

ous system and on infant behavior.^{15,16} Our study demonstrates short-term influences on newborn behavior, caused by changes in the composition of single feedings.

We designed our formulas to maximize the effects on tryptophan transport into the brain, yet we offered newborns amounts of tryptophan or valine that may be present in human milk. We believe that the changes observed in sleep behavior could have been caused by changes in serotonin synthesis and neurotransmission within the newborns' brains.

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SPHENOID SINUSITIS

A Review of 30 Cases

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Abstract We studied 30 patients with infectious sphenoid sinusitis (15 acute cases and 15 chronic cases) in an effort to characterize the clinical presentation, bacteriology, and associated complications of this frequently misdiagnosed infection. Severe frontal, temporal, or retro-orbital headache that radiated to the occipital regions or pain in the trigeminal (V₁ to V₃) distribution or both were the most prominent presenting symptoms. In acute cases, purulent exudate was frequently seen in the middle and superior nasal turbinates. Computerized axial tomography or sinus tomography and cannulation of the sphenoid sinus proved to be the most useful diagnostic studies.

SPHENOID sinus infection is frequently misdiagnosed on initial evaluation.¹ Situated deep in the apex of the nasal cavity, the sphenoid sinus is not accessible to direct clinical examination and is often not seen clearly on routine radiologic examination. The walls of this sinus are adjacent to the pituitary gland, optic canals, dura mater, and cavernous si-

nus, which contain the internal carotid arteries and the third, fourth, fifth, and sixth cranial nerves (Fig. 1). The maxillary division of the fifth nerve may also indent the wall of the sphenoid sinus. The sphenoid walls can be extremely thin; sometimes bone is absent, and adjacent structures are separated from the sinus cavity only by a thin mucosal barrier. Because of the close proximity of the sinus cavity to the cortical venous system, cranial nerves, and meninges, infection of the cavity may spread to these structures, mimic other neurologic diseases, and result in serious complications.²⁻⁴ In the preantibiotic era,

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Teed noted that the sphenoid sinus was involved in 15 to 33 per cent of cases of sinusitis. He also emphasized the high incidence of intracranial infections and high mortality associated with infection of this sinus.⁵

Since 1948, there have been only a few reports of infections of the sphenoid air space in the otolaryngology literature.⁶⁻¹² As a result, many internists and family physicians are unfamiliar with this disease. We describe 30 cases of sphenoid sinusitis treated in our hospitals, in order to emphasize the clinical presentation of this infection in the antibiotic era, as well as review the bacteriology and radiologic criteria for diagnosis and optimal management of the disease.

METHODS

All cases evaluated were seen between 1968 and 1980 at the Massachusetts Eye and Ear Infirmary or Massachusetts General Hospital and were diagnosed as sphenoid sinusitis. Only cases fulfilling the following criteria were included in the study: clinical symptoms (headache, fever, nasal drainage, and so forth), positive radiographic findings, positive sphenoid bacterial cultures, and biopsy specimens demonstrating acute or chronic inflammation of the sinus mucosal lining (23 patients), or clinical and radiologic findings compatible with sphenoid sinusitis followed by clinical and radiologic improvement with antibiotics (7 patients).

Sinusitis was considered acute if the duration of symptoms was less than one month; chronic symptoms were those that persisted for more than one month. The diagnosis of cavernous sinus thrombosis was confirmed by angiography (in three cases) or autopsy (in three cases). All patients with cavernous sinus involvement had deficits of one or more of the cranial nerves passing through the cavernous sinus.

Sphenoid surgery was performed by an external ethmoidal approach in 18 patients and by an intranasal approach in 2, as previously described.⁴ The surgery included removal of the front facet of the sphenoid and drainage of the sinus. Cannulation of the sphenoid sinus was performed as previously described, after administration of 4 per cent cocaine solution to reduce the size of the turbinates for visualization of the ostium.⁴

Bacteriology

Purulent material from the sphenoid sinus was transported to the laboratory and planted within a median time of 45 minutes on Brucella agar plates with 5 per cent horse blood, on cooked-meat medium, on prereduced anaerobic modified Columbia sheep-blood agar, and also on prereduced anaerobic laked sheep-blood agar with kanamycin and vancomycin. The anaerobic plates were incubated in Gas Pak jars. The bacteria isolated were identified by standard methods, and the antimicrobial sensitivities of the sinus isolates were determined by the method of Bauer-Kirby.¹³

Radiologic Evaluation

Twenty-nine of the patients with acute or chronic sphenoid sinusitis were studied radiologically. In one patient, the diagnosis was made at autopsy without radiographs. In the majority of cases conventional sinus films were obtained, including the anterior-posterior, Waters', and lateral views,¹⁴⁻¹⁷ but in a few cases only plain skull films with limited views of the sphenoid sinus were available. In addition, most patients underwent anterior-posterior and lateral tomographic studies. Two criteria were used to define sphenoid sinus involvement: increased opacification of the sinus (localized or diffuse), and increased sclerosis of the bony walls of the sinus.

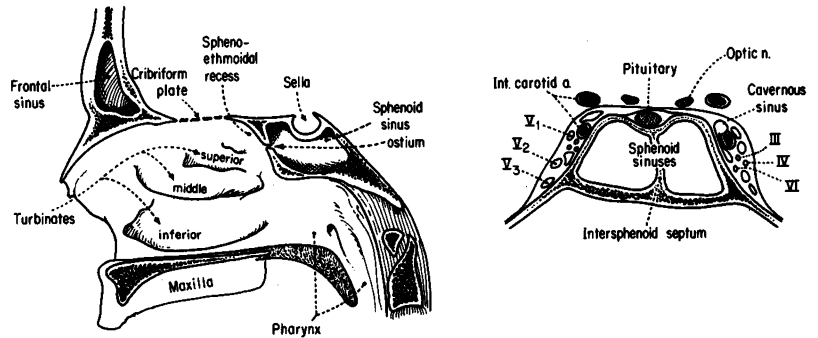


Figure 1. Sagittal and Coronal Sections of the Sphenoid Sinus.

The sagittal section (A) shows the relation of the sphenoid sinus to the sella turcica, cribriform plate, and nasopharynx. The coronal section of the posterior aspect of the sphenoid sinus (B) shows its close proximity to the cavernous sinuses and pituitary gland.

RESULTS

Thirty patients fulfilled our criteria for the diagnosis of bacterial sphenoid sinusitis. Fifteen had acute disease and 15 had chronic infection. During the study period (1968 to 1980), 1087 patients with a diagnosis of sinusitis were admitted to the two hospitals. Sphenoid sinusitis represented 2.7 per cent of the total number of sinusitis cases observed in our two institutions. Patients ranged in age from 9 to 84 years; 11 were less than 30 years old; 17 were women and 13 were men.

Initial Diagnosis

On admission, acute sphenoid sinusitis was often unrecognized, resulting in a delay in appropriate therapy. Only 6 of 15 patients were correctly diagnosed on first arriving at the hospital. Other initial diagnoses included ophthalmic migraine headache (in three patients), aseptic meningitis (in four), and cavernous sinus thrombosis (in two). A correct diagnosis on admission was more frequent in cases of chronic infection (9 of 15). The six unrecognized chronic infections were characterized by atypical headaches of progressively increasing severity that had lasted from six months to three years. Initial diagnoses in these cases included atypical facial pain (in two cases), migraine headache (in one), trigeminal neuralgia (in one), idiopathic third-nerve palsy (in one), and retro-orbital tumor (in one).

Symptoms

Headache was the most common initial symptom, beginning three days to one month before hospitalization in acute cases. Many of the patients with chronic sinus infection had had intermittent headaches for several years. The pain was dull and constant in some patients and intense and sharp in others. The headache interfered with sleep in most cases and was not relieved by aspirin.

Most of the patients reported pain in more than one location. The two most common patterns of pain were unilateral headaches involving the frontal, temporal,

or occipital region (or a combination), as well as pain or paresthesia in the remainder of the V₁ to V₃ area — i.e., the periorbital region, nose, cheek, teeth, and gums; and unilateral or bilateral retro-orbital headaches radiating into the occipital region. Vertex headache was a rare symptom (in four patients, Table 1).

Predisposing Illnesses

Among the acute cases, predisposing factors included deep-sea diving, cocaine sniffing, proton-beam therapy for a pituitary adenoma, and partial obstruction of the sphenoid ostium with carcinoma of the left ethmoid bone. Two patients had diabetes mellitus requiring insulin. Of the patients with chronic infection, only two had predisposing illnesses. One had had fractures of the orbital floor and ethmoid bones; the other had received oral steroids for arthritis.

Physical Findings

Most of the patients with acute disease had fever (38 to 40°C). Examination of the ear, nose, and throat often demonstrated purulent discharge from erythematous superior or middle turbinates. The most frequent neurologic deficit, detected in one third of the patients, was hyposthesia or hyperesthesia of the ophthalmic or maxillary dermatomes of the fifth nerve or both.

Several of the acutely infected patients were lethargic or comatose when they arrived at the emergency room. A depressed mental status was associated with nuchal rigidity, suggesting meningeal irritation. A change in mental status was accompanied by clinical findings (ptosis, chemosis, proptosis, and paralysis of the third, fourth, and sixth cranial nerves) suggesting that infection had extended to the cavernous sinus by the time of admission.

Laboratory Data

The peripheral-blood white-cell count was elevated (mean \pm S.E.M., $14,586 \pm 1056$ cells per cubic millimeter) in the patients with acute sinusitis but was within normal limits in those with chronic sinusitis. Cerebrospinal fluid was obtained from 10 patients. In five the findings were typical of a parameningeal infection — i.e., the white-cell count was between 6 and 1510 per cubic millimeter (428 ± 278), the

Table 1. Headache or Pain in 30 Patients with Sphenoid Sinusitis.

LOCATION	ACUTE SINUSITIS	CHRONIC SINUSITIS	TOTAL
	no. of patients *		
Vertex	2	2	4
Frontal	6	4	10
Temporal	5	4	9
Occipital	8	3	11
Retro-orbital	5	4	9
Orbital-periorbital	3	6	9
Nasal	1	4	5
Face-cheek	5	3	8
Jaw-teeth	1	1	2

*Many patients had pain in more than one site (see text).

Table 2. Bacteriologic Findings in 30 Patients with Sphenoid Sinusitis.*

ORGANISM	ACUTE SINUSITIS	CHRONIC SINUSITIS	TOTAL
	no. of patients *		
Aerobic bacteria			
Gram-positive organisms			
<i>Staphylococcus aureus</i>	5	1	6
<i>Staph. epidermidis</i>	0	3	3
β -Hemolytic streptococci	1	1	2
Viridans streptococci	2	0	2
Enterococci	1	0	1
<i>Streptococcus pneumoniae</i>	3	0	3
Gram-negative organisms			
<i>Hemophilus influenzae</i>	0	1	1
Other (<i>Escherichia coli</i> , <i>Pseudomonas aeruginosa</i> , proteus, <i>Klebsiella pneumoniae</i> , citrobacter, serratia)	2	6	8
Anaerobic bacteria			
Streptococci	3	1	4
Fungi			
Aspergillus	0	3	3

*Sphenoid sinus cultures were obtained from 13 patients with acute infection and from one with chronic infection. Mixed bacteria were found in four acute and four chronic cases.

polymorphonuclear leukocytes and lymphocytes were present in roughly equal numbers, and the glucose concentration was normal (in four patients) or low (in one). Findings in the other five patients were indicative of bacterial meningitis — i.e., the cerebrospinal-fluid white-cell counts were high (6425 ± 4864 cells per cubic millimeter), with a predominance of polymorphonuclear leukocytes, and the cerebrospinal-fluid glucose values were low.

Bacteriology

Blood specimens were obtained from most patients during surgical exploration. In four patients, specimens were obtained by transnasal cannulation of the sinus on admission. Additional specimens obtained during subsequent surgery in two of the patients yielded the same microorganisms on culture.

Staphylococcus aureus, *Streptococcus pneumoniae*, and other streptococci were isolated in specimens from the majority of patients with acute sphenoid sinusitis (Table 2). In two other patients gram-negative organisms were isolated from the sphenoid sinus. Cultures from four patients revealed more than one organism each. Cultures of blood from three patients contained the same pathogen found in the sphenoid sinus: *Staph. aureus* (in two patients) and anaerobic streptococci (in one). Cultures of cerebrospinal fluid from four patients were positive. In each case, the same pathogen was found in the sphenoid sinus and the cerebrospinal fluid: anaerobic streptococci (in one patient), group H streptococci (in one), *Staph. aureus* (in one), and *Str. pneumoniae* (in one).

In contrast to the predominance of gram-positive organisms in acute disease, there were equal numbers of gram-negative and gram-positive organisms in cultures from patients with chronic sphenoiditis (Table 2). Cultures from four of these patients revealed more than one organism. In three cases of chronic infection,

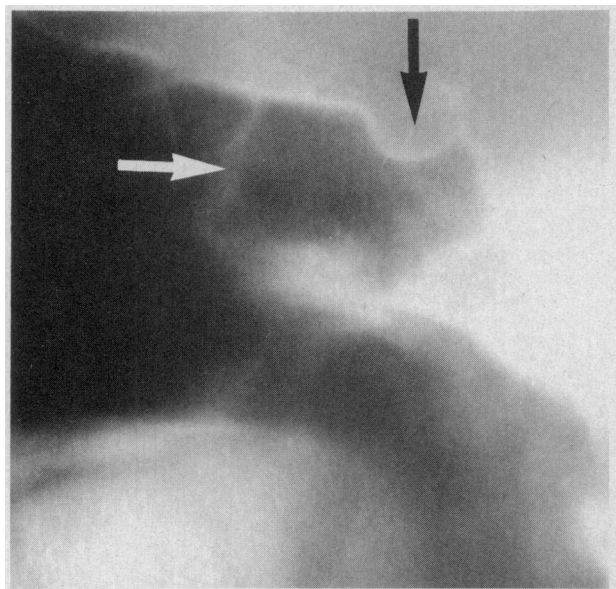


Figure 2. Lateral Tomographic Section of a Normal Sphenoid Sinus.

The vertical arrow points to the sella turcica. The orientation of the radiograph is identical to that of the sagittal section in Figure 1. The horizontal arrow points to the anterior wall of the sphenoid sinus (the wall nearest the nasal turbinates).

aspergillus species were identified by histopathology, which demonstrated superficial invasion of the mucosa by mycelia.

Radiographic Findings

Acute Sphenoiditis

Four patients with acute disease underwent routine skull or sinus radiography, which failed to reveal any abnormality of the sphenoid sinuses. In three cases, the diagnosis was not suspected until autopsy. A fourth case was diagnosed only after sinus tomography. Tomograms of the sinuses were obtained in the other 11 patients with acute infection. (A normal tomogram is shown for comparison in Figure 2.) The entire sphenoid was abnormal in six cases, and unilateral changes were found in five. Mucosal thickening was marked in most instances (Fig. 3). Three tomographic studies also showed sclerosis of the sphenoid bone. Involvement of other sinuses was seen in five patients (two with ethmoid sinusitis and three with pansinusitis).

Chronic Sphenoiditis

Mucosal thickening was marked in all patients with chronic sphenoid sinusitis; six had moderate to marked bony sclerosis (Fig. 4). Among these patients, increased density of the entire sphenoid sinus was found in 10, and unilateral disease in 5. Radiologic abnormalities were frequently observed in the ethmoid sinuses as well (in eight patients). In addition to sphenoid and ethmoid changes, opacification of the maxillary sinuses was apparent in three patients.

There was no evidence of air-fluid levels or bony erosion in any patient with sphenoid sinusitis. Com-

puterized axial tomography was positive in seven patients and indicated spread to the cavernous sinus in one. No correlation was found between the severity of clinical disease or complications and the degree of radiologically demonstrable mucosal thickening or bony sclerosis.

Treatment, Complications, and Outcome

Acute Sphenoiditis

Since the proper diagnosis was often missed on admission, appropriate therapy was frequently delayed. Only six patients received appropriate treatment with high-dose intravenous antibiotics. All six recovered without sequelae. All nine patients whose therapy was delayed had serious complications or died (Tables 3 and 4).

Major pyogenic complications included cavernous sinus thrombosis and bacterial meningitis (Table 3). With one exception, the survivors of delayed treatment were left with permanent neurologic deficits (Table 4).

Frequently, symptoms and signs failed to resolve despite treatment with high-dose intravenous antibiotics. Eight of the 11 surviving patients required surgical drainage for cure. At surgery, the sphenoid sinuses were found to be filled with pus, often under pressure. None of the four patients who subsequently died underwent surgery for drainage of the sphenoid sinus. With one exception, sphenoid sinusitis was not suspected while they were alive. Post-mortem examination revealed purulent sphenoid sinusitis in all four patients, acute septic cavernous sinus thrombophlebitis in three, and cortical-vein thrombosis in one. Bacterial meningitis, predominantly basilar, was found in all four. Additional findings included cerebral infarction, pituitary infarction, and retro-orbital abscess.

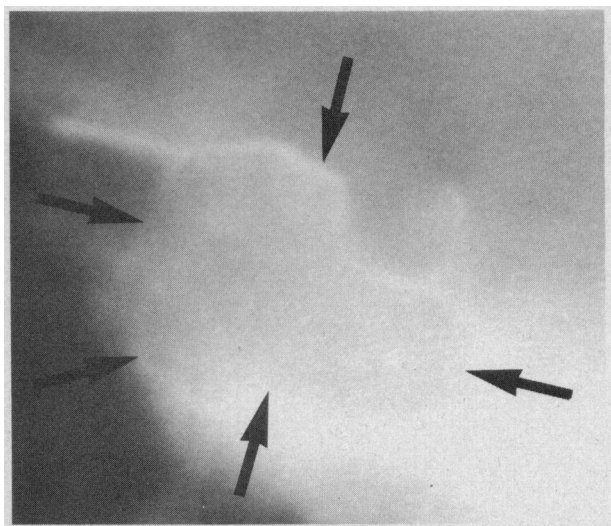


Figure 3. Lateral Tomographic Section from a Patient with Acute Sphenoid Sinusitis, Showing a Completely Opacified Sphenoid Sinus with No Bony Abnormality.

Arrows outline the bony margins of the sinus. The orientation of the radiograph is the same as in Figure 2.

Chronic Sphenoiditis

Of the 15 patients with chronic sphenoid sinusitis, eight had been treated with one or more courses of antibiotics, and symptoms recurred or persisted until surgery. Twelve of the 15 patients eventually required surgical exploration and drainage of the sphenoid sinus. The sinuses were filled with purulent material in nine patients; three others had only mucosal thickening at operation. Further findings included polypoid material (in two patients) and thick granular fibrous material (in two). Most of the patients had postoperative fever that resolved with adequate antibiotic therapy. Morbidity was lower in the group of patients with chronic sinusitis than in the group with acute infection. Three patients with chronic disease had severe complications, but none died (Tables 3 and 4). However, persistent sequelae were seen in four patients. Irreversible blindness developed in one, and three continued to have severe headaches associated with persistent sphenoid opacification on x-ray films. Two of these patients had pain in the region of V₂, which was not relieved by trigeminal rhizotomy (in one) or trigeminal block (in the other). A subsequent re-exploration in one patient again showed pus in the sphenoid sinuses.

DISCUSSION

Untreated infection of the sphenoid sinus can easily spread to the cavernous sinuses, dura mater, and optic canals, resulting in serious irreversible neurologic complications and death. Early recognition and aggressive therapy are critical in preventing such an extension of the infection.

Sphenoid sinusitis is less common than other sinus infections. The incidence at our institutions — 2.7 per

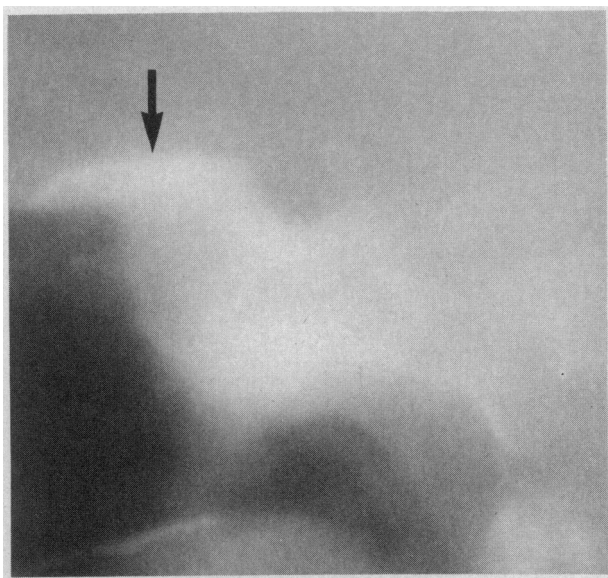


Figure 4. Lateral Tomographic Section from a Patient with Chronic Sphenoid Sinusitis, Revealing Increased Density within the Sphenoid Sinus Secondary to Membrane Thickening and Marked Sclerosis (Arrow).

The orientation of the radiograph is the same as in Figure 2.

Table 3. Complications in Patients with Sphenoid Sinusitis.

COMPLICATION	ACUTE	CHRONIC
	SINUSITIS	SINUSITIS
	<i>no. of patients</i>	
Orbital cellulitis, abscess	1	1
Unilateral ophthalmoplegia	1	1
Meningitis	6 *	0
Cortical-vein thrombosis	1	0
Cavernous sinus thrombosis	5	1

*Four of the patients with meningitis had cavernous sinus thrombosis; the other two presented with a lateralized hemiparetic syndrome.

cent of all sinus infections — is much lower than that described by Teed in the preantibiotic era.⁵ This lower incidence may reflect the use of antibiotics to treat sinusitis. We suspect that the incidence of sphenoid sinus infection may actually be higher in the antibiotic era. Many early cases may be missed on routine sinus x-ray films, and antibiotic treatment initiated for radiologic involvement of other sinuses.

Despite careful evaluation, diagnosis was delayed in the majority of our patients with acute sphenoid sinusitis. This disease should be included in the differential diagnosis of headache. Purulent sphenoiditis can be mistaken for frontal or ethmoid sinusitis, aseptic meningitis, brain abscess, or septic thrombophlebitis. Infection of the sphenoid sinus can also mimic trigeminal neuralgia, migraine headache, carotid-artery aneurysm, or brain tumor.

A clinical feature that may help the clinician recognize this disease is severe headache that interferes with sleep and generally is not relieved by aspirin. Typically, headache steadily increases in severity with time. Although it is often described as a vertex headache, our patients rarely reported headache in this location; most presented with fronto-temporal-occipital or retro-orbital-occipital pain. Another useful clue is pain or paresthesia in the region of the ophthalmic (V₁), maxillary (V₂), and mandibular (V₃) branches of the fifth nerve. Finally, symptoms of photophobia and eye tearing, in addition to raising the possibility of ophthalmic migraine, should also alert the physician to the possibility of sphenoid sinus infection with early spread to the cavernous sinus.

Physical examination may often provide useful clues. In many of our patients abnormal findings were either misinterpreted or ignored on initial evaluation. Sinus tenderness generally cannot be determined, since the sphenoid is located deep in the cranium (Fig. 1); however, a careful examination of the nose frequently reveals pus in the middle and superior turbinates. Hyperesthesia or hypoesthesia in the V₁, V₂, and V₃ dermatomes may indicate early extension beyond the sphenoid sinus walls. By the time other neurologic deficits become apparent, infection will have spread to the cavernous sinus or other intracerebral structures, and the likelihood of full recovery will be markedly diminished.

Radiographic study is the primary method for establishing the diagnosis on admission.¹⁴⁻¹⁸ Although conventional lateral and submental vertical views may

Table 4. Outcome in 30 Cases of Sphenoid Sinusitis.

OUTCOME	ACUTE SINUSITIS	CHRONIC SINUSITIS
	no. of patients	
Complete recovery	7	11
Severe headache	0	3
Irreversible cranial-nerve injury	4 *	1 †
Death	4	0

*Second, third, and fifth (V₂ and V₁) cranial nerves.

†Second cranial nerve.

show moderate to marked sinus opacification with sclerosis, such views failed to demonstrate sphenoid sinus infection in several of our patients. If the history and physical examination raise the possibility of such an infection, detailed anterior-posterior, lateral, and in some cases, basal tomographic cuts are indicated. More recently, computerized axial tomography (CT) has been the most useful radiographic examination for demonstrating sphenoid sinus opacification and bony involvement.¹⁸ In one of our patients an enhanced CT examination was used to detect unilateral cavernous sinus thrombosis. It should be emphasized that in our series no correlation was seen between the extent of opacification or bony sclerosis and the severity of complications. In fact, increased sclerosis tended to be associated with localized sinus infection. Therefore, any radiologic abnormality of the sphenoid sinus should be a matter of concern.

As in the previous studies of acute maxillary sinusitis, *Str. pneumoniae* and other streptococcal species were the most common organisms in our patients with acute infection.¹⁹⁻²¹ However, unlike previous investigators, we also frequently cultured *Staph. aureus*. Intrasinus exudate for culture was obtained from surgical specimens, as it was in the previous studies. In addition, the same organism was found in specimens of blood, cerebrospinal fluid, or sphenoid exudate (obtained by transnasal cannulation on admission) from several of our patients. Gram-negative bacilli were the most common organisms in our patients with chronic infection. Staphylococci also predominated in these patients — a finding previously noted by Frederick and Braude in their review of chronic sinusitis.²²

Treatment

Early and aggressive treatment is critical for prevention of the frequently fatal complications of this infection. Delay in treatment of acute infection was associated with 100 per cent morbidity in our series. In most patients with simple acute sinusitis, a trial of nasal decongestants and oral antibiotics can be started.

However, the patient who presents with severe hemi-cranial, retro-orbital, occipital, or vertex headache should have cranial CT examination to include the paranasal sinuses. Conventional tomography should be used if CT is not available. If involvement of the sphenoid sinus is confirmed, admission to the hospital for further evaluation and therapy should be considered.

The initial hospital evaluation should include can-

nulation of the sphenoid sinus to obtain specimens for Gram's stain and culture. If cranial-nerve deficits are noted, orbital and skull CT scans with contrast dye should be obtained. Cavernous sinus venography may also be performed to evaluate the patency of these vessels.

Parenteral antibiotics should be started soon after admission. If there are no clues to the bacterial cause on Gram-stained smears of the sinus exudate, high-dose penicillinase-resistant penicillin is the antibiotic of choice. Therapy should be continued for a minimum of two weeks. If symptoms persist or neurologic signs develop after the initial antibiotic treatment, the sphenoid sinus should be drained surgically.

Patients with chronic sphenoid sinusitis are generally not as critically ill as those with acute infection, nor is the morbidity and mortality as high as it is in acute cases. Treatment with parenteral antibiotics for staphylococci as well as gram-negative organisms is recommended. Surgery may be required for drainage and relief of symptoms.

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