

FACIAL AND THORACIC DEFORMITIES INCIDENT TO OBSTRUCTION BY ADENOID HYPERTROPHY IN THE NASO-PHARYNX.

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The disease which occasions the particular form of obstruction, prone to result in the series of deformities about to be described, is variously known as "adenoid hypertrophy in the naso-pharynx," "adenoid vegetations" and "hypertrophic naso-pharyngitis;" and in multiplicity of cases, gravity of consequences, facility of operative treatment, and the brilliancy of results, it may be said to outrank any other affection of the upper respiratory tract.

At the vault of the pharynx a number of muco-lymphoid follicles are grouped together forming a compound gland, analagous to the tonsils and known as the third tonsil, the pharyngeal tonsil or the tonsil of Luschka. In the normal state this is not of sufficient size to deserve such appellation, but when hypertrophied, as it frequently is, it bears some resemblance to the faucial tonsil in a state of enlargement.

In form, contour, and consistency the growth presents many gradations from the soft stalactitic "adenoid vegetations" up to the dense and more fibrous individuate variety, in which the mass is made up, in large part, of but a single neoplasm, of firmer consistence, smooth surface, and more or less irregular contour, according to size and degree of impaction. Fig. 1, accurately drawn

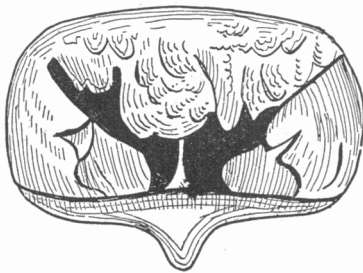


Figure 1.

from nature, is a typical representation of an average case in which the naso-pharynx is seen to be occupied by a fimbriated adenoid mass, which occludes, in large part, the posterior nasal choanæ, and so presses downward the upper lip of the tuber of the left Eustachian orifice as to practically close the channel to the middle ear.

Etiology.—The disease is frequent among children, not uncommon with adolescents, and not at all rare during early adult life. Children of inherited syphilitic or tuberculous diathesis, and

those perhaps otherwise the victims of scrofulosis, are predisposed to it. It is unnecessary to discuss the relationship between these diatheses. Whether as a result of inheritance of one or more of them, or of acquisition through bad hygienic influences, we recognize in the state called scrofulosis a debilitated constitutional condition, one of the characteristics of which is a pronounced tendency toward hyperplasia of the lymphatic and lymphoid structures. The reason therefore one can not state any more than one can formulate a full explanation of the normal cell proliferation. It concerns that of which we know naught—the ultimate essence of life itself. Climatic inequalities furnish adequate exciting causes. Repeated acute congestion from "cold" acting upon a structure already the subject of diathetic predisposition to hyperplasia, serves to establish a chronic hypertrophy of the group of muco-lymphoid glands in the naso-pharynx. This mass is then constantly swathed in a viscid muco-purulent, readily decomposable secretion, the product of its own elaboration, which, by exciting and maintaining irritation, further conduces to the development of the growth.

Symptoms.—The space of the naso-pharynx is, by nature's law, designed to be free, and to serve as a common area of air communication between the five openings which enter it.

The Eustachian tubes open into it, one on each lateral wall posterior to the nasal choanæ, and upon perfect patency of these openings, together with free nasal respiration, the power of hearing is dependent, for ventilation with normal air pressure in the cavity of the middle ear, is essential to correct auditory sense. Deafness, therefore, is frequently a deplorable symptom, and one which is liable to become permanent unless speedy relief be afforded.

Into this space open also the posterior nares, the natural respiratory passage being *via* the nose and naso-pharynx. Adenoid hypertrophy therefore serves as a plug to the posterior nasal openings, and obstructs nasal respiration completely or in part according to the degree of glandular enlargement. From this point we find it a matter of exceeding interest to trace the origin and development of each successive step in the series of deformities consequent upon this condition. The plugging up of the posterior nares necessitates oral breathing, and the constantly open mouth interferes with the normal adaptation of certain facial muscles, which in turn effects radical changes in the contour of the soft and developing bones of the face, the whole resulting in a physiognomy characterized by a vacant, stupid, almost idiotic expression of countenance, which can be better illustrated by photographs from nature than described. (Fig. 2.)

The hanging lower jaw causes the face to appear elongated. The nose is pinched or its alæ

distended, while the angles of the mouth and eyes have a drawn appearance.



Figure 2.

Moreover, as pointed out by Henri Chatellier,¹ of Paris, cited by Hooper,² of Boston, the air cavities in communication with the nose, as the frontal, maxillary, sphenoidal, and ethmoidal sinuses, which are essential to the proper expansion of their respective bones, cease to develop when the circulation of air through the nose is interfered with, thus altering nature's intent regarding the dimensions of the face and head, and still further deforming the physiognomy. Dr. Hooper has also described and illustrated the next link in the chain of deformities—the high-arched hard palate and V-shaped indenture. The naturally rounded arch of the roof of the mouth is formed, in large part, by the palate process of the superior maxillary bone, which also constitutes a corresponding portion of the floor of the nose. Augmentation of atmospheric pressure upon the buccal surface of the palate process and the impact of air currents to and fro during mouth-breathing, together with diminution of intra-nasal air pressure incident to nasal obstruction, gradually force upward the centre of the hard palate, and change thus the obtusely rounded Romanesque arch into one of Gothic shape—the pointed or high-arched palate invariably existing in association with long continued and excessive adenoid development during childhood. (Fig. 3.)

Elevation of the palatal arch lessens the transverse diameter of the jaw and causes it to grow pointed in front—the so-called V-shaped indenture—and with the resulting contraction of the alveolar process, the teeth, especially those near the point, are crowded into various grotesque ag-

gregations, or are rotated on their axes—a condition depicted in Fig. 5, drawn from a typical case, in which the two central incisors overlap, and the two lateral incisors undergo a quarter rotation and stand at right angles to the alveolar process.

Dentists have long recognized what they call the "habit" of mouth-breathing as a prolific source of irregular indentures, and S. C. G. Watkins, in a recent number of the *Ohio Journal of Dental Science*, says: "I know a child which at birth and up to the third year had a perfectly formed and normal arch; but it acquired the habit of breathing through the mouth, and the arch is becoming narrow—so much so that you cannot do more than place your finger in the centre of the arch. Now, at the age of 9 years, there has been a radical change in the shape of the mouth, and from no other reason than oral breathing."³

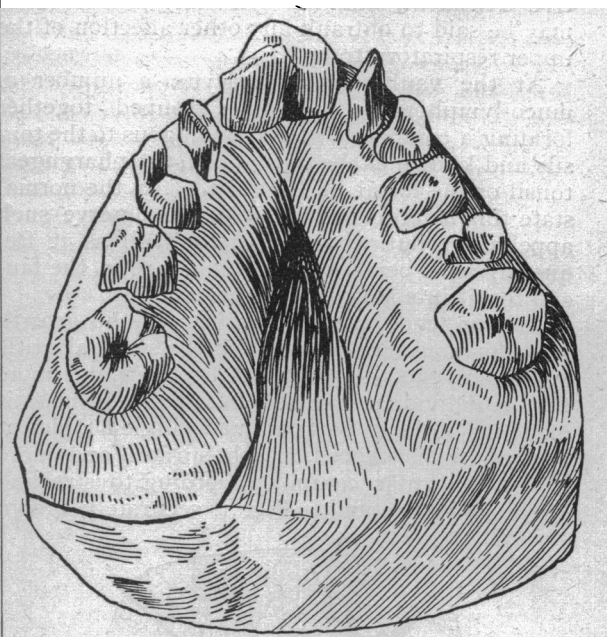


Figure 3.

We would advance a step and assert that mouth-breathing is but rarely a mere "habit." It conduces to so much discomfort that even the babe would feel inclined to close this aperture could he secure the requisite amount of air through the nose and naso-pharynx. Aside from greatly debilitated states, it is occlusion to some degree of these passages which necessitates the habit.

Next, elevation of the palatal arch must produce contortion within the nose. Deflections of the septum are rarely congenital; indeed, the only part of the septum which is ossified at this period is the vomer, the whole ethmoid bone, including the vertical plate, being in a soft cartilag-

¹ Des Tumeurs Adenoides du Pharynx, Paris, 1886.

² "The Mechanical Effects of Adenoid Vegetations in Children." Reprint from Medical and Surgical Report of the City Hospital of Boston, Fourth Series, 1889.

³ It is proper to state that other dental authorities deny this influence of oral breathing in the causation of deformed indenture, and attribute the latter solely to a perverted formation of the permanent set of teeth. The association, however, between adenoid vegetations and the high-arched palate is so constant that an etiological relationship must exist.

inous state. Traumatism, as shown by Robertson in the investigation of 217 cases, cited by Delavan,⁴ cannot fairly be made to account for much over half the number. Now, the septum, composed of the vomer, the perpendicular plate of the ethmoid bone and its cartilaginous portion, is unequal in power of resisting compression to the bones by which it is incased. Designed by nature to fill vertically the natural space between the roof of the nose and its floor, the abbreviation of this space by elevation of the palatal arch through the instrumentality of naso-pharyngeal adenoid hypertrophy cannot result otherwise than in forcing the septum to provide for itself by bending and curving laterally in various directions—a condition which is diagrammatically represented in Fig. 4, and which explains the etiology of the remaining moiety of cases of deviation of the septum narium. Furthermore, guided by the contorted septum, even the external nose may be twisted to one side—a deformity common enough in its milder aspects, and not unfrequently seen in aggravated forms. Within the nares also the septal deflection acts as an additional impediment to nasal respiration and drainage, and becomes a potent factor in the evolution of hypertrophic rhinitis, or that form of nasal catarrh characterized by enlargement of the turbinated bodies. (Fig. 5.)

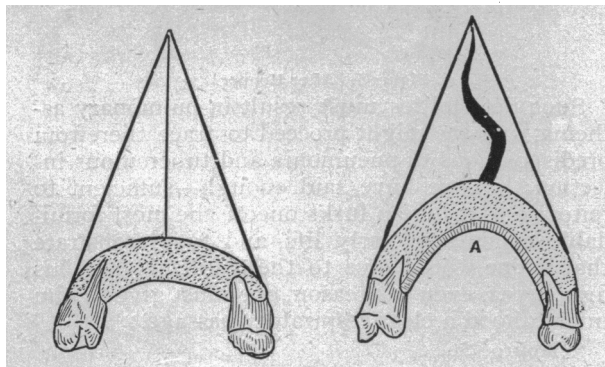


Figure 4.

Finally, not only, as before said, do these unfortunates *look* stupid, but they really *are* stupid and exhibit abundant evidence of mental hebetude, with inability to fix the attention to learn, to memorize, or to reason, the whole evidencing an impairment of cerebral function which Dr. Guye,⁵ of Amsterdam, has recently described under the name of *aproxexia nasales*. Indeed, we hold it not illogical to believe that in extreme cases of long duration, associated perhaps with deafness, such alteration of cerebral function might ensue as to result in absolute idiocy.

Three varieties of thoracic deformity are observed to accompany obstructive naso-pharyngeal adenoid hypertrophy, the association of one or

other form, in advanced cases, being so constant that a direct causal relationship, although difficult of absolute demonstration, can reasonably be assumed.

For the induction, however, of two of these forms, the "pigeon-breast" deformity and the "barrel-shaped chest," the intermediation of still another symptom—bronchitis, seems essential.

The influence of hypertrophic rhinitis and obstructive deflection of the septum narium in the causation of certain cases of bronchitis is now a matter of common observation. Plethora of the blood-vessels of the nasal mucous membrane tends to develop a like plethora in the bronchial mucous membrane, and anæmia induced in the turbinated tissues tends to effect an anæmic state of the bronchial tubes. The physiological relationship between the two regions—the nasal erectile tissues being designed to warm and moisten the inspired air—demands, through the vaso-motor system, an intimate correspondence between their blood supplies. As might therefore be expected, a pathological correspondence also obtains, and without entering into a discussion of the hypothetical details of nervous mechanisms, we simply state the oft-observed fact, that turgescence and vaso-motor paresis of the nasal erectile tissues may occasion vaso-dilation, congestion, and inflammation of the bronchial mucous membrane.

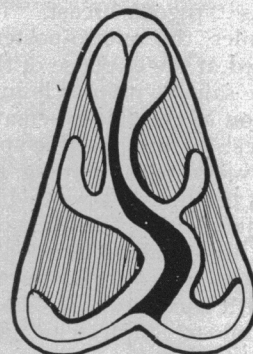


Figure 5.

Now, we have just seen how adenoid vegetations, persisting through early childhood, can occasion obstructive deflection of the septum with hypertrophic rhinitis, and, in many instances, chronic bronchitis is thereupon superinduced.

Furthermore, adenoid hypertrophy is, in itself, an etiological factor in the production of chronic bronchitis. Especially in neurasthenic individuals, it is exquisitely sensitive to reflex producing impressions, and its irritation may result, reflexly, in spasm of the glottis, cough, asthma, and parietic vaso-motor bronchitis.

Miss A., for instance, was affected by large adenoid vegetations, and suffered also with profuse bronchitis and from alarming, rapidly recurrent attacks of spasm of the glottis. On two occasions we were able to excite the glottic spasm

⁴ Reference Handbook of the Medical Sciences, Vol. v, p. 213.

⁵ "On Aproxexia," by Dr. Guye, Professor of Otolaryngology in the University of Amsterdam. Journal of Laryngology and Rhinology, December, 1889.

in our office by simply titillating the adenoid mass by a probe. They culminated with extreme suddenness and endured for a couple of minutes, the second paroxysm being so severe as to suggest hasty preparations for tracheotomy and to cause us to desist from further experimentation in this direction. Surgical removal of the adenoid growth, somewhat incomplete for want of time, resulted in a disappearance of the bronchitic symptoms, and in such amelioration of the glottic spasms that they ceased to be a serious inconvenience.

Again, for example, in the case of a young boy who suffered from chronic bronchitis with frequent asthmatic paroxysms, the removal of an associated adenoid mass from the naso-pharynx resulted in an immediate disappearance of all symptoms for a period of five months, but with subsequent rarer and milder recurrences. Indeed, in all cases of chronic bronchitis with children, adenoid hypertrophy should be sought for, as the upper and lower respiratory tracts constitute one continuous surface, having intimate nervous and vaso-motor connections, and the removal of adenoid vegetations, if present, will eliminate at least one, and possibly the chief, source of irritation.

With the continuance of bronchitis during the early developing years, the irregular convulsive action of the respiratory muscles incident to cough and dyspnoea is productive of the thoracic deformity known as "pigeon-breast," in which the sternum protrudes, the antero-posterior diameter being lengthened at the expense of the transverse diameter. The same symptom is influential also in the production of the emphysematous or "barrel-shaped" chest, as the coughing spells and powerful expiratory efforts, by forcing the air to the superior part of the lungs, serve to distend the antero-posterior diameter of the chest at this point.

The culmination, under like conditions, of one or other of these two deformities, is seemingly determined, with some by age—young subjects being little prone to emphysema and the deformity tending toward "pigeon-breast;" and with others, by the existence primarily of any degenerative change in the alveolar walls which might predispose to emphysema and in consequence to the "barrel-shaped" deformity.

The third variety of thoracic deformity—the "flat chest," is due directly to obstruction by the adenoid growth itself, and is an indrawing of the chest walls, especially a shortening of the antero-posterior diameter which results from an insufficient air supply to the lungs. The impairment of the normal respiratory relations by exclusively oral breathing, is such that complete expansion of the chest does not occur and a sufficient amount of air cannot be obtained, a fact which any one can test for himself by comparing the ease of deep inspiration through the nose with the laboriousness and incompleteness of similar efforts continued through the mouth alone.

The chest in these cases becomes flat and thin (Fig. 6, after Hooper), has a sunken appearance over the lower part of the sternum, perhaps a deep concavity at the ensiform cartilage with depressed intercostal spaces—a permanent, mild degree of the retraction observed during the dyspnoea of membranous croup, and due, as Löwenberg,⁶ cited by Hooper,⁷ has explained, to excess of atmospheric pressure on the outside of the chest, together with the labored action of the diaphragm and intercostal muscles in efforts at inspiration.

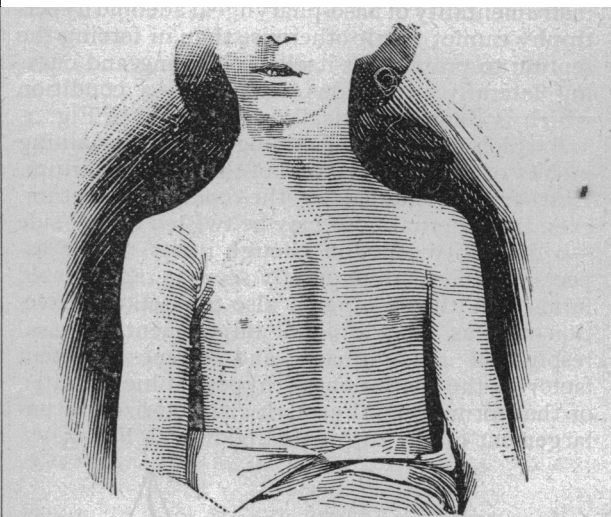


Figure 6. After Hooper.

Such deformities must result in pulmonary asthenia, and we might proceed to trace therefrom predispositions to pneumonia and tuberculous infection, but we have said enough—sufficient to reiterate that herein lurks one of the most formidable enemies of early life, and to demonstrate that if one would save to the adult his face, his figure, yea, even his reason, one must give to him in childhood a clear respiratory passage.

70 Monroe St.

A SIMPLE AND RELIABLE ASTIGMOMETER.

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It is an old saying that "haste makes waste;" and every medical man will do well to have these three words inscribed in golden letters over his desk; for three-fourths of all the mistakes made in medical practice can be traced to hasty examination. But while we should spare no time in

⁶ Tumeurs Adenoides du Pharynx nasal, Paris, 1879.

⁷ The Mechanical Effects of Adenoid Vegetations in Children. Reprint from the Medical and Surgical Reports of the City Hospital of Boston, 1889.