Theatre Staff of the Royal National Throat, Nose and Ear Hospital for their helpful co-operation, to Dr J V Dadswell, Lecturer in Clinical Pathology, for assistance with this paper, and to Mr J Conolly for the photomicrographs.

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Mr B S Carter
(*North Staffordshire Royal Infirmary, Stoke-on-Trent*)

Secretory Otitis as a Cause of Conduction Deafness in Children

My interest in secretory otitis as a cause of conduction deafness in children dates from a chance finding just two years ago. At that time, secretory otitis was regarded as a condition occurring not very frequently in adults, with a characteristic history and a characteristic appearance of the drum, and often affecting one ear only. Myringotomy and inflation would produce clear yellow fluid with immediate improvement in the hearing, but this treatment often had to be repeated. At the same time, deafness in children, due to eustachian tube dysfunction, was recognized as a common indication for removal of tonsils and adenoids. In some 10% of cases, the deafness was not completely cured by this operation, and these were treated at first by the application of radium to the nasopharynx, and later by careful dissection of adenoids, especially in the lateral recesses of the post-nasal space.

Early in 1961, impacted wax was being removed from the ear canal of a child, under general anæsthesia preparatory to removal of tonsils and adenoids for deafness, and the drum, seen through the operating microscope, appeared opaque and full at the back though with no evidence of

inflammation. A small myringotomy opening was made and suction produced thick sticky mucopus; the appearance of the drum returned to normal, and subsequently the deafness was cured. It was then decided to carry out routine inspection of the ear drums, with myringotomy and suction, in a consecutive series of 100 children seen with conduction deafness, excluding only those with a patent perforation of the drum, and to record the results.

The ages of the children ranged from 3½ to 13 years, the average being 7 years, and they were referred equally by the family doctor and the school health service, either with a definite complaint of deafness or as candidates for removal of tonsils and adenoids. On questioning, the parents of 89 children admitted that deafness had been noted at home for periods as long as four years, yet often they had done little about it. One frequently heard such expressions as, 'I thought he was putting it on' and 'He can hear when he wants to'. In 34 children there was a past history of one or more attacks of otorrhœa, and in 43 children of attacks of earache without otorrhœa: in addition, healed perforations of the ear drum were found in 8 children where a history of previous otorrhœa was denied, so that over 80% had evidence of previous otitis media. Twenty-six children had been operated on previously for removal of tonsils and adenoids, and one for adenoids only: at re-operation, 8 of these had marked residual adenoids requiring removal. The hearing loss shown by pure tone audiometry was most commonly in the 20–50 dB range.

At operation, 80 ear drums out of 200 were found to be opaque and white with posterior fullness. Forty-four drums showed healed perforations, and in these the thin scar was sometimes depressed and sometimes bulging outwards with a crinkled appearance like crepe paper. Forty drums were retracted and hyperæmic, usually with a leash of blood vessels behind the handle of the malleus. Only 8 drums showed the blue or amber 'oily' appearance of adult secretory otitis.

Myringotomy was performed on 185 ear drums, a small incision in the postero-inferior quadrant. Only occasionally did this produce a gush of fluid, but often pulsation of the myringotomy opening was seen, suggesting a release of tension. The fine end of the Zoellner's sucker was introduced into the middle ear and would remove the thin fluids, but for the thicker fluids it was found necessary to use the fine end to bring the exudate into the hole, and then remove it by applying the wide bore of the sucker (minus the fine tip) to the surface of the drum. This technique often had to be repeated several times before the middle ear was cleared. On a few occasions, the wide bore sucker was passed through the drum into the middle ear, and though this made a big hole, the drum always healed up without trouble.

Fluid was found in 147 out of 185 ears examined, or, in terms of children, in 92 out of 100. A serous effusion was found in only 18 ears; all the rest were mucoid fluids, either translucent or opaque. The thicker varieties of these were almost semi-solid, and resembled calves-foot jelly or condensed milk.

Of the children who had not had previous operations, tonsils and adenoids were removed in 52, and the adenoids only in 23. There were no immediate complications of the surgical treatment.

All the cases have been followed-up for periods of one to two years, and in 85 no further treatment has been considered necessary. After a period of improvement, 15 children were found to have become deaf again, but 10 of these seemed quite satisfactory after a second myringotomy. The 5 refractory cases have been in hospital for treatment on 18 occasions between them, and 2 children from this group I would like to mention in more detail:

Case 1
Girl aged 3½ years with thick mucopus in both ears. At the third myringotomy the fluid from the right ear was more definitely purulent, so her mastoid was opened and found to contain mucopus and softened bone: since then she has had no further trouble.

Case 2
Boy aged 7 years with a dark blue drum and an amber coloured serous effusion in one ear only, the other side being quite normal. The fluid reaccumulated so quickly after three myringotomies that drainage of the mastoid was considered advisable, and at operation, dark fluid was present in the normal-looking cell system. However, he was still not cured, so at the fifth operation a polythene tube was inserted through the inferior margin of the drum. This had extruded itself at examination two months later, and so far he has had no further recurrence and his hearing remains good.

Certainly in Stoke-on-Trent this condition of seromucinous fluid behind an intact ear drum is a common one. In the twenty-two months since the first case in March 1961, a total of 223 children have been treated.

Obviously the main question that arose was why this common cause of deafness had not been

recognized before 1961, and the only possible answer seems to be ignorance of the condition. Although secretory otitis is mentioned in current textbooks of otology, there is nothing to suggest that it occurs in children to the extent found here. Even after a search of the literature, in this country only papers by Bateman (1957), Colman (1958) and Stevens (1958) were found.

However, in the American literature there are many articles, several of them dealing mainly with secretory otitis in children, but nearly all of these date from after 1940. Yet by 1950 Hoople considered that secretory otitis was found in 3–4% of all E.N.T. patients, and described it as a challenge to otolaryngology. Suehs (1952) and Armstrong (1957) reported similar findings. Various reasons have been suggested for the apparent increase of cases, including allergy, antibiotics, hypometabolism, &c., but the most important has probably been a greater awareness of the condition. The editor of the *American Journal of Diseases of Children* added as a postscript to an article by Freeman & Freeman (1960) on secretory otitis in children: 'This represents an ear, nose and throat point of view of which many pædiatricians may be unaware.'

Certainly during the latter part of the nineteenth century, it was evidently well known, and the first English edition of Politzer's textbook (1883) has a full description and recommends myringotomy. In this country, Hinton (1874) gives an account of secretory otitis in a doctor's son where, after myringotomy, he 'drew out a string of viscid mucus which extended, without giving way, an inch beyond the orifice of the meatus'. Even earlier, Wathen (1755) mentions inspissated mucus in the middle ear as a cause of deafness, and cured patients by syringing the eustachian tubes through a nasal catheter with a solution of honey of roses in warm water.

Yet in 1908 Macleod Yearsley, in the chapter in his textbook on catarrhal inflammation of the middle ear, said that cases with exudate were rare in this country though common in America: he had only seen 10 cases out of 5,000 aural conditions. Gray (1910), discussing catarrhal exudate in the middle ear, advises strongly against myringotomy as suppuration will almost certainly ensue.

The place of the school medical service: In Stoke-on-Trent we are fortunate in having, since 1960, an audiology clinic run by one of the school medical officers, Dr K J Roberts, who has been trained in Professor Ewing's department in Manchester. He does much of the follow-up testing for me, for which I am very grateful, and we meet regularly to see new children as candidates for surgical treatment.

Results of treatment on hearing: Only 4 out of 100 were unchanged, 3 of them being cases of extensive fibrosis after chronic suppurative otitis media and one probably juvenile otosclerosis. Seventy-two obtained normal hearing, which was defined as no loss exceeding 20 dB at any frequency in either ear, and it was particularly pleasing that 9 of these showed healed perforations. The remaining 24 were all improved and, even though they did not reach the normal level, it was in this group that the social benefit was outstanding. The level at which speech in a free field was heard improved in several cases from 70–80 dB to 30–40 dB; the schools reported better educational progress, and one girl even became top in her class examination. Five children had previously worn hearing aids and were able to manage without them. The treatment of these children has been most satisfying work.

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Mr K G Malcomson (*Bristol*) said that secretory otitis in the adult was almost a separate problem and he would confine his remarks to the condition in children; in them the *incidence* was difficult to assess accurately and there was no doubt a geographical variation. In 1957–8, in a series of 522 children in Bristol admitted for removal of tonsils and/or adenoids, he had found deafness due to effusion (all forms) in 55 (10%); 20% had been quoted by some writers. The problem was therefore of some importance.

Early diagnosis was delayed because the condition was often *painless* (62% in the above series) and when earache occurred it was often transient and did not alarm the parents; moreover suspicion that their child might be hard of hearing took root slowly in the minds of the majority of parents.

Audiometric finding of a conductive deafness was usual, and this might vary from week to

week; it was clinically frustrating that the degree of hearing impairment could not be closely correlated with the drum changes, which might vary in the same ear independently of the hearing loss.

The management of the condition was now fairly well established and was successful in the majority of patients. In a few mucus recurred or persisted notwithstanding repeated paracenteses; in such children the viscosity of the mucus was usually considerable and it might have to be pulled out of the ear if suction failed. When the eustachian tube was inflated via the external auditory canal it was invariably patent.

For a time he had carried out cortical mastoidectomy in these resistant cases. In 8 such children the mastoids had been either acellular or of the small cell type, with thickened mucosa; in one of these a small solitary cholesteatoma was found in the antrum. Despite mastoidectomy tenacious mucus continued to cause deafness in 6.

On the basis that the condition might be a secretomotor disturbance he had divided the tympanic branch of the glossopharyngeal nerve in 4 children. Three had been previously treated by multiple paracenteses and cortical mastoidectomy; after Jacobson neurectomy 2 children had no further deafness and the other 2, in addition to viscid mucus, had dense peri-ossicular adhesions which had clearly been responsible for persistent hearing loss.

Observation of resistant cases for over five years had shown the development of dense interstitial fibrosis and calcification of the drum in some children (unrelated to the sites of paracentesis) and also, he suspected, tympanosclerosis; indeed, organization of retained masses of homogeneous mucus might be the foundation for the latter condition. Moreover, tympanotomy had yielded disquieting evidence of the mucosal reaction in children with very tenacious mucus – sheets and bands of fibrous tissue particularly around the ossicles.

When adults presented with adhesive and tympanosclerotic deafness, and had no history of ear trouble in childhood, the above findings should be sufficient to arouse the most complacent in the battle against painless acquired deafness in children.

Mr William McKenzie (*London*) agreed with Mr Carter in his conclusion that middle ear deafness in children was due to fluid, but he questioned the wisdom of a routine myringotomy at the time of the removal of the adenoids and tonsils. Mr McKenzie had often been surprised by the improvement in hearing after simple removal of the adenoids, however small they were, and the same applied to adenoid remnants, in London at all events.

A small point in diagnosis of fluid in the middle ear was that the Zeiss microscope was usually set with the patient lying on a couch, and the fluid level could not, of course, be seen. In this position, however, a few air bubbles might be seen in the posterosuperior quadrant while the remainder of the drum showed the familiar brown oily colour.

Mr H Zalin (*Liverpool*) said that Dr Senturia had suggested, on the basis of his animal experiments in which he cauterized the nasopharyngeal tubal orifice, that the essential mechanics of secretory otitis media involved an ascending infection of the middle ear via the lumen of the eustachian tube, and that there was little or no evidence of tubal obstruction at any stage. It seemed that the diagnosis of tubal obstruction was by no means easy, and Mr Zalin felt that Dr Senturia had by no means proved his point there. A tube which could be inflated was not necessarily one which opened physiologically during swallowing. The inflation test was artificial and unphysiological. His own conception was of a combination of tubal infection and obstruction, the former being largely responsible for the latter. Without tubal obstruction it became impossible to explain the late sequelæ of middle ear exudate, in particular collapse of the tympanic membrane and suppressed pneumatization of the mastoid. Furthermore, if transluminal tubal ascending infection was the sole factor, surely Dr Senturia's therapeutic technique of continuous tubal inflation was irrational and hazardous. The fact that it did seem to help suggested that there were other factors present, in particular tubal obstruction.

The condition of exudative otitis media was misnamed in so far as it implied a middle ear lesion only. It was in fact a tubotympanomastoiditis and, where cholesterol granuloma formed in the mastoid, a careful cortical mastoidectomy had been found of value in obtaining resolution.

Antibiotics seemed to be of little value in these middle ear exudates and this he attributed to the infection being, in the main, viral in origin.

There was no shortage of upper respiratory tract viruses in the nasopharynx and he was thinking mainly of the adenoviruses.

Finally, Mr Zalin believed that the seeds of chronic suppurative otitis media were germinating during the active stage of exudative otitis media when the middle ear cleft was being devitalized and its normal growth and development frustrated.

Mr D H Craig (*Belfast*) said that attention should be confined to the variety of case characterized by a very viscid exudate. It was quite extraordinary that it was only in the last few years that the condition had begun to be recognized. It was not uncommon for a disease to change its natural history over the years; the waxing and waning of scarlet fever was a case in point. It was interesting to hear from Mr Carter that these cases had apparently been described about fifty years ago.

He was inclined to think that the liberal and irrational use of antibiotics might be a possible explanation of why these cases were beginning to be found now. Certainly he had been impressed by the number of times the condition had occurred in children whose repeated upper respiratory infections had received ten, fifteen or even twenty courses of some antibiotic or other.

He found it a most difficult condition to diagnose. Occasionally bubbles had been seen, but this was uncommon; even with a microscope it might be difficult to be certain that an exudate was present, although he agreed that the drum did sometimes take on a brownish tinge.

These cases were difficult to treat. He found it easier to remove the exudate if more than one incision was made in the tympanic membrane; he now made three. In the earlier cases he had rolled back the drum as in a stapedectomy and it was striking how much fluid could be aspirated from the region of the mastoid antrum after the middle ear was apparently clear. Encouraged by this, he had done a number of mastoid operations on these children, but their hearing had not been made any better; indeed, some of them had been worse than those treated by aspiration alone. Some, in spite of repeated aspirations, did not do well. He had found a characteristic sequence of events in all of them. At first there was the presence of this extraordinary gluey fluid with no naked-eye changes in the mucous membrane of the middle ear. Then the mucous membrane became obviously thickened and rather dense granulation tissue formed, particularly in the region of the stapes. When this occurred there was a peculiar ring-shaped depression in the drum, in the region of the posterosuperior quadrant. He had found that, when this stage was reached, the child's hearing defect was permanent.

Dr Stuart Carne (*London*) said that this subject had interested him greatly and for the past five years he had been trying to trace the natural history of this disease among the patients in his practice. He agreed with Mr Carter that it was extremely common. As it was not associated with pain and as the deafness was not severe – often less than 20 dB – it was unlikely that otologists in Great Britain saw more than a small proportion of all the cases. This was particularly true when it was remembered how hard it was to see the ear drum through the inadequate otoscopes still used by many general practitioners and pædiatricians; in addition, the survey of otitis published in the *Lancet* (1957, ii, 510) a few years ago had shown that the majority of patients were treated *in toto* by their general practitioners.

He was not so optimistic as Mr Carter about the prognosis. He was more interested to know whence this recurrent illness came. It was not always easy to recognize details on the drum of an infant; when it cried the drum looked red. When he got a good view he often saw the posterosuperior bulge in babies with an upper respiratory infection. He wondered whether inadequate antibiotic and chemotherapeutic treatment for this or other diseases led to incomplete resolution. It seemed to him that in this decade the deafness due to chronic suppurative otitis media had been replaced by the deafness due to secretory otitis media.

Mr Maxwell Ellis (*London*) said that the problem of serous middle ear effusions in children had certainly been overlooked for a long period and had been rediscovered with a vengeance. The treatment, generally speaking, was reasonably successful and often enough the cases resolved spontaneously. However, it was the cause of the condition which was more at issue. He therefore asked Dr Senturia how he was so sure that the changes he described were inflammatory. Dr Friedmann's changes were produced by the introduction of a pyogenic organism and were understandable. As he understood his description, Dr Senturia cauterized the nasopharyngeal orifice of the eustachian tube and the subsequent

changes he described occurred automatically; Mr Ellis was not at all clear why they did or should. He presumed there was some evidence that infection did occur for, if not, this thesis was built on a *non sequitur*. He also did not believe that the majority of the clinical cases he saw were due to infection.

Mr I B Thorburn (*Blackpool*) said that exudative otitis media was a common and frequently overlooked cause of conductive deafness about which very little was known. It was important to realize that it could have a silent or insidious onset at any age, but was more common in children. He stressed that the underlying pathological changes tended to be of a progressive type affecting essentially the mucous membrane lining the whole tubotympanomastoid cleft. It was important that the condition should be recognized and arrested in its early stages since this might prevent the development, possibly years later, of a diffuse cholesterol granulomatosis.

He endorsed Mr Carter's suggestion that the ears of all children suspected of being hard of hearing should be examined with the operating microscope. If a small incision through the postero-inferior part of the drum followed by suction demonstrated the presence of thick fluid he liked to introduce a small polythene tube which could be retained safely for many weeks. Rarely did anything drain subsequently through the polythene tube unless assisted by inflation or suction. It was his experience that neither the incision of the drum nor the polythene drain promoted infection even when combined with removal of tonsils and/or adenoids and antral lavage.

He wished to draw Dr Senturia's attention to a peculiar observation frequently noted on microscopic examination of the ears of patients under closed endotracheal general anæsthesia: an initially retracted tympanic membrane which would gradually distend with gas or air. There was no pressure within the pharynx and it was his impression that the gas did not come from the eustachian tube, but that it might be an anæsthetic gas, possibly nitrous oxide, liberated from the tympanic mucous membrane.

Mr N W MacKeith (*Southampton*), referring to those cases in which the fluid in the middle ear was mucus, said he thought it likely that (though infection might be a contributory factor) the cause was a disturbance of the physiology of the tubotympanic mucosa. Few of these cases were seen in adolescents or adults and the condition seemed comparable to the mucopurulent nasal discharge of some children which also cleared up as they reached the age of 10 or 12 years.

Mr Terence Cawthorne, Mr E H Miles Foxen, Mr P H Huggill and **Mr Colin Johnston** also took part in the discussion.

Dr Ben H Senturia, in reply, expressed pleasure that Dr Friedmann had confirmed their own findings in his human biopsies.

He did not see as many children with conductive deafness as did Mr Carter, but he had observed striking improvement in hearing following the aspiration of mucopurulent effusions from the middle ears of selected children. Although he had not sufficient proof at the present time, Dr Senturia believed the portentous aspect of this problem was the sensory-neural deafness which was found in follow-up observations on some of these children. He agreed that they were exchanging chronic conduction deafness and possibly inner ear deficiencies for suppurative otitis media and mastoiditis.

Mr Malcomson had spoken of adhesions in the middle ear. Dr Senturia's experience had been like that expressed by Mr Cawthorne in a symposium on adhesive otitis before this Society (1956, *J. Laryng.* **70,** 559). With a rare exception, Dr Senturia did not see adhesions in the middle ear. This was not unexpected, since adhesions did not occur readily in cavities lined by endothelium or ciliated epithelium such as the pleural spaces or the middle ear. The tympanic membrane was often found tightly adherent to the medial tympanic wall, but it could be readily peeled off without evidence of adhesions. The exceptions, of course, were those ears in which surgical interference or severe streptococcal disease had resulted in the destruction of large contiguous areas of lining membrane.

Dr Senturia regretted that he could not support with any pathological observations Mr Malcomson's suggestion regarding tympanosclerosis. When he first examined their experimental sections of long-term middle ear disease he thought they had produced tympanosclerosis in the dog. After more careful comparison with the findings in human biopsies it had become

obvious that several important pathological components of tympanosclerosis were absent. He had no doubt that infection played an important part in tympanosclerosis as well as in middle ear effusions but they were unable to relate tympanosclerosis and middle ear effusions.

Dr Senturia agreed with Mr Zalin's statement that middle ear effusions were associated with a tubotympanomastoiditis. He stressed the fact that the tubal component in humans and animals resolved promptly, leaving the smouldering middle ear and/or mastoid portion of the disease.

It was his belief that serous effusions were due to many causes, viz. tubal obstruction, aero-otitis, nasopharyngeal malignancy, myringitis bullosa hæmorrhagica, &c. He had ridden up and down the altitude chambers at the USAF School of Aviation Medicine and produced serous effusions in normal human volunteers by simply asking them not to swallow during descent from altitude. He had suggested that irritation of the mucoperiosteum of the tympanum might take many forms such as negative pressure, virus infection, &c., and would cause serum to leave the vascular channels and enter the lumen of the middle ear, producing a serous effusion.

He did not yet know how, when or why disease extended from the nasal fossæ or nasopharynx to the middle ear. It was known that trauma to the torus tubarius or vault of the nasopharynx would not induce an effusion, yet irritation of the nasopharyngeal orifice of the eustachian tube would do so. They had theorized that some unknown factor interfered with the ciliary beat in the tube, allowing upward migration and propagation of those bacteria which happened to be present in the nasopharynx.

Mr Ellis was quite right when he stated that Dr Senturia had not presented observations on the bacteriological or pathological changes which occurred at the nasopharyngeal orifice of the eustachian tube; it had not been possible to include everything they would have liked to discuss. Dr Senturia could confirm the fact that it was possible to scarify the nasopharyngeal orifice but that it was practically impossible to seal off the tube completely. He believed, however, that the important point lay in the fact that infection did not persist at the point of trauma in the nasopharyngeal orifice of the tube or in any part of the eustachian tube after the third day. Yet active inflammatory changes continued for months and permanent sequelæ were visible six months after cautery in the middle ear and mastoid.

Routine cultures had been taken of the nasopharynx and external auditory canal at regular intervals before, during and after cautery. The nasopharynx, external ear and aspirated effusions had been cultured aerobically and anaerobically at the time of the sacrifice of the animal. These studies needed to be extended, but it could be shown that, after cautery, exogenous organisms frequently appeared in the nasopharynx and shortly thereafter in the middle ear.

Dr Carne had emphasized the need for greater understanding of this problem and for proper indoctrination of the general practitioner and the pædiatrician. Dr Senturia had no statistics, but it was his impression that in the United States the overworked pædiatrician did not provide therapy for a long enough period of time to eliminate the infection from the middle ear completely. Disease therefore smouldered on. It was also highly possible that they were dealing with an attenuated organism which did not cause acute pain or fever yet produced a subacute otitis media which was disregarded by the parents or hidden by the child who was fearful of being restricted in his activities.

They had tried various forms of the corticoids for the treatment of middle ear effusions without impressive results. In those patients with much allergic rhinosinusitis they thought that the ear complication had been controlled temporarily, but the condition had recurred. Incidentally, those who considered the eosinophil as an indicator of the allergic state would be interested to know that among the hundreds of effusions which they had studied cytologically they had found only two or three which showed any significant increase in eosinophils. Needless to say, Dr Senturia was very much in favour of the use of corticoids and antihistamines for the treatment of persistent allergic rhinosinusitis which progressed posteriorly and produced nasopharyngitis and then middle ear effusions.

Dr Senturia shared the apprehension expressed by Mr Johnston regarding the performance of simultaneous myringotomy and adenoidectomy. He recalled the classical experiments of Dr Theodore Walsh (1943, *Laryngoscope* **53**, 75) with regard to the frontal sinus and the nasofrontal duct. If bacterial contamination of the lumen of the frontal sinus was present and the duct was undisturbed, disease did not occur. If, on the other hand, one irritated the region of the orifice of the nasofrontal duct in the middle meatus and then implanted organisms into the frontal sinus, an infection was produced. From a practical point of view Dr Senturia found it necessary to perform

myringotomy and adenoidectomy together, but he was careful to give broad-coverage antibiotics pre- and postoperatively. Despite these precautions they saw an occasional postoperative suppurative otitis media which required active treatment.

During the period of their service in the USAF, Dr Fred Ogden and Dr Senturia had developed a good deal of skill and understanding regarding the use of continuous-pressure politzerization. With this knowledge Dr Senturia found he could inflate the eustachian tube of almost anyone. Most children over 5 years of age would co-operate after a little coaxing. Pressures were routinely set at 15 mm Hg (0·5 lb). If tubal resistance was encountered, the pressure was raised progressively to 30, 60 or 90 mm Hg (1, 2 or 3 lb). It was necessary to move slowly in apprehensive patients since sudden inflation of a retracted tympanic membrane was frightening. It was possible to over-inflate or rupture a drum containing a monomeric membrane. Frequently it was necessary to provide the patient with a sip of water prior to inflation. The inflation was repeated several times on any single visit. Very occasionally Dr Senturia requested the patient or parent to purchase a positive-pressure source and instructed him to inflate the tubes twice daily at home.

Mr Huggill and Mr Cawthorne had called attention to the high incidence of serous effusions among adults and the large percentage of mucopurulent effusions in children. This was a good generalization, but Dr Senturia emphasized that mucopurulent effusions occurred occasionally in adults and serous effusions were found frequently in children. It was true that the adult was usually treated early since the effusion interfered with his daily activities while the child's condition seemingly developed insidiously and many parents tended to ignore it or attributed a hearing deficiency to inattention. Many of the so-called poor results in the chronic cases were obviously due to the otologist's impatience since, as could be seen on their sections, regression of the tremendously thickened lamina propria was a slow process.

In reply to Mr Thorburn, Dr Senturia said he had always assumed that when he removed the mucous plug from the tympanic orifice of the eustachian tube the positive pressure in the nasopharynx forced the gases up the tube. He recalled Politzer's early descriptions in which myringotomy had been followed by a flow of bubbles to the surface. The differential pressure between the nasopharynx and middle ear and the degree of 'locking' of the tube by intratympanic negative pressure played an important role.

Mr B S Carter, in reply, said that adenoidectomy and myringotomy had deliberately been done at the same operation, as an integral part of the investigation, and had produced no harmful effects.

In reply to Dr Carne, he agreed that a 20 dB loss would produce a handicap in a young child; most of the audiograms in the cases classified as normal would lie above that level.

Meeting May 3 1963

The subject of the meeting was **Polytomography of the Temporal Bone and its Clinical Implications.** The opening speakers were Dr F G M Ross (Bristol), Mr H D Fairman (Bristol) and Mr J Angell James (Bristol). Dr James Bull, Mr T Boyle, Mr G Livingstone, Dr P W E Sheldon and Dr I Simson Hall took part in the subsequent discussion.

(To be reported in the *Journal of Laryngology*)