Thesis.

Adenoid Vegetations in the Naso-pharynx

-A Clinical and Pathological Study of 214 Cases

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Introduction

A study of adenoid vegetations in the nasopharynx may appear to the casual observer an insignificant theme on which to found a thesis, but I hope to show in the following pages its importance, and far reaching consequences that have not yet received, either by the Specialist or General Medical Practitioner, the careful attention it deserves.

Most of our general Medical and Surgical text books scarcely more than mention the existence of a hypertrophied pharyngeal tonsil, and even treatises on diseases of the Throat & Nose give little consideration to the manifold signs and symptoms presented by subjects suffering from these adenoid vegetations; and it is only too certain, that for many people childhood is made miserable and adult age a failure by a condition, which, is easily recognised when looked for, and perfectly curable when rightly treated.

The material I have had to carry on this investigation has been obtained at the Hospital for Sick Children, Newcastle-on-Tyne, where, for over a year, I have been Resident Medical Officer. It has consisted of 214 cases of adenoid vegetations operated on, and the examination of the throats of 500 children at the Out Patient Department in connection with the Hospital. It is often said,

(1) 'Archiv fur Ohrenheilkunde', and Med. Chir. Trans. Vol LIII 1870

(2) 'Die Anwendung der Galvanocaustik im Innern des Kehlkopfes u. Schlundkopfes', p. 66, seqq. Wien, 1867.

and justly I think, that the data received from Hospital patients is more likely to be misleading and erroneous than a history obtained in private cases, but 85% of these cases operated on were In-patients, residing in the Hospital for at least a week, so that having them under constant observation during that period corrects, and more than compensates any faulty observations by the parents or friends.

History

The history relating to adenoid growths in the nasopharynx is of comparatively recent years. They were first accurately noted and described by Meyer(1) of Copenhagen in 1870 who designated them "adenoid vegetations".

Before this time, as far as I have been able to ascertain, we only possess the description of 5 cases of diseases of the nasopharynx which could be interpreted as cases of adenoid vegetations.

Voltolini(2) of Breslau, a few years previously, described two cases in which he noted cylindrical growths in the nasopharynx, situated in the first case on the roof and the side walls, and in the other spread over the whole surface of the cavity. In the first case mention is only made of the deafness of the patient, but in the second a few of the symptoms of adenoid vegetations are described.

The remaining three observations are

(1) 'Archiv fur Ohrenheilkunde' 1868

(2) 'Hospital Tidende' 1895 N° 647

by Lowenberg[1], of Paris, who describes vegetations in the nasopharyngeal cavity under the title of "pharyngitis granulosa" but makes no mention of any symptoms present.

Before this time the symptoms of adenoid vegetations were evidently looked upon as caused by enlargement of the tonsils, although most authors recognized that excision of the tonsils was not always followed by the relief of the symptoms complained of.

In order to detect how far back undoubted evidences of the existence of adenoid vegetations may be traced, Meyer[2] has studied the various portraits and busts in numerous European collections, and as a proof of adenoid vegetations at the beginning of the present century numerous portraits of the eminent sculptor Canova are mentioned. They all depict the artist with an open mouth, narrow nose and languid expression; and Canova it is said suffered from deafness. He states that several Ancient Roman statues and busts show undeniable evidences that adenoid vegetations existed as far back as Roman art goes, whereas he did not find a single instance which might serve as proof of the existence of adenoid vegetations in Ancient Greece. This may be explained by the fact, that,

(1) La Maladie et la Mort de François II, Roi de France, J. ap. 1894.

Greek artists had a tendency to idealise the human features, and we might scarcely expect to find an adenoid physiognomy unaltered to the Grecian idea of beauty and grace.

M. Potiquet[1] a few years ago made a contribution to the medico historical literature of this subject. He states that the young King, Francois II of France, the first husband to Mary Queen of Scots, showed many unquestionable evidences of having suffered from "adenoid vegetations, which may be regarded as the indirect cause of his death. He was described as an unwholesome, anaemic youth, with open mouth and marked dulness of intellect, and was known to be dull of hearing. He died of meningitis and intracranial abscess. In their rude pathology the process was expressed as a "formation of corruption in one of his ears which discharged the function of the nose", or, as Henry II expressed it, "the humours of the little king were discharged by a wrong channel owing to his not being able to blow his nose"

Meyer in his original article gave so full an account of the disease, that subsequent observers have really only corroborated its accuracy.

Anatomy and Physiology of the Nasopharynx.

The nasopharynx is the post nasal space, or the upper part of the pharynx situated above the soft palate and behind the nasal cavities. It is irregularly cubical in shape and cannot be obliterated by muscular action, although its size varies with the movements of the soft palate. Its roof is formed by the basilar process of the occipital bone, together with a small part of the posterior portion of the body of the sphenoid, while it terminates below in an imaginary plane opposite the border of the palate. The posterior wall is formed by the spinal column, the prominence of the arch of the atlas being often recognized at about the point where the vertex of the palatal arch in contraction impinges upon the pharyngeal wall. From this upwards the wall curves forward. The anterior boundary is formed by the two oval openings of the posterior nares, together with the posterior border of the vomer or septum, which presents a somewhat sharp edge below at its articulation with the hard palate, but expands somewhat above to articulate with the rostrum of the sphenoid. Each lateral wall is marked by the opening of the pharyngeal orifice of the Eustachian tube, which presents as a somewhat elongated or ovoid funnel-

-shaped orifice. The opening of the tube is partially surrounded by a well defined cartilaginous ridge, which is mainly formed by a projection of the cartilage which enters into the formation of the tube proper. This point has to be remembered when adenoid vegetations are removed by forceps. The eminence is well marked posteriorly and above, while anteriorly it is less prominent, and immediately below the orifice it is absent. As the mucous membrane is reflected over this cartilaginous ridge or cushion of the Eustachian orifice, as it is usually termed, it is thrown into a fold as it passes from the lower termination of the posterior section of the ridge to the pharynx below, forming what has been designated by Luschka as the plica salpingo-pharyngea, while by its reflection from the anterior portion of the ridge a less prominent fold is formed, which extends from the anterior border of the tube to the soft palate. This is called by Luschka the plica salpingo-palatina. Immediately behind the Eustachian orifice, and lying between the cartilaginous cushion and the posterior wall of the pharynx, is noticed an elongated depression, the fossa of Rosenmüller. This fossa varies in shape and depth in different subjects although it is usually elongated and much broader above than below.

It is from this fossa particularly that adenoid vegetations have to be thoroughly removed to prevent recurrence.

The soft palate forms the floor of the cavity. The body of the axis corresponds with the level of the soft palate, but in the child owing to the small depth of the fall, the body of the axis projects below the soft palate so that it can be touched with a straight probe introduced through the mouth. This is of importance as showing the relative smallness of the vertical diameter of the nasopharynx in children, and also the increase of the vertical measurement that takes place consequent to the growth of the bones of the face at puberty.

The capacity of the nasopharynx varies in children according to age, and also differs greatly in different individuals, without reference to their physical development. I have estimated on the 500 children I have examined that the average capacity is in children of

one year — 2 c.c.
six years — 5 c.c.
12 years — 9 c.c.

The fibrous basement structure of the nasopharynx consists of a thick aponeurosis, which has its attachment to the basilar process of the occipital bone and the petrous portions of the temporals. Beneath this tissue are

(1) "Hyperplasie der Rachen Tonsille"
Berlin 1884

found muscular structures involved in the movement and support of the head. Its internal surface is lined with mucous membrane covered with columnar ciliated epithelium. It is endowed with mucous glands of both the tubular and racemose variety.

The feature, however, which gives the pharyngeal vault an especial interest is the presence of a definite collection of adenoid tissue which stretches across the posterior wall between the openings of the Eustachian tubes. It is known as the pharyngeal tonsil or Luschka's tonsil. The mucous membrane that stretches over the surface is thickened and thrown into a number of folds.

According to Trautmann[1] the pharyngeal tonsil may be divided into 2 parts, an anterior and posterior: the former is subdivided by 6 antero-posterior clefts, whilst the latter is marked by furrows running transversely outwards towards the fossa of Rosenmüller.

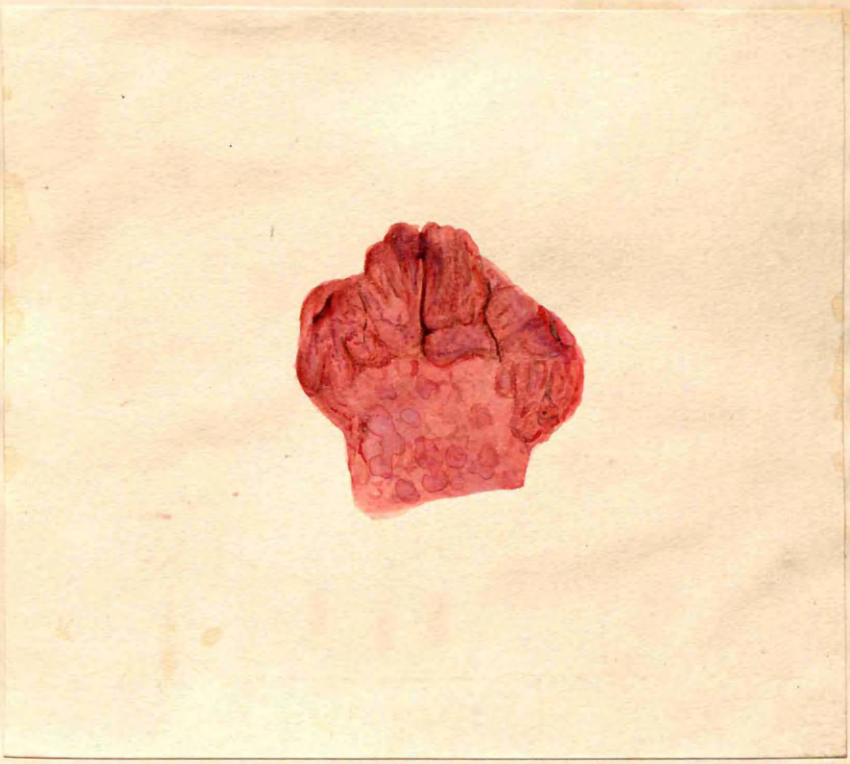
In a number of cases I have examined post mortem this division has always been apparent but not so definitely subdivided as indicated by Trautmann.

The two drawings (page 9) taken from post mortem preparations, present the posterior and lateral walls of the nasopharynx folded out with the pharyngeal tonsil in the posterior

'Normal' Nasopharynges

posterior + lateral walls

Drawn from P.M. preparations



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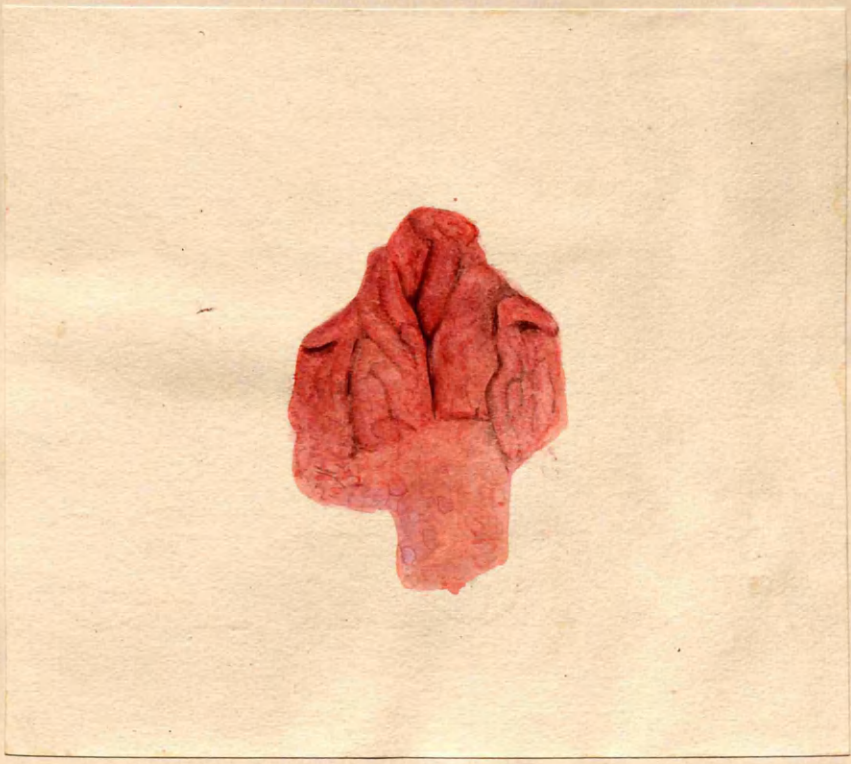


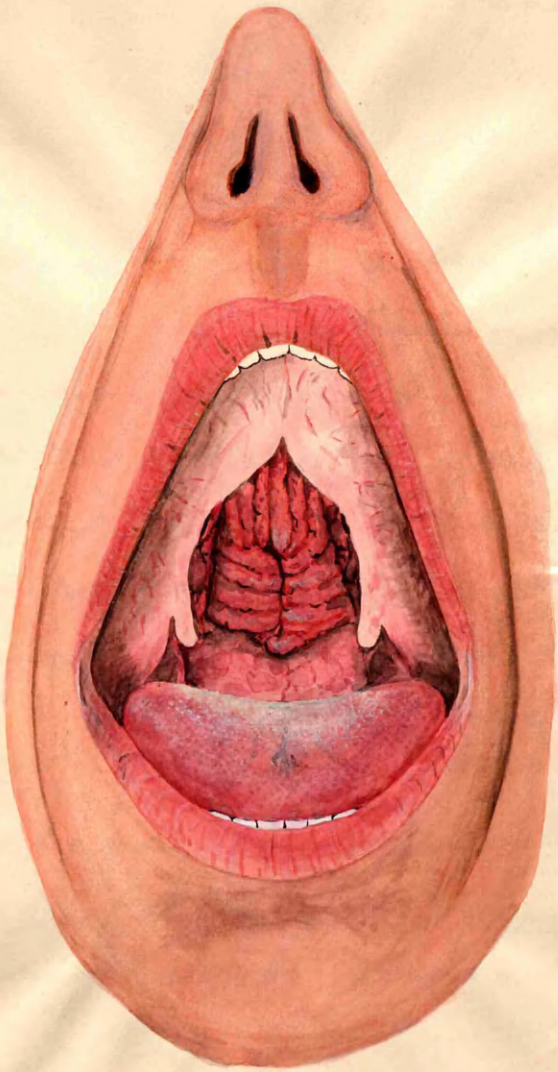
- Nº 2 -

wall. They also exhibit adenoid tissue on the lateral walls around the orifices of the Eustachian tube: this is important as showing how adenoid hypertrophy need not be confined to the pharyngeal tonsil on the posterior wall, but will also likely extend at the same time to the adenoid tissue on the lateral walls.

One constant condition is the division of the thickened mucous membrane into two halves on the posterior wall by a median recess the recessus pharyngeus medius, or pharyngeal bursa as it is often called.

Although the fairly normal nasopharynges I have examined post mortem have not presented the very definite division described by Trautmann, still in one case in the hypertrophied condition, this division was exhibited with almost mathematical accuracy and regularity. This case I allude to had a wide cleft palate and a splendid view of the nasopharynx could be obtained.

On page 11 is a drawing I made of this boy's mouth. Above in the vault of the nasopharynx will be observed 6 longitudinal clefts stretching from before backwards, while below is the posterior portion divided by transverse furrows extending from the



George Rutter, aged 3½, Cleft palate
'Adenoids'

recessus pharyngeus to the fossa of Rosenmüller on each side. In this drawing also are observed the orifices of the Eustachian tubes.

The nasopharynx receives a large supply of blood from the following sources, viz:- from the ascending pharyngeal artery, the sphenopalatine branch of the internal maxillary artery and the tonsillar & palatine branches of the facial artery. From these arteries fine arterioles and capillaries are distributed abundantly to the mucous membrane & lymphoid tissue of the nasopharynx.

The veins form a plexus covering the outer surface of the nasopharynx communicating on the inside of the internal pterygoid muscle with the pterygoid plexus.

Lymphatics are numerous surrounding the follicles: they pass into the superior deep cervical lymphatic glands.

Definition

Adenoid vegetations may be simply defined as a hypertrophy of adenoid tissue normally existing in the nasopharynx. The adenoid tissue we have seen is chiefly collected in a mass in the vault & posterior wall of the post nasal space forming what is usually designated the nasopharyngeal tonsil, but also extends, as shown in the drawings on page 9 over the lateral walls into the fossa of Rosenmüller and around the Eustachian tube orifices.

To ascertain exactly the extent of the increase of adenoid tissue in the nasopharynx that constitutes a pathological condition, and the frequency of this affection in children making no complaint of the throat or nose, I examined the nasopharynx in 500 cases at the Out-patient Department of the Hospital with the result that I considered in 34 patients, or 6·8%, there was distinct adenoid hypertrophy.

Aetiology

Age is an important aetiological factor in the development of adenoid vegetations in the nasopharynx. All authors agree that it is almost entirely a disease of youth. In early life the secreting and absorbent glands are in greatest activity: after

(1) Edin. Med. Journal, April 1897

puberty the balance is in favour of absorption, because secretion and nutrition are carried on more slowly than in youth. In youth then it is probably the activity of the glands which predisposes to hypertrophy at that period.

In the 214 cases operated on the ages were as follows :-

Under one year	– 3 cases		
1 year	– 5 cases		
2 years	– 10 "	0 – 5 years	
3 "	– 12 "	73	
4 "	– 18 "		
5 "	– 23 "		
6 "	– 31 "		
7 "	– 22 "	6 – 10 years	
8 "	– 16 "	98	
9 "	– 14 "		
10 "	– 15 "		
11 "	– 12 "		
12 "	– 10 "	11 – 15 years	
13 "	– 8 "	42	
14 "	– 7 "		
15 "	– 5 "		
16 "	– 1 "		

This table agrees generally with similar ones of authors who have contributed this subject e.g. McBride & Turner[1] who give analyses of 500 cases as follows.

0 – 5 years	– 57 cases
6 – 10 "	– 141 "
11 – 15 "	– 115 "
16 – 20 "	– 86 "
21 – upwards	– 101 "

(1) "Guy's Hospital Gazette", Oct. 1890.

The adenoid vegetations occurring in the 101 of Mc Bride's cases in patients over 21 years were small, and he designates them chiefly "adenoid remains".

Symonds in 107 cases noted that 90 were under 15 years of age.

It will be observed from the first table that 3 cases necessitating operation were children under one year, and one of these children presenting well marked adenoid vegetations was only 3 months old. From the above data it may reasonably be concluded that adenoid vegetations in the nasopharynx may occur at an early age, even in the first months of infancy, & that the tendency to the formation increases with years until the 5—7 years are reached, and afterwards that the liability to their development gradually gets less.

When these growths are detected so early in life it is quite possible that some of them may be congenital.

In connection with these statements there are one or two points that have to be remembered, viz:- that, the ages given are those when the operations were performed but the symptoms of 'adenoids' had been observed in most cases months or years previously, so that would tend to place the presence of 'adenoids' more frequently at an earlier period than tabulated. In my table of cases it will be seen that

73 cases occurred between the ages of 0 – 5 years against 140 between the ages of 6 – 15, whereas in the table of Mc Bride & Turner only 57 occurred as against 256 in the same periods. I am of opinion, then, that adenoid vegetations occur and will be found & looked for more frequently in the first five years of childhood than is generally imagined. Again, the age limit of the Hospital being about 14 years, the comparative numbers occurring in later periods of life are by necessity excluded from the table.

Sex

Meyer thought that adenoid vegetations were more frequent in boys than in girls. Mr Symonds(1) reports differently, in his 107 cases, 65 were females & 58 male patients.

In Mc Brides 500 cases there were 263 males & 237 females. The 214 cases I bring forward also support Meyers supposition, there being 115 boys and 99 girls. The difference however is so small that it would not appear as if Sex exerted much influence in the aetiology.

Heredity

There can be little doubt that the members of certain families are specially liable to suffer from adenoid vegetations, as several children of the same parents are often similarly

affected. In my 214 cases,
42 had one brother or sister with well marked symptoms of adenoids,
10 had 2 brothers or sisters,
4 had 3 brothers or sisters, and in one family, composed of 7 members, the entire number presented in youth symptoms of 'adenoids' and some of them were operated on for their removal.

I have found it impossible to give any definite data regarding the occurrence of 'adenoids' in the parents of the children. This may be explained by the fact that the detection of 'adenoids' is a matter of recent years, and vegetations, although they had existed in the parents would have formerly escaped notice. In one family however where 2 children were affected, an uncle & 2 aunts had been operated on for the removal of adenoid vegetations.

As the total result of these figures it may reasonably be concluded that family predisposition is a strong element in the aetiology of adenoid vegetations.

Climate

Most authors suppose that a cold damp climate is most favourable for the development of these growths, but the geographical distribution of the disease is as yet too imperfectly known to enable one to state

(1) Hospitals-Tidende, 1895, No. 647.

2 Journal of Laryngology, June 1893.

(3) Lagow Annual, 1889, Vol IV D 23

positively the full bearing of climatic conditions in relation to adenoid growths.

Meyer[1] in an elaborate article on the distribution of these vegetations, endeavoured to collect evidence of the existence of adenoid growths in different parts of the world and in different races. He stated that Dr. Helme found that in 60 Esquimaux children, between 6 & 14 years of age, only 16 were free from adenoids, while the rest, - 73·3% - suffered. In South Dakota, Dr. Quarry reported the frequent occurrence of adenoids. Dr. Cantlie of Hong-Kong states that adenoids are very prevalent amongst the natives there. Meyer concludes that adenoid vegetations are found with about equal frequency in Europe, America & Asia, but that a warm climate seems less favourable to their development than a cold one. Dr. Hamilton[2], of Adelaide, states that adenoids are very common in South Australia & that he has seen as many cases from inland dry regions as from the coast. Dr. Walker of New Plymouth, New Zealand, in a letter to the present writer stated that adenoids were very prevalent amongst the Maories.

Massei[3] affirms that adenoid growths are rare in Italy, and those

(1) 'Monatschr f. Ohrenh.', Berlin, Oct. 1896.

found seldom present extensive development.

One point at least is certain, that is that it is during cold damp weather that the symptoms of adenoid vegetations become most aggravated and during the Summer months the symptoms are less obvious, so that it would appear that a cold damp climate at least favours the development of adenoids, whilst a warm dry atmosphere tends to retard their growth.

Cleft Palate

It has been noted that in cases of cleft palate adenoid vegetations are usually extensive. The three cases of cleft palate I had amongst the 714 cases substantiate this statement. It is most probable that the irritation of the food in these cases tend to produce the adenoid hypertrophy.

Deaf Mutism

This loss of speech due to acquired or congenital deafness is associated in a large percentage of cases with adenoid vegetations in the nasopharynx.

Frankenberger(1) gives statistics of 59 cases & states that 49% of them presented well marked adenoid growths. In the same paper he states that Lemche found adenoids in 58% of cases of deaf-mutism, and Aldrich places it as high as 75%. In

healthy children on the other hand he reports that Doyer found adenoids in 5% and that both Wroblewski & Rafemann discovered them in 7% of cases examined.

In the 1,500 children I examined I found adenoid vegetations in 34 of them = 6.8%. It would appear then, with such a larger percentage of deaf-mutes presenting adenoids, that adenoid vegetations causing deafness may be an important and frequent cause in the development of acquired deaf-mutism. This supposition is the more plausible when we remember that extensive adenoid vegetations may be present in infancy before the child learns to speak. These vegetations may, as I will afterwards show, lead to sclerotic changes in the tympanum with subsequent deafness.

In my 214 cases I had 3 deaf-mutes all presenting extensive adenoid hypertrophy with 'adenoid' symptoms of long duration. all three were able to say the primitive words papa & mama and in two of them there was a history of a prolonged double otorrhoea in childhood. One of the three, a boy of 7 years old, acquired quite a large vocabulary when he was two years of age. Subsequent to this however he gradually lost most of his words

and when admitted to Hospital could only say papa, mama, penny bread, milk. This boy and one of the other two, a girl of 6 years, could apparently hear the tick of a watch by bone conduction. Six months after operation in both of these cases the mothers stated that they considered their children heard a little since the operation and gave as examples, that the boy would look round when she came in at the door even when his back was turned to her, that he had gone suddenly to the window and pulled up the blind when a hand was passing the house playing: that the girl had looked round when the mother had accidentally dropped a plate behind her. In both these cases we may reasonably take it that hearing returned to a slight degree after removal of the adenoid growths but not sufficiently to effect a learning of speech. These cases may be quite well explained by the occurrence of adenoid vegetations in infancy with inflammatory extention to the ear and subsequent deafness that would interfere with the learning of language. With an advanced sclerotic process in the ear a return of hearing after operation would only

be partial, & not likely to be followed by an improvement of speech except with careful training.

From these cases I consider that the presence of adenoid vegetations in the nasopharynx may be the primary cause in a number of cases of acquired deaf-mutism in children, which if operated on early, before sclerotic changes occurred in the tympanum, would in all probability prevent the deafness, and allow speech to be naturally acquired.

As exciting causes may be mentioned cold, repeated nasal catarrh inducing gradual chronic rhinitis, with chronic congestion and discharge of secretion differing somewhat from normal. The more or less acrid secretions in the nasal cavities replace the bland healthy ones, and pass back constantly over the pharyngeal tonsil which having to 'tackle' irritating secretions gets swollen & inflamed. The smallness of the nasopharynx in the child, with the chronically inflamed pharyngeal tonsil leads to the early blocking of the nose, which still further favours the retention, stagnation & decomposition of the altered discharge that keeps up the irritation and induces in time destruction of the epithelial lining and hypertrophy of the adenoid tissue.

In the same way the Specific

fevers especially scarlet measles, diphtheria & whooping cough, if great watchfulness is not exercised in the convalescence chronic inflammatory changes are liable to be set up in the nasopharynx which lead latterly to hypertrophy. The regurgitation of food in infancy has the same effect, setting up chronic inflammatory changes followed by hypertrophy of the adenoid tissue.

We shall see later in the section of pathology the almost constant association of pyogenic organisms with these vegetations

Symptomatology and Semeiology

Effect on the Lungs and Respiration.

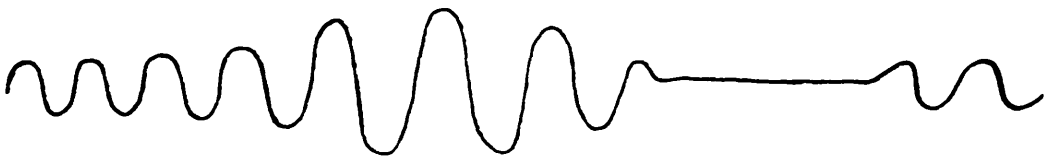
Children affected with adenoid vegetations in the nasopharynx have a difficulty in breathing. This dyspnoea varies generally according to the extent of the occlusion of the post-nasal space. At first the obstruction to the nasal breathing is slight, and scarcely perceptible, and is only revealed by the child opening his mouth to breath after the least exertion, or it is often first observed with the increased congestion attendant on attacks of 'sore throat', and mothers will then state that the child breathes badly and constantly snores at night when it has a 'cold in the throat'. As soon as the 'cold' disappears the breathing becomes calm and easy.

This difficulty of breathing gradually increases with the growth of the adenoid vegetations until nasal respiration is impossible, and, of necessity, a habitual mouth breather produced. The oral respiration is constant where the nasopharynx is entirely occluded or where the obstruction is too great to allow the required amount of air to pass during inspiration and expiration.

When the cavity is only partially obliterated, as in the latter condition often three four or more small

inspirations and expirations are taken entirely through the nose, followed by two or three longer and stronger oral respirations. The mouth breathing comes as a relief, and is followed by a pause or period of apnoea from 2 to 4 seconds.

It will be seen that here we have a condition that simulates closely Cheyne Stokes respiration, and clinically may be mistaken for it in some cases.



A B C

A short nasal respirations.
B stronger oral respirations.
C period of apnoea.

It can be easily distinguished however from the typical Cheyne Stokes breathing by the smaller respirations being nasal, and the increasing and larger inspirations oral: also, the waxing and waning of the amplitude of the respirations are not so smooth - the declining stage being more [illegible] & the succeeding cycles not so constant as in the usual Cheyne Stokes respiration.

This condition I have found typical in 16 cases & it has been

most marked during sleep. It is usually, but not always, accompanied by some snoring. It may be explained by the partial asphyxia during the imperfect nasal respiration that causes stronger respiratory movements sufficient to produce oral respiration. When the asphyxia is relieved the stronger respiratory movements abate.

In some children, even with little adenoid hypertrophy, the breathing is constantly oral: this is usually caused by some previous attack of inflammation that has occluded the nasopharynx, and, at the time necessitated mouth breathing, and before the inflammation subsides the habit is formed and continues.

Occasionally the ~~inspiratory~~ act can be performed apparently naturally through the nose whilst nasal expiration is impossible. The reverse equally holds true, but is not so common. These anomalies are explained by some valve action of the adenoid growths according to their position & size. By obliteration of the nasopharyngeal space or by the above valve action sneezing or blowing the nose is often impossible.

Snoring during sleep is almost constant in adenoid hypertrophy, and it is this that generally attracts forcibly the attention of the parents and

(1) Clinical Manual, 2nd Edition, p. 398.
(3) Text-Book of the Principles & Practice of Medicine, 3rd Edition, Vol I, p. 879
(2) A Practical treatise on Diseases of Children, 2nd Edition, p. 281

causes them to seek advice regarding it. Parents describe this snoring in some cases as 'terrific', & as 'can be heard across the street'. With the nasal obstruction, and the increased salivation that often takes place in these children, and the accumulation of thick viscid se-cretion that collects at the back of the throat, some of these cases are in imminent danger of suffoc-ation and wake up in terror often half asphyxiated and spluttering.

Should it be wondered that in such a condition as this we might expect to get a reflex spasm of the glottis - laryngismus stridulous-occurring?. I think not, and adenoids in these cases, if their presence is sought for, I consider will rarely be found absent. In no less than 48 cases were these attacks found to occur at some period, and in some of them several sudden onsets of this affection. Laryngismus Stridulus has been universally associat-ed with, or attributed to Rachitis by Finlayson[1], Eustace Smith[2] Fagge[3] etc. Eustace Smith states that the association of laryngismus stridulus with rickets is indisputable, but goes on to say that it is important to remember, in investigating this point, that the patients do not always show a marked degree of

(1) Journal of Laryngology + Rhinology, Oct. 1891

rickets; exactly the degree that they often show is just such as to mistake the appearance of the pale flabby lethargic backward, restless at night adenoid child for rachitic manifestations. In 3 of the 48 cases there were definite signs of rickets but this percentage is so small as to cast a grave doubt whether the rachitic diathesis enters at all into the aetiology of laryngismus stridulus. The cases I bring forward at any rate show how frequently the presence of adenoids is accompanied by this spasm of the glottis, and would appear to justify the conclusion of how largely this factor, which has formerly been overlooked or neglected, enters into the aetiology of laryngismus stridulus.

Dr Robertson[1] pointed out that infantile respiratory stridor, crowing during inspiration, is seldom unaccompanied by adenoids. Dr Robertson attributes the stridor to bilateral paralysis of the posterior crico arytenoid muscles which expand the rima glottidis, caused by reflex action by stimulus carried to the medulla. This condition appears to me more spasmodic than paralytic, but at any rate the removal of adenoids in the nasopharynx cured the spasm in one of my cases - the only one

amongst the 214 who presented this special symptom.

It is a well established fact recognised by all authors who have studied nasal obstruction how prone the subjects of this defect are to inflammatory affections of the whole respiratory tract. When there is obstruction to nasal breathing in children affected with adenoid vegetations in the nasopharynx, the natural heating, moistening, and filtrating nasal chamber is cut off & the mucous membrane of the throat larynx & lungs is directly exposed to inspired air of various temperatures, devoid of the bland humidity, but presenting all the deleterious and irritating particles & germs of disease contained in the atmosphere. In this way these children are victims of 'sore throat', laryngitis, bronchitis, pneumonia, phthisis pulmonalis, etc., and a common expression with parents is that the child is "never without a cold & cough". Chronic bronchitis with emphysema is the most frequent lung condition associated with adenoids. I have estimated that no less than 34% have some chronic bronchitis with or without emphysema. By preventing the free expansion of the chest adenoid vegetations tend to retard the growth of the lungs, and small lungs are vulnerable

(1) Montreal Medical Journal, Aug. 1894.

lungs. In the same way, and for the same reason, it is common to find some collapse of the lungs especially in the upper lobes anteriorly: This is associated with deficient resonance and weak harsh breathing, and, when added to this is hollow respiration conducted down from the pharynx, it is easy to imagine how naturally a condition such as this, may be mistaken for phthisis pulmonalis, and a slight bleeding from vascular irritable adenoids may appear to confirm the diagnosis. If the nasopharynx was examined in cases of supposed incipient phthisis, and in many other lung conditions I am sure that, in children at least, an easy solution would be afforded to some of the puzzling disorders of the respiratory system.

Hamilton (1) reported a case of asthma that, after resisting all treatment was cured by the removal of adenoids, & other similar cases have been reported.

In none of my cases have the patients suffered from typical asthma, but a few of them have had 'asthmatic attacks' along with bronchitis and these 'attacks' have not returned since operation.

I have stated that patients suffering from adenoids have small lungs. I took cyrtometric tracings and measurements of the chest at the level of the nipple in 80 cases

suffering from adenoid vegetations, and compared the measurements thus obtained with similar ones taken in 100 cases of healthy children. The following table gives the average measurements in inches in both cases with the age of the patients opposite (See cyrtometric tracings page (122 - 127)

Age last birthday	Chest Measurements with adenoids	Chest Measurements without adenoids
2 years	16 3/4	18 1/2
3 "	17 1/2	19 1/4
4 "	18	20
5 "	18 1/2	21 1/4
6 "	19 1/2	22
7 "	20 1/4	22 3/4
8 "	21 1/4	23 1/4
9 "	22	23 3/4
10 "	22 3/4	24 1/4
11 "	23 1/4	24 3/4
12 "	23 3/4	25 1/4

This table shows the deleterious effect of adenoid growths obstructing respiration and retarding a healthy development of the chest. The chest measurements it will be observed, in cases with adenoids, are 1½" - 2" less than in those without adenoid vegetations, or to put it differently, it shows that the presence of adenoid growths in the nasopharynx so retard the healthy development of the lungs that a child of any age - from above 4 years to 12 years - has

only a chest measurement equal to that of a healthy child 2 to 3 years younger. The importance of this statement need only be mentioned here: its effect on general nutrition is obvious.

Not only is the chest measurement diminished, but the form of the chest is often greatly altered. In the 80 cases in which I took cyrtometric tracings 75% exhibited some departure from normal. On pages 122-127, some of these tracings are shown. It will be observed that they manifest no special type and the deviations from normal mostly resemble the "flat chest", "pigeon-breast", "rickety chest" or present some unilateral retraction: others again resemble the emphysematous chest and are really caused by emphysema. A more or less constant deformity, that exists alone or is superimposed on the above ones in nearly all cases, is depression of the ensiform cartilage and symmetrical retraction of the infra-mammary region.

How far the presence of adenoids causing obstruction to free respiration enters into the aetiology of these chest malformations it is difficult to affirm, but they occur with such frequency in these cases as to lead an

unbiased observer to suppose that these adenoid growths enter largely into their causation, either directly by the obstruction to breathing, or indirectly through lung complications.

When it is remembered that the chest measurements are so altered in cases of adenoids and how marked is the improvement following operation, it can be more easily imagined how often adenoid vegetations may cause these deformities of the chest

Effect on the Ear & Hearing

It is for the treatment of ear affections that many of these children have visited the Hospital. The guardians state, usually, that their children are dull of hearing or have a discharge from or pain in the ear. Many of these cases though revealing a typical adenoid physiognomy have made no complaint or reference to the throat or nose, but the nasopharynx is nevertheless the starting point of these morbid changes although, alas, it generally receives little attention.

The treatment in these cases is not as a rule directed at the primary lesion but confined to the ear affection per se. It is a well established fact recognised by all authorities on this subject how frequently

(1) Med. Chir. Trans., Vol LIII, 1870.

adenoids are associated with ear affections. Meyer[1] in 175 cases of adenoid vegetations reported no less than 130 with an ear trouble. Bonner found in 90% of his cases the ears affected.

In the 214 cases I bring forward, there was some ear affection in 186 of them, that is in 87%.

The ear being in direct communication with the nasopharynx septic infection can readily extend either along the lymphatics of the Eustachian tube, or directly through the lumen of the Eustachian tube itself. In either of these ways an otitis media purulenta, of any degree of intensity, may be established, attended with the usual subjective and objective symptoms of acute or chronic suppuration in the middle ear. Again, the adenoid vegetations in the nasopharynx may by their direct mechanical pressure interfere with the natural discharge of secretion from the Eustachian tube and tympanic cavity, causing it to be retained and thereby exciting inflammatory changes in the middle ear. The chief consequence of these ear affections in the 186 cases has been deafness. In every one of them that could be properly tested there was impairment of hearing to a certain degree. The loss of hearing has varied from unobserved

dulness to complete deafness. In some the tick of the watch is heard well whilst ordinary conversation is scarcely recognisable & vice versâ.

The dulness of hearing varies from time to time, being worse during the recurrent attacks of inflammation. The extent to which the hearing is impaired is in almost all cases greater, I think, than what is generally supposed. It is impossible to give accurate statistics of this point in children but from the cases where reliable answers could be depended upon, I have estimated that over 60% are unable to recognise the tick of a watch, that should normally - in the one used - be heard at 40 inches, until it is placed within 10 inches from the ear. On pages 111-113 I give a series of cases where accurate records could be made, and these may be looked upon as fairly typical average impairments of hearing.

During an acute attack of inflammation in the middle ear the mucous membrane lining the tympanum and recesses & covering the ossicles is swollen & thickened. There is a copious secretion and usually a perforation of the membrane and discharge. These changes interfere with the smooth transmission

of sound along the ossicular chain, and hearing is impaired. When the inflammation has become chronic sclerotic changes develop in the tympanic lining with scarring and indrawing of the tympanic membrane, binding and rigidifying the ossicles, that destroy all elasticity and render almost impossible the reception and transmission of the delicate waves of sound. In children with adenoid vegetations in the naso-pharynx the inflammation in the middle ear is always prolonged, owing to the obstruction in the upper pharyngeal cavity that constantly excites and keeps up the otorrhoea.

The history in most cases is an otorrhoea, which has lasted for years, varying in intensity from time to time, being more aggravated with each recurrent attack of inflammation in the nasopharynx. It is usually greater during the Winter & Spring & subsides somewhat during the Summer months

In my 214 cases there was a history of past suppuration in one ear in 90 of them, and in both ears, in 7 cases. In 39 cases there was active otorrhoea in one ear, and in 12 both ears were 'running'. This gives a

(1) 'Bulletin de la Société Anatomique' Paris, March 18th, 1892.

total of 78 cases in the 214 or 36·4% presenting an otorrhoea at some time or other.

In the 78 cases the otorrhoea extended to :-

1. -Acute inflammation in the mastoid cells, in 8 cases.
2. -Acute periosteal mastoid abscess, in 9 cases.
3. -Caries & necrosis, in 4 cases.

I have no doubt that in many others these processes occurred to a minor degree, but these 21 cases came under my observation & required immediate operation.

The impairment of hearing may occur apart from any septic extension from the throat as follows :-

1. By the retention of the normal or abnormal secretion of the tympanum and mastoid cells, or antrum. This secretion when it accumulates, forms through time, a kind of cholesteatomat-ous mass in the tympanum and mastoid cells, that seriously interfers with hearing and is often followed by inflammatory changes.
2. By, according to Pilliet(1) by sclerotic processes occurring in the adenoid stroma of the mucous membrane of the Eustachian tube and middle ear. These sclerotic process I would expect would be preceded by one or

more attacks of acute non-suppurative inflammation of the middle ear and would most likely be the result of such attacks.

3. By occlusion of atmospheric air from the cavity of the tympanum.

The impairment of hearing has been accounted for by some e.g. by Yearsley[1] who holds that the sounds reach the ear more readily through the Eustachian tube than by the external auditory meatus, and gives as a proof that deaf persons often open their mouths to hear. As against this supposition we have the easily appreciated fact that shows the opposite, namely, that a watch held in front of the open mouth is not heard so distinctly as opposite the ear.

Other physiologists believe that the freedom of the Eustachian tube is necessary to admit of the motion of the air in the tympanum, when it vibrates under the influence of the membrane of the drum. It is not the vibration of the air in the tympanic cavity that produces the sound, however, but the transmission of the vibrations along the ossicular chain from the membrane of the drum.

The true explanation of the loss of hearing by the closure of the

Eustachian tube appears to me to be more likely explained by the statement of Waller(1) named, that hearing is temporarily dulled whenever air-pressure within the tympanic cavity is less or greater than that upon the outer surface of the membrane. This statement can be experimentally proved by artificial inflation or extraction of air from the tympanic cavity.

When the tube is closed the vacuum caused by loss of air in the tympanum places the membrane of the tympanum under atmospheric pressure. The membrane is pushed in and made tense, and its vibrations on the impulse of sound are greatly impeded. This sucking in or pushing in of the membrane of the ear I have rarely found absent in cases of adenoids. It may of course be drawn in by advancing sclerotic processes in the tympanum, but this pushing in of the membrane by atmospheric pressure that I allude to here, occurs chiefly in non suppurative cases.

Effect on the voice & Speech

The degree to which the voice or speech is affected in adenoid vegetations of the nasopharynx varies according to the situation and extent of the hypertrophied masses.

If the changes are slight amounting only to a thickening of the mucous membrane in the nasopharynx, the alteration in the voice is correspondingly slight and articulation is simply dull and resembles that of a chronic cold. When the hypertrophy of the adenoid tissue increases the dull phonation becomes more aggravated and constant. The voice loses its tone and natural variations and flexibility, that add so much to its expression and becomes altered to an unvarying monotony which has been designated by Meyer as "dead". It is often termed "nasal", a singularly inapt expression, as it is the want of the resonance in the nasal chambers, and the absence of the harmonies developed in the nasal cavities that largely produces the 'nasal' tone. When the obstruction is complete or where the breathing is much impeded, the voice habitually presents the "dead" characters. The m and n sounds are represented by b and d sounds, so that instead of "common" the patient says "cobbod", instead of "nine", "dine" and so on. In many cases with the monotonous, thick, indistinct, blurred phonation it is even impossible for the initiated to understand, so that parents who are accustomed to such pronunciation from their

(1) Quain's Anatomy, vol iii, part iii, p. 140.

children often find it quite impossible to follow a sentence. The want of confidence thus established in these children renders their speech still more backward and uncertain, and this is easily appreciated by the manner the altered speech is dribbled out in an indistinct low mumble. The dulness of hearing usual in these cases has a certain effect in preventing them from acquiring orthoepy.

Stammering has been observed in some cases of adenoid hypertrophy. It has not been present in any of the cases I bring forward.

Effect on Sense of Smell

Before any odour can be appreciated it is necessary for the odoriferous particles to come into contact with the olfactory cells in the Schneiderian membrane. The olfactory region, or that part in which the olfactory nerve is distributed includes in man only the uppermost part of the nasal fossa, (superior turbinal & corresponding part of the Septum[1]). When the nasopharynx is occluded there is no nasal respiration and the odoriferous particles do not reach the olfactory cells, so that in these cases I have found the sensation of smell entirely absent. The Sense of Smell was examined in

most of the 214 cases but excluding those children too young to give reliable answers, and those too dull of hearing or stupid for credence to be placed on their statements, I have estimated that 43% had apparently no sense of smell, 33% presented great impairment while the remainder 24% responded fairly readily to the test and answered correctly. These tests were carried out by the penetrating well known odour of a raw onion.

In a number of cases where the nasopharynx was not entirely occluded with adenoid growths, the sensation of smell was apparently entirely lost. This may be explained by the fact of the air current being too weak, and not carried to the upper part of the nasal fossa, or, by the narrowing of the nose with or without Septal deviation preventing the current from reaching the olfactory region, or, by inflammatory changes or alteration in the nerve cells themselves.

Effect on the Sense of taste

Taste is in a large measure affected by aroma: anything that suppresses the sense of smell tends to lessen the acuteness of taste. In adenoids then, where according to the above statistics 43% have the abolition of smell,

and 33% have it impaired we would expect to find the sensation of taste lessened, and such it is, but it is more difficult to find exactly how far it is interfered with. This may be explained by the greater difficulty in carrying out the test, as one trial almost spoils subsequent ones at the same time. In 55 children examined where definite and reliable answered could be obtained, 4 could not distinguish salt from sugar, 23 could appreciate both after some hesitation or on a second trial, showing distinct impairment, whilst the remaining 28 answered correctly + readily.

The cases where the sensation of taste was most impaired were habitual mouth breathers. It may be remarked, that the constant oral respiration may have a direct effect on the taste bulbs. The mouth tissues in most of these cases are in a chronic inflammatory condition, but in a large number of them where a little congestion is just manifested there are traces of past ulceration. It is easy to imagine, then, how by ulceration or by slight superficial inflammation even amounting to a constant congestion, that sclerotic changes would develop and compress the delicate taste bulbs. This

appears to me to be the most likely explanation but I can offer no definite proof beyond the clinical history and macroscopic pathological appearances of the tongue

Pathological analogy of other chronic inflammatory conditions would support this view regarding the development of sclerotic changes around the taste bulbs.

Effect on the organ of sight

This is a subject that has been practically neglected, and indeed it may appear at first sight rather imaginative to suppose that adenoid vegetations in the nasopharynx can have any influence in causing eye affections; still such is not the case and the presence of eye affections more or less connected directly with adenoid growths is not a supposition but a reality.

In children suffering from adenoids mouth breathing is generally habitual at night, and with this oral respiration the cheeks are stretched depressing the lower eyelid & exposing the eyeball to atmospheric influences. This imperfect closure of the eye during sleep is present in about half the cases, and leads in most cases to some inflammatory

changes in the eye. These changes are usually slight amounting only to a hyperaemia of the conjunctiva. This hyperaemia may give little trouble, and only in the morning when the child awakes are their eyes irritable and the eyelids heavy. This condition however in a number of cases extends to some of the forms of conjunctivitis with abnormal discharge, photophobia and lacrimation, or marginal blepharitis may be caused. In the same way by constant exposure of a portion of the surface of the eyeball during sleep allowing the dust particles and microorganisms an entrance to the eye, phlyctenular ophthalmia may be developed or corneal ulceration. In the 214 cases, not including hyperaemia which is very common, there have been 10% of them affected with these or one of these eye affections, so that I have no hesitation in affirming that adenoid vegetations enter largely into the aetiology of these eye affections. Again just as pyogenic infection travels by the Eustachian tube to the ear so also it extends by the nasal duct to the lacrymal sac and affects the eye. The extension of septic inflammation to the eye is less common than to the ear

owing to the situation of the orifice of the nasal duct & to the constant washing of the duct with lacrymal secretion.
In two cases however this retention occurred and was in both cases reasonably attributed to extension from the retained and abundant muco-purulent discharge from these adenoid growths.

More interesting and important is the effect they sometimes have on refraction and accommodation.
In 4 cases, out of a number that complained of headache & indistinct vision, a form of astigmatism that is not "with the rule" was found to be present i.e. cases in which the meridian of greatest curvature was not the vertical as is usual but the horizontal

The astigmatism in all these cases was compound and the factors of the astigmatism may be noted as for glasses to correct it as follows:-

1. R. eye, +.75 D sph: + 1D cyl. ax. horiz.
   L. eye +1.5 D sph: + 1D cyl. ax. horiz.

2. R. +2 D sph: + 1.5 D cyl. ax. horiz
   L. +1.5 D sph: + 1.5 D cyl. ax. horiz.

3 R. +1·5 D sph ⊃ +1·5 D cyl. ax. horiz.
L. +3 D sph ⊃ +1·5 D cyl. ax. horiz.

4 R. +1 D sph ⊃ +·75 D cyl. ax. horiz
L. +1·5 D sph ⊃ +·75 D cyl. ax. horiz.

I have stated that it was only in a few cases that complained of defective eyesight that the eyes were tested, so that I am unable to give accurate statistics of how often this form of astigmatism occurs in cases of adenoid vegetations, but I have no doubt had the others been examined more would have been found to present this form of astigmatism

As an explanation of this compound astigmatism not "with the rule" I have a theory to offer, which at any rate explains thoroughly what is really found to exist we shall see later how the obstruction to the nasal breathing interferes with the growth of the antrum and a glance at the photographs on pages 117+118 show the falling in of the face over that cavity. In the same way then, the floor of the orbit that forms the roof of the antrum will be likely depressed, and this will increase the vertical diameter of the orbital cavity. In this way the natural

support of the eyeball will be diminished in its vertical meridian while not interfered with in its horizontal meridian. The effect of this on the eye will be that the pressure is relaxed in the vertical axis whilst the normal pressure is exerted in the horizontal. This will tend to increase the vertical diameter of the eye (Fig A) while at the same time diminish the curvature in the vertical meridian (Fig B)

Fig A.

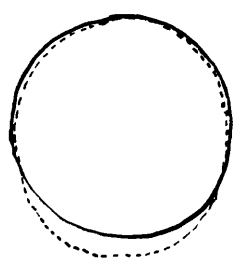


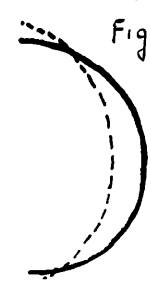
Fig B

The dark line indicates the normal eye: the dotted line the alteration.

The alteration in the curvature of the cornea will be general, but chiefly confined to the vertical meridian, so that besides the hypermetropia produced by the general effect of lessening of the corneal curvature we will obtain astigmatism by the unequal alteration in the two planes, and that astigmatism will be not "with the rule" because in this case the greater curvature will be horizontal (Fig C) and the lesser one vertical (Fig D) ~~n~~ (Fig B)

Fig C

Fig D



(1) "Transactions" American Langng. Association 1887

Effect on the features and bones of the face.

The distortion of the features in subjects with well marked adenoid hypertrophy is one of the most interesting and striking characters of the disease. Most cases present what may be termed the 'adenoid physiognomy' to some degree, but it is only children in whom the obstruction to respiration has lasted for a long period that a marked distortion of the features is exhibited. The earlier the onset of the hypertrophy of the nasopharyngeal tonsil and the more complete the obstruction to nasal breathing, the greater is the facial deformity.

We have seen how the obstruction in the nasopharynx necessitates mouth breathing, and D. Delavan[1] has stated that a diagnostic sign of a habitual mouth breather is a high arched palate associated with deflection of the nasal septum, and the analyses of the cases the present writer brings forward coincide with that opinion.

In my 214 cases, no less than 168 had high arched palate and the 46 children, in whom no distinct elevation was present, the adenoid hypertrophy was recent or the obstruction was so slight as not to necessitate

oral respiration.

The height and character of the arch varies according to the degree of the obstruction, the age of the patient and the duration of the adenoids.

When the nasal respiration is impeded in a growing child during first dentition, the palate assumes a higher elevation and in course of time appears dome shaped instead of a slight curvature as in the normal palate (Fig A + B or plaster casts)

Fig. A.

Fig. B

vertical sections
palate

The alveolar margin which in a healthy child is semicircular (Fig C or Casts) now forms an ellipse (Fig D) so that

Fig. C

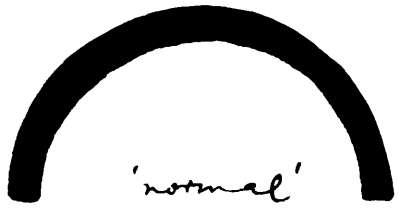
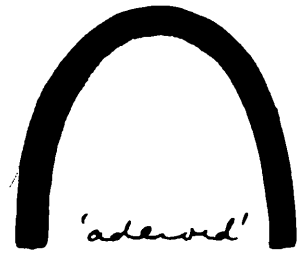


Fig. D



alveolar margin

the transverse diameter is shortened, whilst the antero-posterior measurement is increased. The whole alveolar process appears small but the temporary

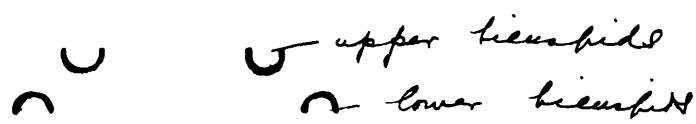
teeth are all in their natural position and as a rule no bend or angle is observed anteriorly during first dentition. As age advances the deformity of the palate still increases, the arch becomes higher and may become pointed above. The labial alveolar margins approach each other still further and the anterior alveolar margin forms an angle instead of a curve as formerly, assuming the V shaped form familiar to dentists, whilst the anterior portion of the alveolar process becomes protruded and instead of being vertical as in the normal bone, slides to an angle of 60° degrees or so. During these changes the whole growth of the upper maxilla is delayed.

These changes affect the size and position of the teeth. As long as the temporary set of teeth are present they appear arranged in the elliptical curve which gradually may become elevated anteriorly, so that when the mouth is closed a △ space is formed, the lower incisors forming the base and the upper incisors the sides as:-

By the period of second dentition the maxilla has assumed more or

less the v shape. The central incisors now do not fall anteriorly but are turned sideways, the inner surfaces facing each other. The other teeth are small and are crowded together owing to the retarded growth of the superior maxilla and are often irregularly arranged.

The inferior maxilla during this period is undergoing natural growth, so that with the narrowing of the lateral diameter of the upper maxilla the upper bicuspids or molars may fall within the lower bicuspids or molars as:-



Again, by the protrusion of the anterior portion of the upper maxilla the upper incisors are often carried far beyond the lower ones and by their oblique position make perfect approximation impossible:-

The teeth in the upper maxilla being irregularly placed are more liable to become carious. The constant oral breathing favours this development and indeed it is exceptional to find a healthy set of teeth in these children.

The formation of the high

arched palate may be explained in a number of ways. The growth of the nose is delayed when nasal respiration is impeded just as atrophy or arrest of growth occurs in any organ with disuse. In this way the nasal cavities become smaller and the palate elevated. When the mouth is shut the tongue's dorsal surface rests upon the palate and gives it support, but when the mouth is open, by the oral respiration, the tongue falls to the floor of the mouth and the cheeks are stretched tightly over the face and exert a constant pressure on the maxilla. This pressure is only exerted laterally, not anteriorly, so that the lateral borders of the alveolar process gradually approach each other whilst a projection or bend occurs anteriorly. In this way an angle is formed in front, the lateral margins approach each other and the whole anterior portion is projected forwards relinquishing its vertical position. The approaching of the lateral alveolar processes and the bending anteriorly tend to raise the palate still further.

When adenoid vegetations are developed after the 2nd dentition the characteristic V shaped palate is seldom observed; the teeth have found their natural position

and relations to one another so that a regular arrangement of the teeth is usually present and the palate is high arched and elliptical. It has been stated that when the nasal respiration is impeded the nose does not develop or develops slowly. The nasal bones remain small and approach each other a small narrow nose being the result. This narrowness is more apparent on account of the congestion & swelling that is usual at the root of the nose between the eyes. The nostrils are narrowed and the ala of the nose dimpled in a cicatrix manner giving rise to the appearance of the whole nose being sucked in. It is not only the development of the nose that is impeded but the air cavities entering into it apparently remain small. The maxillary antrum seems arrested in growth and the cheeks sink in below the eyes. The malar bone on either side is drawn inwards and depressed, whilst the external angle of the frontal bone is tilted downwards and inwards with the malar bone. The arrest in development of the air sinuses opening into the nose may be explained by disuse of the nose as a respiratory chamber. The alteration in the position of the malar bone and adjacent process of the frontal bone is caused by the narrowing of the nose, the smallness of the maxillary antrum together

with the constant pull of the cheeks downwards in habitual mouth breathing

These alterations of the bones of the face affect the features, so that besides the narrow nose, flattened dimpled alae & open mouth, we have added the drooping of the outer eyebrow, sinking in of the cheeks with hollows descending one on each side from the inner canthus of the eye downwards and outwards over the antrum of Highmore. The upper lip is short and often raised & protruded, exhibiting a kind of Δ shaped mouth.

The solemn face occasionally presenting a degree of lividity but usually pale & pasty with anxious vacant stare, devoid of almost any expression of intelligence and exhibiting the above deformities in a varying degree make a truly striking picture for a physiognomy of this disease (See photographs pages 117, 118

Effect on the nervous system

It is a universally appreciated fact that severe reflex disturbances in childhood may be 'generated' by even a very minor pathological lesion. In children suffering from adenoid vegetations in the nasopharynx, that constantly impede the important function of respiration and interfere with all the special senses, we

would expect to find various reflex disturbances and such is the case.

Cough. I have found is the most common, a spasmodic single barking cough generally hoarse but sometimes clear and high pitched, occurring frequently apart from any apparent disease in the lungs or larynx.

Laryngismus stridulus formerly mentioned, on page 28, where its frequency and aetiology is discussed.

Headache, giddiness and tinnitus aurium are frequent accompaniments with adenoid vegetations. The pain in the head is sometimes caused by errors in refraction or may be due to congestion but may exist as a simple reflex phenomenon caused by the presence of adenoids.

Menière of Paris reported an interesting case in which intense and daily cephalalgia, of 2 3 months duration, resisting all treatment was found to be due to adenoid growths which when removed brought about complete recovery.

Cases of asthma due to reflex disturbances from adenoid vegetations have been formerly mentioned (page 30).

In 2 cases incontinence of urine was cured by the removal of adenoids, having formerly resisted all treatment. In one case of 'petit-mal' after

(1) Lancet, May 25th, 1895

(2) Lancet, March 2nd 1901

a long course of bromides with little if any effect great improvement followed the excision of adenoid vegetations, so that whereas the patient used to have about 4 or 5 fits a day, now he has only 2 or 3 such attacks.

Eustace Smith[1] has reported cases of general convulsions brought on by adenoid growths. In a number of my cases there was a history of general convulsions but whether or not the adenoid growths caused the onset of these convulsions, it is different to affirm.

Effect on the Mental development

Guye[2] of Amsterdam wrote a most interesting paper on the connection between intellectual torpor and nasal obstruction, pointing out the bad effects of nasal obstruction on cerebral circulation by lymphatic obstruction. This impaired mental function was termed by him aprosexia and the interference with the cerebral lymphatic system was advocated by him to the cause of the delayed mental development in children with adenoid vegetations. There appears to be a certain amount of truth in this statement as by the embarrassed respiration the venous return of blood from the head must be somewhat obstructed, but in

the impairment of hearing I think we find a truer and deeper explanation. Almost all these children we have seen are dull of hearing, and this is to great in most cases as to seriously interfere with the hearing of ordinary conversation. He does not hear what is said to him and therefore cannot understand. In that way his vocabulary is limited and his mental progress delayed.

At school through the impairment of hearing he miss half what is taught and appears stupid and backward: he is easily surpassed by his more fortunate companions and, by his constant liability to 'catch cold' his attendance at school is erratic. In reading he makes little progress and cannot be taught to speak properly a long as the adenoids are left unattended to. Add to this the vacant adenoid physiognomy with open mouth and expressionless face and it will appear how easily these children may be considered or looked upon as mentally weak or idiotic.

That the impairment of hearing is the chief cause of the delayed mental development and not the obstruction to the cerebral lymphatics has been manifested by the fact

that I have found that the mental stupor varies more, according to the impairment of hearing than to the amount of nasal obstruction. In a number of cases where there was little or no obstruction but marked dulness of hearing, I found the mental development greatly retarded, and on the other hand in some cases where the obstruction was almost complete but the hearing well retained the 'aprosexia' was scarcely manifested.

Should the obstruction be the chief cause of the lethargy and diminished mental capacity the opposite result of which I have stated would have been observed.

Chiefly through the impairment of hearing then, children with adenoid vegetations in the naso-pharynx make slow mental progress. Being easily tired, listless and disinclined for exertion they show a want of sustained attention & exhibit a mental torpor and lethargy. Taking little interest in games their imagination is not fostered and, by the bodily inactivity, a healthy mental capacity and development is delayed.

Effect on Digestion

The presence of adenoid vegel-

ations in the nasopharynx has a more or less direct effect on digestion & assimilation.

In the infant alimentation is affected through the difficulty in nursing caused by nasal obstruction: if the child is nursed at the breast the lips have to be removed from the nipple in order that it may breathe & similarly in 'bottle babies' the bottle is constantly expelled and may lead to the erroneous idea that the child is satisfied.

In older children it is noticed that they are longer at their meals: they make a noise, splutter and become breathless when eating, and the food is often gulped down in order that breathing may be established. Owing to the congested state of the faucs and the constant irritation of these vegetations, nausea & sickness is easily induced

The constant swallowing of puriform thick mucus tainted with septic germs is liable to and often appears to set up a gastritis or enteritis. The unhealthy condition of the mouth & teeth also favours such occurrences by the swallowing of decomposing particles. Mastication is interfered with by the carious condition of the teeth and by their malposition formerly described on page 51: this also delays digestion which is

still rendered more difficult by the diminished respiratory capacity and the general bodily inactivity.

In these various ways the presence of adenoids interfere with and deranges digestion but, alas, too often in these cases is the existence of adenoid vegetations seldom suspected, and the child's suffering progresses under the administration of grey powder or bismuth.

Effect on the Circulation

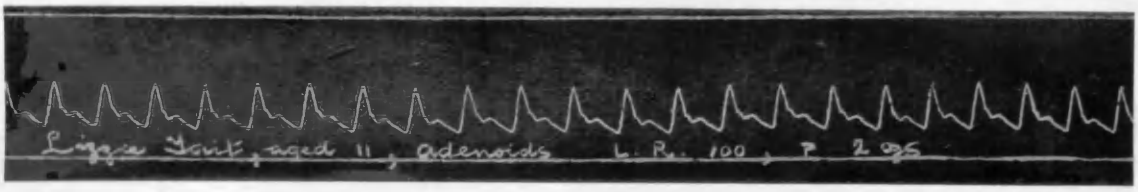
I have shown how the important functions of respiration and digestion are interfered with in children presenting adenoid hypertrophy, how lethargic and disinclined for play they are, & also, how anaemic and ill nourished.

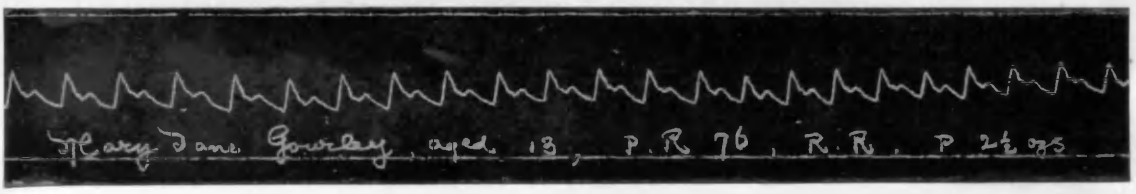
We would naturally expect to find in these patients a weak vacillating circulation and such is the case. There is no organic disease of the heart but the sounds are usually weak and there is occasionally a basal systolic murmur soft & blowing, in the pulmonary area. The pulse is usually soft & compressible low tensioned to some degree (See Sphygmographic tracings page 61) and is easily accelerated by the slightest exertion or physical disturbance. From the weak

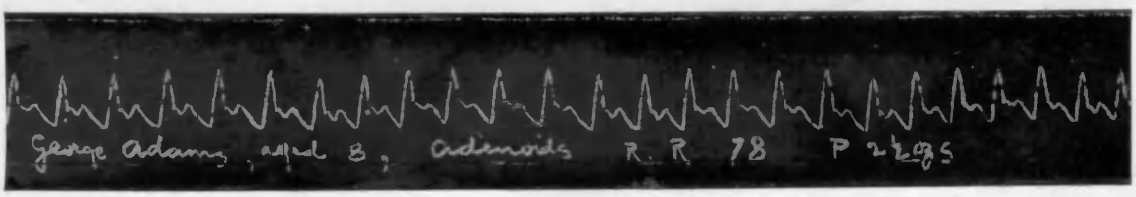
Sphygmographic tracings

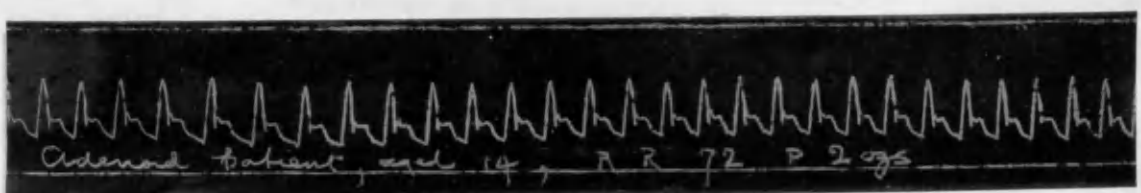
in cases of Adenoid Vegetations

showing degrees of low tension









condition of the heart and anaemia these children have cold extremities and are very liable to chilblains.

For the same reason a feeling of faintness is common especially on getting up in the morning.

Effect on the general health.

The deteriorating effects of adenoids on the respiration and digestion have already received some attention on the above pages. By the imperfect development of the lungs and constant gastric derangements, assimilation is interfered with & signs of defective nutrition established. These children become [illegible] and anaemic from defective haematosis. They are ill-nourished, thin & flabby; have languid circulations & are disinclined for exertion or play. They complain of being tired and exhausted on the slightest provocation and take little interest in games, books or toys. The constitution becomes weakened and undermined & they are predisposed to various diseases.

To estimate to what extent exactly healthy development is hindered, I had the weight and height of 100 children taken before operation with the following result shown in the

tables

The first column gives the age of the child, the 2nd his height in inches, the 3rd is an abstract from Roberts' Anthropometry giving the normal height in the English artisan class, whilst the 4th column shows the average defect in height in children suffering from adenoid vegetations

Age last birthday	Height without shoes 'Adenoid' children	Normal	Defect in height
4 years	Inches 37	38·3	1·5
5 "	39·2	41	1·8
6 "	40	43	3
7 "	41·5	45	3·5
8 "	43·5	47	3·5
9 "	45·2	49	3·8
10 "	47·5	50·5	3
11 "	48·2	51·5	3·3
12 "	50·4	53·5	3·1
13 "	52·3	55·5	3·2
14 "	53·5	58	4·5

The above table illustrates forcibly the extensive defective development occurring in children the subjects of adenoid hypertrophy in the naso-pharynx.

The next table is a similar one showing the loss in weight or the defective weight of 'adenoid'

Children

Age last birthday	Weight without Clothes			Defect in weight
	Adenoid children		Normal	
4 years	lbs	34	37	3 lbs
5		36	43	7 "
6		39	47	8 "
7		44	49	5 "
8		47	51	4 "
9		49·5	54	4·5 "
10		53	58	5 "
11		58	62	4 "
12		60·5	66	5·5 "
13		63	70	7 "
14		71	76	5 "

The 2nd table is just as striking as the first in showing the deteriorating effects of adenoid growths in physical development.

Under this section I may mention the great liability to enlargement of the cervical glands that occurs in children affected with adenoid hypertrophy in the nasopharynx. It is the deep cervical chain that is affected from above downwards. At first the enlargement is apparently simple & inflammatory, caused by the absorption of pyogenic organisms which I shall afterwards show are almost constant on the surface,

in the follicles and extending into the mucous membrane and adenoid tissue. These micrococci or their ptomaines enter the lymphatics and are carried to the glands and there set up irritation according to the extent and quality of the infection. If the pyogenic infection overcomes the phagocytic action in the gland suppuration results, whereas, if the gland is able to overcome the infection, swelling and induration are alone observed.

In 85% of the cases there was distinct enlargement of the deep cervical glands and in 9% 'suppuration' resulted. An important question is the occurrence of tubercular disease in these glands. I shall show afterwards, in the section of pathological anatomy, that the tubercular bacillus growing in the adenoid growths themselves is rare, but, that the mucous membrane is so weakened and altered as to allow the tubercle bacillus an easy entrance into the lymphatics leading to the deep cervical glands and there setting up tubercular disease. The frequency with which tubercular disease occurs in the glands of the neck is recognised by all and in these cases where the deep cervical glands are

affected adenoid growths in the nasopharynx are rarely absent.

Diagnosis

There need be little doubt regarding the diagnosis of adenoid vegetations in the nasopharynx. The alteration of speech and expression, with the symptoms of nasal obstruction occurring in a child is sufficient to direct the attention of the surgeon to the nasopharynx; and when these symptoms are associated with deafness the existence of adenoids may be strongly suspected. This suspicion is strengthened if the faucial tonsils are enlarged & if there is some granular pharyngitis present. A positive diagnosis can only be arrived at by a direct examination of the nasopharynx itself, either by posterior rhinoscopy or by digital manipulation. The latter method is distinctly preferable, in children at least, and affords more accurate data regarding the precise condition and extent of these growths than can be obtained by illumination of the post nasal space. As Meyer[1] remarks "the practiced finger used gently will, without giving real pain, reveal with sufficient clearness the number, situation, size, shape, consistence & vascularity of the vegetations".

The only real difficulty is not referring the secondary catarrhal symptoms to their primary lesion, and the mistake usually made is not to look for the presence of adenoids in these cases. An examination of the nasopharynx should never be omitted in cases of cervical glandular enlargements, hypogenic ear affections, laryngismus stridulus, repeated bronchial catarrh, & errors in refraction, especially when these occur in young subjects.

With the arrested development, defective nutrition & restlessness at night, cases of adenoids are liable to be mistaken for rickets, especially where there is some chest deformity and enlargement of the abdomen due to digestive derangements. In adenoids, however, there is no alteration in the ends of the bones and the obstruction to respiration is generally definite enough, but, should there be any doubt an exploration of the post nasal space easily settles the matter. It is to be remembered that rickets may be present in any child as well as adenoids although there is apparently no association of the one with the other.

In infancy with the altered shape of the nose, and snuffling

in the nose due to the obstruction & excessive secretion, cases of adenoids simulate infantile Syphilis. The nares, however, are not cracked or coated with dry crusts. There are no linear ulcers and the obstruction is not so complete as in infantile Syphilis, so that the child can usually take the breast better.

From polypi they are distinguished by their position & lesser mobility. They are not as a rule pedunculated, and their red colour contrasts with the gray oyster-looking appearance of genuine polypi.

Fibroma in the nasopharynx is rare: it would be firmer & more defined than adenoid vegetations and would not exhibit the same tendency to bleed on digital examination.

Sarcoma in the nasopharynx is also comparatively rare. In Sarcoma there is a great feeling of intumescence and the swelling very likely would not be symmetrical.

'Idiopathic' hypertrophic rhinitis may present symptoms of adenoids. The absence of the latter growths in the nasopharynx and the appearances presented by anterior rhinoscopy render the diagnosis easy.

In a similar way tuberculous rhinitis may simulate adenoids and may be differentiated in the same manner.

Prognosis

The prognosis in case of adenoids is very good if prompt and proper treatment be adopted.

When left alone unoperated on, most authors are agreed, that after puberty, they tend to disappear spontaneously. If left unmodified by treatment however, they induce, as formerly shown, serious and important changes in the organs of special sense, in the respiratory and digestive systems, &, in mental & physical development. These changes may prove directly fatal particularly by lung complications and by pyogenic infection to the brain through otitis media purulenta. When operated on, & the obstruction removed, the symptoms more or less rapidly disappear according to their character, duration & intensity, and the general condition of the patient is as a rule highly satisfactory.

Recurrence, although it is said to occur, is exceptional after free removal. The operation is a minor one & is practically devoid of danger. The exact degree of improvement that occurs, in the various secondary changes induced by adenoid hypertrophy, will be considered later on page 116 et seq.

(1) Medical & Surgical Reporter
Philadelphia Aug. 11th 1894

Treatment.

It has been already stated that the hypertrophied adenoid tissue tends to disappear spontaneously, and the question naturally arises, Why not leave it alone? This is not advisable for many reasons. In the first place the time when it may subside is uncertain. At puberty the symptoms of nasal obstruction are generally supposed to begin to abate. This may be caused by advancing sclerosis in the adenoids themselves or by the more rapid growth of the nasopharynx at that period of life. The latter condition is often overlooked in considering this subject. The growth of the nasopharynx at puberty is so great as to allow of free nasal respiration instead of the former oral breathing, even should the adenoids remain stationary in development. This would afford a partial relief of symptoms without the adenoid growths themselves diminishing in size. It would appear then that atrophy of adenoid growths at puberty is only apparent in some cases, and Dr. Delevan(1) has pointed out that they may continue to exist in adults for many years. In the second place, their presence induces the deteriorating effects formerly mentioned, on the organs of special sense, the frequent and serious

nature of which has been already discussed. Then there is the liability to lung complications, the slow mental and physical development and the defective general nutrition, all of which are presented more or less as long as the adenoid growths are left unattended to.

In considering whether or not an operation is advisable in all cases, we have to remember that the amount of adenoid tissue in the nasopharynx varies widely in different individuals, and, what would be pathological in one, might quite well be physiological in another; and that a slight hypertrophy if situated in the region of the Eustachian tube might give rise to serious ear complications, whilst a much larger increase in other situations might manifest no symptoms. Cases of adenoids not presenting any symptom are very unlikely to consult a Surgeon or Physician regarding the presence of adenoids. Their presence then is not detected till some obvious sign or symptom occurs, and then the question of operation is discussed.

When Adenoids are distinctly present operation should be advocated, as I think we are seldom justified in adopting

an expectant course of treatment, knowing full well the grave possibilities attending procrastination in these cases, and the more satisfactory results following early operation. Again, the adenoid growths never disappear entirely at puberty, and in many cases remain as a permanent source of nasopharyngeal catarrh.

As definite indications for operation I might mention the following:-

1. When adenoid vegetations give rise to symptoms of nasal obstruction, i.e. when they interfere with respiration & promote oral breathing; when the voice is altered in quality: when there is impairment or loss of the sense of smell or where an adenoid physiognomy is developing.
2. Ear complications.

Where there are recurrent attacks of pain in the ear operation is urgent as these attacks may be the first indication of extension of inflammatory process.

Middle ear deafness is one of the most constant concomitants of adenoid vegetations, and operation should not be delayed in these cases as the longer and the longer it is deferred the more permanent is the damage to the hearing, and the less beneficial

will the operation in the end be.

Operation is advisable in all these cases of middle ear deafness, in children at least, but those of long standing, with sclerosis and signs of anchylosis of the head of the stapes, little effect in the hearing may be expected after the removal of adenoids, although judicious after treatment by inflation usually improves the hearing.

3 Otitis media purulenta, with or without further extension of the suppurative process to the mastoid cells or brain.

The mechanical obstruction to the orifice of the Eustachian tube, and the inflamed condition of the adenoid tissue around it tend to prolong the otorrhœa, whilst excision of the adenoid vegetations is usually followed by a rapid subsidence of the otorrhœa.

When the otorrhœa is very foetid it is advisable to treat it locally for a time before removing the adenoids, as the risk of infecting the wound & producing acute inflammation is less. In the acute suppurative inflammations of the middle ear or in cases of extension to the mastoid cells or brain, the secondary suppurative consequences should be treated at once and the adenoids removed as early as possible

So also in acute periostitis over the mastoid, or where there is caries or necrosis, the excision of the adenoids should be postponed till immediately after the urgent operation.

4 Glandular enlargements in the neck

Here again an early removal of adenoids is of the highest importance, as when adenoid growths are excised early the glandular swellings usually rapidly subside. Should however operation be deferred, septic or tubercular infection frequently follows

5 Deaf Mutism

When there is reason to suppose that the deaf mutism is acquired and adenoid vegetations present, they should be removed, as I have already shown, the adenoid vegetations appear to be the primary cause in some cases of this affection. To obtain a satisfactory result, however, the operation must be performed very early

Medical treatment

The simpler methods of general medication are often indicated on account of impairment of the general health. I have found equal parts of Cod liver oil and Syr. ferri Phos. Co. most serviceable

administered in dose of ℥ss thrice daily.

The question as to the value of local medical treatment is an important one. The secretion may be removed and the parts disinfected by various alkaline and antiseptic lotions. The hypersecretion may be lessened and the chronic hyperaemic swelling and thickening partially removed by astringents. All that can be accomplished by this is the limiting of the amount of the secretion, and the securing of a small absolute reduction of the size of the growths. A cure cannot be looked for except in the very early stages of the disease when the growths have attained but a limited size.

I have a distinct objection to the using of the nasal douche as I consider repeated ear trouble has frequently followed its use. A much safer method is the application of a medicated spray by means of an atomizer.

As above stated, all that I have noted and all I think that we can reasonably expect from local medication is a modification in the symptoms: a radical cure can only be accomplished by thorough extirpation of the offending vegetations, so that

Surgical measures ought to be resorted to in preference to all others. The application of Caustics as a distructive agent no doubt will remove the vegetations, and is said to be followed with successful results. It is a painful indefinite operation, devoid of precision, and involving a long course of treatment. For these reasons it is not to be recommended.

Surgical treatment.

The modes of operating are numerous; and various instruments are employed by different Surgeons. Each one advocates some special advantage in the instrument he is in the habit of using, and employs it in preference to any other. All the instruments are devised for the same object and a skilful manipulation of any one of them will remove the morbid growths. The precision and rapidity in using, and the total eradication in removing depend more on the surgeon than on the instrument

The methods of operating may be classed as follows, according to the manner in which the vegetations are removed, or according to the instrument employed.

1. Natural finger nail
2. Artificial finger nail

3. Jaeger's Ring Knife
4 Gottstein's Curette
5 Hartmann's Curette
6 Lowenberg's forceps
7 Snare
8 Electric Cautery
9 Sharp Spoons

(See page 78

Other instruments are used but they are only modifications of one or other of these.

I have already stated that I consider that the total extirpation of adenoid vegetations depend more on the dexterity of the manipulation of any single instrument rather than on the merits of the instrument itself, still I think there are serious objections to some of the instruments and methods employed.

The ~~artificial~~ natural finger nail is advocated by Guye, Pritchard, Richardson of New York & Lennox Brown When it is alone used it is very difficult, nay, even impossible in some cases, to remove the entire growth.

The furrows as we have seen are chiefly longitudinal and the narrow nail too often slides along the furrows, leaving active growing ridges; and again, the growths may be so filmy as to resist a most powerful finger nail. The only case in which the natural finger nail operation is at all satisfactory is where the vegetations are soft.

(1) Ibid, 1895.

Fig. 1

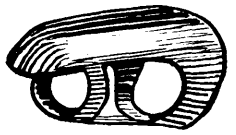


Fig. 2

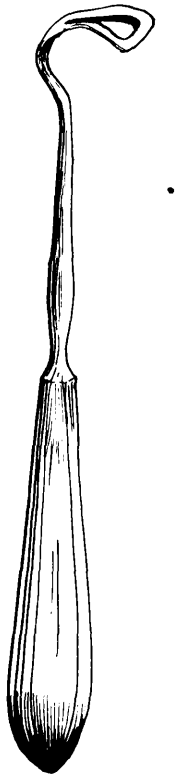


Fig 3

Fig 4

Fig. 5

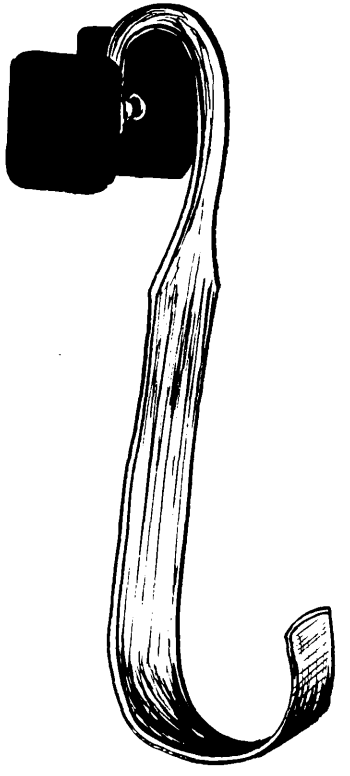


Fig 6

Fig. 1. 'Dalby's Nail'

Fig. 2. Gottstein's Curette

Fig. 3. Hartmann's Curette

Fig. 4 Löwenberg's Forceps

Fig 5 Ridley's Gag

Fig. 6 Meyer's Ring Knife

Semon[1] states that in his experience in no class of cases is a repetition of the operation more frequently required than in those in which the finger nail has been used as the instrument for removal.

These objections cannot be urged against the artificial nail of Dalby. This instrument I have not used.

Löwenberg's forceps I consider dangerous and unsatisfactory instruments: dangerous in so far as that the cartilaginous portion of the Eustachian tube may be readily enclosed and drawn away with the forceps and, unsatisfactory because they prolong the operation & the extirpation of the adenoid growths in the end is not so complete. In the same way the Snare operation, advocated by Chiari[2] of Vienna who introduces the snare through the nose and guids it by posterior rhinoscopy, is unsatisfactory. It is tedious and incomplete in the end & offers no advantage over the Curette operation.

The application of the electric cautery, as first recommended by Voltolini, makes a very prolonged & different operation requiring numerous sittings and in the end not at all radical.

The instrument I am in the habit of using is Gottstein's Curette

(1) Lancet Sept 14th 1895

This instrument is used by Barr (1) with a modification to retain the adenoid growths, by Mc Bride Politzer, Rotenberg & Semon. The whole vault of the nasopharynx and the posterior wall can be cleared of the entire adenoid vegetations in a few sweeps with this curette and by turning it laterally the sides of the cavity is denuded of any hypertrophied adenoid tissue. In this way a complete operation is accomplished in a short time, and any outstanding vegetations around the orifices of the Eustachian tubes are cleared, whilst the Eustachian tube itself cannot be torn away or damaged by the gliding cutting movement. To complete the operation I introduce the forefinger of the right hand and explore the cavity to ascertain whether any vegetations still remain and if these cannot be completely removed with the finger nail the curette is reintroduced to divide the remaining vegetations.

The finger nail is of use especially in clearing out the adenoid tissue from the fossa of Rosenmüller & from the recess immediately below and descending from the orifice of the Eustachian tube. Into these recesses the curette does not penetrate and I consider this clearing out of the fossa

of Rosenmüller and the descending folds from the Eustachian tube orifice very important, both as regards the prevention of a recurrence and the immediate success of the treatment.

Should these recesses not be thoroughly evacuated of the adnoid tissue recurrence to a degree is almost sure to follow, and I have no hesitation in saying that if this point is not attended to the success of the operation regarding hearing will be unsatisfactory.

Other forms of curette are used. Mayer employs a ring knife which he introduces through the nose. This method I have no experience of, but I would imagine that the operation would take longer than that with the curette & in the end not be so complete.

Hartmann's curette is practically the same as Gottstein's but works laterally instead of vertically.

The sharp spoon of Juraschek & Brautmann is similar in action to the curette.

Choice of an Anaesthetic

Much discussion has occurred on this subject. Many aural surgeons operate without any anaesthetic, regarding the anaesthetic as unnecessary or, as adding so materially to the danger of the operation as to

(1) Ibid Aug. 1894

be prohibitory.

Others employ a local anaesthetic, the one usually employed being cocaine.

Hartz (1) states that he has used cocaine & antipyrin locally as anaesthetic and the adnoids were removed without pain and the patients did not complain of pain afterwards.

I have found in my experience that operation in children without a general anaesthetic is unsatisfactory and incomplete. Most children even object strongly to have a Eustachian catheter passed and a prolonged painful operation is only accomplished after some brutality, whilst the straining & movement of the child interfere with the precision of the operation. The fright the child receives with such manipulation I would expect to give rise occasionally to some serious after effects.

Mayer operated without an anaesthetic on his 102 cases with the ring knife introduced through the nose & guided posteriorly with the left index finger.

He justly states, under these conditions, that one operation rarely succeeds in removing all the vegetations so that it often requires to be repeated and that in some cases neither the operation nor the after treatment

succeed in displacing all trace of the vegetations. He adds that it causes a headache and produces a slight stupor for a few hours.

For these reasons a general anaesthetic in children at least should always be employed as affording the only satisfactory result and allowing of a complete operation at one time.

In administering an anaesthetic the first consideration is to place the patient in the best position for operating and also in a position that prevents the blood from being drawn into the larynx during operation. Some operators prefer the head hanging over the end of the table; the only drawback to this is that the bleeding is more profuse. Mr. Rovell & others operate with the head on the same level as the body and trust to sponging the back of the throat to prevent blood running down into the pharynx.

Dalby sits his patient on the end of a couch and bends the patient forwards to an angle of 40°.

Butlin & Hewetson[1] operate with the patient on his side, the thighs flexed and the head bent a little forwards on a low pillow.

Mr. Brain, giving nitrous oxide and ether, first places the patient in a semirecumbent position and when

narcosis is complete flexes the trunk forcibly on the thighs.
In this position the blood escapes from the mouth and nose into a basin between the knees.
Most operators consider anaesthesia by nitrous oxide gas alone or with ether, too short for a complete operation. This depends somewhat on what instrument is used.
Three fourths of the cases I have operated on have had nitrous oxide alone and I have always had sufficient time to do a complete operation. When adenoid vegetations alone require removing I prefer nitrous oxide gas, but when the faucial tonsils are also enlarged I generally give chloroform and excise the tonsils and adenoids at the same time.
The patient is placed in the dorsal position with the head slightly depressed resting on the top left side of the table. Ridley's gag is introduced before the anaesthetic is administered. Standing at the top of the table, with the patient in the above position, as soon as anaesthesia is induced Gottstein's Curette is introduced behind the soft palate and carried to the extreme vault of the nasopharynx. With a number of firm backward and downward sweeps the entire

vault & posterior wall is thoroughly denuded of its hypertrophied adenoid tissue. The curette is then turned to each lateral wall which is cleared of its growths by a number of downward & outward cutting movements of the curette. The patient is then turned over completely on the left side and the right forefinger introduced to complete the operation, special care being taken to thoroughly clear out the fossa of Rosenmüller and the recess descending on each side immediately below the Eustachian tube. The dorsal position is more favourable for obtaining precision in removing the vegetation with the curette, whilst the slight droop of the head retains the adenoid growths and blood in the nasopharynx until the patient is turned over on his side. The side position is also favourable for the introduction of the finger. With this method of operating there is little haemorrhage and no chance of asphyxia. I have never had to use any haemostatic.

Should any blood be blocking the larynx or bronchia the best method of expelling it is to have the patient well round on the left side with the head somewhat depressed, then taking hold of his

(1) British Med. Journal, Jan 13th 1894
(2) Medical Press, Aug. 1894

right arm raised from the side and administer a sharp knock with the flat of the hand on the interscapular area. This is sufficient to eject any clot into the throat or mouth where it can easily be removed.

Should the haemorrhage give rise to any anxiety it can always be easily controlled by a small sponge introduced into the naso-pharynx by a pair of curved forceps or kept in position as a pad with a ligature brought through the anterior nares.

Holloway[1] & Wood[2] make an earnest plea for nitrous oxide gas to remove adenoids. Lennox Brown & Dundas Grant employ nitrous oxide as its being perfectly safe. Other operators as W. Brain prefer gas and ether as the stage of anaesthesia by the combination lasts longer.

Many Surgeons prefer ether to chloroform on the grounds that with ether, they say, the power of coughing or swallowing is generally retained, that the heart's action is not so liable to be depressed. With ether however, there is usually great venous turgidity and copious bleeding; and the noisy

(1) Brit. Med. Journal, Apr. 21st, 1894

(2) Lancet

(3) Brit. Med. Journal, Jan 27th, 1894.

(4) Brit. Med. Journal, May 5th 1894

breathing instead of being a safe-guard is more liable to draw blood into the lungs. The profuse mucous secretion that collects at the back of the throat is also an additional drawback to the use of ether. For these reasons I prefer chloroform instead of ether. I have administered it in about 100 cases of adenoids and in none of these has there been the least trouble or anxiety regarding the pulse or respiration.

Semon[1] strongly objects to the induction of complete anaesthesia. Barr[2] I consider puts it correctly when he says that whilst the corneal reflex is abolished the cough & swallowing reflex should not be entirely in abeyance, and yet whilst avoiding deep narcosis it is important that it should relax the muscles of the pharynx and prevent retching during operation.

Grant Morris[3] & Dyke Dowse[4] recommend chloroform in preference to nitrous oxide as it gives more time for a complete operation.

After treatment

The bleeding following operation is usually slight, and practically ends before they leave

the Theatre. Secondary Haemorrhage is not liable to occur and the present writer has not met with a case. Should it happen it would be best treated by the means adopted to stop bleeding immediately after operation.

Sucking ice stops oozing, prevents swelling and tends to lessen the tendency to sickness if chloroform has been administered.

Very often a quantity of blood is vomited after the operation: this blood is usually dark coloured and is what has been swallowed during or immediately after operation. If bright red blood has been vomited, the nasopharynx should be inspected to see that bleeding has really ceased.

The patient should be confined a few days in bed. Most of these cases have been kept for a week in the Hospital, and during that time the diet is liquid & cool. At the end of a week they are discharged: at that time the wound is covered with granulation tissue and there is little chance of infection. During the second week they are confined to the house and at the end of that period return to the Hospital for inspection. With these precautions

the present writer has found inflammatory trouble following operation rare.

Formerly it was the usual custom at the Hospital to operate on adenoids as Out-door patients, the children returning to their unsanitary homes a few hours after operation. At that time I understand every third or fourth child returned with an otorrhoea, whereas in the cases now brought forward only 4 of them developed an otorrhoea after operation and in 3 of these it was questionable whether or not there was a slight discharge prior to operation, the reaction in the nasopharynx only aggravating the condition for the time.

Retaining patients in the house in a well ventilated room with an equable temperature for a few days is important in preventing complications following operation.

The nasal douche I discountenance, considering it a source of danger.

The atomizer with a medicated spray is distinctly preferable and reduces the chance of septic extension to the ear

Attending to the mouth with antiseptic lotions both before & after operation has a beneficial effect in lessening septic infection.

It is advisable in many cases to continue the medicated spray for a considerable time after operation and astringent preparations may be judiciously administered. By this means any tendency to recurrence is checked.

Inflation of the middle ear by Politzer's method is advisable in most cases after operation and generally improves the hearing. This, however, should not be adopted till the healing process in the nasopharynx is completed or almost entirely so, as earlier inflation may cause purulent extension to the ear. The number of times Politzer's method has to be performed is guided by its effect on hearing.

Oral breathing often has to be stopped by forced education in nasal respiration

Phonation frequently requires a prolonged course of careful training before speech loses its 'nasal' quality.

As complications of the operation I may mention

1. Septic infection extending to the ear. As preventative measures the entire preparations in everything should be made as for an ordinary aseptic operation. The mouth both before & after operation should

should be attended to by antiseptic lotions. The nasal douche should be avoided, so also early inflation.

2 Acute pharyngitis.

In one case an acute pharyngitis followed the removal of adenoids: in this case operation was performed during an acute adenoiditis. In the evening of operation the temperature recorded 104°, the pulse was highly contracted & the pulse rapid. He complained of sore throat, 'muscular' pains and painful deglutition, &, indeed all the local & general symptoms of acute follicular pharyngitis were observed. With the administration of calomel & salicylate the symptoms rapidly subsided. A double otorrhoea was present in this case and appeared uninfluenced by the acute throat inflammation.

To prevent this complication operation should be avoided during an acute attack of inflammation in the adenoid tissue and by taking the ordinary precautions as above.

3 Contracting some Contagious disease.

Scarlet fever, measles, erysipelas have been said to follow, occasionally, soon after operation. In only one case did a contagious disease occur after operation in the 214 cases brought forward. This patient was

dismissed a week after operation 'well'.
He went home and contracted diphtheria and died in 4 days.
On inquiry it was ascertained that the drains in connection with the house were in process of repair & that the main drain passed below the floor of the room in which the child was placed. This drain had been opened into through the floor.

4 Legs of tissue loosened but not detached. This only occurs in an incomplete operation where the finger nail alone has been used or any of the instruments inefficiently.

5 Stiff neck or any neck.
The author has not met with these complications and expects they more frequently follow cases where no anaesthetic has been administered.

6 Hypertrophic rhinitis.
This is a condition which often exists prior to the removal of adenoid vegetations and is usually greatly improved after ablation of the adenoids. The septic centre in the nasopharynx tends to develop this condition particularly in the posterior portion of the inferior turbinated bone which lies adjacent to the adenoid vegetations, and the removal of these adenoids

should be the first treatment for the hypertrophic rhinitis.

Some cases of chronic hypertrophic rhinitis appear to take on an acute action after excision of the vegetations. In 5 cases this occurred: 4 of them yielded readily to treatment, whilst in the 5th one the former hypertrophy has still increased.

In 2 cases the operation seemed to induce a hypertrophic rhinitis, but in these cases it is possible that some hypertrophy of the posterior ends of the inferior turbinated bones existed previously.

As an explanation of the causation of these changes it might be suggested that after operation the current of air passing through the nose is much stronger and more constant than before operation. A greater physiological activity in moistening & warming the air is therefore demanded necessitating increased blood supply. Should care then not be exercised after the removal of the vegetations and during the period of reaction, inflammatory changes in the turbinated bones will probably ensue.

Pathological Anatomy and Bacteriology.

Adenoid vegetations usually spring from the roof and posterior wall of the nasopharynx, where normally a collection of adenoid tissue exists called the pharyngeal tonsil. They are also generally present on the lateral walls of the nasopharynx, in the fossa of Rosenmüller and about the orifices of the Eustachian tubes or in the recesses below the opening of the tube. In the latter situations the vegetations are as a rule smaller and never reach the size they attain on the roof & posterior walls

Meyer mentioned a few cases where growths were observed on the upper surface of the soft palate. The present writer has not detected them in that situation although the soft palate has been frequently observed to be thickened and more firm than in the natural state. It has been said that the largest vegetations spring from the roof & posterior wall and there the hypertrophy usually assumes the folds and projections of the normal pharyngeal tonsil. With the enlargement these folds become greatly increased in depth, & when pressed against each other they give the situation to the

fingers as of one soft fleshy mass.
More frequently, however, the vegetations hang loose & free, so that whilst completely closing the nasopharynx they give way to the fingers and feel very much like a bunch of earthworms. When they are removed in toto the folds are more definitely apparent.
A median recess divides the tonsil into 2 lateral halves and these halves are further divided by transverse clefts in their lower portions and vertical or antero-posterior divisions in the upper segments that cover the roof of the nasopharynx.
This arrangement is well observed on the drawing (page 11)
The number of divisions or clefts varies from 4 to 6 on each side above and below. The colour of the vegetations does not differ materially from that of the surrounding mucous membrane which is invariably congested & swollen. The mucous membrane lining the posterior wall of the oral part of the pharynx is usually dotted with enlarged follicles presenting the granular condition known as pharyngitis granulosa.
In 68% of the cases the faucial tonsils were in a state of chronic enlargement. The pillars of the fauces besides being in a state of

chronic congestion are thickened & deficient in mobility and approximate the posterior wall of the pharynx.

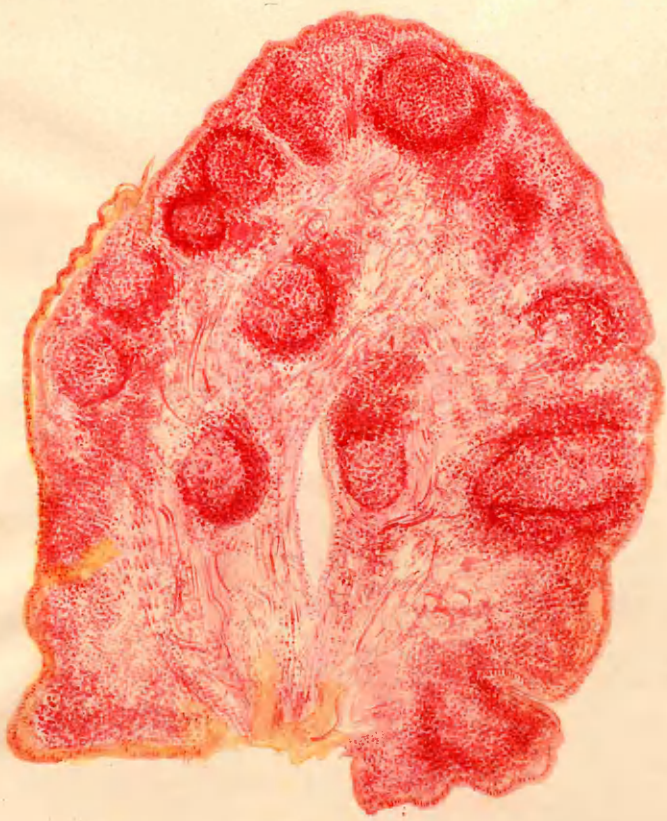
The soft palate is similarly affected.

The mucous membrane lining the nasal cavities generally participates in these inflammatory changes and is frequently observed to be swollen and altered.

When an adenoid vegetation is examined by the naked eye it is seen to be of a reddish colour presenting a number of very small granules on a shining or velvety surface. The consistence of the growth varies considerably some being soft like jelly others being firm and almost fibrous.

When a section is examined under the low power of the microscope (see drawings page 97 & 98 or sections) the surface is seen to be covered by an epithelial lining on which open penetrating glands, whilst the main mass of the tumour is composed of lymph tissue collected into masses - lymph follicles - with a fibrous tissue stroma of varying density supporting the lymph corpuscles, glands & vessels.

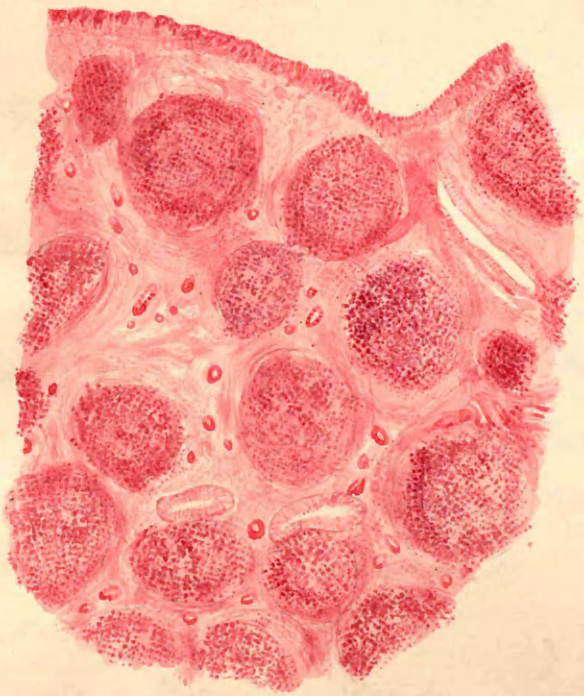
Under a higher power - $\frac{1}{6}$ objective or $\frac{1}{12}$ oil immersion - these various structures can be more definitely observed and examined. First with regard to the epithelium, the normal ciliated



Adenoid Vegetation, low power

stained with picrocarmine

– Slide No 3 –



-Adenoid Vegetation, low power

stained with logwood + eosin

- Slide No. 10 -

columnar variety is present in some of the sections at some points on the surface in a natural healthy physiological state (Fig A, page 100, r sections) Long regular columnar cells with delicate cilia on the surface, containing a deeply placed oval nucleus & branching below, whilst within the branches are irregularly cubical & spindle cells supported on a fairly definite basement membrane. The distinction or division between the mucous membrane on the surface and the lymphoid tissue below is definite.

On the surface of the mucous membrane are occasional 'leucocytes' whilst escaping between the columnar cells others are observed reaching the surface.

In sections cut from a healthy normal nasopharynx the entire surface is covered by that columnar ciliated epithelium but in adenoid hypertrophy this delicate epithelium is observed to be changed in character and converted to quite a different type. This change is generally only over the surface but occasionally the ciliated columnar epithelium lining the glands is similarly changed. The variety to which the epithelium is generally converted is the stratified squamous, which may vary in thickness to from 3 - 12 rows or more of cells

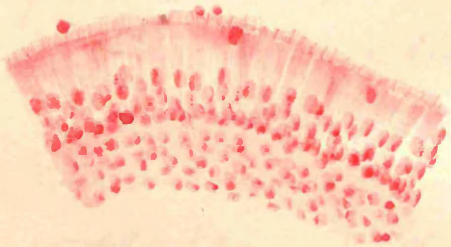


Fig. A.

Fig. A. Normal Ciliated Columnar Epithelium
-alum carmine

Slide N° 4.

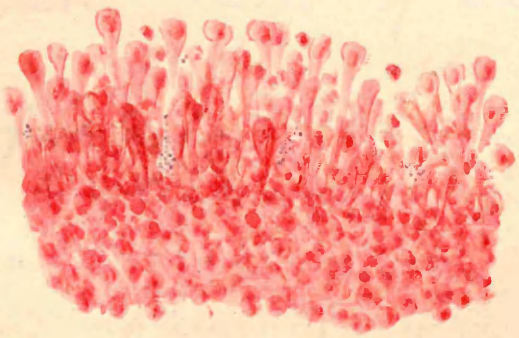


Fig. B. Slide N° 7.

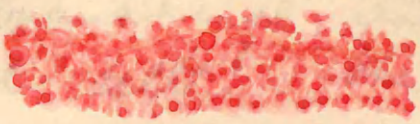


Fig. C. Slide N° 24

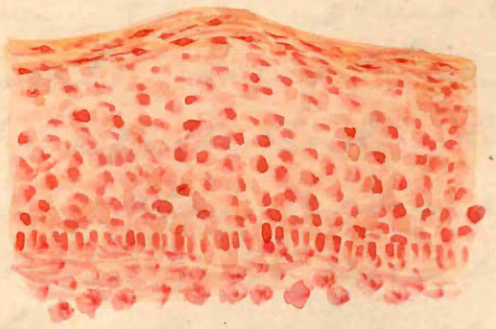


Fig. D. Slide N° 8

Figs B, C & D -Altered epithelial surfaces

C alum carmine
B Gram's method
D picrocarmine

The ciliated lining is now entirely absent (Figs B. C. D. page 100), and the distinction between epithelium and lymphoid tissue is not so definite, the one almost gradually merging into the other. In some sections the epithelium is represented by an irregular row of cells (Fig C) composed only of one layer, so that the lymphoid tissue of the follicles come closely up to the surface, separated from it by a single indefinite layer of cells. At other places and in other sections (Fig B) the epithelial lining is thickened and proliferating, presenting large cubical cells with clear nuclei in their upper part, whilst below a thick process descends and branches. Around and between these 'branchings' are oval or irregularly cubical cells. These cells almost appear continuous with the adenoid tissue, and the adenoid tissue itself seems more vascular than in the normal condition the vessels being traced readily up to the epithelial lining.

In other sections (Fig D) the epithelium is greatly thickened and presents rows of squamous cells flattened out, and, taking a deep yellow colour with the picrocarmine stain approaches the appearance of the stratum corneum of true skin

The alteration in the variety of the epithelium may be explained by the functional inactivity of the hypertrophied adenoid tissue & the result of pressure & chronic irritation.

If the pressure be continuous the mucous membrane will be thinned, if it is intermittent a thick stratified squamous variety will be developed.

These changes in the epithelium have no relation to the age of the child from which the sections were cut. The function of lymphoid tissue in the pharyngeal region is generally accepted to be the elaboration of secretions used in swallowing and the production of leucocytes, which either enter the circulation or escape on the surface to destroy micro-organisms; and also some connection with absorption.

When the natural ciliated lining of the mucous membrane is destroyed discharges collect on the surface, and the hypertrophied condition with deep recesses tend to favour the stagnation of secretion & suppuration.

In a number of sections stained by Gram's method these pyogenic organisms are observed dotted over the surface and extending deep down into the adenoid tissue. This is well shown in a number of the sections and (Fig 8 page 100) exhibits the entrance of these organisms.

With these pyogenic organisms in

the surface and the mucous membrane thinned, weakened, destroyed or altered septic absorption readily take place, and it is easy to imagine how the tubercle bacillus could gain an entrance into the lymphatics in this region where the natural barrier is so changed & weakened.

In this manner septic or tubercular infection is carried to the deep cervical glands and readily accounts for the frequency with which these glands are affected.

From the mucous membrane fibrous strands of varying density descend, dividing up the vegetations into compartments in which are situated the lymph follicles.

The follicles consist of clumps of dense retiform tissue, the meshes of which are closely packed with lymph corpuscles and pervaded by fine capillaries. These follicles vary in size, some of them come close up to the epithelium as to almost touch it, whilst others are deeply placed. Between the follicles the fibrous tissue stroma is denser and stronger and contains larger vessels, and in a number of the sections numerous mucous glands. The proportion of the lymphoid to the fibrous tissue varies in different cases. In soft vegetations the

lymphoid tissue preponderates and in fewer ones the fibrous tissue is more attendant and denser. The age of the patient has nothing to do with the relative proportion of fibrous to lymphoid tissue as in some of the youngest subjects the fibrous tissue has been observed to be most dense. In one way there would appear to be some connection between the development of the fibrous tissue and the size of the growths. When the tissue becomes fibrous the lymph follicles are compressed and so lessened in size, whilst the contraction of the fibrous tissue is associated with diminution in the lumen of the vessels.

These sclerotic changes are often noticed first around the lumen of the vessels and the walls of the vessels are observed to be surrounded by concentric rings that appear to be exercising a certain amount of pressure on the vessel so contracting it's lumen. It is possible that by this process of perivascular sclerosis the blood supply of the tissue is lessened and 'spontaneous' disappearance in time of these vegetations may be explained.

(1) Ann. d. Mal. de l'oreille, du Larynx etc.,
Paris 1894 p 979

(2) Bull. acad. de Méd.
Paris, April 30 1895

(3) Op. cit

(4) Berl. Kin. Wchschr., Hefte 31 & 32 Aug. 1896

The deep cervical glands are a favourite seat for tubercle and the lymphatics entering into this chain are chiefly derived from the tonsils and nasopharynx. In most of these cases the tonsils were healthy or only moderately hypertrophied and the question naturally arises, are adenoid vegetations tubercular manifestations involving the glands secondarily? This has been discussed by a number of Continental writers.

Lermoyez[1] in 1894, reported a case of adenoids that required a second operation and histological examination of the tissue removed showed it consisted almost entirely of tubercular tissue with tubercle bacilli. A second recurrence followed operation. A histological examination of the primary vegetations was not made.

Dieulafoy[2], in 1895, examined microscopically the growths removed from 75 cases with the result that 4 he said presented evidences of tubercle.

Brindel[3] in 1896 made sections of 64 cases of adenoid growths and found evidence of tubercle in 8 of them and in one section said he was able to stain 4 tubercle bacilli near a nodule.

Gottstein[4], in 1896, in 33 pharyngeal tonsils found microscopical evidence

of tubercle in 4 of them. Giant cell nodules were present in these cases but no tubercle bacilli

Bleeder & Fischer[1] in the same year found tubercle bacilli in 5 cases out of 12 cases examined

McBride & Turner[2] in 1897 made a histological examination of 100 cases of adenoid vegetations and discovered evidence of tubercle in 3 of them. Giant cells were detected but no caseation & the tubercle bacillus was not able to be stained in any of the three.

The present writer has examined histologically adenoid vegetations in 40 cases and has failed to detect evidence of tubercle in any one of them. In some of the sections there are giant cells - polynuclear cells - but not characteristic enough to afford definite evidence as to the positive nature of these vegetations. In the cases above stated where evidence of tubercle have said to have been found, it will be seen that the constant factor leading to the conclusion of the tubercular nature of these growths has been the presence of giant cells: Now giant cells may be present in all cases where there is inflammation and new formation of tissue, and the presence of giant cells per se does

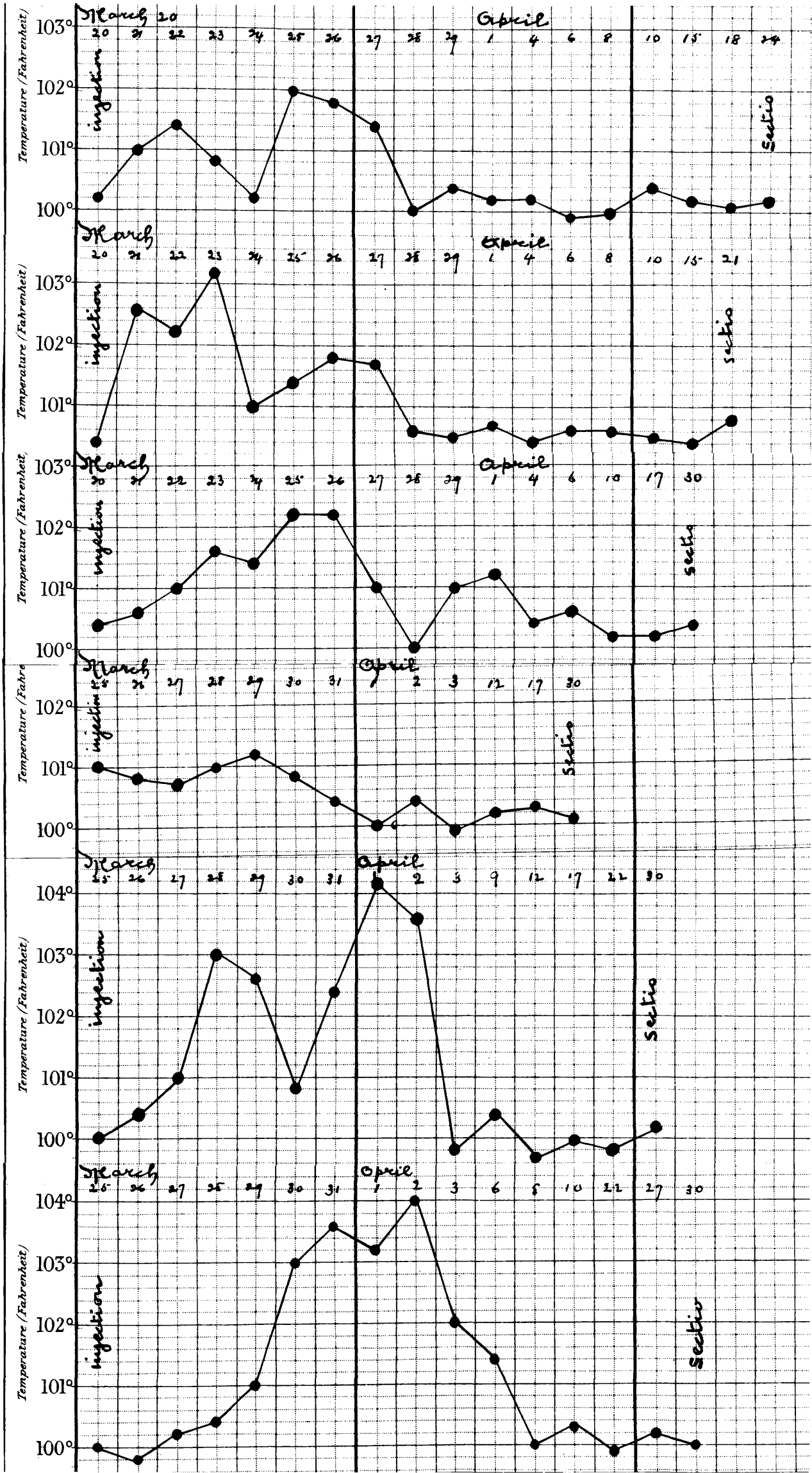
not afford sufficient proof of the tuberculous nature of the lesion.

Before it can be said to be distinctly tuberculous caseation & Koch's bacilli must be present and with the exception of the one case reported by Lermoyez and the cases of Glender and Fisher, definite tuberculous foci in adenoid vegetations cannot be said to have been thoroughly demonstrated.

To ascertain further if adenoid vegetations were tuberculous the author at his request had 6 guinea pigs injected by Dr. George Murray of Newcastle, with adenoid vegetations from 6 cases. The adenoid growths were removed aseptically and washed thoroughly in sterilized water. They were afterwards made into an 'emulsion' with sterile meat broth and injected into the groins of the guinea pigs.

The temperature was taken daily for a time and the records are shown on the accompanying charts, page 108. All the guinea pigs continued to live and did not lose weight. Necropsies was performed on each of them 5 to 6 weeks afterwards, with negative results as regards tubercle. None of the guinea pigs developed tuberculosis. The abdominal glands

Charts of Guinea Pigs
from injection to sectio



were small & apparently healthy.
Microscopical sections of them failed to detect any abnormality or evidence of tubercle. These experiments afford much more reliable data, regarding the consideration whether or not adenoid growths are tubercular, than merely the histological examination of sections of the vegetations. The tubercle bacillus is not always easy to find in even definite tubercular glands, but the injection of tubercular tissue into the susceptible guinea pig reveals readily the true nature of the tissue. The glands in the neck were removed in two of the cases from which the adenoids were excised for injection and were found to be tubercular. If these tubercular glands were secondary to 'tubercular' adenoids it would have been expected that the primary centre by injection would have produced tuberculosis in the guinea pigs but such was not the case. In none of the 6 cases was tubercle produced in the guinea pigs, and, as formerly stated in none of the 40 cases in which a histological examination was made were evidences of the tubercular bacillus detected, so that the present writer feels justified in concluding that adenoid vegetations

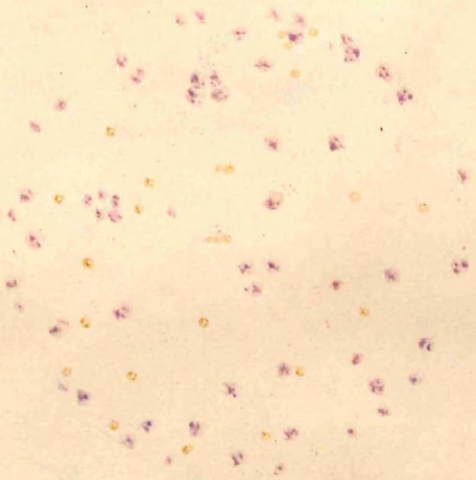
are not themselves tubercular. That they may be the seat of tubercle or have tubercle implanted in them occasionally is not doubted, but as one has endeavoured to show the hypertrophy is not the result of tubercular infection and cannot be regarded as a tubercular manifestation. The mode in which the tubercle bacillus infects the glands is by gaining entrance through the altered weakened mucous membrane on the adenoid growths. It can be carried directly to the glands without necessarily forming tubercular nodules in the adenoid tissue, just as tabes mesenterica may be caused by the tubercular bacillus entering the 'lymphatics' of the bowel, through some weakened condition in the epithelial lining without necessarily any tubercular ulceration of the bowel and without any tubercular foci in the closed follicles.

Although no tubercular disease was caused by the injecting of the adenoid 'emulsion' localized abscesses followed in 4 of the 6 cases. The adenoid growths it was already mentioned were removed by an aseptic Gottstein's curette. They were washed in sterile water and made into an 'emulsion' with the sterilized meat broth. The syringe and needle for injection were boiled and the groins of the guinea pigs were thoroughly prepared before injection. The puncture

was covered with a collodeon dressing.
In the 2nd, 3rd, 5th & 6th guinea pigs an abscess formed at the seat of puncture within a week and was incised. The pus was stained in each case for tubercle bacilli with negative results. Pyogenic organisms, however were found in all four. The drawings, on pages 112 & 113, show the pus corpuscles and the pyogenic organisms. Cultures of the pus was grown in nutrient gelatine & agar agar from each of the cases and the organisms isolated were as follows:-

No 2	Guinea pig	- Staphylococcus pyogenes albus
No 3	"	" - aureus
No 5	"	" " "
		& Streptococcus
No 6	"	Streptococcus
		& Staphylococcus pyogenes aureus

That pyogenic organisms are usual not only on the surface of adenoid vegetations but also extending into the crypts and the tissue itself the present writer has further proved by the following experiments.
From 25 cases of adenoids gelatine cultures were made: 15 of these were innoculated from the surface of the vegetation after it was removed from the nose.

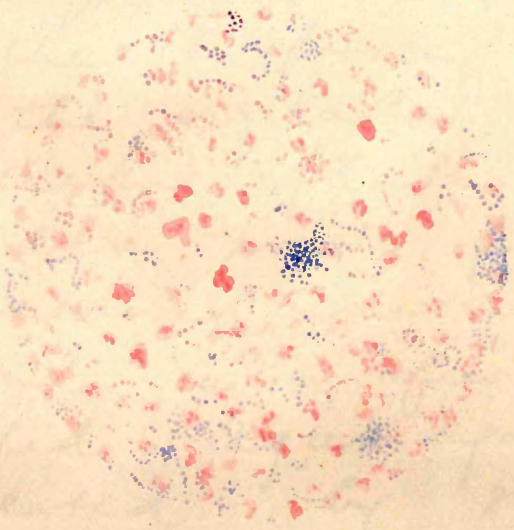


Pus from abscess

No 5 Guinea Pig.

Ziehl-Neelsen stain

- Slide No 30 -



Pus from abscess

No 6 Guinea pig

Stain - Grams Method

- Slide No. 32 -

pharynx, whilst 10 cultures were taken from the inside of the growth by the following method.

The surface of the vegetation was first cauterized and an incision made through the cauterized portion with a sterilized knife. The sterile platinum needle took a scraping from the side of the incised margin and the gelatine media inoculated by a stab culture.

A growth occurred in 13 of the 15 former tubes and in 5 of the 10 latter.

Gelatine plate cultures were then made from the growths that occurred on the gelatine tubes, and in a few days colonies developed. Agar agar tubes were inoculated by streak cultures and microscopic specimens prepared from the growth that occurred on the agar agar tubes.

With this method the following is a tabulation of the pyogenic organisms found.

1. Staphylococcus pyogenes aureus
2. " " "
3. " " " + Streptococcus
4. No growth
5. Staphylococcus pyogenes albus + aureus
6. No growth
7. Streptococcus

8 Staphylococcus pyogenes albus
9 " " aureus + Streptococcus
10 " " " " "
11 " " "
12 " " "
13 " " aureus
14 " " albus
15 " " " + Streptococcus

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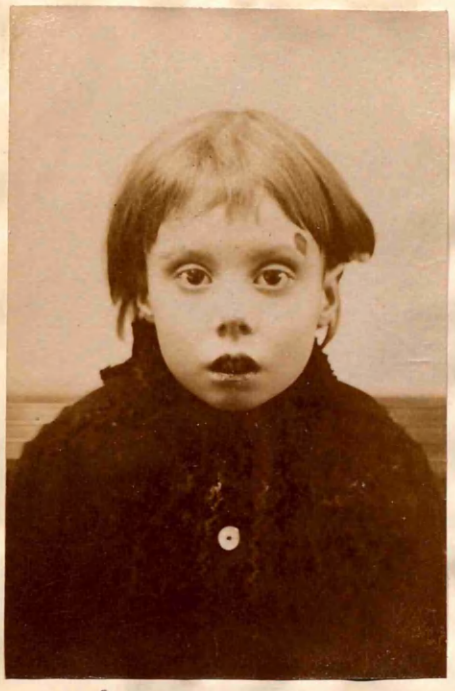
16 Streptococcus
17 Staphylococcus pyogenes aureus
18 " " "
19 No growth
20 "
21 Staphylococcus pyogenes albus
22 No growth
23 No growth
24 Streptococcus
25 No growth.

Results.

The general improvement following operation is usually rapid & complete. It has often been astonishing with what rapidity the marked adenoid physiognomy has vanished and in one case, 3 days after operation a mother refused to believe that a certain child was her boy until he insisted that he was her son. In most cases, however for a few days there is little alteration observed owing to the blood clot and exudation in the nose & nasopharynx. By the end of that period quiet breathing is established and they pass restful nights. The face gradually assumes a natural expression, the vacant anxious depressed stare gives place to a look of intelligence and buoyancy (see photographs page 117, 118) The mouth closes and becomes natural. The nose develops and the swelling and congestive appearance at its root between the eyes subside.

The superior maxilla enlarges and the arching of the eyebrow becomes less apparent.

The various facial muscles develope and the range of expression is increased. Sometimes the adenoid face persists after operation and requires forced education



before operation

6 months later

Christina Orions, aged 5.

before operation

6 months later

Cissy Hesketh, aged 3.

Before operation

6 months later

Stanley Beveridge, aged 8.

Before operation

6 months later

Percy Moore, aged 14.

to overcome the habit of oral respiration. The general condition of the patient is still more striking. Mothers in returning affirm that the temperment of their children is quite altered, that whereas formerly they were dull and lethargic now they are bright and active.

They make rapid progress at school & take a healthy delight in every exertion. They no longer complain of being tired and weary but delight to run & play. Anaemic girls lose their anaemia without the administration of iron. To estimate the increase of weight and height following operation I had 100 children over 4 years weighed and measured before operation (see tables pages 63, 64), and records made 6 months later with the result of an average gain in weight of 6½ lbs in the 6 months. The average gain in weight of a healthy child is according to 'Robert's Anthropometry' about 4 lbs in a year or 2 lbs in 6 months. This shows that the gain in weight following excision of adenoids is over 3 times what it naturally is. The increase in height was equally striking giving an average of 2¾" in 6 months. The normal increase in

height, from the same table, is 2" in one year, i.e., 1" in 6 months.

After excision of the regulations then the growth during the first 6 months is about 3 times what it normally should be. These figures speak well for the beneficial effect of operation, and enforce the statement of the advisability of early operation.

Not only are the features and general health greatly improved but the derangements of the nervous system, special senses & respiratory system are cured or greatly abated.

Headaches and spasmodic cough have been relieved, 2 cases of incontinence of urine cured & one case of petit mal greatly modified

There has been no return of attacks of laryngismus stridulus in any of the patients.

Nausea & sickness is not so common.

The beneficial effect of operation is possibly more marked on the respiratory system than on any other.

Sleep is quiet & constant, when formerly it was noisy and interrupted.

The constant tendency to catch cold diminishes, children who before operation were never without some bronchitis aggravated into an acute exacerbation on the slightest

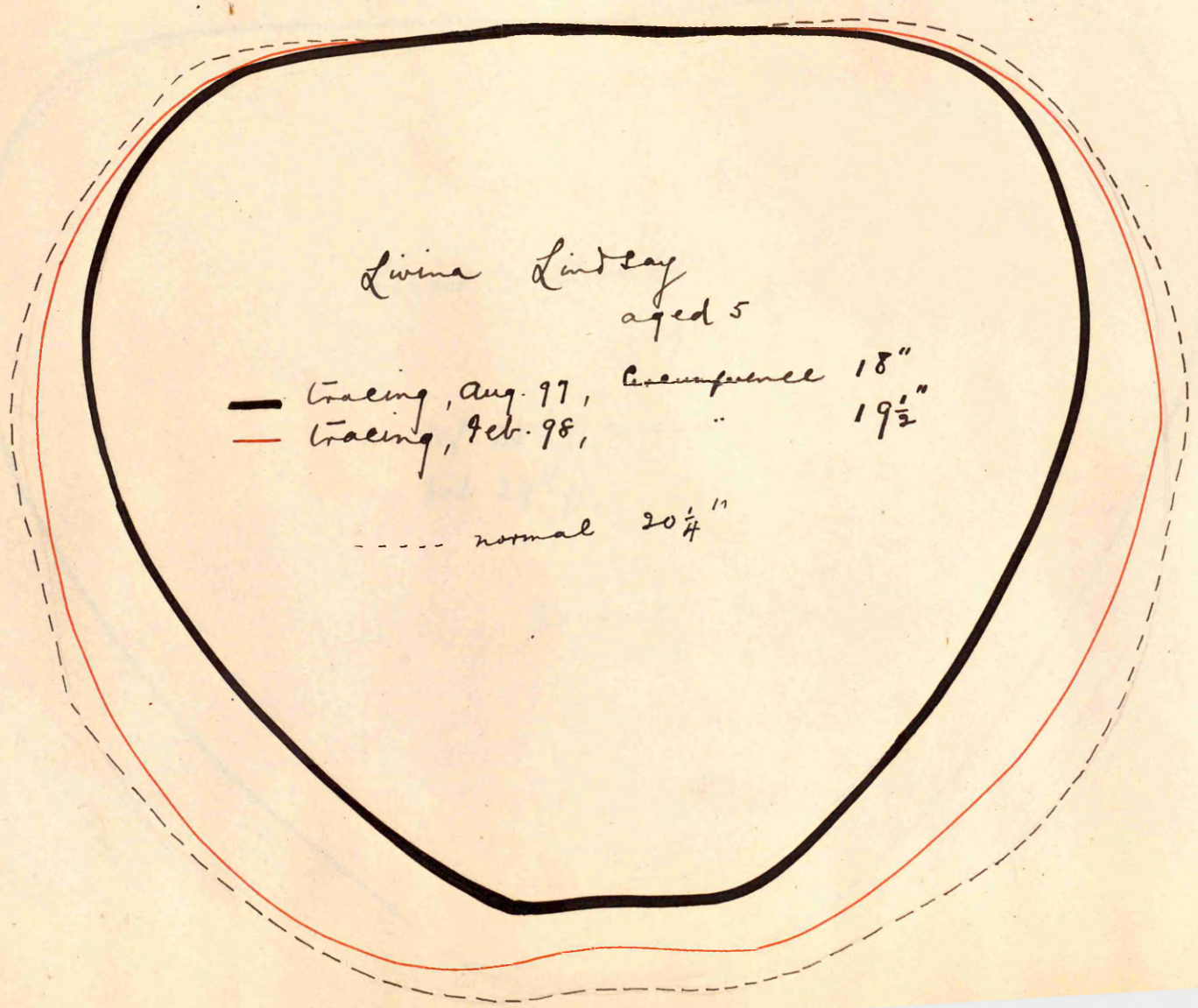
exposure now are able to run about in all weathers without cough or lung trouble.

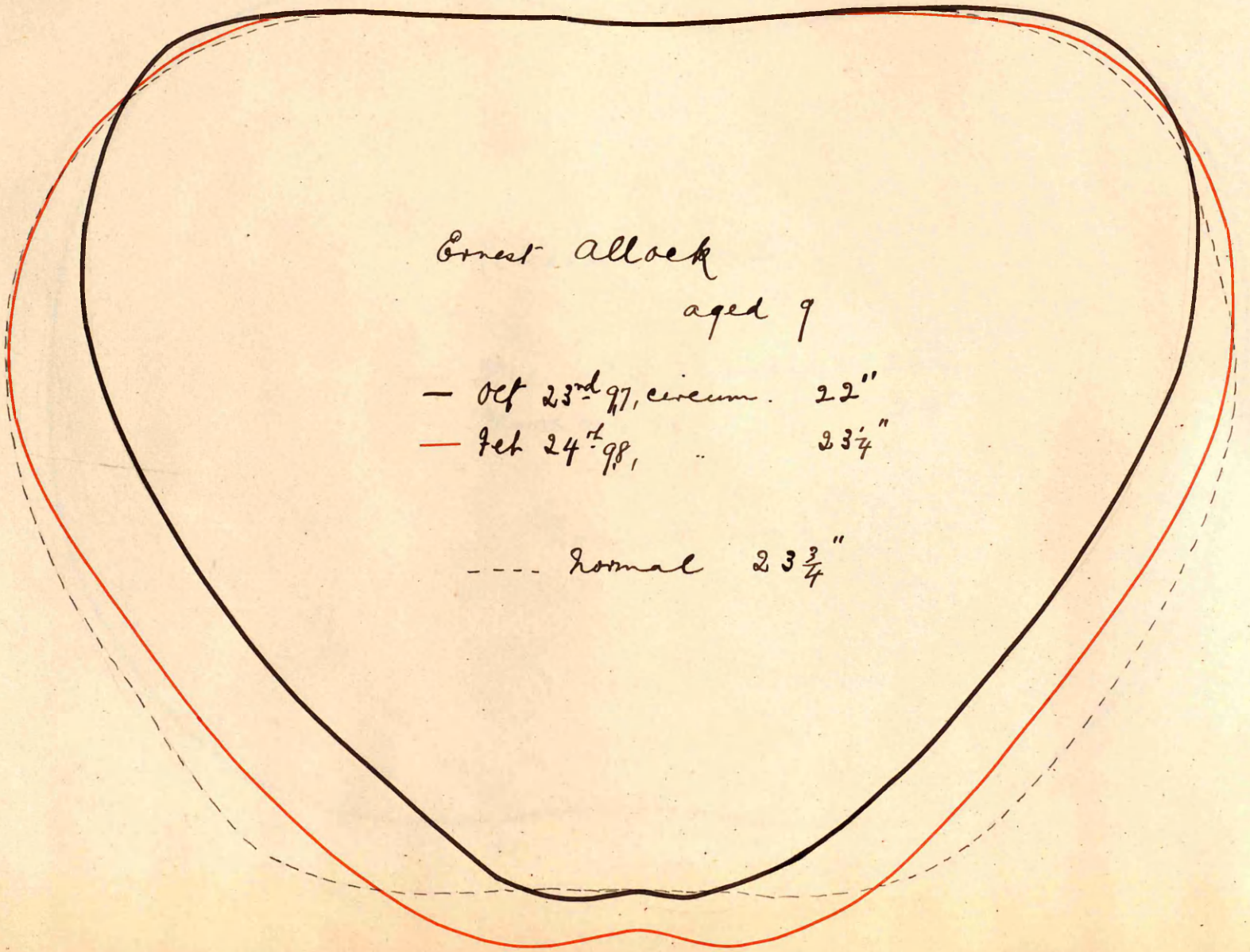
The expansion of the chest showing the increased development of the thorax and the larger respiratory capacity is well shown in the accompanying cyrtometric tracings (page 122-127)

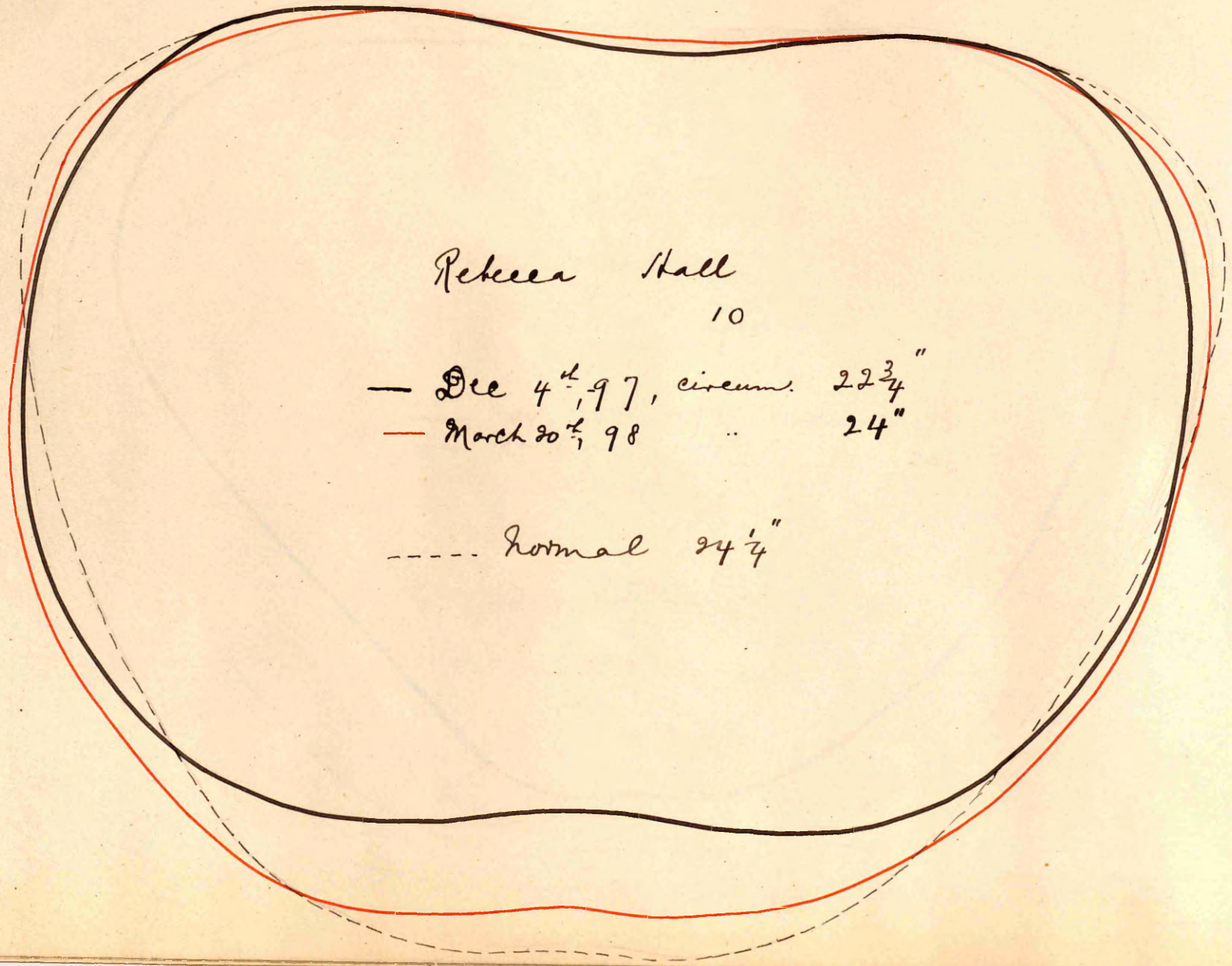
These are a few out of a large number and may be taken as average examples of the increased chest measurement with a gain of about 1½" in 5 months. It will be observed also that the tracing after operation resembles more the normal type.

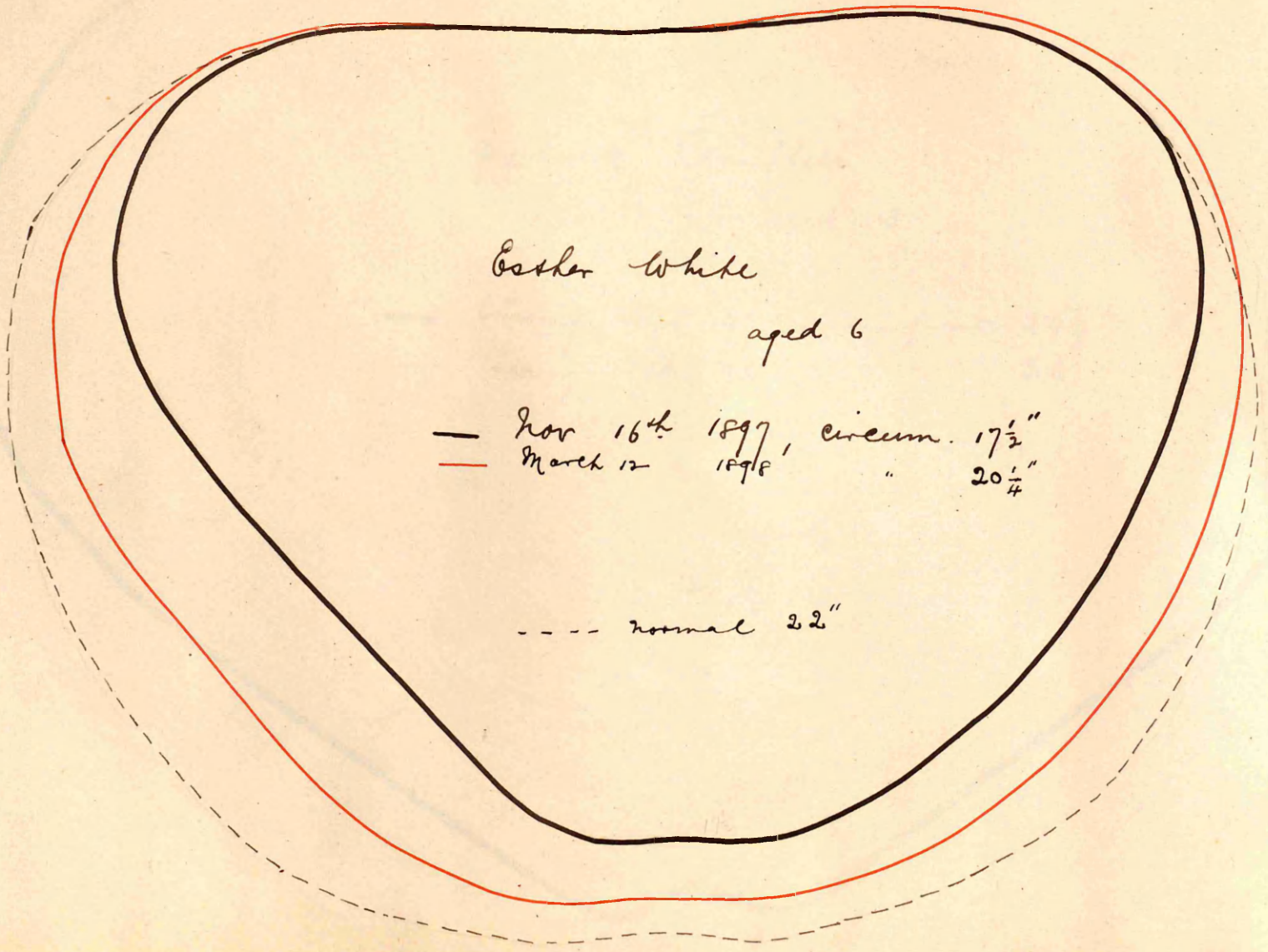
The effect on hearing has been an important one and one that also shows the value of early operation.

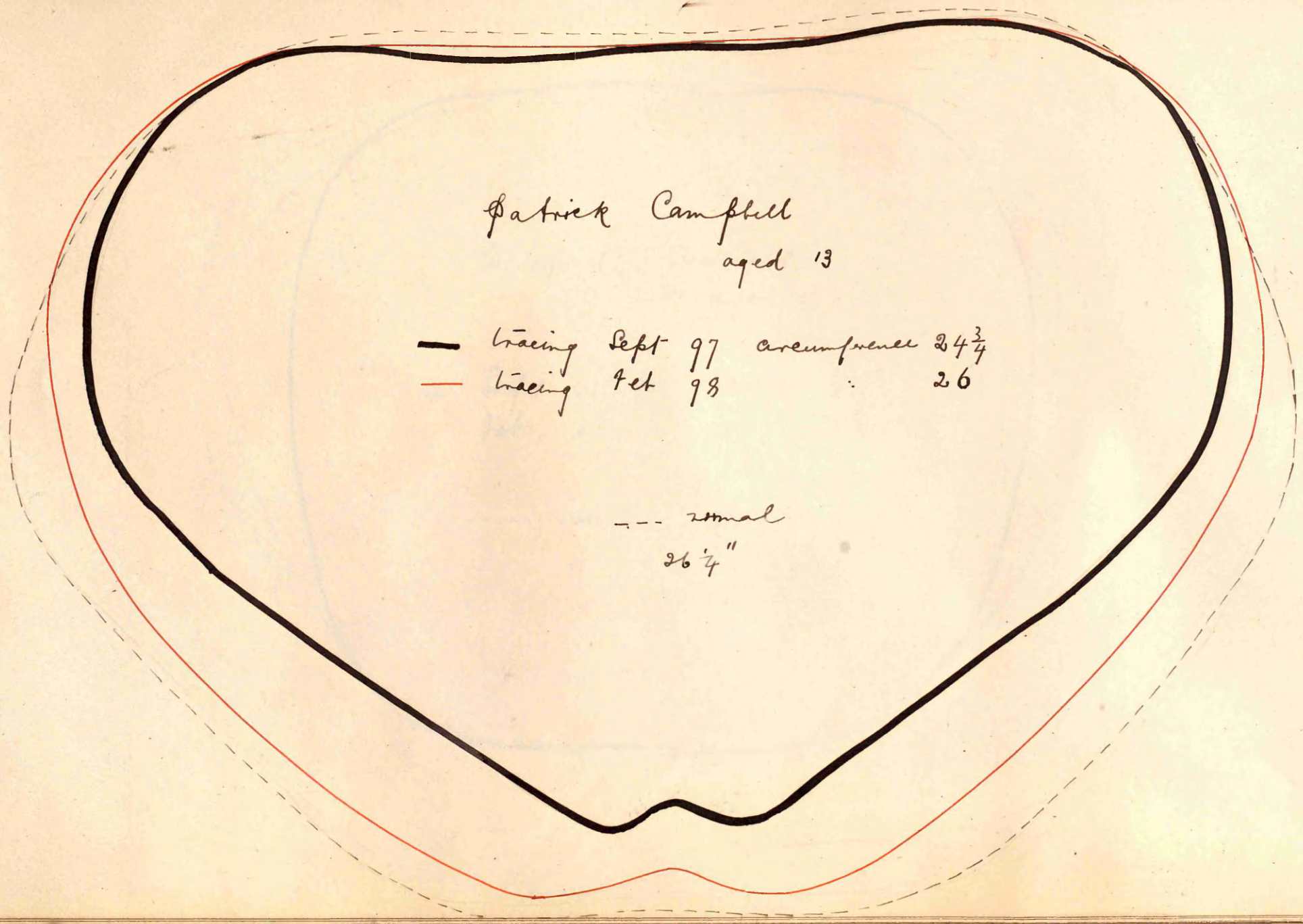
The hearing of the older patients who could give reliable answers was noted before operation with the 'watch', 'whispering' & ordinary conversation & tested again at intervals of a day, a week & 6 months. Less favourable results have been obtained in cases where operation has been performed after ear complications in the shape of otorrhoea has presented itself, especially where the otorrhoea has persisted for a long time, and in cases of long standing adenoid vegetations without otitis media

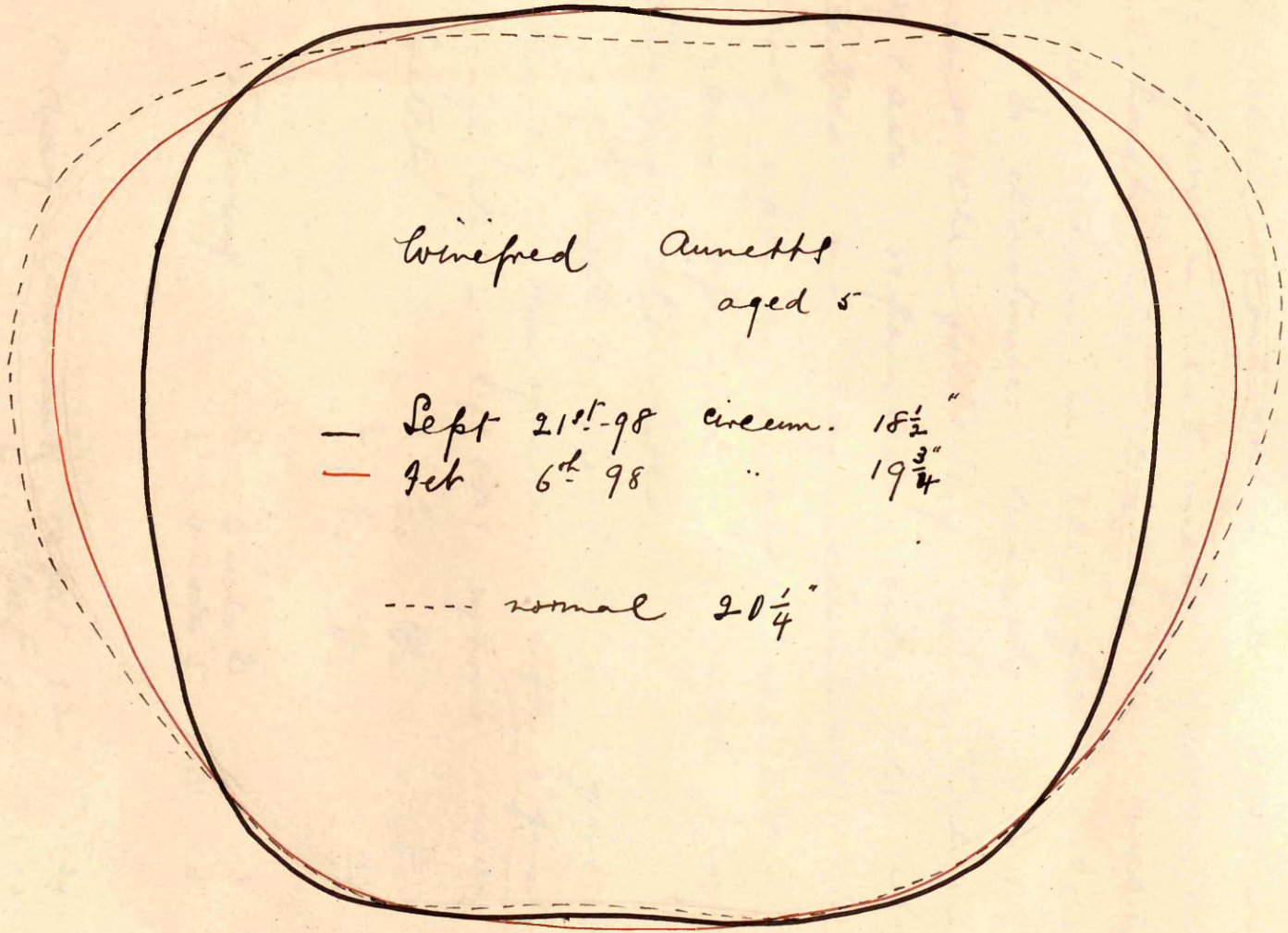

Livina Lindsay
aged 5
Tracing, Aug. 97, Circumference 18"
Tracing, Feb. 98, " 19½"
normal 20¼"


Ernest Allock
aged 9
— Oct 23rd 97, circum. 22"
— Feb 24th 98, " 23¼"
---- Normal 23¾"


Rebecca Hall
10
— Dec 4th, 97, circum. 22¾"
— March 20th, 98 " 24"
----- Normal 24¼"


Esther White
aged 6
— Nov 16th 1897, circum. 17½"
— March 12 1898, " 20¼"
---- normal 22"


Patrick Campbell
aged 13
tracing Sept 97 circumference 24¾
tracing Feb 98 " 26
--- normal
26¼"


Winifred Annetts
aged 5
— Sept 21st-98 circum. 18½"
— Feb 6th 98 " 19¾"
----- normal 20¼"

purulenta. but with great retraction of the membrana tympanum.

On the other hand, cases that are operated on early before septic infection has extended to the ear and before the middle ear is much contracted or 'sclerosed,' operation is extremely favorable even although the deafness is much greater than in the former case.

To illustrate exactly what is meant the following is a tabulation of cases separated into the above classes and may reasonably be look upon as average results

1. Cases operated on with long standing otorrhoea.

A.G., aged 10, otorrhoea for 5 years both ears.

Hearing (R=right side L=left side)	before operation	After operation 24 hours	1 week	6 months
'Watch'	R. $\frac{8}{40}$	$\frac{12}{40}$	$\frac{12}{40}$	$\frac{15}{40}$
	L. $\frac{5}{40}$	$\frac{11}{40}$	$\frac{15}{40}$	$\frac{12}{40}$
'Whispering'	R. 3 inches	2	3	4
	L. 4 inches	5	5	10
'Ordinary Conversation'	R. 10 feet	12	14	14
	L. 8 feet	15	13	17

L.M., aged 9, otorrhoea intermittent 6 years, (double)

'Watch'	R.	$\frac{1}{40}$	$\frac{4}{40}$	$\frac{4}{40}$	$\frac{7}{40}$
	L.	$\frac{6}{40}$	$\frac{7}{40}$	$\frac{8}{40}$	$\frac{10}{40}$
'Whispering'	R.	4 inches	5	5	9
	L.	2 inches	4	7	9
'Ord. Convers.'	R.	2 feet	4	7	7
	L.	7 feet	9	10	14

2 Cases operated on after acute otorrhoea of short duration.

A. C., aged 8, otorrhoea 2 weeks

Hearing		before operation	after operation 24 hours	1 week	6 months
'Watch'	R.	$\frac{4}{40}$	$\frac{16}{40}$	$\frac{20}{40}$	$\frac{28}{40}$
	L.	$\frac{2}{40}$	$\frac{10}{40}$	$\frac{15}{40}$	$\frac{25}{40}$
'Whispering'	R.	·1 inch	4	4	7
	L.	3 inches	5	5	10
'Ord. conver.'	R.	4 feet	10	21	22
	L.	8 feet	9	17	18

P. L., aged 7, otorrhoea 10 days

'Watch'	R.	$\frac{2}{40}$	$\frac{10}{40}$	$\frac{12}{40}$	$\frac{18}{40}$
	L.	$\frac{7}{40}$	$\frac{16}{40}$	$\frac{17}{40}$	$\frac{16}{40}$
'Whispering'	R.	1 inch	3	2	5
	L.	2 inches	4	6	8
'Ord. Conver.'	R.	2 feet	10	12	15
	L.	6 feet	15	18	19

3 Cases operated on with long standing deafness, no perforation but membrane greatly indrawn

A. G. aged 10, deafness 6 years.

Hearing		before operation	after operation 24 hours	1 week	6 months
'Watch'	R.	$\frac{0}{40}$	$\frac{1}{40}$	$\frac{2}{40}$	$\frac{3}{40}$
	L.	$\frac{3}{40}$	$\frac{4}{40}$	$\frac{4}{40}$	$\frac{9}{40}$
'Whispering'	R.	0 inches	½ inch	½	1
	L.	1 inch	2	2	5
'Ord. Convers.'	R.	½ foot	2½ feet	3	4
	L.	5 feet	7 feet	10	12

4 Early operation before otitic media has developed or before much in-drawing of the tympanic membrane

F. D. aged 7 'adenoids' 3 months

Hearing.		before operation	24 hours	1 week	6 months
'Watch'	R.	1/40	17/40	24/40	29/40
	L.	4/40	13/40	28/40	32/40
'Whispering'	R.	2 inches	3	10	10
	L.	1 inch	4	6	9
'Ord. Convers'	R.	2 feet	10	24	26
	L.	5 feet	12	20	29

J. D. aged 7 'adenoids' 4 months

'Watch'	R.	4/40	20/40	31/40	33/40
	L.	15/40	16/40	32/40	32/40
'Whispering'	R.	2 inches	3	7	9
	L.	5 "	7	12	13
'Ord. Conv.'	R.	8 feet	8	29 feet	29
	L.	17 feet	16	32	35

The sensation of smell was largely improved by the operation. Before operation 43% of the cases could not distinguish the odour of an onion whereas testing 6 months afterwards only 9% failed to correctly respond to the sensation.

The sensation of taste was equally

satisfactory, the 4 cases who could not distinguish salt from sugar detected afterwards both correctly, and, in the cases where it was formerly impaired the sensation appeared improved.

The change in speech has been rapid and decided. In most cases the 'nasal' tone persisted to some degree for weeks or months but the characteristic "dead" phonation was lost. The long chronic inflammatory changes in the soft palate and fauces render the mobility of these parts less active & delicate, so that a considerable training in phonation is often required before the speech becomes satisfactory and the persistence of the palate deformity tends to favour the delay in acquiring correct pronunciation.

It is almost too early to expect any alteration in the bone deformities, that accompany adenoid vegetations, to have occurred in the space of a year. The deformity of the chest certainly has greatly modified in a number of the patients but no decided or obvious alteration has been noted in the shape of the palate or the altered bony conformation of the face beyond an apparent increase in the size of the antrum and nose.

It is probable that the deformity

in the palate will diminish through time but its persistence and effect on the voice render an early operation the more imperative to prevent the high arched ⊢ V shaped palate from developing.

It is too soon after operation to give statistics of any special value regarding recurrences. In only one case has there been a return of the adenoids and that patient was operated on again not so much for the size of the vegetations but for dulness of hearing persisting unaltered after the first operation.