CHRONIC EAR DISEASE

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Preface

During the past thirty years, the invention of the operating microscope, refinements in anesthesia, and the introduction of antibiotics have led to considerable improvements in the treatment of chronic middle ear disease. Interest in devising better methods of management has stimulated the development of a new system of surgical treatment. The purpose of this monograph is to provide an assessment of the current state of the art in general, and of tympanoplasty in particular. The basic principles of tympanoplasty were, of necessity, worked out on a trial and error basis. The accumulation of data has led to the evolution of certain concepts which now appear likely to justify the aspirations of the original tympanoplastic surgeons.

In this monograph the possible etiological processes of chronic suppurative otitis media are reviewed, the surgical techniques which have been most widely used described, and the results obtained with them reported. There are still a number of situations for which we have no satisfactory solution. These are defined and the means by which they might be better managed in the future is discussed.

Acknowledgments

I cannot adequately thank two friends who labored so strenuously in the production of this monograph, with unbelievable willingness and cheerful cooperation. For them (and for the writer) it has been a mammoth task which, without them, could not have reached completion. Miss May Weller prepared the manuscript with devoted care, and the beautiful illustrations were drawn by Miss Helen McIlhenney.

My sincere gratitude is also due to Dr. T. H. Hassard, M.Sc., MIS, Ph.D., who analyzed the numerous statistics, to Professor A. G. Kerr, M.B., FRCS, who interpreted the histologic material derived from revision operations and animal experiments, and to Mr. Matthew Thompson for his helpful proofreading.

Finally, because the work with which this monograph is concerned depended upon the cooperation of many thousands of patients, whose fortitude I admire and appreciate, I would proffer my most sincere thanks to all my younger medical colleagues, nursing staff, and audiometricians who made it possible.

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PATHOLOGY OF CHRONIC OTITIS MEDIA

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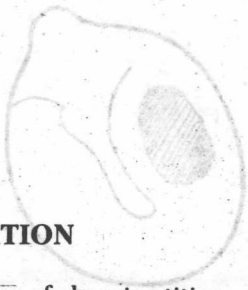

CLASSIFICATION

The classification of chronic otitis media is bewildering. Although most texts define a number of inflammatory pathologies of the middle ear, and treat them as separate clinical entities, this is not necessarily consonant with recent advances in knowledge. It does seem correct that many subtotal or total perforations of the tympanic membrane, with or without an ossicular defect, were formerly the result of prior episodes of acute inflammation complicating viral infections such as morbilli, but this traditional view is less tenable in the light of recent experience. Acute necrotizing otitis media from any cause is now uncommon in most developed communities, and we must therefore find another explanation for the central defects of the tympanic membrane which are still frequently observed.

Since its first description by Cruveilhier[6] in 1829, surgeons and pathologists alike have been largely preoccupied with a particular type of chronic otitis media, cholesteatoma, and much confusion has arisen from attempts to formulate a single hypothesis to explain the etiology of this condition. Only since the introduction of microsurgery have other, less sinister forms of middle ear disease begun to assume importance. Furthermore, whereas the eradication of invasive disease was once paramount, the recent development of techniques for functional restoration has stimulated the search for a method to combine both goals in the composite procedure of tympanoplasty. However, because of conflicting attitudes to different pathologies, and variety in skill and motivation, a number of procedures have evolved whose principles do

not always appear to agree. Hence, for example, we have at present an unresolved disagreement between the "open" and "closed" schools of thought regarding the treatment of cholesteatoma.

Having studied the diverse theories of pathogenesis which have frequented the literature on chronic suppurative otitis media (CSOM) during the last century, and having attempted to correlate these with clinical observation of the condition, I have concluded that persistent middle ear effusion (MEE) has a fundamental role in all commonly occurring varieties of CSOM. Hence, I have argued that tubotympanic disease, cholesterol granuloma, the atelectatic ear, and cholesteatoma, being not necessarily separate clinical entities, are in effect the pathologic results of differences between host resistance to infection and variations in virulence, anatomy, and therapy.[24]

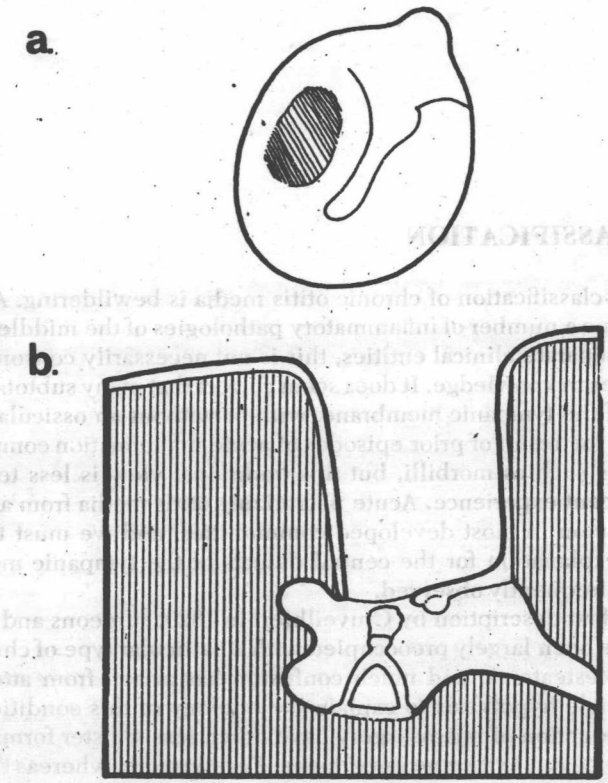

Fig. 1–1 (A) Otoscopic appearances of "scarred" but intact tympanic membrane; (B) Schematic representation of the thin posterior tympanic membrane.(Smyth, G. D. L.: Middle ear effusions: A consideration of factors involved in their aetiology, maintenance and treatment. *Clin. Otolaryngol.*, 3:479, 1978.)

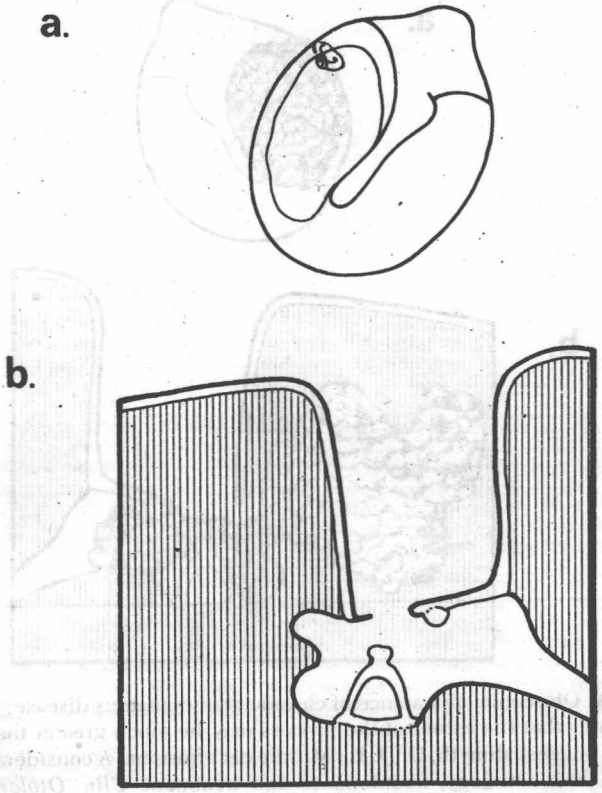

Fig. 1–2 (A) Otoscopic view of a defect in the posterior tympanic membrane; (B) Schematic respresentation of this pathologic entity. (Smyth, G. D. L.: Middle ear effusions: A consideration of factors involved in their aetiology, maintenance and treatment. *Clin. Otolaryngol.*, 3:479, 1978.)

If the foregoing concept is correct—and the evidence from (1) repeated observations of the ears of patients with MEE; and (2) the now well known potential for cholesteatoma to recur after surgical treatment strongly suggest that it is—then a practical and more easily understood classification of CSOM is possible, and certainly necessary. I find such a concept particularly attractive because it eliminates the need to conceive multiple etiologies to explain the various clinical entities observed in practice.

Although as already stated CSOM can, in cases where a perforation of the tympanic membrane fails to heal, be the direct result of acute suppurative otitis media (ASOM), this is rarely observed. As a result of clinical observation and reasoning I have come to believe that, practically without exception,

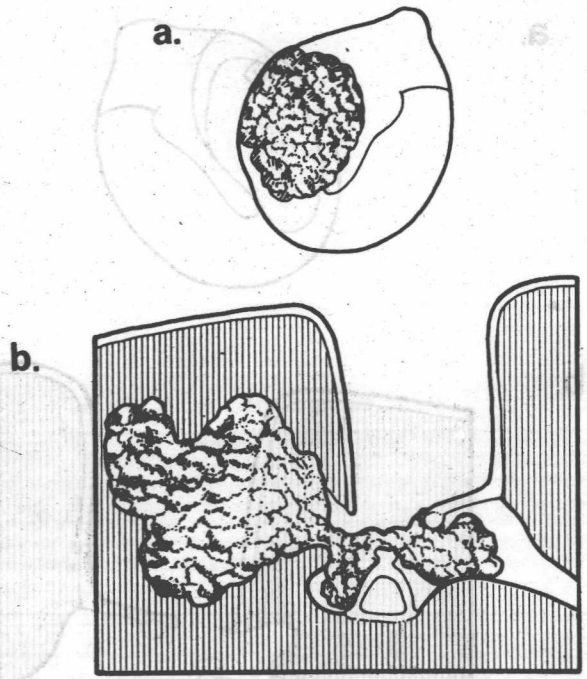

Fig. 1–3 (A) Otoscopic appearance of chronic granulomatous disease; (B) Schematic view illustrates that the extent of the process may be much greater than the clinical appearances suggest. (Smyth, G. D. L.: Middle ear effusions: A consideration of factors involved in their aetiology, maintenance and treatment. *Clin. Otolaryngol.*, 3:479, 1978.)

CSOM is the result of unresolved MEE. My postulate is that, because of the action of enzymes in the effusion, there develops an area of the tympanic membrane which lacks substance owing to loss of its fibrous and elastic tissue layers. At this stage there are four possible outcomes:

1. Nothing changes, and in later life it will be noted that there is a "healed scar" of the tympanic membrane which does not significantly impair hearing (Fig. 1–1).

2. The weak area will break down during a subsequent episode of ASOM, leading to permanent perforation of the membrana tensa. Examination at this stage will reveal a tympanic membrane defect of variable size, a view of the medial wall of the tympanum, and possibly of the stapes and long process of the incus (depending on the size of the defect and the state of the ossicles) (Fig. 1–2). Granulation tissue and mucoid or mucopurulent discharge may be present. It should be emphasized that the *development* of this

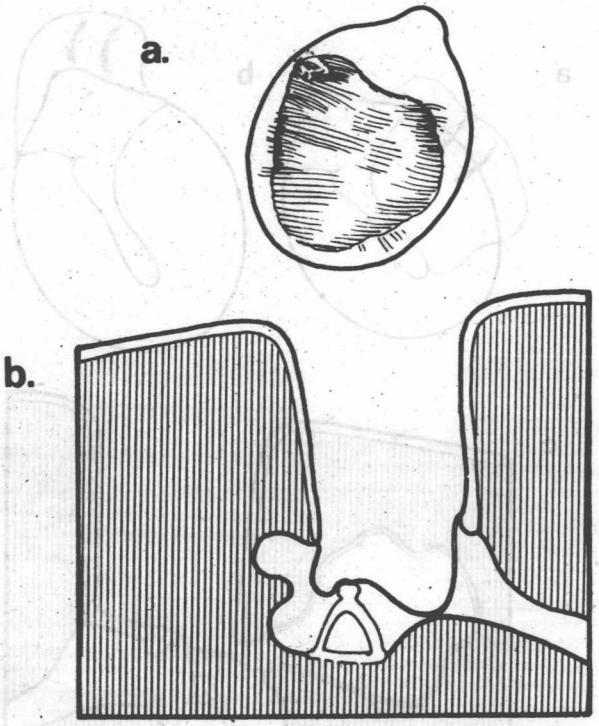

Fig. 1–4 (A) Otoscopic appearance of a collapsed tympanic membrane plastered onto the medial wall of the mesotympanum; (B) Schematically, pockets may form around the remnants of the ossicular chain. (Smyth, G. D. L.: Middle ear effusions: A consideration of factors involved in their aetiology, maintenance and treatment. *Clin. Otolaryngol.*, 3:479, 1978.)

phase is rarely documented by otologists because it usually occurs in patients who, having been without symptoms, were not under prior observation. Although the initial acute infection can usually be brought under control, a chronic tympanic membrane defect constitutes a permanent threat of reinfection either from the external ear canal via the perforation, or by insufflation of secretions from the nasopharynx because of the altered aerodynamics of the tubotympanic cleft.

The range of clinical presentations is extreme extending from a simple central perforation with little or no mucosal or ossicular disease to a more generalized mucosal involvement causing prolific granular mucosal reaction (cholesterol granuloma) and chronic osteitis of the ossicles and bone of the mastoid air cell system resulting in persistent odoriferous discharge with progressive hearing loss (Fig. 1–3).

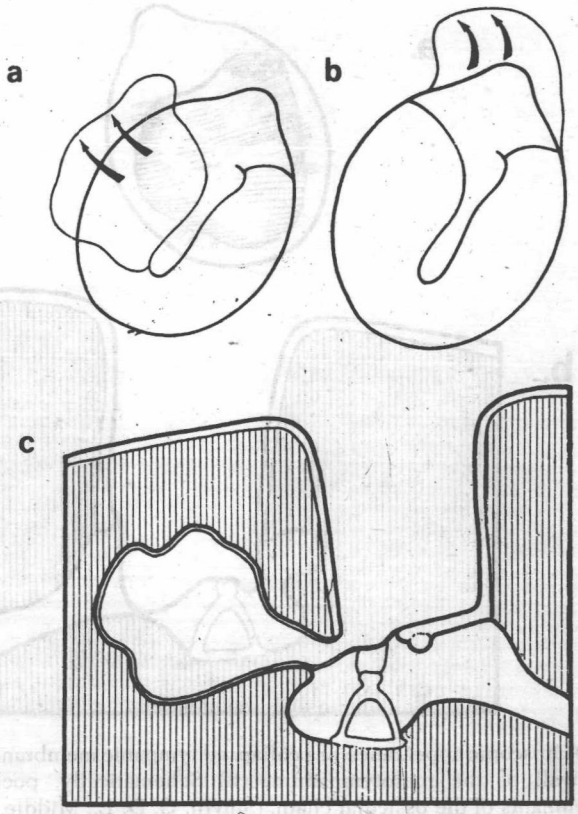

Fig. 1–5 (A) Retraction pockets extend into the facial sinus and; sinus tympani or (B) epitympanum; (C) Schematically, such a pocket is shown reaching the far extent of the retrotympanic air cells. (Smyth, G. D. L.: Middle ear effusions: A consideration of factors involved in their aetiology, maintenance and treatment. *Clin. Otolaryngol.*, 3:479, 1978.)

3. The third possible outcome of persistent MEE also derives from a weakened area of the tympanic membrane. In this case a large area of the membrane is affected and, possibly as a consequence of persistent reduced intratympanic pressure, the traction of adhesions, and an altered direction and rate of growth – all acting separately or in conjunction – the weakened tympanic membrane recedes medially to form an invagination which lines the mesotympanum and drapes any remaining ossicular tissue (atelectasis). As a result a self-cleansing stratified squamous epithelial pocket, with a broad aperture, is formed within the mesotympanum (Fig. 1–4).

4. Alternatively, (1) an already-formed atelectatic pocket in the meso-

tympanum loses its self-cleansing powers, becomes infected and, owing to the stimulus of inflammation, actively grows into the epitympanum and retrotympanic spaces (Fig. 1–5); or (2) persistent middle ear effusion provokes a cellular response either in the basilar lamina of the tympanic membrane or in epitympanic mesenchymal rests, either of which may lead to the development of keratinizing squamous epithelium within the middle ear cleft. Although pathologists do not yet agree upon the precise mechanism whereby the specious pathologic entity termed cholesteatoma develops, a cause and effect relationship between middle ear effusion and cholesteatoma is now increasingly accepted.

Thus, I propose that MEE is the underlying cause of virtually all forms of chronic suppurative otitis media, ranging from benign uncomplicated perforations of the tympanic membrane, through atelectasia of the middle ear, to life-threatening cholesteatomatous disease of the petrous bone.

The main purpose of this chapter is to discuss the pathology of the clinical entities which are treated by tympanoplasty. However, both because prevention of the disease or its treatment at an early reversible stage are the ultimate goals, and because I believe that each of the clinical entities treated by tympanoplasty develops from MEE, current attitudes to this disorder will be considered initially.

MIDDLE EAR EFFUSION

The now voluminous literature on MEE illustrates the confusing and contradictory views about the factors which initiate and maintain this effusion. The dimensions of the problem are apparent from the numerous and varied descriptive terminologies for the condition which have been used during the past decade, and which include secretory otitis media, serous otitis media, catarrhal otitis media, seromucinous otitis media, and even the mundane "glue ear." For purposes of general discussion the title "middle ear effusion" is probably more suitable than most, since it encompasses all varieties of the condition with minimal prejudice.

It is notable that most otologic textbooks contain illustrations of the Eustachian tube, which together with their commentary on its opening mechanisms seem to confer an enormous emphasis on dysfunction of the tube as the prime cause of MEE. To attribute such an overwhelming importance to the Eustachian tube and to consider it in isolation could well be misleading, particularly if in so doing the influence of other factors, such as abnormal nasopharyngeal air-pressure changes, are ignored. For better understanding of the complex and interrelated forces which may initiate and maintain MEE, a model comprising the external ear, the tympanic membrane, the middle-ear space, mastoid air cells, and the Eustachian tube in communication with the nasopharynx and nose is proposed (Fig. 1–6). Most importantly, such a model takes into account the range of frequent volumetric and air-pressure changes

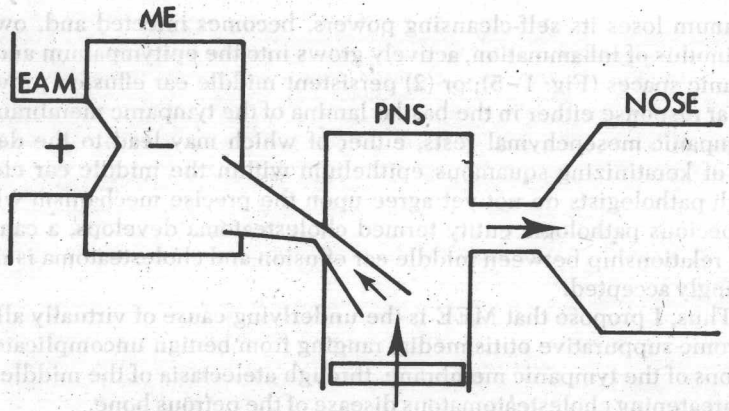

Fig. 1–6 Model designed to demonstrate air pressure relationships between external auditory meatus (EAM), middle ear (ME), postnasal space (PNS) and nose. During swallowing, elevation of the soft palate results in a larger displacement of air anteriorly into the nose than posteriorly into the Eustachian tube.

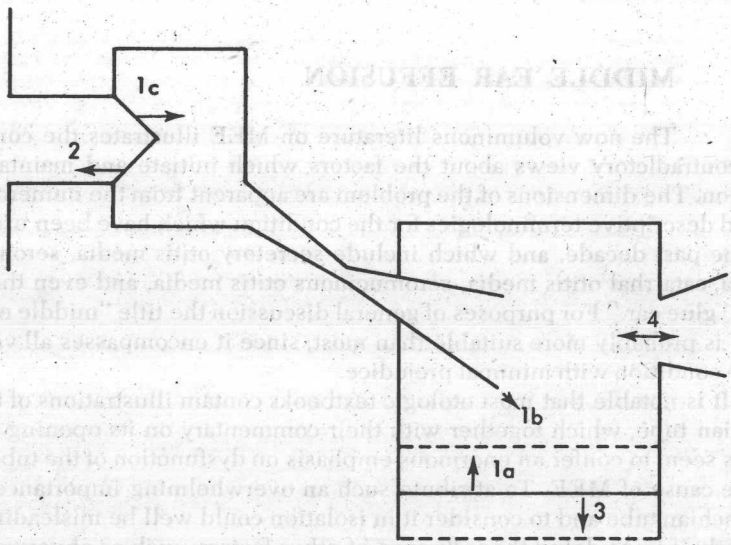

Fig. 1–7 During first phase of swallowing, ascent of the palate (1a) elongates the cartilaginous Eustachian tube (1b) and produces a temporary inward movement of the tympanic membrane (1c). At the moment of tubal opening air enters the middle ear and allows the tympanic membrane to move outward (2) until the second phase of swallowing begins with relaxation and depression of the palate (3). Throughout both phases air passes freely to and from the nose (4).

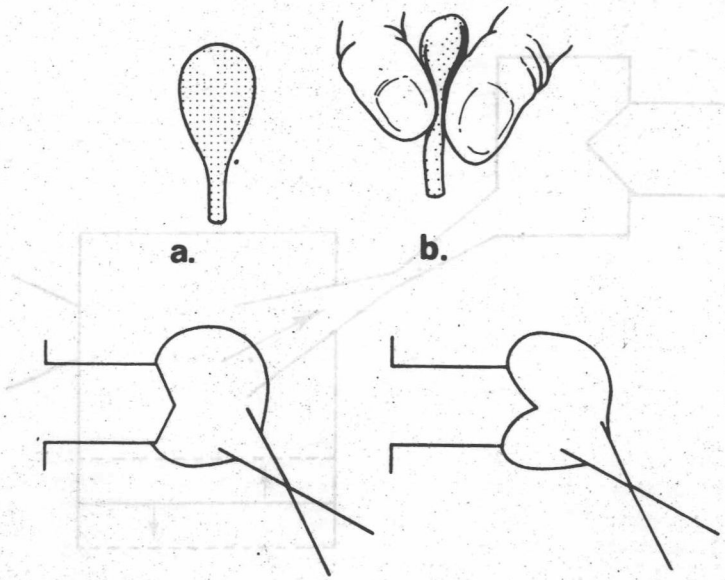

Fig. 1–8 (A) The middle ear is likened to a medicine dropper; (B) The development of negative pressure in the middle ear creates a potential for aspiration which is assisted by the elasticity of the tympanic membrane.

occurring routinely in the upper respiratory tract, the understanding of which is vital for a better comprehension of this fascinating and chimaeric disease.

The need for periodic opening of the Eustachian tube to replenish oxygen absorbed from the middle ear is universally recognized and undisputed. Lamp,[9] in a discussion of etiologic factors in chronic secretory otitis media, makes several interesting observations on tubal function which can be confirmed clinically. Because of their relevance to the workings of the previously proposed composite model, some of these observations will be used to illustrate its mechanism.

During the first phase of swallowing (Fig. 1–7), traction on the soft tissue of the Eustachian tube elongates the column of air within its lumen, with the consequence that the inevitable immediate reduction of middle ear air pressure produces an inward movement of the tympanic membrane, which acts as a diaphragm pump. The situation at this point is similar to that existing when the end of a medicine dropper is compressed (Fig. 1–8). With further opening of the Eustachian tube the elasticity of the tympanic membrane, now cocked inward, aspirates air into the middle-ear space to replenish that which had previously been absorbed. As the palate relaxes, during the second phase of swallowing (Fig. 1–7), the tube closes, trapping within the middle ear a