Notes on

RETROBULBAR NEURITIS

with

Five Acute Cases due to Rheumatism & Chill

Two Cases of Blindness due to Ethmoidal & Sphenoidal Sinus Disease; with notes on Ozaena, Ethmoidal & Sphenoidal Sinus disease

A Case of Brain (Cerebellar) abscess from middle ear disease. The abscess in somewhat unusual Situation.

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Notes on
RETROBULBAR NEURITIS

It is unfortunate that the meaning of the term Retro-bulbar Neuritis is not accurately defined in textbooks on eye diseases, for it is the cause of much confusion in the minds of most medical men.

Of course it implies an inflammation of the optic nerve behind the globe. But by that are we to understand that we have an optic neuritis without changes in the fundus of the eye, as revealed to us by the ophthalmoscope, yet giving rise to defective vision, central scotoma, etc.

Or, is it a neuritis which is accompanied only by pallor of the optic disc - limited to the temporal side - and certain visual changes, central scotoma, etc.

Or, is it a neuritis which may be accompanied by swelling of the disc - which swelling may be very small in amount or so marked as to be termed "papillitis" - and associated with certain definite symptoms, such as rapid loss of vision, pain on movement of the eye, central scotoma, etc., and all of which symptoms tend later on to disappear.

Apparently since Samelsohn described the first case of Retrobulbar Neuritis the term has been used to describe other forms so that it is now applied to a large class of cases, which vary in type, yet at some
period of their course shew a central scotoma.

Samelsohn described the first case of Retrobulbar Neuritis. The case was one P.K., shoemaker, aged 63, who consulted Samelsohn in 1877 for his sight which had deteriorated in the course of a few weeks. He was emmetropic as regards refraction. But R.V. \(\frac{15}{70}\) L.V. \(\frac{15}{100}\). His visual field appeared to be normal at the first examination. It was only on using small squares 5 m.m. in size at the second examination that a relative central scotoma for red and green was found, which extended about eight degrees round his fixation point. He could discern blue in all parts of the field, it only seeming duller in the region of the central scotoma. No changes were found in the fundus. There was no history of excessive use of tobacco or alcohol. After two months his visual acuity was further diminished, although he entirely abstained from alcohol and tobacco. The central scotoma was now relative for all colours, and was surrounded by an area in which the sensation for colour was much duller than in the more peripheral parts of the field. The diagnosis was probably retrobulbar neuritis. Inunction of Blue ointment was prescribed without, however, any improvement. Two years after the first examination the temporal halves of the discs shewed a whitish decoloration; further, the discs looked hazy. The central scotoma was now absolute, and vision amounted to count-
ing fingers at 18 feet. The patient died in 1879 of heart disease.

Examination of the optic nerves, after death, revealed flattening of the nerve in front of the optic foramen up to the entrance of the central artery of the Retina. There was no effusion into the sheath of the nerve. A section of the nerve near the eyeball shewed an area of grey decoloration occupying a sector on the temporal side. The apex of the sector being towards the centre. This grey area, on microscopic examination, was found to consist chiefly of thickened connective tissue. The trabeculae being much thickened, and the intertrabecular spaces being much contracted and filled with granular debris - the remains of the nerve fibres.

Since Samelsohn's contribution appeared, among others who have worked at the subject - as is well known - are Nettleship, Unthoff, Edmunds, Lawford, etc.

The slow course of Samelsohn's case is similar to the form of retrobulbar neuritis we sometimes see in cases of disseminated sclerosis, often before the latter disease has distinctly shewn itself; such cases may be termed chronic in the same way as we apply the term chronic retrobulbar neuritis to tobacco cases because of their gradual onset and somewhat slow course. But there is a form of Retro-ocular neuritis much more acute, and it is with that form I am chiefly concerned.
Some of these cases are described in a discussion which took place in 1897 at several meetings of the Ophthalmological Society (Transactions of the Ophthalmological Society Vol.XVII). The discussion, which was opened by Mr Marcus Gunn, was "limited chiefly to forms of optic neuritis (with or without papillitis) caused by local disease, such as periostitis in the orbit or optic canal, gumma or other focal inflammation, or growth upon the nerve, in the form of idiopathic neuritis (with or without papillitis) of one nerve or both, which occur after infective diseases, e.g. influenza, in the course of disseminated sclerosis, or without any apparent cause".

This introduction at once shews what a large ground the term covers.

My interest in cases of Retrobulbar Neuritis has been aroused by reading the reports of the discussion, and in doing so one could not help noticing the great diversity of opinion which prevailed amongst leading ophthalmologists and neurologists as to what constituted a retrobulbar neuritis apart from toxic cases. And in a discussion which I opened at the Northumberland & Durham Medical Society last year, I was again struck with the indefiniteness of men's views as to what constituted retrobulbar neuritis when non-toxic in origin.

Medical men, apart from Specialists, have a very vague idea as to the meaning of the terms retro-ocular
or retrobulbar neuritis; and I have come to the conclusion that it would be better to give up using these terms altogether.

Mr Berry, who took part in the discussion, suggested the following classification for cases usually placed under the heading of Retrobulbar Neuritis:

(a) Neuritis associated with serious cerebral disease other than such as produced papillitis, e.g. those existing along with chronic alcoholism, syphilis, etc.

(b) Unioocular neuritis - which was probably almost invariably the result of chill.

(c) Binocular neuritis, as found in toxic amblyopia.

If this classification were adopted the term papillitis could be reserved for cases of Meningitis, tumour of the brain, etc., where we invariably get a papillitis or choked disc, the term neuritis being reserved for other cases, thus dropping out of use the term retro-ocular neuritis altogether.

For my own part - putting aside toxic and nervous cases - if I can satisfy myself from the history, symptoms, etc., that the site of the lesion is somewhere between the central opening of the optic foramen and the globe, no matter the ophthalmoscopic appearances, I call that retrobulbar neuritis. Such a group would include gouty, rheumatic, and "chill" cases, cases due to infec-
tious disease, cases due to orbital cellulitis, periostitis, and gumma, etc.

Two of the commonest causes of acute retrobulbar neuritis, in my experience, and I have met with a considerable number, are rheumatism, and sudden chilling of the face after coming out of a warm atmosphere.

During the past twelve months I have kept notes of five such cases which I append.

Two of the patients gave a distinct history of rheumatism; three gave a distinct history of exposure to cold after coming out of a warm atmosphere. Three of the patients were males, two females. One eye only was affected, and curiously enough in all it was the left one.

I have specially noted rheumatic and chill cases because of the variety of views expressed in the above-mentioned discussion, as to rheumatism and chill being causes of retrobulbar neuritis.

Gunn says: - "Sometimes an orbital cellulitis seems to follow great exposure to cold". Again he says: - "The nerve in its passage through the optic canal is not only exposed to inflammation from the bony walls and the periosteum, but also from the sphenoidal sinus, only separated from it here by a thin lamina which may be imperfect. It is probable that a considerable number of cases of retro-ocular neuritis traced to exposure to cold are examples of inflammation attacking the nerve in this part of its course", but he says - "Both nerves
are often affected at the same time or almost simultaneously'.

This is an important point and might influence the differential diagnosis greatly.

My two cases of blindness from sphenoidal disease confirms Gunn's statement.

Sir W. Gowers said, "Acute Rheumatic Cellulitis causing prominence of the eyeball, and involving the membranes on that side and affecting the optic nerve, was an affection that had scarcely been recognised".

Mr Berry in his B. Class of neuritis cases would put "Unioocular neuritis" which was probably invariably the result of chill, "whatever other conditions co-existed".

Again Dr Hill Griffith says, "In a fair proportion of cases rheumatism is certainly present".

Mr Silcock said, "Mr Gunn had attributed some cases of retrobulbar neuritis to cold. It really passed his comprehension how this could be - So in rheumatism, what was meant by rheumatism", and "He was not familiar with rheumatic cellulitis of the orbit".

Dr Macnaughton Jones stated, "With a fairly large experience, he had never known retrobulbar neuritis follow on acute rheumatism".

In the discussion I opened at the Northumberland & Durham Medical Society, one Ophthalmic Surgeon practically told me the one or two cases I read as being due
to rheumatism and cold were not cases of retrobulbar neuritis at all. Thus it will be seen that the opinion as to causation differs very considerably, and, as I have already tried to show, so does the opinion as to what constitutes a retrobulbar neuritis.

The chief symptoms in my cases were:-

1. Rapid loss of vision coming on as a grey mist, not like the fogginess of glaneoma. The mist gradually got worse until in all total blindness supervened.

2. Pain in the region round the eye, and often over the side of the head, face and jaws. Over the face and jaws there was generally tenderness to the touch, which is sometimes more marked still over the supra-orbital and infra-orbital nerves.

3. Pain on lateral or vertical movement of the eyes with the head fixed. The pain is dragging in character. Gunn says, "Hoch believes this pain to be due to stretching of the optic nerve: - I, myself, believe the pain to be muscular, or due to a Tenonitis. I have experienced this pain, as well as tenderness over the face and jaws, after being exposed to a cold draught of air coming on to the side of the face, and I have found the same symptom in others after a similar cause without vision being in any way affected.

4. Pain sometimes on pressure of the eye backwards.

5. Pain on pressure over some particular point
of the globe often just beneath the upper lid.

6. In two of my cases each thought he had got a foreign body into his eye at the commencement of the trouble.

7. In three of the cases the pupil was larger on the affected side; one as the result of iridoplegia which at a later examination was found to be accompanied by cycloplegia.

8. In another case there was diplopia ptosis etc.

9. All the cases soon shewed signs of early recovery.

Case I. Mr C., of Middlesbrough-on-Tees, aged 30, Hairdresser. On Whit Monday 1899, Mr C., went to Richmond in Yorkshire, and got a wetting which set up rheumatism. Ten days after this, the rheumatism was so acute that it was found necessary to keep him in bed for four days. A week later the sight of the left eye became affected. It began as a grey mist which rapidly got worse, and in about forty-eight hours the patient was totally blind in that eye. He had pain over the temple and side of the head and face. There was pain on sudden lateral movement with the head fixed. A week after this loss of sight, namely on July 4th, he came to consult me on the recommendation of Dr Young of Middlesbrough, and on making an ophthalmoscopic examination, I found the optic disc very markedly swollen. In fact, it might have been termed a papillitis or a "choked disc".
The retinal veins were over-distended and tortuous, and the arteries narrower than normal, and tortuous. There was a well-marked striated appearance of the swollen disc, and the vessels in curving over it were hidden at points. No haemorrhages were observed. I should have mentioned that the pupil of the affected eye was larger than the other, and reacted sluggishly to direct light. I ascertained that no drops had been applied to the eye. The fundus of the other eye was quite normal, vision also. After a careful examination I expressed the opinion to his doctor that it was a case of rheumatic retrobulbar neuritis, and suggested that the case should be treated like a rheumatic joint, viz:- the eye kept covered with cotton wool and a flannel bandage, Salicylate of Soda to be given internally, and the patient to be kept in bed if there were any swelling of the joints. This to be followed up with Potassium Iodide and blisters to the temple. Whilst expressing this opinion I recommended an examination of the urine for sugar and albumen. This examination I learnt gave a negative result. I should further mention that no specific history could be elicited, and no nervous symptoms found. The patient was a very moderate drinker. He was in the habit of smoking two ounces of twist tobacco weekly, a factor which I think had no direct bearing on the case. A week later the sight began to improve and steadily did so. About six
weeks after my first examination I made the following notes:—L.V.7.10 swelling of left disc has disappeared and is now paler than the right, arteries slightly attenuated. Has a central scotoma for red and green. No contraction of his field of vision.

Case 2. was that of Mr L., aged 24, Signalman, residing at New Shildon near Darlington, and sent to me by Dr Thompson of that town.

Patient said he had just left his cabin at 2 a.m. on June 2nd 1899, when he thought he got a foreign body into the left eye. The eye was irritable for a day or two. On June the 8th, he noticed a grey mist cover over the same eye, on June the 10th the sight was quite gone from it; on the 11th, there was some tenderness of the eye on pressure, especially just beneath the centre of the upper lid. There was pain on sudden lateral movement of the eyes, and some pain in the circumorbital region. On the 12th, when I first saw him, there was slight divergence of the affected eye. The pupil of that eye was larger than the other and did not react so readily to light, and he had the barest perception of light in it. An ophthalmoscopic examination shewed the optic disc to be distinctly congested and hazy, and indefinite in outline. It appeared to be filled in and slightly raised; the veins were too full and there was an appearance of oedema round the disc. The patient was a non-smoker and steady living man as his occupation demanded that he should be. There was no
history of influenza, rheumatism, or syphilis, no nervous symptoms, no disease of the nose, and no history of recent illness. He was inclined to attribute the trouble to the foreign body, but there was no trace of any foreign body, and there had never been any active inflammation as a result of such. In my opinion, the cause was sudden chilling of the face on leaving the warm stuffy atmosphere of the Signal Cabin at 2 o'clock in the morning. It so happened that at this time even in the month of June of 1899 - and the fact was publicly commented upon - we had several sharp frosty mornings, and it was during the time these occurred that the patient's trouble began. On pointing out to him that chill was the likely cause, he stated that he had found it very cold on leaving his cabin in the early morning just then, and he agreed it was probable cold was the cause. The urine was examined in this case also and gave a negative result.

The diagnosis was Retrolublar neuritis due to sudden exposure of the face to cold atmosphere. The treatment suggested was, the wearing of dark glasses, desisting from using the eyes for near work. The administration internally of Salicylate of Soda, to be followed later by Potassium Iodide and blisters to the temple.

On August 9th 1899, that is after an interval of two months, I had a further opportunity of examining
the case. I found the vision markedly improved, in fact when some compound hypermetropic astigmatism was corrected V. was $\frac{5}{6}$ or nearly normal. The left disc, however, was distinctly paler than the right, and the arterial vessels were slightly attenuated. There was no contraction of the field of vision, but there was a small central scotoma for red and green.

Case 3. was that of Miss D., aged 25, of Stockton-on-Tees, and sent to me by Dr Dale of Stockton-on-Tees on April 20th 1899. She stated that a few days previously the sight of the left eye began to get dim, the defect shewing itself at first in the form of a mist and now the eye was quite blind. There was a slight amount of circumorbital pain and distinct, but not marked, pain on sudden lateral movement of the eyes. There were no pupil changes. The ophthalmoscopic signs were not very marked, but sufficiently evident. The left disc shewed distinct want of definition as compared with the right. There was an appearance of haziness; and there was also hyperaemia, the vessels shewing up more, and appearing more numerous in the disc and its neighbourhood in the left eye. Patient was otherwise quite well, is not neurotic or hysterical, there is no history of injury, influenza, rheumatism, or specific disease, and no disease of the nose. Further, there is nothing in the urine. An enquiry as to whether she had been exposed to a cold draught of air elic-
ted the fact that on the day prior to the commencement of the trouble, that she had caught cold in the evening on coming out into the open air after sitting, for several hours, in a hot close atmosphere. I diagnosed the case as being one of Retrobulbar neuritis, due to sudden chilling of the face, and again recommended Soda Salicylatus internally; also repeated small blisters to the temple. Later, Dr Dale informed me that, the patient got quite well. This patient could not give me the opportunity of examining her again for the purpose of reporting on her case.

Case 4. was also sent to me by Dr Dale. Mrs I., of Stockton-on-Tees, stated that in the beginning of May 1899, she had slight headache, followed in a few days by a sudden feeling of stretching in the left eyeball, and she noticed that she saw things double, and objects looked crooked. The sight of the left eye began to get misty, this gradually got worse, so that at the end of a fortnight she could only barely distinguish light from darkness. The upper eyelid now began to droop, and marked ptosis followed, accompanied by oedema and slight proptosis. There was pain on pressure over the eyeball, and pain on sudden lateral and vertical movement. There was also a good deal of circumorbital pain which extended over the side of the head and face. There is no history of recent illness or injury. There is no specific history, and nothing
in the urine. The patient had sciatica five years ago, and frequently has pain and swellings about the large joints.

The note as to the condition of the fundas was:- Disc very hyperaemic; hazy in outline; veins too full.

**Diagnosis.** Probably a case of rheumatic orbital periostitis or cellulitis, involving the optic nerve and the divisions of the third nerve. The treatment consisted in wearing dark glasses, and the administration of Salicylate of Soda and Iodide of Potash internally. The patient was practically well in six weeks excepting for some pain on sudden lateral or vertical movement of the eye with the head fixed. This pain remained for a considerable time and would tend to confirm the diagnosis of orbital periostitis or cellulitis. I have very recently examined this case, and it seems to have recovered perfectly except for slight pallor.

There is no contraction of the field of vision, and there is no central scotoma, neither for white nor colours. This case may be likened to the case described by Sir William Gowers as acute rheumatic orbital cellulitis. His case was that of a young man of gouty heredity, who after a bath developed symptoms which included paralysis of the left ocular nerves, transient proptosis, and tenderness on pressing back the eye. In addition, he had one or two convulsions beginning on the right side and indicating cerebral irritation.
on the side of the affected orbit. I cannot say that there were any cerebral symptoms in Dr Dale's patient.

In the common rheumatic cases due to cold Sir W. Gowers says, it is chiefly the nerve sheaths which are affected. This explains, to my mind, not only the rapid loss of vision, but also, why there is a tendency to early recovery. There is an effusion into the subvaginal lymph space, and the nearer this effusion is to the globe, the more marked are the ophthalmoscopic changes. The deeper the effusion the slighter the ophthalmoscopic signs. These cases, therefore, are in contrast to toxic, alcoholic, and nervous cases, which are more chronic in character, and in which the more central part of the nerve is involved, and which may at no time shew very marked ophthalmoscopic signs.

Case 5. Geo.C., aged 31, bricklayer's labourer, residing at 49 Hatherley Street, Middlesbrough, came to the North Riding Infirmary on February 24th 1900, complaining that three or four days ago, whilst at work, he thought he got something into his left eye. After that, the sight became misty and had gradually got worse until now he was quite blind.

Examination:— Pupil of the left eye widely dilated and does not react either to direct or indirect light. Movements of the eye good. Has had no kind of drops put in the eye. Has a certain amount of circumorbital pain. Pain in the eye on sudden lateral movement. No
pain in the eye on pressure backwards. Some tenderness on pressure over the upper eyelid. No history of any recent illness. Nothing to be found in the urine. Absolutely denies any specific history. States he is a bricklayer's labourer and works at the Steel Works, says it is his duty to go into the furnaces before they are cool for the purpose of repairing them; when inside the furnace he perspires freely, as if in a Turkish bath, and after an interval comes out to cool. The Steel Works are very draughty, and on the evening on which he states he felt the foreign body go into the eye, it was very cold, and at the time he had just come out of a furnace bathed in perspiration. Here we have a most distinct history of chilling of the face after being in a warm atmosphere. He is not rheumatic. Has no disease of the nose. The fundus showed the disc to be distinctly hyperaemic, hazy in outline, appears to be filled in, and the veins are out of proportion to the arteries.

For treatment - 10 grain doses of Salicylate of Soda were prescribed, and a shade to be worn over both eyes. On March 3rd, I again saw the patient, he reports little or no improvement. Same treatment ordered to be continued. On March 11th, considerable improvement V. 50, pupil still widely dilated. Fundal changes unaltered. Same treatment continued. On March 18th, very considerably better. Wishes to re-
sume work. V. 15 (one or two letters) slightly improv-
ed by -ID. Reads large type with difficulty; with
-3D added on reads J.I., thus giving evidence of paraly-
sis of the ciliary muscle. Tested roughly with the
hand, the field of vision is normal. Has a central
scotoma for red and green. Fundal changes not nearly
so distinct. I have omitted to mention that the pa-
tient is a moderate smoker and drinker. But the his-
tory of the case, and its acute character, point alto-
gether to the sudden changes of temperature as being the
cause. I regret I am not able to report further on
this case.

These cases, to my mind, afford pretty clear evi-
dence that we can have a rheumatic retrobulbar neuritis
and a retrobulbar neuritis due to a sudden cooling of
the face. Probably, in both, we have the same set of
pathological changes at work.

As regards treatment, it is difficult to speak dog-
matically as to the effect of drugs. It may be said
that these cases would have recovered without treatment.
That is probable, but perhaps more slowly. I think
both local and general treatment exert a beneficial ef-
fact. Warmth as a local remedy, in such forms as dry
heat, hot bathing, cotton wool and flannel bandage, etc.,
is very beneficial. Whilst I am inclined to place a
good deal of faith in the administration of Salicylate
of Soda, the alkalies, Iodide of Potash, Alkaline waters,
and other antirheumatic remedies.

In connection with this subject, Percival draws attention to an anatomical point in the Northumberland & Durham Medical Journal for January this year. He says, "The subdural and the subarachnoid spaces of the optic nerve may each be injected from the cranial cavity separately, in most individuals. These spaces usually cease abruptly at the scleral opening. In children, however, the subarachnoid space is sometimes continuous with the lymphatic space of the choroid. Hence, in such cases, it is not unusual for a pseudoglioma to present itself after a fever attended by delirium e.g. Meningitis. Pseudoglioma is a distension of the choroidal lymph space with exudation detaching the anterior layer of the choroid and the retina. In some few cases, the subarachnoid space is closed at the optic foramen. When this occurs, an exudation in the subarachnoid space of the brain may be checked at the optic foramen from extending forwards, and may set up a retrobulbar neuritis. Some years ago, I shewed a case in which both these conditions were present. The child, who had recovered from some form of meningitis, had retrobulbar neuritis in one eye and pseudoglioma in the other, presumably owing to the peculiar anatomical condition of her subarachnoid space that she got these curious affections in the place of double optic neuritis."
One case I saw of retrobulbar neuritis which shortly afterwards developed symptoms of cerebral tumour. In this case, I suppose, the subarachnoid space was occluded at the optic foramen, hence retrobulbar neuritis was set up, and not papillitis. I merely mention this as illustrating the importance of looking for symptoms of retrobulbar neuritis in doubtful cases of cerebral tumour where papillitis is absent.
TWO CASES of BLINDNESS
ASSOCIATED with OZAENA, DISEASE of the ETHMOIDAL & SPHENOIDAL SINUSES &c.

Three cases, similar to the above-mentioned, are quoted by Gunn in the Transactions of Ophthalmological Society (Vol.XVII. Page 117). One was published by Holmes. Holmes opened the sphenoidal sinuses and two days later there was a discharge of thick creamy pus, followed by relief of pain and improvement of vision, a central colour scotoma remaining. Brailly shewed a similar case in 1884, and Sandford exhibited a specimen in 1894, shewing disease of the sphenoidal sinuses, which had been the cause of double optic atrophy. McBride mentions a case recorded by Rouge in 1872. A lady suffered from nasal speech, then from left sided exophthalmos and strabismus, and, later on, deafness and blindness occurred. After death, pus was found in the sphenoidal sinus.

My two cases were associated with ozaena and extensive disease of the nose and accessory sinuses. In both, the sphenoidal sinuses were punctured and pus allowed to escape. In one case the patient died very soon after the onset of the blindness; the other case is still under treatment.

To understand the treatment of these cases aright, I shall discuss shortly the etiology of ozaena, and disease of the ethmoidal and sphenoidal sinuses.
True ozaena, also called Chronic Atrophic Rhinitis, and Foetid Rhinitis, is a disease which is more common in females than in males, and it usually appears about the time of puberty. There are three leading symptoms, viz: - Foetor, Crust formation, shrinkage of the nasal mucous membrane and inferior and middle turbinates, causing the nose to be "roomy".

A number of authorities consider it a general disease of the nose, the atrophied mucous membrane throwing off a secretion which rapidly dries, and after drying, becomes very offensive.

Habermann, and others, have found marked fatty degeneration of the glands of the nasal mucous membrane, and he is inclined to consider the changes in the soft parts as secondary to this cause.

Zanfal believes that in cases of ozaena the nose is arrested in growth, and that the turbinates are unnaturally small; as a consequence decomposition is favoured by diminished force of the respiratory current.

Michel first threw light on ethmoidal and sphenoidal disease, and he considers that these sinuses are always affected in ozaena, which he describes as a chronic non-ulcerative disease of the nose, with foetor, atrophy, and crust formation. In short, he holds that ozaena is secondary to disease of the sinuses.

Grunwald holds similar views on this subject.

Noebel of Zittau, and Löhnberg (Assistant) in the
Berliner Wochenschrift (Mar. 12th, 19th, 26th 1900) discuss the whole question very thoroughly. Noebel says, Ludwig Grunwald, in pointing out the relations of ethmoidal and sphenoidal disease to ozaena, has placed it in the clear light of physical investigation, and after him there is nothing more to add.

Therefore, Noebel believes in the focal (herd) theory of the disease as against its being a disease of the mucous membrane, or of the turbinates. Noebel says Grunwald's investigations have not advanced treatment, and he has some very important suggestions to make on that point.

He does not believe in heredity as a predisposing cause. He considers ozaena to be, in most cases, a residue of infectious disease e.g. Scarlet fever, Diphtheria, etc., and this explains why it is seen in more than one member of the same family. As to the foetor, he says, "just as cavities in the mastoid, lung, etc., have their odour, so have cavities of the nose, and it is due to saphrophytic parasites which produce the gases formed from the products of bone necrosis.

As regards treatment - Where there is ethmoidal disease he scrapes away the granulation tissue, diseased bone, etc., and opens the ethmoidal cells to give exit to pus.

Noebel's plan of opening the sphenoidal sinuses is worth noting - Previous to operation, he applies a
20% solution of cocaine, in nervous cases partially anesthetizes, but prefers cocaine.

Armamentarium. In addition to Grunwald's forceps and sharp spoons, he has three modifications of Bechmann's scissors; one pair of scissors curved on the flat to the right to cut through the left middle turbinate. Another pair curved to the left to cut through the right middle turbinate. Third, a pair curved on the edge down towards the fingers. Lastly, a pair of punch forceps to punch out as large an opening as possible in the anterior wall of the sphenoidal sinus. The dressings are sterilized and the nose well douchèd out.

1st Step. Separate the posterior half of the middle turbinate with the curved scissors cutting transversely through the middle turbinate. Cut away the posterior half with the scissors curved on the edge. Then haul it away with snare, or forceps, and plug the nostril. A few days after this, having first found the ostium of the sphenoidal sinus with a probe, we enlarge it with a sharp spoon so wide that a blade of the punch forceps passes into the cavity. Now snip away the bony anterior wall of the sinus until a large opening is got as in figure —

If by now caries is demonstrated, we remove the granular degenerated mucous membrane with a sharp spoon tak-
ing great care at the roof, where is the brain, and at the outer side, where is the optic nerve and the carotid artery. If the mucous membrane is not seriously diseased, leave alone, but make the opening as large as possible for it has a tendency to diminish in size, as shewn by Moritz Schmidt.

After cleansing the nose introduce Iodoform ganze into the cavity, and into the nostril a tampon saturated with europen oil, then send the patient to bed. Plug afresh daily. After the fourth day, douche with astringents. Two effects of the operation manifest themselves, viz:- cessation of headaches, and disappearance of foeter. Both are explained by the free exit of the discharge. Naturally suppuration cannot cease until the cavity is healed.

Finally, Noebel says, "all attempts to solve the disease (ozaena) from the point of view of the turbinate theory has proved unfruitful."

The focal theory has cleared up the essence of the disease from all points of view. The (herd) focal theory teaches us that the nasal suppurations - ozaena and others - depend chiefly on disease of the ethmoidal and sphenoidal sinuses, and to obtain cure or relief the treatment required is surgical, or it is without success.

Grunwald and Schäffer have been in the habit of using a trocar to puncture the sphenoidal sinuses. Their plan is described along with Case 1.

Spiess employs a Trephine.
For cleansing the nose in ozaena, after the crusts have been mechanically removed with a probe and forceps, I find nothing better than Vaselini Alba liberally put into the nostrils at bedtime and snuffed well up; the nose in the morning being well douched with a solution of Permanganate of Potash. This may be done twice in the twenty-four hours. The petroleum keeps down the crust formation, and the Permanganate solution the foc- tor. At present, I have a case of ozaena practically cured by this treatment.

I must not omit to mention Schäffer’s name in connection with sphenoidal sinus disease, for to him belongs the credit of first diagnosing chronic empyema of this cavity, and also of first treating it.

Hering, Greville Macdonald, Bronner, etc., have also observed cases.

**Case 1.** Ozaena: Necrosis of Nasal Septum: Ethmoidal and Sphenoidal Sinus disease: Brain Complication: Death.

On September 8th 1896, I saw with Dr Hunton in the Stockton Union Hospital, a youth (H.G.) aged 18, grocer’s assistant, with the following history:-

On August 31st 1896, he was grinding a tea-mill when he suddenly became unconscious and was convulsed for an hour. On September 2nd, he felt quite well again. On September 3rd, he had another fit. He experienced a cloud over both eyes and went totally blind within 48 hours.
He was removed to the Union Hospital. The temperature was 99.2. There was no albumen and no sugar in the urine. The temperature never rose above 100. He was conscious, comfortable, and had no pain whatever. The pupils were widely dilated, reacted very slightly to light, but there was no perception of light. The left disc was pale, ill-defined, and the veins over-distended. The right was slightly pale. Up to this illness he had enjoyed very good health, and there was no history of syphilis or tubercle. His heart and lungs were healthy. The opinion now formed was that of some disease about the body of the sphenoid, viz: sarcoma, or perhaps gumma. A bad odour from the patient led me to examine his nose, which was found full of greyish green ozaena crusts. He now stated that he had seen pieces like bone and skin come away from his nose. As douches were useless, the nose was cleared of crusts with probe and forceps on September 25th, and the following observed:

The nose is very roomy, turbinates much atrophied, nearly the whole of bony septum is away, horrible fetid pus is coming from the open ethmoidal cells, and the perpendicular plate of Ethmoid is necrosed and loose. We now diagnosed sphenoidal sinus disease with secondary brain mischief in the region of the optic commissure.

After free douching, cocaine was applied and the ethmoidal cells were scraped out, and part of the
necrosed perpendicular plate removed.

On October 2nd, as the patient was no better, the sphenoidal sinuses were explored after the plan of Grunwald & Schäffer, viz:- by passing a fairly large trocar along the lower border of the middle turbinate until the point came against the basisphenoid, then the point was tilted upwards and backwards so that the trocar lay across the middle turbinate and was then pushed firmly and very steadily on, until the point entered the sinus (a solid perforator with a shoulder a quarter of an inch from the point answers better). After the operation the discharge was increased in amount, and the patient felt better. Sight, however, did not improve. Later, he was put on a mixture of Potassium Iodide and Nux Vomica, and kept quietly in bed without however improving.

On October 7th, he had another fit, became unconscious, and developed facial paralysis and divergent strabismus. He sank and died three days later.

I greatly regret to state that, owing to the attitude of the authorities, a post mortem could not be made.

In this case, I think, there had been an escape of pus into the brain cavity followed later by a suppurative meningitis.

The family history was difficult to obtain, the boy's parents were dead, and he was practically without relations. He had the flattened nose usually seen in
these cases, but there were no definite indications of hereditary syphilis.

**Case 2.** K.S., of Loftus-in-Cleveland, a girl aged 14, was sent to the North Riding Infirmary, Middlesbrough, by Dr Stephen, on March 9th 1900, suffering from blindness which had come on a fortnight previously.

**Personal history.** She had inflammation of the eyes 5 or 6 years ago lasting about 3 months. The present illness began 3 months ago with a small sore in the roof of the mouth. This healed, patient then began to complain of pain in the head, and became listless and drowsy. Sight began to fail about two months later, and was entirely lost in four or five days. A discharge from the nose began three months ago and has increased.

**Family history.** Father and mother living. Father denies any specific disease. Patient is the third and only living child. The first died immediately after birth; the second lived six weeks.

**Condition on Admission.** Patient is extremely pale and unhealthy looking. Very tremulous when examined, otherwise inclined to be drowsy and sleeps a great deal. The pupils are widely dilated. Has old corneal opacities, more marked in the right eye. Not the faintest perception of light, pupils do not react to light, movements of the eyes good. Does not complain of pain. Walks easily. Nothing definite in the fun-
dus on examination with the opthalmoscope. Prescribed Iron, and ordered urine to be carefully examined. The foetor of ozaena was not detected at this examination, and nothing had been said about discharge from the nose. A few days later, I again examined the patient; she was in statu quo. Sleeps a great deal. Temperature varies between 98 and 100.

I detected a very bad odour from the patient, and observed distinct flattening of the nose. As nothing had been found in the urine, I proceeded to examine the nose and found it full of typical greenish ozaena crusts. Thinking now of the previous case, I began to suspect sphenoidal disease. I ordered frequent douching of the nose with strong Soda solution. This, however, had no effect, so I cleared the nasal passages with probe and forceps, and after douching away the stinking pus which had accumulated under the crusts, I observed the following:— There is a very large perforation of the nasal septum, in fact the whole bony septum seems to be absent. The ethmoidal region is occupied by granulation tissue covered with pus. There is dried pus on the nasopharyngeal wall. The nose is very roomy. The Inferior turbinate is hardly recognizable. The middle turbinate much smaller than normal. The movements of the soft palate are to be seen on swallowing, and one can also recognize the eustachian orifices. A further examination of the fundi, at this time, satis-
fied me that no definite changes were to be observed. No scar is to be seen in the roof of the mouth; there are no scars about the angles of the mouth. The teeth are fairly good and not peg shaped; the hearing is good. The opacities in the corneae are probably the result of interstitial Keratitis, they interfere, to a certain extent, with the examination of the fundus.

We had now two things in favour of hereditary specific disease, viz:— the corneal opacities, and the early deaths of the two other children. I accordingly prescribed Syrup Ferri Iodide & Potassium Iodide Grs. x. and decided to puncture the sphenoidal sinuses at an early date. The temperature at this time was slightly subnormal.

On April the 1st, the sphenoidal sinuses were punctured in the same manner as in the previous case, next day there was considerably more discharge from the nose, and continues so.

Examination April 7th. — Patient not so drowsy, still very pale and tremulous. Bears the cold very badly. Temperature still rather unsteady, pupils not so widely dilated but do not react to light. Patient still quite blind.

Ophthalmoscopic Examination. Optic discs shew decided change since previous examination, they are distinctly pale, evidently a commencing atrophy. Nose very clean - inspissated pus on nasopharyngeal
wall looks as if it were trickling down from the opening in the sphenoidal sinus.

In this case the differential diagnosis, of course, lies between specific disease in the region of the optic commissure and retrobulbar neuritis, due to sphenoidal disease. The evidence, in my opinion, is in favour of the latter, and I shall proceed as early as possible to enlarge the opening in the sphenoidal sinuses in the manner suggested by Noebel.

In conclusion, I would suggest that in all cases where there is a history of rapid loss of sight without any apparent cause, that a history of discharge from the nose be sought for, and the nose carefully examined.

Since writing the above the patient has died, and I now give the further history of the case with post mortem examination confirming my diagnosis.

On April the 9th, the temperature in the morning was 98°, in the evening it was 99°. On the morning of the 10th of April I passed a probe into the sphenoidal sinuses through the puncture openings previously made. Nothing further in the way of operative interference was done. The temperature on the morning of the 10th was 98.8°, in the evening 99.2°. From this date until the 13th the temperature varied between subnormal and 99.4°. On the evening of the 13th, the patient after being up in the afternoon, complained to her father,
who was visiting her, of feeling very sleepy and drowsy -
more so than usual. On the morning of the 14th, the
temperature was 101.4°, and the patient was very somno-
 lent. Later in the morning she fell into a comatose
condition from which up till late in the afternoon she
could be roused by shouting to her. In fact, this
could be done until half-an-hour before death. which took
about 4:30 P.M.
The pulse, on the morning of the 14th, was dis-
 tinctly slow - from 55 to 60 per minute. In the af-
ternoon, and shortly before death, it was very slow -
from 45 to 50 per minute. Prior to the onset of the
coma the patient had not complained of pain in the head,
or any where else, only of the drowsiness. There were
no sweatings or rigors.

Post Mortem Examination. This was performed by
the House Surgeon, Dr Donaldson, in my presence, the
head only being examined, and the following changes
will, I think, be sufficient explanation of the cause
of the blindness and fatal termination.

The base of the brain was very adherent to the
anterior fossae of the skull, and the base of the brain
corresponding to the anterior fossae was covered by a
considerable quantity of fairly firm lymph. The cribr.
iform plate of the Ethmoid and the whole of the Ethmoid
was soft and carious, the whole breaking down under
slight pressure of the fingers causing pus to exude
freely from the ethmoidal cells. The base of the
skull occupied by the sphenoid was removed, and on exam-
ing the sphenoidal sinuses the openings made by the
trocars were readily found. The anterior walls of the
sinuses were carious and soft, and the sinuses contain-
ed pus.

The whole brain was soft and puttylike in consist-
tence. Evidently the result of a general softening.
There was no fluid in the ventricles.
A CASE of BRAIN (CEREBELLAR) ABSCESS

Resulting from Middle Ear Suppuration,

The Collection of Pus in somewhat Unusual Situation

Mr L., aged 41, residing in Middlesbrough-on-Tees, had been troubled with discharge from the right ear for over a year; along with this gradually increasing deafness accompanied by occasional severe attacks of headache.

On 11th of October 1899, he saw his doctor and complained of the discharge and pain in his ear, also of severe headache, chiefly over the right side and in the occipital region, and of giddiness.

On October 30th, his doctor visited him at home and found the condition worse. The Patient has having perspirations, and the pain and giddiness were much worse. The temperature was 101° and variable, pulse 90. From this time the headache increased greatly, but the leading symptom was giddiness. Patient said it was like to "throw him off his feet", he really could not stand it, and asked to be shot it was so distressing.

On November 7th, he was admitted into the North Riding Infirmary, Middlesbrough. Temperature was 101° and pulse 120. Has great deal of pain and giddiness, also deafness. The face is pale and pasty looking, the patient is drowsy at times, at other times restless. Has tenderness on percussion over the right side of the head. Later, the patient developed right facial paralysis, and speech became slow and rather confused. The
fundi were carefully examined with the ophthalmoscope but there was no optic neuritis. There was never any tenderness over the mastoid, or thickening, or tenderness in the neck below the mastoid. The symptoms were not at all typical of brain abscess, so before proceeding to extreme measures it was decided to open up the mastoid antrum.

The mastoid was found to be sclerosed, very hard, and unusually vascular. No pus was found. We then proceeded to explore the temporo-sphenoidal lobe by trephining an inch and a quarter above and slightly behind the centre of external auditory meatus. On getting away the circle of bone, the membranes and brain were pulsating, but the parts were very tense. The membranes were incised and turned aside and a Maciewen’s canula was inserted in three different directions, but no pus found. The patient was put to bed but no improvement took place. Three days later, the following note was made - Patient more comatose, relapsing unconsciousness, temperature above normal (101) and variable. It was now decided to explore the cerebellum, this was done by trephining an inch and a quarter behind the external meatus, and quarter of an inch below Reid’s base line. This was done but no pus found. Patient died two days after.

**Post Mortem Examination.** An abscess the size of a nutmeg was found situated between the right hemisphere
of the cerebellum and the Medulla oblongata. That is, it was enclosed between the necrosed and softened lobes - Flocculus, Amygdala, and Biventral - and the Medulla oblongata.

The nerve roots in the neighbourhood of the abscess appeared softened, and there was general congestion of the meninges, also turbid lymph in the fourth and lateral ventricles. The track of the exploring canula was within half an inch of the collection of pus.

The chief features in this case were the extremely dangerous site of the abscess owing to its proximity to the Medulla oblongata, and the difficulty attending in exploring for such an abscess, and further of getting it evacuated.

The chief feature in the symptoms was the giddiness which the patient complained of as being terrible. We must also note the absence of optic neuritis, which is often found in cases of brain abscess. We must further note that the temperature and pulse were not typical, for in brain abscess the pulse is often slow and the temperature subnormal.

It is more than probable that the giddiness was due to destruction of parts of the cerebellum, which we know is the centre for equilibration.

The temperature and pulse may have been due to the proximity of the abscess to important centres in the medulla, or to some accompanying Meningitis.
Macewen was the first to operate for Cerebellar abscess as a result of ear disease, this he did in 1889. Abscess of the Cerebellum is not so frequent as abscess of the Cerebrum.

Barr states that of 75 cases of Brain abscess due to ear disease, 55 were in the cerebrum, and 13 in the Cerebellum.