

Review Article

The aetiology and management of atrophic rhinitis

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Abstract

Atrophic rhinitis is a chronic, debilitating and recalcitrant disease of the nasal cavities that is prevalent in several parts of the world. It has unique epidemiological features and clinical characteristics. Clinicians and researchers for decades have tried to postulate theories for the aetiology of the primary form of the disease. Management of the disease has seen several medical therapeutic regimens including alternative forms of medicine. Surgical options for the condition are also not completely satisfactory with a number of failures and recurrences. The authors provide here a comprehensive review of the existing literature as regards the aetiology and management of this refractory condition.

Key words: Rhinitis, Atrophic; Aetiology of; Management of; Ozaena

Introduction

Atrophic rhinitis (AR) is a debilitating chronic nasal mucosal disease of unknown aetiology. The condition is characterized by progressive nasal mucosal atrophy, progressive atrophy of the underlying bone of the turbinates, abnormal widening/patency of the (roomy) nasal cavities (with paradoxical nasal congestion) and formation of viscid secretions and dried crusts leading to a characteristic fetor (ozaena).^{1,2} AR is sometimes referred to as coryza foetida, atrophic catarrh, rhinitis atrophicans, acute necrotizing rhinitis or rhinitis chronica foetida.^{3,4} The primary form of the disease is also known as 'ozaena' (a stench) because of the characteristic foul smell emanating from the nasal passages.⁵ 'Subacute' and 'acute' forms of atrophic rhinitis and 'simple' and 'ozenous' atrophic rhinitis have also been described.⁶

Epidemiology

Primary atrophic rhinitis has decreased markedly in incidence in the last century and this is probably related to the increased use of antibiotics for chronic nasal infections.⁷ The reported prevalence of primary AR ranges from 0.3 to 1 per cent of the population in those countries with a high prevalence. The disease is extremely common in swines and cattle and is described as 'progressive atrophic rhinitis'. It has been studied extensively in these animals and several

treatment forms including vaccines have been evaluated.^{8,9} The porcine model has been used for evaluating the pathophysiology of this disease.¹⁰

The condition is unlikely to occur before puberty. It is prevalent predominantly in young and middle-aged adults. Several authors have reported a predominance in females (M:F = 1:5.6).¹¹ It is a common condition in tropical countries such as India, Pakistan, China, Philippines, Malaysia, Saudi Arabia, Egypt, Central Africa, Eastern Europe (Poland), Greece, Mediterranean areas and Latin and South America.^{1,12} A literature review shows that the majority of publications on AR are from India, China, Poland and other regions where the condition is common. Primary AR seems to have a high prevalence in the arid regions bordering the great deserts of Saudi Arabia.¹³ It has been reported that the condition is common in Asians, Hispanics and African Americans. The incidence of AR is low in the natives of equatorial Africa.²

In one study, the significance of environmental factors was reinforced by the findings that 69.6 per cent of the patients were from rural areas and 43.5 per cent were industrial workers.¹¹ That the contraction and development of the disease is by a combination of inheritance and environment has been lucidly described in another study.¹⁴ The disease appears to be more common in the lower social classes, poor populations and those living in poor hygienic conditions.¹⁵

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Accepted for publication: 29 April 2005.

Aetiology

Primary atrophic rhinitis

The aetiology of primary AR is unknown. The diagnosis is essentially clinical and one of exclusion of other conditions that may produce atrophic features secondarily.

Hereditary factors. The disease may be polygenetic and hence heritable. Primary AR has been reported in families where females are affected. One interesting study showed that 27.4 per cent of cases displayed an inheritance pattern of which an autosomal dominant pattern was seen in 67 per cent and a recessive trait in the rest.¹⁶ In another study, 20 per cent of cases had more than one member of the family suffering the same disease.¹⁷ A positive family history may be obtained in about 15–30 per cent of the cases. Congenital inhibition of the development of the nasal mucosa and turbinates has also been proposed.

Infections and infectious agents. Chronic bacterial infection of the nose or the sinuses is implicated as a cause of primary AR.^{1,18–20} In one study in Thai patients, 58.7 per cent had evidence of sinusitis, and the causative organisms in nasal swab cultures were *Klebsiella* species, especially *K. ozaenae*.¹¹ Other organisms that have been implicated include: *Coccobacillus foetidus ozaena* (*Coccobacillus* of Perez), *Coccobacillus* of Loewenberg²¹, *Bacillus mucosus* (Abel's bacillus)²², diphtheroids, *Bacillus pertussis*, *Haemophilus influenzae*, *Pseudomonas aeruginosa* and *Proteus species*.¹⁵ There is however little evidence to suggest these organisms cause the disease; they may be secondary invaders. *Bordetella bronchiseptica* and *Pasteurella multocida* are implicated in AR in pigs.²³ It is possible that superinfection with a mixed flora of these organisms causes ciliostasis with resultant destruction of epithelium and progressive mucosal changes. Cilioinhibition by *K. ozaenae* has been studied as a mechanism in the pathogenesis of AR.²⁴

Developmental disorders. Structural changes during development of the nasal and faciomaxillary regions perhaps have a role to play in the development of AR. Poor pneumatization of the maxillary sinuses and congenitally spacious nasal cavities, excessively patent nasal cavities in relation to the shape and type of skull and platyrrhinia are associations that are mentioned.^{1,15,25}

Nutritional deficiency. Poor nutrition is considered to be an important factor in the development of AR. Some authors consider this to be an iron deficiency disease.²⁶ Fat-soluble vitamin deficiency (especially vitamin A) is also believed to be a factor.¹⁵ An interesting study from Poland attempts to find out why ozaena is almost absent in the well-developed regions and why it commonly occurs in the developing and underdeveloped countries where the everyday diet is poor in iron, proteins and vitamins.²⁷

Phospholipid deficiency. The possible role of surfactant deficiency in AR has been studied. Biochemical analysis of the nasal aspirate in cases of primary atrophic rhinitis revealed a significant decrease in the total phospholipids compared to normal cases.²⁸

Autonomic disorders. Excessive vasoconstriction from autonomic imbalances as a reason for development of AR has been described.⁵ There is also a hypothesis that vasomotor rhinitis and primary atrophic rhinitis are diseases at the two ends of an autonomic spectrum and, further, that the dimensions and sensory receptors of the anterior nasal aperture play a vital role in the aetiopathogenesis of both these conditions through reflex autonomic action.²⁹ AR is also compared to a transient regional osteoporosis with related neurogenic pathogenesis.^{30–32} An initial stage of vasodilatation and hyperaemic decalcification of the turbinates is followed by collapse of the bones as seen in reflex sympathetic dystrophic syndrome.^{31,32} The autonomic theory is also supported by the effectiveness of attempts to overcome the vasoconstriction in AR by stellate ganglion blocks and cervical sympathectomy.

Endocrine imbalances. Oestrogen deficiency is implicated as a causative factor by some authors.^{1,33} The incidence of the disease in pubertal girls, the aggravation of symptoms during menstruation and pregnancy, and the improvement of symptoms in some cases with oestrogen therapy support this theory.

Allergy and immune disorders. Type I allergy was evident in 85 per cent of Thai patients with AR in a recent study.¹¹ Another study evaluated cellular immunity in patients with AR using the leucocyte migration and spontaneous rosette test *in vitro*;³⁴ this confirmed altered cellular reactivity or loss of tolerance to nasal tissues. Various factors such as viral infections, malnutrition and immunodeficiency may trigger a destructive autoimmune process with the release of antigens of nasal mucosa into the circulation. A study of immunoglobulins revealed a rise in serum IgM without a rise in IgG and IgA levels in AR.³⁵ In a recent study on immunology in a family affected by ozaena, positivity for IgG class anticardiolipins was correlated with disease manifestation.³⁶ The disease certainly develops and is transmitted by a multitude of factors and multigenic modes.¹⁴

Secondary atrophic rhinitis

AR may be secondary to a number of conditions. *Extensive accidental maxillofacial and nasal trauma* can cause extensive damage to nasal and sinus mucosa with resultant crusting and atrophy. *Extensive nasal surgery* such as submucosa resection of the septum, turbinate surgeries and sinonasal surgeries may also result in mucosal denudation, crusting and progressive atrophy of underlying tissues. The malady 'empty nose syndrome' associated with extensive turbinate surgeries has been discussed exhaustively.³⁷

Turbinate surgeries have been both accused and acquitted in the literature as aetiology for secondary atrophic rhinitis.⁷ *Recurrent acute and chronic suppurative infections* of the nose and paranasal sinuses may result in AR. It is believed that with longstanding suppurative sinusitis in childhood, there is proliferation of connective tissue cells that contract, leading to reduced nourishment to the nasal mucus membrane, and hence shrinkage and atrophy. Viral exanthems during childhood may also result in AR. *Chronic granulomatous disorders* of the nose such as *tuberculosis, lupus vulgaris, syphilis, leprosy, rhinoscleroma (atrophic stage), yaws and pinta* have been strongly associated with secondary AR.^{38,39} Some cases of AR have been described in *AIDS patients in Zambia*.⁴⁰ *Radiation exposure* of nasal and sinus cavities may result in excessive nasal crusting and atrophy of the tissues. Radiotherapy for nasopharyngeal cancer may result in atrophic rhinitis that may be made worse with the use of chemotherapeutic agents and decongestants.⁴¹ *Unilateral AR* has been observed in patients with gross deviation of the nasal septum.⁴² *Anhidrotic ectodermal dysplasia (Christ-Siemens-Touraine syndrome)* is a rare genetic disorder of the ectoderm that may rarely present with atrophic rhinitis. This may be a cause of congenital AR.^{43,44} *Osteochondroplastic tracheobronchopathy* is a rare disease with accumulation of bony and cartilaginous nodules in the tracheal and bronchial mucosa and may be sometimes associated with ozaena.⁴⁵ *Ichthyosis vulgaris* is another autosomal dominant condition that may be associated with AR.⁴⁶ AR has been reported as an unusual complication of *typhoid fever*.⁴⁷ *Occupational exposure to phosphorite and apatite dusts* is associated with AR.⁴⁸

Pathology

Atrophic rhinitis is characterized by '*atrophic changes of all parts of the nose*'. The normal respiratory epithelium changes to cuboidal or stratified squamous epithelium (metaplasia). Partial to severe metaplasia may be found with or without keratinization. There is atrophy of the cilia and the mucosal and submucosal glands. The mucosa becomes pale with thick, viscid, scanty secretions, drying up to form greenish or grayish yellow crusts and scabs. Secondary bacterial infections produce a foetid odour. Characteristic changes of squamous metaplasia in Thai patients with AR have been described.¹¹ The lamina propria and submucosa may show chronic cellular infiltration, granulations and fibrosis.

A rarefying osteitis of the inferior turbinates and the ethmoid turbinates is observed with resultant atrophy. Taylor and Young⁴⁹ showed a positive alkaline phosphatase reaction in the endothelial cells lining the capillaries that suggested active bone resorption. The nasal cavities thus become extremely roomy. Two characteristic types of vascular involvement have been documented by these authors.⁴⁹

AR Type I

In this common type (50–80 per cent of all cases), there is endarteritis obliterans, periarteritis and periarterial fibrosis of the terminal arterioles as a result of chronic infections with round cell and plasma cell infiltration. Logically, these are the patients that benefit from the vasodilator effects of oestrogen therapy.

AR Type II

Believed to be less common (20–50 per cent of all cases), there is capillary vasodilatation in this variety. The endothelial cells of dilated capillaries have more cytoplasm than normal and show a positive alkaline phosphatase reaction suggesting active bone resorption, which is a feature of the disease. This variety is not amenable to oestrogen therapy. However, in one study, 80 per cent of cases were compatible with the Type II histopathological classification with capillary vasodilatation.¹¹

Immunology

The leucocyte migration test and spontaneous rosette test were performed *in vitro* to assess the cellular immunity status of patients with AR.³⁴ With the leucocyte migration test, the mean migration percentage in patients with AR was lower than in the control group indicating recognition of the antigen by circulating leucocytes. In the spontaneous rosette test, patients with AR did not form or formed poorly a rosette compared to the control group. These features suggest poor cellular immunity and reactivity in AR. Also, a study of immunoglobulins in AR revealed a rise in serum IgM without a corresponding rise in the levels of IgG and IgA.³⁵

Clinical features

Primary AR is nearly six times more common in females and is usually bilateral. The following symptoms are observed in patients with primary AR.^{2,15} The nose emits a foul smell, due to crusting and secondary infections, which is the main characteristic of the disease. It is this factor alone that leads to social problems for the patient by keeping relatives and friends away. It is famously said of an AR patient:² "The patient is often only made aware of the loathsome effluvium surrounding her by the reluctance of others to come within her vicinity."

The patient herself is commonly anosmic due to the following reasons: the atrophic process involves the olfactory epithelium and the bipolar nerve cells and nerve fibres; there is an insufficient and non-uniform air blast that may not reach the olfactory areas; and there may be a genuine obstruction with large crusts blocking the air blast to the olfactory area in the roof of the nose. Some patients suffer from cacosmia when they smell a foul odour even with pleasant ones.

Nasal obstruction is often the chief complaint and is brought on by a number of factors such as: inability of the patient to perceive the passage of air in the

nose due to anaesthesia of the nasal mucosa ('blunting effect'); large crusts causing obstruction to air flow and disruption of the normal laminar and eddy current pattern of the inspiratory and expiratory air flows. Patients may also complain of headache. Thick purulent discharge with a foul smell due to anaerobic bacteria is common. Periodically, dark greenish crusts are brought out through the nostrils and are foul smelling. Rarely, crusts may dislodge into the pharynx to cause foreign-body sensation and choking. Minor bleeds from the nose may be observed with dislodgement of crusts. Occasionally, patients may complain of dryness in the nose and throat (pharyngitis sicca).

The signs of primary AR are as follows:^{2,15} The patient is usually gloomy and there is presence of foetor sometimes detected from a distance. There is a wide capacity of the nasal passages. Greenish yellow and black crusts of various sizes may be noticed lining the nasal cavities. Bleeding and ulcerated mucosa is seen when crusts are separated. The turbinates, especially the inferior and middle turbinates, may be shriveled significantly. The nasal mucous membrane appears lusterless because of absence of mucous glands. A probe test may reveal insensitivity of the nasal mucosa. A spatula test will reveal widely patent nasal airways. Clinical features of sequelae such as sinusitis, atrophic pharyngitis and laryngitis and otitis media, and features of complications such as septal perforation, saddle nose deformity and myiasis (maggots) may be seen. In cases of secondary AR, features of the primary disease such as tuberculosis, leprosy, and so on may be observed.

Sequelae and complications

Nasal septal perforation and saddle nose deformity. Severe cases left untreated may be complicated by destruction of nasal bone and cartilage. This may eventually lead to septal perforations and saddle nose deformities. Correction of saddle nose deformity and septal perforation in cases of long-standing atrophic rhinitis are formidable tasks.⁵⁰ The thick and puckered skin secondary to longstanding disease makes the creation of a dorsal subdermal pocket difficult. These patients do not tolerate synthetic implants well and also show an unusually high rate of absorption of autologous bone grafts.

Secondary rhinosinusitis. It is difficult to tell whether this is the cause or an effect of AR. There is no doubt however that rhinosinusitis is a co-morbidity in AR patients.

Local and systemic spread of infection. Spread of infection to the pharynx, larynx, lungs and ears, and intracranial spread in immunocompromised patients is possible. A case of pyogenic liver abscess and septic pulmonary emboli associated with *Klebsiella ozaenae* bacteraemia has been reported.⁵¹ A case of cerebral abscess caused by *K. ozaenae* has also been reported.⁵²

Atrophic pharyngitis and laryngitis. Pharyngitis sicca is a frequent co-morbidity in AR with a dry pharyngeal mucosa. Dislodged crusts may cause choking episodes.^{2,3}

Chronic dacryocystitis. A rare complication of AR in the form of dacryocystitis has been noted where a successful endoscopic dacryocystorhinostomy was performed.⁵³

Nasal myiasis. This is an extremely distressing condition seen in neglected cases of primary AR, especially in patients of lower socioeconomic status living in poor hygienic conditions.² The putrefied nasal debris and foul smell attract flies of the genus *Chrysomya* (*C. bezianna vilteneauve*). These flies lay eggs which later hatch into larvae known as maggots. Tens to hundreds of maggots may infest the nasal cavities and eat away the mucosal soft tissues down to bone! They create tunnels in the soft tissues of the nose, sinuses, nasopharynx, pharyngeal walls, orbital tissues, lacrimal apparatus, facial tissues and skull base, and may even cause meningitis and death.⁵⁴ They may also cause bone destruction resulting in nasal dorsal deformities, facial deformities, and septal and palatal perforations.

Investigations and diagnosis

Diagnosis is by a high index of clinical suspicion: the triad of characteristic foetor, greenish crusts and roomy nasal cavities is diagnostic of the condition. General and systemic examination of a patient should include thorough evaluation of possible causes of AR such as tuberculosis, leprosy, scleroma and syphilis. All the classical features and so called 'stigmata' of the above conditions are looked for. Chronic sinus suppuration on its own, suppurating adenoidal disease in adolescents, and neglected foreign bodies/rhinoliths in unilateral cases have to be excluded before 'labeling' a patient as primary AR.

Investigations are considered only to rule out secondary causes of AR and other nasal granulomatous conditions. These include haematological studies, radiological assessments and biopsy.^{2,15} *Haemoglobin* estimation is necessary as some patients have anaemia. *A total and differential count* of the leucocytes along with a *peripheral blood smear* study is essential; it may show leucocytosis with infections and a microcytic hypochromic picture with iron deficiency anaemia. Raised *erythrocyte sedimentation rate* is found especially with granulomatous infections such as tuberculosis. *Blood sugars*, both fasting and two-hour post-prandial levels, are assessed for diabetes mellitus. *Total iron binding capacity and total serum iron levels* may have to be estimated if the peripheral smear shows a microcytic hypochromic picture. Patients with malnutrition may require estimation of *serum proteins and plasma vitamin levels*. An immunological study including *autoimmune assays* may be necessary. *Leucocyte migration and spontaneous rosette tests* are of academic value. *Nasal swab* for stains, culture and antibiotic sensitivity may be useful. X-rays, *Water's and*

Caldwell's views, of the paranasal sinuses are sometimes considered especially prior to a proof puncture, but routine radiography is of limited value and has been replaced largely by limited high-resolution computerized tomographic (CT) scanning. *An antral proof puncture* may be necessary to treat a co-existent acute or subacute sinusitis. Lavage is both diagnostic (for Gram stain, culture and antibiotic sensitivity) and therapeutic. *VDRL* and other *serological tests for syphilis* will have to be requested if there is a strong suspicion of syphilis. A *chest X-ray* and *Mantoux test* may be ordered if secondary AR due to tuberculosis is being considered. Hansen's disease (leprosy) may be investigated by *ear lobe puncture/smear* and nasal biopsy (bacteriological and morphological indices). *Nasal biopsy* may be performed for Young and Taylor classification and for secondary AR related to nasal granulomas such as leprosy, lupus, syphilitic gumma and scleroma. If a surgeon is embarking on sinonasal surgery for disease clearance, complete *CT scanning* (high resolution with bone windows) of the nose and sinuses is essential. In one study, 60 per cent of the patients showed thick bony walls and a small cavity of the maxillary sinus that were confirmed on antroscopy.²⁵ Twenty-five per cent of the patients in the same study showed signs of infection including mucopurulent secretion in the maxillary sinuses.

Conservative treatment

The mainstay of treatment for AR is conservative. Medications and therapy may be locally or systemically administered.^{2,15}

Nasal irrigation and douches. An ideal alkaline nasal douche mixture consists of 28.4 g of sodium bicarbonate (helps in dissolution of crusts), 28.4 g of sodium diborate (acts as an antiseptic, is also bactericidal as an acid, and helps to buffer the bicarbonate in the mixture) and 56.7 g of sodium chloride (makes the solution isotonic). One teaspoonful of the above mixture (pulverized triborisis) in about half a pint (280 ml) of luke-warm water is used to douche the nasal cavities vigorously to clear off the crusts. This can be done three or four times a day using a 10 or 20 cc syringe, nasal catheters, douche rubber bulbs, douche cans or siphon bags, asepto syringes, Birmingham glass syringes, fountain syringes, Higginson syringes or enema cans, or even by snuffling up the solution through the nostrils. It is useful to instruct the patient to bend forward during the procedure and keep saying 'K, K, K ...' so that the nasopharyngeal isthmus is relatively closed and there is little risk of aspiration. What comes into the pharynx may be brought out through the mouth.

Several commercial brands of nasal irrigation systems are available today incorporating sea-salt solutions that can be used as nasal sprays followed by douching. An interesting study from California discusses the benefits of nasal irrigation in sinonasal disease. Use of pulsatile hypertonic saline nasal irrigation for the treatment of sinonasal disease including atrophic rhinitis for three to six weeks

resulted in statistically significant improvements in nasal symptoms.⁵⁵ There may be a need for nasal toilet periodically with the use of nasal endoscopes for removal of crusts.

Glucose-glycerine nose drops. Twenty-five per cent glucose is used to inhibit saprophytic infection and proteolytic bacteria (glucose on fermentation produces lactic acid and an acidic pH that inhibits bacterial growth), and promote the growth of commensal flora. Glycerine helps as a lubricant and hygroscopic agent (adsorbs water from the atmosphere and moistens mucosa, and hence impedes crust formation). Glycerine may also cause some degree of irritation and hence improve vascularity.¹⁵ These nose drops should be applied three or four times a day after douching the nose.

Liquid paraffin nose drops. Although liquid paraffin is effective in lubricating the nasal mucosa and in removal of crusts, long-term use is not recommended in view of reports of paraffin granulomas and inhalational lipid pneumonias.

Oestradiol in arachis oil. This combination is available for instillation into the nose as drops and sprays (10 000 units/ml).² It must be remembered that oestrogens are only useful for Young and Taylor Type I AR; oestradiol may worsen the situation in the Type II variety. It is advisable to obtain a histological typing before considering such an option of treatment.

Kemicetene antiozaena solution. This again is a popular solution containing 90 mg of chloramphenicol, 0.64 mg of oestradiol dipropionate, 900 IU of vitamin D2 and propylene glycol in each millilitre. This is used in the form of nose drops after douching.²

Chloramphenicol/streptomycin drops. These medications are also available for use after douching. Local treatment with injection of a mixture of streptomycin and novocaine has been tried with satisfactory results.⁵⁶

Placental extract injections. Filletto's biogenic theory supports use of submucosal injections of placental extracts to produce vasodilatation. Local nasal injections may also produce a mass effect in reducing the size of the roomy cavities. The benefits of this option are attributed to the biogenic, angiogenic and mitogenic stimulatory properties of the placental extracts. The extract (0.5 ml) is injected into each nasal cavity per week for 24 weeks. However, recurrence of symptoms has been noted within eight weeks of cessation of therapy. Intranasal injections used for two years have produced 93.3 per cent relief of symptoms, while systemic human placental extracts have also been used with 80 per cent improvement in two years.^{57,58}

Acetylcholine with or without pilocarpine. This has also been used topically or hypodermally to produce vasodilatation and improve the secretomotor activity of the mucous glands.

Antibiotics and antimicrobials. Keeping in mind that the causative agent is *Klebsiella* species some authors have recommended the use of systemic (intravenous) aminoglycoside (Tobramycin) therapy for two weeks in addition to topical gentamicin.⁵⁹ Good results have been reported in one study where Rifampicin 600 mg once daily for 12 weeks was administered.⁶⁰ More recently, ciprofloxacin in a daily dose of 500–750 mg bid for one to three months has been tried successfully, i.e. measured by the disappearance of crusts, odour and *K. ozaenae*.⁴

Iron, zinc, protein and vitamin (A and D) supplements. These have been recommended especially in cases of malnutrition and established deficiencies. The use of potassium iodide by mouth with the object of increasing nasal secretion has been recommended.²

Vasodilators. Xanthinol nicotinate, cyclandelate, dipyridamol and nicotinic acid, for example, may be tried systemically to improve the vascularity of the nasal tissues. However, their vasodilatory properties are not selective and local. Nicotinic acid and its derivatives have been used both systemically and topically by several authors in the past.^{6,61,62}

Prostheses. Non-surgical closure of the nasal vestibule using prostheses has been described, including occlusion of the nostril with an obturator made from dimethylpolysiloxane.¹² This is useful in cases of secondary AR where formal closure of the nostril is contraindicated in view of the treatment necessary for the primary disease. Another similar device made of clear acrylic resin called a 'pin-hole nasal prosthesis' has been described more recently.⁶³

Vaccines. Vaccines have been evaluated in pigs with progressive AR using various challenge models including *Bordetella bronchiseptica* and *Pasteurella multocida* infection. The results need to be further studied and evaluated for human therapeutic applications.⁹ Autogenous vaccines to *Perez bacillus* have also been tried.

Decongestants or antihistamines. It must be remembered that the use of these is **strongly contraindicated** in AR as they worsen the pathology and hence the clinical course of the disease.

Surgical treatment

The principles of surgery may be divided largely into four groups. There may be some surgical procedures that achieve more than one therapeutic goal among the ones listed below. There may be an overlap of some of the principles as well.

- (1) *Decreasing the size of the nasal cavities.* This helps in reducing the turbulence of air currents in the roomy cavities and hence prevents drying of the mucosa and crusting.

- (2) *Promoting regeneration of normal nasal mucosa.* This may be achieved by allowing the nasal cavities to rest by temporary closure (complete or partial) of the nostrils and hence reduction of air turbulence or blast into the roomy cavities. This promotes the regeneration of the nose's own normal anatomy, histology and physiology in many ways.
- (3) *Increasing lubrication of the dry nasal mucosa.* This is achieved by increasing the secretory abilities of the nasal cavities or by introducing secretions from elsewhere.
- (4) *Improving vascularity of the nasal cavities.* This is achieved either by blocking the sympathetic nervous system (stellate ganglion block or cervical sympathectomy) subserving the nose or by introducing grafts (e.g. placenta, maxillary mucosal flaps, buccal flaps) that improve vascularity.

Decreasing the size of the nasal cavities

Lautenschlager was the first surgeon to embark on a definitive surgical procedure for AR. He reduced the size of the roomy cavities to bring in some normalcy to the nasal air flow by medializing the lateral nasal wall or the medial wall of the maxillary antrum using a Caldwell-Luc type of approach.⁶⁴ Reducing the size of the roomy cavities, sometimes referred to as 'recalibration' of the nasal fossae, has seen several modifications and led to the use of various substances and implants that are endonasally or sublabially inserted into the floor, septum or lateral walls of the nose.⁶⁵ The list includes autologous bone (cortical or cancellous chips), cartilage, fat, muscle; homologous lyophilized bone, cartilage, fat, placenta; synthetics such as glass beads, acrylics, plastic gauzes, Teflon, Proplast, Dacron, silicone or silastic grafts, glycerine, paraffin or dental pastes, and so on.

Good results from the submucosal injections of a suspension of powdered Teflon in 50 per cent glycerin paste have been reported.⁶⁶ An autologous medullary (cancellous) bone graft as a single long piece of bone has been tried.⁶⁷ Autologous costal cartilages have been used for reducing the nasal cavity size with good outcomes.⁶⁸ An osteoperiosteal flap from the anterior wall of the maxilla has been described.⁶⁹ Hydroxylapatite implants have been placed under the mucosa of the septum and floor with favourable long-term results.⁷⁰ A method of submucous implantation using pedicled auto-flap of cheek muscle and maxillary periosteum of the piriform aperture has been described in 32 patients with AR, with stable long-term outcomes.⁷¹ Triosite implants with fibrin glue have been used to reduce the size of the nasal fossae via a labial vestibule route. No rejection occurred in the nine patients that had the implants and the osseocoalescence, as evaluated by CT at six months, was good.⁷² Good results have been reported by inserting plastipore plates (high-density polyethylene sponge with micropores to enable tissue ingrowths) into submucosal pockets in the floor and septum, and thus reducing the volume of the nasal fossae.⁷³

Promoting regeneration of normal nasal mucosa

Young's operation.^{74,75} This procedure essentially closes one nostril and hence the nasal cavity completely under local or general anaesthesia. The surgery involves raising skin folds in the vestibule by circumferential incisions and suturing them together. The advantages are that the blast and turbulence of air through a roomy cavity is removed giving rest to the nasal mucosa, a medium with the CO₂ and pH conducive for regeneration of respiratory epithelium is created, and finally a negative post-choanal pressure that is produced with the closure of the nostril may cause a certain degree of vasodilatation within the cavity. It has been shown that partial surgical closure of the nostril in AR has a beneficial effect on the nasal mucosa and reverses the basic pathologic alterations in its microanatomy.^{76,77} The disadvantages of Young's operation include difficulty in raising the skin flap, suture-line breakdown, excessive scarring, and hence vestibular stenosis. Some patients may experience snoring and sleep apnoea. The following are modifications to Young's operation.

*Sinha modification.*⁵⁸ Because there is complete closure of the nostril, Young's procedure cannot be done bilaterally. Sinha *et al.* modified the technique leaving a small 3mm-diameter hole within the skin membrane. Any further increase in size of the hole decreased the success rate rapidly. As such, this procedure may be carried out on both nostrils simultaneously.

*Gadre modification.*⁷⁸ In this technique, instead of reflecting the skin of the vestibule forward as suggested by Young, mucosal flaps are developed backwards from the mucocutaneous junction and sutured in the midline. The end result is a three-layered membrane with an outer epithelial layer continuous with the skin, an inner mucosal layer continuous with the nasal mucosa, and a middle layer of fibrous tissue.

Ghosh's vestibuloplasty. Based on the hypothesis that primary AR belongs to a group of conditions under the heading 'reflex sympathetic dystrophic syndrome', the author has developed an interesting surgical technique termed 'vestibuloplasty'. A flap is raised from the lateral wall of the nasal vestibule antero-posteriorly and is sutured on itself, decreasing the lateral flow of air into the nasal cavity. Thus, the technique directs the stream of air away from the turbinates and dissipates the force of the air currents.^{31,32}

*El Kholy modification.*⁷⁹ Recently, El Kholy *et al.* have described an interesting septal mucoperichondrial flap for closure of the nostril in AR. The technique overcomes the difficulties and shortcomings of the classical Young's operation and some of its modifications.

*Reversal of Young's operation and its modifications.*⁸⁰ It is useful to visualize periodically with the nasal

endoscope (Hopkins rod-lens telescope or fibreoptic scope) the nasal cavities of patients who have undergone Young and modified Young operations. Diagnostic endoscopy would demonstrate reduction of crusts following surgery, reappearance of free mucus suggesting respiratory mucosal regrowth and hence the need to plan for reversal of the procedure.¹³

Recanalization of a 'Young's obturator membrane' after months or a year is a daunting task. Reporting a five-year follow up of AR patients who had undergone this surgery, Young⁷⁴ wrote: "the aperture that is created (by excision of the membrane) tends to close and more than one operation may have to be done before the opening is satisfactory." Gadre *et al.*⁸⁰ have described a useful three-flap technique involving reopening of the obturator membrane followed by stenting with tubes for six weeks.

Increasing lubrication of the dry nasal mucosa

*Wittmaack's operation (1948).*¹⁵ This procedure increases nasal secretion and lubrication by implantation of a Stensen's (parotid) duct into the maxillary antrum.

*Raghav Sharan's operation.*⁸¹ A conventional Caldwell-Luc procedure is carried out to include an intranasal antrostomy through the inferior meatus. The mucosa of the maxillary antrum is elevated and brought into the nasal cavity on each side through the antrostomy. The benefits are perhaps three-fold: decreasing the size of the cavity, and improving lubrication and vascularity.

Improving vascularity of the nasal cavities

Stellate ganglion injections. With the sympathetic distribution (sympathetic innervation causes vasoconstriction in the nasal cavities) to the nasal cavities and the paranasal air sinuses coming from the cervical sympathetic chain, a stellate ganglion block is a logical means of improving the blood supply to these regions. The procedure is performed through an anterior paratracheal approach. Ten to 15 cc of 1 per cent xylocaine is injected slowly. The success of the procedure is judged by the appearance of Horner's syndrome, congestion of the ipsilateral tympanic membrane and congestion of the ipsilateral nasal mucosa. In cases of unilateral AR, daily blocks on the affected side are recommended. In bilateral cases, the two sides are treated on alternate days to avoid the risk of bilateral recurrent laryngeal nerve palsy. It is reported that foetor and crusting are relieved within eight to 10 blocks and that this is maintained for up to four to eight days after cessation of blocks. Anosmia however persists.⁸² The procedure carries a big risk of transient recurrent laryngeal nerve palsy, is in many ways a blind technique with large vessels in the vicinity of the sympathetic chain, and has no sustained effect. Patient compliance is another factor that is against such a daily cumbersome procedure.

Cervical sympathectomy. This is a classic surgical technique based on the same principle as stellate

ganglion blocks but with obvious permanence in its effect. However, it is a technique endorsed by very few for this condition.⁸²

Novocaine block of the pterygopalatine fossa and juxta-nasal sympathectomy. These procedures has also been tried for AR,^{83,84} but have not been duplicated by other authors.

Miscellaneous surgical procedures

Clearance of sinonasal infections. The role of endoscopic sinus surgery (ESS or FESS) in AR has been studied by few authors.^{85,86} The general conclusions are that ESS in combination with adequate post-operative antibiotic therapy can significantly improve AR. Also, candidate selection is critical for the success of AR treatment using ESS.

Middle turbinectomy. One study reported cure of AR and its features paradoxically by performing a middle turbinectomy. However, these results have not been duplicated by any other centre.⁸⁷

- **This paper reviews the aetiology, presentation and management of atrophic rhinitis**
- **Primary atrophic rhinitis, more common in arid regions, is decreasing in incidence**
- **Many types of treatment have been described. The rationales for these varying surgical and medical treatments are discussed**

Alternative forms of medicine

Chaulmoogra oil has been used in the past with variable results. Available as 'Nirdosh' in India, it was used orally at 250–500 mg twice daily for nine months after meals (to avoid gastric irritation). The oil has also been applied locally in the nose in conjunction with the oral administration. The active ingredients, chaulmoogric acid and hydnocarpic acid, have bacteriostatic and bactericidal actions specifically against acid-fast bacilli. Also contained is an endotoxin that may help develop immunity. The results are satisfactory for as long as the patients are on the medication, with recurrence of symptoms after stopping treatment.^{15,88} Powder of sea ka has been used in the past for ozaena with favourable results.⁸⁹ Some authors have tried vibratory massage of nasal turbinates using Mandl's paint or Balsam of Peru carried on cotton-tipped probes to encourage glandular activity. Authors from China have satisfactorily treated 23 cases of AR by deep puncture at three points (acupuncture) in the nasal region.⁹⁰ The authors have shown functional changes in the nasal mucosa, mucociliary transport rate, surface temperature of the conchal mucosa, acid-base scale of nasal secretion, and volume of nasal secretion after such treatment.

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Dr S N Dutt takes responsibility for the integrity of the content of the paper.

Competing interests: None declared
