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(*Founded in* 1887 *by* MORELL MACKENZIE *and* NORRIS WOLFENDEN)

August 1952

LARYNGEAL NODES AND THE SO-CALLED AMYLOID TUMOUR OF THE CORDS

By H. D. BROWN KELLY and J. E. CRAIK (Glasgow)*

IN December, 1949, at a meeting of the Scottish Otolaryngological Society, one of us (H.D.B.K.) presented a case of so-called vocal cord amyloidosis. In the discussion which followed, the late Dr. Ewart Martin drew attention to the uncertain status of the pathology of this condition and suggested that further investigation might be profitable. With this end in view, we have studied all the laryngeal material that has come to this laboratory for histological examination in the past five years. All cysts and epithelial tumours whether simple papillomas, intra-epithelial carcinomas or frankly invasive carcinomas were excluded : there were no examples of malignant tumours other than carcinoma. We were then left with 37 lesions from 35 patients (Table I) which form an apparently heterogeneous collection of œdematous, angiomatous and myxomatous polyps, angiomas, fibromas, varices, singer's nodes, amyloid tumours and one case previously reported as chondroma (Kelly, 1950). These apparently unrelated lesions we consider to be different manifestations of one condition, the simple laryngeal node, the various pathological terms having been used merely to describe the histological appearances. This view of the interrelationship of these so-called simple tumours of the larynx was first put forward as long ago as 1895 by Chiari and has more recently been sustained by Friedberg and Segall (1941) and by Ash and Schwartz (1944). The present paper is a clinico-pathological study of the 37 laryngeal nodes selected on a purely histological basis with the addition of one case

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(Case 1) from which no biopsy was taken. The salient clinical and pathological findings of all these cases are given in Tables I, II, and III, and some illustrative case histories are in the appendix. It should be noted that in Case 7 the lesion was bilateral and that Cases 27 and 32 had recurrences, labelled respectively 27B and 32B.

Synonyms

The laryngeal node has various synonyms, the more common being singer's node, teacher's nodule, chorditis nodosa and chorditis tuberosa. In children it is referred to as screamer's node.

Aetiology

That vocal abuse is the chief factor in the formation of laryngeal nodes is well recognized. The voice may be suddenly and violently strained by shouting or screaming at, for example, a football match. In most instances, however, the vocal abuse is constant and long continued, usually being associated with the patient's occupation: thus singers and school teachers are frequent sufferers. A most important factor is bad voice production such as speaking or singing in a high pitch to which the larynx is not suited. Those who spend hours conversing with deaf relatives are often affected. Several of our cases were workers in machine shops who carried on their daily conversation above the noise of machinery. Of our 35 patients, 19 (54 per cent.) gave a definite history of overstraining the voice (Table I).

Jackson and Jackson (1937) drew attention to a less obvious type of voice abuse when they pointed out that some individuals phonate in a way peculiarly irritating to the vocal cords and this they referred to as the "throaty voice". Maljutin (1931) reported a series of stroboscopic examinations on 200 pupils at the State Conservatory of Moscow. He found that at the beginning of vocal exercises, vibratory excursion was greater in one cord, usually the right in right handed pupils and the left in left handed individuals. Occasionally vibrations were entirely confined to one cord, the opposite one remaining tense and immobile: it was in these cases that laryngeal nodes tended to develop. We do not find any preponderance of nodes in the right cord. Out of 32 cases in which the side was recorded, 2 were bilateral, 15 right sided and 15 left sided (Table I).

Nasal obstruction and sepsis have been suggested as factors in the production of nodes. Thus it is easily imagined that in the presence of impaired nasal respiration, the mucus covering the cords might become dried and leave the cords unprotected. Purulent post-nasal discharge bathing the cords is sometimes observed. In our series marked nasal obstruction or sepsis was present in 8 cases (23 per cent.).

The effect of excessive smoking on the vocal cord has been recorded by

Myerson (1950). Under the term "Smoker's Larynx", he described lesions similar to laryngeal nodes in both clinical and histological appearances: he recorded 143 cases, three being cigar smokers and the remainder smoking from 20 to 120, mostly over 40, cigarettes daily. In the early œdematous stage he observed complete disappearance of the lesion within a day of stopping smoking. Heavy smoking has not been a feature of our cases.

Very often nodes are not accompanied by laryngeal catarrh or injection of the cords. This observation has led StClair Thomson and Negus (1937) to suggest that "the basis of the condition is muscular strain". We regard the absence of inflammatory reaction to be due to the relative absence of blood vessels in the cords (Fig. 1) as explained in the section on Pathology. Jackson and Jackson (1937) consider that as many as 4 per cent. of all cases of hoarseness are due to speaking with the false cords, "dysphonia plicae ventricularis". On phonation, in these patients, the ventricular bands come together before the true cords, which are thus prevented from vibrating. It is possible that aberrant voice production by the false cords may produce laryngeal nodes of the ventricle or ventricular bands, such as in case No. 2. We have not been able to find any case in the literature attributed to such a sequence of events.

After all these conditions have been considered, there are still cases of laryngeal node for which no cause can be found. Further, since thousands of people abuse their voices, but only a very few develop laryngeal nodes there must be other ætiological factors. Of these we consider the most important to be that unscientific but human variable, the "reaction of the individual". This individual reaction to stress suggests a possible relationship to Selye's General Adaptation Syndrome (1946). In this connection it will be shown later that, at least in the case of the so-called amyloid laryngeal node, the histological features are similar to those of some of the collagen diseases which are generally regarded to-day as diseases of adaptation.

Clinical Features

Reference to Table I shows that of the 35 patients, 25 were male and 10 female. The ages ranged from 24 to 71 years. In one case (No. 2) the node originated in the ventricle. The exact anatomical relationships were recorded in 17 cases, and of these only one node was situated on the posterior third of the cord; the remaining 16 were on the anterior or middle third. On stroboscopic examination both the movements of the cords and the shape of the glottis can be seen to vary considerably according to the vocal register. In the production of head tones only the free edges of the cords vibrate, while in chest tones the cords show much stronger vibrations of greater excursion along their whole length. With an ascending pitch, the glottic chink becomes shorter; at first it is

TABLE I

CLINICAL FINDINGS IN 38 LARYNGEAL NODES FROM 35 PATIENTS

Case No.	Ref. No.	Age	Sex	Duration of symptoms	Vocal abuse	Onset	Site of lesion	Type of node
1	—	24	F	1½ years	Yes	Gradual	Bilateral. Junction ant. and mid. 1/3	——
2	386/50	69	M	2 months	Yes	Gradual	Left ventricle	Œdematous
3	1933/47	53	M	2 months	Yes	Gradual	Right ant. 1/3	Granulomatous
4	1290/49	50	F	6 months	No	Gradual	?	Granulomatous
5	158/50	38	M	2 months	Yes	Intermittent	Right ant. 1/3	Granulomatous
6	1317/50	40	M	3 months	No	?	Left cord	Granulomatous
7A	3137/51	52	M	3 weeks	Yes	Sudden	Left ant. 1/3	Granulomatous
7B							Bilateral. Right ant. 1/3	Fibromatous
9	157/47	64	M	7 weeks	Yes	Gradual	?	Angiomatous
10	2193/50	47	M	2 years	Yes	Gradual	Left ant. 1/3	Angiomatous
11	2964/51	71	M	1 year	No	Gradual	Left cord	Angiomatous
12	248/51	41	F	4 months	No	Intermittent	Right ant. 1/3	Varicose
13	1162/49	55	M	6 months	Yes	Gradual	Left cord	Fibromatous
14	1293/49	53	M	3 months	Yes	Intermittent	Right post 1/3	Fibromatous
15	1182/47	64	M	3 months	No	Gradual	Left ant. 1/3	Fibromatous
16	1887/47	53	F	2 years	No	Gradual	Right junct. ant. and mid. 1/3	Fibromatous
17	1508/48	43	F	6 months	No	Gradual	Left cord	Fibromatous
18	2022/48	61	F	6 months	No	Gradual	?	Fibromatous
19	28/50	51	M	9 months	Yes	Gradual	Right ant. 1/3	Fibromatous
20	249/51	58	F	years	No	Gradual	Right cord	Fibromatous
21	469/51	60	F	6 months	Yes	Gradual	Left cord	Fibromatous
22	2118/51	51	M	2 months	Yes	Intermittent	Right mid. 1/3	Frbromatous
23	3383/51	26	M	years	Yes	Gradual	Right mid. 1/3	Fibromatous
24	3021/51	31	F	7 years	No	Gradual	Left ant. 1/3	Fibromatous
25	1445/48	52	M	5 months	No	Gradual	Left ant. 1/3	Nodular hyaline
26	1283/50	52	M	3 months	No	Gradual	Left cord	Nodular hyaline
27A	3218/50	41	M	2 years	No	Gradual	Right cord	Nodular hyaline
27B	294/51	41	M	6 months	No	Gradual	Left cord	Nodular hyaline
29	2838/50	44	M	3 months	No	Gradual	Right cord	Nodular hyaline
30	779/49	59	M	3 months	No	Gradual	Cord	Diffuse hyaline
31	1596/50	24	M	1 month	Yes	Gradual	Right cord	Diffuse hyaline
32A	1723/48	40	M	1 year	Yes	Intermittent	Right ant. 1/3	Diffuse hyaline
32B	2630/50	42	M	Less than 2 years	Yes	Gradual	Right cord	Diffuse hyaline
34	1449/49	57	M	4 years	Yes	Gradual	Left cord	Hæmorrhagic infarct.
35	1676/47	60	F	14 years	No	Gradual	Left cord	Hyaline mass.
36	3061/51	48	M		Yes			Hyaline mass.
37	358/51	50	M	6 months	No	Gradual	Right cord	Hyaline mass.
38	2500/49	50	M	2 years	Yes	Intermittent	Right ant. 1/3	Chondroma

Case No. 7 had bilateral nodes, 7A and 7B.
Cases 27 and 32 had recurrences, 27B and 32B respectively.
" Ref. No." is the number of the original histological report.

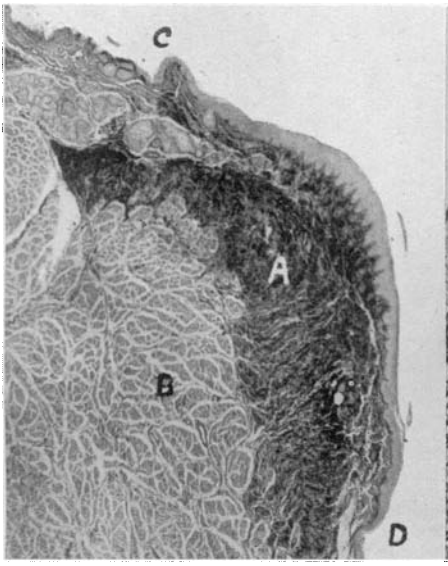

FIG. 1.

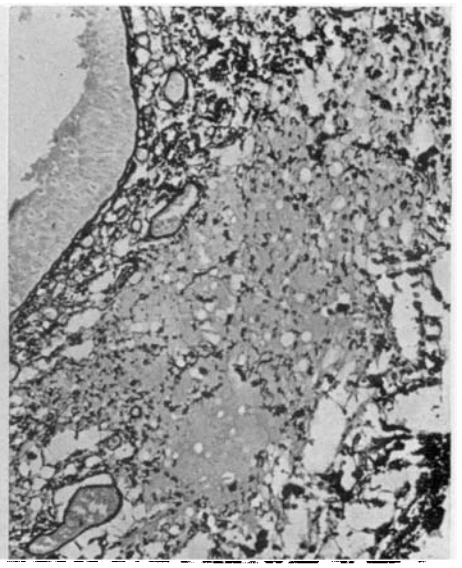

FIG. 2.

FIG. 1.

Transverse section of normal vocal cord.

A. Reinke's triangle with rich network of elastic fibres, stained black, and poor blood supply. B. Striped muscle. C. Superior arcuate line. D. Inferior arcuate line. Note the close apposition of the epithelium to the muscle at the arcuate lines.

Weigert's resorcin-fuchsin. Minus blue filter: pan. plate. ×22.

FIG. 2.

Case 2. Subepithelial œdema and disruption of elastic fibres. Œdema fluid extends across the centre as a greyish pool in which the black stained fragments of elastic tissue are scattered.

Weigert's resorcin-fuchsin. Orange filter: pan. plate. ×170.

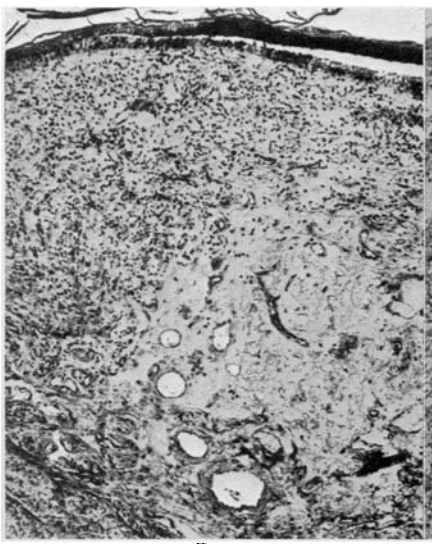

FIG. 3.

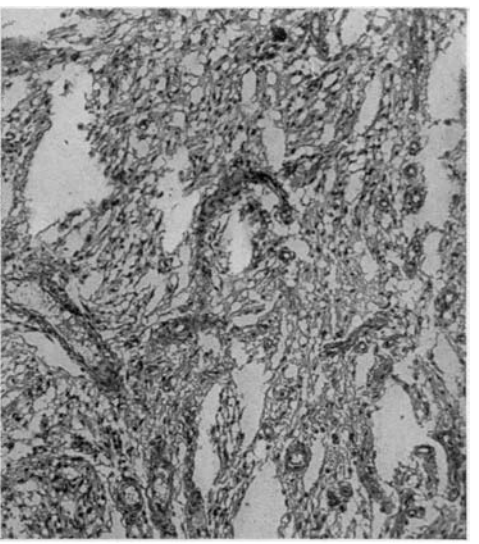

FIG. 4.

FIG. 3.

Case 3. Granulomatous node: vascular and highly cellular granulation tissue. The cells are lymphocytes, plasma cells and fibroblasts.

H. and E. Blue-green filter: process pan. plate. ×65.

FIG. 4.

Case 10. Angiomatous node: irregular vascular channels with little stroma. This might justifiably be termed a true angioma.

Picro-Mallory. Orange filter: pan. plate. ×100.

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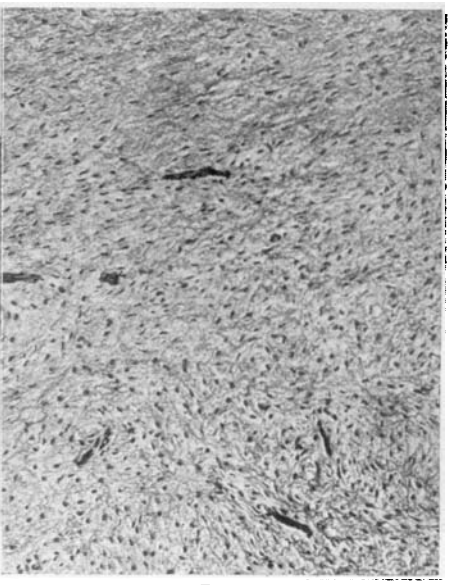
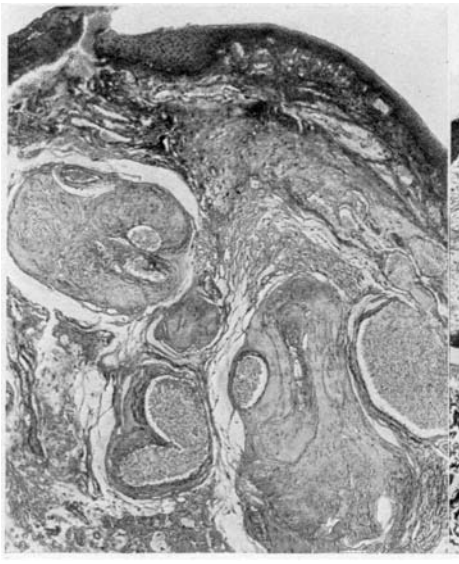


FIG. 5. FIG. 6.

FIG. 5.
Case 12. Laryngeal varix: wide spaces containing a few cells and albuminoid fluid.
Picro-Mallory. Orange filter: pan. plate. ×68.

FIG. 6.
Case 23. Fibromatous node: cellular fibrous tissue arranged in intersecting and whorling bundles. The structure is similar to that of a true fibroma.
Picro-Mallory. Minus blue filter: pan. plate. ×100.



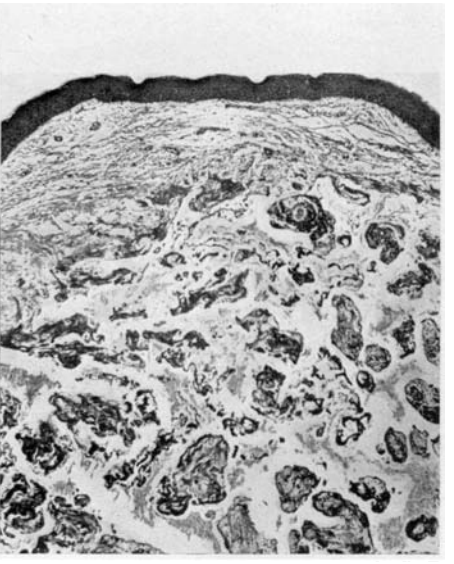


FIG. 7. FIG. 8.

FIG. 7.
Case 30. "Amyloid tumour": hyalinized thrombi in greatly dilated vessels. The massive thrombi are recanalized by smaller vessels. The so-called amyloid substance appears greyish and structureless. Compare with Figs. B and C.
Picro-Mallory. Orange filter: pan. plate. ×42.

FIG. 8.
Case 27A. Hyaline node showing amyloid staining reaction: hyalinization of thrombi in and around multiple plexiform vessels. The so-called amyloid substance is stained red and appears black in the photomicrograph.
Congo red for amyloid. Blue-green filter: pan. plate. ×42.

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bounded by the anterior two-thirds of the cords, and finally by the anterior halves, the posterior halves being held firmly together and taking no part in the vibrations. In the lower tones, the cords appear to be humped up, and to vibrate forcibly against one another in the middle thirds of their length (Kallen, 1932). The anterior half is therefore the region of the cord that has to withstand the greatest mechanical strain : thus nodes are confined almost entirely to this area.

The most recent statistical review is that of Holinger and Johnston (1951) who present an analysis of 1,197 " Benign Tumours of the Larynx ". Out of this total 775 were conditions which we would prefer to classify simply as variants of the laryngeal node. The incidence of these lesions was : polyps 521, nodules 193, polypoid degeneration 112, non-specific granulomas 37, hæmatomas 5, hæmangiomas 4, lymphangiomas 1 and amyloid tumours 2. Twelve other granulomas followed intubation of the trachea and have therefore been excluded by us on the ground that they are not true laryngeal nodes.

Laryngeal Appearances

StClair Thomson and Negus (1937) and Jackson and Jackson (1937) consider that vocal nodes are usually bilateral, and even when there is a well formed mass on one cord only, the opposite cord always shows at least a recognizable swelling, however insignificant. Even without node formation, bilateral œdema can cause narrowing of the glottis to such an extent that the voice becomes deep and vibrating. In our series only Cases 1 and 7 were bilateral : in the remainder, the cord opposite to that bearing the nodule appeared to be normal. Kallen (1932) has demonstrated that in some cases closure of the cords is not prevented, because the node may be as far as one millimetre from the margin. Singers may have such nodes for years without disturbance to the voice.

According to Jackson and Jackson (1937), in the very early stages the cords lose their clear white surface, and become dull, succulent looking, and injected at the point where the node is likely to form. When œdema has developed, the anterior two-thirds of the cords lose their normal shape, becoming soft, flabby and of a greyish-red colour. To this condition Hajek (1925) applied the term " Reinkesche Ödem " after the anatomist who first described the subepithelial layer of the cord. The appearances of the fully formed node vary, and there is often a remarkable absence of laryngeal injection or inflammation. There may be a mere localized thickening which is of the same colour as the rest of the cord. The œdematous polyp is pale grey or translucent : it is usually sessile but occasionally pedunculated, in which case it lies hidden below the cords, popping up on phonation to become visible. The angiomatous node is of a darker colour varying from pink-yellow to dark crimson. The tumour in which the largest amount of hyaline material was found (Case 34 and

Fig. 16) presented as a mamillated swelling of a dirty yellow colour. Occasionally there is generalized swelling of the cord simulating malignant disease as in the case recorded by Williman (1912).

Pathology

An appreciation of the minute anatomy of the vocal cords is necessary for a full understanding of the development and progression of the laryngeal node. The architecture of the cords was first described in 1895 by Reinke, but it was not until 1925 that Hajek correlated the clinical features with the peculiar anatomical structure. Fig. 1 is a transverse section of a normal vocal cord taken at autopsy, showing the true cord covered by stratified squamous epithelium which changes to columnar epithelium at the superior and inferior arcuate lines. Between the epithelium and the muscle is a triangular space, the space or triangle of Reinke, bounded above by the superior surface of the cord, medially by the inner surface of the cord and infero-laterally by the underlying muscle. The basal angles of this triangle are at the arcuate lines, at which points the epithelium approaches the muscle very closely. Reinke's space is extremely rich in elastic fibres which are stained black in the figure but it contains very few blood vessels. This lack of blood vessels means that irritation of the cords results primarily, not in a granulomatous reaction, but in œdema, and this œdema remains confined to the space of Reinke because of the close apposition of the epithelium to the muscle at the arcuate lines. The localized œdema produces swelling which disrupts the elastic fibres (Fig. 2). Since elastic tissue has poor powers of regeneration it will be largely replaced by less pliable fibrous tissue, so that prolonged œdema of the cord can itself cause irreversible changes. Sooner or later, however, continued irritation will stimulate the ingrowth of new blood vessels and fibroblastic proliferation with the formation of granulomatous tissue (Fig. 3). When the blood vessels are numerous the tissue will have the appearance of an angioma (Fig. 4) and dilation of blood vessels or lymphatics will produce a varix (Fig. 5). More commonly, fibroblastic activity is relatively greater than new vessel formation, so that a fibromatous mass (Fig. 6) results. An inflammatory round celled infiltration may be superadded to any of these lesions but this is usually confined to the subepithelial layers and is rarely a striking feature. Epithelial hyperplasia occurs only occasionally in true laryngeal nodes; atrophy is much commoner. Because all these changes are localized, they result in larger or smaller swellings of the cord which are usually described as " nodes " when sessile and " polyps " when pedunculated. Some of them may appear clinically to be papillomas but this term should be reserved solely for true tumours which are proved histologically to be of epithelial origin. In Tables I and II our cases are arranged into groups according to the histological appearances described above.

Laryngeal Nodes and Amyloid Tumour of the Cords

Ash and Schwartz (1944) have attempted to arrange the lesions in four chronological stages: (1) fibroid, (2) polypoid, (3) varix, (4) hyalin or amyloid. But in Table II in their paper, as they themselves state, the duration of clinical symptoms cannot be correlated with the so-called stages. We find the same difficulty with our cases: the various histological stages are related neither to the duration of symptoms nor to the severity of voice abuse. For instance, two fibromatous nodes (Table I, Cases 20 and 23) had produced symptoms for many years, and 7 out of 13 cases of so-called amyloid tumour had been present for 6 months or less. We thus prefer to regard these changes as differing types of reaction to irritation rather than as developmental stages. Similar reactions to irritation are seen in any part of the body, an obvious and appropriate example being the irritation granulomas, and the so-called angiomas and fibromas so common in the mouth. Thus we feel that all the varieties so far described should be classified primarily as laryngeal nodes and the descriptive histological types added as a qualifying term. The various conditions would therefore become œdematous, granulomatous, angiomatous, varicose and fibromatous laryngeal nodes (Table II).

TABLE II

HISTOLOGICAL FINDINGS IN 24 NON-HYALINE LARYNGEAL NODES

Case No.	Staining reactions		Fibrinoid material	Illustrations	Type of node
	Congo red	Gram			
1	—	—	—	Fig. 20	No biopsy
2	Neg.	—	Nil	Fig. 2	Œdematous
3	Neg.	Pos.	Fibrils	Fig. 3	Granulomatous
4	Neg.	—	Nil	—	Granulomatous
5	Neg.	—	Nil	—	Granulomatous
6	Neg.	—	Nil	—	Granulomatous
7A	Neg.	Pos.	Fibrils	—	Granulomatous
7B	Pos.	Pos.	Fibrils	—	Fibromatous
9	Neg.	Pos.	Fibrils and granules	Fig. 12	Angiomatous
10	Neg.	—	Nil	Fig. 4	Angiomatous
11	Neg.	—	Nil	—	Angiomatous
12	Neg.	—	Nil	Fig. 5	Varicose
13	Neg.	Neg.	Nil	—	Fibromatous
14	Neg.	Neg.	Nil	—	Fibromatous
15	Pos.	—	Granules	—	Fibromatous
16	Neg.	Neg.	Nil	Fig. 19	Fibromatous
17	—	—	Nil	—	Fibromatous
18	Pos.	Pos.	Fibrils	—	Fibromatous
19	Pos.	Pos.	Small plaques	—	Fibromatous
20	Pos.	Pos.	Loose thrombus	Fig. 11	Fibromatous
21	Neg.	Neg.	Nil	—	Fibromatous
22	Neg.	Pos.	Granules	—	Fibromatous
23	—	—	Nil	Fig. 6	Fibromatous
24	Neg.	Pos.	Plaques	Fig. 9	Fibromatous

"Pos." indicates that congo red positive amyloid material or gram positive fibrinoid material is present even if only in minute amounts. A stroke indicates that special staining reactions were not carried out.

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Fig. 9.

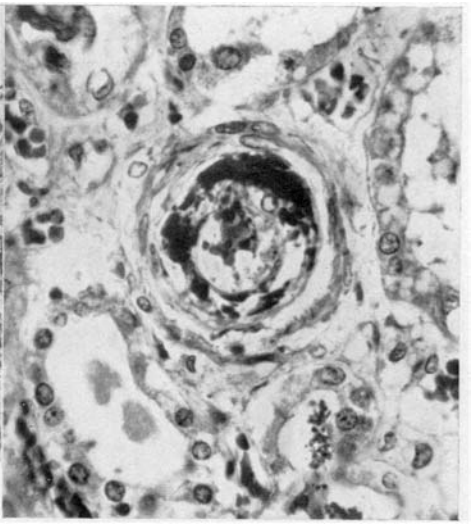

Fig. 10.

Fig. 9.

Case 24. Interstitial hyaline in fibromatous node: fibrinoid material (black) is present in the interstitial tissue and in the wall of the dilated vessel situated under the epithelium. In susceptible individuals this material becomes hyalinized to form the so-called amyloid substance.
Picro-Mallory. Blue-green filter: pan. plate. ×85.

Fig. 10.

Scleroderma. Fibrinoid material in wall of a renal vessel. Figs. 9, 13, 14 and A all show comparable fibrinoid changes in laryngeal nodes.
Picro-Mallory. Blue-green filter: pan. plate. ×475.

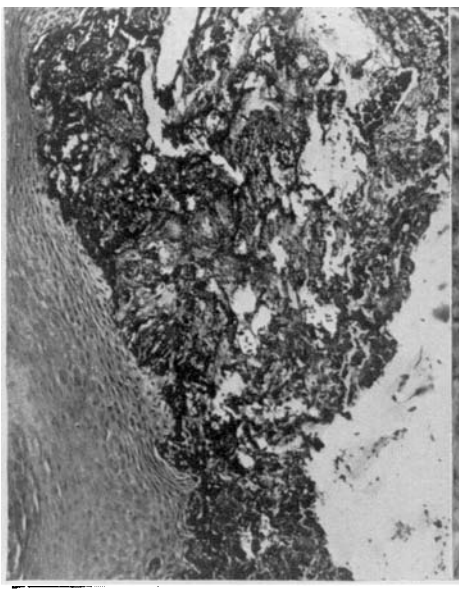

Fig. 11.

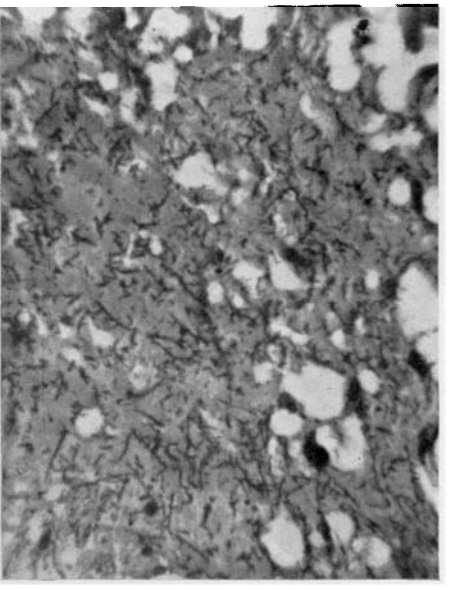

Fig. 12.

Fig. 11.

Case 20. Subepithelial hæmorrhage with fibrin clot: fibrin stained black. Hyalinization of the fibrin produces the so-called amyloid substance, in susceptible individuals.
Gram's stain for fibrin. Minus blue filter: pan. plate. ×100.

Fig. 12.

Case 9. Serous exudate in angiomatous node: black fibrils of fibrin are seen in the greyish serous fluid. This fibrinous exudate is one of the precursors of interstitial "amyloid" substance.
Picro-Mallory. Blue-green filter: pan. plate. ×400.

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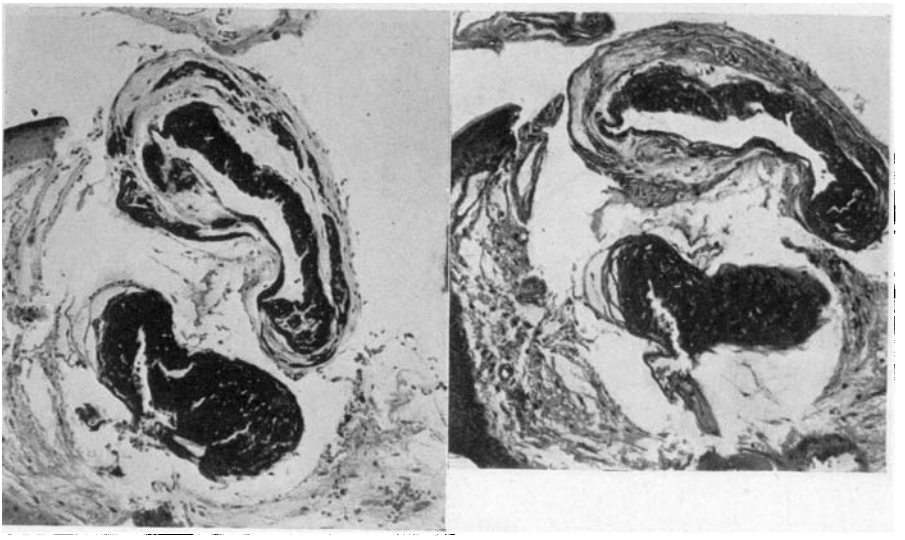

FIG. 13. FIG. 14.

FIG. 13.

Case 32A. Fibrinoid exudate in artery wall: same vessel as in Figs. 14 and A but stained to show the Gram positive fibrin reaction.
Gram's stain for fibrin. Minus blue filter: pan. plate. × 100.

FIG. 14.

Case 32A. Fibrinoid exudate in artery wall: same vessel as in Figs. 13 and A but stained to show the Congo red positive amyloid reaction.
Congo red for amyloid. Blue-green filter: pan. plate. × 100.

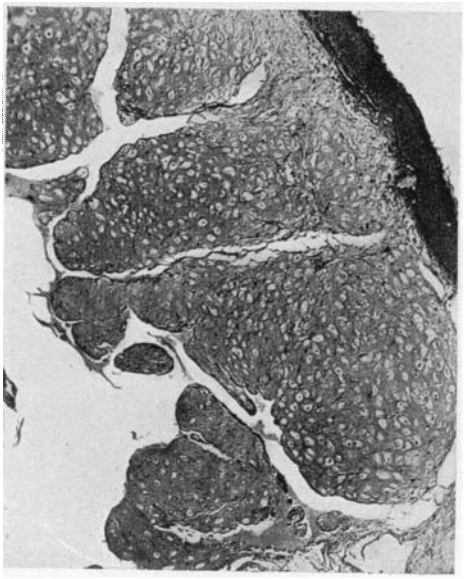

FIG. 15.

Case 38. " Chondroma " of vocal cord: there is a homogeneous matrix in which are a few fibrillary processes and many lacunae containing single cells, a picture simulating cartilage: the whole mass appears to have been a lobulated thrombus and the endothelial lining is still apparent.
Picro-Mallory. Orange filter: pan. plate. × 40.

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All the above changes can be regarded as normal reactions to stress. But a number of these nodes show a type of reaction which is rarely seen in similar lesions in other parts of the body (Tables I and III, Cases 25-37). We refer to the so-called " amyloid tumour " of the larynx for which Ash and Schwartz (1944) prefer the term " hyalin tumour ". This change was aptly described by Wingrove (Wylie, 1924) as " fibro-plastic thickening ", and it is noteworthy that he does not mention the term amyloid. We also do not regard the condition as a true amyloidosis, but since there is now a large literature built around this term (Beavis, 1934 ; Kramer and Som, 1935 ; New and Erich, 1938 ; Eggston and Wolff, 1947 ; Ash, 1942 ; Ash and Schwartz, 1944 ; Holinger and Johnston, 1951) we will deal in more detail with so-called amyloidosis of the larynx under it.

Amyloidosis of the Larynx

There is no general agreement about the nature of amyloid, indeed the chemical composition would appear to vary. Cohen (1943) sums up the position by saying that amyloid appears to be a complex protein and that its deposition depends on a disturbance of protein metabolism. An excellent classification of amyloidosis is given by Reimann, Koucky and Eklund (1935) ; for our present purposes this can be simplified and it may be said that there are three independent conditions in which amyloid or amyloid-like material may be deposited in the larynx :

1. Secondary amyloidosis.
2. Primary amyloidosis.
3. Amyloid tumour in a laryngeal node.

Secondary amyloidosis is the condition which is generally understood when the term " amyloidosis " or " amyloid disease " is used. It is always associated with a chronic infective condition such as tuberculosis or osteomyelitis, or with diseases in which there is hyperglobulinæmia such as multiple myelomatosis. There is a highly typical distribution : the kidneys, spleen, liver, adrenals and intestinal mucosa are principally affected, and the amyloid substance is deposited in the walls of the smaller blood vessels. The specific staining reactions, to be described later, are intense. In myelomatosis the staining reactions are often less distinct, and the anatomical distribution may resemble that of primary amyloidosis. Very occasionally the amyloid substances may be laid down in the larynx but in this case it is merely part of the generalized disease and is of little importance.

Primary amyloidosis is a rare condition in which amyloid-like material is laid down in the stroma of the myocardium, of the skeletal muscles and of the skin, and also in the media of small blood vessels throughout the body. It is " primary " in the sense that it is not associated with chronic infection or other precipitating disease entity. In this condition the

amyloid substance does not give the specific staining reactions as readily, as diffusely, or as strongly as it does in secondary amyloidosis (Lubarsch, 1929). Ætiologically, anatomically and chemically, therefore, it appears to differ from secondary amyloidosis despite the similarities both macroscopical and microscopical: but it is not possible always to distinguish clearly between the primary and secondary forms. Thus Howie (1950) reports a case of amyloidosis of the tonsils, arytenoids and epiglottis having the histological appearances and the anatomical distribution of primary amyloidosis. The patient, however, was suffering from active tuberculosis from which she ultimately died. Again Williams (1952) describes a case of primary amyloidosis in which the staining of the amyloid material was as typical as in secondary amyloidosis and in which the kidneys were extensively and severely affected. The larynx is affected in a high proportion of cases of primary amyloidosis and it would appear that frequently the upper respiratory tract alone is involved.

The literature on primary amyloidosis is both confused and confusing, especially with regard to classification: most reviewers consider only one aspect of the condition. For instance, Beavis (1934) reports five cases of "Local Amyloid Disease of the Upper Air Passages". From his description it is apparent that four of the cases are hyaline laryngeal nodes ("amyloid tumours") and that only Case No. 3 is a true primary amyloidosis; yet all five are considered together as examples of the same entity. Again, the interest of Parkes Weber, Cade, Stott and Pulvertaft (1937) in syndromes, restricted their consideration to the truly generalized primary amyloidosis of which they described a case and found only 10 others in the literature. They intentionally omitted cases in which the amyloidosis was confined to localized areas, e.g. tongue (macroglossia), upper respiratory tract or lungs, yet they regarded these as examples of the "incomplete syndrome". At about the same time Kramer and Som (1935) tabulated from the literature 94 cases of "idiopathic local amyloid tumours" of the upper respiratory tract and added one of their own. They rejected cases of laryngeal amyloidosis occurring in generalized amyloidosis because they thought these should be classified separately. Reimann, Koucky and Eklund (1935) also considered the two conditions to be separate entities. In our opinion, however, a mere variation in the extent of the anatomical distribution does not justify the view that the underlying pathology of localized primary amyloidosis differs from that of the generalized form. Beckert (1917) considered that there was a gradual transition from localized to generalized primary amyloidosis and described a case of amyloidosis of the larynx in which generalized amyloid disease developed several years later. Thus it is desirable that authors describing cases of primary amyloidosis involving the larynx should be careful to state whether the condition is localized or generalized. It is not necessary to postulate two separate entities.

TABLE III

HISTOLOGICAL FINDINGS IN 13 HYALINE ("AMYLOID") LARYNGEAL NODES

Case No.	Staining reactions		Illustration	Type of Hyaline
	Congo red	Gram		
25	Pos.	—	—	Nodular
26	Neg.	Neg.	—	Nodular
27A	Pos.	Neg.	Fig. 8	Nodular
27B	Neg.	Neg.	—	Nodular
29	Pos.	Pos.	—	Nodular
30	Neg.	—	Fig. 7	Diffuse
31	Neg.	Some pos. material	—	Diffuse
32A	Pos.	Pos.	Figs. 13, 14 and A	Diffuse
32B	Neg.	—	Fig. C	Diffuse
34	Pos.	Pos.	Fig. 16	Hæmorrhagic infarct
35	Pos.	Pos.	—	Massive
36	Neg.	—	Fig. B.	Massive
37	Pos.	Neg.	—	Massive

For explanation see Table II.

Positive staining material is much more extensive in the hyaline nodes than in the non-hyaline.

Amyloid tumour of the cords is an unusual condition which affects some laryngeal nodes (Table III and Figs. 7, B and C). It has often, in the past, been confused with primary amyloidosis of the larynx, for instance, the cases of Beavis on which we have already commented. New (1919) stated that if an isolated deposit of amyloid was confined to the vocal cord it could be assumed to be a singer's node that had "undergone hyaline degeneration". This shrewd observation is too often disregarded even today. The amyloid tumour is not a true amyloidosis in that (1) it occurs in single or occasionally in multiple nodes which are confined to the larynx, and it is never part of a generalized disease process; (2) the deposition of the material is nearly always related to intravascular thrombosis; and (3) the reaction of the amyloid-like substance to amyloid stains is never typical, often weak and sometimes absent. We, therefore, prefer the term "hyaline laryngeal node".

Ash and Schwartz (1944) recognized three sites of formation of amyloid in this condition: (1) Interstitial, which results from fusion and hyalinization of "interstitial fibrils", but they fail to comment on the possible nature of these fibrils. (2) Hyalinization of intravascular thrombi. (3) Hyalinization of the vascular walls. As a rule all three types are to be found in each case, but usually one predominates. They say that of 25 specimens selectively stained not one gave the specific amyloid reaction, hence their contention that the term "hyalin" should be substituted for "amyloid". They make no attempt to explain the process of formation of the hyaline material. Eggston and Wolff (1947) regard amyloid deposition in a laryngeal node as a degenerative process.

They mention the frequency of thrombosis but they do not associate it with the deposition of the amyloid-like substance.

The so-called amyloid tumour is usually highly characteristic in its histological appearances : plaques of a hyaline material are laid down in a pre-existing laryngeal node. The plaques may be small and multiple giving a plexiform structure (Fig. 8), or they may be large, in which case they are either homogeneous (Figs. 7 and C) or reticulated (Fig. B). This hyaline material is often present in small amounts, but only when it forms the greater part of the node is it referred to as an " amyloid tumour ". In our series there are 13 cases which contain a large proportion of the hyaline material (Table III, cases 25-37) ; only Cases 30-37, however, are characteristic hyaline or " amyloid " tumours. In the remainder, the hyaline substance, though just as typical in appearances and staining reactions, is less diffuse and all pervading (e.g. compare Figs. 7 and 8). These cases would probably not be described as " amyloid tumours ", but their relationship to and progressive development into such tumours is so obvious that we include them among our hyaline nodes (Tables I and III). It is by studying these less fully formed nodes that an understanding is obtained of the deposition and development of the hyaline material. Histological study of our cases confirms the distribution described by Ash and Schwartz (interstitial, intravascular and in the vessel walls). We find, however, that the amyloid is always related to fibrin or fibrinoid material, which becomes structureless and hyalinized, and that this applies no matter which of the three sites is involved. The intravascular type is always thrombotic (Figs. 7, B and C). In the vessel walls there is a deposition of fibrinoid material (Figs. 9 and A) similar to that seen in polyarteritis nodosa, disseminated lupus erythematosus, scleroderma and other collagen diseases (Fig. 10). In the interstitial tissue we have found three precursor states, and these can be demonstrated even in some non-hyaline nodes (Table II, cases containing fibrinoid material) : (*a*) hæmorrhagic with formation of a loose fibrin clot (Fig. 11) or (*b*) extension of fibrinous exudate through a vessel wall into the adjacent stroma (Figs. 7, B and C) or (*c*) gross œdema of the stroma with serous exudate and deposition of fibrils of fibrin (Figs. 9 and 12). These are presumably the " interstitial fibrils " of Ash and Schwartz and it is noteworthy that they also found " exudation of serum " in the earlier stages of the laryngeal node. Holinger, Andrews, Anison and Johnston (1947) give some excellent photomicrographs showing these fine deposits of fibrin, but amyloidosis is outwith the scope of their paper and is not mentioned. In this connection it is of interest that Barnard, Smith and Woodhouse (1938) in describing two cases of primary generalized amyloidosis say that " as far as the interstitial tissue (amyloid) is concerned the appearance is that which would be found if the organ had been infiltrated by a coagulable fluid which had coagulated ". Further,

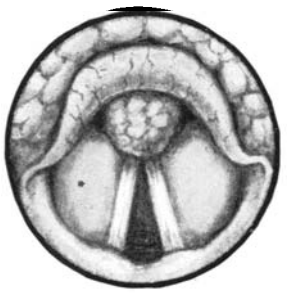

Fig. 16.

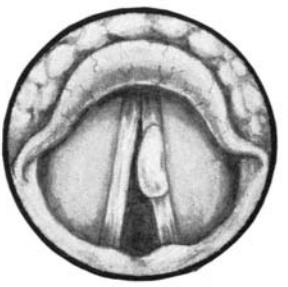

Fig. 17.

Fig. 16.

Case 34. Laryngeal appearances of the hyaline node: the mamillated tumour situated in the midline anteriorly was attached to the left vocal cord. Histologically this is an "amyloid tumour".

Fig. 17.

Case 38. Laryngeal appearances of the "chondroma" of the right cord. (Direct Laryngoscopy)

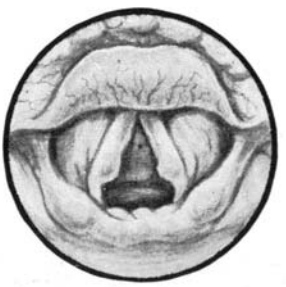

Fig. 18.

Laryngeal appearances of simple bilateral œdema of Reinke's space (after Wessely).

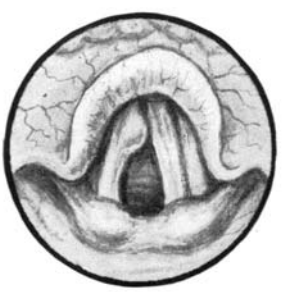

Fig. 19.

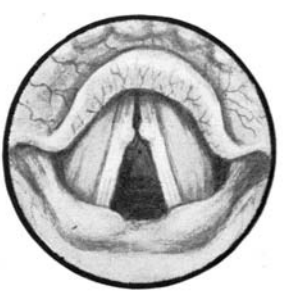

Fig. 20.

Fig. 19.

Case 16. Laryngeal appearances of a simple polypoid node. This case had the histological structure of a fibromatous node.

Fig. 20.

Case 1. Laryngeal appearances in earliest stages of laryngeal node formation. These bilateral nodes subsided without surgical interference.

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they describe their Fig. 7 as a large intramural amyloid deposit, but to us it looks very like a hyalinized thrombus in a vessel. If there is in fact a fibrinoid exudate in the interstitial tissue, in addition to thrombosis of vessels, in primary amyloidosis, it may be that this condition also belongs to the group of collagen diseases. It is thus possible that amyloid tumour of the larynx and primary amyloidosis are more closely related than present accepted evidence would indicate.

Staining Reactions

The fibrinoid material in the laryngeal node, as in polyarteritis nodosa and other conditions in which it is a feature, gives the staining reactions of fibrin: it is Gram positive (Fig. 13) and with the trichrome stains of Mallory type it is fuchsinophil (Fig. B). The picro-Mallory technique of Lendrum and McFarlane (1940) is a particularly effective method of demonstrating it ; the erythrocytes are stained bright yellow and contrast sharply with the red fibrinoid material. As the fibrinoid deposits become hyalinized to form the so-called amyloid substance they tend to lose these staining reactions. With the trichrome stains they become bluish or purplish (Fig. C) instead of red, and the Gram positive reaction becomes less intense ; but it is rare to find a plaque that does not contain some Gram positive areas. With material as small as laryngeal nodes chemical analysis is not possible, so the identification of amyloid is entirely dependent on histological methods. True amyloid is structureless, homogeneous and waxy in appearance: it takes on a blue-black colour with iodine and sulphuric acid, gives a metachromatic reddish-purple reaction with methyl violet and stains bright red with congo red. The hyaline material of laryngeal nodes is structureless and homogeneous but not really waxy in appearance. The specific staining reactions are often but by no means always positive (Figs. 8 and 14) and are never as intense as in secondary amyloidosis. We have used congo red to identify " amyloid " in our cases because, although it is less specific than iodine or methyl violet, it is more permanent, a point of considerable importance when the number of sections available is limited.

Development of Hyaline Laryngeal Nodes

There are four possible headings under which the production of hyaline material in laryngeal nodes can be considered :

(1) Excessive or prolonged trauma or irritation.
(2) Hæmostasis and thrombosis in dilated blood vessels.
(3) Hæmorrhage and thrombosis due to twisting of the pedicle of a pedunculated tumour.
(4) Idiopathic or hypothetical.

(1) As already mentioned trauma is the most important single entity in the production of all laryngeal nodes, but it cannot be more than a contributory cause of fibrinoid exudate. A study of Table I, Cases 25-37

shows that the majority of hyaline nodes do not have a longer history than the other types of laryngeal node. Nor is there a history in these cases of unusually vigorous voice abuse.

(2) Hæmostasis in dilated vessels is an attractive possibility because all cases are associated with thrombi and in many there are large, dilated angiomatous vessels (Figs. 7, B and C). In others, however, the vessels are small (Fig. 8) and this explanation would not account for the interstitial or the intramural fibrinoid change. Hæmostasis and thrombosis is therefore not the universal explanation but a possible one in certain cases.

(3) Twisting of the pedicle can only occur in pedunculated tumours yet most of the amyloid tumours are sessile. Torsion always results in engorgement of vessels along with hæmorrhage, but no gross engorgement is discernible in most of our cases, and hæmorrhage although often present is not invariable and is rarely gross. Nevertheless we consider that Case 34 may well belong to this category.

(4) We are left, then, with no satisfactory general explanation of the formation of the hyaline node. There can be no question that irritation is the primary cause as with laryngeal nodes in general, but there must in addition be some peculiarity of individual reaction to the irritation. As we have already pointed out, the eccentric fibrinoid degeneration of vessel walls (Figs. 9, 13, 14 and A) and the perivascular fibrinoid exudate (Figs. 7, 9 and B) are very suggestive of similar lesions in the collagen diseases (Fig. 10). It may be that these lesions are very localized expressions of Selye's General Adaptation Syndrome, and that the continued minimal trauma of phonation, in susceptible subjects leads to hyalinization instead of absorption of the fibrin. For example, we have recently seen, in this laboratory, a papillomatous squamous carcinoma of skin surrounded by a localized ring of amyloid-like material which gives the staining reactions of fibrin as well as those of amyloid. It seems probable that repeated small traumata to the warty tumour produced a fibrinoid exudate which, by a peculiarity of individual reaction as in amyloid tumour of the vocal cord, has become hyalinized instead of being absorbed. Again, two of our patients with amyloid nodes (Cases 27A and 32A) later developed second nodes and these also were found to be of amyloid type (27B and 32B) showing that these two patients were susceptible to this peculiar change. (It should be noted that all cases of laryngeal node were classified histologically by one of us (J.E.C.) under serial numbers and not under patients' names. It was not known at the time that Cases 27B and 32B were relapses yet each had been allocated to the same group as its antecedent lesion.)

That amyloid hyalinization is not the only fate which overtakes massive thrombi in laryngeal nodes is shown by our last case (No. 38). In Fig. 15 the outline of the original lobulated clot with its endothelial

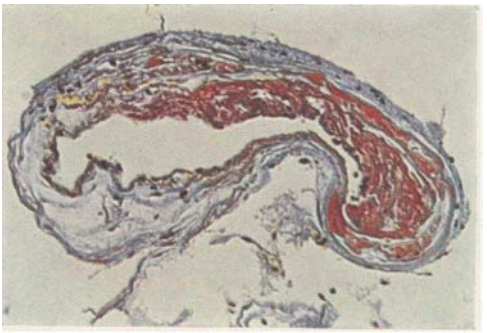

FIG. A.
Case 32A. Ektachrome photomicrograph of fibrinoid exudate in artery wall: the fibrinoid material is stained red, erythrocytes yellow and fibrous tissue green; smooth muscle has almost entirely disappeared. This vessel lies just outside the area of the "amyloid" tumour. Same vessel as in Figs. 13 and 14.
Picro-Mallory. ×125.

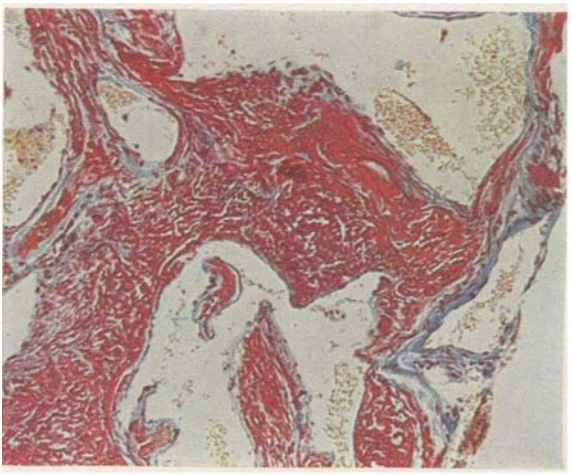

FIG. B.
Case 36. Ektachrome photomicrograph of early so-called amyloid tumour: at this stage the relationship of the "amyloid" to fibrinoid is more obvious. Greatly dilated vessels are seen containing erythrocytes, stained yellow, and small fibrin clots stained red. The hyalinizing red stained interstitial fibrinoid (amyloid) between the vessels is very obvious. Compare with Fig. C.
Picro-Mallory. ×100.

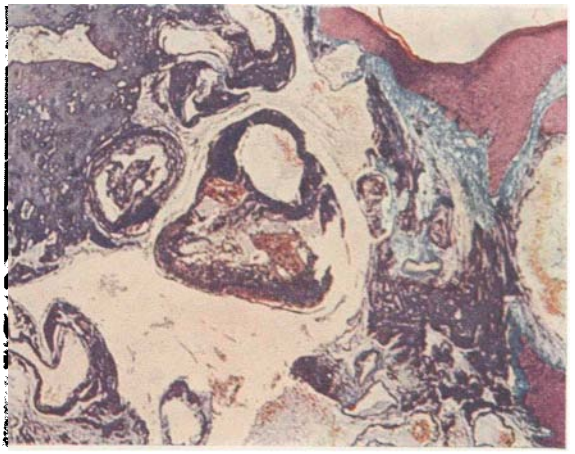

FIG. C.
Case 32B. Ektachrome photomicrograph of fully developed so-called amyloid tumour: the fibrinoid material in this case is completely hyalinized (stained bluish purple) and closely resembles true amyloid. Erythrocytes are stained orange. This tumour was a recurrence in the same patient as Case 32A—Figs. 13, 14 and A.
Picro-Mallory. ×42.

lining can still be seen. The mass of the thrombus has been converted into a chondroma-like tumour with a homogeneous cartilaginous matrix complete with lacunae containing cells. Staining reactions for fibrin, amyloid and even mucin were negative.

Treatment

In the early œdematous stages, as in Case 1, laryngeal nodes will resolve without surgical intervention. Rest to the voice should be as complete as possible for three to four weeks. In appropriate cases whispering may be allowed. In the meantime arrangements should be made for the patient to receive instruction in proper voice production. For instance, many patients, especially women school teachers, strain their voices by speaking stridently and have to be taught to speak in a lower key. The singer who develops nodes may be found to be a natural baritone attempting to sing as a tenor, and will have to be trained again in his proper register. Children must be restrained from shouting or screaming and inquiries must be made about singing at school (Moure, 1896). A search must be made for any nasal obstruction or sepsis in the upper air passages, and appropriate treatment arranged. Smoking, especially of cigarettes, should be restricted.

The larger nodes are removed by direct laryngoscopy. Cup-ended laryngeal forceps are employed, and great care must be taken to ensure that the tumour only is removed ; a " bite " must not be taken out of the edge of the cord as this would result in permanent damage to the voice. For accurate work careful anæsthesia is of paramount importance. To attempt removal of a laryngeal node in a subject whose cords, when touched, approximate in spasm is to court disaster. We find that a combination of local and general anæsthesia is usually preferable.

Complete removal of the nodule is by no means the termination of treatment. In all cases every effort must be made to eliminate the underlying vocal strain in order to minimize the risk of recurrence. In our group of 35 patients, three at least had relapses (Cases 10, 27 and 32).

We have used neither A.C.T.H. nor cortisone in the treatment of our cases and do not intend to use them. Although we regard the hyaline node as being a localized manifestation of the Adaptation Syndrome, we would emphasize that the changes are so far advanced as to be irreversible. A.C.T.H. and cortisone might be of use, in susceptible persons, to prevent the formation of hyaline nodes, but it would be medically unsound and economically impossible to give these drugs to all singers and school teachers in the hope of preventing a few laryngeal nodes !

Summary

Clinical and histological findings are presented of 38 laryngeal nodes from 35 patients. The so-called simple tumours of the larynx, namely polyps,

granulomas, fibromas, myxomas, angiomas, varices and singers' nodes are not true tumours. They indicate differing individual reactions to irritation, and are not related to the duration or severity of voice abuse.

A review of the literature on amyloidosis is given and a simplified classification is suggested. The localized " amyloid tumour " of the larynx is not a true amyloidosis, and the term " hyaline tumour ", or better, " hyaline laryngeal node " is preferred. Some nodes contain thrombi and fibrinoid exudate which, in susceptible subjects, become hyalinized to form the " amyloid " substance. The staining reactions of the fibrinoid material, and its distribution closely parallel those of the collagen diseases, to which group we believe the hyaline nodes belong.

Zusammenfassung

Die Autoren berichten über die klinischen und histologischen Befunde in 38 Fällen von Tumoren der Stimmbäder in 35 Patienten.

Die sogenannten einfachen Geschwülste des Kehlkopfes, nämlich Polypen, Granulome, Fibrome, Myxome, Angiome, Varizen und Sangerknötchen sind keine Geschwülste im wahren Sinne das Wortes. Sie stellen verschiedene individuelle Reaktionen zu Reitzzuständen dar und sind nicht abhängig von der Dauer oder der Schwere des Stimmverbrauches.

Eine Literaturübersicht der Amyloidose ist gegeben und eine vereinfachte Klassifizierung wird vorgeschlagen. Die lokale " Amyloidgeschwulst " des Kehlkopfes ist keine wirkliche Amyloidose und die Bezeichung " hyaline Geschwulst " oder besser " hyaliner Kehlkopfknoten " ist vorgezogen. Manche Knötchen enthalten Thromben und Fibrinexsudate, welche in empfindlichen Personen hyalinisiert werden und die Amyloidsubstanz bilden.

Die Farbreaktionen des Fibrinmaterials und ihre Verteilung sind ähnlich den Erkrankungen des Kollagens, zu welchen die hyalinen Knötchen nach der Meinung der Autoren gehören.

Résumé

Les observations cliniques et histologiques sont obtenues par 38 tumeurs des cordes vocales de 35 malades.

Les ainsi-nommées tumeurs simples des larynx, c'est-à-dire les polypes granuleux, fibreux, myxomateux, angiomatiques, variqueux et noeuds de chanteurs ne sont pas de vraies tumeurs.

Ils indiquent différentes réactions individuelles à l'irritation, et n'ont aucune relation à la durée on à la sévérité de l'abus de la voix.

Une revue de la litterature sur l'amyloidose est donnée, et une classification simplificé est proposé.

Une " tumeur amyloide " locale du larynx n'est pas une vraie amyloidose, et l'expression " tumeur hyalinique " ou, " noeud hyalinique du larynx " est préférable.

Certains noeuds contiennent des exudates thrombiques et fibreux qui chez certains sujets susceptibles se transforment en une substance " amyloide ".

Les réactions de couleur du material fibreux et sa distribution se rapprochent parallèlement à celles des maladies collageniques.

C'est pourquoi que les auteurs considérent les noeuds hyaliniques appartenir à la groupe collagénique.

Acknowledgments

We are glad to be able to express our gratitude to Mr. Alex. Strang and Mr. T. O. Howie for permission to publish their cases; to Mr. Forbes M. Walker for the clinical notes on, and permission to publish Cases 7 and 24; to Dr. W. B. Davis for much helpful advice and friendly criticism and for permission to use the pathological material; to Mrs. C. Moorhead Hamilton for the laryngeal drawings, and to Mr. Hugh Gray, Clinical Photographer of this Hospital for processing all the photomicrographs including the ektachromes.

Appendix

The following cases with their pathological reports illustrate the inter-relationship of the various types of laryngeal node.

CASE 1 (Fig. 20). Bilateral œdematous nodes.

M.M., female, aged 24, school teacher, complained of hoarseness of 18 months' duration. She had been teaching for 3 years, and had to control a large class of "noisy, rude boys". The classroom was dusty and dry. At first the loss of voice was intermittent, and eventually became so bad that she was absent from school on several occasions. Examination of the larynx showed a small node on each cord at the junction of the anterior and middle thirds (Fig. 20). These nodes came into contact on phonation, and prevented the complete approximation of the posterior parts of the cords.

Treatment consisted in complete voice rest for 3 weeks and instruction in speaking in a lower and less strident key. There was some nasal obstruction due to turbinal hypertrophy, which was improved by cautery. Three months later her voice was normal. Mirror laryngoscopy showed that the nodes had vanished, and that the cords approximated perfectly.

CASE 2 (Fig. 2). Œdematous node: fragmentation of elastic tissue.

R.S., male, aged 69, an engineers' pattern maker, complained of hoarseness of only 8 weeks' duration. There had been no previous trouble with the voice, although for some years he had worked in noisy surroundings, and had frequently to shout.

Examination showed an apparent prolapse of the left ventricle, the left cord being round, moist and succulent looking.

Histological examination showed gross sub-epithelial œdema with rupture of elastica (Fig. 2). There was no inflammatory exudate.

CASE 3 (Fig. 3). Granulomatous polyp.

W.G., male, aged 53, master baker, complained of hoarseness of about 2 months' duration. He gave a history of frequent bouts of coughing with scanty spit, occasionally blood stained, during the past 2 years. Examination showed a nodule on the anterior end of the right vocal cord which was removed.

Histological examination showed subepithelial granulation tissue with marked œdema and lymphocyte and plasma cell infiltration (Fig. 3).

CASE 7. Bilateral nodes: left, granulomatous; right, fibromatous.

W.McC., male, aged 52, cattle drover, complained of hoarseness of three weeks' duration. He had no pain but suffered considerable discomfort from laryngeal obstruction. Examination revealed large pedunculated polyps on the anterior thirds of each cord.

Histological examination of the node from the left cord showed a subepithelial infiltration of plasma cells, lymphocytes and fibroblasts. The node from the right cord was purely fibromatous with no inflammatory exudate: it contained considerable areas of interstitial fibrinoid.

CASE 10 (Fig. 4). Angiomatous node. Recurrence within 12 months.

H.F., male, aged 47, travelling salesman, complained of repeated attacks of hoarseness over the past two years, the attacks lasting several weeks. There had been complete loss of voice for the past month. He had to talk a lot in the course of his work, and he was a fairly heavy drinker and smoker. He had had similar hoarseness 20 years before. Examination showed a nasal septum deviated to the right, the mucosa being red and congested. On laryngoscopy, a small growth was seen lying in the anterior commissure, attached to the anterior third of the left cord. It was reddish in colour and about the size of a millet seed. Removal was carried out with cup-ended forceps.

Histological examination showed a highly vascular node, both young and mature vessels being arranged in a plexiform structure (Fig. 4). The stroma was loose and œdematous in places.

A year later this man was again hoarse and had a tiny node on the anterior commissure, which was removed but not examined histologically.

CASE 12 (Fig. 5). Laryngeal varix.

E.B., female, aged 41, housewife, complained of intermittent hoarseness, lasting four months. No special vocal strain. Examination showed a curious heaped up appearance of the anterior third of the right vocal cord. It was rather a diffuse swelling compared with the usual well defined polyp. This was removed and a month later the voice was clear and giving no trouble.

Histological examination showed an angiomatous polyp (Fig. 5). The greatly dilated vessels contained albuminoid fluid and a few leucocytes. There was some œdema of the surrounding fibrous tissue and considerably increased vascularity, but inflammatory exudate was absent.

CASE 16 (Fig. 19). Fibromatous polyp, partly myxomatous.

A.McF., female, aged 53, a housewife, complained of gradually increasing hoarseness for two years. She was a garrulous type, but did not admit to any vocal abuse! Examination showed a small colourless polyp on the right vocal cord at the junction of the anterior and middle thirds (Fig. 19).

Histological examination showed a fibromatous tissue which in places was loose and myxomatous. There was no œdema and no increased vascularity.

CASE 24 (Fig. 9). Fibromatous node: interstitial and vascular fibrinoid.

S.B., female, aged 31, housewife, complained of hoarseness of seven years' duration. The hoarseness had been intermittent at first, but constant for the

past two years. She denied any vocal abuse. Examination revealed a small tumour with irregular surface on the anterior end of the left vocal cord.

Histological examination showed increased vascularity with dilatation of many vessels, the largest of which contained thrombi. Fibrinoid material was present in the vessel walls and in the interstitial tissue (Fig. 9).

CASE 27, A and B (Fig. 8). Hyaline laryngeal nodes; nodular type.

J.S., male, aged 41, painter, complained of hoarseness of two years' duration. He did not admit to any vocal abuse. Examination a year previously had shown only a granular pharyngitis. On laryngoscopy a polyp was seen lying in the anterior commissure. It appeared translucent and hung from the anterior end of the right cord into the subglottic region. On phonation the tumour was forced up between the cords and lay on their upper surfaces. Six months later another polyp was removed from the left cord.

The histological appearances of these two nodes were similar. They consisted of nodular masses of hyaline (amyloid) material which appeared to be hyalinized thrombi in a plexiform angioma. These masses gave a positive reaction for amyloid (Fig. 8) and there were still some traces of Gram positive fibrinoid material.

CASE 32, A and B (Figs. 13, 14, A and C). Hyaline laryngeal nodes: diffuse type.

P.C., male, aged 40, aero-engine inspector, was first seen in October, 1948, complaining of hoarseness of one year's duration. His work entailed much shouting above the noise of aeroplane engines which were being tested. Examination showed a small nodule on the anterior third of the right vocal cord. It did not differ in colour from the remainder of the cord. Removal with cup-ended forceps presented no difficulty.

The patient remained well for two years, and then became hoarse again. He still subjected his voice to considerable strain. Examination showed a recurrence of the node on the right cord. This time it was pink in colour.

Histological examination showed similar changes in each of these nodes. There were wide angiomatous vessels containing hyalinized thrombi and massively hyalinized interstitial tissue (Fig. C). Amyloid staining in Case 32A was positive but negative in the recurrence. In addition the earlier node contained a small artery showing the eccentric fibrinoid exudate which is so typical of the "collagen diseases" (Figs. 13, 14 and A).

CASE 38 (Figs. 15 and 17). "Chondroma" of the vocal cord.

This case is included because, although the histological appearances are those of a chondroma, it is likely that it is really a laryngeal node which has undergone pseudo-chondrification. This view is supported by the history of vocal abuse, the site and clinical appearance of the tumour, and by the soft consistency of the tumour at operation.

R.B., male, aged 54, an engineer, suffered from hoarseness of two years' duration. At first this was intermittent, the voice being quite strong between attacks, but for three months aphonia had been constant. Working in a

machine shop, he had had to shout above the noise, and admitted to considerable vocal strain.

Mirror laryngoscopy showed a pale, œdematous swelling of the right cord, but as the appearances were inconclusive, direct examination was carried out. Lying on the surface of the right cord was a sausage-shaped tumour, so translucent that at first sight it was difficult to distinguish from a blob of mucus (Fig. 17). On probing, it was found to be attached to the medial border of the cord, about the junction of the anterior and middle thirds. The translucence, and the fact that it lay in the long axis of the cord, provided an illusion in the mirror of the cord itself being œdematous. With cup-ended forceps the mass was completely removed.

Histological examination showed a chondroma-like tumour (Fig. 15). It consisted of a smooth homogeneous matrix with a few fibrillary strands, and large numbers of lacunae each containing a single cell, a picture exactly like hyaline cartilage. Special staining methods failed to reveal mucus, elastic fibres, amyloid or fibrin. The mass as a whole resembled a lobulated thrombus and even appeared to be lined by an endothelium which passed down into the fissures. It is probable that this has been a laryngeal node which has undergone massive thrombosis, the thrombus having been converted into a cartilage-like mass.

This case was shown to the Scottish Otological Society in December, 1949.

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RHINITIS CASEOSA

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RHINITIS CASEOSA is an uncommon, if not rare, condition, and the cases which have been described in the literature have been reported either as isolated instances or in very small series. Few examples may be seen in a lifetime, even by those with wide experience, and for this reason the case reported here may be of interest.

The condition is characterized by the formation of masses of caseous material in the nose and sometimes in the maxillary antrum, though it appears that the antrum is rarely if ever involved alone. In fact, there is only one possible case of this reported in the literature (Cartaz, 1908). As a rule only one side of the nose is affected, there being no more than three cases described with bilateral involvement (Nyulasy, 1892; Schleicher, 1890; Laocarret, 1898). The condition is accompanied by nasal discharge which is often fœtid, and in the later stages by intranasal and extranasal deformity due to destruction and expansion of the tissues by pressure of the caseous mass.

Historical

Rhinitis caseosa was first described as a distinct clinical entity by Duplay in 1868, though the date is more often reported as 1874, when he correlated the reports of five cases described in the literature in the previous 20 years. A few isolated cases were described over the next 20 years, and these were reviewed by Potiquet in 1889. Reports of several series of cases, usually very small in number, followed thereafter from time to time, principally during the last decade of the nineteenth century and the earlier years of the twentieth. The literature on the subject was reviewed in 1936 by Meyersburg, Bernstein and Mezz, and again an excellent review with a very full and comprehensive bibliography was produced by Polson in 1942, analysing the reported cases. This last exhaustive review of the literature revealed information of some kind concerning about 158 cases, including those only briefly mentioned by various clinicians in discussion, but detailed reliable records were available of only 75 cases. Since then the only reference to the subject seems to have been by Farrior in 1942, who described one of the only three cases presented in the American literature.

Aetiology

Various theories have been postulated from time to time to account for the condition, the principal ones being as follows:

1. *Tuberculosis.* There was thought to be a causal relationship between pulmonary tuberculosis and the pressure of the caseous material in the nose, the caseation being supposed to be due to decay of the nasal epithelium arising from infection by tubercle bacilli. Support was lent to this theory by the description by Cozzolino (1884) of an organism resembling the tubercle bacillus in the caseous material.

2. *Syphilis.* The condition was also considered to be in some way connected with tertiary syphilis and the destruction caused by the formation and breaking down of gummata.

3. *Erysipelas.* It was suggested by Duplay (1868) that extension of erysipelas from the face to the nose caused epithelial desquamation with the consequent collection of a mass in one of the nasal passages, a theory which was evidently founded on the coexistence of the two conditions in one case, and which received little support.

4. *Cholesteatoma.* The condition was later thought by Cozzolino (1889) to be analogous to aural cholesteatoma, and for several years it was referred to under this description. Histological and chemical examination by Kelson (1904) showed definite differences between the two conditions, notably the absence of any cholesteatomatous membrane or the typical laminae and the absence of cholesterin from the nasal material, but the theory retained its popularity for the first two decades of this century.

In supporting the cholesteatoma theory, Heimendinger (1907) and Winkler (1910) each separately described the condition as arising from two independent causes. The first, " true cholesteatoma ", was held to be a developmental anomaly of fœtal origin where some disturbance of the normal embryonic growth caused proliferation of epidermis into the bones of the face. This later acted as a nidus for the formation of a cholesteatomatous tumour, deep seated and involving the overlying bone, which was assumed to be a factor in causing subsequent suppuration.

The second type, " false cholesteatoma ", was thought to be a product of existing sinus suppuration, and to be due to the encroachment into the sinus cavity of diseased atrophic nasal mucosa which had undergone metaplasia to the squamous type. It was assumed that the squamous epithelium, meeting the inflamed ciliated epithelium of the sinus cavity, was unable to unite with it and was thrown off and died, another layer soon taking its place. This condition of " false cholesteatoma " was said to be more superficial than the " true " type, involving only the mucosa and not the adjacent bone.

5. *Specific infection.* Infection by the strepthrix alba was believed by Massei (1896-7) to be the specific cause of the disease, and the presence of the organism was demonstrated by several other observers, but the fact that many other workers failed to find the streptothrix in material from their cases suggested that in cases where it was present the organism was a secondary invader.

Other bacteria and fungi have also been demonstrated in the material from time to time, usually in single cases or in extremely small series.

6. *Polyp degeneration.* It was suggested by Bories in 1889 that the caseous material was the product of degeneration of mucous polypi in the nose, as he found similar appearances on microscopic examination of the caseous material and of polypi which had undergone degeneration. This theory met with little acceptance, however, and furthermore the presence of polypi is by no means an invariable accompaniment of caseous rhinitis in an analysis of the reported cases as carried out by Polson. Polypi were a prominent feature in the case described below, and acted as an obstruction which completely concealed the mass of caseous matter, and thus doubtless contributed to its collection.

7. *Suppuration complicated by obstruction.* The most generally accepted view today is that the phenomenon of caseous rhinitis is due to the retention in the nose and sinuses of the products of suppuration owing to some form of obstruction, under certain conditions, bacteriological and physical, which favour its inspissation. The process was described by Avellis (1900) as " Verkasung ", or metamorphosis of the secretion into a caseous mass, which he assumed to take place during the course of chronic sinusitis when the lining membrane recovered gradually till it was able to overcome the attacks of the infecting organism. The secretion contained in antrum, being prevented from escaping by some form of mechanical obstruction, became stagnant, lost its moisture, and fatty degeneration of the pus cells seemed to result, so that after a while the mass became more or less solid with the consistency of soft cheese.

The factor of obstruction by some means or other has been mentioned in a large number of the reported cases, though not always emphasized, as also has the presence of chronic suppuration in the nose. The suppuration has been described in several cases as being due to a foreign body or a rhinolith, or in others to chronic sinusitis. As is apparent from the various theories held in the past regarding the cause of the condition, it has not always been clear whether the suppuration constituted a causative factor or an effect of the caseous rhinitis.

Polson found that sinusitis was noted as being present in only 21 per cent. of cases, too low an incidence to permit the conclusion that it is an important factor, but it may be pointed out that in many cases the condition of chronic sinusitis could easily have been hidden and overlooked in the presence of the grosser phenomenon of the caseous mass. He also drew attention to the fact that sinus suppuration is common, and that therefore rhinitis caseosa should be commoner than it is if the two conditions have a causal relationship, but it must be remembered that in addition to suppuration and obstruction to the escape of secretions there must be other favourable conditions present for the development of rhinitis caseosa. Inspissation must occur, probably due to stagnation

followed by evaporation of the moisture of the secretion by air currents, and presumably the bacteriological conditions must be exactly right. These favourable conditions are largely hypothetical, and the unlikelihood of all the necessary factors occurring together would account for the rarity of the disease.

The cause of mechanical obstruction to the secretion has varied in the cases which have been sufficiently fully described for this to be assessed. Polypi have been present in many cases, but by no means invariably. In other cases large septal crests or deflections have been blamed, or foreign bodies, granulation tissue, neoplasm situated in the anterior part of the nose, gumma, septal hæmatoma and perichondritis, hypertrophic changes in the mucosa, especially of the turbinates, have all been present to cause obstruction. In the case reported here there seemed to be no doubt but that the mass of polypi in the right ethmoidal region constituted a most effective obstructive factor.

Pathology

The morbid changes found in the nose are as various as the numerous conditions described above as giving rise to obstruction and suppuration.

The caseous material itself has been described as yellowish or grey, semi-solid, resembling the contents of sebaceous cysts, or likened to rotten cheese or putty. Its quantity varies greatly in different cases, and in most cases its offensive odour has drawn comment. Specimens examined histologically have been found to consist chiefly of pus cells and amorphous debris. Fatty cells and some crystals have occasionally been described, and a trace of cholesterin has been found on a few occasions.

The organisms discovered at different times have varied widely, and include streptothrix, fungi and other bacteria as already discussed under the heading of ætiology.

The pathological changes in the mucosa and structure of the nasal passages depend on the degree of suppuration which is present and on the duration and extent of the condition. They vary from lesser degrees of inflammatory infiltration and thickening in early cases to complete mucosal degeneration and destruction, while in the later stages of the disease the accumulation and expansion of the caseous mass leads to slow destruction by pressure of the adjacent soft tissues and bone resulting in external deformity and fistulæ.

Symptomatology

It is important to bear in mind that rhinitis caseosa is not a disease in itself, but is an incidental development in the course of nasal sepsis when other factors such as obstruction and inspissation of the secretions are also present. The history is most likely to be one of increasing unilateral nasal obstruction, often associated with offensive discharge which may

either be purulent or serous. There may be frequent exacerbations of infection of the upper respiratory tract, but on the other hand the infection may be a low grade one and the general symptoms may be confined to vague complaints such as headache and lassitude arising from the chronic sepsis.

On examination of the nose the caseous material may be directly visible through the anterior nares, or it may be seen on posterior rhinoscopy obstructing the choanæ. In other cases the mass may not be immediately apparent owing to obstruction of the view by a septal deflection, hypertrophied turbinates or whatever abnormally caused retention of the secretion in the first place. In the case described below for example, the caseous material was entirely concealed from view by the mass of ethmoidal polypi, and was not discovered until these had been removed at operation.

In addition to the caseous material, nasal examination will reveal signs of chronic suppuration, whether arising from the paranasal sinuses or from some source in the nasal passages themselves such as a foreign body. A purulent or serous discharge will usually be found either in the nares or postnasal space, the nasal mucosa may be engorged or hypertrophied, and crusting may be present. Foetor of the breath appears to be a common accompaniment of the condition, though it is by no means invariable and it was not present in the case reported here.

In the later stages of rhinitis caseosa when the mass has caused extensive destruction by erosion various degrees of facial disfigurement may be seen, such as swelling of the face, broadening or collapse of the nose, external fistulæ near the inner canthus of the eye, or ulceration through the palate. Such extreme damage will not arise, however, unless the nasal condition has been neglected over a long period, and it is probably unlikely to be seen today. While the case with external deformity is usually one of long standing and slow development, however, it is not always so, and Farrior's case was remarkable for the comparatively rapid development of the whole condition. There was apparently only one year between the beginning of any nasal complaint at all, in the form of an attack of acute sinusitis which failed to resolve satisfactorily, and the development of noticeable external deformity which took the form of unilateral swelling and broadening of the nasal bridge, and which remained permanently after the disease had been dealt with.

Treatment

Once the caseous mass has been discovered it may be dealt with by simple means, such as scooping out with a spoon followed by repeated irrigation to ensure its complete removal, and from the published descriptions of several cases it seems that adequate removal of the mass and of any obvious obstruction in the nose is not followed by reaccumulation of

subjectively and on examination, and she felt that her general health and wellbeing had benefited considerably.

My thanks are due to Mr. Stephen Young, in whose wards the patient was treated, for permission to publish the details of this case.

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relevant publications leaves some doubt as to the origin of these discrepancies and the question still seems to be unresolved.

In Great Britain the devising of adequate standards for audiometry may be said to have received its impetus when in 1939 the Section of Otology of the Royal Society of Medicine approached the British Standards Institution with a view to the preparation of suitable performance specifications. This work was interrupted by the war but is now approaching completion. In the same connection the National Physical Laboratory was requested, through the medium of the Electro-Acoustics Committee of the Medical Research Council, to establish the necessary basic standards of measurement, and the Laboratory undertook a programme of work, some parts of which are still in progress, with this end in view. The investigation described in the present paper is a part of this programme and was undertaken with the object of providing additional threshold data obtained under accurately controlled and reproducible conditions, which it was hoped would assist in the establishment of a standard reference zero. A brief and preliminary account of the work, in which reference is also made to other work at the National Physical Laboratory bearing on audiometry, has appeared in the Proceedings of the Second International Conference on Audiology held in London in 1949.

Experimental Procedure

An important consideration which must be faced at the outset of an investigation of the present nature is the choice of subjects. In this connection the Laboratory has followed the recommendations of the Electro-Acoustics Committee of the Medical Research Council in Appendix VIII of its Report Ref. 261. The tests were thus restricted to otologically normal subjects between the ages 18 to 25 years inclusive.

It might, of course, be suggested, as an alternative, that the subjects should be a random sample of the otologically normal population, with a similar distribution of ages. This, however, is open to objection on the ground that, even if precautions were taken to exclude subjects with obviously defective hearing or ear disease, the selection would certainly include persons with high-tone deafness of the kind normally associated with advancing age. It seems natural to regard the primary function of a pure tone audiometer as being the comparison of the hearing of a subject with a standard representing hearing which is free from impairment, and it thus seems more satisfactory to adopt as a standard of reference a group of subjects whose auditory state is the most likely to be free from impairment of any kind.

In accordance with the above considerations, the prospective subjects, consisting of just over 100 members of the Laboratory staff, were submitted to an otological examination to ensure that no subjects were included having symptoms or signs of ear disease, or wax in their external

auditory canals. Only subjects judged as normal at this examination were accepted for the threshold test, which was successfully completed by a total of 99, comprising 45 men and 54 women.

The test consisted of the measurement of the r.m.s. voltage at the terminals of a type 4026A earphone corresponding to the least sound pressure audible to the subject, and was carried out with pure tones at 14 frequencies, viz. 80, 125, 250, 500, 1,500, 2,000, 3,000, 4,000, 6,000, 8,000, 10,000, 12,000 and 15,000 c/s. Precautions were taken to ensure the adequate suppression of harmonics or other extraneous components in the test tone, and all such components—which in any case were at least 40 decibels below the level of the fundamental—were well below normal threshold. The circuit included an on-off switch by which the tone could be interrupted by an independent operator; this switch was arranged to have a time constant of about 0·2 sec. in which circumstances no clicks or transients were audible on switching the tone on or off. All the tests were conducted in a highly silent absorbent room in which the ambient noise level was too low to permit of measurement with a sound level meter and was estimated to be below 20 phons. Subjects of normal hearing wearing the earphones were not conscious of any disturbing sounds other than those of physiological origin or due to their own movements. The test was carried out separately on each ear, the earphone being worn on a headband of the type normally supplied with this pattern of earphone and having at its other end a dummy earphone of the same type. The force of application exerted by the headband, when properly adjusted to give a comfortably firm fit on individual subjects, varied in practice over a range from 350 to 500 gm., averaging about 450 gm. In order to avoid auditory fatigue the tests on each subject were carried out in two sessions separated by several days at each of which seven frequencies were covered on the two ears. Each session was divided into two parts with an interval of several minutes whilst the subject changed from one ear to the other, and a short interval was allowed between successive frequencies.

In determining the threshold the tone was first reduced by the operator in steps from an easily audible level until it became inaudible, and then increased from a level well below threshold until it again became audible. The threshold value was taken as the mean of the two lowest levels at which the tone was consistently heard by the subject.* At each level the tone was switched on and off by the operator several times, for successive durations of the order of 2 secs. with similar intervals, the subject being instructed to signal by flashing a light when the tone was heard. In the first place the threshold was located approximately by varying the intensity in 5 db. steps, but the final threshold point was determined by a closer examination in the threshold region using 2 db. steps.

* In the great majority of cases these two levels were identical.

The results of the threshold determination may clearly be expressed in terms of sound pressure in two different ways. The simplest procedure is to determine the sound pressure set up by the earphone, in an approved type of artificial ear, when the earphone is actuated by the voltage corresponding to the normal threshold excitation. When expressed in this manner, the results relate, of course, only to a particular pattern of earphone and a particular type of artificial ear, but nevertheless provide an unambiguous definition of the normal threshold with reference to the particular type of earphone concerned. The second procedure is to determine, using the same type of earphone, the corresponding acoustical pressures set up at a specified position in real ears. This could reasonably be expected to render the results independent of particular designs of

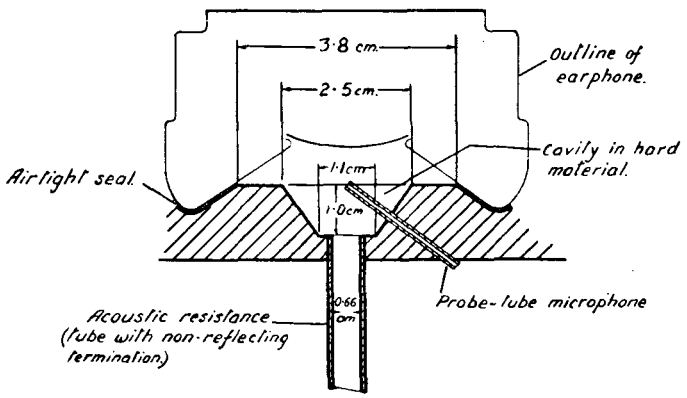

FIG. 1.

Diagram of artificial ear employed for calibration of audiometer earphones. (Section through axis.)

N.B.—The 3·8 cm. seating diameter indicated in the diagram applies to earphones (such as the type 4026A) having deep earcaps; for earphones having shallow earcaps (such as the normal British type of P.O. telephone receiver) the seating diameter adopted is 2·5 cm. This choice of seating diameters has the object of ensuring that the air volumes enclosed by earcaps of various sizes on the artificial ear approximate closely to the volumes which obtain on average human ears.

A Committee of the British Standards Institution is preparing British Standards for artificial ears.

earphone, and to enable them to be compared with previous determinations of the minimum audible pressure such as that of Sivian and White. In the present work both these methods have been followed.

The earphone employed has been calibrated using an artificial ear of the general type recommended in the Report of the Electro-Acoustics Committee of the M.R.C. (Appendix III and Fig. 13*d*). The artificial ear, which is based on the original design of West at the Post Office Research Station, with modifications introduced at the National Physical Laboratory with special reference to the calibration of audiometer earphones, is shown in Fig. 1. It has been established that this type of artificial ear

will provide objective comparisons of the acoustical output of earphones of a given pattern which are in close accord with the subjective comparisons obtained by equal-loudness balancing on human ears, and it is the type now regularly used at the National Physical Laboratory for the calibration of audiometer earphones.

The corresponding threshold pressures developed in real ears have been determined by direct comparison of the pressures generated, for given electrical excitation of the earphone, in the artificial ear and in human ears, making use of probe-tube microphones. These measurements were carried out on 20 ears of 20 subjects, 10 male and 10 female, with the earphone applied to the ear in the same manner and with the same force of application as in the threshold tests. It has proved convenient to define the normal threshold (minimum audible pressure) in terms of the acoustical pressure at the entrance to the external auditory meatus, rather than at the ear drum which is relatively difficult of access. In order to obtain results fairly representative of a small region at the entrance of the meatus, the pressure was measured at two positions, one averaging about 0·7 cm. outside, and the other about 0·3 cm. inside, the entrance to the meatus. In both cases the final results were the mean of two separate determinations.

Results of Threshold Determination

The results of the determination of normal threshold, expressed in terms of the sound pressure in the artificial ear when an earphone of type 4026A is actuated by the voltage corresponding to threshold excitation, are given in Table I, together with the standard deviations for the 198 ears of the group of 99 subjects. It is customary to define the normal threshold as the *modal* value for the group of subjects, but for completeness Table I also gives the mean and median values. It will be seen that the mean, modal and median values agree closely at each frequency. Four examples of the distribution of the results, which are sufficiently typical of those obtained over the full range of test frequencies, are shown in Fig. 2.

For all test frequencies in the range 80 to 4,000 c/s, 90 per cent. of the observations were included in a total range of 25 db.: and for test frequencies in the range 500 to 3,000 c/s, 90 per cent. of the observations fell within a total range of 20 db. From 6,000 to 12,000 c/s the corresponding total range for 90 per cent. was 30 db., but 75 per cent. still fell within a range of 20 db. The results for 15,000 c/s need to be considered separately as 17 of the subjects failed altogether to hear this frequency. The results given in Table I thus apply in this case to 164 ears of the 82 subjects whose hearing extended to 15,000 c/s, and of these 90 per cent. fell within a total range of 35 db. and 60 per cent. within a range of 20 db.

No significant difference was found between the mean thresholds for men and women, or between those for the left and right ears, the actual differences in the two cases being normally in the region of 1 db.

In the course of the otological examination of the subjects an approximate measure of the sizes of their ear canals was obtained, in four groups,

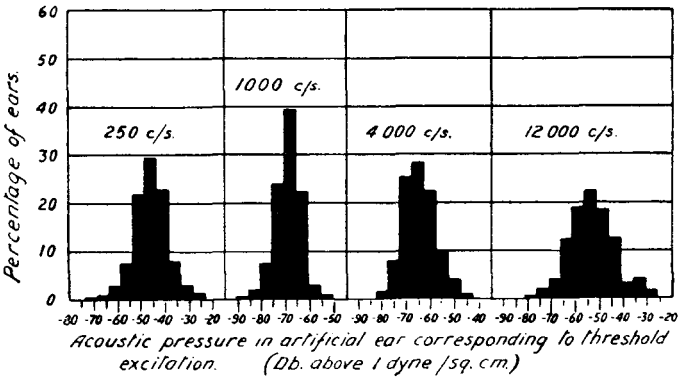

FIG. 2.
Distribution of threshold pressures for 198 ears; age limits 18-25.

TABLE I
RESULTS OF THRESHOLD DETERMINATION

Frequency cycles per second	R.M.S. acoustic pressure in artificial ear for threshold excitation of 4026A earphone (Db. above 1 dyne/sq. cm.)			Standard deviation (db.)
	Modal value	Mean value	Median value	
80	−13·5	−13	−12·5	8·0
125	−29·5	−28·5	−28·5	6·8
250	−45·5	−46	−46	7·3
(256)	(−44)	(−43)		
500	−62·5	−61·5	−62	6·5
(512)	(−61·5)	(−61·5)		
1,000	−68	−68·5	−68·5	5·7
(1,024)	(−68·5)	(−68·5)		
1,500	−66·5	−65·5	−65·5	6·1
2,000	−64·5	−63·5	−63	6·1
(2,048)	(−65·5)	(−65·5)		
3,000	−68	−67	−67·5	5·9
(3,072)	(−68)	(−67·5)		
4,000	−65·5	−64·5	−65·5	6·9
(4,096)	(−64)	(−63·5)		
6,000	−65	−63·5	−63·5	9·1
8,000	−65·5	−65	−65	8·7
(8,192)	(−64)	(−63·5)		
10,000	−58·5	−57	−58	9·0
12,000	−53	−53·5	−54	9·6
15,000	−37	−35	−35·5	10·7

Figures in brackets are the corresponding values obtained in the investigation by the Central Medical Establishment of the R.A.F. referred to in the text.

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in terms of the size of insert fitting best suited to each individual. This factor was also found to have no significant effect on the results.*

Since the completion of the N.P.L. investigation, measurements have been made on 1,024 ears of 512 otologically normal male subjects, in the same age group, at the Acoustics Laboratory of the Central Medical Establishment of the Royal Air Force. These measurements, which were made at the seven frequencies 256, 512, 1,024, 2,048, 3,072, 4,096 and 8,192 c/s, are described in full in another paper in this issue by Wheeler and Dickson, but by permission of the authors have been

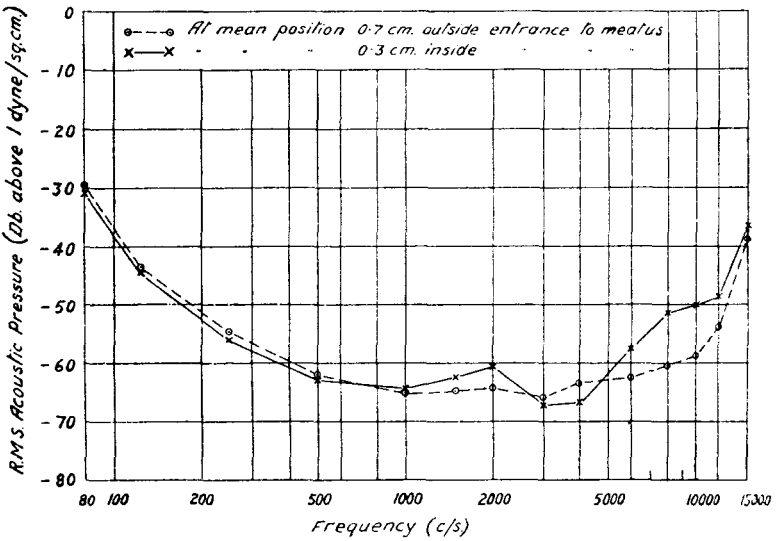

FIG. 3.

Minimum audible pressures at two positions close to the entrance to the external auditory meatus—modal values for 198 ears (164 ears in the case of 15000 c/s.)

included in Table I for purposes of easy comparison with the N.P.L. results. It is important to note that the C.M.E. tests were made with an earphone which was not only of the same type as that used by the present authors, but was also calibrated at the N.P.L. on precisely the same equipment. Thus the basis of objective measurement was identical in the two investigations. The modal values at corresponding frequencies nowhere differ by more than 1·5 db. and on the average the results at

* Some calculations have also been made at frequencies from 80 to 10,000 c/s of the coefficients of correlation between the deviations from normal of the threshold levels of the right and left ears of the subjects. Significant correlation was present at each frequency the coefficient varying from about 0·35 to about 0·65 but with no regular dependence on frequency; the average value, 0·50, is very close to that observed in the U.S. National Health Survey in the case of a group of 1,242 subjects. The correlation coefficient associated with the *average* level of the audiogram over the range 80 to 10,000 c/s is appreciably higher, viz. 0·7. No doubt a number of factors are involved in the degree of correlation between the hearing acuities of the two ears of a subject when listening with earphones, but analysis of these factors would be outside the scope of this paper.

the seven pairs of corresponding frequencies differ by only about 0·5 db. This must be considered very satisfactory agreement, and seems to provide strong evidence for the reliability and representative nature of the results of both investigations.

The threshold acoustic pressures in the ear canal corresponding to the results in Table I are shown in Figs. 3 and 4. Fig. 3 gives separately the results at the two positions near the entrance to the external auditory meatus; it will be seen that the pressures are substantially identical at the low frequencies, but show a difference varying with frequency over

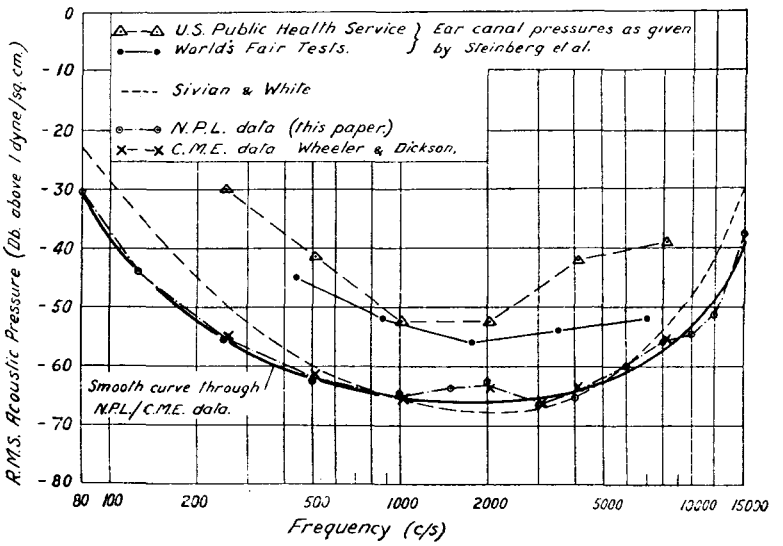

FIG. 4.

Minimum audible pressure at the entrance to the external auditory meatus (mean of two positions in Fig. 3)—modal values for 198 ears (164 ears in the case of 15000 c/s) showing comparison with ear canal measurements reported by other investigators.

the upper half of the frequency range. The latter effects must be associated with the wave motion in the ear canal and auricle, and illustrate the importance in all measurements of this kind of properly specifying the position in the ear cavity to which the data are supposed to relate.

Fig. 4 gives the average results for the two positions and may perhaps be regarded as a fair representation of the minimum audible pressure in a small region at the entrance to the external auditory meatus. The corresponding results of the C.M.E. investigation at 7 frequencies, which have been derived from their measurements of the threshold pressures in the artificial ear combined with the N.P.L. comparisons of the pressures in the artificial ear with those set up in real ears, are also included in Fig. 4. The curve given by Sivian and White and the results for the two U.S. surveys previously mentioned are also shown for comparison.

smaller curvature, falling appreciably lower at the ends of the range and slightly higher in the middle portion.

It is not possible to be certain of the exact reasons for the departures at the ends of the range, especially as the curve given by Sivian and White is not the result of any one investigation, but represents their assessment of several sets of data, probably not all of equal merit. In some investigations to which they refer, the number of ears involved is rather small, and in many cases the ages of the subjects do not seem to be known. The restriction on age to the range 18 to 25 in the present work may partially explain the increased sensitivity at high frequencies, and the choice of the entrance to the meatus rather than the ear drum as the measurement point may result in a tendency in the same direction. It may be remarked that errors in calibration and in the derivation of the actual acoustic pressures at the ear drum, such as may arise at low frequencies from uncertainty in the fit of earphones and at high frequencies from wave motion in the ear cavity, are on the whole more likely to be in evidence at the ends, rather than in the centre, of the frequency range. Careful attention has been paid throughout the present investigation to ensuring both the validity of calibrations and consistency in the experimental conditions, such as the fit of the earphones, from which uncertainties might otherwise arise.*

As will be apparent from Fig. 4, the present results differ rather widely from those of the two Surveys carried out in the United States under the auspices of the U.S. Public Health Service and the World's Fair respectively. The results shown in Fig. 4 for the Surveys apply in each case to male subjects in the age group 20-29 years and are taken from the paper by Steinberg, Montgomery and Gardner (1940). It is understood that the U.S. Public Health Service results in Fig. 4 relate to 684 ears of subjects with a clinical history of normal hearing for speech in both ears, and that the World's Fair results relate to 3,287 ears, although it is not clear in this case to what extent the sample includes persons having defective hearing. In comparing the Surveys, Steinberg, Montgomery and Gardner state that the differences between them may arise partly from true differences in hearing acuity of the groups of people tested, and partly from differences in the general conditions of test and the techniques adopted, fit of earphones, and background noise. They also state that the calibrations by which the ear canal pressures were established may have been subject to error.

From the point of view of this paper, the most interesting comparison

* With regard to the increased sensitivity at low frequencies, it may be of interest to add that in the course of a study of the equal-loudness contours threshold observations are being made at the National Physical Laboratory under free field conditions on about 100 subjects. These measurements are not yet complete, but have so far indicated an increase in sensitivity at low frequency by comparison with the Fletcher-Munson curves which is consistent in order of magnitude with the present departure in the case of earphone listening.

Laboratory of the R.A.F. on 512 subjects in the same age group. The results, when expressed in terms of the acoustic pressure at the opening of the auditory meatus are also in very substantial agreement, over the range 250 to 8,000 c/s, with the results given by Sivian and White in their classic paper on the subject. Outside this frequency range the results diverge from Sivian and White's curve in the direction of greater sensitivity.

3. The present results diverge rather widely from those of the Surveys conducted under the auspices of the U.S. Public Health Service (1935-6) and the World's Fair (1939), the data obtained in the former, for the 20-29 age group, having now been incorporated in an American Standard Specification. The precise causes of these discrepancies are not known with any certainty but some possible contributory factors are discussed. Some clinical evidence exists which suggests that the reference zero to which audiometers should be calibrated should lie a good deal closer to the present results.

4. It is suggested that the time may be ripe for further international discussion of the question in the light of the additional data now available, with a view to possible agreement on an international standard.

Acknowledgments

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They express their thanks to Mr. C. S. Hallpike, Director of the Otological Research Unit of the Medical Research Council, who organized the clinical examination of the subjects. They are also indebted to Air Vice-Marshal E. D. D. Dickson, C.B.E., and Mr. L. J. Wheeler, of the Central Medical Establishment of the Royal Air Force, for giving them the opportunity of seeing the results of their investigation before publication. The work described in this paper was carried out as part of the research programme of the National Physical Laboratory and the paper is published by permission of the Director of the Laboratory.

Abstract

The paper describes a determination of the normal threshold of hearing by earphone listening recently undertaken at the National Physical Laboratory, the primary object of the work being to provide additional data, obtained under controlled conditions, to assist in the establishment of a reference standard for the calibration of audiometers, which is greatly desired by otologists. The objective basis of calibration at present in use at the National Physical Laboratory is briefly described.

The results of the threshold determination are in excellent agreement with recent measurements made at the Central Medical Establishment of the Royal

Air Force. They are also in substantial agreement, over the frequency range 250 to 8,000 c/s—i.e. the range of most importance in audiometry—with the classic results of Sivian and White. The results differ rather widely from those obtained in two recent U.S. Surveys, one of which now forms the basis of an American Standard. The significance of the results from the point of view of possible agreement on an international standard is discussed and it is suggested that the time is ripe for further international discussion with that aim in view.

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THE DETERMINATION OF THE THRESHOLD OF HEARING

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(London)

Introduction

It is a well established practice to measure hearing acuity by audiometric methods; due mainly to the absence of a well defined and universally accepted base line this lacks the accuracy of scientific measurement. The calibration of the majority of audiometers in use today is based on the results of the measurements made during the National Health Survey (U.S.A., 1935-36). We do not consider that the degree of control maintained during these measurements was strict enough to render them readily repeatable. Nor do we believe that the conditions were such as to produce figures on which the absolute threshold value of a person's hearing could be judged.

As no officially recognized standard for the value of the minimum audible sound pressure exists in this country we decided to undertake a series of experiments with the aim of determining the absolute value of the threshold of hearing for certain pure tones.

For the optimum results in determining a value for normal hearing the following factors must always be taken into consideration:

(1) Age of the subjects tested.

(2) Clinical condition of the ears tested.

(3) Ambient level of the background noise to which the listener is subjected.

(4) The good fitting of the earphone used to provide the sound.

(5) Mechanical pressure exerted between the earphone and the ear.

(6) Accuracy and stability of the equipment used.

During the American experiments referred to above, little or no attention was paid to item 2. The results thus obtained from this Survey were the outcome of testing a general cross-section of the population which would include some people having a hearing deficiency, whereas for the purposes of audiometry an assessment of normally good hearing is required.

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A considerable amount of work on this subject has already been accomplished by the National Physical Laboratory in connection with the establishment of basic standards for use by the Electro-Acoustics Committee of the Medical Research Council. Their results are reported fully elsewhere in this issue. One of their conclusions was that there was no difference in the threshold value for male and female ears and as a result the experiments to be described in this paper were carried out on male subjects only. In addition it was decided to extend the measurements to include a thousand ears of subjects between the ages of 18 and 23 years.

Clinical Examination

A pre-requisite in an investigation of this nature is a thorough and expert clinical examination before any testing is undertaken. Any condition which is likely to interfere with the accurate determination of the threshold of hearing must be excluded. Subjects were rejected who

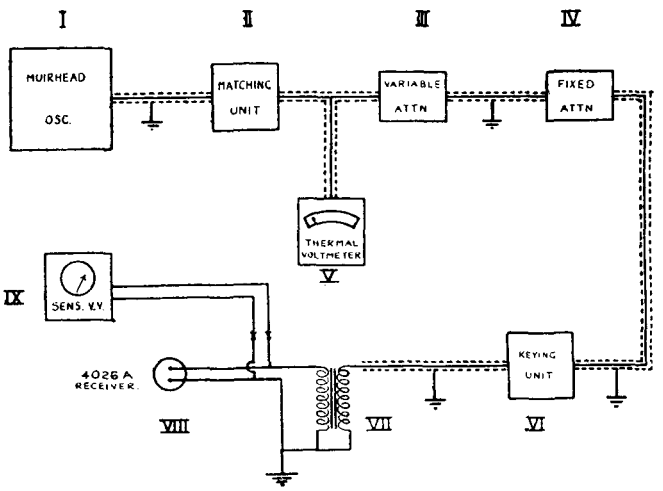

FIG. 1.

had gross hearing defects due to disease or suffered from an active middle-ear infection or had tinnitus or any whose intelligence prevented them understanding the test or appreciating what was expected from them. The latter subjects were those who responded normally to the forced whisper voice but showed gross variations when tested by audiometry, due not to deafness but to poor intelligence.

In our investigation the subjects were males between the ages of 18 and 23. They were all examined at two centres, after a preliminary screening by the Unit Medical Officer. Clinical examination at these two

centres was carried out by experienced otologists and the hearing was tested by tuning forks and the time-accepted forced whisper test. A standard technique was adopted whereby the ear not under test was masked (not blocked) by an assistant and the possibility of lip-reading was eliminated by shielding the candidate's eyes. A background of ambient noise not exceeding 40-50 decibels was aimed at and words with a low pitched and high pitched content were used. Before being tested by the special equipment set up for the purpose the subjects underwent pure tone audiometry with one or other of the well-known commercial sets at present in use in this country. It will thus be seen that those who ultimately became subjects for the estimation of their thresholds of hearing were a screened and selected group with presumably normal hearing.

Regardless of the result of the pure tone test all the subjects passing the clinical and 20 feet whisper tests were accepted for the threshold determination on our standard special equipment, with the proviso that those showing a loss by pure tone audiometry of more than 30 decibels up to 3072 and more than 35 decibels at 4096-8192 c.p.s. would be monitored during the threshold determination.

These are the subjects who provide the extremes on the right hand side of the distribution curve (Figs. 3-9, pages 385-392) and do not affect the modal value which formed the basis of our estimation.

Description of Apparatus

In Fig. 1 is shown a block schematic diagram of the layout of the apparatus with each item labelled for reference to the text.

I. Oscillator

This is a Muirhead Wigan Decade instrument type D-105A in which the individual frequencies can be selected by the manipulation of four dials labelled respectively "thousands", "hundreds", "tens" and "units".

It is used to provide the electrical signal which drives the earphone. Its frequency accuracy is $\pm$ 0·2 per cent. or $\pm$ 2 cycles whichever is the greater and the total harmonic content of its output is less than 1·25 per cent. of the fundamental.

II. Matching Unit

This is a valve amplifier having unity voltage gain and was specifically designed to match the Muirhead oscillator to the 600 ohm attenuator networks at all frequencies. The extra distortion this unit adds to the signal does not exceed 0·1 per cent. due to the extensive feedback circuitry.

Physical Laboratory. The earphone was mounted on a spring steel headband; to the other side was fitted an earphone case to produce a comfortable and balanced fit.

Setting up the Apparatus

It should be pointed out here that the final values for the minimum audible sound pressures are NOT those actually present in the auditory canal under the condition of threshold excitation; they are in fact the pressures recorded when the earphone is energized to threshold level and coupled to a 3 c.c. Artificial Ear of accepted design. The National Physical Laboratory have, however, correlated these pressures with those obtaining in real ears.

It was decided that when the variable attenuator was set to 0 db. the sound pressure level produced by the earphone (according to the artificial ear) should be 1 dyne/sq. cm. From the calibration chart of the earphone the corresponding voltage at each of the test frequencies was determined and the Oscillator (I) output adjusted until this voltage was indicated on the sensitive Valve Voltmeter (IX). A note was then made of the reading on the Thermal Voltmeter (V) and this value ("setting up voltage") was used to set the oscillator output during the tests. This particular operation was carried out twice daily during the course of the experiments; at no time did the "setting up voltages" vary in magnitude by more than 0·5 per cent. of the scale readings. These figures are shown in Table I.

TABLE I

Frequency in c/s	256	512	1024	2048	3072	4096	8192
Volts on 4026A for 1 dyne/sq. cm. ..	3·12 mV	4·16 mV	4·72 mV	4·20 mV	2·42 mV	3·26 mV	5·28 mV
Oscillator output ..	5·00 V	6·40 V	7·15 V	6·30 V	3·45 V	4·78 V	8·37 V

It is necessary in such experiments to maintain a constant mechanical pressure between the earphone and the ear under test. It seemed that the simplest way to achieve this would be to ensure that the headband was always distended by the same amount. Accordingly, a series of measurements were made to determine the overall distance between the outside faces of the earphone and the dummy case when they were being worn. This distance was measured on 100 heads and noted to vary between the limits of 8·1 in. and 9·1 in.; it was therefore decided that the headband should always be distended beyond the limit to 9·25 in. In practice this was achieved by inserting the requisite number of thin

sheets of rubber between the dummy earphone and the ear not being tested.

The actual pressure exerted by the headband under these conditions of stress was later measured and found to be 13·5 oz. (383 gm.). The diagram in Fig. 2 shows how this pressure was measured.

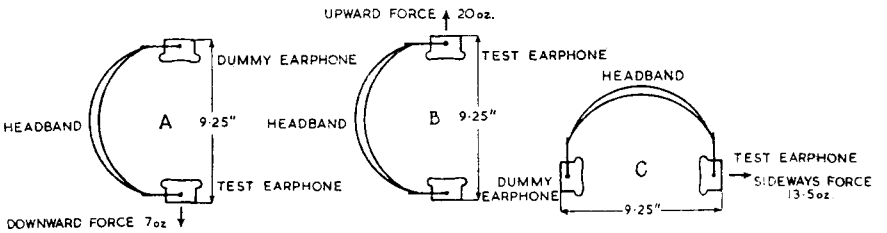

FIG. 2.

Difference between A and B = Twice weight of earphone = 13 oz., therefore weight of earphone = 6·5 oz. In A tension in headband = downward force + weight of earphone = 13·5 oz. In B tension in headband = upward force — weight of earphone = 13·5 oz. In C tension in headband = sideways force = 13·5 oz.

Operation of Equipment

The subjects were seated in a standard fashion in a sound-proof booth having an ambient noise level at all times less than 20 db. relative to a sound pressure of 0·0002 dynes/sq.cm. The subject maintained communication with the operator outside the booth by means of a simple lamp signalling system. It was the operator's responsibility to attend to the correct fitting of the earphone and correct tensioning of the headband. The temperature in the booth varied during the experiment through the range 65° to 70° F.

The oscillator output was adjusted to the "set up" value for the appropriate frequency (*vide* Table I) and the variable attenuator set to produce a sound which it was estimated could be heard comfortably by the subject. The sound pressure level was then reduced progressively until the subject could no longer indicate or distinguish correctly between the "on" and "off" conditions of the test tone, and the attenuator setting at this point was noted.

From this value the sound pressure level was reduced by a further 10db. and then from that point progressively increased until the subject's signals again synchronized with the operation of the interruptor key. This second attenuator setting was also noted, though it seldom varied by more than 2 db. from the first, generally being smaller by that amount. The arithmetic mean of these two values was taken as the threshold value for the ear under test and was expressed in decibels below a sound pressure level of 1 dyne/sq. cm. (As plotted in the histograms, by subtracting the reading from 74, the answer can be expressed in decibels above 0·0002 dynes/sq. cm.)

The Determination of the Threshold of Hearing

In order to simplify the plotting of the histograms it was decided that the variable attenuator setting should always be a multiple of 2 decibels. That was the smallest allowed incremental value for the changes in sound pressure, except for the initial approach to the threshold region when steps of 6, 8 or 10 db. were used to save time.

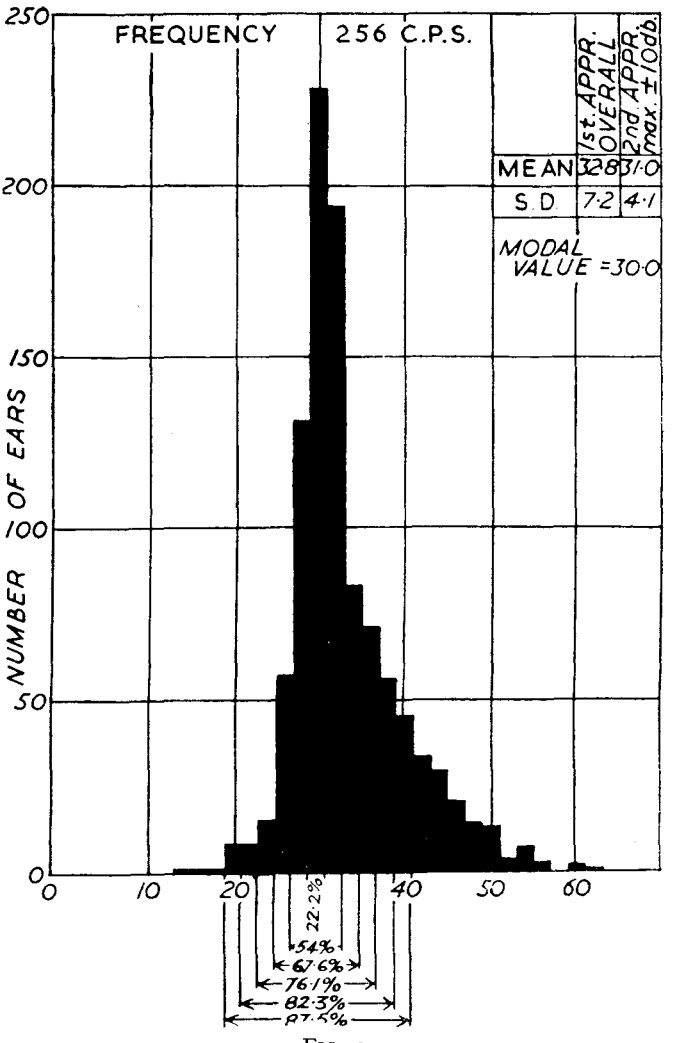

FIG. 3.

Sound pressure decibels relative to 0·0002 Dynes/cm² as measured on a 3 c.c. artificial ear

The tests were made at seven frequencies, viz. 1024 c/s, 512 c/s, 256 c/s, 2048 c/s, 3072 c/s, 4096 c/s and 8192 cycles in that sequence. To avoid fatigue, the subjects were allowed a short rest between each frequency and a longer one between the testing of each ear.

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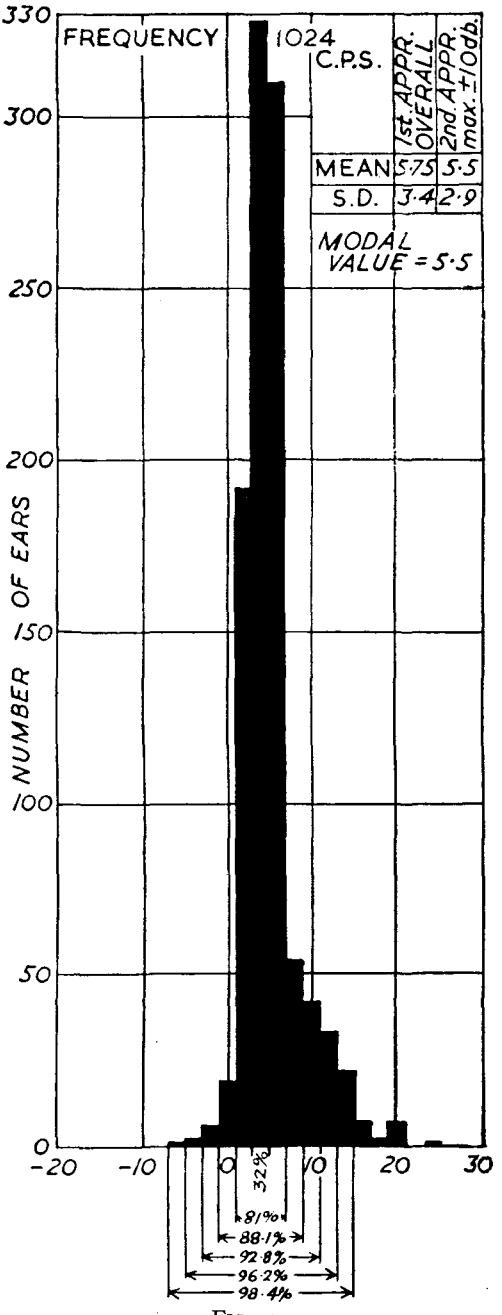

FIG. 5.

Sound pressure decibels relative to 0·0002 Dynes/cm² as measured on a 3 c.c. artificial ear.

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measurements at each of the test frequencies are shown in Figs. 3 to 9 inclusive.

It will be noticed from these histograms that by far the greater number of the subjects (between 87·5 per cent. and 98·4 per cent.) fall within the limits of a band 20 db. wide centred on the value of the maximum occurrence. These subjects can truly be described as being representative of normal good hearing. The asymmetry of the curves also shows that more people are expected to be slightly below than above average.

To obtain the mean value of the threshold from these curves the calculations were restricted to this 20 db. band of " normally good ears " ; because of the close agreement between the modal values and these second approximations it would appear that the definition is a sound one. For all practical purposes, however, the modal value of the curves should be taken as the threshold rather than the arithmetic mean, as it is reasonable to assume that more people conform to the actual threshold value than to any other single value above or below it.

Whilst the experiment was in progress the results were being continually analysed, partly as a further check on the consistency of performance of the equipment, and partly to determine what effect, if any, the number of ears tested would have on the final results. From Table II it is obvious that the performance of the whole equipment was quite consistent and that providing the clinical examination is thorough and efficient, any measurements on a smaller scale can and will produce a similar result.

TABLE II

MINIMUM AUDIBLE SOUND PRESSURES IN DECIBELS REL. TO 0·0002 DYNES/SQ. CM. STEP BY STEP ANALYSIS FOR EACH 50 SUBJECTS.

Subjects	Frequency in cycles per second													
	256		512		1024		2048		3072		4096		8192	
	Mean	Modal	Mean	Modal	Mean	Modal	Mean	Modal	Mean	Modal	Mean	Modal	Mean	Modal
To 50 ..	30·2	29·3	12·9	12·8	6·1	6·0	7·4	7·5	6·7	5·9	10·6	9·6	9·3	9·1
To 100 ..	30·4	29·4	11·9	11·9	5·5	5·5	8·4	8·3	6·9	6·1	9·8	10·4	9·6	9·4
To 150 ..	30·4	29·4	11·7	11·8	5·5	5·4	8·8	8·6	6·7	6·0	9·9	8·9	9·7	9·4
To 200 ..	30·6	29·9	11·7	11·7	5·5	5·4	8·6	8·5	6·5	5·8	10·2	9·2	10·3	9·7
To 250 ..	30·7	29·8	11·8	11·7	5·7	5·6	8·8	8·6	6·7	5·9	10·6	9·6	10·2	9·7
To 300 ..	31·1	30·1	12·3	12·2	5·9	5·9	9·1	8·7	6·7	5·8	10·3	9·3	10·3	9·7
To 350 ..	31·2	30·2	12·4	12·4	6·6	6·4	8·6	8·6	6·6	5·8	10·3	9·3	10·2	9·7
To 400 ..	31·2	30·2	12·4	12·4	5·6	5·7	8·7	8·7	6·6	5·8	10·7	9·7	10·0	9·6
To 450 ..	30·8	29·9	12·4	12·4	5·3	5·5	8·7	8·7	6·6	5·8	10·3	9·3	10·1	9·6
To 500 ..	31·2	30·2	12·3	12·3	5·7	5·5	8·6	8·7	6·7	5·8	10·3	9·8	10·3	9·8

A similar step by step check to the above was also kept on the ages of the subjects and the individual values in this case did not vary by more than 1¼ per cent. from the final value of 19·7 years.

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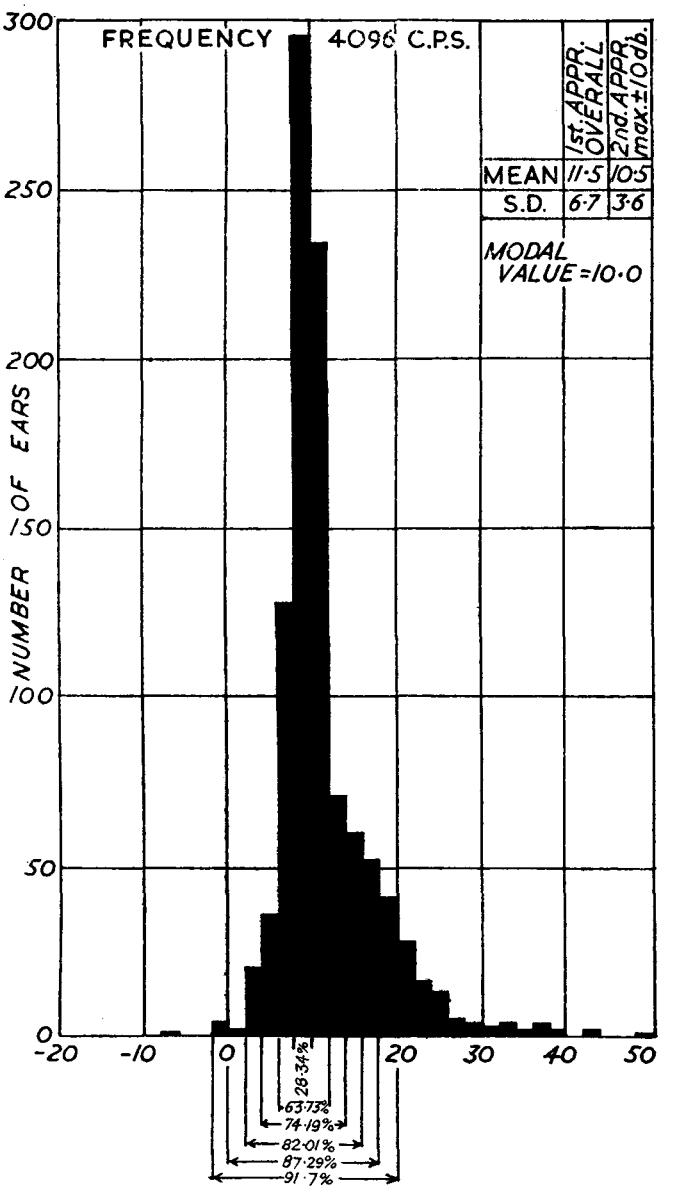

FIG. 8.

Sound pressure decibels relative to 0·0002 Dynes/cm² as measured on 3 c.c. artificial ear.

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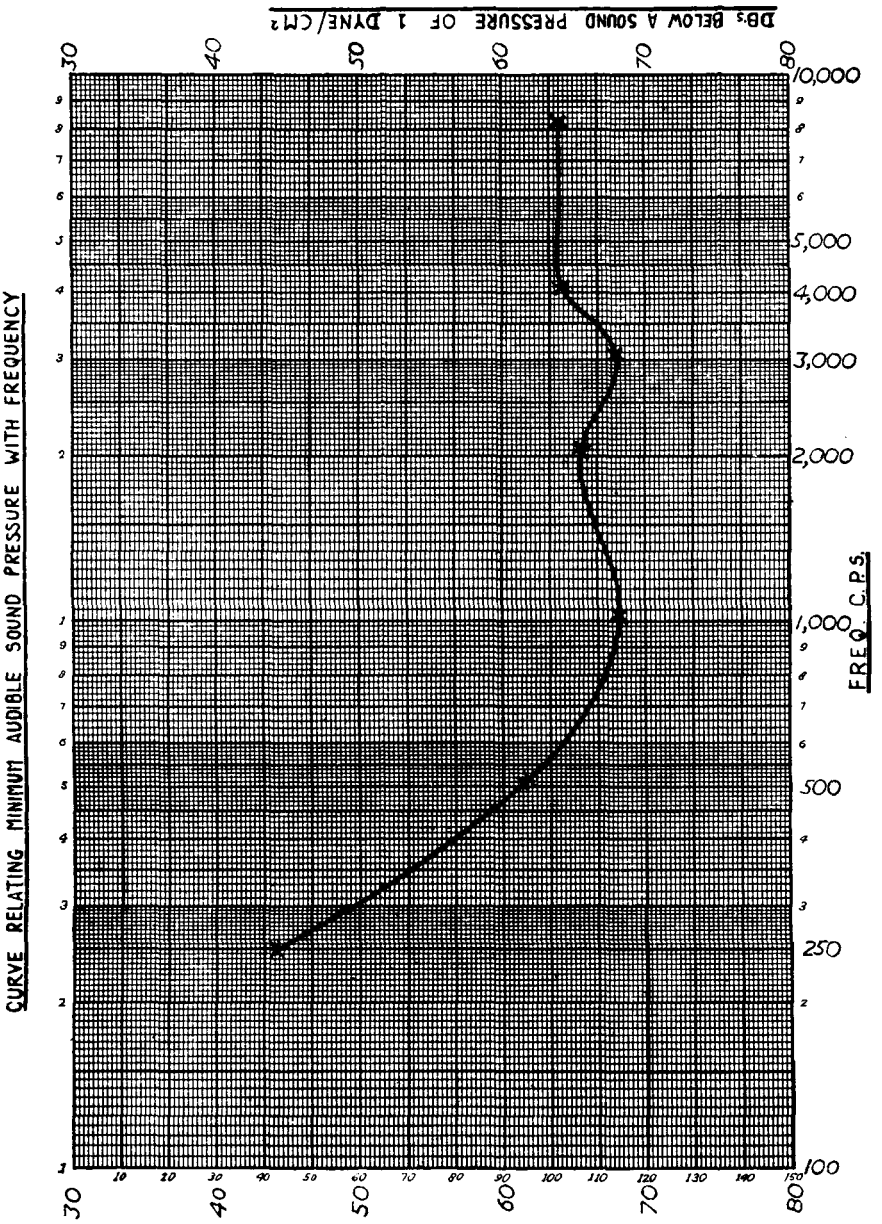

FIG. 10.

Curve relating minimum audible sound pressure with frequency.

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figures and those obtained by the National Physical Laboratory and the reader is referred to Mr. Dadson's paper for a fuller analysis of this agreement.

Acknowledgments

We are glad to record our appreciation of the help and collaboration Dr. T. S. Littler, Director of the M.R.C. Wernher Research Unit on Deafness, has given in this investigation. In his capacity as adviser to our Department he has scrutinized equipment, measurements and the ultimate content of this report. To Professor Bradford Hill and Dr. Armitage, both of the London School of Hygiene and Tropical Medicine, we express our gratitude for guidance in the statistical planning of this research.

We also wish to thank the Senior Medical Officers of Groups for organizing the attendance of subjects. Without their co-operation the scheme, which involved the movement of a large number of people, would not have been possible.

Finally, to the members of our staff, particularly Sergeant R. F. H. Jellis, who helped in the testing and checking, we tender our thanks.

Each candidate carried this sheet with him and presented it to the examiner at the time of examination. Any candidates with a history of ear trouble were examined first, and in addition to auriscopic inspection were examined with a Siegle's attachment as well.

Clinical Examination

1. *Hearing*

Entrants were first tested for hearing. This was performed by standing the entrant 20 ft. from the examiner and facing directly away from him. An orderly made a masking noise by intermittent compression of the tragus of each ear in turn while the examiner whispered three or four words for the opposite ear.

Any entrant unable to hear this was re-examined later and his degree of deafness assessed by the number of feet at which he could hear this same forced whisper.

2. *Auriscopy*

Entrants were next examined with an electric auriscope. Firstly the degree and quality of any wax present was noted. Following this all wax was syringed out.

Secondly, any abnormalities in the external auditory meatus were noted and all exostoses and deformities were drawn on the entrant's sheet.

Thirdly, the tympanic membrane was carefully inspected over its whole area and any irregularities and abnormalities noted.

Finally, any entrant giving a history of ear trouble, or presenting any abnormal signs, was given a complete examination with tuning forks, direct auriscopy and movement of the tympanic membrane with the Siegle's speculum.

All results were recorded on the entrant's typed sheet as he presented himself. Every candidate was examined, and all the pathological ears tabulated by myself, thus eliminating individual differences in recording.

Definition of a Normal Ear

For the purpose of this survey an ear was considered to be normal if it exhibited the following characteristics.

1. A forced whisper can be heard at 20 feet with each ear separately, the other being masked by intermittent compression of the tragus.

2. Pinna is of average shape and size. No accessory auricles or fistulæ are present.

3. There is no evidence of any past aural operation.

4. External auditory meatus contains no more than a small amount of wax usually obscuring a small part of the tympanic membrane.

5. External auditory canal is smooth and passes medially slightly downwards and forwards.

6. About four-fifths of the whole tympanic membrane can be seen

after angling the speculum in every direction. The portion most commonly invisible is the anterior recess.

7. Tympanic membrane : handle of the malleus stands out clear and white. Light reflex is present extending from umbo to annulus tympanicus in the antero-inferior quadrant. It usually occupies an arc of 15° to 30°, and is at 100° to 140° to the handle of the malleus. The drum is of a homogeneous pearly translucency, and has a smooth contour indrawn at the umbo—moving as a complete membrane on Siegling or on inflating the eustachian tube.

Any folds in the attic region should be present in the same position in the other ear.

Every effort was made to ensure that all candidates understood and answered questions in their histories correctly. Instructions and testing were standardized to eliminate personal error as far as possible. All illustrations of pathological ears were made by one observer on a standardized diagram, and care was taken to reproduce pictures as accurately as possible with regard to size and position of lesions relative to the normal landmarks, and the four quadrants of the drum.

Findings

Over all it was found that 105 recruits, i.e. 10·5 per cent. exhibited some pathology in one or both ears. In the remaining 895 no abnormality could be detected.

Histories

Data from the history sheets is tabulated in Table I.

Probably of most significance is the fact that in the pathological group the majority (73·4 per cent.) gave no history of previous ear trouble.

A detailed analysis of those with active lesions in the ear showed that of 7 cases with active chronic suppurative otitis media only 3 gave a history of any ear trouble, while in 2 cases, with dry perforation, no history was obtainable at all.

There appears to be no difference between the normal and pathological groups with regard to tonsillectomy or adenoidectomy.

TABLE I

Group	Total number of cases	Percentage with previous aural history	Average age of ear trouble	Percentage cases with tonsils and adenoids removed	Average age in years of operation	Percentage cases having had measles or scarlet fever	Average age of infection
Normal ..	895	8·6	9 years	30·5	6	70	6 years
Pathological	105	22·6	7 years	33·3	8	60	6 years

The significance of a previous history of measles or scarlet fever is of great interest.

It is a common belief (Kerrison, 1930; Ballenger, 1943; Lederer, 1946) that measles and scarlet fever are frequently responsible for otitis media in patients who give no history of the latter.

From the figures obtained in this series of cases, there appears to be no grounds whatsoever for this belief. The fact that one has had measles or scarlet fever does not imply that any past otitis media was due to that cause.

Further, the age incidence of measles and scarlet fever does not support a correlation between these two diseases and evidence of past otitis media with no history.

The average age incidence of measles or scarlet fever in both normal and pathological groups was six years. There are few adults who will not remember a primary attack of acute otitis media when they were six years of age—even though in association with an infectious disease. In the pathological group, of which 23 per cent. gave a history of ear trouble, the average age at which it started was seven years, i.e. presumably the remainder, who gave no history, had trouble much earlier than this.

Very few, if any, adults remember events associated with teething. This is a time when many cases of otitis media occur, and the condition is frequently overlooked by both parent and physician since it usually settles when the teeth have erupted. Examination of the ears is difficult even for the expert at this age, while without any apparatus one can follow the course of the erupting teeth. For these reasons the constitutional disturbance is attributed to the teeth and the ears seldom examined.

It seems, therefore, that in those patients who show signs of old otitis media without any history, it is probable that in the majority the trouble occurred in infancy perhaps in association with teething, and not in later childhood as a sequel of exanthemata.

Examination

I. Cerumen

For purposes of analysis and interest ear wax was divided into three broad categories as regards consistency:

Hard—the consistency of soft wood.

Soft—the consistency of thick oil, usually unpleasant smelling and sufficiently moist to be soaked up on a bit of cotton wool.

(Care was taken NOT to include any active C.S.O.M., or low grade otitis externa amongst these cases.)

Medium—half-way in between the other two.

(Doubtful cases were always classed as medium; only wax which was quite definitely very hard or very soft was put in the other categories.)

It was also divided into three classes by virtue of the quantity of wax present :

O—No wax was present or insufficient to obscure vision to any part of the tympanic membrane.

+—Sufficient wax was present to obscure part but not all of the tympanic membrane, no matter how the aural speculum was angled.

++—No portion of the tympanic membrane could be seen because of the amount of wax present, in spite of angling the aural speculum in every direction.

To try and gain a true impression of the amount of wax present and in what proportion of the population, the possibility of syringing had to be taken into account.

All ears syringed presumably had wax at the time of syringing. Ears syringed over one year prior to examination were not taken into account, as it was thought that any tendency to wax formation would manifest itself within this period. Therefore, the total number of ears containing wax was calculated as the number of ears syringed during past year plus the number of ears containing wax which had NOT been syringed during the past year. As syringing would have altered the quantity and possibly the consistency of wax in the ears, those cases which had been syringed were not counted in an analysis of the consistency of wax in the ears. It was assumed that in them the wax would have proportionately the same consistency overall as in those which had not been syringed. In pathological cases, ears with an active discharge were not included in the count of wax.

Table II contains a summary of all the data appertaining to wax.

II. External Auditory Meatus

Pathological group—total 105 cases (pathology in one or both ears) contained :

(*a*) Active external otitis in three cases (five ears). Two cases gave a history of discharge and irritation from the ears.

(*b*) Exostoses in three cases (five ears).

All cases gave a history of regular swimming activity.

Peripheral portions of the postero-inferior, and antero-inferior quadrants were obscured by exostoses.

(*c*) Stenosis in four ears. (Two of these were secondary to a cortical mastoidectomy.)

TABLE II

FINDINGS WITH REGARD TO QUANTITY AND CONSISTENCY OF CERUMEN IN NORMAL AND PATHOLOGICAL EARS

Group	Total number of ears	Percentage of ears containing wax	Percentage of ears containing wax, which totally obscures T.M.	Consistency of wax		
				Medium	Hard	Soft
Normal ..	1,790	27·6	22·2	90·5%	5·1%	4·4%
Pathological	210	23·3	30·7	—	—	—

Consistency of wax in the pathological group is not included because of insufficient cases to be of significance.

III. CORTICAL MASTOIDECTOMY

Nine cortical mastoidectomies out of 105 pathological cases, of which one was an active chronic suppurative otitis media; two were stenosed along the meatal wall (partially obscuring the tympanic membrane); one showed no visible scar even though the whole drum could be clearly visualized and was subjected to Sieglization.

IV. TYMPANIC MEMBRANE

Although every pathological drum had been drawn individually by a single observer, a system of codification was evolved to enable one, without actually seeing the lesions, to gain a fair idea of their size, position and nature. By this means one could tabulate and collate all lesions of a particular type and compare them with other groups.

The following is the system used and enables one to interpret the tables below.

(*a*) *Position of lesion in tympanic membrane*

The drum was divided into its four separate conventional quadrants. One line passes through and along the handle of the malleus, continued to separate the drum into two halves anterior and posterior. A second line passes at right angles to the first and through the umbo separating the drum into two more halves, upper and lower. The attic region is, of course, separated from the two upper quadrants by the anterior and posterior malleolar folds. As there were only a few lesions in the attic they are all mentioned separately.

(*b*) *Size of Lesion indicated by*

+=less than half the area of a quadrant.

++=more than half but not quite a whole quadrant.

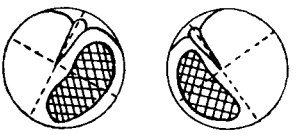
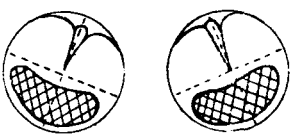
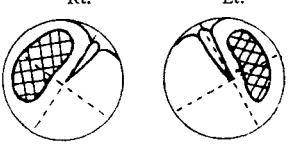
+++=larger than one quadrant but not larger than 1½ quadrants.

++++=more than 1½ quadrants, but not greater than 2 quadrants.

Analysis of the Ears of a Thousand Young Men

TABLE III

ANALYSIS OF POSITION, SIZE AND NATURE OF LESION IN TYMPANIC MEMBRANE

No.	Ears Rt.	Lt.	B.	Quadrant Ant-Sup.	Ant-Inf.	Post-Inf.	Post-Sup.	F.	Comments
									Typical Group I lesion Rt. Lt.
									
Group I								I	
1	x	–	–	xx–xx		–	–	–	Fixed posteriorly
2	cx	–	I	cc–cc			xxx	–	
3	–	x	I	xx–xx		–	–	–	
4	cx	–	I	ccc		x	–	–	
5	–	c	–	ccc		–	–	–	Marginal calcareous patch
6	–	cx	–	ccc		xx	cc	–	M.h.p. separating 2 calcareous patches
7	–	cx	–	ccc		xx	cc	I	M.h.p. separating 2 calcareous patches
8	cx	–	I	xxx c		–	c	–	Calcareous patch in ant. fixed scar
9	–	cx	I	xxx–cc		–	xx	–	
10	cx	–	I	ccc		x	–	–	
11	–	c	I	ccc		–	–	–	
12	cx	–	–	ccc		x	–	–	
13	cx	–	–	ccc		x	–	–	
14	x	–	I	xxx		–	xx		
									Typical Group II lesion Rt. Lt.
									
Group II								–	
15	x	–	I		xx–xx			I	Thin ballooning scar
16	x	–	–		xx–xx			–	Grossly adherent. F.W.=6 ft.
17	cx	–	–		ccc		x	–	
18	cx	–	I		ccc		x	–	
19	cx	–	–		ccc		x	–	
20	–	cx	I	xx	ccc		–	–	
21	–	c	–		ccc		–		
									Typical Group III lesion Rt. Lt.
									
Group III									
22	–	cx	–	cc–cc			xx–xx	–	
23	–	cx	I	cc–cc			xx–xx	–	
24	cx	–	–	ccc			xx–xx	–	
25	cx	–	I	ccc			xx–xx	I	Healed perf. adherent to incus.
26	x	–	I				xx–xx	–	Rt. cortical mastoidectomy
27	x	–	I				xx–xx	I	
28	x	–	–				xx–xx	I	Grossly adherent. F.W.=4 ft.
29		cx	I	c	–		xx–xx	–	
30	–	x	–	x	–		xx–xx	–	2 separate m.h.p.
31	–	cx	I	–	c		xx–xx	–	Fibrous band between calc. patch and (m.h.p.
32	–	x	–				xx–xx	–	
33	x	–	–				xx–xx	–	Firmly adherent to incus.
34	–	cx	–	–	c		xx–xx	–	
35	x	–	–				xx–xx	–	
36	–	cx	I				xx–xx ccc	–	Scar contains calc. patch and moves on (respiration
37	–	cx	–				xx–xx c	–	Calcareous patch in m.h.p.
38	x	–	–				xx–xx	–	
39	–	cx	I	c	–		xx–xx c	–	Calcareous patch in m.h.p.
40	cx	–	–	–	cc		xx–xx	–	
41	x	–	I				xx–xx	–	

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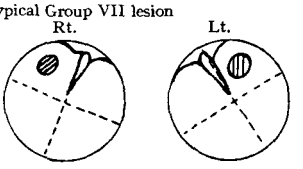
Analysis of the Ears of a Thousand Young Men

No.	Ears Rt.	Lt.	B.	Quadrant Ant-Sup.	Ant-Inf.	Post-Inf.	Post-Sup.	F.	Comments
									Typical Group VII lesion 
Group VII									
89	–	x	1				xx		
90	x	–	–				xx	1	Firmly fixed to incus
91	–	x	–				xx		
92	–	x	1				xx	1	
93	–	x	1				x		
94	x	–	–				x		
95	–	x	1				x		
96	–	x	–				x		
97	–	x	–				x		
98	–	x	–				x		
99	–	x	–				x		Left cortical mastoidectomy
100	–	x	–				x		Left cortical mastoidectomy
101	–	x	–				x		Left cortical mastoidectomy
102	x	–	–				x		Right cortical mastoidectomy
103	x	–	–				x		
Group VIII									
104	OS	–	1			OS+			C.S.O.M. active ++
105	–	OS	1				OS+		Left cortical mastoidectomy Post-Sup. marginal perforation. Granulations +
106	–	OD	1			OD +++			Dry posterior perforation
107	OS	–	–			OS+			Post-Inf. marginal perforation Active C.S.O.M. + granulations
108	OS	–	–	OS++					Ant-Sup. perforations and granulations
109	–	OS	–	OS+					Anterior attic perforation
110	OS	–	–			OS++			
111	OD	–	–	OD+					Dry attic perforation
112	–	OS	1		OS++				Perforation with granulation

This is in disagreement with Kerrison, who is of the impression that calcareous patches are situated usually in the posterior half of the drum. Frequently there is no visible scar in the tympanic membrane (e.g. Cases Nos. 5, 11, 21, etc.). The whole drum moves normally without any irregularity and it appears as if the calcareous patch is merely a deposit in the middle fibrous layer of the drum. It is difficult to know whether this is a physiological or pathological process.

Frequently also one finds a mobile healed perforation separated by apparently normal drum from a calcareous patch, which also looks pathological, i.e. the portion of drum containing it does not move quite evenly and regularly (e.g. Cases Nos. 22, 23, 24).

Other not uncommon findings are two calcareous patches separated by a healed perforation; and also conversely a calcareous patch completely enclosed in the substance of the scar of a healed perforation, e.g. Cases Nos. 37 and 39.

Marginal calcareous patches, i.e. about 2 mm. from the annulus tympanicus and passing through two or more segments are quite common, e.g. Cases Nos. 11 and 21. These nearly all seem to be in the substance of

the drum and may have no connection with a perforation. Some authorities regard them as senile degenerations, but they are relatively so common in this age group that one wonders if that is a correct interpretation.

3. *Scars in tympanic membrane*

One point which became quite evident, the more cases that were studied, is that the size of the scar of a healed perforation does not seem to have any direct relation to its thickness. The scar of a healed perforation involving three quadrants may move fairly evenly and regularly with the rest of the drum the scar apparently having about the same thickness and elasticity as the drum, the difference being chiefly evident only at the junction of scar and normal drum, e.g. Case No. 47. Alternately, a small scar occupying only one quadrant may be tissue-paper thick and balloon out on Sieglization, like an inner tube bulging through a split tyre.

Generally it is supposed that the fibrous middle layer of the drum does not reform after a perforation. Many of these scars are so firm that one feels they must have all the elements of a normal drum, including a fibrous layer.

It seems reasonable, in assessing these ears with regard to prognosis, particularly for flying, that more importance should be placed on the thickness, and mobility of the scar than on its size. Another factor which becomes apparent was that the position, degree and extent of adherent scars did not bear any direct relationship to hearing. An ear with a large, tightly adherent posterior scar of the tympanic membrane might have clinically normal hearing, while one with a smaller inferior scar might be associated with a quite marked hearing loss.

4. *Individual cases of interest*

No. 36. J.H. Gave no previous history of ear trouble. Tonsils or adenoids not removed. Measles, aged 3 years.

Examination revealed a right ear with a large healed posterior perforation firmly stuck down to the incus, a large anterior calcareous patch and normal hearing. The drum was quite fixed on Sieglization.

Left ear, revealed a large healed posterior perforation, with a calcareous plaque in its substance.

This healed perforation was not only *NOT* fixed, but it was actively mobile with respiration, inwards on inspiration, outwards on expiration. There was no effect on the hearing.

One usually finds about one of these cases of respiratory tympanic membrane in every thousand cases.

No. 110. M.P. History of left otorrhœa, aet. 6 years, for which he had treatment. Tonsils and adenoids removed, aet. 6 years. Never had measles or scarlet fever.

Examination: Right ear. Forced whisper 20 feet. External auditory meatus—normal. Tympanic membrane—normal Rinne + ve.

Left ear. Forced whisper, 1 foot. External auditory meatus—filled with soft waxy discharge completely obscuring tympanic membrane. On cleaning this revealed: tympanic membrane—small central attic perforation, which discharged profusely on Sieglization Rinne — ve. Weber —→ Left.

Chief interest in this case was how such a marked degree of hearing loss in one ear had escaped notice. Apparently common practice at medical boards is to make the patient block his own ear. In this case he was told to cup his hand over each ear in turn. Recruits are frequently told that they have wax in their ears but as their hearing is normal their ears are all right. This is, of course, quite fallacious as patients with dry perforations secrete wax like normal persons and it is not at all uncommon to remove dry hard wax and find a large dry perforation behind.

Another instance of the value of a proper system for testing the hearing of recruits is illustrated in the following case.

C.K. History. No previous ear trouble. Never had tonsils and adenoids removed. Scarlet fever, aged 14. Passed at Medical Board as normal ear, nose and throat.

Examination: Right ear. Forced whisper 2 feet. External auditory meatus—completely occluded by dense hard wax. After removal of wax tympanic membrane normal and forced whisper 20 feet.

Left ear. Forced whisper 6 feet. External auditory meatus—also completely occluded by dense hard wax. After removal of wax tympanic membrane normal and forced whisper 20 feet.

In this case the patient said that at his medical board two examiners had examined his hearing one blocking his ear and the other whispering, but he was facing the whispering examiner.

On further investigation it became obvious he was a perfect lip reader, and was quite ignorant of the fact.

He was practically deafened by noise after the removal of the wax. Though we have seen many ears completely occluded by wax and with a diminished hearing this is the first case we have on record of natural lip reading secondary to wax deafness.

Conclusions

In a group of 1,000 healthy young men one may expect to find evidence of some previous aural pathology in about 10 per cent., and of these cases about 10 per cent. are unfit for present day service requirements, e.g. because of chronic suppurative otitis media. A few of these cases give a history of previous ear trouble, but the majority, even of those with active lesions, do not. This is because most cases occurred in infancy, prior to the development of conscious memory. The value of an adequate and standardized clinical hearing test cannot be overestimated in assessing

a man. To pass an ear as normal without being able to see every portion of the drum (e.g. when obscured by wax) is quite fallacious even if the hearing is normal and there is no previous aural history. In assessing the functional loss of a scarred drum, thickness and mobility of the scar are of more importance than size. The role of calcareous patches is difficult to determine, but they appear to be frequently associated with scars. Marginal calcareous plaques appear to be physiological entities and are quite common in this age group.

Table IV contains a summary of all the aural conditions found.

SUMMARY OF PATHOLOGICAL EARS IN 1,000 YOUNG MEN

Total number of cases (pathology in one or both ears) 105.

External ear :	
Otitis externa	5 ears
Exostoses	5 ,,
Stenosis	4 ,,
Cortical mastoidectomy	9 ,,
Middle ear :	
Scars	54 ears
Calcareous patches	15 ,,
Calcareous patches plus scars	34 ,,
Active chronic suppurative otitis media	7 ,,
Quiescent chronic suppurative otitis media	2 ,,

Summary

Of a thousand fit young men examined, 10 per cent. showed signs of previous aural disease.

All lesions of the tympanic membrane were graded, and tabulated to assess the numbers of a given size of lesion in any particular position.

Conclusions are drawn as to what may be considered a normal ear and the importance of a standard system of examination is emphasized.

Acknowledgments

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CLINICAL RECORDS

THREE CASES OF INFECTED LARYNGOCELE

By JAMES FREEMAN (Bristol)

THREE cases of infected laryngocele, treated recently at the Bristol General Hospital, are recorded, as it is felt that the manner in which they presented may be of some interest.

CASE I

J.V., aged 58, a toolmaker, attended on Boxing Day, *1951*, owing to complete inability to swallow. He had had increasing dysphagia for the previous week, but after eating his Christmas dinner with great difficulty, he had been unable to swallow anything, even liquids.

He had no hoarseness, and no dyspnœa (though he had had some cough and breathlessness on exertion for several years).

He had noticed a swelling on the right side of the neck for the past three days, and he remarked that he had had a similar swelling, associated with difficulty in swallowing, about three years before, and that the trouble had settled after a few days treatment with penicillin at home, and had not recurred until the present occasion.

He was found to have a soft tender fluctuant swelling, about 2½ inches across, reaching from below the angle of the jaw nearly to the midline anteriorly, and to the lower border of the mandible above.

Indirect laryngoscopy was difficult owing to the large amount of saliva in the throat, but there appeared to be an irregular mass in the region of the right arytenoid, with some superficial slough over it, and the right cord appeared to be fixed. It was felt that the condition was either an infected laryngocele, or else a carcinoma of the larynx with secondary infection.

A course of intramuscular penicillin was started, and by the next day he was able to swallow fluids, and thereafter his dysphagia was steadily relieved, though the swelling in the neck remained unchanged.

On *December 31st* the swelling was aspirated, and 32 c.c. of thick brown pus was withdrawn (this was sterile on culture).

On *January 3rd, 1952*, direct laryngoscopy showed a large smooth swelling in the region of the right false cord, but no evidence to suggest a neoplasm now. An oblique incision was then made over the swelling in the neck (which had by this time refilled); the wall of the sac was then isolated, dissected out, and traced over the upper edge of the thyroid cartilage, down to its origin from the laryngeal ventricle, and then removed.

Microscopic examination of the tissue removed confirmed the diagnosis of infected laryngocele.

Subsequent progress was straightforward, and laryngeal appearances are now normal.

CASE II

F.P., aged 69, a labourer, attended in *May, 1950*, with a history of hoarseness for 7 to 8 years, sore throat for the past month, and dyspnœa for the past fortnight.

Laryngoscopy showed a carcinoma of the larynx with subglottic extension.

Total laryngectomy was performed on *June 1st, 1950*, and deep X-ray treatment was given subsequently.

The pathological report on the specimen removed, showed that both true cords were largely destroyed by malignant growth, more extensive on the left side, where the growth also involved the lower aspect of the false cord. On this side, there was an abscess cavity above the false cord, about ½ in. across, filled with pus, which drained into the lumen of the larynx just above the true cord.

Fig. 1 shows a low power magnification of the left true and false cords, with the abscess cavity above them. The other photographs show higher magnification of the areas noted in the diagram.

The whole lining of the cavity and of the saccule (which forms the track leading from the cavity to the ventricle), is heavily infiltrated with inflammatory cells, and pus is present in the cavity, the saccule, and the outlet from the saccule into the ventricle.

The lining of the cavity is partly stratified squamous epithelium (see Fig. 2), but pseudo-stratified columnar ciliated epithelium is present in some areas (see Fig. 3), and this epithelium also lines the track leading from the cavity to the ventricle (see Fig. 4). This suggests that the abscess was really a true internal laryngocele, which had become infected, and that the prolonged exposure of the ciliated lining to chronic infection had resulted in metaplasia to squamous epithelium.

The medial aspect of the false cord is covered by healthy squamous epithelium (see Fig. 5), but on the lower aspect the epithelium is replaced by squamous carcinoma, which extends up just to the point at which the saccule opens into the ventricle ; beyond this point the epithelium is columnar ciliated, heavily infiltrated with inflammatory cells (see Fig. 6).

Fig. 7 shows the squamous cell carcinoma which has completely destroyed the true cord.

CASE III

A.T., aged 38, a plumber, attended in *1950* complaining of " strangled speech " for the previous four months. He said this had started after having a sore throat which took a long time to clear up.

He noticed that his voice had changed, and that a swelling appeared in the right side of the neck when he swallowed, and that this was getting larger. There was no dysphagia or dyspnœa at that time.

He was found to have a soft mobile reducible swelling, attached deeply in the region of the hyoid bone, on the right side of the neck. There was no swelling inside the larynx. X-ray showed that the swelling contained air, and he was advised to have the laryngocele removed.

However, he could not be persuaded to have the operation, so he was advised to cease blowing down pipes to clear them (as he was accustomed to do

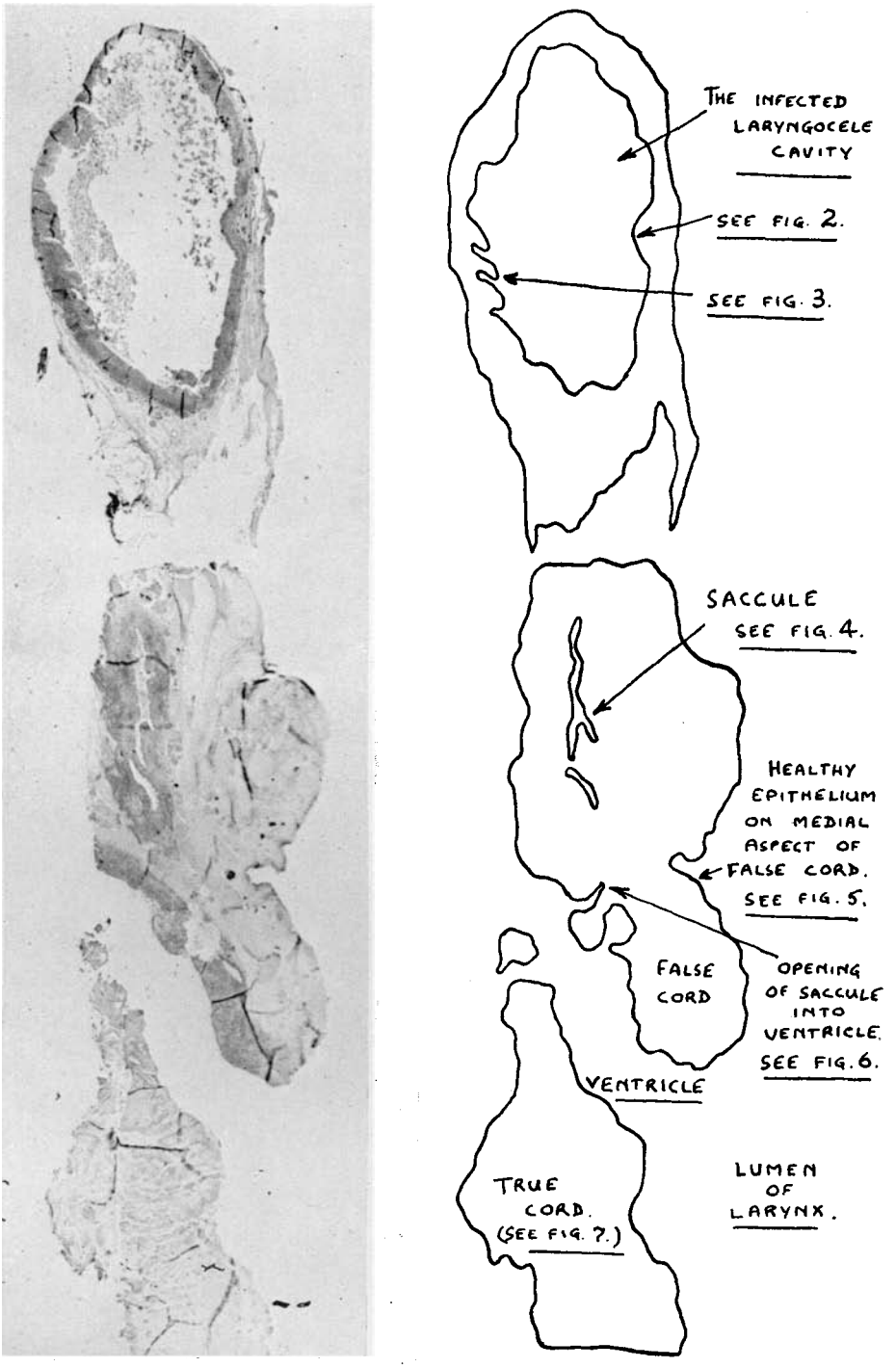

FIG. 1.

To show left false and true cords, laryngeal ventricle and saccule, and the infected laryngocele above them.

[*face p.* 410

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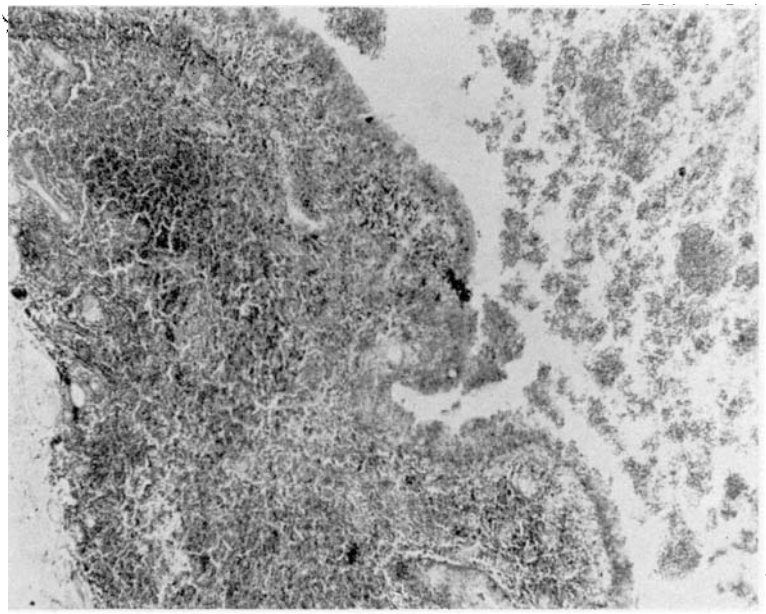

FIG. 2.
Squamous epithelium lining part of the cavity.

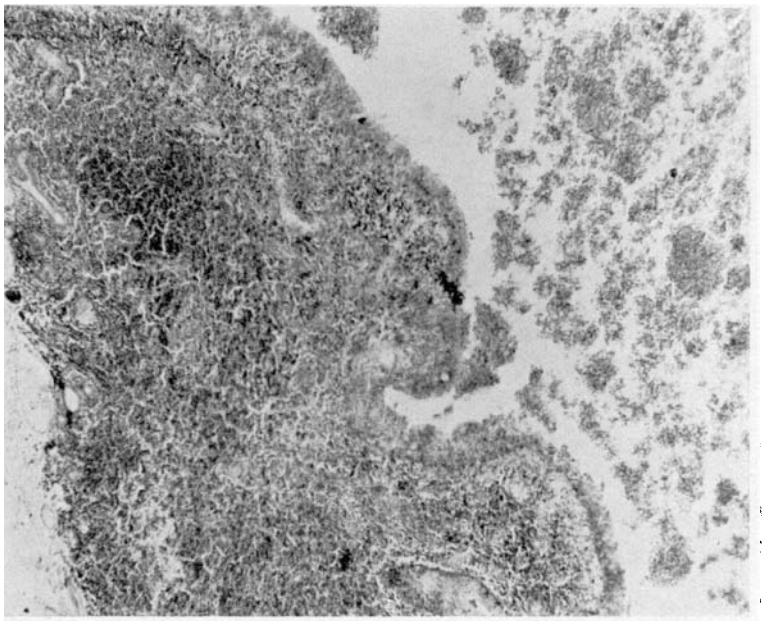

FIG. 3.
Columnar ciliated epithelium lining the cavity on the opposite side.

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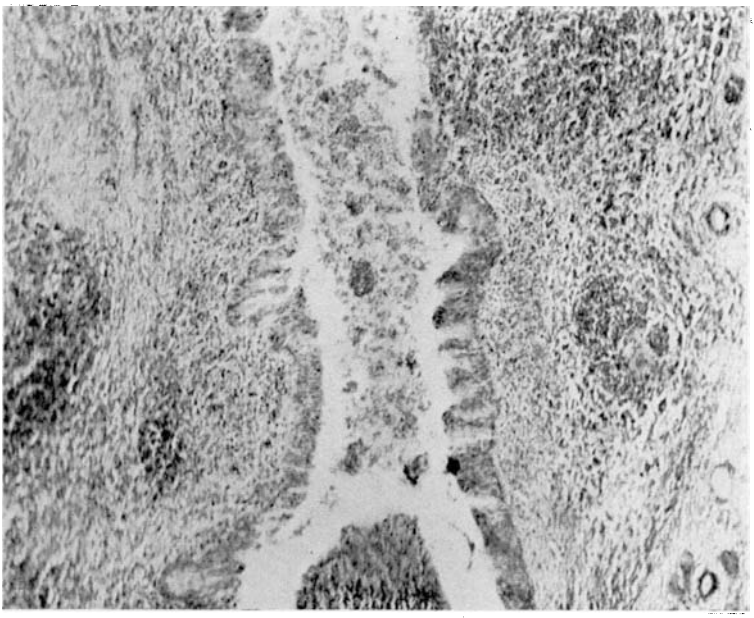

FIG. 4.
Columnar ciliated epithelium lining the saccule.

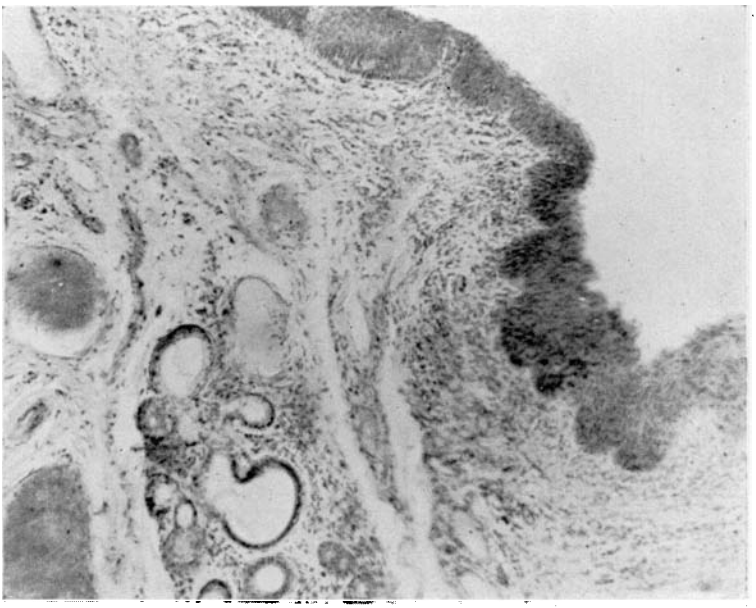

FIG. 5.
Healthy squamous epithelium on the medial aspect of the false cord.

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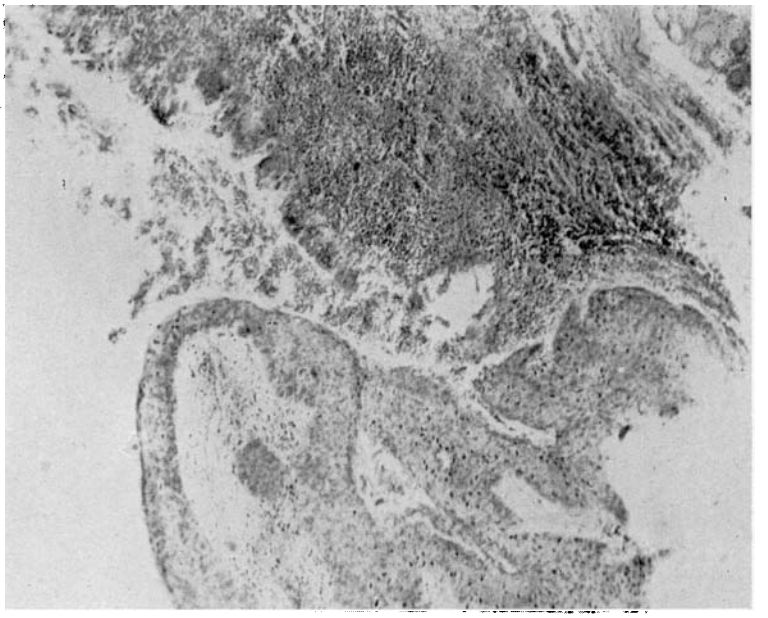

FIG. 6.

To show the outlet of the saccule into the ventricle, with columnar ciliated epithelium above, and squamous cell carcinoma below.

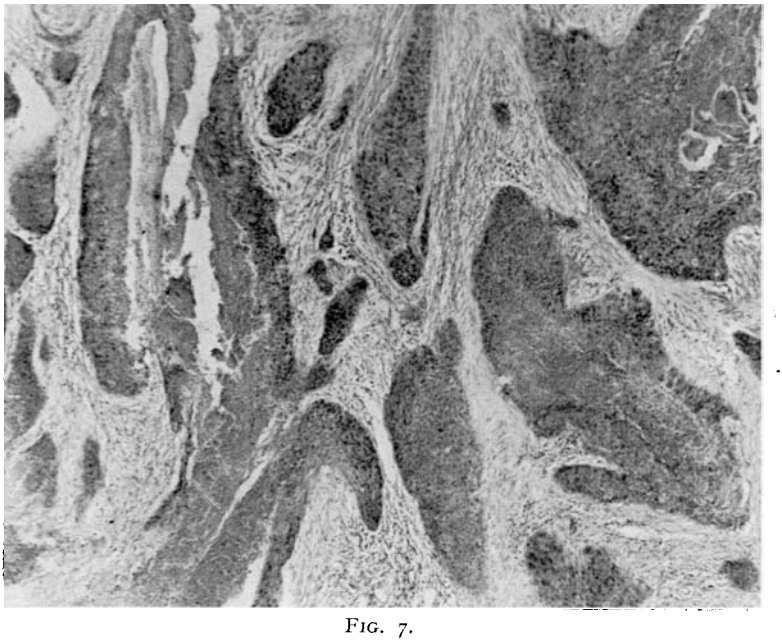

FIG. 7.

To show the squamous carcinoma which had destroyed and replaced the true cord.

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in the course of his work), in the hope that this would prevent the condition getting worse.

Some months later, he returned as he was becoming increasingly hoarse, though he still had no dysphagia or dyspnœa.

Examination now showed a swelling visible inside the larynx, and half occluding it. The external swelling was still reducible, and X-ray showed that it still contained air.

Two weeks later, the external swelling was larger and firmer (but fluctuant), and no longer reducible. X-ray showed it to be opaque, and it was clear that it now contained fluid. There was now dysphagia, and occasionally a choking sensation, and the patient was now willing to have an operation to get relief.

At operation on *February 23rd, 1951*, through a right collar incision, the sac was dissected out ; its deeper part was very adherent ; it was punctured, and 60 c.c. of pus was withdrawn (this was sterile on culture). There was no direct communication with the laryngeal lumen at this time. The sac was dissected down to its origin at the laryngeal ventricle, and removed, and the thyro-hyoid membrane repaired. Progress since then has been uneventful and the voice is now good.

Discussion

In the literature referring to laryngoceles, the complication of infection appears to be relatively rare, and the association of carcinoma of the larynx with a laryngocele to be rarer still.

Shambaugh (1915) described a case of a lady aged 69 whose laryngocele discharged large quantities of pus day and night, constantly waking her up, and who was able to obtain partial relief by pressure over the external sac, evacuating as much as a quarter of a tumbler of pus at a time. She had had attacks of hoarseness since childhood.

Allmann and Cordray (1942) recorded a case of a patient who had a laryngocele associated with hoarseness for five years, where direct laryngoscopy showed the left cord and ventricle to be chronically inflamed, and a thick greyish mucoid material draining from the left ventricle.

Lothrop (1943) described a " laryngopyocele " in a patient who had been hoarse for 18 months.

Schall (1944) described a case of laryngocele associated with carcinoma of the larynx, and a history of hoarseness for two years, but there is no record as to whether this laryngocele was infected or not.

Taylor (1944) reported a case of a man of 35 who had had recurring attacks of hoarseness as long as he could remember. He was found to have a laryngocele, which after removal was reported on as containing a quantity of limpid serous fluid ; microscopically, there was evidence of chronic inflammatory changes in the wall of the sac.

O'Keefe (1951) records a case of a laryngocele in a man aged 45 who had been hoarse for six months. The lining of the cyst was respiratory type epithelium, densely infiltrated with inflammatory cells.

The presence of dysphagia as a prominent symptom of a laryngocele, as in Cases 1 and 3, appears to be quite unusual : since in both these cases the laryngoceles were filled with pus, it seems reasonable to suppose that the

dysphagia was due to extension of inflammation and œdema backwards, so as to involve the pharynx, and that conversely, if a laryngocele is known to be present, dysphagia indicates that it is probably infected.

In Cases 1 and 2 there was no history to the effect that either patient had ever blown wind instruments or had abnormally raised the intraglottic pressure in any other way. In Case 3 repeatedly blowing down pipes to clear air locks probably contributed to the development of the laryngocele, or at least aggravated the condition if it was already present as a congenital abnormality.

The finding, in Case 2, of an unsuspected chronically inflamed laryngocele, associated with hoarseness of 7 to 8 years duration, and the association of prolonged hoarseness with infected laryngoceles in the other cases previously recorded, suggests that persistent or recurrent hoarseness may be due to this cause (or at least due to chronic infection of the ciliated epithelium lining the laryngeal ventricle or saccule), more often than is realized, and an attempt is now being made to investigate this matter further.

The association of carcinoma of the larynx with an infected laryngocele raises the question of whether or not chronic infection in a laryngocele or laryngeal ventricle may be a contributory factor in the development of carcinoma of the larynx.

It is suggested that, when a larynx is examined after laryngectomy, a routine search should be made for any evidence of chronic infection in the laryngeal ventricle saccule, or in an unsuspected laryngocele.

I wish to thank Mr. G. R. Scarff and Mr. J. Angell James (under whose care these patients were admitted), and Dr. A. L. Taylor (Director of the Pathological Department of the Bristol General Hospital), for permission to record these cases, and for their advice and encouragement.

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A CASE OF GROSS HYPERPLASIA OF THE TONSILS IN A MAN AGED 77

By B. S. CARTER (Stoke-on-Trent)

It is a matter of common observation that there is a steady decline in the size of the tonsils with age. Some authorities consider that this is due to replacement of the normal cellular constituents by fibrous tissue, and Stieglilz (1949) describes a process termed autoclasis, in which pieces of lymphoid tissue are cut off from the main body of the tonsil by fibrous bands.

Kelemen (1943, 1945) however, states that the cellular and basal reticular constituents of the tonsil remain unchanged in form and activity throughout life, and further says that there is a characteristic period of increased tonsillar activity at the onset of senescence corresponding to the similar period before puberty. He mentions that often in old age the tonsils are large and fully developed, but says nothing about gross hyperplasia.

Thorpe (1946) reported a case of a woman aged 78½ where the tonsils were so grossly hypertrophied as to meet in the midline and cause marked dysphagia. Tonsillectomy was at first considered out of the question but later was performed under local anæsthesia. The operation was commenced as a dissection, but this caused respiratory distress, so the tonsils were removed with a large guillotine very easily; the post-operative course was uneventful.

Certainly old age in itself does not appear to be a contra-indication to tonsillectomy. Wilkinson (1929) reports that out of 10,000 cases done at the Mayo Clinic from 1923 to 1927, 13 were age 70 and over, and Kelemen (1946) maintains that in later years the indication for operation is as urgent as in earlier life, and that technically intervention is simpler. This latter point was certainly confirmed in the case here described.

Case Report

Mr. W.G., aged 77, had a sore throat in *January, 1950*, following which he experienced increasing difficulty in swallowing and some interference with respiration. At examination in *May, 1950*, he had enormous pedunculated tonsils which almost completely occluded the pharynx, the right one being much the larger. There was no clinical evidence of malignancy, and tonsillectomy was advised. Anæsthesia (Dr. E. Isaacson) was induced with nitrous oxide and oxygen, and maintained with vinesthene-æther mixture through a nasal intratracheal tube. The tonsils were dissected very easily, with minimal bleeding, and the only difficulty was due to the large size of the tonsils, which made it necessary to cut off the freed upper part of the right tonsil so as to get at the lower pole. The patient was encouraged to move about the ward on the following day, and made an uninterrupted recovery. He died from coronary thrombosis in *December, 1951*, without any further trouble from his throat.

Dr. A. J. McCall reported that the right tonsil weighed 25 grammes and the left tonsil 10 grammes: section showed simple hypertrophy associated with marked keratosis of the crypts and chronic inflammation.

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CLINICAL NOTE

PURE TONE AUDIOMETRY

Technique for Determining Thresholds of Hearing for Pure Tones

The following notes, prepared jointly by the Medical Research Council Committees on Medical and Surgical Problems of Diseases of the Ear and on Electro-Acoustics, are intended for the guidance both of designers and users of pure tone audiometers.

The Audiometer

In order to achieve uniformity both of performance and of the test procedure, the general design and layout of audiometers should be as closely standardized as possible. This applies particularly to the mode of operation and to the relative positions of the different controls.

For general work a series of fixed frequencies is to be preferred to a continuous sweep frequency, and the following should be provided: 250, 500, 1000, 2000, 3000, 4000, 6000, 8000 cycles per second.

An " on " switch should be included which can be either held or locked in the " on " position.

It should be possible to test hearing for pure tones by air and by bone conduction. There should be a masking device and twin headphones should be available and so arranged that when the test tone is delivered to one ear a masking sound can be delivered to the other. All controls should be silent in operation.

Finally, the standard of threshold on which the calibration of the audiometer is based should be clearly stated both on the audiometer and on the audiogram.

The Test Room

Noise must be excluded as far as possible from the test room. If the ambient noise level is greater than 40 db. as measured on a sound level meter conforming with the American Standard Specification, the room should not be regarded as quiet enough for accurate threshold tests, and this fact should be recorded on the audiogram. For ears having nearly normal hearing a sound level of 40 db. may well be excessive for accurate determination of threshold. The test should be carried out in a room separate from the general clinic, and the room should be large enough to contain both subject and tester. For a successful and reliable test a quiet room free from visible as well as audible distractions is necessary.

The Subject

The subject should be comfortably seated, and unable to see the face of the audiometer and any movements of the controls.

The Tester

The tester must of course be familiar with the audiometer in use, and the accuracy of the instrument must be frequently checked.

Clinical Note

The Test

With the possible exception of screening tests the pure tone audiometer test should only be carried out after an otological examination. The subject must clearly understand what to expect, and it may sometimes be necessary for a printed card to be available for the subject to read before the test. The following test has been found to be helpful.

" Hearing Test "

" In order to test your hearing you will be asked to listen through headphones to a series of simple tones. Each ear will be tested separately and every time that you hear a tone, no matter how loud or soft it may be, you are to say ' YES ' (or press the signal button or raise the hand).

" There may be a continuous hum or buzzing noise in one ear : this is to prevent that ear hearing test tones. Ignore the buzzing sound, fixing your attention on the test tone and say ' YES ' or give a signal only when you hear the test tone."

It is customary to test all tones in one ear before starting on the other.

Each test tone should be presented to the subject by switching the sound on two or three times for half to one second, with a similar interval between the sounds. At first with each tone the intensity should be well above the estimated threshold, and if heard should be sounded again with the intensity reduced by 5 to 10 decibels. This should be continued until the sound is no longer heard. The threshold of each tone should be noted as the lowest level at which the tone is heard, the steps of intensity being arranged at 5 decibel intervals.

Should the difference in hearing loss between the two ears at any frequency be greater than 40 decibels, the test should be repeated on the less sensitive ear while the more sensitive ear is masked by a suitable continuous masking noise. If known the intensity and nature of the masking noise should be noted on the audiogram. As each tone is tested the threshold should be noted on the audiogram, and the subject should be warned when the test with a new tone is about to begin.

Audiometry for bone conduction is in an early stage of development and investigations to determine most suitable apparatus and techniques are still proceeding. Pending the results of these investigations it is recommended that, when testing for bone conduction the ear under test should not be occluded or covered in any way, as any external obstruction may appreciably influence the result of the test. During the test the untested ear must be masked using, for example, one of the headphones provided. The bone conduction receiver should be placed just behind the auricle on a level with the external meatus being tested, the receiver being held in position by a springy headband. The tones to be used should include 250, 500, 1000, and 2000 cycles per second.

At the end of the test the audiogram should be signed by the tester, and a clear indication of the meaning of the symbols, date of test and name and age of the subject added, together with any remarks about masking, conditions of the test room, and any doubt about the accuracy of the subject's responses.

GENERAL NOTES

BRITISH ASSOCIATION OF OTOLARYNGOLOGISTS
RUEDI TRUST FUND

THE British Association of Otolaryngologists has been entrusted with a sum of money to be devoted to the financial assistance of British postgraduates studying otolaryngology in Switzerland. Information can be obtained from the Secretary of the Association, 45 Lincoln's Inn Fields, London, W.C.2.

THE INSTITUTE OF LARYNGOLOGY AND OTOLOGY

The Academic Board announces the following Special Lectures to be given in the Lecture Hall of the Institute at Gray's Inn Road. They are open to all members of the Speciality and to post-graduate students.

1952

September 12th, at 5 p.m. DR. NORTON CANFIELD, of Yale University, will open a discussion on the analysis of clinical hearing records.

FIFTH INTERNATIONAL CONGRESS OF OTO-RHINO-LARYNGO-BRONCHO-ESOPHAGOLOGY

AMSTERDAM, JUNE 8TH-15TH, 1953

ORGANIZED BY THE DUTCH OTO-RHINO-LARYNGOLOGICAL SOCIETY

President :	Prof. Eelco Huizinga.
General Secretary :	W. H. Struben.
Scientific Secretaries :	J. Bijtel and P. G. Gerlings.
Reception Secretaries :	Miss A. C. Schippers and A. Dokkum.
Treasurer :	Mrs. F. Velleman-Pinto.

The International Committee for the International Congress for Oto-Rhino-Laryngology has invited the Dutch Oto-Rhino-Laryngological Society to organize the Fifth International Congress in the Netherlands.

This Congress will take place on June 8th-15th, 1953, in Amsterdam.

Apart from the combined sessions (selected subjects) the Congress Members will be invited to smaller sessions.

The selected subjects will be :

TUMOURS OF THE BRONCHUS

A. Soulas et P. Mounier-Kuhn. " Tumeurs bénignes des bronches."
L. Pietrantoni. " La clinique et l'endoscopie des cancers des bronches."
Louis H. Clerf. " Cytology of Tumours of the Bronchus."

AUDIOLOGY

C. S. Hallpike. " Physiological Problems of Audiology."
George E. Shambaugh. " Clinics of Audiology."
S. R. Silverman. " Social Aspects of Audiology."

General Notes

Allergy

G. Dohlman. " Theory of Allergy."
French H. Hansel. " Methods of Allergic Examination."
R. Melchior. " L'hypersensibilité d'origine non-allergique."

Ladies' Programme

A Ladies' Committee will take care of the Associate Members during their stay in Amsterdam. A special programme will be arranged for visiting different points of interest.

Communications

Communications of two sorts are invited, and these should be in one of the official languages : English, French or German.

(*a*) *Original Papers*

Speakers are limited to 15 minutes. The number of original papers is limited and the Secretary will inform Members before February 1st, 1953, whether time in the programme has been allotted to them. Members presenting papers are requested to give the Secretary a full typescript copy (4 pages of 500 words each, figures included) immediately after they have been read. If this is not done, it will not be possible to print the paper in the Proceedings. Discussion of the original papers will be limited to a total of a quarter to half an hour.

(*b*) *Contributions to the Discussion of the Selected Subjects*

These speakers will be limited to five minutes. About one hour will be devoted to the discussion of each selected subject.

All Members taking part in discussions are asked to give a written résumé of their contribution (10 lines) to the Secretary at each meeting.

Films

Scientific films will be shown at intervals throughout the Congress.

Subscriptions

The Subscription for Members has been fixed at 100 Dutch guilders (or the equivalent in other currency).

The subscription will include the cost of the Proceedings of the Congress when published. Ladies and other relatives accompanying Members may be registered as Associates at a fee of 25 Dutch guilders.

The registration fee may be transferred beforehand by the Intermediary of your Bankers to the Twentsche Bank, Amsterdam, in favour of the account of the International Congress of O.R.L.B.E., 1953.

General Information

Accommodation in or around Amsterdam : In the next notice you will find a form for hotel accommodation which will be provided for by the Organizing Committee. This first notice is sent to all those who may possibly want to attend this Congress. Those interested may receive further circulars concerning details on registration, payments, accommodation, entertainments and receptions, excursions, programme, etc., from the Secretary.

General Notes

It will greatly facilitate the clerical work and tend to avoid mistakes if all names and addresses are either typewritten or written clearly in block capital letters.

Letters of registration, enquiries and suggestions should be addressed to the Secretary of the Congress: W. H. STRUBEN, J. J. Viottastraat 1, Amsterdam-Z.

EXAMINING BOARD IN ENGLAND

OF THE

ROYAL COLLEGE OF PHYSICIANS OF LONDON

AND

THE ROYAL COLLEGE OF SURGEONS OF ENGLAND

DIPLOMA IN LARYNGOLOGY AND OTOLOGY

THE Secretary reports to the two Royal Colleges that at the First Part of the Examination for the Diploma in Laryngology and Otology, held on June 6th, 9th and 10th, 1952, by Mr. G. H. Livingstone and Mr. F. C. W. Capps, 50 Candidates presented themselves, 27 of whom passed; and that at the Second Part of the Examination, held on June 13th, 16th, 17th, 18th and 19th, 1952, by Mr. J. H. Cobb and Mr. R. J. Cann, 40 Candidates presented themselves, 17 of whom passed.

The following 17 Candidates, having complied with the regulations of the Board, are eligible to receive the Diploma, viz.:

Name.	Address.	Qualification.	School.
Ackerley, Anthony George.	c/o Officers Mess, R.A.M.C. H.Q., Millbank, S.W.1.	M.B., B.Ch. Cambridge, L.R.C.P., M.R.C.S.	King's College.
Atkinson, Edmund Clegg.	298 Meadow Head, Sheffield 8.	M.B., B.S. London, L.R.C.P., M.R.C.S.	St. Barts.
Banerji, Sushil Chandra.	c/o Barclay's Bank, 9 Russell Square, W.C.1.	M.B., B.S. Lucknow.	Lucknow.
Brown, James Stinson.	96 Napier Court, S.W.6.	M.D. Manitoba.	Manitoba.
Bushell, Basil Wyvern Michael.	11 St. Mary's Road, Harborne, Birmingham.	M.B., Ch.B. Birmingham.	Birmingham.
Clifford, Patrick Paul.	Springmount, Ballyard, Tralee.	M.B., B.Ch. N.U.I.	Dublin.
Fleming, Frank.	1A The Oval, West Hartlepool, Co. Durham.	M.B., B.S. Durham.	Durham.
Halfhide, Anthony William.	62 Castlebar Road, W.5.	B.Ch. Cambridge. L.R.C.P., M.R.C.S.	St.George's.
Itchhaporia, Savitri.	Canterbury Hall, Cartwright Gardens, W.C.1.	M.B., B.S. Punjab.	Punjab.
Juby, Herbert Bernard.	The Durdans, Stowmarket, Suffolk.	M.B., B.S. London, L.R.C.P., M.R.C.S.	St. Barts.
Khambatta, Kershaw Burjor.	Bleak House, Bleak House Road, Karachi 4, Pakistan.	M.B., B.S. Bombay.	Bombay.
Ross, Edgar Hughan.	c/o D.M.S., Penang, Malaya.	M.B., B.S. Sydney.	Sydney.
Rousseaux, Georges Emile.	R.A.M.C. Officers Mess, Military Hospital, Wheatley, Oxon.	B.M., B.Ch. Oxford.	Oxford.

General Notes

Name.	Address.	Qualification.	School.
Scales, Peter Raglan McHaffie.	50A West Kensington Mansions, Beaumont Crescent, W.14.	L.R.C.P., M.R.C.S.	Guy's.
Sinha, Rama Heramb Prasad.	c/o Dr. R. V. P. Sinha, M.S., F.R.C.S., Medical College Hospital, Patna, Bihar, India.	M.B., B.S. Patna.	Patna.
Taylor, Thomas Cochrane.	2 Windsor Road, Saltburn by Sea, Yorks.	M.B., B.S. Durham.	Durham.
Wood, Philip Henry.	9 Fernwood Court, Fernwood Road, Jesmond, Newcastle-upon-Tyne 2.	L.R.C.P., F.R.C.S.	University College.

LATE NOTICE

INSTITUTE OF LARYNGOLOGY AND OTOLOGY, ACADEMIC BOARD

Special Lecture

1952

September 5th. PROFESSOR JOHN R. LINDSAY, of University of Chicago. "Observations on the Obliteration of the Ductus Endolymphaticus, Aquaeductus cochlearis and the accompanying venous channels in Experimental Animals."

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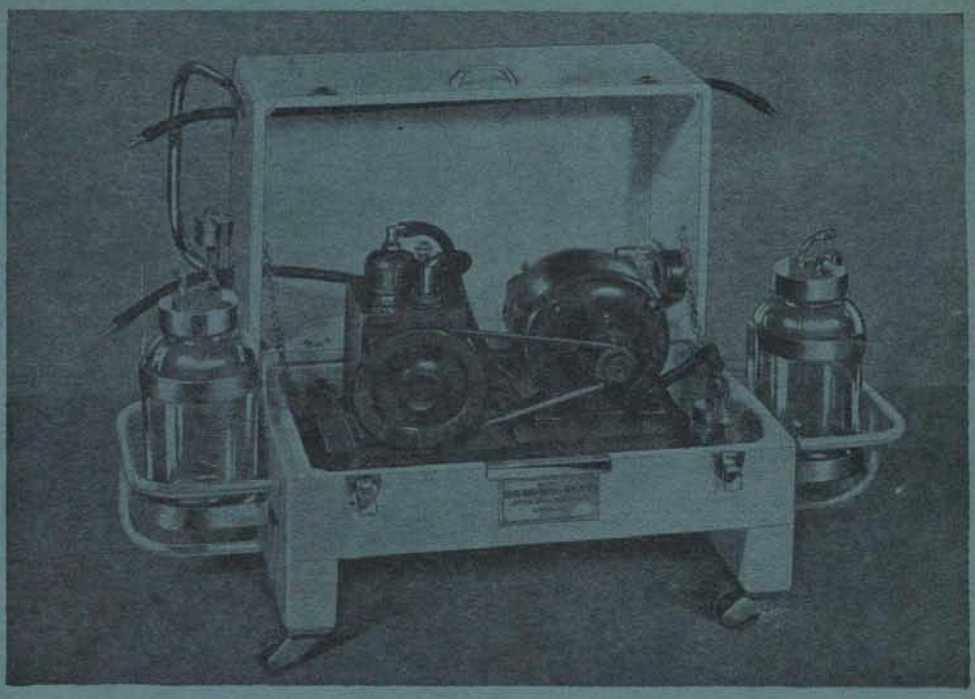

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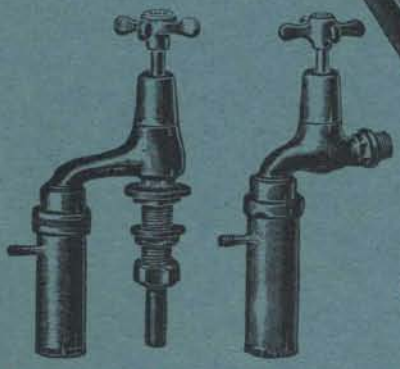

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