# **Chapter 4**

# **Acute Frontal Sinusitis**

# 4

Douglas Reh, Peter H. Hwang

# Core Messages

- Although uncomplicated acute frontal sinusitis (AFS) is a self-limited disease, complications associated with it can be catastrophic
- Uncomplicated AFS is most often associated with a viral upper respiratory tract infection. Bacterial infection is suspected if symptoms are persistent for at least 10 days
- The diagnosis of AFS is considered in patients who meet the general diagnostic criteria for acute sinusitis and have symptoms localized to the forehead region
- The predominant organisms cultures from patients with uncomplicated AFS are Hemophilus influenza, Streptococcus pneumoniae, and Moraxella catarrhalis
- When indicated, uncomplicated AFS should be treated with 10 to 14 days of antibiotics
- Complicated AFS is suspected when symptoms are protracted and severe
- Work up of complicated AFS should include CT scans with IV contrast
- Intracerebral abscess is the most common intracranial complication of AFS
- Patients with complicated AFS should be admitted for intravenous antibiotic therapy, intravenous hydration, and serial neurologic examinations
- Treatment of complicated AFS often requires surgery in addition to antibiotic therapy

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# Introduction

Acute sinusitis is one of the leading diagnoses made in ambulatory medicine. The National Ambulatory Medical Care Survey (NAMCS) estimates that 20 million cases of acute bacterial rhinosinusitis (ABRS) occur each year [1]. The incidence of acute frontal sinusitis (AFS) specifically is considerably lower, less common than maxillary sinusitis in adults and ethmoid sinusitis in children. Medical therapies for acute sinusitis result in expenditures of \$3.5 billion per year in the United States. Of all antibiotics prescribed in 2002, 9% of pediatric prescriptions and 18% of adult prescriptions were written for a diagnosis of acute sinusitis [1].

AFS occurs most commonly in adolescent males and young men. While the reasons for the male predilection are unknown, the age predilection appears likely due to the peak vascularity and peak development of the frontal sinuses between the ages of 7 and 20. Although AFS is largely a self-limited disease, complications of acute sinusitis can have catastrophic clinical consequences if not detected promptly.

## Etiology and Pathophysiology of Acute Frontal Sinusitis

Acute frontal sinusitis is most commonly preceded by a viral upper respiratory tract infection. Human rhinovirus is implicated in 50% of cases, but other viruses may include coronavirus, influenza, parainfluenza, respiratory syncytial virus, adenovirus, and enterovirus. The peak prevalence of these viruses occurs in early fall and spring, which parallels the peak incidence of ABRS. Viruses upregulate pro-inflammatory cytokines such as interleukin-1, interleukin-6, interleukin-8, tumor necrosis factor- $\alpha$ , as well as other inflammatory mediators such as histamine and bradykinin. Viruses also suppress neutrophil, macrophage, and lymphocyte function and can thereby inhibit the immune response [2]. The viral induction of the inflammatory cascade results in acute mucosal edema, occlusion of sinus ostia, and impaired mucociliary clearance. Mucus stasis can then favor the proliferation of pathogenic micro-organisms, resulting in acute bacterial sinusitis. Other risk factors for acute sinusitis include a variety of host factors: septal deviation, nasal polyposis, and immunodeficiency/immunosuppression, among others.

While acute sinusitis typically affects the ethmoid and maxillary sinuses, progression of disease to involve the frontal sinus may be influenced by anatomic variations of the frontal sinus. The frontal sinus begins developing at age 3. Four frontal pits along the upper lateral wall of the embryological middle meatus differentiate into the anterior ethmoid cells. The second of these furrows evaginates from the anterior ethmoid region into the frontal bone, creating the frontal sinus [3]. Because the frontal sinus is embryologically derived from pneumatization of the ethmoid, frontal sinus outflow is thus influenced and defined by the degree of pneumatization of the ethmoid labyrinth. A variety of ethmoid-derived structures that comprise the frontal recess can thus narrow the outflow tract and predispose to acute frontal sinusitis. These structures may include agger nasi cells anteriorly, the bulla lamella posteriorly, supraorbital ethmoid cells laterally, and type I–IV frontal cells [3].

#### **Uncomplicated Acute Frontal Sinusitis**

#### Diagnosis

AFS is principally a clinical diagnosis based on type and duration of symptoms. CT scans, when ordered to diagnose acute bacterial sinusitis, may yield false positives. Gwaltney et al. showed that 87% of adults with acute onset of upper respiratory tract infection (URI) symptoms demonstrate CT evidence of nasal cavity mucosal thickening and sinus opacification [4]. They also showed that after 2 weeks without antibiotic therapy, repeat CT scans showed improvement in 79% of 14 patients with these findings. Sinus aspiration studies have shown that significant bacterial growth occurs in approximately 60% of patients with URI symptoms lasting for 10 days or more [5]. Therefore persistent or worsening symptoms after 10 days may indicate a bacterial infection [1].

In 1997 the American Academy of Otolaryngology-Head and Neck Surgery Foundation assembled the Rhinosinusitis Task Force (RSTF) to develop clinical definitions of rhinosinusitis. Rhinosinusitis as defined by the RSTF is "inflammation of the nasal cavity and paranasal sinus" [6]. The RSTF subclassified rhinosinusitis into three major clinical categories based on duration of symptoms: acute, with symptoms lasting less than 4 weeks; subacute, between 4 and 12 weeks; and chronic, greater than 12 weeks.

By RSTF guidelines, patients with rhinosinusitis must meet a variety of symptomatic major and minor criteria.

The major criteria defined by the RSTF include:

- Facial pain or pressure
- Nasal congestion
- Nasal obstruction
- Purulent rhinorrhea
- Hyposmia or anosmia
- Fever (for acute rhinosinusitis only)
- Purulence on nasal exam

The minor criteria defined by the RSTF include:

- Headache
- Nonacute fever
- Halitosis
- Fatigue
- Dental pain
- Cough
- Ear pain or pressure

A diagnosis of rhinosinusitis requires either two major factors, one major and two minor factors, or purulence in the nasal cavity on physical exam [6].

There are no site-specific criteria for the diagnosis of AFS. Generally frontal sinus symptoms are localized to the brow, temple, and frontal bone region. Frontal headache is the most prevalent symptom of AFS [7]. Thus, a diagnosis of AFS should be considered in patients who meet RSTF criteria for acute sinusitis, in whom symptoms localize to the forehead region. In some cases, the acute onset of frontal headache, even in the absence of more classic symptoms such as nasal congestion and rhinorrhea, should prompt the physician to consider a diagnosis of AFS. This is especially true in those patients without a history of chronic headache.

Although most cases of acute rhinosinusitis can be diagnosed by symptoms alone, the physical examination can provide helpful adjunctive diagnostic information. Transillumination and palpation, while classically described for physical exam of the sinuses, are relatively nonspecific tests. Anterior rhinoscopy and nasal endoscopy, however, can be useful adjunctive diagnostic tools. Examination of the nasal cavity may reveal mucosal edema, purulent discharge, or anatomic obstructions such as septal deviation or polyposis. Purulent secretions may be aspirated under endoscopic visualization and cultured to guide antimicrobial therapy. During aspiration and culture, the endoscope should be used to retract the nasal vestibule away to minimize contamination of the culture device by normal nasal vestibular flora.

Unless a complication of acute sinusitis is suspected, imaging studies such as CT and MRI are not necessary in making the diagnosis of AFS.

## Bacteriology

While the bacteriology of acute maxillary sinusitis has been well documented by maxillary tap studies, the bacteriology of AFS has not been well studied. Data are limited principally because of the difficulty of accessing the frontal sinus for cultures. Brook obtained aspirates from the frontal sinuses of 15 patients with acute infection [8]. Twenty isolates were grown from 13 of the specimens. The predominant aerobic and facultative organisms were *Haemophilus influenzae* (6/13), *Streptococcus pneumoniae* (5), and *Moraxella catarrhalis* (3). B-lactamase producing organisms were isolated in 33% of the specimens. Limitations of this study were its small numbers and the lack of documentation of sampling technique.

Given that AFS typically occurs in conjunction with acute maxillary and ethmoid sinusitis, it seems reasonable to extrapolate data for acute maxillary sinusitis to that for AFS. Indeed, the organisms cultured in the Brook study did parallel those obtained from the maxillary sinuses in other studies; namely, *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Moraxella catarrhalis* [1].

#### Treatment

The goals of treating uncomplicated AFS are:

- to control the infectious component of the disease process using antimicrobial therapy
- to reduce the edematous, obstructive component of the disease process and restore sinus patency using decongestant therapy
- Uncomplicated AFS is almost exclusively treated medically; surgical therapy is rarely indicated.

Antibiotic therapy should be selected for coverage of the primary organisms associated with acute rhinosinusitis: *S. pneumoniae*, *H. influenzae*, and *M. catarrhalis*. Drug resistance has become an increasing concern in the treatment of ABRS. Since the early 1990's, the rates of penicillin resistance in *S. pneumoniae* have increased dramatically, with 15% of isoTable 4.1. U.S. penicillin resistance rates of *S. Pneumoniae* by region, 1999–2000

Geographic Location	No. of isolates	Intermediate resistance (%)	High-level resistance (%)
West			
San Diego, CA	30	10.0	23.30
Los Angeles, CA	51	5.9	15.70
San Francisco, CA	52	9.6	23.10
Portland, OR	22	22.7	31.80
Seattle, WA	50	18.0	18.00
Denver, CO	51	21.6	13.70
Salt Lake City, UT	50	16.0	16.00
Phoenix, AZ	59	10.2	35.60
Midwest			
Iowa City, IA	54	11.1	16.70
Indianapolis, IN	56	10.7	19.60
Chicago, IL	41	14.6	12.20
Milwaukee, WI	53	11.3	32.10
Detroit, MI	58	8.6	5.20
Cleveland, OH	52	7.7	34.60
East	50	10.0	22.00
Rochester, NY	50	18.0	22.00
Boston, MA	31	6.5	19.40
New York, NY	59	15.3	20.30
Philadelphia, PA	52	19.2	7.70
Washington DC	20	5.0	35.00
South			
Chapel Hill, NC	41	9.8	56.10
Mobile, AL	49	10.2	16.30
Houston, TX	55	20.0	38.20
Dallas, TX	44	11.4	15.90
Miami Beach, FL	21	19.1	28.60

From [10]

lates showing intermediate resistance and 25% showing high resistance. Macrolide- (18%) and trimethoprim/sulfamethoxazole (TMP/SMX) (20%)-resistant strains of *S. pneumoniae* are also significant in the United States [9]. Thirty percent of *H. influenzae* and greater than 95% of *M. catarrhalis* cultured are B-lactamase-producing isolates [1]. Resistance patterns and prevalence differ by geographic region. Table 4.1 shows differences in bacterial resistance by U.S. region [10].

The Sinus and Allergy Health Partnership recently published antibiotic recommendations for the treatment of mild to moderate ABRS. These recommendations are based on clinical efficacy and reflect drug resistance patterns. These recommendations are summarized in Table 4.2 [1]. AFS should be treated with a minimum of 10 to 14 days of antibiotics when possible. If the patient's symptoms fail to resolve, the antibiotic course should be extended by 2 weeks [11] and consideration should be given to endoscopic exam and culture.

Adjunctive medical treatment in AFS is aimed primarily at re-establishing the patency of the frontal recess and ostiomeatal complex through which the frontal sinus drains. Topical (oxymetazoline, phenylephrine) and oral (pseudoephedrine) decongestants and mucolytics (guaifenesin) may improve drainage of the affected sinuses. Selected patients with known inflammatory dysregulation, such as those with atopic disease, aspirin sensitivity, or nasal polyposis may benefit from oral steroids. When used in carefully selected patients, steroids can acutely reduce inflammation and facilitate drainage of affected sinuses [11].

#### **Complicated Acute Frontal Sinusitis**

#### Diagnosis

Occasionally, patients with AFS may present in acute distress with toxic clinical features. Clinical findings such as prostration, severe headache, or orbital complaints should raise suspicion for an infectious complication of AFS.

Complications from AFS principally involve:

- extension to intracranial structures
- the orbits may occasionally be affected

Although the true incidence of AFS-related complications is unknown, a study of 649 patients admitted to the hospital for sinusitis showed an intracranial complication rate of 3.7% [12].

The frontal sinus is susceptible to extrasinus spread of infection in part because its venous drainage occurs through diploic veins that traverse the posterior table and communicate with the venous supply of the meninges, cavernous sinus and dural sinuses. These venous channels may be more porous in the developing sinus, and thus adolescents and young

Initial therapy	Calculated clinical efficacy (%)	Calculated bacteriologic efficacy (%)	Switch therapy options (no improvement after 72 hours)
Mild disease with no recent antimicrobial use in past 4–6 weeks			
Amoxicillin/clavulanate (1.75–4 g/250 mg/d)	90-91	97–99	
Amoxicillin (1.5–4 g/d)	87-88	91–92	Gatifloxacin/levofloxacin/moxifloxacin
Cefpodoxime proxetil	87	91	Amoxicillin/clavulanate (4 g/250 mg)
Cefuroxime axetil	85	87	Ceftriaxone
Cefdinir	83	85	Combination therapy
B-Lactam Allergic			
TMP/SMX	83	84	
Doxycycline	81	80	Gatifloxacin/levofloxacin/moxifloxacin
Azithromycin/erythromycin/clarithromycin	77	73	Rifampin plus clindamycin
Mild disease with recent antimicrobial use in past 4–6 weeks or moderate disease			
Gatifloxacin/levofloxacin/moxifloxacin	92	100	
Amoxicillin/clavulanate (4 g/250 mg)	91	99	Reevaluate patient
Ceftriaxone	91	99	
B-Lactam Allergic			
Gatifloxacin/levofloxacin/moxifloxacin	92	100	Reevaluate patient
Clindamycin and rifampin			

Table 4.2. Recommended antibiotic therapy for adults with mild or moderate ABRS

From [1]

adults (especially male) are at increased risk for complications of AFS.

Suspicion for complicated AFS should be elevated when:

- Symptoms are protracted or more severe than would be expected for a typical case of acute sinusitis
- On physical examination, there is periorbital edema or discoloration, which can indicate a preseptal cellulitis, or painful or restricted eye movement, which may indicate an orbital cellulites or abscess
- Neurologic findings such as altered mental status, seizure, or cranial neuropathy are present, which may indicate intracerebral complications

As in uncomplicated AFS, nasal endoscopy may yield cultures of purulent material that can guide antimicrobial therapy. Lumbar puncture may also be indicated to obtain CSF cultures and to rule out meningitis. Consultations with an ophthalmologist, neurosurgeon, neurologist, or infectious disease specialist should be considered.

In contrast to uncomplicated AFS, radiologic studies play an important role in confirming and characterizing the extent of extrasinus infectious involvement. CT scan is the imaging modality of choice in evaluating intracranial or orbital complications of AFS. Studies should be performed with IV contrast in axial and coronal planes. With bone and soft tissue algorithms, CT scans can characterize bony erosions of the frontal sinus as well as phlegmons or fluid collections in adjacent orbital and intracranial soft tissue. Serial imaging studies should be considered in patients who appear clinically unresponsive to initial treatment.

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Intracerebral abscess is the most common intracranial complication of AFS. The frontal lobe is most frequently involved, although hematogenous seeding of distant brain structures may be observed less commonly [12]. Headache is the most common early symptom, although subsequently there may be a quiescent asymptomatic phase during which an abscess has coalesced [13]. Overall mortality reported in the literature ranges widely from o% to 53% [13,14].

Meningitis is another important neurologic complication of AFS [12].

Symptoms suggestive of meningitis include:

- High fever
- Photophobia
- Neck pain or stiffness
- Severe headache
- Mental status changes

Mortality is reported as high as 45% [15]. While meningitis is the second most common intracranial complication of acute sinusitis in general, the frontal sinus as a site of origin is less common than the sphenoid (most common) and the ethmoid sinuses. Advanced cases of frontal sinusitis with meningitis may also be associated with subdural or epidural abscesses. When these abscesses occur they typically develop immediately posterior to the frontal sinus along pathways of venous drainage [14].

Osteomyelitis of the frontal sinus may be caused by direct extension of infection or by thrombophlebitis of the diploic veins. Of all the paranasal sinuses, the frontal sinus is most commonly associated with osteomyelitis. When osteomyelitis involves the anterior table, a subperiosteal abscess may develop, presenting as a subcutaneous fluctuant protuberance over the brow or forehead. This abscess is known as Pott's Puffy Tumour, which was first described by Sir Percival Pott in 1775 [16]. Strictly an infectious complication and not neoplastic in any way, Pott's Puffy Tumour may present with severe headache, fever, and photophobia.

Cavernous sinus thrombosis and superior sagittal sinus thrombosis comprise another important class of complications associated with AFS. Patients with cavernous sinus thrombosis develop:

- Ophthalmoplegia
- Proptosis
- Visual loss
- Trigeminal nerve (V2 and V3) deficits

Early clinical recognition is important, as symptoms can quickly progress, and mortality exceeds 30% [17–19]. Superior sagittal sinus thrombosis is associated with subdural abscess and has a high mortality rate, 80% [18].

Isolated AFS rarely causes orbital complications. However, AFS in the context of pansinusitis is associated with 60–80% of orbital complications [20,21]. Although direct spread to the orbits from the frontal sinus is possible, the ethmoid sinuses are more commonly implicated in the development of orbital complications.

#### Bacteriology

The organisms cultured from the sinuses of patients with intracranial abscesses include [12]:

- Staphylococcus aureus
- Anaerobic streptococci
- Streptococcus epidermidis
- Streptococcus pneumoniae
- Staphylococcus intermedius
- Beta-hemolytic streptococci
- Gram-positive aerobes and anaerobes are the predominant bacteria in complicated AFS

Table 4.3 summarizes the organisms cultured from paranasal sinuses in patients with intracranial complications [12]. Table 4.4 shows Goldberg et al.'s summarization of the common organisms associated with AFS complications and the recommended primary antibiotic therapy based on the Sanford Guide to Antimicrobial Treatment [14].

#### Chapter 4

### Treatment

Treatment of complicated AFS includes aggressive medical therapy and surgery to drain both the involved sinus and the abscess collection if present.

Because of the acuity and morbidity of complicated frontal sinusitis, patients should be admitted for

Table 4.3. Organisms cultured from paranasal sinuses with associated intracranial complications

Organism	n (%)
Negative cultures	5 (21)
S. aureus	5 (21)
Anaerobic streptococci	3 (12)
S. epidermidis	2 (8)
S. pneumoniae	2 (8)
S. intermedius	2 (8)
b-Hemolytic streptococci	2 (8)
S. viridans	1 (4)
Actinomycoses sp.	1 (4)
Fusobacterium necrosporum	1 (4)
Bacteroides melaninogenicus	1 (4)

intravenous antibiotic therapy, serial neurologic examination, and intravenous hydration. Empiric antibiotic therapy should be initiated immediately, choosing broad-spectrum agents that have favorable penetration of the blood-brain barrier. If cultures can be obtained, antibiotic therapy may be tailored accordingly. It should be noted that a significant percentage of cultures from patients with intracranial complications are negative. This may perhaps occur because antibiotic therapy is often initiated emergently before cultures can be obtained. Antila et al. obtained 103 frontal sinus cultures in patients with AFS and simultaneous maxillary sinusitis [22]. Only 30% of these cultures were positive for bacteria. Twenty-one percent of the cultures in Clayman et al.'s study were negative [12]. In such cases, bacteriologic data from historical cohorts may be used to guide antibiotic selection.

Depending on the degree of morbidity, many patients will require continuation of intravenous antibiotic therapy as an outpatient after resolution of the acute phase of illness. Oral antibiotic therapy may be appropriate in selected patients. Duration of treatment varies with the nature and severity of the complication, as well as the response to initial therapy.

The use of intravenous corticosteroids in patients with AFS complications is controversial. Some stud-

Table 4.4. Common organisms associated with ABRS-related complications and recommended empiric antibiotic therapy

Disease	Most common organism	Primary drug choice	Alternative 1
Pott's tumor (acute osteomyelitis)	<i>S. aureus</i> , streptococci, anaerobes, polymicrobial	Pencillinase-resistant penicillin and metronidazole, consider vancomycin	Third-generation cephalosporin and vancomycin and metronidazole
Intracranial abscess	Streptococci, Bacteroides sp.	3 <sup>rd</sup> generation cephalosporin and metronidazole	High-dose PCN G and metronidazole
Orbital complication	S. pneumococcus,H. influenzae, M. catarhalis, S. aureus	2 <sup>nd</sup> and 3 <sup>rd</sup> generation cephalosporin or ampicillin/ sulbactam	Ticarcillin/ clavulanate or piperacillin and tazobactam
Meningitis	S. pneumococcus, H. influenzae	3 <sup>rd</sup> generation cephalosporin and vancomycin	Meropenem and vancomycin
Dural sinus thrombophlebitis	S. aureus, group A streptococcus, H. influenzae, fungal organisms	Pencillinase-resistant penicillin and 3 <sup>rd</sup> generation cephalosporin	Imipenem or meropenem and vancomycin

From [14]

From [12]

ies have advocated their use in patients with cerebral edema and clinical deterioration [23], while others argue that they may interfere with antibiotic penetration and immune response [12]. No prospective studies or animal models have conclusively shown that steroids improve mortality or morbidity associated with cerebral edema; thus the use of corticosteroids should be considered on an individual basis.

Treatment of complicated AFS often involves surgery in addition to antibiotic therapy. Patients with intracranial abscesses may require neurosurgical drainage concurrently with surgical treatment of the frontal sinus.

Methods of draining the frontal sinus include:

- Trephination
- Endoscopic frontal sinusotomy
- External ethmoidectomy

# Advantages and Disadvantages of Trephination

#### Advantages

- Technical simplicity
- Efficacy of draining the sinus
- Access to the sinus lumen for irrigation

#### Disadvantages

- Scar
- Potential injury to the supraorbital nerve
- The critical area of impaired outflow of the sinus is not addressed

In experienced hands, endoscopic frontal sinusotomy may be an alternative surgical technique in complicated AFS. The endoscopic approach provides a minimally invasive means of draining the sinus and anatomically improving frontal outflow. Disadvantages of the endoscopic approach include its technical complexity as well as the difficulty of adequate visualization in the acutely infected milieu. External frontoethmoidectomy is less commonly used in managing complicated AFS. This technique may be associated with frontal mucocele formation (20%–30% of cases) and frontal stenosis [24].

## References

- Sinus and Allied Health Partnership (2004) Antimicrobial treatment guidelines for acute bacterial rhinosinusitis. Otolaryngol Head Neck Surg 130(1):1-45
- 2. Patel J, Faden H, Sharma S, et al (1992) Effect of respiratory syncytial virus on adherence, colonization and immunity of non-typable Haemophilus influenzae: implications for otitis media. Int J Pediatr Otorhinolaryngol 23:15–23
- Kuhn FA (2001) Surgery of the Frontal Sinus. In: Kennedy DW, Bolger WE, Zinreich SJ "Diseases of the Sinuses", B.C. Decker, Hamilton, Ontario, pp 281–301.
- 4. Gwaltney JM Jr, Scheld WM, Sande MA, et al (1992) The microbial etiology and antimicrobial therapy of adults with community-acquired sinusitis: A fifteen year experience at the University of Virginia and review of other selected studies. J Allergy Clin Immunol 90:457–461
- 5. Gwaltney JM Jr, Phillips CD, Miller RD, et al (1994) Computed tomographic study of the common cold. N Engl J Med 330:25–30
- 6. Report of the Rhinosinusitis Task Force Committee Meeting (1997) Alexandria, Virginia, August 17, 1996. Otolaryngol Head Neck Surg 117(3 Pt 2): S1–68
- Seiden A, Martin V (2001) Headache and the frontal sinus. Otolaryngol Clinics North Am 34(1):227–241
- Brook I (2002) Bacteriology of acute and chronic frontal sinsusitis. Arch Otolaryngol Head Neck Surg 128:583-555
- 9. Hoban DJ, Doern GV, Fluit AC, Roussel-Delvallez M, Jones RN (2001) Worldwide prevelance of antimicrobial resistance in *Streptococcus pneumoniae, haemophilus influenzae*, and *Moraxell catarrhalis* in the SENTY antimicrobial surveillance program, 1997–1999. Clinic Infectious Disease 32(Suppl 2): \$81–93
- Doern GV, Heilmann K, Brueggemann A, et al (2001) Antimicrobial resistance among clinic isolates of streptococcus pneumoniae in the United States during 1999–2000, including a comparison of resistance rates since 1994–1995. Antimicrob Agents Chemother 45(6):1721–1729
- Maccabee M, Hwang P (2001) Medical therapy of acute and chronic frontal sinusitis. Otolaryngol Clinics North Am 34(1): 41–7
- 12. Clayman GL, Adams GL, Paugh DR, Koopmann CF (1991) Intracranial complications of paranasal sinusitis: A combined institutional review. Laryngoscope 101:234–239
- Giannoni CM, Stewart MG, Alford EL (1997) Intracranial complications of sinusitis. Laryngoscope 107(7):863–867

- 14. Goldberg AN, Oroszlan G, Anderson TD (2001) Complications of frontal sinusitis and their management. Otolaryngol Clinics North Am 34(1):211–225
- 15. Singh B, Dellen VJ, Ranjettan S, et al (1995) Sinogenic intracranial complications. J Laryngol Otol 1109:945
- 16. Pott P (1775) The Chirurgical Works of Percival Pott. London, Hayes W. Clarke and B. Collins
- 17. Morgan PR, Morrison WV (1980) Complications of frontal and ethmoid sinusitis. Laryngoscope 90:661
- 18. Southwick FS, Richardson EP, Swartz M (1986) Septic thrombosis of the dural venous sinuses. Medicine 65:82
- Stankiewitz JS, Newell DJ, Park AH (1993) Complications of inflammatory diseases of the sinuses. Otolaryngol Clin North Am 26:63
- 20. Jackson K, Baker SR (1986) Clinical implications of orbital cellulitis. Laryngoscope 96:568

- 21. Schramm VL, Curtin HD, Kennerdell JS (1982) Evaluation of orbital cellulitis and results of treatment. Laryngoscope 92:732
- 22. Antila J, Suonpaa J, Lehtonen O (1997) Bacteriological evaluation of 194 adult patients with acute frontal sinusitis and findings of simultaneous maxillary sinusitis. Acta Otolaryngol Suppl (Stockh) 529:162
- 23. Gallagher RM, Gross CW, Phillips D (1998) Suppurative intracranial complications of sinusitis. Laryngoscope 108: 1635
- 24. Lang EE, Curran AJ, Walsh MA, et al (2001) Intracranial complications of acute frontal sinusitis. Clin Otolaryngol 26:452-457