Ocular affections due to nasal disease / by Henry Eales.

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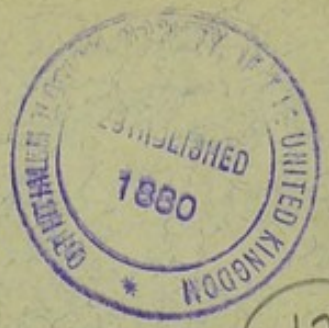

OCULAR AFFECTIONS DUE TO NASAL DISEASE.

BY

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OCULAR AFFECTIONS DUE TO NASAL DISEASE.

By Henry Eales, M.R.C.S.

The sub-division of medical and surgical practice into numerous specialities increases the necessity for co-operation in the diagnosis and treatment of disease, as is well shown in the cases I propose to bring before you.

Ophthalmologists have long suspected that some of the conditions coming under their notice were the result of nasal disease, but it is only with the very modern development of rhinology as a speciality, and by co-operating with the rhinologist, that it has been possible to show this connection in many cases.

As a rule the subjects that interest the ophthalmologist are so special and technical as to be hardly suitable to bring before this Branch, but the subject I am now dealing with appears to me to be one that all may be interested in, specially the rhinologist, and so I deemed it suitable to bring before you, not so much in hope of myself adding materially to what is known on the subject, as from a desire of gaining knowledge of value by mutual discussion with rhinologists, to whom we must look for any advance of our knowledge in this class of cases.

I do not attempt to deal with the whole subject of the connection between ocular and nasal disease, which would be impossible in the time at my disposal; but to confine myself to the results observed by me in some cases of acute suppuration, or distension, of the accessory nasal sinuses; more especially the ethmoidal cells, sphenoid sinus, and the antrum of Highmore.

I shall leave out of account cases of obstruction of the nasal duct, as outside the purpose in view, nor do I propose to deal with the frontal sinus, disease of which rarely invades the orbit.

I would here remind you that these accessory sinuses discharge their secretions into the nose by very narrow canals or openings, which may easily become occluded; or if the secretion is very free, as in acute catarrh, with swelling of the mucous membrane, may be insufficient, and so lead to distension of the sinus.

1. The anterior ethmoid cells discharge with the frontal sinus, into the middle meatus, through the infundibulum, a long, narrow canal. The antrum of Highmore also discharges into this meatus.

2. The posterior ethmoid cells discharge into the upper meatus on each side by one or sometimes two openings. The sphenoid sinus also discharges into the upper meatus by a small opening on each side, the sinus being a double one, with a bony septum between the two sides.

The inaccessibility of these latter openings is one of the great obstacles to diagnosis of disease in these sinuses, which are so calculated to cause damage to the optic nerve.

It may be that in many of the cases coming to the notice of the ophthamologist there has been a long standing previous chronic disease in the sinuses, indeed the history often suggests this, but no serious ocular lesions occur until there is some sudden obstruction to the free discharge from these sinuses, leading to distension; or acute suppuration takes place in them.

In short, I propose to deal only with

1. Cases of rapid proptosis, due to sudden invasion of the orbit, in suppuration of the accessory sinuses.

2. Cases of retrobulbar neuritis, due to extension of disease in the posterior ethmoid cells, or sphenoid sinus, to the optic nerve.

I intend only to relate the salient features of the cases, and not to weary you with ophthalmological or rhinological technicalities or details, the latter indeed being beyond my knowledge.

PROPTOSIS.

1. It is sometimes one of the most difficult questions in surgery to determine the cause of proptosis; but the cases I am now dealing with have a character that makes them easily recognised by the following facts :—

The proptosis is *rapid*, coming on in the course of 24 hours or a few days, and is accompanied by headache, malaise, and often a rise of temperature, and often there is a history of preceding nasal discharge, and often of a preceding attack of influenza. The following two cases are fairly typical :—

W.P., aged 15, a delicate, spare, overgrown lad, came to the Eye Hospital on September 8, 1908, and said that for six days previously the eye itched and felt gritty, and three days before began to "swell." On examination, he had marked proptosis of the left eye, directly forwards, but no loss of movement. No optic neuritis was present, or pallor of disc, and there was no marked injection of conjunctiva or chemosis. R.V.=$\frac{6}{18}$, L$\frac{6}{36}$, apparently due to hypermetropia with astigmatism. Distension of ethmoid cells was diagnosed, probably due to acute suppuration. He was sent to the Ear and Throat Hospital, and next day Mr. Seymour Jones removed the middle turbinate, and curetted the ethmoid cells, evacuating much mucopus, and the following day the proptosis was much less, and in three or four days was almost quite gone, and eight days later was scarcely perceptible. When last seen (November 17) he was apparently quite well, and had no discharge from the nose.

2. A.J., a lad aged 18, came to the Eye Hospital on November 27, 1908, and said that six days ago he had a bad cold, followed by severe headache, and two days later the upper lid of the left eye drooped, and the left eye became pushed outwards and forwards.

On examination, I found considerable proptosis, the left eye being displaced outwards a litttle. There was some tenderness on pressure over the inner wall of the orbit, and movements of the eye were very slightly limited, upwards, outwards, and

downwards. He complained of diplopia, and the false image (viz., of left eye) was to the right of and below the image of right eye, showing the left eye to be displaced out and up. The fundus was normal, and there was no conjunctival injection or chemosis.

Diagnosis of acute distension of the ethmoid cells, probably due to suppuration, was made, and the case sent to the Ear and Throat Hospital, where this was confirmed by Mr. Seymour Jones, who found some polypi and œdema of middle turbinate, causing obstruction to the outflow from the anterior ethmoid cells. The polypi were removed, and part of middle turbinate. Next day he was a little better, the following day the proptosis much less. On December 2 I noted all proptosis gone, movements of the eye normal, no diplopia, V.=$\frac{6}{6}$, fundus normal.

In my Middlemore Lecture (1905) I recorded an almost identical case in a girl aged 10, which terminated satisfactorily and rapidly, in the same way after similar treatment at the Ear and Throat Hospital, and so far as I know, she has had no further trouble since, though I have not seen or heard of her for some two years now. In the same lecture I reported a similar case, which, however, delayed in seeking treatment, and when seen an abscess had formed at the inner side of the orbit, which had burst through os planum and the conjunctiva, and was discharging over the cornea, which became infected by a destructive ulceration, which caused permanent and serious damage to vision by nebula of the cornea resulting, showing the importance of dealing with these cases early by intra-nasal curetting if necessary.

A worse result still sometimes occurs, when general orbital cellulutis and abscess is set up by the orbital tissues becoming invaded, causing optic neuritis, with subsequent blindness from atrophy of the nerve, or more often from thrombosis of the retinal artery taking place; though it is remarkable how completely the optic nerve recovers from the neuritis set up in these cases sometimes, if the suppurative process rapidly subsides.

Many years ago I saw a case in a lad about 12, in which proptosis of right eye, with intense optic neuritis of this eye, both rapidly disappeared on the spontaneous discharge within a few days of onset of pus from the nose, the optic neuritis leaving no permanent defect of vision, or even ophthalmoscopic evidence of its previous existence, and a similar result of complete recovery of the optic nerve after neuritis in orbital abscess has been recorded by others. Until recent years it was the habit of ophthalmic surgeons to treat these cases on the expectant principle, and if abscess formed in the orbit, to open it, and drain, but the cases I have described indicate the expediency of at once dealing with any nose trouble found, and so preventing this if possible. I agree entirely with the opinion expressed by Sinclair Thompson in an instructive paper in the *Ophthalmoscope* for April this year that bearing in mind the intimate association between the nasal accessory cavities and the orbit, I have no doubt that future experience will show nearly all cases of orbital abscess and periostitis and cellulitis to be due to extension of disease from the nose; unless it may be those cases of periostitis and abscess under the external angular process of the frontal bone, which are frequently met with, and are perhaps due to the exposure of this part of the orbit to injury; but that even abscess in this position may be due to nasal disease is shown by a very interesting case recorded in my Middlemore Lecture (1905), in which an abscess, which formed in this region, and which repeatedly recurred, and was opened by me, was found to be due to abscess in the posterior ethmoid cells, which had burst through into the apex of the orbit, and formed a sinus along the roof of the orbit, pointed near the external angular process, which was opened, and in syringing out which sinus fluid passed into the nose, and in which Mr. Marsh found extensive disease of the posterior ethmoid cells, which he curetted.

ANTRUM.

Empyema of the antrum invades the orbit through the floor, along the course of the canal for the infraorbital vessels

and nerve, causing displacement of the eye upwards and abscess, which points usually at the outer end of the lower lid, where it must be opened if necessary; but it is important if possible to prevent this by taking early measures to drain the antrum in the usual way.

It may be thought that empyema of the maxillary sinus would not occur in the infant, in whom the sinus is rudimentary, but I have twice had to deal with an abscess evidently arising in the nidus of the maxillary sinus, bursting through the floor of the orbit, in children only a few weeks old. (See Middlemore Lecture, 1905, in *Medical Review*, 1906.

It would seem that in the early stage of these cases the proptosis is due to bulging of the inner orbital wall from distension of the underlying ethmoid cells, and that this rapidly subsides on obtaining free drainage of these cells into the nose; but if much delay occurs before this is secured we find abscess and cellulitis of the orbit rapidly taking place from invasion of the orbit in the suppurative process.

RETROCULAR NEURITIS.

Ophthalmologists have long been familiar with cases diagnosed as retrocular neuritis. In these cases there is a history of sudden or rapid failure of vision in one or both eyes with diminution or loss of pupil reflex to light, in which, while the F.V. is full, there is a marked failure of central vision, and central scotoma is found for colours, or is even absolute. There is often no evidence of disease on ophthalmoscopic examination, and often some pain on ocular movement or on pressing back the eyeball, but after an interval of five or six weeks the disc will be noticed to become paler, and ultimately atrophic.

Now if, in such a case, as is often found, only one eye is affected, then the presence of consensual reflex to light, though direct reflex is lost or bad, is absolute evidence that the lesion is in the optic nerve, demonstrating, as it does, that the efferent impulses from the brain to the eye, that the third nerves are unimpaired, and that the phenomenon is therefore due

to interference with the efferent impulses from the eye to the brain, carried up by the optic nerve, and our experience that in cases of fracture of the skull, in which the optic nerve is damaged in the neighbourhood of the optic foramen, and in which no change is shown for five or six weeks at the disc, which then becomes gradually atrophic, indicates that in these cases, too, the lesion is probably in the neighbourhood of the optic foramen, or at the apex of the orbit.

Now Onodi has shown that the optic nerve in the optic foramen is often only separated from the sphenoid sinus and the posterior ethmoid cells by the thinnest bony partition, often no thicker than a piece of paper, and even in some rare cases bone being deficient in this partition at places where it is only membranous, so we see how very exposed is the optic nerve to danger of damage from retention of pus or mucus in these cells, and the following cases I am bringing forward, in which disease in these sinuses was present, and the way in which in some sight at once improved after attention to the nose, would seem to emphasise the connection between the nasal affection and that of the optic nerve.

No doubt our past experience shows that some of these cases recover without operative interference, while in others vision, though defective, may be retained, but I am disposed to think that in these cases it is imperative to ascertain if possible if there is retention of pus or mucus in the ethmoid or sphenoid sinus, and if possible to evacuate it early before too much damage has been sustained by the optic nerve, and hence in these cases it is my practice to seek the advice of the rhinologist early, on whom must of course devolve the decision as to whether the sphenoid sinus and ethmoid cells should be explored when they do not present obvious appearances of being affected by empyema.

The rapid failure of vision in these cases, and its almost equally rapid recovery, is to my mind in favour of compression of the nerve rather than extension of inflammation to it, being the cause of failure, while the absence of evident neuritis at the disc also favours this view; moreover, the way in which rapid

proptosis occurs without signs of orbital inflammation, and evidently caused by bulging of the orbital wall in the cases of proptosis, just described, shows how easily these thin walls yield when there is retention in the underlying cells; but in cases where there is much inflammatory change in the muco-periosteum of these cavities with such thin walls, and where retention is maintained unrelieved for any length of time, it is probable that the inflammatory condition extends to the periosteum on the other side of these thin bony walls, which at the optic foramen is closely associated with the sheath of the optic nerve, which would therefore almost certainly become involved in the inflammatory process.

The first case of which I have notes, in which there appeared to be nasal trouble as the probable cause of the visual failure, is that of L.C., which was reported fully in the *Lancet* for September, 1905, by the Resident Surgical Officer to the Eye Hospital, Mr. Percival Hay, and Dr. Glegg.

She attended first on January 20, 1905, complaining of being unable to continue her work at the telephone office, owing to inability to see the keys, especially on her right-hand side, and having to turn her head to see this side—she also complained of dull headache and of diplopia—which had come on after an attack of influenza, about Christmas before. On examination I found R.V.=$\frac{6}{12}$, and L.V.=$\frac{6}{18}$ and charts taken showed the R.F.V. to have lost almost the entire temporal half, while there was a similar but less deficiency in the temporal half of the F.V. in the left eye also. I also found she had apparent complete paralysis of the right external rectus (6th nerve), moreover the left eye did not deviate much beyond the middle line in attempts to look at the right side; while on looking to extreme left, movements of both eyes were jerky in this position. The fundus was normal in each eye.

I diagnosed retro-ocular neuritis, from probable sphenoidal or post-ethmoidal sinusitis, following influenza, and sent her to the Ear and Throat Hospital on January 31st. Dr. Glegg found slight muco-pus at the roof of the nose on the right side, in the spheno-ethmoid recess, which was again observed

on February 3, and on February 15 Dr. Glegg opened the post part of the ethmoid labyrinth, and evacuated a small quantity of pus with the curette.

After this procedure her vision at once began to improve, and next day there was considerable enlargement of her F.V. in the right eye, on the temporal side. On February 21st the L.F.V. was normal, and the right nearly so, only a deficiency of about 10 deg., and V=$\frac{6}{8}$ in each eye, and there was marked recovery in the paralysis of the right external rectus, the right eye moving out well beyond the middle line. March 27 V=$\frac{6}{6}$ in each eye. F.V. was full in each eye. Paralysis of right external rectus was practically gone. From that date she had had no further trouble with her eye, so she told me when I last saw her, on December 4, 1908, having sent for her to report herself, when I found her eyes in all respects well, as last noted.

In the *Lancet* Dr. Hay discusses the paralysis of movement at much length and some complexity, but I am not sure that the explanation is not much simpler, being due to paralysis of the right external rectus, while the left internal rectus was simply inhibited voluntarily or involuntarily in attempts to look to the right, owing to a desire to avoid diplopia, which was very annoying to the patient, while the jerky movement in looking to the extreme left may have been due to want of proper control by the weak right external rectus when this movement took place.

R. C., female, aged 33, came to see me first on November 13, 1906, stating that six days previously her sight, which was good on rising, began to fail about 10 a.m., and got worse all day, but during the last three days had been improving. Frontal headache, which was present at first, was now gone. On enquiry, I learned that she had had an operation on the nose, at the General Hospital, in 1905, for diseased bone. Says she still has discharge at the back of the nose.

On examination, I found R.V.=$\frac{6}{12}$ part, and L.V.=$\frac{6}{12}$ part. The F.V. of left eye was contracted to a small central area, and there was considerable contraction of nasal side of F.V. in

right eye. The fundus appeared normal in each eye, except for some fulness of the retinal veins.

Right pupil reacts sluggishly to light, the left only very slightly. She was sent to Dr. Foxcroft, who evacuated a cyst in the ethmoid cells. On November 20 I found R.V.$\frac{6}{8}$ part, L.V.$\frac{6}{8}$ part, but already there was considerable expansion of F.V. in each eye, though considerable loss of temp. half in left eye, and some contraction on nasal side right eye, both fields were larger on December 4, and on December 11 both F.V. were full, and R.V.$=\frac{6}{6}$, and L.V.$=\frac{6}{8}$ full. I saw the patient a few days ago, and found her vision$=\frac{6}{6}$ in each eye, and learned she had had no further trouble with her sight since attending here in 1906.

A very similar case, a man aged 23, of blindness in one eye, with rapid recovery after resection of the post end of the middle turbinate, which was under the care of my colleague, Mr. Wood White, is recorded by Mr. Evans in his paper in the *Ophthalmoscope* for April last, in which paper Mr. Evans also gives an epitome, of which the following gives the salient points, of my third case, which is worth including here, and which came under my care first on January 8, 1907.

Amy Marion, age 34, gave the history that she had been unable to see to work for six months. No nose trouble; pupils sluggish to light. No pain on pressure or movement of globe. R.V$=\frac{6}{18}$, $\frac{6}{6}$ partly C.+75, L.V$=\frac{6}{36}$, not improved. Temporal contraction of both F.V. Large scotoma in left eye. Fundi normal. No nasal disease found on examination of nose. Fortnight later, R.V.$=\frac{6}{18}$ partly, L.V.$=\frac{6}{60}$ L.F.V. cannot be taken. Fortnight later, R.V.$=\frac{6}{12}$, L.V.$=\frac{6}{24}$, colour scotoma in both eyes, left most marked. Mr. Seymour Jones operated several times, removing polypi from the left posterior ethnoid cells, and opening up a purulent right sphenoid sinus.

In this case both F.V. showed extraordinary changes while under observation, but ultimately became good examples of bi-temporal hemianopsia about May, 1907, since which they have remained the same, as also her vision, which then was R.V.$=\frac{6}{8}$, L.$=\frac{6}{18}$, while the temporal sides of optic discs also became very white and atrophic, and when last seen,

December 4, 1908, her condition was precisely the same. She then said she had had no attacks of failure of vision for eight or nine months, and had no trouble with her nose.

E. S., age 22, came to the Eye Hospital on September 7, 1908, complaining that the right eye went blind on September 3rd. On examination, showed right eye V.=$\frac{6}{60}$ only. The fundus appeared normal, and the pupil reflex was sluggish, though consensual reflex good. Left eye V=$\frac{6}{6}$. Fundus normal, pupil reflex normal. Charts taken showed slight loss of nasal side of F.V. in right eye, scotoma for colour right eye—not in left. She said she had had bad cold in her head all the year. Brought to my notice first September 18, when R.V. = fingers only six inches. Diagnosis of retro-ocular neuritis, probably due to distension of post ethnoid cells or sphenoid sinus was made, and she was sent to Ear and Throat Hospital, where Dr. Seymour Jones found the septum deflected to right and right middle turbinate boggy, and compressed against side of nose, and hypertrophic rhinitis present.

September 26 Mr. Seymour Jones operated on deflected septum, and a week later removed the posterior end of middle turbinate, and opened the sphenoid sinus and drained it. September 28 R.V. = fingers at two feet only.

October 3, R.V.=$\frac{6}{18}$, temp. side of disc for first time noticed to be a little pale, and this has increased since. Sight has gradually improved, and F.V. gets larger. The left eye has remained normal throughout V=$\frac{6}{6}$ and F.V. full.

November 24, R.V.=$\frac{6}{12}$, F.V. nearly full. Slight nasal contraction. Still colour scotoma. Disc very pale. Left eye normal. Pupil reflex good in each eye. No headache. No obvious nasal trouble now.

L. M., age 21, attended the Eye Hospital on September 22, stating that her sight went rapidly one Saturday morning five months ago, but soon got better, but has varied ever since. "At times can see a treat."

On examination, R.V.=$\frac{6}{24}$, L.V.=$\frac{6}{36}$. Large colour scotoma in each eye. Both discs pale, the left most marked. Sees best in afternoon and at twilight. Pupil reflex sluggish

in each eye. R.F.V. contracted a little on nasal side. L.F.V. contracted on both nasal and temp. sides.

Brought to my notice October 27. Diagnosis of retro-ocular neuritis, probably from sphenoid or ethmoidal sinusitis, with retension.

November 2, a case sent to Ear and Throat Hospital, where Mr. Seymour Jones found muco pus in both olfactory fisssures, and boggy appearance at post ends of middle turbinate, which was removed.

November 16. Post-ethnoid labyrinth curetted. Since this sight has improved, and when last seen, on December 8 last, says her sight does not now go dim if she hurries, as it did before. R.V. $= \frac{6}{12}$, L.V. $= \frac{6}{18}$. F.V. practically full in each eye, but scotoma for colour still in each eye, and discs very pale, especially left.

It will be noted that in cases 1 and 2, which were treated early, there was no evidence of any permanent damage to the nerves, while in case 3, in which the disease was long standing, and the affection of the sphenoid and the ethmoid sinuses was more severe, the nerves are severely damaged, and vision did not recover fully, and there appears to be a permanent bi-temporal hemianopeia.

Cases five and six are more recent, and doing well, and though I fear case 5, in which the left nerve is badly damaged, and in which the vision has been affected eight months now, will probably not recover completely, I hope good vision will be regained. I quite hope case 4 will make a perfect recovery.

I can not but believe that most, if not all, of the cases reported of blindness, following influenza, some of which have recovered, and in some of which atrophy of the optic nerve with more or less permanent blindness followed, probably owe their origin to sinus disease.

A careful study of these cases convinces me that the nose and eyes trouble are associated as cause and effect, and confirms me in the view, that these cases should receive the

earliest attention of the rhinologist, when first coming to our notice, a practice which I shall certainly continue to pursue.

I have sufficiently detained you, and do not propose to deal with the question of paralysis of the 6th, as possibly caused by sphenoid sinus disease to-day, though the proximity of the nerve to the sinus, and paralysis noted in one of the cases included in this paper, and some cases reported by others makes this probable.

I would like to take this opportunity to thank Dr. Foxcroft and the staff of the Ear and Throat Hospital for the ready way in which they always attend to all cases sent to them, as without their co-operation it would be impossible for me to ascertain the presence of nasal disease in any of my patients.

My thanks are also due to Mr. Hird, our late resident surgical officer, and our present resident surgeons, for valuable assistance in keeping records of cases, no easy matter in an out-patient department with an attendance often exceeding 300 in the morning.