ATROPHIC RHINITIS

By JAMES ADAM (Glasgow)

MUCH has lately been written regarding atrophic rhinitis; it was a special subject of discussion at the International Congress at Madrid in 1932*; yet there is little agreement as to its ætiology. Predescu-Rion[1] says it is a mystery; recent text-books are also non-committal, though Sir St Clair Thomson[2] cautiously admits that sinusitis is becoming a more probable factor than had been believed hitherto. What strikes one about this literature is that great attention is paid to the last stage of the malady, much less to the early clinical history. Surely this is a false start; neglect of the obvious, causes obscurity and leads to mystery and search for the recondite.

In a paper[3] read at the British Medical Association twenty-four years ago, I advanced clinical and pathological evidence in favour of Grünwald's theory that atrophic rhinitis is the end stage of an hyperplastic, i.e. inflammatory, process, that sinusitis often plays an important part in keeping up the chronicity of the disease, and that this sinusitis is often missed. That paper was based on 30 cases; this one, based on 141 seen during the last thirty years, goes, after consideration of the clinical and pathological evidence, to support the previous one. The main addition I would make relates to the importance of deficiency of vitamin as a factor in the early stages.

When Leroux-Robert and Costiniu say that *the* characteristics of atrophic rhinitis are atrophy, crusts and fœtor, and are the signature of it, would it not be wiser to add that this signature is subscribed at the end of a long clinical document? They are far from alone in thinking thus, yet not one of these three things is of the essence of the disease. As well say that general paralysis or locomotor-ataxia is the signature of

* The chief report is by Leroux-Robert (Paris) and Costiniu (Bucharest). It extends, with a voluminous bibliography, to 117 pages and covers the ground so completely that it is taken as an up-to-date presentation of the subject and has served in the present paper as a basis for criticism. They have re-published their report in Portmann's *Rev. de Lar. Oto. Rhinol.*, January and February, 1933. They will be referred to here as the Madrid reporters, though there were others.

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syphilis and neglect the chancre. *With some distinct exceptions*, the clinical history of atrophic rhinitis begins as suppurative rhinitis during or before school days—" a dirty nose " needing many handkerchiefs unless (and worse) none is used at all. This I pointed out in my former paper. Douglas Guthrie[4] has since instanced similar cases, as also have Moure[5] and others. It is mainly with this type that this paper deals.

It is convenient to divide the series into three groups :— (A) Those in whom nasal discharge began in the first seven years of life ; (B) Those in whom it began in the next seven ; and (C) Those of later date.

The reasons for this division are :—

(1) Fourteen is the school-leaving age (and many of these patients were examined in connection with schools), while at seven they are beginning to be referred for special examination ; (2) atrophic rhinitis is recognized as often influencing facial development, which in turn is closely related to the eruption of the teeth and the development of the paranasal sinuses. At the age of seven the permanent teeth are beginning to erupt, at fourteen most of them are set and the nasal and paranasal cavities are well established ; (3) after fourteen, puberty begins, and most writers discuss puberty and ozæna together as being in some way related.

In an analysis of the series it is to be noted that the older the patients the less definite they are apt to be in dating the beginning of their trouble, so that figures stating the duration of the disease are often short of the truth, and doubtless many patients of group (C) should come under (B) and of (B) under (A).

Indifference and neglect as to nasal symptoms until the condition becomes ozæna and socially repugnant, are astonishingly common and partly account for the so-called mystery and the dispute as to cause.

In Table I. the cases are divided according to the age at which symptoms began :—

TABLE I.

Age at or before which symptoms began.

GROUP :—	(A)	(B)	(C)	TOTAL.
Age	1–7	8–14	15 and later.	
Number of Cases	59	51	31	141

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That is, in at least 42% the disease began before the age of 8, in 36% more, before the age of 15. In only 22%, therefore (probably much less), did it begin after the school-leaving age; in fully 78% (probably a distinctly higher figure) it began during the important years of facial development and before puberty. This high figure for an early beginning contrasts strongly with those quoted by Leroux-Robert and Costiniu and shows how misleading as to ætiology are figures based on the age of the patient when first seen, and which link the disease mysteriously with puberty.

The average age of patients of group (A) when first seen was 16½, the average duration of their disease thirteen years. The oldest was 52, one was 41; the youngest, in whom the disease had existed at least six months, was 14 months; three others were under 2 years. Reference will be made to five of these early cases presently. In group (B) the average age at which the patient was first seen was 21—the oldest was 60, the youngest 9; the average duration of the rhinitis 14 years, the shortest 1 year, the longest 40. An attempt to get similar figures for group (C) was useless owing to the indefiniteness of patients' statements. It is very important to note that only 6 of the 31 patients of class (C) could give anything approaching an accurate date for the beginning of their nasal trouble. This point will be discussed with the ætiology. Probably the whole of the remaining 25 should come under groups (A) and (B).

Alleged Cause

In group (A) of 59 patients, 7 ascribed the disease to scarlet fever, 6 to measles, 3 to a combination of these, 7 to diphtheria, 3 to whooping cough, 1 to trauma and 1 to chill. Three were probably parasyphilitic cases. Five others call for short notes :—

(1) A girl of 22 attributed her disease to being infected by a school-mate, who had used a brush for her own " dirty " nose, putting it into the patient's; her nose, however, according to her mother had been " stuffy " from babyhood.

(2) A baby of 14 months had had nasal discharge for at least six months, when her father noticed fœtor. Her mother, aged 37, had had atrophic rhinitis for over thirty years (multisinusitis, Abel's Bacillus, Friedlander's Bacillus and one resembling Klebs-Loeffler, etc.). Being anosmic the mother had not noticed fœtor

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in the child (in whose nose pneumococci, staphylococcus aureus, streptococcus and pneumococci, but nothing resembling B. Klebs-Loeffler, was found). Pus and stinking crusts were found in the baby's right nostril; the left was not affected. Antiseptic treatment cured the child; when seen again at the ages of 8 and 22, the nose was normal.

This was clearly a case of infection from mother to child; the different varieties of organisms in the two cases does not preclude direct infection; the flora in the same case of ozæna is found to vary from time to time.

(3) The next case, also unilateral, is one of infection from mother to child, but in a different way. The mother says that after the birth she was laid up for six weeks owing to sepsis in the uterus. She noticed nasal discharge in the baby within forty-eight hours of birth. Seen when five months old, it showed profuse purulent discharge from the right nostril. No foreign body was present, and as one does not puncture an antrum in so young a baby, argyrol was prescribed. There was no improvement when the child was seen again at the age of two. In the interval the diphtheria bacillus had been reported in pure culture but was not found at my second examination. Puncture of the antrum yielded thick pus. An opening was made below the inferior turbinate but always tended to close despite repeated enlargement, and the discharge still persists despite antiseptic treatment. Had I not missed the sinusitis at the first examination would there not have been a better chance of cure?

The right to include this case under atrophic rhinitis may be challenged, but it is deliberately included because it is in line with the clinical history of other unchallengeable cases. Moreover, the right nostril is already wide, the turbinates look as if they are beginning to shrink. Microscopic examination, by Dr. F. E. Reynolds, of the antral party wall showed the mucosa to be densely infiltrated by cells of a chronic inflammatory type, with only an occasional polymorphous cell; the connective tissue stroma was increased in density, the walls of many blood-vessels were markedly thickened. (These are similar to the changes found by Lautenschläger in ozæna.) The mucosa still retains its columnar lining; and mucous glands, though few, are not atrophic. This is what one might expect in so young a case. Certain of the bony trabeculae show absorption by osteoclasts, no deposition of bone is occurring. This again is what we should expect at the beginning of this disease. The mucosa of the lower turbinate shows many actively secreting glands and is densely infiltrated by cells of chronic inflammatory type. Treatment of sinusitis in so young a child is supremely difficult; the Caldwell-Luc operation is, of course, barred. One may ask how can the ethmoid escape involvement in such a case?

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(4) Of equal interest is the fourth case :—

A schoolboy, aged 13, of repulsive appearance, with flat nose, blepharitis, conjunctivitis, rhagades round his mouth, and stinking discharge from his right nostril. The cause was a gaming-die which he had put into his nostril seven years before and which had been missed by the medical inspector at school and by the attendants at a hospital to which he had been sent for scarlet fever. His facial appearance suggested syphilis but the Wassermann reaction was negative. There was prompt improvement on removing the die, but I saw little of him till now, five years later. His nose shows the changes of atrophic rhinitis, and he has still to undergo operation for maxillary sinusitis which, as in the previous case, I had missed.

(5) The fifth case was that of a girl of 15, whose nose was fractured at birth during delivery with forceps, neither mother nor child being expected to live. The nose presented crusts, fœtor, atrophy, disappearance of ethmoid sponge, small sinuses and a depressed bridge. The face was similar to that of the preceding case.

Thus, in 33 cases (56%) of group (A), there was strong evidence that the nasal disease was due to infection.

In group (B) of 51 cases, scarlet fever or measles was blamed in 5, chill in 2, diphtheria in 1, trauma in 1 ; in the 31 cases of group (C), trauma in 3, diphtheria in 1. In the older patients of the last two groups it was naturally more difficult to get at a definite cause, but the evidence against a specific organism is fairly strong ; it is in favour of the disease beginning as an inflammatory process and against its being of a degenerative nature.

Corroborative evidence that the atrophy and ozæna appearing after puberty form the end-stage of an inflammatory process of the nasal mucosa beginning years before, is the frequent coincidence of hyperplasia with atrophy. This occurred in 22 cases of this series. The hyperplasia in such cases is usually in and around the middle turbinate, while the lower is atrophic. In 8 cases polypi were present, in 2 of them inside the antrum. One of these is the ozænatous mother who infected her baby. She was first seen when 28, before marriage. She had typical facies, crusts, fœtor and atrophy, especially of the lower turbinates, sinusitis of both antra, both ethmoids and the left frontal and sphenoidal sinuses. The usual

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types of organisms were found, including Abel's, and one indistinguishable from B. diphtheriae. The histology of the lower turbinate showed multi-layered, horny epithelium, fibrous stroma, and thick-walled vessels, but the mucous glands were fairly preserved. The sinuses were operated on in addition to other treatment. When seen thirty years later, she had long been able to give up her nasal douche, there being little crust; but the atrophy of the turbinates and ethmoid was marked.

Adenoids, another form of hyperplasia and a fertile cause of purulent rhinitis, had to be removed in some of the children. The relationship of adenoids to sinusitis was not systematically investigated, but in 8 cases of which I have notes, sinusitis was found in 7.

Sinusitis

This was proved in just over half of the cases in each group, 72 in all. The prolonged stooping posture, suction, proof puncture, transillumination and X-ray, were the means used. If unilateral darkness on transillumination, together with unilateral rhinitis (purulent or mucopurulent or crusts) were accepted as proof of sinusitis, 21 cases more could be added, making 66%. This is a low percentage, for in 20 cases there was not fair opportunity for investigation, but it is high compared with that generally given. Leroux-Robert and Costiniu, for instance, conclude that the influence of sinusitis in producing atrophic rhinitis is not proven, that sinusal complications of ozæna are rare. They make the extraordinary statement that maxillary sinusitis is generally of dental origin! Predescu-Rion found among 460 patients with ozæna only 32 with sinusitis. In Coates and Jackson's text-book, J. M. Ingersoll, writing on atrophic rhinitis, says (p. 55) "the cause is unknown"; and Harmon Smith (p. 230) says of atrophic pharyngitis that it is, in many cases, associated with sinus disease and most often secondary to atrophic rhinitis; while L. A. Coffin (p. 138), writing on sinusitis in children, says:—"One of the most frequent and serious sequelae of chronic sinusitis in children is atrophic rhinitis and ozæna." In Dan McKenzie's text-book sinusitis is not mentioned; St Clair Thomson inclines to think that the importance of sinusitis has been under-estimated.

In this connection it is important to emphasize that all the sinuses but the frontal sinus are present at birth or shortly after,

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though the maxillary and the sphenoidal sinus may be little more than slits. At any rate, within a year or two they are developed enough to be distinct. Examination for sinusitis, especially in refractory children, is not easy. The absence of sinusitis can rarely be proved in one sitting and diagnosis usually requires several examinations. The more one examines atrophic rhinitis the oftener one finds sinusitis. Negative antral puncture is apt to be taken as proof of absence of sinusitis. Negative puncture may be misleading for various reasons. It was so in one case of this series, in which the antrum was full of pus ; in a case of bronchiectasis there was sinusitis caseosa which had been missed by various rhinologists, the pultaceous mass coming away only after two minutes' douching. But what of the sphenoid and ethmoid cells ? I have seen these involved in atrophic cases, yet the antrum was free from infection. In the adult atrophic nose it is easy to see the sphenoidal ostium and, often, to get pus from it, but its investigation is not practicable in children. St Clair Thomson, however, justly remarks on the likelihood of the sphenoidal cavity being affected in the purulent rhinitis accompanying adenoids, and also on the diminishing frequency of atrophic rhinitis since the operation for adenoids has become a common practice on schoolchildren. A similar remark applies to the ethmoid of children ; when they have purulent rhinitis lasting for years the ethmoid cannot escape. Proof of this is seen after puberty ; often the ethmoid is then a mere shell with the orbital plate and stump of the middle turbinate as its walls, the spongy structure having vanished.

The X-ray has rendered investigation of the sinuses easier, but it has increased rather than diminished the percentage of sinusitis ; examination of the earlier cases in this analysis was before the advent of the X-ray and perfection of its technique.

The antrum was involved in 62 cases, the ethmoid in 30, the frontal in 5, the sphenoid in 10. In relation to each other these figures are not worth much, except perhaps as regards the frontal sinus, which is the last to develop, has an outlet favouring drainage, and is therefore less often involved. Of the sinuses, the antrum naturally invites most attention but Mosher says the ethmoid is in the key position in relation to the other cavities. In at least 15 cases there was multi-sinusitis.

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Sex

The proportion of females to males was 2 : 1 :—3 : 1 is the figure commonly given. This predominance of females may be partly due to the fact that at puberty, when fœtor and self-consciousness come into play, the female pays more attention to a disability which tends to social ostracism and makes her seek treatment. This may not be the whole explanation. Brindel finds the proportion between the two sexes nearly equal.

Familial Incidence. There were eight families with more than one member affected.

Case VII.—Woman of 38; disease began after scarlet fever at 6. She came, not because of her nasal condition, but because of atrophic pharyngitis, with crusts extending to the larynx. Sinusitis of ethmoid and sphenoid; antra negative. One of a family of ten, she had two sisters similarly affected—her son, when 16, had double antral disease cured by operation. She herself refused operation.

Case VIII.—This is the case mentioned of the mother who infected her child (p. 377).

Case IX.—Woman with one sister similarly affected, and a son in the hyperplastic stage.

Case XI.—A woman of 43. Her disease, probably due to measles of which she had three attacks, had lasted twenty years. She had atrophic pharyngitis. She was seen in 1906 and had acquired syphilis in 1900 *after* her three children were born. After two years' treatment crusts had disappeared except for sticky secretion in the pharynx. The three children, girls, aged 12, 10 and 8, all had nasal trouble suggesting atrophic rhinitis, and the antrum was certainly affected in the eldest one.

Cases XXIV and XXV.—A brother (19) and sister (10). Operation on sphenoid cured the brother; the sister was cured without operation; her left antrum was doubtful on transillumination.

Cases LVIII and LIX.—A brother (32) and sister (38), both with disease lasting for years and both with sinusitis. The former was cured, the latter improved after operation. The latter still has sinusitis.

Case CXV.—A boy (7), whose disease was due to diphtheria. His mother had the facies of atrophic rhinitis but refused examination.

Case LXXXVII.—A girl of 15 (with sinusitis); her mother had atrophic rhinitis.

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The inference is, therefore, that heredity is not an important factor in producing the disease as compared with infections, especially those common in childhood. In one case the infection was clearly from mother to child, in another probably so. In line with this is Viggo Schmidt's[6] case in which a mother and seven of her nine children were ozænatous, while the father acquired ozæna *six years after his marriage*.

Tuberculosis and Syphilis

These have been much discussed as having a causative rôle in atrophic rhinitis. The present series negatives the idea, not only because of the absence of the well-known stigmata of lues or of tubercular lesions, personal or familial, but the robust health of most of the adult patients and of many of the children was one of the most striking features of the series. In three patients phthisis appeared during atrophic rhinitis; two of them are known to have died. Nasal syphilis is quite a different thing from atrophic rhinitis and has, so far as is known, been excluded here; but three possibly parasyphilitic cases have been included—the family history suggesting this, despite a negative Wassermann reaction in the mother in one case. It is possible that, even in the absence of congenital syphilitic stigmata, a hereditary taint, or a family tendency to tubercular disease, may weaken resistance to those infections which produce nasal suppuration in children, but this can be so only in a minority of cases.

Bacteriology

Bacteriological examination (never by myself) was carried out regularly only in the earlier cases of the series. A great variety of organisms was found; one observer has listed 56. The organisms vary not only in different cases, but in the same case from time to time. Recent writers have concentrated on three—B. Abel-Loewenberg, the diphtheriform of Belfanti and della Vedova, Perez' bacillus. The first I have found in every case with crusts and fœtor, the second commonly, the Perez never. Abel's is associated with the crusts; when they are absent, so is the bacillus. It is certainly not the cause of the disease; opsonic observation made on an early case while vaccines were tried from a culture giving a pure growth, showed a positive but no negative phase, thus indicating

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absence of specificity. This organism seems to be a saprophyte, finding a suitable nidus in the atrophic nose, though Leroux-Robert regards it as causing the abundant secretion of mucus. The diphtheroid he regards as the diphtheria bacillus which has become atoxic, but capable of forming crusts instead of false membrane. The relationship of diphtheria to atrophic rhinitis is important because some have held that this infection is the usual precursor of the nasal disease, and because the diphtheroid is a common denizen of the atrophic nose; yet without the virulence test it is indistinguishable from the true B. diphtheriae. The finding of the diphtheroid in ozæna, which certainly began with true nasal diphtheria (two cases), lends colour to this contention. The nine cases given in Table II. were associated with diphtheria bacilli proved to be virulent.

TABLE II.

Cases of Atrophic Rhinitis following Diphtheria.

Case No.	Sex.	Age.	Duration.	Side.	Turbinates:		Sinus.
					Inf.	Mid.	
XLI	M.	33	1	(L)	x		? A (L)
XLVI	F.	25	13	(B)	x	x	A (R)
LXXVII	F.	12	1	(B)			E
LXXX	F.	9	1	(L)	x		O
LXXXIII	F.	17	13	(B)	x	x	E and ? A
CXIV	F.	8	2	(B)	x	x	E (B), A (B)
CXV	M.	7	Several	(B)	x	x	E (B), A (B)
CXXXIII	F.	12	27	(L)	x	x	S (L), E (L)
CXLI	F.	1½	½	(B)	x		A

Duration is in years; (R), (L), (B)—initial letters of Right, Left, Both.

A, E, F, S,—initial letters of the sinuses.

x—atrophy.

Pus, crusts and atrophy were present in all; sinusitis certainly in all but three.

CASE XLI infected his wife with nasal diphtheria; his left antrum was dark.

CASE LXXX was probably infected from her twin sister who had faucial diphtheria. Except for atrophy of the anterior end of the inferior turbinate, the nose is now (fifteen years later) well.

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CASE CXIV was away from school for four years owing to virulent B. diphtheriae in her nose which, in spite of vigorous serum and other treatment, did not disappear until Lautenschläger's operation was done on both nostrils. Even then it was difficult to keep the antral openings patent. She has still (eight years later) affection of the ethmoid but not much discharge or crust formation.

CASE CXV was similar, but got well with the same operative treatment.

CASE CXLI was an unhealthy child of 18 months who had been admitted to hospital over a year before with pneumonia, double otorrhœa, and purulent rhinitis. It was dismissed after recovery from the pneumonia, but re-admitted a year later for broncho-pneumonia. After four months, during which diphtheroids had been found, and during which sporadic cases of diphtheria had been occurring in the wards, it was sent to me for the first time for nasal examination. Atrophy, crusts and sinusitis were found; and virulent B. diphtheriae. The child was then in a fever hospital for eight months owing to persistent virulence of the bacilli. The early report of diphtheroids was probably responsible for the sporadic cases of diphtheria.

My failure to report Perez' bacillus except in one doubtful case is probably due to defective technique on the part of the bacteriologists. Its culture is admittedly difficult. Perez claimed it as the cause of the disease, Leroux-Robert and Costiniu as the cause of the fœtor. It may share with Abel's bacillus in causing fœtor. It is certainly not the cause of the disease. Perez found this organism in dogs and supposed the infection was transferred from them to man, but the numerous cases in the present series in which there was no association with dogs, the multitude of dog fanciers in this country whose children have no ozæna, as well as the production of the disease by other organisms, are all against this claim. If, however, the B. Perez is the same as that of Friedlander, as has been suggested, then this organism was frequent in this series.

The truth seems to be that the non-functioning nose, devoid of cilia, is a good culture chamber for a host of organisms. The curious thing is that I have seen no report of the presence of Vincent's organisms, although they are commonly found in the mouth; over forty times I have found them in the ears, only once—and even that was doubtful—in the atrophic nose.

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Pathology and Ætiology

Histological examination of five cases confirms the finding of the six cases reported in my previous paper. They are similar to those of Lautenschläger and others, are well known, and need only be summarized. Change from columnar ciliated to squamous epithelium, sometimes multi-layered; infiltration of the submucosa with inflammatory round cells, prominent round the vessels, and changing later to fibrous tissue with marked thickening of the arterial wall; mucous glands at first numerous and active, later decreasing in size and number owing to the fibrosis which ultimately shrinks the whole of the soft tissue of the turbinate. This process was seen in its earlier stages in the case of the child, aged 2, already mentioned as having been infected at birth (p. 378). The mucosa and periosteum of the turbinate are intimately bound together. Therefore, one can understand how such a fibrotic process will inevitably affect a thin layer of bone like this, especially in children. It will at least prevent the turbinate bones from growing. The same is true of the ethmoid. But one of the most interesting things in the pathology is, that while the lower turbinate bone atrophies the antral party wall from which it springs usually thickens, at least in cases with sinusitis. This thickening may be so great that puncture with an ordinary trocar is difficult or impossible. In a lad of 17, whose rhinitis started twelve years before from scarlet fever, even the lateral nasal wall was found to be nearly half-an-inch thick at the radical operation; there was more wall than cavity, the antrum being a mere slit holding only a bead of pus. The operation cured the case in respect of discharge, crusts and fœtor. It is noteworthy that though the atrophy was worse on the side of the sinusitis, both nostrils were affected. Radiograms nearly always show that, though only one sinus may be affected by sinusitis, the others fail to expand normally and remain small with thick walls, especially on the side of the sinusitis. This is more marked in unilateral ozæna. Lautenschläger holds the same view.

In short, the bone reacts to suppuration of the mucous membrane when it becomes chronic—the turbinate and ethmoid sponge by atrophy, the antral wall by thickening; the difference being due to the throttling of the blood supply of the thin lamella constituting the turbinate. The spongy

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mass of the ethmoid reacts similarly; it vanishes, leaving only the walls. This statement is to be taken to apply not to adults but to the developing skull of children. Apart from the rare cases due to trauma and to chronic diphtheria, both of different type from those discussed here, it is doubtful if atrophic rhinitis occurs in adults. In this respect the analyses of cases of group (C) (p. 376) in which the disease could not be accepted as starting before the age of 15, is illuminating. Of the 31 cases in this group, only six gave a definite statement as to the date of origin of the disease. Of these one was a man of 33 who had had nasal diphtheria for a year. There was distinct atrophy of the mucosa but not of the bone. It was not like the typical case of atrophic rhinitis dating back to childhood, even when of diphtheritic origin. For that reason the case might be challenged; but for that very reason it is set down here as it shows the difference of response to chronic nasal diphtheria in a man and in a child. Two cases started from trauma. A woman of 32 attributed her trouble to a blow on the nose four years before. Anosmia, moderate atrophy of both inferior and middle turbinates, crusts on the latter; antra dull on transillumination, proof puncture negative. After three submucous injections of paraffin, the discharge diminished and the sense of smell began to return.

A boy of 17 got a kick on the nose when 15. Some atrophy of lower and middle turbinates, wall of left antrum so thick that trocar failed to penetrate; transillumination clear; discharge sometimes fluid pus, sometimes crusty.

Of the remaining three cases, the ages of the patients when seen were 27, 32 and 38, and the ages at which symptoms began, 15, 16 and 20. In the first of these, infraturbinal drainage of one antrum cured the case. The second was a unilateral case with extreme atrophy and sinusitis; this case, however, refused treatment. The third was similar.

These few and exceptional cases go to prove the rule that atrophic rhinitis is a disease *beginning mainly in childhood, while the face is developing*, and only in such cases giving the typical picture.

The rôle of sinusitis in the disease is that *it keeps alive the inflammatory process which began in the mucosa of the nasal cavum* and prevents resolution. This is especially so in children, in whom sinusitis is much more difficult to treat than in the adult. While sinusitis has not been proved in 33% of

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the cases, one thing must be remembered of this minority—the effect of adenoids. The comparatively small nasal cavity of a child, blocked by adenoids, is reduced to much the same state as that of an adult suppurating sinus, but with the nostril for ostium, and with the further disadvantage that the discharge from the nostril is against the ciliary flow. It is no wonder that after years of a struggle like this, the cilia give up and the epithelium becomes squamous, though the fibrotic change in the depth of the membrane has probably more to do with it. Hence the modern prevalence of the adenoid operation has reduced the prevalence of atrophic rhinitis. The nasal cavity gets a chance of drainage in a normal way and for the sinusitis to clear up. All the sinuses benefit by the adenoid operation, but especially the ethmoid and sphenoid.

Of great interest here, and of special value in regard to pathology and ætiology, is chronic suppuration of the middle ear in children. And this apart from the fact that a large number of these cases have had either chronic non-suppurative otitis, or—and more often—purulent otitis, and some of them have been driven to their first consultation by their aural symptoms and not by the nasal condition which caused them. In chronic aural suppuration, the walls round the tympanic antrum, like those round the maxillary antrum, react by sclerosis to the inflammation which has begun in the mucous membrane, and if the suppuration has begun during childhood the mastoid process remains small. Sections of bone removed from such mastoid processes in children show the same sclerotic process as in the wall of the maxillary antrum. Moreover, and as in the case of the latter, the tympanic antrum may remain or become small. Four times in a thousand mastoid operations I have found it obliterated by this sclerosing process in old-standing middle-ear suppuration. The aditus, when traced back from the attic, was found to be a simple canal of the same dimension right on to a blind end in the mastoid, there being no enlarged cavity that could be called the antrum. This is one of Nature's ways of cure. That this sclerosing process is secondary and a reaction to the suppuration in the mucous membrane, no surgeon who uses X-ray films as a routine measure before operation can have much doubt. In most cases he can diagnose from the films which is the affected ear ; the unaffected mastoid process shows cellularity, the other sclerosis.

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TABLE III.*

	Atrophic Rhinitis.	*Otosclerosis.*
Begins—	Usually in childhood	Usually after puberty
Progress—	Tends to improve after 30	Tends to get worse after 30
Nature—	Inflammatory	Degenerative
Pus—	Present	Absent
Bacteria—	Legion	Nil
Accessory Suppuration—	Paranasal sinuses usually affected	Mastoid cells usually unaffected
Sclerosis of Walls—	Of sinus walls, common	Mastoid cells not involved
Special Sensation—	Smell impaired, but may recover	Hearing impaired; does not improve
Common Sensation—	Hypersensitive except in late stage	Hyposensitive
Sex—	More common in females	Much more common in females
Hereditary tendency—	Infrequent	Frequent

Some have fancied an analogy between atrophic rhinitis and otosclerosis, partly because of the preference for the female sex, partly because of reduced sensitivity of the nasal mucosa in the rhinitis and of the external auditory meatus in otosclerosis; partly, perhaps, because a few cases of atrophic

* This is not the place to discuss Wittmack's views on the "sclerosed" mastoid, but the facts stated above have obvious relation to them. On his theory the dense, small mastoid is a reaction to aseptic inflammation of the tubo-tympanal mucosa begun before or shortly after birth; the mastoid cells consequently fail to develop. It is, after all, a reaction of bone to mucosal lesion. The facts given in the present paper are in line with this to the extent that they go to show that the "sclerosed" mastoid is a reaction of bone to *septic* mucosal inflammation during the later development of infancy and adolescence, as also occurs with the sinusitis so often accompanying atrophic rhinitis. Mouret and Portmann at the Copenhagen Congress of 1928, while supporting Wittmack to some extent, state that they "agree" with him in regarding the acellular mastoid as "primary", the cause and not the consequence of the chronicity of suppurative otitis, and advance in support of their view the fact that sclerosis of bone and shrunken cavities do not occur in connection with paranasal sinusitis. This objection fails before the facts mentioned in the present paper.

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rhinitis have been reported after trauma causing descending neuritis. Notwithstanding Fowler's interesting paper[7] connecting otosclerosis with a chronic inflammatory process, the association of otosclerosis with von Recklinghausen's disease (as shown in three cases described by Albert Gray[8]), and with osteogenesis imperfecta (as well as the clinical history familiar to everyone), all support Gray's view that otosclerosis is a degenerative process. It is true that in some cases of atrophic rhinitis the mucosa becomes less sensitive, but my usual experience has been just the opposite—hypersensitiveness. One patient, after thirty years of atrophic rhinitis with pronounced atrophy, used to peel onions so as to stimulate her nose to a flood that washed away the crusts. Another with an almost empty nose after nineteen years of ozæna, had his first attack of hay-fever last year, with sneezing, profuse rhinorrhœa, followed by a mass of crusts which had not troubled him for some years. One treatment by zinc-ionization stopped the hay-fever ; but his nose had to be cocainized first. While chronic aural suppuration has been common in the present series, otosclerosis was noted only twice. The real aural analogue to atrophic rhinitis is not otosclerosis but chronic aural suppuration with its attendant sclerosis. The argument is summarized in Table III.

Other Theories Regarding Ætiology

Paratubercular and parasyphilitic factors have already been discussed. They play only a minor rôle by reducing resistance in a minority of cases. Endocrine deficiency has been emphasized by Leroux-Robert and Costiniu as a cause, especially in view of the more common incidence in females and of the appearance of fœtor and crusts at puberty. The sex incidence is probably exaggerated, as has been mentioned. Brindel divides the sex incidence equally. At puberty when fœtor begins to be marked and the patient has to fight for a place in life, the girl is much more sensitive about odours than the male. A girl buys scents, a boy does not. There is, however, one way in which the endocrine factor may be important.

It is now being realized that for normal endocrine function and for resistance to infection, a due supply of vitamins is essential, and that where there is vitamin deficiency mucosal inflammation is common. In one institution where there are 400 children, I used, twelve years ago, to have a constant troop

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of children with "running" noses. Vitamins, open-air exercise, and handkerchief drill were made routine; now it is rare to see a case; when one does occur there is often sinusitis.

The Neuritic Theory

Descending neuritis has been invoked as a cause in a few cases. The Madrid reporters mention seven cases in addition to ten collected by Laval in which atrophic rhinitis appeared after damage to the fifth nerve, all due to trauma except in one case of cerebral tumour and one of meningitis. (I have noted five as due to trauma.) In most of these cases, apart from the effect of the nerve lesion in lowering resistance to infection, the initial lesion must have involved sepsis; the presence or absence of sinusitis is not indicated. The majority were adults. Such cases are a small class by themselves and not in line with the typical case discussed here. The reporters regard neuritis as an essential factor in producing the atrophy; only after atrophy does the fœtor, ozæna, come. They regard the nasal mucosa as having emunctory as well as respiratory and olfactory functions. Owing to syphilitic or hereditary taint, to the infections common in infancy, and, with the approach of puberty, to renal, endocrine and calcium deficiency, this emunctory function becomes enfeebled. The nasal secretion is no longer effective in dealing with the toxins excreted by the nose or with the toxins elaborated locally during nasal infection. The nerves bathed in these toxins react first by irritation and then by atrophy; the mucosa first by hypertrophy, later by atrophy. The bone atrophies. Once atrophy occurs the bacilli responsible for fœtor and crusts—the Abel-Loewenberg, the Perez, and the diphtheroid—get a suitable nidus in a nose which has lost its normal cleansing and protective functions. Such is the theory in brief. Its complexity seems a breach of the law of parsimony, and arises from putting the emphasis in the wrong place—on the end-stage and not on the effect of chronic purulent rhinitis (usually plus sinusitis which prevents resolution) *during the stages of facial development*, that is, before puberty. One factor, however, not mentioned by these Madrid reporters, may influence prejudicially not only the nasal epithelium but endocrines and nerves also, is deficiency of vitamin A. This will be referred to presently. But as shown above, the nose, even in pronounced atrophy, may be highly sensitive. The

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emunctory function of the nose, which is an essential part of the theory, seems problematical and has little if any place in the physiology of lower animals to which the nose is even more important than to man.

Treatment and Results

At first treatment was mainly by douche, pack and vaccines, with submucous injection of solid paraffin as surgery; but as the frequency and importance of sinusitis was realized, drainage of the sinuses became more frequent with better results. The antrum at first claimed most attention, usually by an opening below the lower turbinate, rarely by radical operation. In children one hesitates to open up the ethmoid cells, but Nature destroys them if the disease is allowed to persist, with, in addition, loss of smell and reduction of the middle turbinate to a stump, whereas both these evils can be prevented by drainage of the cells. Where I have had courage to deal with these after failure from other measures, there has been gain. Lautenschläger's operation I have dropped as unnecessarily severe.

Of 60 patients traced, 9 are *in statu quo*, 27 improved, and 24 cured. Cure means absence of crusts, fœtor and need of douching. As 15 have been seen 10-30 years later than the first examination, I give them in table form. (Figures denote years, letters sinuses.)

Paraffin injection, practised in the earlier cases, was often distinctly helpful; submucous implantation of cartilage may be even better.

In Case LXXV a perfect result was obtained, so that now it is impossible to tell that this girl showed the condition described. She was seen in 1918, when 8, with crusts and semi-fluid pus in both nostrils, starting after diphtheria at 5. Owing to septal deviation the right nostril was much the wider; both the inferior and both the middle turbinates, and the right ethmoid looked extremely shrunken, and the mucosa as smooth as that of the lip; the right sphenoidal ostium was easily seen. The patient was hoarse, the vocal cords red. After trying other treatment for a year the *left* antrum was opened and much pus obtained. After this the discharge diminished and fœtor vanished, but it was not till cartilage was implanted in both walls of the right nostril in 1923 that all symptoms disappeared and the turbinates took on a normal appearance.

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TABLE IV.

Case No.	Age when first seen.	Duration when first seen.	Interval between first and last examination.	Age when last seen.	Sinusitis.	Operation.	Result :—		Remarks.
							Improved.	Cured.	
VII	38	32	30	67	E, S	0	X		Nose still sensitive
VIII	28	20	30	58	A, E, F, S	X	XXX		Does not need douche
IX		29	28	65	A			X	Does not need douche
XXIII	13	3	25	38	0	X		X	Son of (IX); seen in hyperplastic stage
XXVI	1	½	21	22	0	0		X	Baby of case (VIII)
XXVIII	16	12	22	38	A	X		X	
LVII	38	Many	20	58	A, E	X	X		Still further improved since ethmoid operation
LXI	19	5	18	37	E	0	X		Antrum opened but negative
LXV	39	20	10	49	A, E			X	
LXXIV	24	20	18	42	A, E	X	X		
LXXV	8	8	15	23	A, E	X		X	
LXXIX	18	15	15	33	0	0		X	Otosclerosis
LXXX	9	1	15	24	0	0		X	Diphtheria case Anosmia
C	41	25	10	51	A	X		X	Began after trauma
CIX	16	14	10	26	A, E, F, S	X	X		Still has frontal sinusitis

The treatment, however, is preventive. All rhinologists with thirty years' experience are convinced that atrophic rhinitis is becoming a rarer disease. One factor has been the nearly universal removal of adenoids in schoolchildren suffering from nasal discharge and mouth-breathing. Another is the recent attention paid to vitamins. The fat-soluble vitamin A and its precursor, carotene, so widely spread in the plant world and so often wanting in the dietary of the class from which atrophic patients commonly come, are specially important here. To quote Professor Cowell[9]: "In the absence of this vitamin, the cells lining the respiratory passages, as well as those of certain glandular organs, lose their normal structure and tend to be changed into layers and nests of keratinized epithelium. This change is of great importance for the reason that the altered epithelial surfaces permit the invasion of bacteria so that infections are more likely to occur in animals suffering from a deficiency of this vitamin in their diet." The importance of this in relation to atrophic rhinitis, when viewed not as a degenerative disease but as an infection, and an infection not related to any special bacterium, is obvious. I have instanced clinical proof of it above. Moreover, Cowell continues: "There is evidence that deficiency of vitamin A leads to a variety of nervous lesions caused by degeneration of peripheral nerves and tracts in the central nervous system." Of the truth of this I have had personal experience. Attention to this vitamin factor, together with removal of adenoids when necessary, will often clear up purulent rhinitis and sinusitis in children; if discharge persists, *sinusitis is almost certainly present*, particularly in the ethmoid, and must be treated surgically. It is too often missed. With attention to these three factors—adenoids, vitamins and sinusitis—before the school age is over, the mystery of the connection of ozæna with puberty is not likely to trouble rhinologists.

Summary

Analysis of 141 cases of atrophic rhinitis shows:—

(1) That in at least 78% the affection begins before puberty, and in at least 42% during the first seven years of life. These are the years during which the face, nose and its accessory cavities are normally developing most quickly. This development is consequently retarded. When the disease begins in

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adult life, as it rarely does, the typical facies of atrophic rhinitis does not occur.

(2) The disease begins as inflammation of the nasal mucosa and in at least half the cases, probably over 66%, there is sinusitis. This tends to keep the inflammation alive and is often missed. The ethmoid is more often affected than is realized. This also applies to adenoids. The present custom of dealing with adenoids early has reduced the incidence of atrophic rhinitis.

(3) Another factor is deficiency in vitamins, especially vitamin A. This deficiency impairs defence against infection and lowers endocrine function. It may also impair nervous function.

(4) The mucosa reacts first by hyperplasia ; later, owing to fibrosis, there is glandular atrophy and change from columnar epithelium to stratified epithelium ; but if the sinusitis is conquered early enough by proper surgical and other measures there may be considerable recovery with disappearance of crusts and fœtor.

(5) The bone of the thin lamellae of the turbinates and the ethmoid cells reacts by atrophy, that of the walls of the accessory cavities by sclerotic thickening. Similar sclerosis is found in the mastoid process of children with chronic suppuration of the middle ear. The paranasal sinuses may fail to reach their full development with consequent facial modification.

For much histological work special acknowledgment is due to some Glasgow pathologists :—the late Professor Teacher, Royal Infirmary ; Drs. John Anderson and J. A. M. Cameron, Victoria Infirmary ; and Dr. F. E. Reynolds, Stobhill Hospital.

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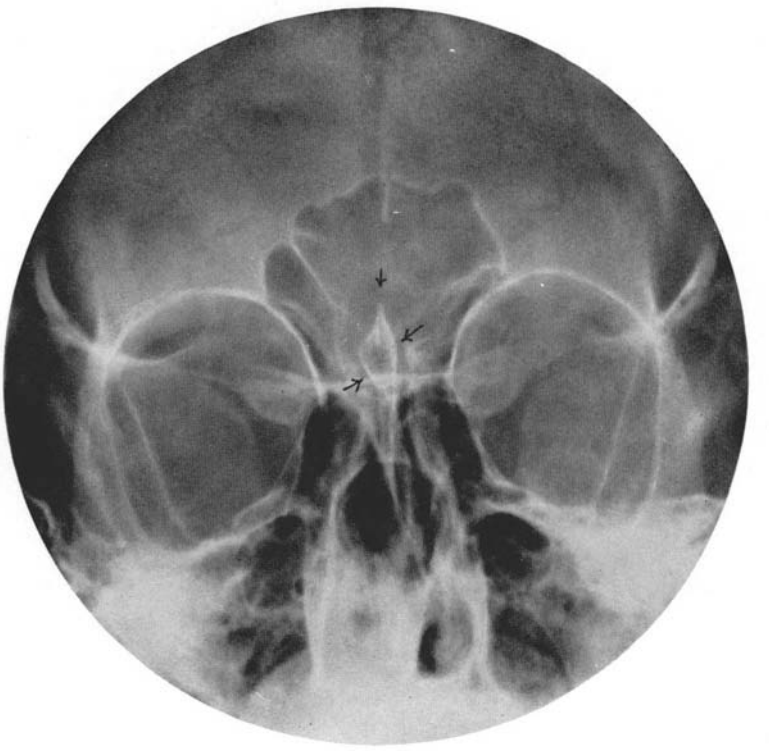
Nach dem Studium von 141 Fällen von atrophischer Rhinitis kommt der Verfasser zur Schlussfolgerung, dass mindestens 78% der Fälle vor der Pubertät beginnen. Die Krankheit setzt mit einer Entzündung der Nasenschleimhaut ein und in mindestens 60% der Fälle schliesst sich eine Nebenhöhlenentzündung an. Er glaubt, dass Vitaminmangel eine wichtige Rolle spielt, der die Widerstandskraft gegen Infektionen herabsetzt und die endokrinen Funktionen vermindert. Er beschreibt im Einzelnen die histologischen Veränderungen, die in den erkränkten Geweben beobachtet werden.

. . . .

L'auteur conclut de l'étude de 141 cas de rhinite atrophique, qu'au moins 78% des cas débutent avant la puberté que l'affection commence comme une inflammation de la muqueuse nasale et que dans au moins 60% de cas il y a association de sinusite. Il pense que la carence en vitamines joue un rôle important en raison du fait qu'elle affaiblit la défense de l'organisme contre l'infection et diminue les fonctions endocriniennes. Il décrit les altérations histologiques que cette maladie produit dans les tissus.

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CASE II.
X-ray showing a Crista Galli cell not infected.

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