

existed in such quantity, or have been developed in such positions, or have been so affected by inflammatory processes, as to cause interference with free nasal respiration, the effect of the same is always felt in the development of the nose as a whole, and in the majority of cases, can be seen in the shape of the outer nose.

The *adenoid nose*, is essentially a weak one, and one showing signs of irregular and unsymmetrical development; and its shape often mars a face which would have had much beauty, had not the regular growth of the nose been interfered with. The delay in the development of the nasal bones causes a flattening of the bridge of the nose. This delay is due to lack of stimulus from the active advance of the septum. The nasal bone lacks support. In the same way the lateral cartilages of the nostrils—owing to disease of the nostrils during complete stoppage of the nasal passages and to their unequal use when one side of the nose is freer than the other, and to their want of full support from the septum—become usually developed, or are developed not at the proper planes to each other; and, as a result, the nostril-walls thicken, or at times are too thin in certain places, and lose their normal curves. The later development of the facial bones and of the nose, removes, in a great measure, many of these faults due to nasal obstruction; but where excessive adenoids have existed for a long period of time, the nose, no matter how vigorous its development after the restoration of nasal respiration, will always tell plainly its early history. The septum is also affected by the stoppage to nasal respiration, and a certain proportion of the cases of thickening of the cartilaginous portion, especially in its upper posterior aspect, can, I am inclined to think, be traced to the early existence of adenoids.

It may be of interest to mention here, in passing, two conditions, which, though I am unable to prove them to be directly, or indirectly, dependent the one upon the other, are often enough associated to make an observer wonder whether they are the results of a common cause; and, if so, whether the one condition may not have some influence in determining the other. The two conditions are:

1st. A hypertrophic state of the upper and posterior part of the cartilaginous septum of one side—so hypertrophic that it furnishes an obstruction to the direct passage of air from the nostril entrance to the middle and superior turbinates, and often preventing any anterior view of the middle turbinate.

2nd. A marked diminution of the power of hearing on the side corresponding to this enlargement of the septum, as compared with the hearing power of the opposite side; and this when the air passages of the lower and posterior parts of

the nose are apparently sufficiently free. Often, in older children, and in adults, these adenoids, or their remains, are the cause of a hypertrophic condition of the turbinates, which it is useless and unscientific to try to reduce with acids or the cautery as long as the adenoids are left in the nasopharynx, and which disappear without further treatment as soon as they have been removed. Epistaxis is of not infrequent occurrence in these cases, and it is probable that its source is to be sought in some small ulceration caused either by the nature of the catarrhal discharges or by pressure of the swollen turbinates.

II. *Effects upon the Eye.*—In writing of the delay in development of the nose bones caused by nasal obstruction, mention was made of the ethmoid bone, whose orbital plate furnishes not an inconsiderable part of the bony surface of the orbit. It is in the highest degree probable, that continuous nasal obstruction in early childhood delays the development of this plate, as well as of the rest of the ethmoid bone, it being an integral part of this bone; and, if so, the orbit is necessarily prevented from developing properly. This is a highly important point, for the eyeball is contained in the orbit, and the shape of the orbit necessarily determines, to a greater or less degree, the shape of the eyeball.

And just here, I believe, is to be sought the explanation of the fact, that the majority of children who have suffered from nasal obstruction, and post-nasal adenoids, as the chief cause of this condition, are far-sighted, and to a degree higher than can be explained by inheritance. The eyeball being in the orbit is influenced in its development within certain limits, though the laws of inheritance stamp their plan upon it while it is in embryo, by the development of the orbit. And here, again, in the faulty development of the orbit, is to be found the explanation of some of those sporadic cases of astigmatism which one meets with from time to time—that is, certain of those cases of astigmatism which are not inherited, and which cannot be explained by influence of previous inflammatory conditions of cornea, etc. I will cite one example here which is striking enough.

Mr. X, aged 22, has a father, mother, three brothers and a sister whose eyes may be called normal, showing but a slight amount of hyperopia; no myopia in the family. Mr. X, himself, shows marked astigmatism in one eye, while his nasal history is one of obstruction to a greater or less degree, greater in one side of the nose than the other, and lasting for a number of years.

Most writers on refraction of the eye say that in many cases the degree of hyperopia decreases as the child attains its growth. The degree of nasal stenosis, due to post-nasal adenoids, grows less and less as the child grows older; and so more and more of one of the necessary stimuli to