Original Articles.

ON SOME FORMS OF ADENOID DISEASE WHICH ARE OFTEN OVERLOOKED, AND ON CONDITIONS WHICH MAY SIMULATE ADENOID DISEASE.¹

BY JOHN W. FARLOW, M.D., BOSTON.

THE terms "adenoid disease," "adenoid hypertrophy," "post-nasal obstruction," "mouth-breathing," are so commonly associated that they are considered as almost synonymous. It is not uncommon for patients to be told that there can be no adenoid hypertrophy because there is no snoring or open mouth, and by no means infrequent that cases are referred to specialists for operation where the only symptom is inability to keep the mouth closed. So many of such cases have come to my notice that I have thought the possibility of one condition existing without the other was not sufficiently borne in mind.

The two points which I wish to make are: first, that adenoid disease often exists and causes symptoms demanding operation, without there being mouth-breathing or apparent nasal obstruction; and, second, that mouth-breathing may be caused by many other conditions than adenoid disease.

At birth the vault of the pharynx is occupied by ridges or folds of lymphoid tissue arranged like an open fan, or the cerebral convolutions, with depressions between them, the central depression being generally the largest. If the ridges increase and hang down as separate masses, the name "vegetations" is often given to them, while the term, "pharyngeal tonsil," is commonly applied to a more or less uniform, rounded, firmer enlargement with but little prominence of the ridges. The post-nasal space of the young child is small, and is merely a part of a tube which connects the anterior part of the nose with the larynx. There is practically none of the dome-shaped cavity with which we are familiar in the adult. This narrow tube is easily encroached upon by even a slight hypertrophy in any part of its length. Hence in the infant a very small amount of adenoid disease may cause a considerable amount of obstruction to breathing.

Let us suppose a child has reached the age of twelve years and the post-nasal space has developed into a good-sized cavity while the adenoid tissue has remained small. From a succession of colds, the grip, or a contagious disease, the membranes of the nose and throat have become much inflamed, and with them the adenoid tissue, where the inflammation is very prone to remain long after the rest of the nose and throat are well. As a consequence, we may have a chronically inflamed, but not necessarily obstructive, gland which pours out secretion, forward into the nose, and downward into the lower pharynx, larynx and esophagus, causing rhinitis, pharyngitis, cough, hoarseness, loss of appetite, and indigestion, sensitiveness to cold, etc. I have seen this state of affairs started up by a cold in November, continue through the winter and till warm weather.

We will call this the small catarrhal adenoid, which can occur without causing mouth-breathing in patients whose post-nasal space is of good size. I have had a number of cases under my care, from fourteen to eighteen years of age, where the growth and enlargement

of the bony framework of the post-nasal space seemed to be accompanied with quite a marked secretory activity of this pharyngeal tonsil, and the frequent and prolonged colds have given rise to the idea that the children had outgrown their strength, when, in reality, all that was needed was removal of a focus of irritation and disease. The idea that the physiological atrophy of an organ is a normal process and needs no medical or surgical interference has certainly not proved true in my experience. This atrophy often takes place at a later date than is usually assigned to it, and after the obstructive symptoms have disappeared an abnormal amount of secretion in the throat is a sign that, although the gland may be smaller, it is certainly not without activity and may require attention.

After the catarrhal symptoms have abated, and also quite apart from them, there may be disturbances caused by this relatively small growth, that is, small in comparison with the space which contains it. I think all will agree that the size of the growth is not a certain index of the number or severity of the symptoms. Some large growths may be accompanied by a minimum of discomfort, whereas in certain cases a small growth may be the starting-point for many reflex troubles. As a consequence, we are often called upon to treat cases where there is no mouth-breathing, but where various nervous phenomena demand the removal of what adenoid tissue there is. This may be true in croup, chorea, nervous twitchings, hoarseness, hacking cough, scraping of the throat, to say nothing of diseases of the ear. We may make a comparison with chronically diseased tonsils. It is not by any means hypertrophy alone which requires treatment. Small tonsils with diseased areas and follicles prone to inflammation demand attention equally with the *hypertrophied* glands. Many young persons go through a period of so-called delicate throats from diseased adenoid or tonsillar tissue without hypertrophy, and recognition of the existence and importance of non-obstructive adenoid disease should bring about a diminution in the number and duration of such cases.

Coming now to the second point, conditions which may simulate adenoid disease, any obstruction in the upper air-passages above the uvula may necessitate mouth-breathing: deviations, spurs and ridges of the septum; weakness and falling in of the alæ; catarrhal secretion which children neglect to blow out, and which adheres to and stops up the nose; chronic eczema of the anterior nares, with formation of crusts which block the nostrils. It is not very uncommon to find the openings of the nostrils too small. The interior of the nose may not be large enough for the individual. The septum may be straight and the turbinates not large, but the nose seems built on too small a plan, a sort of misfit. Children's noses are small enough at best, and any reduction in size may lead to snoring and mouth-breathing. It is often a matter of great difficulty to determine whether a given nose is large enough for its proper function. Its weak points are best observed at night. I have in mind a number of patients who habitually wear some form of internal expander at night to dilate the nostrils. As children they had many symptoms which suggested adenoid disease, but repeated examinations showed the absence of post-nasal obstruction.

Hypertrophy of the turbinates, especially of their posterior ends, in young adults may have a close resemblance to adenoid disease. The two may be asso-

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ciated, but not necessarily so. The rare congenital closure of the posterior choanæ by a membranous or bony diaphragm, especially when the occlusion is not total, may easily give rise to the idea that the more common adenoid growth was present.

In young adults I have seen a number of instances of bilateral hypertrophy of the submucous tissue of the posterior end of the vomer, which had persisted for some time and which had been considered to be post-nasal obstruction requiring operation.

Retro-nasal fibromata springing from the basilar process may simulate adenoid disease, but when they are large they cause a much greater degree of obstruction than adenoid disease. Polypi in the posterior part of the nose, especially when projecting into and filling the post-nasal space, are a possible source of error, but are so uncommon at the age when adenoid disease is usually found that they seldom cause the mistake to be made.

I have often noticed that the post-nasal space itself was small, the distance between the Eustachian eminences much reduced, and, at times, these eminences enlarged and encroaching on the calibre of the space. This condition may go with a narrow nose, and both may have been brought about by former adenoid hypertrophy, which has atrophied subsequently leaving the bony parts too small. The post-nasal space, being thus smaller than it should be, serves to hold back somewhat the air from the nose, especially when there is any acute swelling or undue amount of secretion, and the same obstructive symptoms may exist as were present before the disappearance of the adenoid disease. This similarity of symptoms sometimes leads to disappointment on the part of parents when they fail to see the great improvement in breathing which they had hoped for as a result of operation on their children.

The distance from the soft palate to the posterior pharyngeal wall may be much diminished by a forward projection of the upper vertebræ, which is not by any means uncommon, or the soft palate itself may extend backward so far as to leave but a small cleft for the passage of the air, and hence mouth-breathing may be necessary. In such cases it is often very difficult to obtain a view of the post-nasal space with the mirror through the narrow opening, and it may be almost impossible to pass the finger up behind the palate.

The jaws and teeth may be the sole cause of the open mouth, and children with irregular and very large upper teeth often find it impossible to keep the lips closed over the teeth. The enormous, central, upper incisors which appear in some children at the time of the second dentition are plainly too large for the upper lip, which has not begun to assume adult size. If we were obliged to buy, at eight years of age, a hat that would be the right size for us when we were twenty-one, we should not be very much surprised if for a number of years we had a very poor fit. The same holds true of the teeth. These large incisors, not covered by the small upper lip, and the open mouth cause the parents to believe very naturally that there is some post-nasal growth, especially if some kind friend or neighbor repeatedly suggests this as a probability. If the mouth is kept open from necessity there may ensue pharyngitis, large tonsils, cough, disease of the nose, etc., symptoms strongly suggestive of adenoid disease.

Occasionally I have seen the tongue too large to remain comfortably between the teeth when the mouth

was closed, and, from the resulting open mouth, the post-nasal space was suspected.

The specific snuffles of the young child may simulate adenoid disease very closely; and as the post-nasal space and fauces may be too small for the passage of the finger, it is well to bear in mind the possibility of specific disease whenever nasal or post-nasal obstruction is present, especially in the infant. Specific adhesions, bands, hypertrophies, ulcerations with secretion may cause obstruction later on.

MEDICAL EXPERT TESTIMONY IN THE KELLEY MURDER TRIAL.¹

BY WALTER CHANNING, M.D., BROOKLINE, MASS.

ON April 16, 1897, the dead body of Joseph A. Stickney, cashier of the Great Falls National Bank of Somersworth, N. H., was found on the floor of the bank. Death had been produced by numerous blows on the head and by cutting the throat with a razor.

Suspicion quickly pointed to Joseph E. Kelley, who lived in the adjoining town of Berwick. He was arrested three days later in Montreal, and confessed that he was the murderer.

Experts were employed by the defence to examine into his mental condition, which led to the employment of others by the State for a similar purpose. This procedure was rendered necessary, as the law of New Hampshire specifies that, "where insanity is set up as a defence to an indictment the jury must be satisfied beyond a reasonable doubt that the killing was not produced by mental disease."

Drs. Cowles, Jelly and Channing were the State experts, and with Drs. Bancroft, Gorton and Waterman, the defence experts, examined the prisoner several times before the trial.

As a result of these examinations and the evidence brought out at the trial, all of the experts came to the conclusion that Kelley's brain was imperfectly developed and that, in the full sense of the word, he was not responsible.

This unanimous opinion led the defence to change the prisoner's plea of "not guilty" to "guilty." This change made a very important difference, as the judge under the New Hampshire law could determine the degree of guilt, and a jury was no longer necessary. The jury accordingly was dismissed, and the trial became a hearing before the judge.

It was arranged that after the evidence was all in the experts should testify, those for the defence being called first. The difference between testifying in this manner and that ordinarily pursued was marked. In this case each expert gave his testimony directly to the judge, expressing freely and unqualifiedly his personal opinion and was not made to appear the prejudiced advocate of either one side or the other.

The experts were unanimously of the opinion that Kelley's responsibility was limited because of brain defect, and they all answered in response to the question of the judge, that they thought he did not possess the capacity to form a premeditated and deliberate purpose to the degree that a sane man would.

The court established the degree of murder as that of the second degree, and the prisoner received a sentence of thirty years, as there is no sentence to life imprisonment in New Hampshire.

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