Ocular palsies with nasal sinusitis

BY

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The diagnosis of nasal sinusitis as a cause of ocular palsy has recently fallen into disfavour, particularly since the importance of congenital aneurysm as a cause of third nerve palsy has been recognized.

It is the purpose of this communication to record a series of fully investigated cases of ocular palsy observed over some years, in which the palsies were associated with the presence of a chronic infection of the nasal sinuses. We made a short preliminary report of these cases in 1948.* Since then one further example has been added to the series.

Historical Review

The association of ocular palsies with nasal sinus infection has been recognized for many years. Trantas (1893) described a patient with left exophthalmos and total left ophthalmoplegia, in whom recovery from ocular palsy followed drainage of the infected sinuses, though severe visual loss resulted, with optic atrophy. Trantas mentions the occurrence of other cases of sinus disease with blindness and third nerve palsy in the literature. Further early cases in the continental literature are quoted by Onodi (1914) and Rollet (1933). In recent times the relatively few recorded cases include those with isolated third nerve palsy (Sluder, 1927; Rollet and Parthiot, 1929; Genet, 1929; Rollet, 1933; Yaskin, 1939), isolated fourth nerve palsy (Sluder, 1927; Redslob and Delbos, 1941), isolated sixth nerve palsy (Caliceti, 1927; Sargnon and Parthiot, 1930; Horgan, 1931; Diamant, 1937), and multiple ocular palsies (Sargnon, 1923; Sluder, 1927; Stieren and McKee, 1932; Yaskin, 1939; Kretschmar and Jacot, 1939). Nearly all the patients had short histories and rapid and complete recoveries following treatment of infected sinuses. The only fatal case among these was a man who died 40 days after operation of meningitis (Rollet, 1933). This patient had, in addition to ocular nerve palsy, an optic neuritis.

Pathological Anatomy

Exhaustive studies of the relation of the ocular nerves to the nasal sinuses have been made by Sluder (1913), the Onodis (1914), and Houser (1933). During the course of these nerves from the brain stem through the superior orbital fissure to the orbit, there are two sites where the anatomical relationships predispose to palsies as a result of sinus infection.

The third nerve after it emerges from the anterior end of the cavernous sinus lies in close relation to the upper part of the lateral wall of the sphenoidal and posterior ethmoidal sinuses, especially if pneumatization has extended into the attached root of the lesser wing of the sphenoid. It should be noted that the optic nerve is also in close relation here.

The fourth and sixth nerves are more laterally placed, but may be involved by extension of the infective process from the anterior or posterior groups of sinuses into the superior orbital fissure.

The sixth nerve alone is in relation to the posterior wall of the sphenoidal sinus in Dorello's canal. This canal is bounded by the posterior clinoid process, the apex of the petrous bone, and the petro-clinoid ligament, and transmits the sixth nerve and the inferior petrosal sinus. There is much variation in the extension of the sinus cavity backwards in the body of the sphenoid bone, and in some skulls there may be as little as two millimetres between Dorello's canal and the interior of the sinus cavity (Houser, 1933).

Involvement of the sixth nerve by a spread of inflammation to the apex of the petrous bone from an ear infection, as in Gradenigo's syndrome, is well known. Symonds (1944) suggested that in a proportion of cases this syndrome may result from a thrombophlebitis of the inferior petrosal sinus. In some individuals the sixth nerve reaches the lateral

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wall of the cavernous sinus by first penetrating the inferior petrosal sinus. In view of the relative frequency of sixth nerve palsy in association with otitis media and mastoiditis, it is rather surprising that it should not be encountered more frequently as a result of sphenoiditis.

**Case Material**

The cases are divided into two main groups: ocular palsies with sphenoiditis; ocular palsies with inflammation of the superior orbital fissure.

**Ocular Palsies with Sphenoiditis (Four Cases).—** These cases form a well-defined group. The patients presented with headache and double vision. They were apyrexial and did not complain of nasal symptoms, but radiological examination showed opacity of the sphenoidal sinuses and extreme rarefaction of bone around the sella turcica. Surgical drainage of the infected sphenoidal sinuses was performed, with immediate improvement in the ocular palsies, progressing to partial or complete recovery. Radiological examination after some months showed regeneration of the decalcified bone about the sella.

**Case 1.—** Mrs. F. B., aged 60 years, was admitted to Chase Farm Hospital on August 8, 1943, from the Royal London Ophthalmic Hospital. Five months previously she had noticed double vision on looking to the left; this lasted for two weeks. Three weeks before admission the double vision recurred accompanied by pain in the right side of the forehead and drooping of the right eyelid. About the same time she noticed the sight of the left eye was deteriorating. For many years she had a nasal discharge with loss of sense of smell, but about five years ago the discharge had ceased.

On examination there was bilateral anosmia. She was myopic, otherwise the fundi were normal. The visual field of the left eye showed a slight superior nasal constriction and a hemianoptic temporal defect with sparing of fixation to 3/1500 white. The visual field of the right eye was full. The left visual acuity was 6/36, and the right 6/12 corrected. There was a right partial ptosis, with absence of elevation and adduction; depression was weak. The superior oblique muscle was active. The right pupil was slightly larger than the left but reacted normally. The rest of the nervous system was normal. The blood pressure was 180/100 mm., and there was moderate peripheral arteriosclerosis.

The sedimentation rate was 17 mm. in one hour; the blood count was normal. The blood Wassermann reaction was negative, and the cerebrospinal fluid was normal. Radiological examination showed erosion of the middle of the dorsum sellae, floor and neighbouring parts of the sphenoid, with generalized opacity of the nasal sinuses.

On September 2, 1943, a bilateral Caldwell-Luc operation was performed by D.G.P. with free drainage of pus from the antra, ethmoids, and sphenoidal sinuses. Culture grew haemolytic streptococci. Sulphathiazole was given post-operatively.

On September 6 the ocular movements had improved and ten days after operation were full, apart from slight ptosis. The visual field of the left eye had expanded and the left visual acuity was 6/12 corrected. Radiological examination a month after operation showed some re-formation of the dorsum sellae.

Five years later the patient was well, and the sella turcica was normal apart from slight irregularity of the floor.

Although there were no recent nasal symptoms, a sphenoiditis and pansinusitis produced a right third nerve palsy complete except for the pupillary fibres. There was interference chiefly with the nasal fibres of the left optic nerve. Recovery resulted from drainage of the infected sinuses.

**Case 2.—** Martin W., a man aged 46, was admitted to Chase Farm Hospital on October 10, 1946, from the Royal London Ophthalmic Hospital. He complained of attacks of shooting pains in the right temple for the past six months. One month before admission he started to see double and the right eyelid drooped. There was no history of nasal discharge.

On examination there was no anosmia. The fundi, fields, and visual acuity were normal. The right pupil reacted sluggishly to light and accommodation. There was a right partial ptosis with weakness of elevation, adduction, and depression. Two weeks after admission a weakness of the right superior oblique muscle developed. No clinical evidence of sinus infection was detected.

The sedimentation rate was 12 mm. in one hour, the blood count normal. The blood Wassermann reaction was negative, and the cerebrospinal fluid was normal. Radiological examination showed that the dorsum and floor of the sella were destroyed, with an opacity of the sphenoidal sinus. Bilateral carotid arteriography and air encephalography demonstrated no abnormalities.

On December 17, 1946, at the Brentwood Annexe of the London Hospital the sphenoidal sinus was explored by Mr. Keogh. The posterior part of the bony roof of the sinus felt soft. The sinus was filled with 4 ml. of dark brown fluid under pressure containing pus cells, and on culture grew Staphylococcus pyogenes.

Three months after operation some weakness of elevation and depression remained, but the other movements were full, and there was no ptosis. Radiological examination a year later showed partial re-formation of the floor of the sella.

Paresis of the right third and fourth nerves was produced by an infected cyst of the sphenoidal sinus. There were no nasal symptoms. Partial recovery occurred after drainage of the sphenoidal sinus with re-formation of the bone.

**Case 3.—** J. S., a man aged 60 years, was admitted to the London Hospital on November 10, 1947. Nine months previously he had noticed double vision for
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On October 29, 1947, Mr. Bowen-Davies performed operative drainage of the sinuses. Polypoid mucosa was found in the posterior ethmoidal cells. The left sphenoidal sinus contained some curious material almost of the consistency of dark brown plasticine. The histological changes were those of a chronic pyogenic inflammation.

On November 5, 1947, there was no diplopia and the ocular movements were full. On radiological examination two months after operation the sella was seen to be re-forming (Fig. 1b). Six months later the patient had remained well.

There was a history of catarrh, although there was no clinical evidence of sinus infection. The partial left third nerve paresis recovered after drainage of the infected sphenoidal sinus, and the eroded sella re-formed.

Case 4.—Maurice W., a man aged 45, was admitted to Chase Farm Hospital on November 14, 1942, from the London Hospital. He complained of generalized headache and double vision for two months. He had noticed a blood-streaked, post-nasal discharge for ten years. He was a treated diabetic.

On examination there was no anosmia. The fundi and pupils were normal. There was diplopia on looking up and to the right, suggestive of a left inferior oblique paresis. There was no clinical evidence of sinusitis. Radiological examination showed destruction of the floor of the sella turcica, with opacities of very large sphenoidal sinuses and the maxillary sinuses (Fig. 1a).

On examination there was a right external rectus paresis; otherwise the nervous system was normal. There was no clinical evidence of nasal sinus infection. The blood Wassermann reaction was negative, and the cerebrospinal fluid was normal. Radiological examination of the skull and sinuses showed dullness of the sphenoidal sinuses with erosion of the floor of the sella turcica together with the posterior clinoid processes suggestive of a neoplasm of the sphenoidal sinuses (Fig. 2a).

Fig. 1a (Case 3).—Rarefaction of sella turcica; opacity of sphenoidal sinus.

three weeks, and a dull aching pain in the left temple. The double vision recurred in May and September, 1947, and lasted three weeks on each occasion. There was a history of chronic nasal discharge since a blow on his nose by a cricket ball in 1912. In 1920 a nasal polyp was removed.

On examination there was no anosmia. The fundi and pupils were normal. There was diplopia on looking up and to the right, suggestive of a left inferior oblique paresis. There was no clinical evidence of sinusitis. Radiological examination showed destruction of the floor of the sella turcica, with opacities of very large sphenoidal sinuses and the maxillary sinuses (Fig. 1a).

Fig. 1b (Case 3).—Regeneration of sella; clear sphenoidal sinus (two months later).

Fig. 2a (Case 4).—Absorption of bone of sella turcica.
On March 31, 1943, the ethmoidal and sphenoidal sinususes were explored by D.G.P.; mucous discharge and hypertrophied polypoid mucosa were found. On section this showed granulation tissue infiltrated with polymorphonuclear and plasma cells. Culture grew *Coccobacillus faritidus* ozenae.

There was an improvement of abduction of the right eye three days after operation, and by the twelfth post-operative day the diplopia had cleared. Occasional diplopia recurred over a year, but during the next five years recovery was complete. Radiological examination seven months after operation showed recalcification of the sphenoid (Fig. 2b).

**FIG. 2b (Case 4).—Regeneration of sella (seven months later).**

Sphenoiditis with destruction of bone was present without nasal symptoms apart from some post-nasal catarrh. It must be admitted that the diagnosis of the cause of the sixth nerve paresis was complicated by the presence of diabetes, but permanent recovery appeared to be related to the drainage of the infected sphenoidal sinus.

**Discussion**

Chronic sphenoiditis may be present in the absence of nasal symptoms, although a past history of a period of nasal discharge was usually obtainable from these patients. Cessation of discharge was probably due to blockage of the ostium of the sinus. The increase of tension in the sinus finally produced erosion of its bony wall with the development of ocular palsies. The palsies might be intermittent at first due to changes in tension of the contents of the sinus. Hermann and Hall (1944) have described the case of a patient with periodic headache associated with a partial third nerve palsy due to mucocele of the sphenoidal sinus.

Relative sparing of the pupillary fibres in these patients (Cases 1, 2, and 3) was probably fortuitous. Hermann and Hall found ptosis and an inactive pupil in their patient. Rollet (1933) and Yaskin (1939) also described internal ophthalmoplegia due to sphenoiditis.

Certain clinical features may assist in the differentiation of ocular palsies due to sphenoiditis from those due to supraclinoid aneurysms. The unilateral frontal pain was aching in character and was not intense. The palsy did not develop simultaneously with the pain. There were no symptoms, such as stiffness of the neck, suggestive of a leaking aneurysm. The third nerve palsies were incomplete and other cranial nerves, the second (Case 1) and the fourth (Case 2) were involved. Jefferson (1947) has stated that it is the completeness of an isolated third nerve palsy that is so characteristic of an aneurysm of the posterior communicating artery.

The symptomatology of carcinoma of the naso-pharynx has been studied by Godtfredsen (1947) who found that the sixth nerve was frequently first affected, then the contralateral sixth, and the trigeminal nerve. Palsies of the third and fourth nerves were infrequent.

The appearance of an ocular palsy due to disseminated sclerosis in a patient with sinus infection may be misleading. The patients under consideration were old for the onset of this disease; other signs of disseminated sclerosis were lacking and did not develop during the period of observation.

Neurosyphilis was excluded by the negative serology.

Radiological appearances of pansinusitis may clarify the diagnosis; but if the sphenoidal sinususes only are opaque, with erosion of the sella, exploration is necessary to determine the nature of the lesion.

The site of the lesion of the third nerve in these patients was probably at the medial end of the superior orbital fissure, and of the sixth nerve in Dorello's canal, associated with erosion of the posterior clinoid processes.

**Ocular Palsies with Inflammation of the Superior Orbital Fissure (Six Cases).—**These cases corresponded generally with the *syndrome de la fente sphenoidale* described by Rollet (1933) and Kretzschmar and Jacot (1939), and orbital periostitis (Cairns, 1938; Collier, 1922). The patients presented with unilateral headache, and double vision due to multiple ocular palsies. Proptosis, generally
slight, was present in all. Unilateral papilloedema, trigeminal nerve involvement, and intracranial spread of inflammation were seen in a varying degree. Nasal symptoms were not prominent, but all showed radiological evidence of sinusitis at some period of their illness.

Case 5.—Mrs. L. S. aged 46 years was admitted to the Maida Vale Hospital for Nervous Diseases, London, on April 9, 1947, from the Moorfields, Westminster and Central Eye Hospital (Royal London Ophthalmic Hospital).

Nine weeks previously she had noticed protrusion of the right eye and drooping of the lid. She then started to have an aching pain on the right side of the forehead. Although she had a past history of catarrh there was no recent nasal discharge.

On examination there was no anosmia. The fundi and visual fields were normal. The right visual acuity was 6/9, left 6/6 uncorrected. The right eye was proptosed downwards and forwards. There was partial right ptosis, with weakness of elevation and adduction. The pupils were normal. The rest of the nervous system was normal. The blood pressure was 220/120 mm., and the heart moderately enlarged.

The sedimentation rate was 11 mm. in one hour, and the white count was normal. The blood Wassermann reaction was negative, and the cerebrospinal fluid normal in pressure and constituents.

Radiological examination of the skull showed no abnormality of cranium or orbit. Thickened mucosa or fluid was present in the right antrum, and fluid in the left antrum.

On April 12, 1947, an oval mass about 2 cm. × 0.5 cm. was detected in the posterior part of the orbit, which was thought to be inflammatory and related to sinus infection. Some improvement followed antral puncture and conservative treatment. Subsequently the proptosis increased, and oedema of the lids appeared. Although the fundus and visual field were normal, the right visual acuity deteriorated to 6/18. Radiological examination now showed a patch of osteitis in the roof of the orbit (Fig. 3). The condition was considered to be inflammatory by Mr. Wylie McKissock.

A radical frontal sinus operation was performed by Mr. Howells in July, 1947, at Slough Emergency Hospital; chronic frontal sinusitis was found with necrosis of the anterior wall of the sinus. The patient made a satisfactory recovery.

Maxillary and frontal sinusitis without recent nasal symptoms was diagnosed. There was proptosis, partial right third nerve palsy, and deterioration of right visual acuity due to an inflammatory lesion in the posterior part of the orbit. Recovery took place with drainage of the frontal sinus.

Case 6.—C. C., a boy aged 12, was admitted to the London Hospital on August 26, 1946.

He had complained of headaches for some months. Three weeks before admission he became drowsy, and vomited several times. It was noticed that his right eye was prominent, and he began to see double.

On examination there was a tender swelling in the right temporal fossa extending over the zygoma anterior to the pinna. The right side of the face was warm and flushed. There was papilloedema of the right optic disc, the left disc was normal, and the visual fields were full. There was proptosis of the right eye with incomplete ptosis, weakness of elevation and adduction, and absence of abduction. The rest of the nervous system was normal.

The sedimentation rate was 36 mm. in one hour. The white count was normal. The cerebrospinal fluid pressure was normal in pressure and contained protein 50 mg. per 100 c.c.m., no pleocytosis; Lange curve 12322100. Radiological examination of the skull showed translucency of the lateral wall of the orbit on the right side (Fig. 4a), suggesting infection. There was extensive loss of bone in the posterior and lateral walls of the orbit and destruction of the floor. Both maxillary sinuses showed opacities, suggesting thickened mucosa. There was a roughly spherical opacity in the upper part of the right antrum adjacent to the localized area of erosion in the lower and outermost part of the greater wing of the sphenoid (Fig. 5a).

The patient recovered spontaneously during the next two months and a year later passed the eye examination for a navigation school. Radiological examination 12 and 17 months later showed progressive regeneration.

Fig. 3 (Case 5).—Patch of osteitis in the posterior part of the roof of the right orbit.
of the orbital wall (Fig. 4b), but the appearances of mucosal thickening in the antra persisted (Fig. 5b).

This patient developed a subacute inflammation of the soft tissues and bony wall of the orbit, including the outer wall common with the temporal fossa, and the lateral extremity of the superior orbital fissure. The second, third, and sixth cranial nerves were involved, probably at the apex of the orbit. Apart from an increase of protein in the cerebrospinal fluid, there was no evidence of intracranial extension. Although there were no symptoms of nasal infection, the peculiar radiological appearance of the right antrum was very suggestive of a connexion between the condition of the antrum and the osteitis of the orbit. The sinusitis may, however, have been contemporaneous rather than causative.

Case 7.—A. L., a man aged 49, was admitted to Chase Farm Hospital on March 7, 1946. Three months previously he had had a bad cold, with
pain in the left forehead and behind the eye, radiating over the left side of the head. Two days after the onset of pain he began to see double. The nasal discharge suddenly ceased but the pain and double vision persisted. Chronic catarrh had been present since childhood, and four years before he had undergone an operation for deflected nasal septum. Radiological examination shortly before admission showed a faint opacity of the left maxillary sinus.

On examination there was no anosmia. The fundi and pupils were normal. There was slight proptosis of the left eye, with partial ptosis, weakness of elevation and absent abduction. Appreciation of pinprick was impaired over the distribution of the first and second divisions of the left trigeminal nerve. There was a right lower facial weakness, with increased reflexes in the right arm and leg, and the right plantar response was equivocal.

The sedimentation rate was 22 mm. in one hour. The cerebrospinal fluid was normal in pressure and contained red blood cells. Ventriloculography showed some increase in the subarachnoid space but no other abnormality. The ventricular fluids were also slightly bloodstained.

Radiological examination on March 30, 1946, showed the right maxillary antrum to be semi-opaque, and an appearance of the right frontal sinus suggestive of infection. He had complained of extension of pain to the right orbit and eyeball.

The patient improved spontaneously, but in June, 1946, he had a recurrence of left-sided pain and nasal discharge. There was still slight left-sided proptosis, but the ocular movements were full. The reflexes were brisk and equal. When the patient was seen 15 months later there was no proptosis.

The onset of symptoms was associated with an exacerbation of chronic nasal discharge, which then dried up. The left antrum showed radiological evidence of infection. There was a partial lesion of the left third nerve, involvement of the sensory fibres of the trigeminal, and a complete sixth nerve palsy. The cause of the contralateral pyramidal signs was obscure. The presence of blood in cerebrospinal fluid in the lumbar sac and in the ventricles may have been due to an insidious intracranial venous thrombosis. The course of the illness was unlike a subarachnoidal haemorrhage due to aneurysm. The patient was considered to be suffering from a low-grade infection of the tissues around the left superior orbital fissure, and possibly leptomeningitis.

Case 8.—S. F., a man aged 18, was admitted to Chase Farm Hospital from the London Hospital on February 25, 1942.

Six weeks earlier he had developed right frontal headache, with swelling of the right eyelids, right ptosis and double vision. He had a cough, felt shivery, slept poorly, and sweated at night. Pyrexia was absent, but he had been treated with sulphonamides. Radiological examination on February 11 showed opacity of the right maxillary antrum and sphenoidal sinus.

On examination there was no anosmia. The right optic disc was congested; the right visual acuity was J2, the left visual acuity J1. There was irreducible proptosis of the right eye, with partial ptosis, absence of elevation and abduction, and impaired depression. The right pupil was smaller than the left but reacted normally. The rest of the nervous system was normal. The right nasal cavity was congested, but no pus was obtained on puncture of the right maxillary antrum. Radiological examination of the skull showed no abnormality.

On March 2 the patient developed severe pain and relative hypalgesia with diminished corneal response in the right trigeminal area. The right pupil became sluggish. He had a series of fits of an uncinate type with hallucinations of taste and smacking of the lips.

At repeated examinations, the cerebrospinal fluid was normal in pressure and showed an increase of cells from 30 to 230 per c.mm. (40% polymorphonuclears and 60% lymphocytes), protein 40-60 mg. per 100 c.cm. It was sterile on culture. A ventriculogram was normal.

The white blood count increased from 5,000 per c.mm. (polymorphonuclears 40%) to 13,800 per c.mm. (polymorphs 68%). On March 6 several more uncinate attacks occurred, and on March 7 a right frontal craniotomy and decompression was performed by Mr. Northfield. In the region of the superior orbital fissure the dura mater was grossly indurated, and had a granular external surface. Biopsy showed fibrous granulation tissue, and the culture grew a small clump of Staphylococcus albus only. No abscess was found.

On March 17 the right fundus showed a secondary optic atrophy and there was no perception of light. There was a complete right external and internal ophthalmoplegia.

On May 30 the patient suddenly developed a spastic left hemiplegia associated with pyrexia. Ventriloculography showed displacement of the ventricles to the left. The craniotomy was reopened but only much thickened dura was found in the anterior and middle cranial fossae. He became aggressive with highly organized delusions and was admitted to Napsbury Mental Hospital for five months.

In the following year he returned to heavy work. He had some left-sided fits preceded by an olfactory aura, but these were controlled by phenobarbitone.

In October, 1943, the right visual acuity was perception of light only, there was a right secondary optic atrophy, the ocular movements were almost full, and the left hemiplegia had recovered. During the next five months the visual acuity of the left eye deteriorated from 6/9 to 6/18. On examination there was left anosmia, left primary optic atrophy with a paracentral scotoma, and an upper left temporal defect. On April 13, 1944, the chiasmal region was explored through a left frontal craniotomy by Mr. Northfield. Where the left optic nerve entered the optic canal dense, fleshy adhesions were found, binding down the region of the gyri recti, the olfactory tract and the optic nerve to the surface of the dura mater. The adhesions round the optic nerve were divided and the optic canal was decompressed.
Radiological examination in March, 1944, showed residual destructive changes on the right side, with erosion of the lesser wing of the sphenoid (Fig. 7a), upper border of the optic foramen (Fig. 6a), and enlargement of the superior orbital fissure (Fig. 7a).

In 1948 the patient was working as a packer. He still had olfactory hallucinations but no fits. The left optic disc was pale and the left visual acuity was 6/12 corrected. There was a right secondary optic atrophy with P.L. only. The ocular movements were full without proptosis. Radiological examination showed some enlargement of the right superior orbital fissure but the definition of its outer border had improved (Fig. 7b). There was no deficiency in the outline of the optic foramina but the right (Fig. 6b) was smaller than the left (Fig. 6c).

Radiological evidence of infection of the right maxillary and frontal sinuses was present at the onset of the illness, though clinical effects were probably masked by sulphonamides.
The right second, third, fourth, and sixth cranial nerves, the fifth to a less extent, and possibly the ocular sympathetic were involved in the inflammatory process at the apex of the orbit. Intracranial complications consisted of uncinate attacks and later mental changes due to the thickened adherent dura in the middle and anterior cranial fossæ. The sudden onset of a contralateral hemiplegia suggested a vascular element in the spread of the disease.

The cerebrospinal fluid was normal in pressure and constituents. Radiographs of the skull and an air encephalogram showed no abnormality, but on left carotid arteriography there was delay in the passage of the opaque medium above the carotid syphon. Exploration was undertaken to exclude the presence of a sphenoidal ridge meningioma.

On June 5, 1946, a left frontal craniotomy was performed by Mr. Northfield. No abnormality of the brain was found. The dura mater was thickened and reddened at the medial part of the sphenoidal ridge, and there were some fine adhesions between the dura and arachnoid. The day following operation the patient had a number of right-sided attacks and became unrousable with a pyrexia of 101° F. Re-exploration revealed some hemorrhagic swelling of the brain. The fits continued, and two days later she was still deeply unconscious with a right flaccid hemiplegia. On recovery of consciousness she was aphasic.

A month later she had slight dysphasia with increased reflexes in the right upper limb. The ocular signs were unchanged. Radiological examination now showed clearing of the left ethmoidal sinuses.

Gradual improvement occurred and by November, 1947, though she complained of diplopia, the left ocular movements appeared full. There was slight proptosis. The right-sided pyramidal signs had recovered.

In the absence of clinical evidence of sinus infection, the significance of the radiological findings was uncertain. There appeared to have been sinusitis present shortly after the onset of symptoms, which was more marked on the side of the affected eye. Oedema of the lids, left proptosis, and a partial third and sixth nerve paresis developed slowly. Exploration revealed the presence of inflammatory changes on the meninges adjacent to the left superior orbital fissure. The post-operative complication of a sudden right hemiplegia preceded by right-sided fits was thought to be due to a left venous thrombosis, though the exposed cortical veins appeared normal.

Case 9.—Mrs. M. T. was admitted to Chase Farm Hospital on May 4, 1946.

Fourteen months earlier her left eye had become prominent, and about this time she began to see double. She complained of a dull pain in her temple and a feeling of "something pushing" behind the left eye. She had no nasal symptoms. She suffered from recurrent polyarthritis affecting the small joints of the hands and the knees. Radiographs taken four months after the onset of symptoms showed well-marked changes of maxillary sinusitis, more on the left side, and left ethmoidal sinusitis.

On examination there was no anosmia. The fundi and pupils were normal. The left upper lid was oedematous; the left eye was proptosed downwards and forwards. There was weakness of abduction and elevation. The rest of the nervous system was normal.
pressure and constituents. Radiological examination of the skull and sinuses at this time showed no abnormality apart from some absorption of the dental alveoli.

On November 5 and 9, the patient had a series of dental extractions, and after the second operation developed an external rectus palsy of the right eye.

A right carotid arteriogram showed no abnormality. Right frontal craniotomy was performed on November 21 by D.G.P. to exclude the presence of neoplasm in the middle cranial fossa. A free exposure of the middle and anterior cranial fossae showed no abnormality of the brain or adjacent structures except for a definite bulging of the lateral wall of the cavernous sinus, suggesting localized thickening of the dura.

By March, 1943, the ocular movements had improved, but the right eye was still displaced downwards. The patient complained of persistent pain in the right cheek and in June, 1943, was readmitted to Chase Farm Hospital.

Examination on June 30 showed papilledema of the right optic disc. The eye was displaced downwards, and there was diffuse swelling of the right upper lid and of the outer two-thirds of the superior orbital margin. The ocular movements were full apart from right ptosis. On July 22, 1943, the right gasserian ganglion was injected with alcohol. Unusually tough resistance was detected on passage of the needle through the foramen ovale. There was considerable relief of pain. Radiological examination in July, 1944, showed a slight haziness of the right maxillary antrum.

In May, 1945, the vision of the right eye became misty. There was slight papilledema with reduction of visual acuity to 6/36. The visual field showed a generalized peripheral constriction with a central scotoma to 5/2000 white. The vision continued to deteriorate in this eye and during the next year was reduced to 6/60. The right optic disc showed a secondary atrophy. In 1947 the patient’s condition was unchanged.

At the time of onset of the right-sided facial pain the patient had radiological evidence of maxillary sinusitis which was seen later on the right side only. A year later he developed right proptosis and papilledema, a partial right third nerve paresis, a sixth nerve palsy, and pyramidal signs in the left upper limb. The sixth nerve palsy may have resulted from dental extraction. At craniotomy the appearances were suggestive of thickening of the lateral wall of the right cavernous sinus. Right papilledema proceeded slowly to secondary optic atrophy. Persistent pain in the distribution of the right trigeminal nerve was relieved by injection of the gasserian ganglion. The dura was abnormally resistant to the passage of the needle.

Discussion

The course of the infection in all these patients was subacute or chronic. There appeared to be a high resistance to the infecting organisms, and as the inflammatory process subsided, complete or partial recovery occurred in every patient. Taylor (1949) has commented on the high natural resistance to intracranial infection developing from the sinuses or middle ear.

Unilateral proptosis and edema of the eyelids were present for weeks or months with frontal headache, pain behind the eye, and double vision. Paralysis of the external ocular muscles on the affected side, especially of the elevators and abductors, developed, sometimes with papilledema proceeding to optic atrophy, and slight hypalgesia in the distribution of the first and second divisions of the trigeminal nerve. The condition might recover spontaneously (Case 6), or after drainage of the nasal sinuses (Case 5). Intracranial extension occurred in four patients (Cases 7, 8, 9, 10) with severe trigeminal pain in three (Cases 7, 8, 10), contralateral pyramidal signs, and in one patient (Case 8), uncinate attacks.

The portal of entry of the infection was speculative in five patients (Cases 6, 7, 8, 9, 10). There were no symptoms or signs of acute nasal infection, but the onset of double vision in one patient (Case 7) was associated with sudden suppression of a chronic nasal discharge. Radiological evidence of antral infection was difficult to assess when it consisted of opacity without a fluid level. All that can be said is that sinusitis had been present in every patient at the time of the illness or earlier, and that it appeared worse on the affected side. Exacerbation of ophthalmoplegia followed dental extraction in one patient.

Pathological evidence of infection as shown by an increased sedimentation rate, leucocytosis or changes in the cerebrospinal fluid, was infrequently present.

The inflammatory nature of the lesion was detected by radiological examination or at operation in four cases. In one patient (Case 7) diagnosis depended on relationship of symptoms to nasal discharge, the presence of a raised sedimentation rate and the spontaneous recovery. In one (Case 10) diagnosis rested on the findings at operation in conjunction with the clinical course of the disease observed over several years.

Radiological changes consisted of erosion of bone in the orbital roof (Case 5), destruction of the walls of the orbit (Case 6), and of the lesser wing of the sphenoid and optic foramen (Case 8). Recovery was associated with regeneration of bone, and in one case with reduction in size of the optic foramen (Case 8).

Craniotomy was performed on three patients. In two (Cases 8, 9) pachymeningitis was found in the region of the sphenoidal ridge and middle cranial fossa. When the infection was prolonged over years (Case 8) extension to the opposite side
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occurred. Distortion of the wall of the cavernous sinus and toughness of the dura mater at the foramen ovale suggested the presence of thickening of the dura in a third case (Case 10).

The diagnosis of a chronic infection in the region of the superior orbital fissure is not easy; air studies, arteriography and exploration may be necessary to exclude the presence of other lesions. The idiopathic or "rheumatic" form of orbital periostitis as described by Collier (1922) was characterized by the rapid onset of the ocular palsies, with pain and proptosis. Recovery was usually complete in a few months and intracranial complications did not occur.

The production of proptosis and ophthalmoplegia by a non-fistulous carotid aneurysm in the anterior part of the cavernous sinus has been discussed by Jefferson (1938). Pain was sudden in onset. The ophthalmoplegia tended to be complete and there was dense anaesthesia of the first and second division of the trigeminal nerve. Godtfredsen (1947) considered that exophthalmos and ophthalmoplegia due to penetration of the orbit by a nasopharyngeal growth occurred late and was associated with a poor general state. A meningioma arising from the medial third of the sphenoidal ridge may produce proptosis and ophthalmoplegia, but pressure on the optic nerve usually results in primary optic atrophy with a nasal hemianopia. Hyperostosis of the sphenoidal ridge may be detected on radiological examination. Eosinophilic granuloma of bone is a rare lesion occurring mainly in children and adolescents. It is considered to be related to Hand-Schüller-Christian disease (lipogranulomatosis), "apparently represents a peculiar inflammatory reaction to some as yet unknown agent of infection" (Jaffe and Lichtenstein, 1944), and may recover spontaneously. In the calvarium the lesion presents radiologically as a translucent "punched-out" area. Hill (1949) states that periosteal new bone formation in this condition is usually slight and appears to be confined to the shafts of the long bones. This is in contradistinction to the well-marked radiological appearances of periosteal new bone formation during the stage of recovery in two of our patients (Cases 6, 8). Benign giant cell tumour, reviewed by McNerney (1949) is another rare lesion which may affect the base of the skull; as with other neoplasms this is, untreated, a progressive condition, and the radiological appearance is purely that of bone destruction, with a clear-cut edge. In unilateral exophthalmic ophthalmoplegia and pseudo-tumour of the orbit pain is absent. Insidious thrombosis of the cavernous sinus is usually associated with congestion of the orbital and angular veins and signs of extension to the opposite sinus.

Whether any degree of sinus or venous thrombosis was present in these patients requires consideration. The occurrence of multiple cranial nerve palsies in the posterior fossa due to inferior petrosal sinus thrombophlebitis complicating otitis media has been described by Symonds (1944), though in an earlier discussion of these cases (1927) extradural inflammation was postulated. Extensive postmortem studies recorded by Byers and Hass (1933) and Courville and Neilson (1934, 1935, and 1937) supported the hypothesis of a thrombophlebitis. The multiple cranial nerve palsies in our patients can be accounted for by the presence of local inflammation in the superior orbital fissure, sometimes with osteitis and pachymeningitis. It appears that this fissure may act as a channel for the spread of inflammation into the cranial cavity. In these patients the nasal sinuses provided a possible site for the origin of the infection. The development of an external rectus palsy after dental extraction in a patient with a partial third nerve palsy may have been due to an extension of the local inflammatory lesion or to a spreading thrombophlebitis from the tooth socket. Brain (1947) has described the occurrence of ocular palsies with trigeminal involvement following extraction of a tooth from the upper jaw.

The nature of the lesion causing contralateral pyramidal signs in the last four patients was not clear. In Case 8 there was evidence at craniotomy of the direct spread of infection in continuity to the brain, but the sudden onset of hemiplegia suggested a vascular lesion.

In Case 9 the patient developed a hemiplegia with fits as a post-operative complication. There was some haemorrhagic swelling of the brain but the surface vessels appeared normal. In two patients (Cases 7 and 10) contralateral pyramidal signs were detected on clinical examination. In one patient (Case 7) blood was present in the lumbar and ventricular cerebrospinal fluids due perhaps to the presence of vascular congestion from an insidious cerebral venous thrombosis. In all these patients the pyramidal signs disappeared over a few months.

Symonds (1937) described the occurrence of focal epilepsy, and hemiplegia (with recovery, and without brain abscess formation) in cases of otitis media, and brought clinical evidence for regarding this as due to thrombophlebitis involving the superficial cerebral veins. The last four cases described here may be comparable, though craniotomy in three of them failed to provide visible evidence of venous thrombosis.

No definite recommendations can be made on the treatment of chronic infections in the region of the
superior orbital fissure. Sulphonamides and antibiotics may have an application in the earlier stages of the infection. Drainage or any other local treatment of the sinuses is indicated only if an active infection persists there. Intracranial exploration may be necessary for diagnosis, but vascular complications may result. Decompression of the optic canal and freeing of meningeal adhesions of the optic nerve may be necessary to arrest the development of optic atrophy and visual failure.

Summary

Ten cases of ocular palsy associated with nasal sinusitis, which fall into two clinical groups, are described.

The first group is composed of four patients with palsy of one or two ocular nerves associated with chronic sphenoiditis, and destruction of bone in the region of the sella turcica. Recovery and regeneration of bone followed drainage of the infected sinuses.

The second group consists of six patients with ocular palsies and signs of a subacute or chronic inflammatory lesion in the region of the superior orbital fissure. All showed radiological evidence of sinusitis on the affected side at some time during their illness. Destruction of bone in the region of the superior orbital fissure with later recovery of bone density was demonstrated radiologically in three cases. Involvement of the second and fifth cranial nerves, meninges and cerebrum might occur in the course of the disease.

Resistance of the tissues to the invading organisms appeared high. No deaths occurred. All the patients recovered wholly or in part with subsidence of activity over a long follow-up.

The diagnosis and treatment are discussed.

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