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Editors

The Frontal Sinus

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With 144 Figures, 73 in Color
and 40 Tables

Springer

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- Dedicated to the memory of my mother, Eftihia E. Kountakis, who with her nurturing devotion inspired my pursue of a medical career, and to my loving wife Eleni and children Eftihia, Emmanuel and Nikoleta, who bring meaning to my life.
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  Stil Kountakis

- To my wife, Dana, and my children, Rebecca, Benjamin, Grace, and Anna. A part of you is in each of these pages. Soli Deo Gloria.

  Brent Senior

- To my wife Julia, and to my children Maximilian and Clara for their constant patience.
  It is a pleasure to thank my American colleagues for excellent cooperation in editing this book.

  Wolfgang Draf

Preface

Advances in instrumentation and surgical techniques continue to yield improvements in the surgical management of sinus disease. Rhinologists have developed techniques to address disease in remote areas along the anterior skull base so that many procedures previously performed using an open approach may now be performed endoscopically. Despite these advances, the complex anatomy and remote location of the frontal recess continue to pose challenges in the surgical management of frontal sinus disease. The narrow funnel-shaped aperture and important surrounding structures can predispose to complications most rhinologists hope to avoid they can do without. Because of this, it is not uncommon to hear at rhinology meetings that it is usually best for the average otolaryngologist to avoid instrumentation in this area, especially in primary surgeries. Numerous manuscripts are published describing the anatomy, diagnostic techniques, and medical and surgical management of frontal sinus disease. But as our residents and fellows survey the literature, they often wish they had a single comprehensive source of information related to the anatomy and management of frontal sinus disorders.

This project was initiated in order to fill this void and to provide a valuable source of information not only for academic institutions but also for the private practice environment. Most of the world's leading authorities in rhinology were invited to participate. Chapters in the book are arranged in a logical fashion, providing a comprehensive body of information beginning with the history of frontal sinus surgery and addressing more complex surgical concepts as the reader progresses through the text. Each chapter was written by authors that possess extensive experience on the topic and have previously published on the particular anatomical structure or issue the chapter addresses. The result is the first exhaustive frontal sinus textbook that can be used as a reference source by both academic and practicing otolaryngologists worldwide.

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Chapter 1

History of Frontal Sinus Surgery

1

Hassan H. Ramadan

Core Messages

- With over two centuries of scientific description of frontal sinus surgery, the optimal procedure remains unclear
- Balancing concerns of eradication of disease with cosmesis and restoration of frontal sinus function has resulted in the development of numerous procedures for treatment of frontal sinus disease
- Endoscopic approaches are now widely applied to the management of frontal sinus disease

Contents

Introduction

The first frontal sinus procedure was described in 1750 [36]. Despite more than two centuries since the description of the first procedure on the frontal sinus, the optimal procedure remains unclear. Although frontal sinus surgery makes up only a small portion of all paranasal sinus surgery, the literature is filled with publications on this subject. Ellis in 1954 stated that "surgical treatment of chronic frontal sinusitis is difficult, often unsatisfactory and sometimes disastrous. The many surgical techniques available are expressions of our uncertainty and perhaps so our failure" [11].

The ideal treatment for diseases of the frontal sinus is one that will provide complete relief of symptoms, eradicate the underlying disease process, preserve the function of the sinus, and cause the least morbidity and the least cosmetic deformity. Over the last two centuries a variety of surgical procedures have been described for the treatment of frontal sinus disease. Those procedures flip-flopped from external to intranasal to external and currently to intranasal again. The ideal procedure has not been identified yet despite 2 centuries of various techniques.

The recent advances in imaging and endoscopic techniques have resulted in the resurgence of intranasal procedures for the treatment of frontal sinus disease. Frontal sinus disease, particularly chronic frontal sinusitis, is a highly morbid and sometimes life-threatening condition because of its potential complications. Despite the fact that over the years the incidence of complications has decreased, orbital and intracranial complications, including meningitis, subdural abscess, intracerebral abscess, and osteomyelitis continue to occur.

1

Trephination Era (1750)

Frontal sinus surgery was first described in the 18th century. It is noted that as early as 1750 Runge performed an obliteration procedure of the frontal sinus [36]. The first published report in 1870 by Wells described an external and intracranial drainage procedure for a frontal sinus mucocele [44].

In 1884 Alexander Ogston described a trephination procedure through the anterior table to evacuate the frontal sinus. He then dilated the nasal frontal duct, curetted the mucosa (Fig. 1.1A,B), and established drainage with a tube that was placed in the duct [32].

At the same time Luc described a similar procedure, and two years later the Ogston-Luc procedure was established [26]. However, this technique did not gain popularity because of the high failure rate due to nasal frontal duct stenosis [7].

Radical Ablation Procedures (1895)

At the turn of the century a number of physicians were advocating a radical frontal sinus procedure. Kuhnt in 1895 described removing the anterior wall of the frontal sinus in an attempt to clear disease. The mucosa was stripped to the level of the frontal recess, and a stent was placed for temporary drainage [9]. In 1898 Riedel/Schenke described the first procedure for obliteration of the frontal sinus [34], advocating completely removing the anterior table as well as the floor of the frontal sinus with stripping of the mucosa. This procedure had the advantages of removing osteomyelitic bone as well as allowing for easy detection of recurrent disease. This procedure, however was plagued by the unsightly cosmetic forehead deformity. Killian in 1903 described a modification of the Riedel-Schenke procedure [22]. In an attempt to minimize the cosmetic deformity he recommended preserving a one-centimeter bar of the supraorbital rim. He also recommended an ethmoidectomy with rotation of a mucosal flap into the frontal recess with stenting to prevent stenosis. At that time Killian's technique was embraced because of the success as well as the reduced cosmetic deformity. However the Killian procedure was later abandoned because of the high incidence of late morbidity with restenosis, supraorbital rim necrosis, postoperative meningitis, and mucocele formation, as well as death.

Conservative Procedures (1905)

Because of the significant cosmetic deformity as well as the high failure rate of those ablative external procedures, an era of conservatism followed next. This era consisted of intranasal approaches to the frontal sinus as well as external frontoethmoid techniques. In 1908 Knapp [23] described an ethmoidectomy through the medial wall and entering the frontal sinus through its floor, by which he removed diseased mucosa and enlarged the nasal frontal duct. His operation however never received widespread recognition. In 1911, Schaeffer proposed an intranasal puncture technique to re-establish the drainage and ventilation of the frontal sinus [38]. Numerous complica-

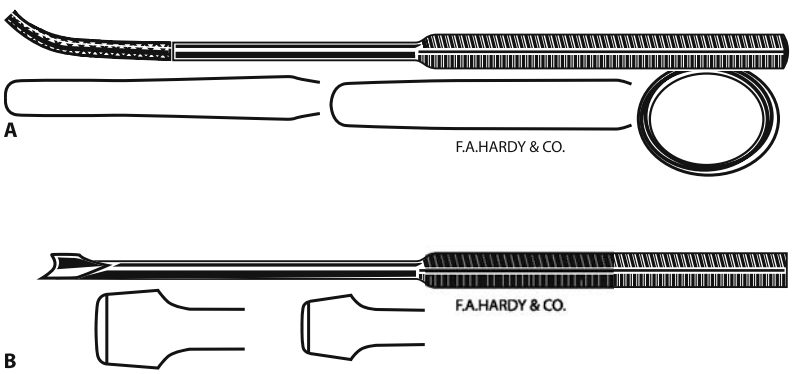

Fig. 1.1A,B.
Instrumentation utilized by Ogston for frontal sinus trephination and curetting frontal sinus mucosa

tions were encountered, however, including intracranial penetration. Between 1901 and 1908, Ingals, Halle, Good, and Wells described several intranasal procedures to the frontal sinus [14, 16, 19, 45]. Halle described a procedure in which the frontal process of the maxilla was chiseled out, and then a burr was used to remove the floor of the frontal sinus [16]. This operation was rarely used because it was associated with a high mortality rate. All of these intranasal approaches were abandoned because of the high mortality and complication rates associated with them. This increased incidence of mortality and complications was a result of the inadequate visualization of the frontal recess.

In 1914, Lothrop described a procedure to enlarge the frontal drainage pathway in a way that would prevent restenosis as well as closure as was reported with other procedures at the time [25]. The procedure described a combined intranasal ethmoidectomy and an external ethmoid approach to create a common frontal nasal communication by resecting the nasal sinus floor, the frontal sinus septum, and the superior nasal septum. Lothrop later admitted that the lack of visualization during the intranasal approach made the procedure dangerous. Further follow-up on those patients also showed that the resection of the medial orbital wall allowed the collapse of orbital soft tissue into the ethmoid area, with subsequent stenosis of the frontal drainage pathway.

External Frontoethmoidectomy (1897, 1906, 1921)

Between 1897 (Jansen [20]) and 1906 (Ritter [35]), the details of frontoethmoidectomy were described in Germany. In the Anglo-American literature, Lynch (1921) [28] in the United States and Howarth [18] in the United Kingdom popularized the principle of frontal sinus floor resection and enlargement of the frontal sinus drainage. Therefore in those countries frontoethmoidectomy was known as the Lynch and Howarth operation [17].

An incision in the medial periorbital area is used (Fig. 1.2), and the frontal process of the maxilla, as well as the lamina papyracea are removed. This allowed access to remove the frontal sinus floor and to curette the mucosa. A stent was then placed in the frontal ostium to maintain communication. The stent was left in place for approximately 10 days. The procedure however was complicated by restenosis and recurrent infections. The problem was somewhat related to the medialization of the orbital soft tissue, as described by Boyden [3], that resulted in nasal frontal narrowing with scarring and stenosis. Failure rates were reported up to 33% with the Lynch procedure.

Despite the failure of the Lynch procedure, interest in it was maintained. Sewall, Boyden, and McNaught modified the Lynch technique in an attempt to in-

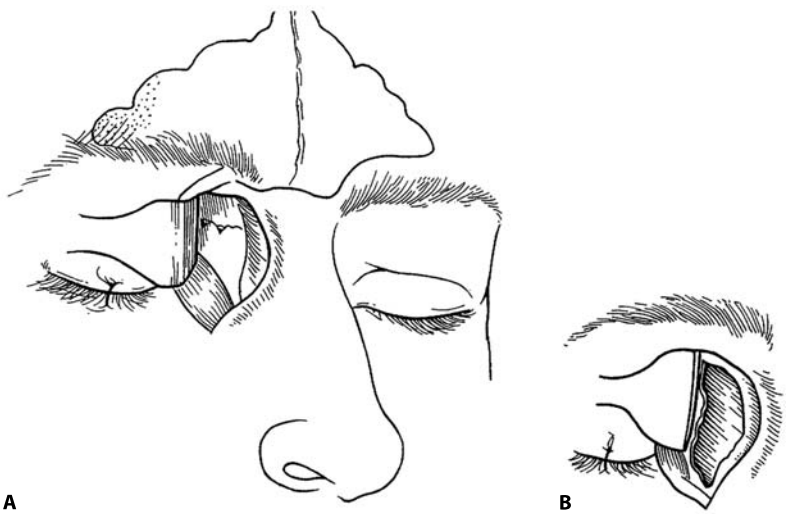

Fig. 1.2.
Lynch incision (**A**) with resulting access to frontal sinus and ethmoid sinuses (**B**)

crease the success rate and decrease failure and restenosis rates [3, 30, 40]. They described using a local mucoperiosteal flap to line and re-epithelialize the nasal frontal drainage pathway area. They also used a silicone tube to stent the frontal ostium, and they recommended leaving the stent in place for 4 weeks. Later several other authors lined the frontal drainage pathway with a mucoperiosteal flap to prevent restenosis and reported early success rates of about 90% [29]. Dedo, using the Sewall/Boyden technique, reported a success and patency rate of 97% at 6 year follow-up [8]. This era of utilizing modifications of the Lynch external frontoethmoidectomy continued to be the procedure of choice extending from its description in 1921 to the 1950s. Walsh in 1943, in an attempt to solve the problem of restenosis and the need for stenting, described a modification of the Lynch procedure in which the frontal drainage pathway membrane was left intact [43]. He came to those observations after he performed an experimental study on three groups of dogs. Brown, in accord with Walsh's idea, reported in 1946 a procedure to preserve the frontal drainage pathway mucosa in an attempt to reduce the failure drainage pathway and restenosis rates [5]. The problem of stenosis was significant enough that many researchers devised stents made of different materials in attempts to solve the problem [12]. Despite those modifications and stent techniques, long-term failure rates up to 30% were still reported, necessitating the continued development of better surgical procedures for the frontal sinus [29].

Osteoplastic Anterior Wall Approach to the Frontal Sinus (1958)

The osteoplastic anterior wall approach to the frontal sinus was described at the turn of the 19th century by several authors including Brieger, Schoenborn, Winkler, and later Beck and others [1, 4, 9, 39]. However, little attention was paid to this technique at the turn of the century, because of the concern about the difficulty of re-approximation of the bony flap to its original position. Osteomyelitis, infection of the bone, was also thought to be a major morbid condition of the procedure. Tato and Bergaglio in 1949 [42], and Lyman in 1950 [27] reported on obliterating the frontal sinus for frontal sinusitis with success and no cosmetic deformity.

In 1958 Goodale and Montgomery reported a series of seven patients who had an osteoplastic flap with an excellent success rate [13]. Montgomery stated that "intranasal probing and attempted enlargement or cannulization of the nasal frontal orifice are mentioned only to be condemned. Once the virginity of the nasofrontal passage has been violated, scarring and stenosis are inevitable." The osteoplastic frontal sinus procedure gained popularity in the 1960s and became the standard during that time (Fig. 1.3). A failure rate of less than 9% made this procedure popular among physicians. The use of a radiographic plate to outline the frontal sinus as described by Becker was a great advantage to safely elevate the bony flap [2].

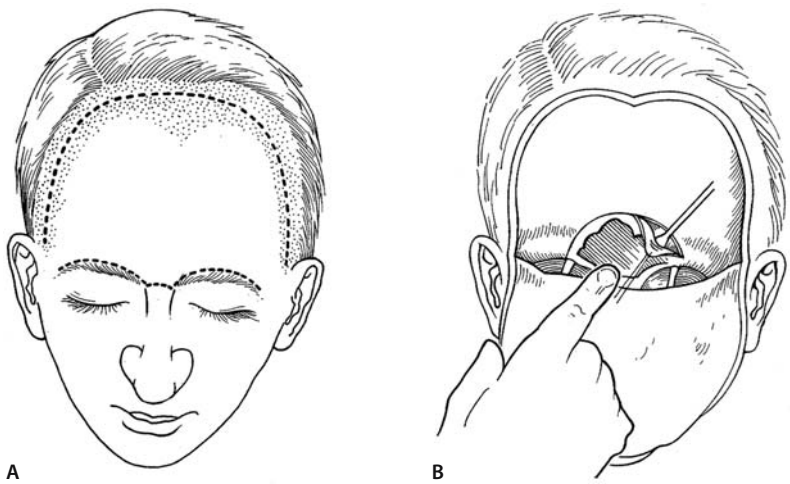

Fig. 1.3.
Osteoplastic frontal sinusotomy illustrating incision options (A) with resulting exposure and elevation of the anterior table of the frontal sinus (B)

A lot of experience accumulated with this technique, and Hardy and Montgomery reported in 1976 a 95% success rate with a median follow-up of 3 years [17]. Wide et al. in 1997 reported a 62% success rate with an additional 21% of patients achieving success after revision surgery [46].

Many otolaryngologists did not feel that the osteoplastic flap with fat obliteration was the answer to frontal sinus disease. They noted that it was an invasive procedure, which is technically difficult. It carries with it a high blood loss with potential for cosmetic deformity and poor scar formation. Many patients experience frontal neuralgias with numbness of the forehead. An additional operative site is needed for harvesting fat with potential morbidities. Long-term follow-up is necessary because of potential mucocele formation, and the presence of fat in the sinus makes it difficult to diagnose other frontal sinus problems [33].

Despite the popularity and the wide use of the osteoplastic flap, many physicians were not satisfied and did not feel that it was the ultimate procedure.

Microscopic/Endoscopic Intranasal Approaches (1991)

Earlier intranasal frontal sinus procedures had a high complication rate due to poor visualization. In 1990, Schaefer and Close reported on the use of the endoscope to treat 36 patients with frontal sinus disease. They performed endoscopic frontal sinusotomy with 12 patients reporting complete resolution of symptoms and 11 reporting improvement [37]. Draf, in 1991, reported on a series of 100 patients in which he used both a microscope and an endoscope to perform intranasal frontoethmoid surgery for frontal sinus disease. He described a concept of three procedures with a 90% success rate. He reported no complications with this endoscopic technique. All 10% of his failures had an open osteoplastic obliterative procedure. The Draf procedures were aimed at opening the frontal ostium intranasally and allowing the sinus to drain. Draf I consisted of an anterior ethmoidectomy with opening of the nasofrontal duct (NFD). Draf II in addition consists of unilateral resection of the floor of the frontal sinus; Draf III is bilateral resection of the frontal sinus floor [10].

With the advent of the endoscope, several authors have recently reported on the use of the endoscope to open the frontal sinus ostium and establish drainage of the frontal sinus. The advantages included lower morbidity rates, a shorter hospital stay, a less invasive procedure, and no external scarring [6, 15, 21, 31].

Kountakis and Gross in 2003 reported on long-term results of the modified Lothrop procedure and noted that with advancement of instrumentation and improved skills of surgeons with endoscopic procedures, success has been similar to that of the open osteoplastic approach with obliteration [24]. Stankiewicz and Wachter in 2003 reported a 90% success rate with the endoscopic approach for patients who had an osteoplastic approach and failed [41].

Conclusion

Currently, most otolaryngologists will initially perform an endoscopic procedure in most cases of chronic frontal sinusitis. An open procedure is usually reserved for patients with absent or distorted intranasal landmarks, failed endoscopic approaches, complicated frontal sinusitis, and evidence of lateral disease or posterior table erosion.

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Chapter 2

Radiologic Anatomy of the Frontal Sinus

2

Ramon E. Figueroa, Joseph Sullivan

Core Messages

- The frontal sinus and its drainage pathway comprise one of the most complex anatomic areas of the anterior skull base, amplified by significant variability
- Improvements in radiologic imaging clarity along with multiplanar demonstration of frontal sinus complex anatomy have paralleled and augmented advances in the surgical management of the frontal sinuses

Contents

Introduction

The frontal sinus and its drainage pathway comprise one of the most complex anatomic areas of the anterior skull base. Its complexity is magnified by the frequency of anatomic variations which impact the direction of drainage, efficiency of mucociliary clearance, and morphology of the frontal recess. Recent significant advances in computed tomography (CT), especially the introduction of multidetector helical scanning and the routine availability of computer workstations, have made demonstration of this complex anatomy easier and more useful to rhinologic surgical approach. This improvement in imaging clarity and multiplanar demonstration of frontal sinus complex anatomy is now of even more clinical relevance in view of the extensive developments in powered instruments, better endoscopic devices, and surgical navigation with CT cross-registration.

Embryologic and Functional Concepts

The sinonasal embryologic development during the first trimester is characterized by the emergence of more than six ethmoturbinals, which progressively coalesce and differentiate into the final anatomy of the lateral nasal wall [6].

The ethmoturbinals give rise to the following structures:

- The most superior remnant of the first ethmoturbinal becomes the agger nasi mound
- The remnant of the descending portion of the first ethmoturbinal becomes the uncinate process
- The basal lamella of the second ethmoturbinal pneumatizes and gives origin to the bulla ethmoidalis
- The basal lamella of the third ethmoturbinal becomes the basal lamella of the middle turbinate.

The nasal mucosa invaginates at specific points in the lateral nasal wall, forming nasal pits that develop into the anlages of maxillary, frontal sinuses, and ethmoid cells [2]. The mesenchyme resorbs around the invagination of the nasal pits, allowing progressive development of the sinus cavity. The embryologic point at which the initial invagination occurs becomes the future sinus ostium. Cilia develop and orient towards this ostium, allowing mucus to flow towards and through the ostium. The efficiency of the mucociliary drainage is then dictated and impacted by the patency, tortuosity, and/or frank narrowing of the resulting drainage pathways, which are progressively modified by the sequential ongoing pneumatization process occurring during the patient's life. Typically the ethmoid cells and the maxillary antra are pneumatized at birth, with the maxillary antra progressively expanding into mature sinuses as the maxilla matures and the teeth erupt. The frontal sinus develops and expands in late childhood to early adolescence, and continues to grow into adulthood. The rate of sinus growth is modified by the efficiency of ventilation and mucociliary drainage, dictated by the sinus ostium and corresponding drainage pathways. The frontal sinus drainage pathway is the most complex of all sinuses, impacted by its anatomic relationships with the agger nasi, anterior ethmoid cells, and pattern of vertical insertion of the uncinate process [3].

Frontal Sinus Evaluation

CT of the paranasal sinuses classically has been performed with continuous coronal and axial 3-mm slices to provide two planes of morphologic depiction of sinus anatomy for presurgical mapping and evaluation [5]. Recent advances in CT scanner designs with the introduction of multidetector helical designs and much larger and faster computing processing capacities now allow for single-plane thin-section high-resolution databases to be acquired and postprocessed to depict the sinus anatomy in any planar projection with high definition of the underlying anatomy. This multiplanar capability has impacted the evaluation of the frontal sinus drainage pathways the most, since depiction of this region in a sagittal plane has become routine.

Typical high-resolution multidetector scanning is performed in the axial plane (Fig. 2.1A) following the long axis of the hard palate, using a low MA tech-

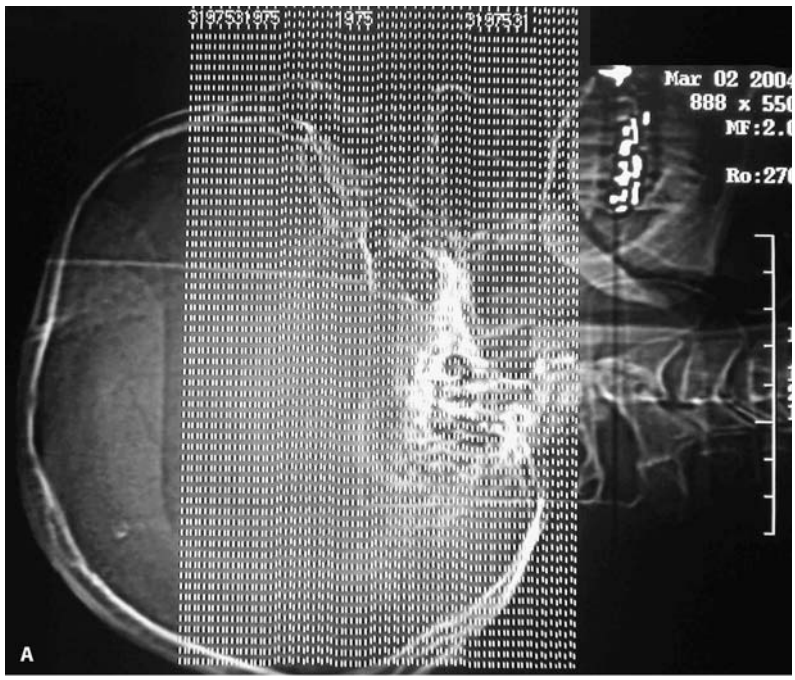
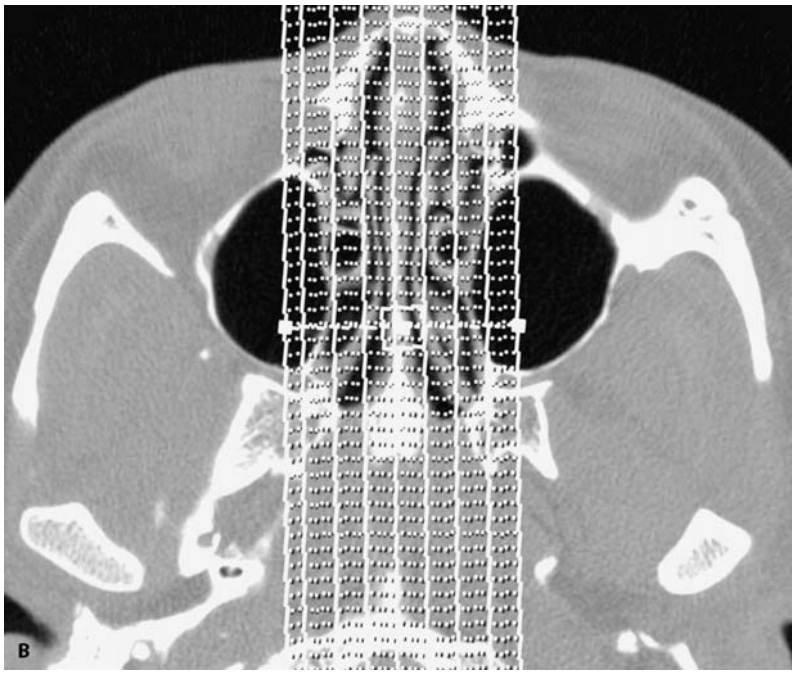


Fig. 2.1A,B.
High-resolution sinus navigation CT protocol. **A** Lateral scout view shows the typical prescription of axial thin section slices. **B** An axial image at the level of the nasal cavity helps prescribe the sagittal reformatted images

Fig. 2.1B.



nique, a small field of view (18–20 cm), and 1.25 mm collimation, with data back-processed in 0.65-mm thickness in bone algorithm and displayed in mucosal (window of 2000, level of –200) and bone (3500/800) detail. Most centers use this pattern of data acquisition for 3D computer-assisted surgical navigation. Interactive evaluation of the data is then performed on the CT workstation to define a sagittal plane perpendicular to the hard palate, prescribing a set of sequential sagittal sections to encompass both frontal sinuses and their corresponding drainage pathways (Fig. 2.1B).

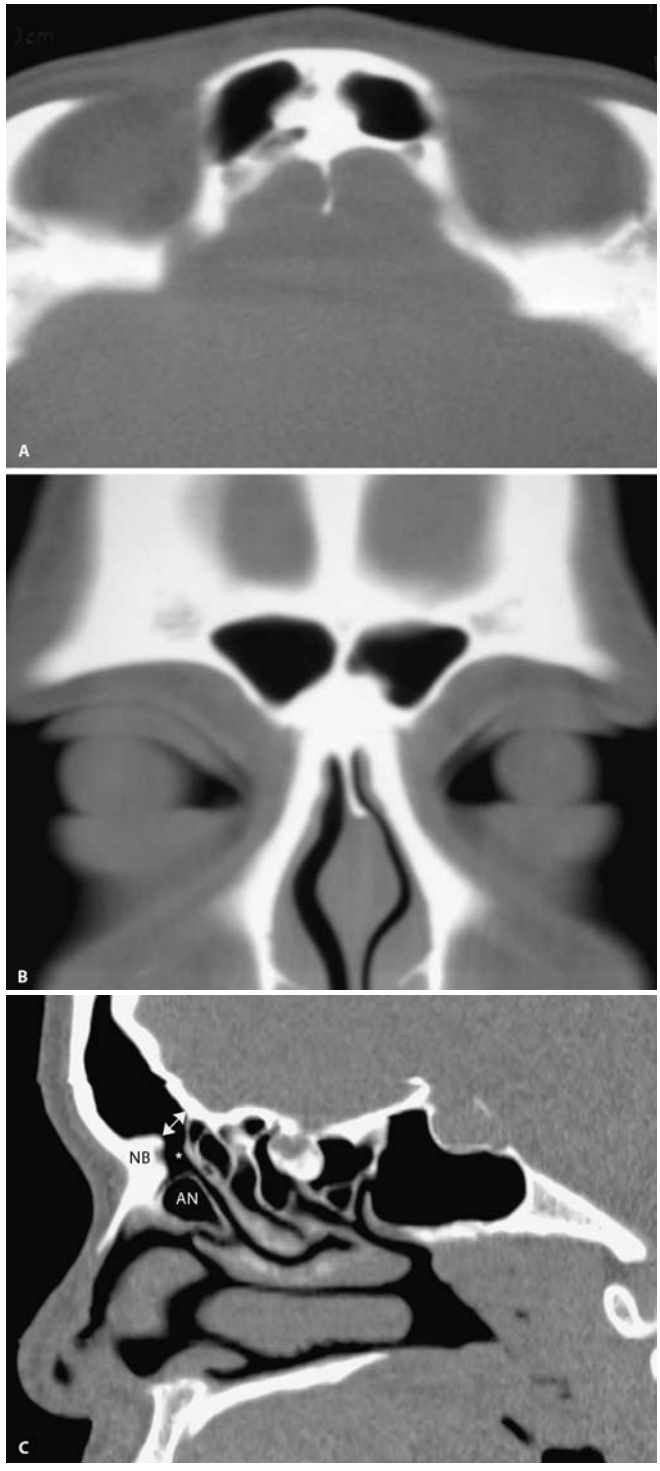
Frontal Sinus Drainage Pathway

The frontal sinus grows and expands within the diploic space of the frontal bone from the frontal sinus ostium medial and superior to the orbital plates, enclosed anteriorly by the cortical bone of the anterior frontal sinus wall and posteriorly by the cortical bone of the skull base and posterior frontal sinus wall (-which is also the anterior wall of the anterior cranial fossa). Each frontal sinus grows independently, with its rate of growth, final volume, and configuration dictated by its ventilation, drainage, and the corresponding growth (or lack of it) of the competing surrounding sinuses and skull base.

The frontal sinus narrows down inferiorly and medially into a funnel-shaped transition point, which is defined as the frontal sinus ostium (Fig. 2.2A,B), extending between the anterior and posterior frontal sinus walls at the skull base level. This point is typically demarcated along its anterior wall by the variably shaped bone ridge of the nasofrontal buttress, frequently called the "nasal beak" (Fig. 2.2C). The frontal sinus ostium is oriented nearly perpendicular to the posterior wall of the sinus at the level of the anterior skull base [3].

The Anatomic Terminology Group defined the frontal recess as "the most anterior and superior part of the anterior ethmoid complex from where the frontal bone becomes pneumatized, resulting in a frontal sinus" [7]. In sagittal plane, the frontal recess frequently looks like an inverted funnel (Fig. 2.2C) that opens superiorly to the frontal sinus ostium. The anatomic walls of surrounding structures dictate its walls and floor. The lateral wall of the frontal recess is defined by the lamina papyracea of the orbit (Fig. 2.3). The medial wall is defined by the vertical attachment

Fig. 2.2A–C.
The frontal sinus ostium. Axial (A), coronal (B), and sagittal (C) images at the level of the frontal sinus illustrate the frontal sinus ostium (arrows), the frontal recess (*), the nasal beak (NB), and the agger nasi (AN) cells

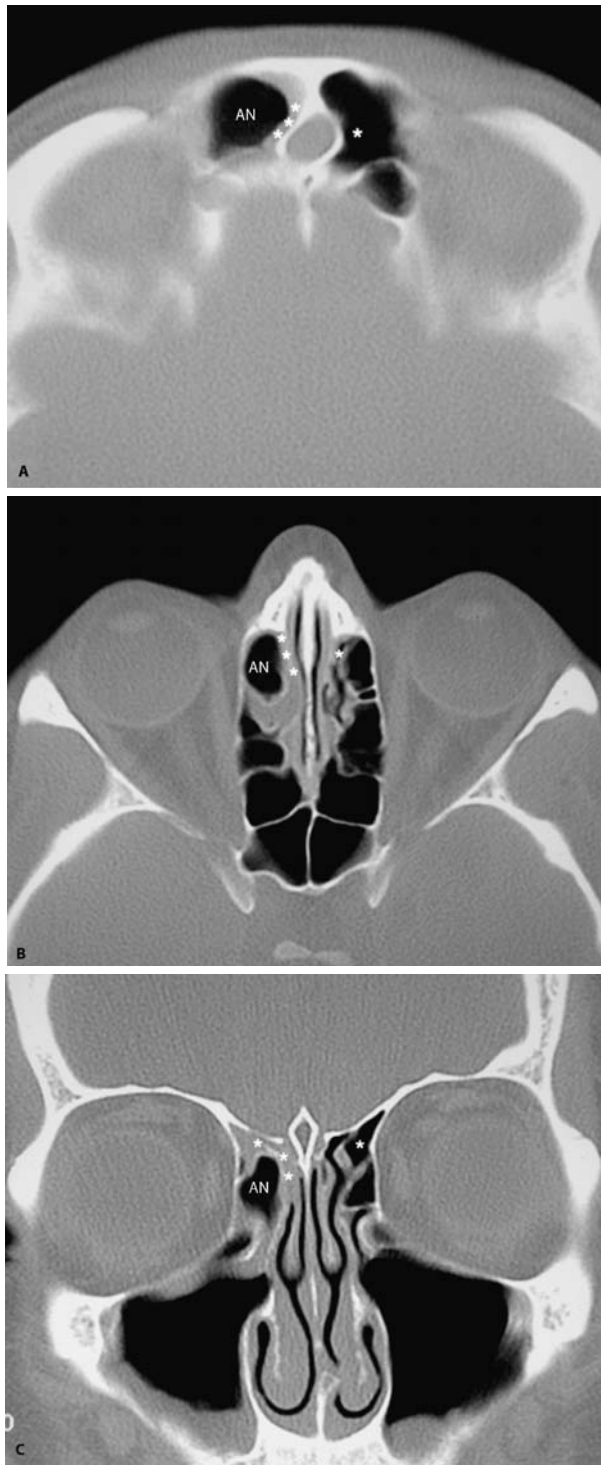


of the middle turbinate (its most anterior and superior part). Its posterior wall is variable, depending on the basal lamella of the bulla ethmoidalis reaching (or not) the skull base, if it is dehiscent allowing a communication with the suprabullar recess, or if it is hyper-pneumatized producing a secondary narrowing of the frontal recess from it posterior wall [2].

The agger nasi cells and the uncinate process dictate the floor and the pattern of drainage of the frontal recess. The frontal recess can be narrowed from the anterior-inferior direction by hyper-pneumatized agger nasi cells (Fig. 2.3). Its inferior drainage is dictated by the insertion of the vertical attachment of the uncinate process, a sagittally oriented hook-like bony leaflet (Fig. 2.4). Whenever the uncinate process attaches to the skull base or the superior-anterior portion of the middle turbinate, the frontal recess drains into the superior end of the ethmoidal infundibulum (Fig. 2.4A). If the uncinate process attaches laterally into the lamina papyracea of the orbit (Fig. 2.4B), the frontal recess opens directly into the superior aspect of the middle meatus, and the ethmoidal infundibulum ends blindly into a "terminal recess".

The ethmoidal infundibulum is a true three-dimensional space defined laterally by the lamina papyracea, anteromedially by the uncinate process, and posteriorly by the bulla ethmoidalis (Fig. 2.5A). It opens medially into the middle meatus across the hiatus semilunaris inferior, a cleft-like opening between the free posterior margin of the uncinate process and the corresponding anterior face of the bulla ethmoidalis (Fig. 2.5B). It is the functional common pathway of mucociliary drainage for the anterior ethmoid, agger nasi, and maxillary sinus mucus. The frontal sinus drainage can also drain through the ethmoidal infundibulum if the uncinate process does not attach to the lamina papyracea of the orbit.

Fig. 2.3A–C.
The frontal recess. A large right agger nasi cell (AN) is stenosing the right frontal recess (***), which is opacified by congested mucosa and can be followed on coronal and sequential axial images. The left frontal recess (*) is well aerated



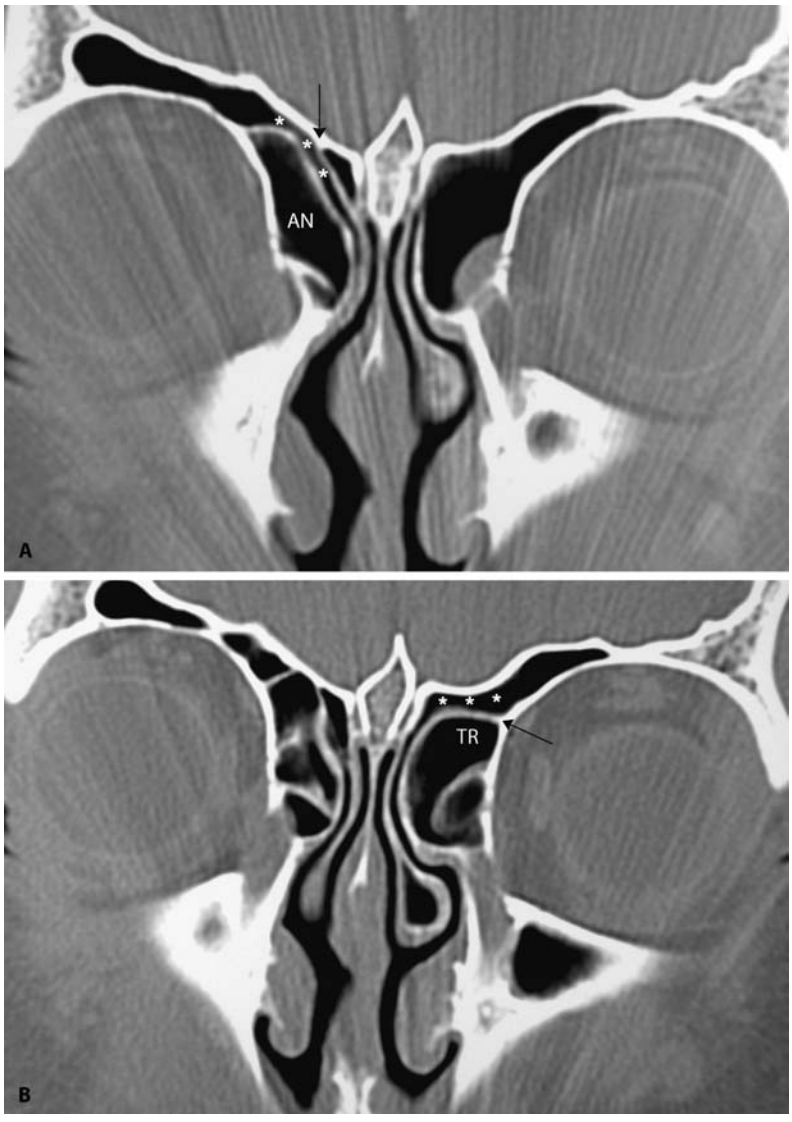
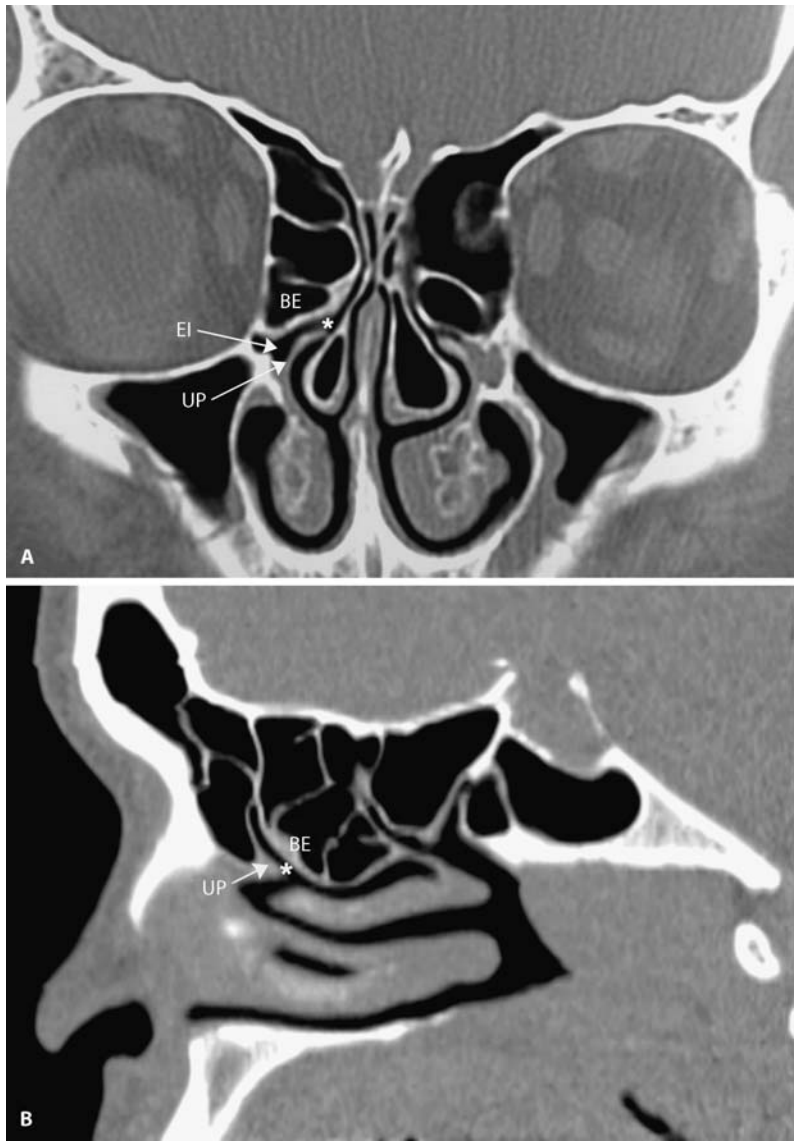


Fig. 2.4A,B.
The uncinate process. In coronal image **(A)** the uncinate process attaches to the skull base (black arrow), with the frontal recess (***) continuing downwards between the agger nasi cell (AN) and the uncinate process. In coronal image **(B)** the uncinate process attaches to the lamina papyracea (black arrow), with the frontal recess (***) opening directly to the middle meatus, and the ethmoidal infundibulum (EI) ending in a blind end or "terminal recess" (TR)

Fig. 2.5A,B.
The ostiomeatal complex. In coronal image **(A)** the ethmoid infundibulum (EI) lies between the uncinate process (UP) and the bulla ethmoidalis (BE), opening into the middle meatus across the hiatus semilunaris inferior (*). Notice the bilateral concha bullosa and the deep olfactory fossae (Keros type III). In sagittal image **(B)** the uncinate process (UP), bulla ethmoidalis (BE), and hiatus semilunaris inferior (*) are shown better as sagittally oriented landmarks



Anatomic Variants

Several important anatomic variants impact on the anatomy of the frontal sinus drainage pathways and the anterior skull base. Familiarity with these anatomic variants is required for safe anterior skull base and frontal recess surgical considerations.

Frontal Cells

The frontal cells are rare anatomic variants of anterior ethmoid pneumatization that impinge upon the frontal recess and typically extend within the lumen of the frontal ostium above the level of the agger nasi cells (Fig. 2.6). Bent and coworkers described four types of frontal cells [1]. All frontal cells can be clinically significant if they become primarily infected or if they obstruct the frontal sinus drainage, leading to secondary frontal rhinosinusitis.

The different types of frontal cells as described by Bent are [1]:

- Type I frontal cell, a single frontal recess cell above the agger nasi cell (Fig. 2.6A)
- Type II frontal cells, a tier of cells above the agger nasi cell, projecting within the frontal recess
- Type III frontal cell is defined as a single massive cell arising above the agger nasi, pneumatizing cephalad into the frontal sinus (Fig. 2.6B)
- Type IV frontal cell is a single isolated cell within the frontal sinus, frequently difficult to visualize due to its thin walls (Fig. 2.6C)

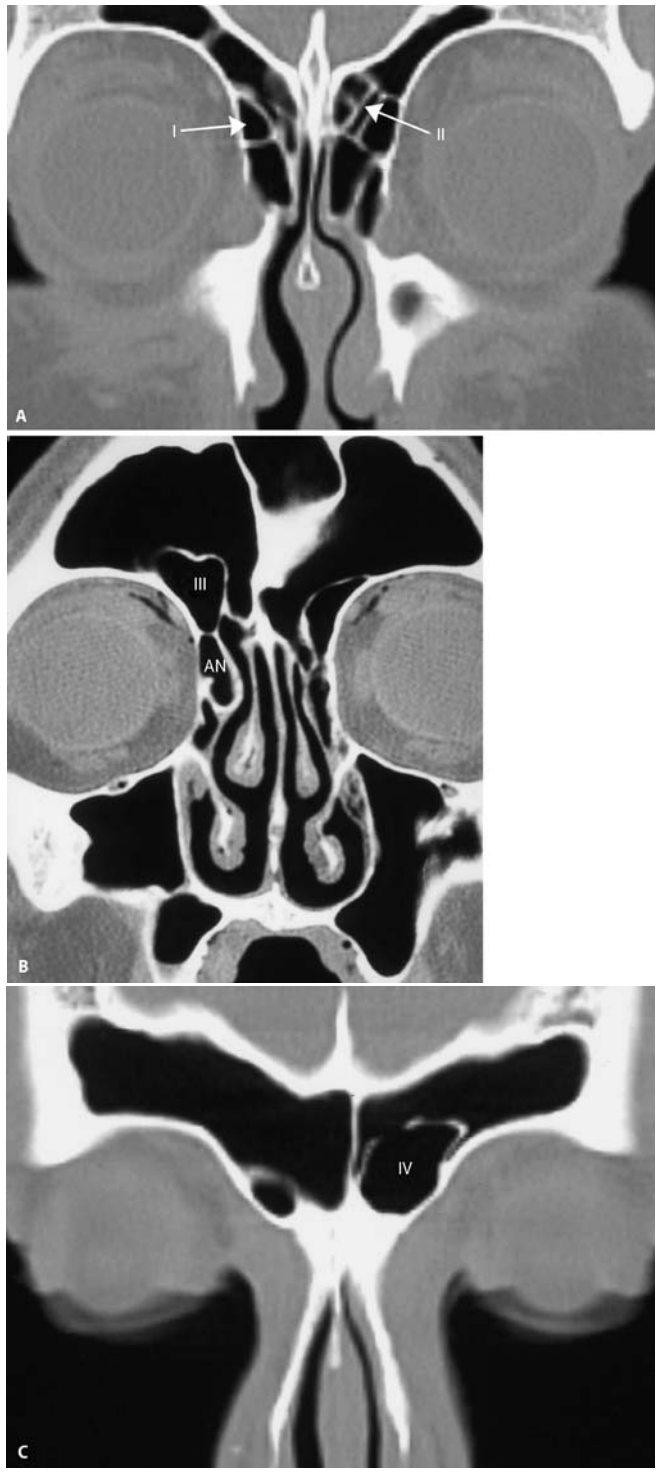
Supraorbital Ethmoid Cell

This is a pattern of pneumatization of the orbital plate of the frontal bone posterior to the frontal recess and lateral to the frontal sinus (Fig. 2.7), frequently developing from the suprabullar recess [2]. The degree of pneumatization of the supraorbital ethmoid cells can reach the anterior margin of the orbital plate and mimic a frontal sinus. Tracing back the borders of the air cell towards the anterior ethmoid behind the frontal recess allows us to recognize this variant better.

Depth of Olfactory Fossa

The orbital plate of the frontal bone slopes downwards medially to constitute the roof of the ethmoid labyrinth (foveola ethmoidalis), ending medially at the lateral border of the olfactory fossa (Fig. 2.8). This configuration makes the olfactory fossa the lowermost point in the floor of the anterior cranial fossa, frequently projecting between the pneumatized air cells of both ethmoid labyrinths [7]. The depth of the olfactory fossa into the nasal cavity is dictated by the height of the lateral lamella of the cribriform plate, a very thin sagittally oriented bone that defines the lateral wall of the olfactory fossa.

Fig. 2.6A–C.
Frontal cells. Frontal cells are rare air cells above agger nasi that impinge upon the frontal recess and frontal sinus. Type I is a single cell above agger nasi, while type II is a tier arrangement above agger nasi. Type III is a single large frontal cell projecting into the frontal sinus lumen. Type IV is a large cell completely contained in the frontal sinus ("sinus within a sinus)



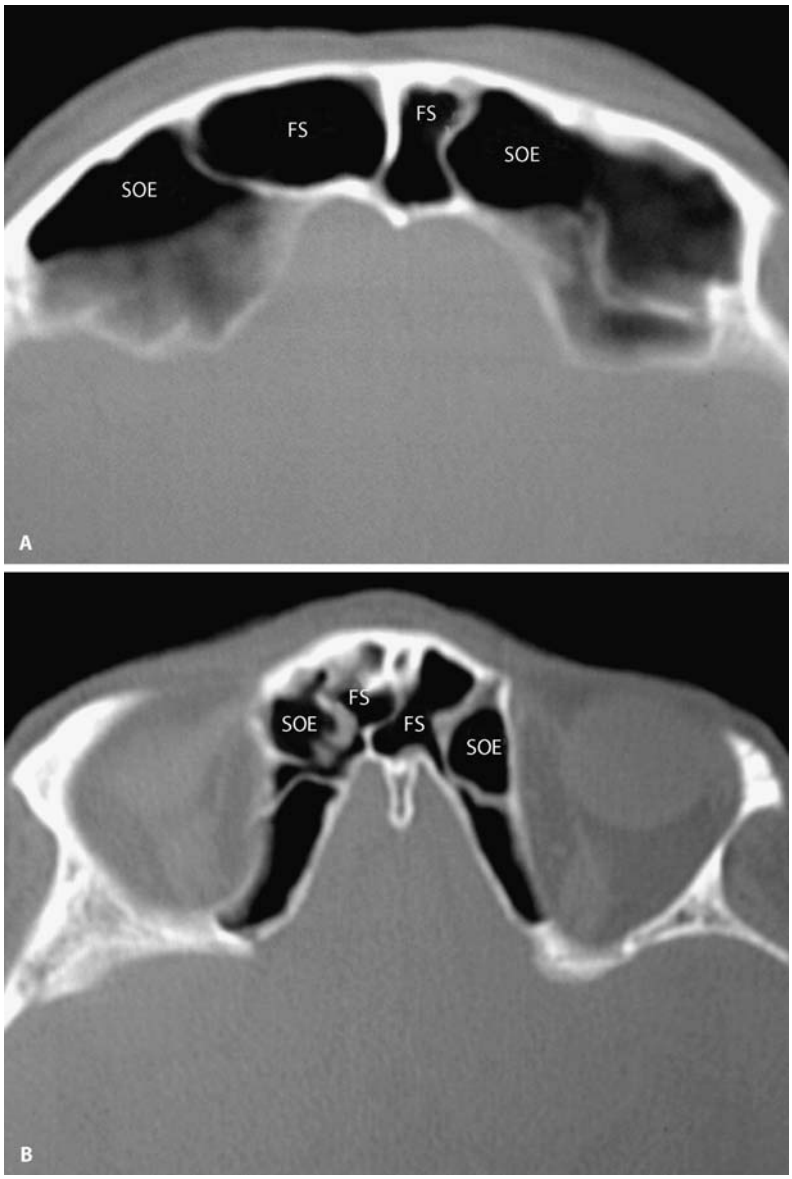


Fig. 2.7A–C.
Supraorbital Ethmoid Cells. In the sequential axial images **A–C** the supraorbital ethmoid cells (SOEs) expand and pneumatize anteriorly into the orbital plate of the frontal bone, not to be confused with the frontal sinus (FS)

Fig. 2.7C.

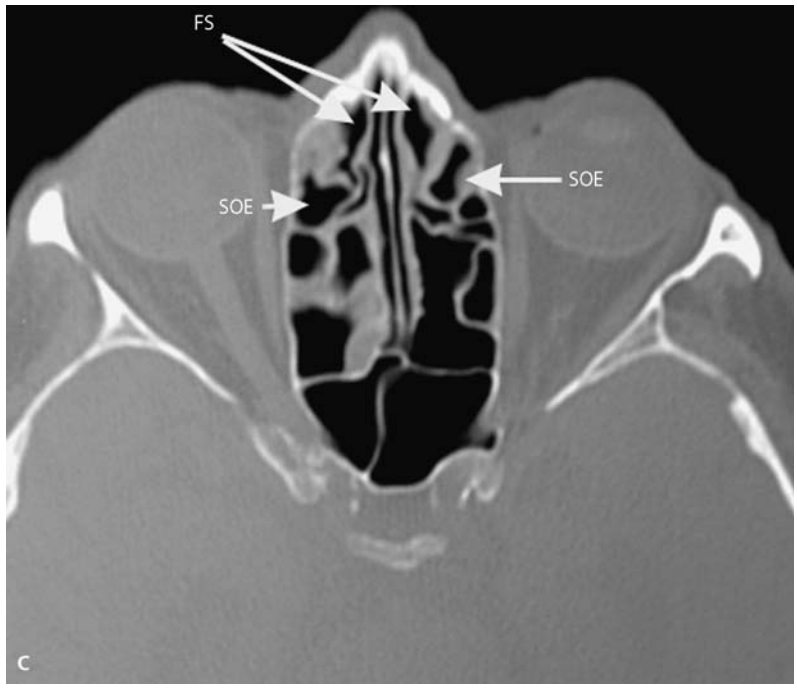


Fig. 2.8A–C.
Depth of olfactory fossa. The length of the lateral lamella of the cribriform plate (white arrows) determines the depth of the olfactory fossa, categorized by Keros in Type I (**A**, 1–3 mm deep), Type II (**B**, 4–7 mm deep) and Type III (**C**, 8–16 mm deep)

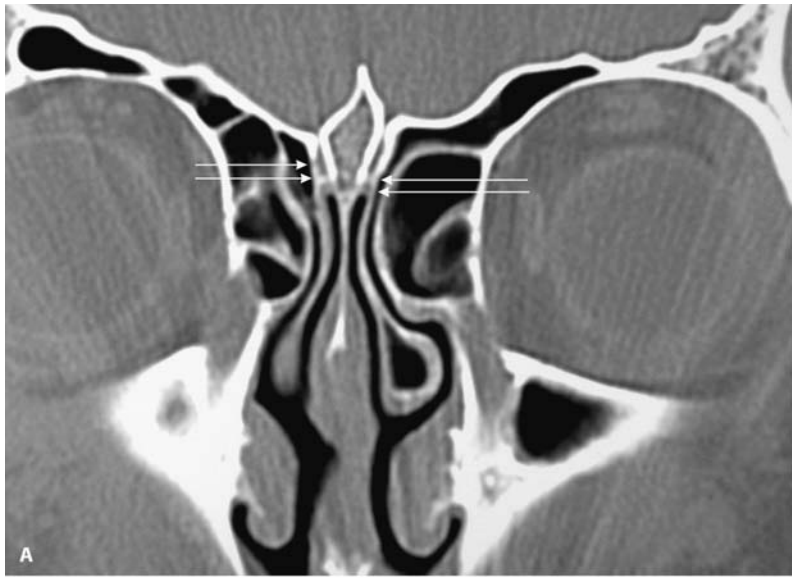
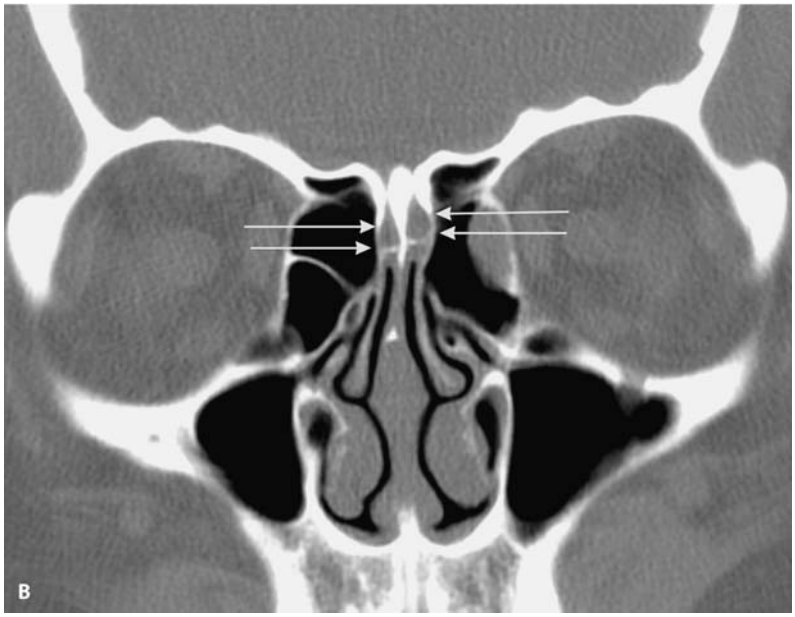
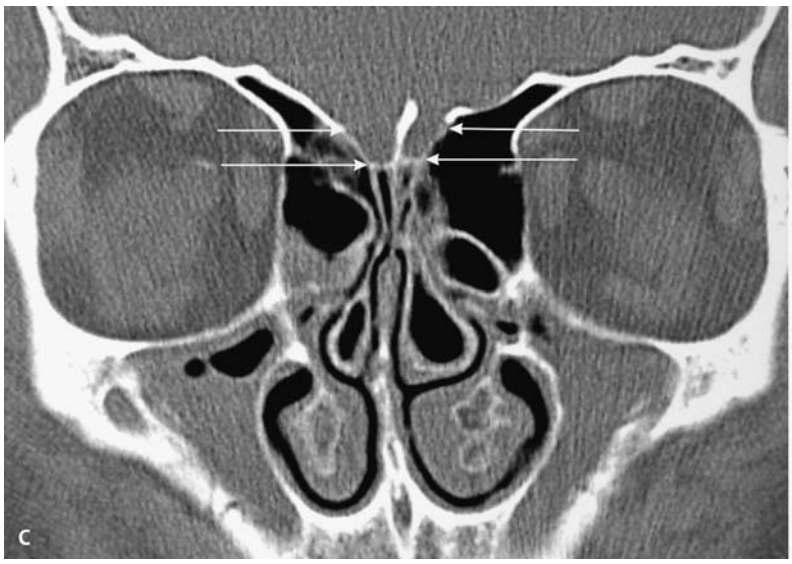


Fig. 2.8B,C.





Keros described the anatomic variations of the ethmoid roof and the olfactory fossa, classifying it in three surgically important types [4]:

- Type I has a short lateral lamella, resulting in a shallow olfactory fossa of only 1–3 mm in depth in relation to the medial end of the ethmoid roof
- Type II has a longer lateral lamella, resulting in an olfactory fossa depth of 4–7 mm
- Type III olfactory fossa has a much longer lateral lamella (8–16 mm), with the cribriform plate projecting deep within the nasal cavity well below the roof of the ethmoid labyrinth.

The type III configuration represents a high-risk area for lateral lamella iatrogenic surgical perforation in ethmoid endoscopic surgical procedures. Occasionally there may be asymmetric depth of the olfactory fossa from side to side, which must be recognized and considered prior to surgery.

Conclusion

The frontal sinus drainage pathways and the surrounding anterior ethmoid sinus represent one of the most complex anatomic regions of the skull base. An intimate knowledge of its anatomy and a clear understanding of its physiology and anatomic variants are required for safe and effective surgical management of frontal sinus drainage pathway problems.

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Chapter 3

Surgical Anatomy and Embryology of the Frontal Sinus

3

Carlos S. Duque, Roy R. Casiano

Core Messages

- **(Overview)** A thorough knowledge of frontal sinus anatomy is critical when performing even basic endoscopic sinus surgical procedures. Every endoscopic sinus surgeon must be aware of all the normal, as well as the abnormal, variants that may exist
- The number and size of the paranasal sinuses are determined early during embryologic development. Disease processes during childhood or early adulthood may modify this anatomy or its relationship to neighboring structures
- The close relationship between the frontal sinus and neighboring orbit or anterior skull base makes it particularly vulnerable to complications from disease or surgery

Contents

Introduction

As with any surgical procedure, a thorough knowledge of anatomy is the one most important factors in minimizing complications and maximizing one's chances of a good surgical outcome. This is particularly important for otolaryngologists performing endoscopic sinus surgery, as each and every one of the paranasal sinuses are in close proximity to critical orbital and skull base structures. A good knowledge of anatomy will enable the surgeon to operate with more confidence, by improving one's ability to correctly interpret normal variants from abnormal or pathological conditions, and determine an appropriate surgical treatment plan to reestablish mucociliary flow to the sinus. This is even more critical for distorted anatomy, due to previous surgery or neoplasms. Furthermore, CT imaging has become an integral part of the diagnostic armamentarium for sinus surgeons. Technological advancements such as intraoperative navigational devices depend on the surgeon's proper identification of normal or abnormal structures on CT or MRI scans. However, despite this technology's intent of reducing complications, failure to know the sinus anatomy or properly identify critical structures on the scan may still result in disastrous consequences.

The frontal sinus hides in the anterior cranial vault surrounded by two thick layers of cortical bone. Its naturally draining "ostium", or frontal infundibulum, remains immersed in an intricate complex area covered by ethmoid cells and other anatomical structures that may not be so easy to find. In order to better understand frontal sinus anatomy, one must begin with its embryological development.

Embryology of the Frontal Sinus

It is important to know that all of the development of the head and neck, along with the face, nose, and paranasal sinuses, takes place simultaneously in a very short period of time. Frontal sinus development begins around the fourth or fifth week of gestation, and continues not only during the intrauterine growth period, but also in the postnatal period through puberty and even early adulthood.

By the end of the fourth week of development, one begins to see the development of the branchial arches, along with the appearance of the branchial pouches and the primitive gut. This gives the embryo its first appearance of an identifiable head and face, with an orifice in its middle, called the stomodeum (Fig. 3.1). This structure is surrounded by the mandibular and maxillary arches or prominences, bilaterally. Both of these prominences are derivatives of the first branchial arch. This arch will ultimately give rise to all of the vascular and neural structures supplying this area. The newly developed stomodeum is limited superiorly by the frontonasal prominence and inferiorly by the mandibular arch [10, 15, 16].

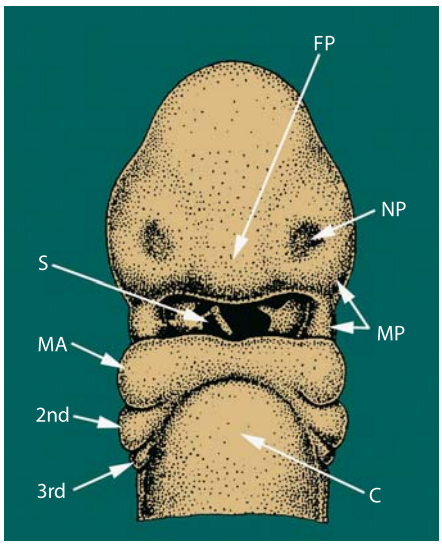

Fig. 3.1. Ventral view of a 5-week-old embryo, showing the stomodeum (S), mandibular arch (MA), 2nd branchial arch (2nd), 3rd branchial arch (3rd), frontonasal prominence (FP), nasal placode (NP), maxillary prominences (MP), and cardiac bulge (C)

The frontonasal prominence differentiates inferiorly with two nasal projections, or nasal placodes, that will be invaded by the growing ectoderm and mesenchyme. These structures later fuse and form the nasal cavity and primitive choana, separated from the stomodeum by the oronasal membrane. The primitive choana will be the point of development for the posterior pharyngeal wall as well as the different sinuses. The oronasal membrane will be fully formed by the end of the fifth week of development, to form the floor of the nose (palate). As the embryo grows, the maxillary processes and the nasal placodes come together in the midline, to form the maxillary bone and the beginning of the external nose. The frontonasal prominence will also develop a caudal mesodermic projection that will form the nasal septum, diving the nose into two chambers [15–17].

Simultaneously, while all these changes are starting to take place, the cranial and facial bones are forming as well. The skeletal system develops from the mesoderm, from which mesenchyme develops, forming the connective tissue (fibroblasts, chondroblasts, osteoblasts) that eventually differentiates into the various support structures of the nose and paranasal sinuses. The neural crest cells and mesenchyme migrate to the occipital area and the future site of the cranial cavity, and disperse in order to form the hyaline cartilage matrix that will later become ossified. Each cranial bone is formed by a series of bone spicules that grow from the center towards the periphery, to occupy its place. At birth, all cranial bones are separated by layers of connective tissue that later become fused and ossified in the postnatal period. Although all of these cranial structures are made out of cartilage and eventually will become ossified, they can still be invaded by neighboring epithelial cells (from the nasal cavity), eventually giving rise to the future paranasal sinuses [16, 17].

Around the 25th to 28th week of development, three medially directed projections arise from the lateral wall of the nose. This begins the process of defining the anatomical structures of the paranasal sinuses. Between these projections small lateral diverticula will invaginate into the lateral wall of the primitive choana to eventually form the nasal meati (Fig. 3.2) [15–17].

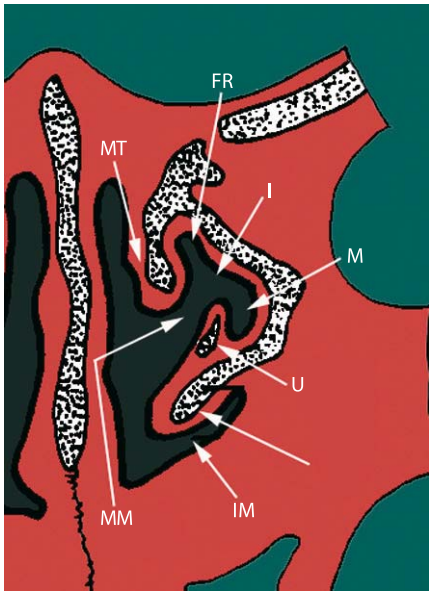

Fig. 3.2. Between the 25th and 28th week of gestation, lateral diverticula will invaginate into the lateral wall of the primitive choana to eventually form the nasal meati. Between these invaginations lie the prominences that later form the middle turbinate (MT), inferior turbinate (IT), and uncinate process (U). The infundibulum (I), maxillary sinus (M), and frontal recess (FR) are seen as small blind recesses or pockets within the middle meatus (MM). The inferior meatus (IM) is also noted

The medial projections of ectodermal tissue form the following structures:

- The anterior projection forms the agger nasi
- The inferior projection (the maxillo-turbinate) forms the inferior turbinate and maxillary sinus [16, 17]
- The superior projection, known as the ethmoido-turbinate, forms the middle and superior turbinate as well as the small ethmoidal cells, with their corresponding draining meati, between the septum and the lateral wall of the nose. Between the already formed inferior turbinate and the middle turbinate, the middle meatus will develop [14–16]

The middle meatus invaginates laterally giving shape to the embryonic infundibulum, along with the uncinate process. During the 13th week of development the infundibulum continues expanding superiorly, giving rise to the frontonasal recess. It has been proposed that the frontal sinus might develop during the 16th week simply as a direct elongation of the infundibulum and frontonasal recess, or as an upwards epithelial migration of the anterior ethmoidal cells that penetrate the most inferior aspect of the frontal bone between its two tables. Primary pneumatization of the frontal bone occurs as a slow process up to the end of the first year of life. Up to this moment, the frontal sinus remains as a small, smooth, blind pocket, and will remain this way until the infant reaches approximately 2 years of life, when the process of secondary pneumatization begins. From 2 years of age until adolescence, the frontal sinus will progressively grow and become fully pneumatized (see Fig. 3.3) [14, 15, 17]. Between 1 and 4 years of age, the frontal sinus starts its secondary pneumatization, forming a cavity no bigger than 4–8 mm long, 6–12 mm high, and 11–19 mm wide. After 3 years of age, the frontal sinus may be seen in some CT scans. When a child reaches 8 years of age, the frontal sinus becomes more pneumatized, and will be seen by most radiological studies. Significant frontal pneumatization is generally not seen until early adolescence, and continues until the child reaches 18 years of age.

Frontal sinus development may be variable. On cadaveric and radiological (CT) studies, the frontal sinus is only identifiable in less than 1.5% of infants less than one year of age [6, 8, 9]. During this period, the frontal sinus remains as a potential pocket and has been referred to as a "cellulae ethmoidalis", since the findings point clearly to its close embryological and anatomical relationship with anterior ethmoid air cells [19, 20, 25].

The frontal sinuses develop within the frontal bones. Each bone remains separated by a vertical (sagittal) suture line that becomes ossified. This will eventually form the frontal sinus intersinus septum. It is not clear which factors trigger the formation of the frontal sinuses. Some authors have speculated that the adolescent growth phase may be stimulated by the different hormonal changes or even by the process of mastication itself [13, 19, 20, 25]. The right and left frontal sinuses develop independently. Each side undergoes separate reabsorption of bone, with the formation of one, two, or even multiple cells, di-

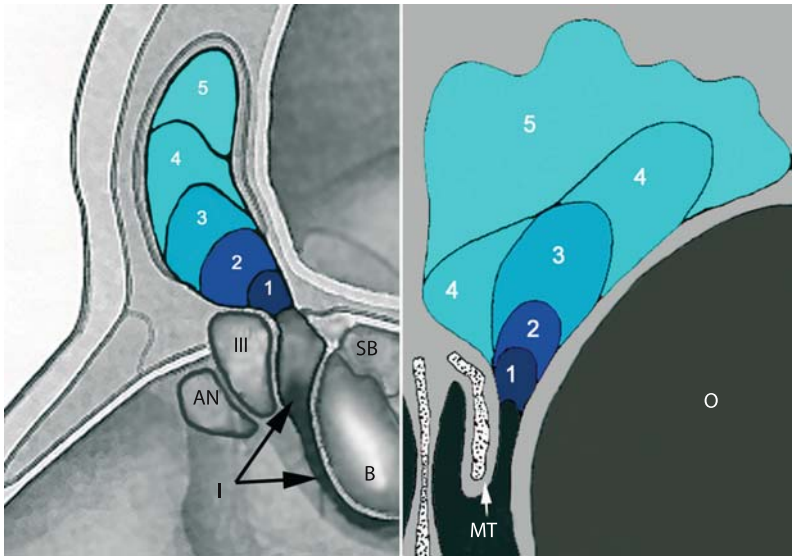

Fig. 3.3.
Sagittal and coronal views of the frontal sinus noting its progressive secondary pneumatization between the ages of 3 and 18 years of age. Between 1 and 4 years of age (1), the frontal sinus starts its secondary pneumatization. After 4 years of age, the frontal sinus may be seen as a small, but definable, cavity (2) . When a child reaches 8 years of age (3), the frontal sinus becomes more pneumatized. Significant frontal pneumatization is generally not seen until early adolescence (4), and continues until the child reaches 18 years of age (5). The agger nasi air cell (AN), type III frontal infundibular cell (III), ethmoid bulla (B), suprabullar cell (SB), middle turbinate (MT), and orbit (O) are marked

vided by various septae. Occasionally, frontal sinuses may develop asymmetrically, or even fail to develop at all. It is not uncommon to find one frontal sinus that is more "dominant" on one side, and a hypoplastic, or even aplastic frontal sinus, on the other side (Figs. 3.4 and 3.5). Aplasia of both frontal sinuses has been reported in 3%–5% of patients, depending on the study. The presence of only one well-developed frontal sinus (with a contralateral aplastic sinus) ranges from 1% to 7%. In some rare cases, pneumatization can be significant, extending out to remote areas like the sphenoid ala, orbital rim, and even the

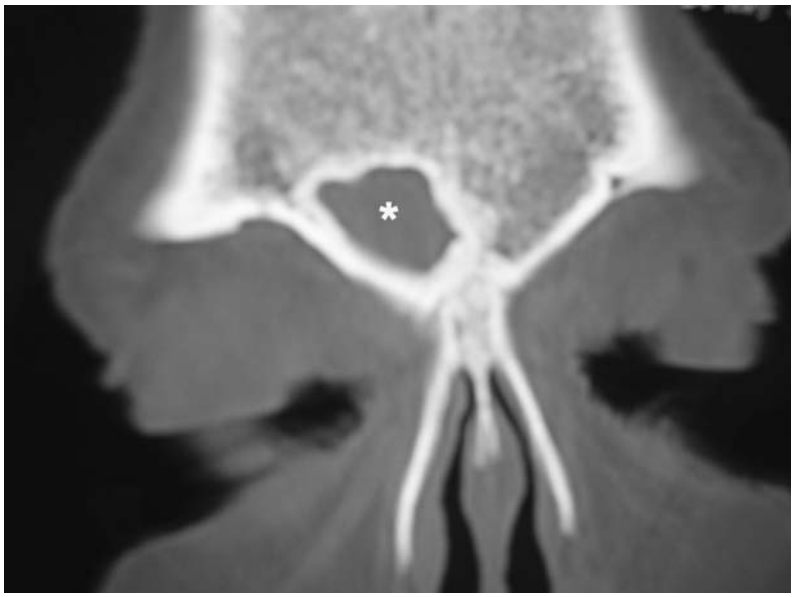

Fig. 3.4.
CT of a patient with chronic rhinosinusitis, a hypoplastic right frontal (asterisk), and aplastic left frontal

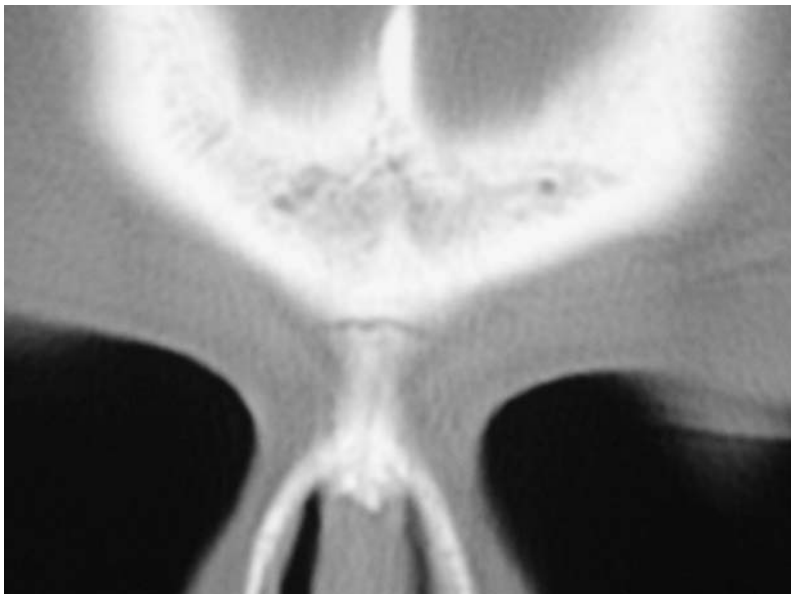

Fig. 3.5.
CT of bilaterally aplastic frontal sinuses

temporal bone. Race, geography, and climate are just a few factors that have been implicated in the abnormal development of the frontal sinus [1, 5, 19, 21, 23]. For example, bilaterally aplastic frontal sinuses have been seen in as many as 43% of Alaskan or Canadian Eskimos. Additional normal variants of frontal sinus development include the formation of as many as five frontal sinus cells, each cell with its own independently draining outflow tract into the middle meatus [5, 20].

Surgical Anatomy of the Frontal Sinuses

As seen in the previous section, the frontal sinus shares a common embryological and anatomical relationship with the ethmoid sinus, to the point that several authors and researchers have referred to this sinus as a "large ethmoidal cell" or simply the termination or upper limit of the intricate ethmoidal labyrinth [14, 15, 17].

In an adult, two frontal sinuses are usually seen. Each frontal sinus cavity takes on the shape of a pyramid, with a thick anterior table and a thinner posterior table.

The anterior wall of the frontal sinus begins at the nasofrontal suture line and ends below the frontal bone protuberance, along the vertical portion of the frontal bone. The height of the cavity at its anterior wall ranges from 1 to 6 cm, depending on the degree of pneumatization [15, 16]. It is made up of thick cortical bone measuring an average of 4 to 12 mm in thickness. This thick anterior table is covered by the pericranium (thick external periosteal layer), followed more superficially by the frontalis muscle, subcutaneous fat, and skin. This very vascularized pericranium is frequently used for reconstruction of large anterior skull base defects or for frontal sinus obliteration [14, 24].

The posterior wall of the frontal sinus forms the most anteroinferior boundary of the anterior cranial fossa, and is in close contact with the frontal lobes, separated only by the dura mater [8, 11, 14, 16, 18, 19, 24]. It has a superior vertical, and a smaller inferior horizontal, portion. The horizontal portion will form part of the orbital roof. Both posterior walls join inferiorly to form the internal frontal crest, to which the falx cerebri inserts (Fig. 3.6). The posterior table of the frontal sinus can also be inherently thin (less than a millimeter in some areas), and prone to gradual erosion and subsequent mucocele formation

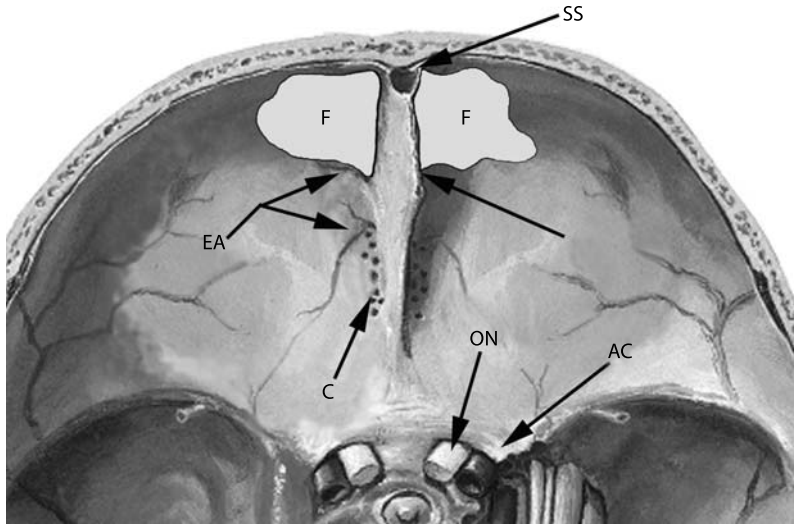

Fig. 3.6.
View of the anterior cranial fossa and orbital roof. The posterior table and extent of the frontal sinuses (F) are identified. The crista galli (CG) and superior saggital sinus (SS) demarcate the approximate level of the intersinus septum separating the right and left frontal sinuses. The crista galli is also continuous with the perpendicular plate of the ethmoid inferiorly. The cribriform plate (C) is seen on either side of the crista galli. Branches of the anterior ethmoid artery (EA) are seen reentering intracranially anterior to the cribriform plate. The optic nerve (ON) is seen entering the optic canal medial to the anterior clinoid process (AC)

from chronic inflammatory conditions [5]. The absence of bony walls cannot be addressed through a physical or endoscopic exam. However, with today's imaging studies this type of abnormality should be easily detected preoperatively.

A triangular-shaped intersinus septum separates the frontal sinuses into separately draining sinus cavities. It is the continuation, anteriorly, of the fused and ossified embryologic sagittal suture line. Although the intersinus septum may vary in direction and thickness as it proceeds superiorly, the base of the intersinus septum will almost always be close to the midline at the level of the infundibulum. At this level, the intersinus septum is continuous with the crista galli posteriorly, the perpendicular plate of the ethmoid inferiorly, and the nasal spine of the frontal bone anteriorly (Fig. 3.7). The falx cerebri inserts into the posterior table of the frontal sinus, at a point corresponding to the posterior edge of the intersinus septum. Additional intersinus septum cells may exist within this intersinus septum. Pneumatization from these intersinus cells may occasionally extend all the way into the crista galli. These cells tend to drain into the nose through their own outflow tract, adjacent to the normal frontal sinus out flow tract, at the level of the infundibulum, on one or both sides of the nose.

Inferiorly, the frontal sinus cavity is limited by the supraorbital rim and wall (or roof), through which the supraorbital neurovascular pedicle courses towards the forehead skin via the supraorbital foramen. At this level, the frontal sinus is funnel-shaped, forming the base of a pyramid. As it forms the roof of the orbit, it is also the point of insertion for the superior oblique muscle. Supraorbital pneumatization may extend as far as the lesser wing of the sphenoid. With the exception of the thin septations of the ethmoidal cells, this inferior wall of the frontal sinus makes up one of the thinnest walls of all the sinus cavities. Like the posterior table of the frontal sinus, this area is also prone to gradual erosion from chronic inflammatory conditions, giving rise to mucoceles with subsequent proptosis and orbital complications. Fortunately, the orbital periosteum (periorbita) acts as an effective barrier to serious consequences, in most of these cases.

Laterally the cavity extends itself as far as the angular prominence of the frontal bone. The superior border of the frontal sinus is the non-pneumatized cancellous bone of the frontal bone.

The frontal sinus outflow tract has been described in many ways and given all sort of names, depending on the surgical approach or perspective by which the frontal sinus is visualized [6, 9, 11]. However, today most authors agree that the frontal sinus outflow tract has an hourglass shape with its narrowest point at the level of the frontal sinus infundibulum (Fig.

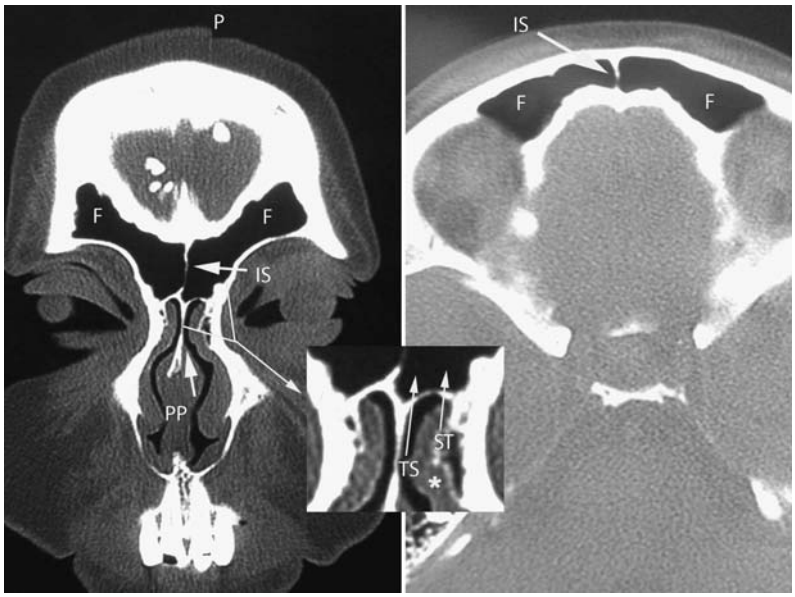

Fig. 3.7.
CT of a normal well pneumatized frontal sinus in an adult. The intersinus septum (IS) of the frontal sinus (F) is continuous with the crista galli posteriorly, the perpendicular plate of the ethmoid (PP) inferiorly, and the nasal spine of the frontal bone anteriorly. In well-pneumatized frontal sinuses, the inferomedial portion of the frontal sinus may be accessible through the nose directly via transseptal (TS) or supraturbinal approach (ST). The asterisk demarcates the anterior attachment of the middle turbinate

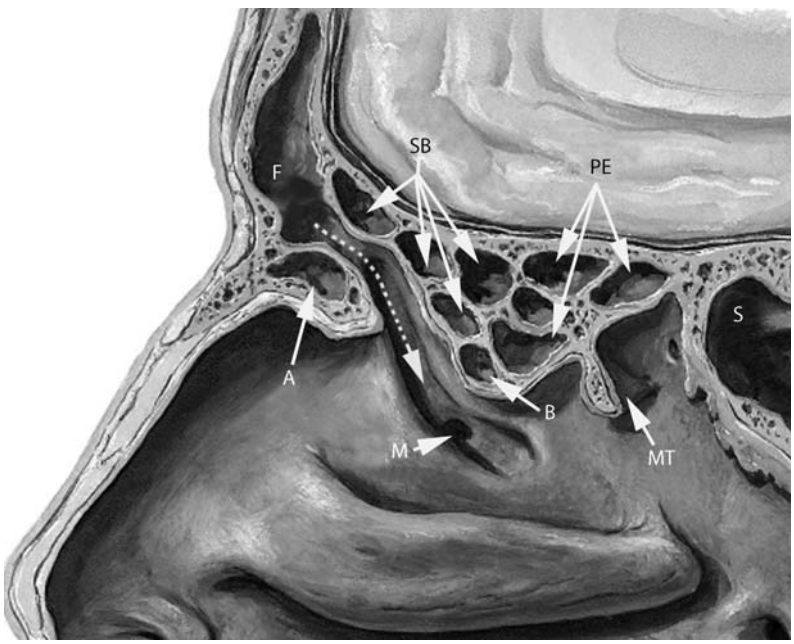

Fig. 3.8.
Sagittal section through the agger nasi (A), ethmoid bulla (B), suprabullar cells (SB), posterior ethmoid (PE), and lateral sphenoid (S). The frontal sinus (F) outflow tract is noted by the dotted arrow, coursing through the frontal infundibulum (the narrowest area in this hourglass-shaped tract), and into the ethmoid infundibulum, before exiting into the middle meatus. The uncinate process has been removed to expose the maxillary ostium (M). The tail of the middle turbinate (MT) is also noted

3.8). The frontal sinus infundibulum is formed by the most inferior aspect of the frontal sinus. It has the form of a funnel that points towards the ethmoid in a posteromedial direction. The angulation (posteromedially) and maximum diameter of this funnel may vary greatly between patients, or even between sides (Fig. 3.9) [2–4, 9, 12, 13, 16, 22].

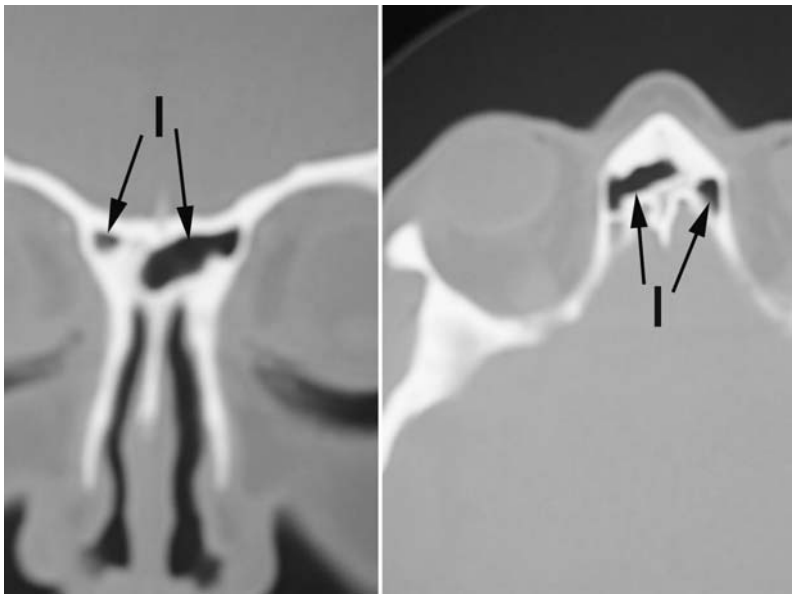

Fig. 3.9.
The right frontal sinus infundibulum is very narrowed and surrounded by thick bone. Unlike the left frontal infundibulum (which is very wide and accessible through a transnasal or supraturbinal approach), this right frontal infundibulum may be more prone to easy obstruction due to persistent inflammatory disease or from inadvertent surgical trauma with subsequent fibrosis or osteoneogenesis

The frontal sinus infundibulum is bounded by the following structures:

- The lamina papyracea laterally in its superior portion
- The middle turbinate anteriorly
- The vertical lamella medially
- The agger nasi anteroinferiorly
- The ethmoid suprabullar air cells posteriorly

A series of "accessory" ethmoidal cells may line the frontal sinus outflow tract along the frontal recess and infundibulum. These cells receive different names according to the location where they impinge on the frontal recess.

These cells include:

- The agger nasi cell
- Frontal intersinus septal cells
- Suprabullar cells (with or without supraorbital pneumatization)
- The frontal or infundibular cells.

It is important to know that these cells might be present in any given patient, not only because they might alter the normal sinus drainage if inflammatory conditions are present, but also because an endoscopic surgeon not aware of these cells might confuse them with the frontal sinus. This could result in a surgical failure due to inadequate reestablishment of frontal sinus outflow drainage and continued frontal sinus symptoms [2–4, 13].

The agger nasi cell is one of these cells. Located anterior to the superior membranous attachment of the uncinate process, the agger nasi cell is sometimes difficult to differentiate on CT imaging and even during surgery. However, with experience, its presence can be documented with CT scan in up to 98% of the cases [2, 3, 9, 13]. It is intimately related to the anterior head of the middle turbinate, along the ascending intranasal portion of the maxillofrontal suture line, and adjacent posteriorly to the lacrimal sac.

The frontal sinus can also be confused with "frontal infundibular cells". These represent a series of anterior ethmoidal cells directly superior to the agger nasi cell, coursing along the anterior wall of the frontal outflow tract. Bent and Kuhn have divided frontal infundibulum cells into four categories, based on

their relationship to the agger nasi cell and the orbital roof (Fig. 3.10) [2, 6, 9, 13].

The types frontal infundibulum cells are:

- Type I frontal cell represents a single air cell above the agger nasi.
- Type II frontal cells correspond to a series of small cells above the agger nasi, but below the orbital roof.
- Type III frontal cells extend into the frontal sinus, but remain contiguous with the agger nasi cell.
- Type IV cell corresponds to a completely isolated frontal cell (not contiguous with the agger nasi cell) within the frontal sinus cavity

Supraorbital cells may also disturb the normal frontal sinus outflow tract in diseased states. On CT these supraorbital cells are essentially suprabullar cells with significant pneumatization over the orbital roof [3, 4, 12].

The frontal sinus obtains its vascular supply from terminal vessels of the sphenopalatine artery and internal carotid artery (via the anterior and posterior ethmoid arteries). Terminal branches of the sphenopalatine artery make their way towards the frontal sinus by way of the nasofrontal recess and infundibulum. The anterior ethmoid artery (and more rarely the posterior ethmoid artery) also gives off some branches to supply the posterior aspect of the frontal sinus cavity. Most of the frontal sinus venous blood supply consists of a compact system of valveless diploic veins, which communicate intracranially, intraorbitally, and with the midfacial and forehead skin. The posterior wall drains into the superior sagittal sinus, intracranially [1, 17].

Microscopic channels provide lymphatic drainage to the frontal sinus through the upper nasal (midfacial) lymphatic plexus, for most of the anterior and inferior part of the sinus. The remaining portion of the frontal sinus drains into the subarachnoid space.

Branches of the ethmoidal, nasal, supraorbital, and supratrochlear nerves provide the frontal sinus cavity with an extensive array of sensory innervation. Autonomic innervation of mucosal glands accompanies the neurovascular bundle supplying the frontal sinus.

The frontal sinus mucosa resembles the rest of the upper respiratory mucosa with its ciliated columnar

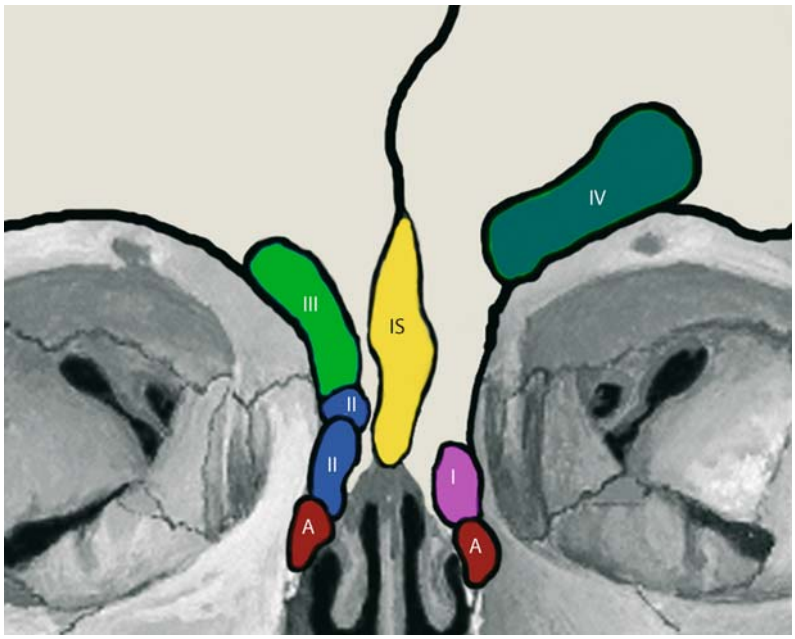

Fig. 3.10.
Bent and Kuhn's classification of frontal infundibular air cells based on its proximity to the agger nasi (A) and orbital roof. Types I, II, III, and IV are shown. In addition, one or more intersinus septal cell (IS) may also exist

respiratory epithelium, along with numerous glands and goblet cells that produce serous and mucinous secretions. The frontal sinus mucosa is constantly producing secretions in order to ensure that the cavity is at all times cleared of particulate matter, and that proper humidification is achieved. Although the final destination of the secretions is the frontal recess, the secretions might recirculate several times through the entire frontal sinus cavity, via its intersinus or intrasinus septae before they finally make their way out into the nose through the frontal infundibulum [8, 11, 13]. Failure to maintain the frontal sinus outflow tract patent (because of edema, fibrosis, polyps, and/or neoplasm) may trigger a vicious cycle of events that results in retained secretions, secondary bacterial colonization, hypoxia, pH changes, and ciliary dysfunction. Any or all of these physiological changes may culminate in chronic rhinosinusitis [13].

Conclusions

Frontal sinus anatomy can be challenging even for the most experience surgeon. A thorough knowledge of the most common normal variants is critical in order to safely navigate through the nose during endoscopic sinus surgical procedures and avoid complications. However, despite great variability in frontal air cell development and pneumatization, the frontal sinus has a predictable mucociliary outflow tract with well established anatomical relationships to neighboring vital structures and ethmoidal air cells.

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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

Contents

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 pred-

ilection 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.

4

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-α, 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 iso-

Table 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			
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

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

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			

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.

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 0% 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].

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 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 4.3. 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)

From [12]

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)	*S. aureus*, streptococci, anaerobes, polymicrobial	Pencillinase-resistant penicillin and metronidazole, consider vancomycin	Third-generation cephalosporin and vancomycin and metronidazole
Intracranial abscess	Streptococci, *Bacteroides* sp.	3rd generation cephalosporin and metronidazole	High-dose PCN G and metronidazole
Orbital complication	*S. pneumococcus,H. influenzae, M. catarhalis, S. aureus*	2nd and 3rd generation cephalosporin or ampicillin/ sulbactam	Ticarcillin/ clavulanate or piperacillin and tazobactam
Meningitis	*S. pneumococcus, H. influenzae*	3rd generation cephalosporin and vancomycin	Meropenem and vancomycin
Dural sinus thrombophlebitis	*S. aureus*, group *A streptococcus*, *H. influenzae*, fungal organisms	Pencillinase-resistant penicillin and 3rd generation cephalosporin	Imipenem or meropenem and vancomycin

From [14]

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].

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Chapter 5

Chronic Frontal Rhinosinusitis: Diagnosis and Management

5

Michael Sillers

Core Messages

- Despite significant advances in surgical techniques, technology, and knowledge of pathophysiology, management of chronic frontal rhinosinusitis remains one of the most challenging problems for otolaryngologists
- Medical therapy for chronic frontal rhinosinusitis is analogous to the therapy for chronic ethmoid rhinosinusitis
- Long-term management success is best achieved in a setting of an integrated medical and surgical approach

Chronic frontal rhinosinusitis represents perhaps one of the most difficult areas within the paranasal sinuses to manage. A current search of the literature will result in numerous publications describing medical therapy, imaging techniques, and surgical procedures specifically for the treatment of symptomatic chronic frontal rhinosinusitis.

This chapter will attempt to discuss a workable rationale for the appropriate diagnosis and treatment of patients with this troublesome disease by presenting the following:

- Anatomic review of the frontal sinus outflow tract
- Current diagnostic criteria
- Endoscopic evaluation techniques
- Advanced CT imaging
- Strategies for medical therapy
- An integrated surgical approach

Contents

Anatomy of the Frontal Sinus Outflow Tract

There has been much confusion regarding the anatomy of and drainage from the frontal sinus. The term "nasofrontal duct" has been entrenched in our literature for many years when, in fact, there is no "duct" leading from the frontal sinus into the nasal cavity [12]. Understanding this complicated anatomic region does not come easily but only after extensive study. The frontal sinus outflow tract (FSOT) can be envisioned as an hourglass with three basic components [16]. The frontal sinus infundibulum is the inferior aspect of the frontal sinus into which "pours" the mucus generated by the respiratory epithelium which lines the frontal sinus. The frontal sinus os-

tium is the inferiormost aspect of the frontal sinus proper, beyond which lays the frontal recess. The frontal recess is a space dependent on the pneumatization of several distinct ethmoid air cells, described by Bent et al., and tends to be the most varied component of the FSOT [2]. The degree to which these cells develop determines the complexity of the frontal recess and in many instances will dictate a specific surgical approach when medical therapy fails.

The frontal recess is bound by:

- The posterior wall of the agger nasi region anteriorly
- The anterior wall of the ethmoid bulla posteriorly
- The lamina papyracea laterally
- The anterior vertical portion of the middle turbinate medially
- The ethmoid roof superiorly

The agger nasi region ("agger nasi" means "mound in the nose") will pneumatize in almost all circumstances [4]. The degree to which it pneumatizes varies and has a great influence on the dimensions of the frontal recess and the frontal sinus (Figs. 5.1, 5.2). Ethmoid cells located above the agger nasi cell are designated as frontal cells and are further classified based on their size and number. Suprabullar, supraorbital ethmoid, and intersinus septal cells can all influence the frontal recess. Each of these cells can be confused with the frontal sinus itself during an attempted endoscopic intranasal frontal sinusotomy, and need to be distinguished on the patient's preoperative CT images and anticipated at the time of surgery. Sagittal reconstructed images are indispensable in the accurate diagnosis of pathology in this region.

Current Diagnostic Criteria

Patients with chronic frontal rhinosinusitis frequently have associated disease in the remaining paranasal sinuses. Isolated frontal sinus disease occurs rarely. Patients present with a history of symptoms of 3 months or more duration as defined by the most recent report of the rhinosinusitis task force [1] (Table 5.1). Symptoms are not generally sensitive or specific for uncomplicated frontal sinus disease. Notable exceptions are frontal sinus osteomas, frontal sinus and supraorbital ethmoid mucoceles, and frontal sinus neoplasm, in which cases patients may have localized pain.

Table 5.1. Diagnostic criteria for chronic rhinosinusitis

1. Continuous symptoms and/or physical finding ≥ 12 weeks
2. One inflammatory sign associated with symptoms
   a. Discolored mucus, nasal polyp, or polypoid swelling
   b. Edema or erythema of the middle meatus
   c. Generalized edema, erythema, or granulation tissue. If not involving the middle meatus or ethmoid bulla, must have radiographic confirmation of inflammation.
   d. Imaging modalities:
      i. CT showing diffuse signs of inflammation
      ii. Plain radiograph with > 5 mm mucosal thickening or opacification
      iii. MRI not recommended

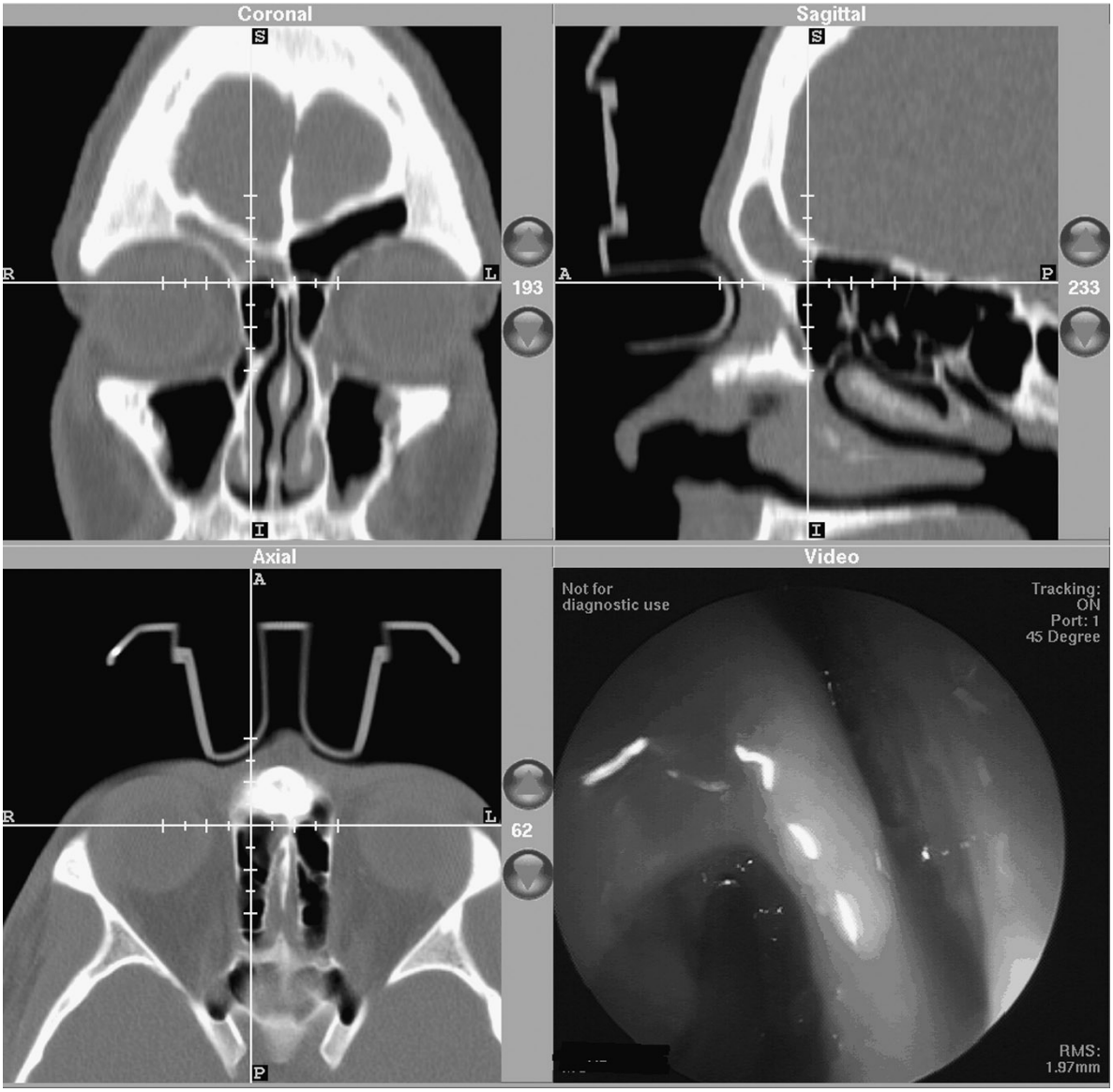

Fig. 5.1. Intraoperative computer-assisted surgery view of large agger nasi cell and associated frontal sinus opacification

Endoscopic Evaluation

Diagnostic nasal endoscopy is the most comprehensive physical examination for the rhinologic patient. The nose should be examined in the natural and decongested state. Careful note is made of differences between sides and in different areas; i.e., middle meatus vs. superior meatus vs. sphenoethmoidal recess. The presence and degree of edema as well as the character and color of secretions should be documented. Abnormal secretions should be collected

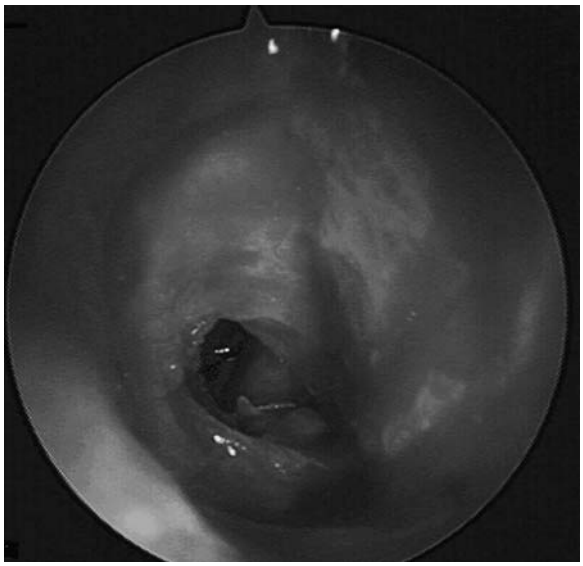

Fig. 5.2. Endoscopic view following removal of the roof of the agger nasi cell

with careful endoscopic technique and sent for appropriate staining and culture. Tantilipikorn et al. found no significant difference between endoscopically acquired cultures obtained through aspiration versus those obtained with a calcium-alginate tipped swab [13]. Often the volume of secretions is small and may be more amenable to a swab technique than an aspirate.

In patients who have undergone previous surgery, frontal sinus disease is suspected when the following are seen:

- Lateralized or amputated middle turbinate
- Synechia
- Polypoid edema in the anterior ethmoid cavity

An angled telescope (30° or more) is almost always required to adequately assess the frontal recess and frontal sinus (Fig. 5.3A–D.)

Advanced Imaging Techniques

Noncontrast CT imaging is the imaging modality of choice for the radiographic evaluation of patients with chronic uncomplicated rhinosinusitis. Standard axial and coronal images are necessary as a preoperative data set but may not be adequate to comprehensively depict the complexity of the FSOT anatomy. Specifically for patients with difficult frontal recess anatomy, sagittal reconstruction is vital. From sagittal images, the anterior to posterior dimensions of the frontal recess can be assessed, and the extent to which frontal recess cells impact the FSOT can be determined [8]. In general, sagittal reconstruction is performed from reformatted axial images. The thinner the axial image slice the better the resolution of the reconstructed sagittal image. These images can be acquired from the workstation in the radiology suite or on a surgical navigation workstation if available.

Computer-assisted sinus surgery has gained wide acceptance and has proven useful in functional endoscopic sinus surgery (FESS) in general. Perhaps one of its greatest areas of utility is in the FSOT. Patients with complex anatomy and/or scarring from prior surgery present a significant challenge to the endoscopic sinus surgeon. The ability to accurately track surgical instruments within a defined surgical volume to which multiplanar CT images are registered has enabled surgeons to safely and successfully treat patients who previously would have required more aggressive open procedures (Fig. 5.4).

Medical Management

There is no medical therapy designed specifically for the frontal sinus. In general there is currently no medical therapy that is FDA-approved for the treatment of *chronic* rhinosinusitis. The choice of therapeutic agents should be made thoughtfully and on an individualized basis. The microbiologic environment of acute rhinosinusitis is different from that in chronic rhinosinusitis and includes primarily *Staphylococcus aureus*, coagulase-negative *Staph* and *Pseudomonas* species. Schlosser et al. specifically cultured

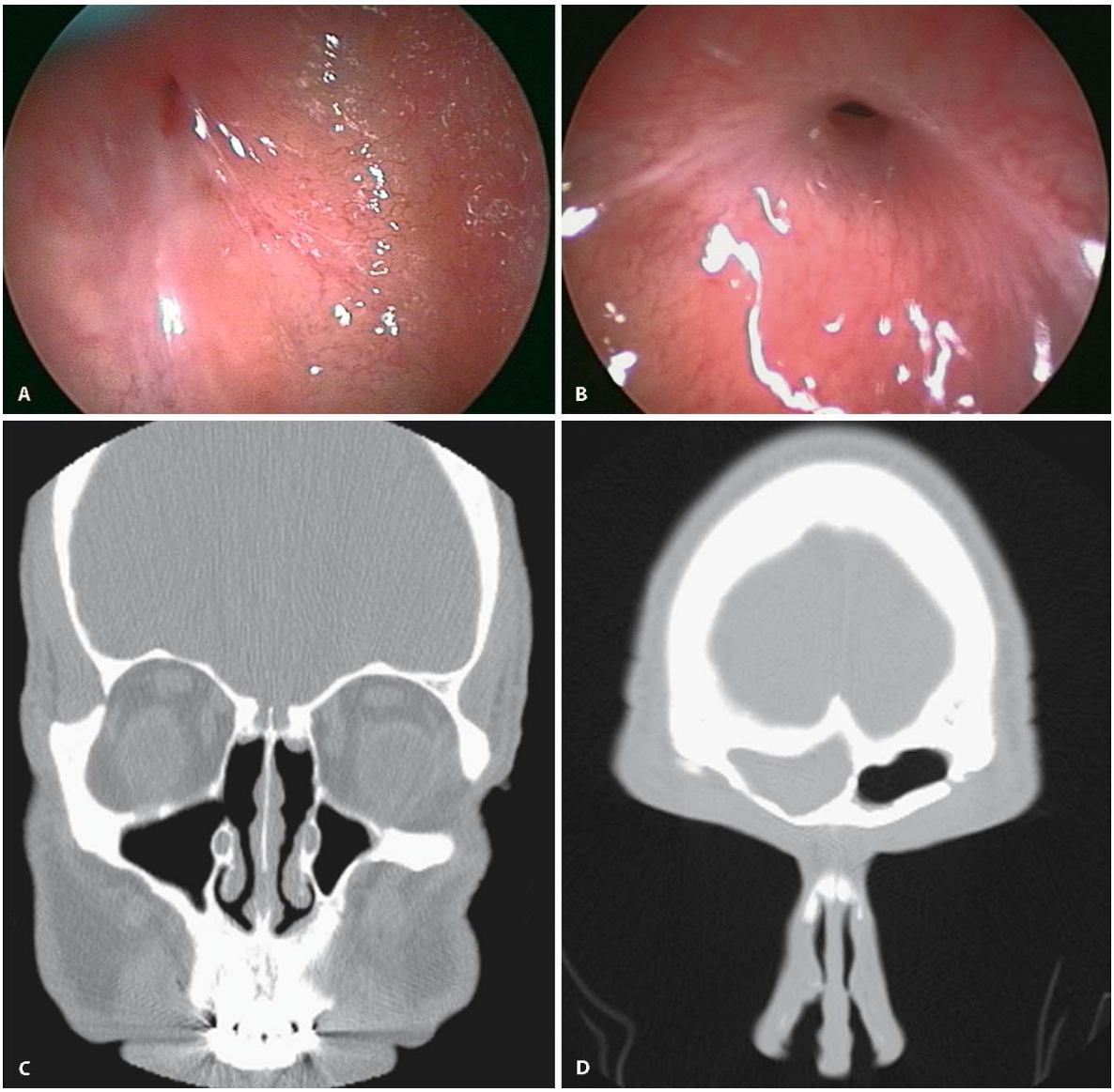

Fig. 5.3. Thirty-degree endoscopic views of left and right frontal recess and accompanying coronal CT in a patient with multiple prior surgeries. **A** Obstructed right frontal recess. **B** Patent left frontal recess. **C** Coronal CT depicting middle turbinate resection and osteoneogenesis along the ethmoid roof. **D** Coronal CT depicting patent left and opacified right frontal sinuses

patients with chronic frontal rhinosinusitis via a mini-trephination approach (Table 5.3). Patients undergoing primary surgery were more likely to have *H. influenza* while coagulase-negative *Staph* was more common in revision cases [11]. Because of these microbiologic differences, culture-directed therapy is likely to result in the most appropriate choice of antimicrobials in each individual patient. Adjuvant therapy focusing on the reduction of inflammation is also frequently recommended.

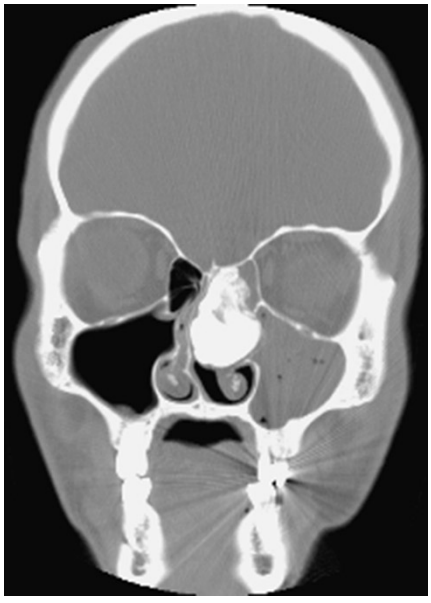

Fig. 5.4. Coronal CT depicting large frontoethmoid osteoma removed via transnasal endoscopic approach utilizing computer-assisted surgery

Adjuvant therapy in chronic frontal sinusitis may include:

- Intranasal and systemic steroids
- Topical and systemic decongestants
- Antihistamines
- Leukotriene modifiers
- Mucolytics
- Nasal saline nasal spray/irrigations

The recommendation for these medications should consider potential side effects, underlying comorbidities and their relative contraindications, drug interactions, and cost. There remains quite a debate as to what constitutes "maximal medical therapy" in both the degree as well as the duration. Adding to the confusion is the difference between patients such as one with aspirin-sensitive asthma and nasal polyposis versus one with limited maxillary and ethmoid infundibular disease. All chronic rhinosinusitis is not the same. In general, patients should have the benefit of therapy for 3–4 weeks followed by a posttreatment CT, at which time an assessment of their clinical response can be made. Symptomatic patients with evidence of chronic inflammatory changes on CT can be considered "medical failures" and appropriate surgery can be recommended.

Table 5.3. Microbiology of chronic frontal rhinosinusitis

Aerobic	
Staphylococcus aureus	– 21%
Coagulase negative Staph	– 21%
Haemophilus influenza	– 9%
Other	– 26%
Anaerobic	– 3%
No growth	– 38%
Fungus	– 4%

An Integrated Surgical Approach

Multiple surgical procedures have been described for the treatment of chronic frontal rhinosinusitis. Montgomery popularized the osteoplastic frontal sinus fat obliteration that was the workhorse procedure for many years [7]. With the advent, widespread acceptance, and technical advances of functional endoscopic sinus surgery, this procedure is much less frequently utilized. As with medical therapy, the choice of approach to the frontal sinus should be made thoughtfully. Factors such as associated ethmoid disease, pneumatization patterns, suspected pathology, need for exposure, and operator experience should all influence the choice an appropriate surgical procedure; no one operation will work for every patient. A stepwise progression should be considered depending on the degree and type of pathology in individual patients (Table 5.4). Weber et al. published combined retrospective results of frontal sinus surgery in 1286 patients: 85% of patients underwent an endonasal approach while only 15% required an external procedure. They achieved success ranging from 79%–97.8% in patients with chronic frontal rhinosinusitis, neoplasm, and trauma [14].

It is important to recognize that mucosal disease in the frontal sinus is usually the result of outflow obstruction in the inferior portion of the frontal sinus outflow tract, i.e. the frontal recess. Notable exceptions include frontal sinus osteoma, inverting papilloma, and de novo mucocele. As a consequence, in

many patients with limited mucosal thickening in the frontal sinus, the most appropriate procedure is a careful anterior ethmoidectomy, taking care not to violate the mucosa in the frontal recess. An intranasal frontal sinusotomy which entails the removal of all ethmoid air cell partitions in the frontal recess, preserving boundary mucosa, and visually identifying the frontal sinus is appropriate in patients with more severe frontal sinus disease, patients with severe polypoid disease in the frontal recess, and those who have failed prior anterior ethmoidectomy (Fig. 5.5). More advanced/aggressive intranasal endoscopic ap-

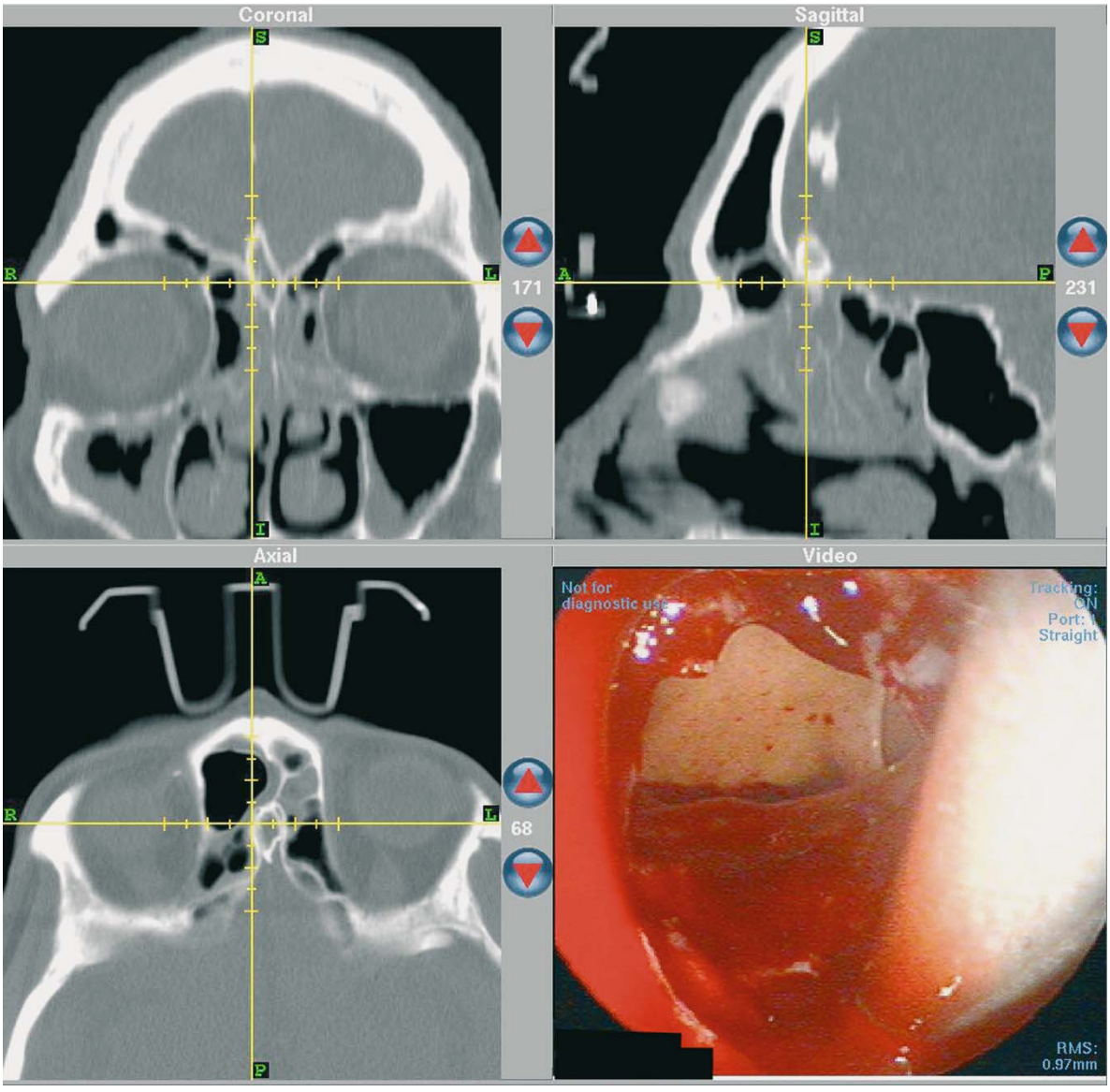

Fig. 5.5. Intraoperative computer-assisted surgery image of an obstructing Type III frontal cell removed to visualize the frontal sinus

Table 5.4. Integrated surgical approach

Procedure	Indication
Endoscopic anterior ethmoidectomy	Limited frontal sinus mucosal thickening
Intranasal frontal sinusotomy	Extensive frontal sinus mucosal thickening/opacification, nasal polyps, and/or failed ethmoidectomy
Frontal sinus rescue procedure	Failed intranasal frontal sinusotomy
Draf II/III, endoscopic modified Lothrop, trans-septal frontal sinusotomy	Extensive frontal sinus disease, neoplasm, osteoneogenesis, and failed intranasal frontal sinusotomy
Frontal sinus trephination	Frontal sinus pathology inaccessible via intranasal approach alone
External frontal sinusotomy	Neoplasm, trauma, or CSF leak requiring wide exposure

proaches (Draf II/III, frontal sinus rescue procedure, endoscopic modified Lothrop procedure, and transseptal frontal sinusotomy) are chosen based on the patient's unique anatomy and usually the failure of prior endoscopic techniques [5, 6, 9, 10, 15].

External procedures are much less frequently required with advanced endoscopic techniques and computer-assisted surgery. A frontal sinus trephination can be performed in conjunction with endoscopic techniques when the disease process in the frontal sinus cannot be adequately reached from an intranasal approach alone. Laterally based frontal sinus mucoceles, small osteomas, and Type III/IV frontal cells are examples of pathology that may be successfully addressed by this "combined" approach [3] (Figs. 5.6, 5.7). External frontal sinusotomy via Lynch incision or through an osteoplastic flap approach is generally considered when wide exposure and visualization are needed such as with frontal sinus neoplasm, trauma, and frontal sinus CSF leak.

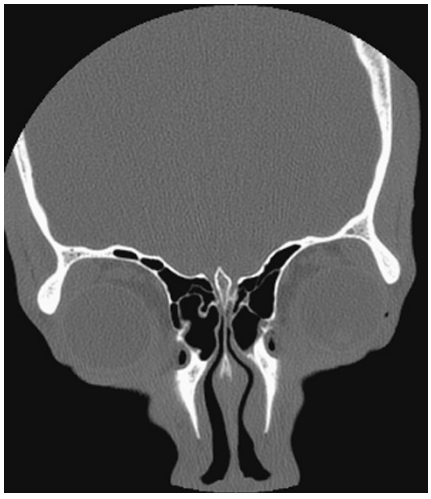

Fig. 5.6. Coronal view of Type III frontal cell

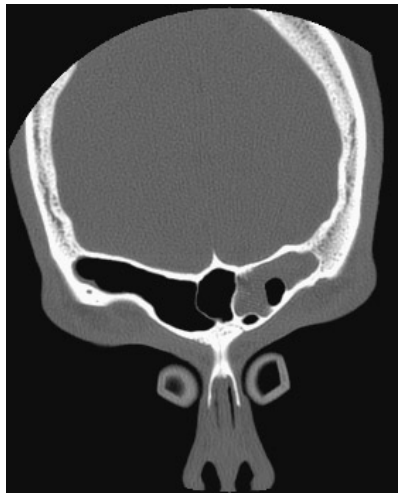

Fig. 5.7. Coronal view of superior extent of Type III frontal cell and associated frontal sinus opacification

Conclusion

Despite the tremendous advancements that have been made in the medical and surgical treatment of chronic rhinosinusitis, it remains one of the most challenging disease processes managed by otolaryngologists today. In 1946 Harris Mosher stated "frontal sinus surgery in my hands has been bitterly disappointing. Temporary favorable results have been common. Permanently favorable results I could never guarantee." His sentiment is true today, and only with long-term follow-up can we determine if our current treatment methods will result in consistent "permanently favorable results."

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Chapter 6

Microbiology of Chronic Frontal Sinusitis

6

Birgit Winther, Jack Gwaltney

Core Messages

- Because of limitations in sampling techniques, microbiology of chronic frontal rhinosinusitis remains poorly understood
- Obstruction of the frontal sinus outflow in the presence of pathogenic bacteria may yield frontal infection
- Surgical manipulation of the sinuses appears to impact subsequent microbiology of the disease process

Contents

Introduction

The microbiology of chronic rhinosinusitis is poorly understood. A major problem has been the sampling method used for collecting specimens for cultures. Most studies have employed surgical or swab specimens obtained during endoscopic surgery. It is not possible to know whether these specimens are contaminated with bacteria from the nasal passages as the result of surgical manipulation during the procedure. Only a few studies have employed the technique of aseptic sinus aspiration prior to beginning the surgical procedure. Another problem is that the bacteriological findings from pre- and postsurgery patients have often not been distinguished, although the two conditions are obviously different.

Chronic frontal sinusitis is less common than chronic maxillary sinusitis. A limited number of published studies have reported the microbiologic findings in patients with chronic frontal sinusitis. This chapter will discuss the pathology of frontal sinusitis and review current knowledge on its bacteriology.

Definitions

The clinical definition of acute infectious sinusitis has been based on a combination of various signs and symptoms and demonstration of a high titer ($\geq 10^4$ cfu/ml of sinus secretion) of bacteria in the sinus aspirate.

Histopathologic findings include [4]:

- Edema
- Massive infiltration with neutrophils
- Increased lymphocytes and plasma cells

- Microabcesses
- In severe cases thrombosed blood vessels and necrotic foci

The epithelial surface remains intact. Neutrophil infiltration has also been reported in viral rhinosinusitis [18]. The clinical definition of chronic sinusitis also depends on selected signs and symptoms, but a bacteriologic criterion is not well established. In fact the role of bacteria in the initial etiology of chronic sinusitis is not well established.

Pathologic findings in chronic sinusitis include [13]:

- Swelling of the ciliary membrane
- Formation of compound cilia
- Dropping of epithelial cells
- Metaplasia

The number of inflammatory cells correlates with the thickened antral mucosa and with amount of purulent secretion [7].

Frontal Sinus Outflow Anatomy and Patency by CT Scanning

The normal frontal sinus is fully aerated and believed to be sterile except during periods of transient bacterial contamination. CT study utilizing application of intranasal contrast medium has suggested that there is an open and easy communication from the nasal cavity to the frontal sinus cavity. Nasal fluid containing contrast medium can be detected in the frontal sinus after noseblowing in normal adult volunteers (Fig. 6.1). In this study, noseblowing generated an intranasal pressure of 60–70 mmHg, which is sufficient to propel nasal fluid through the frontal duct into the sinus [8]. At times polypoid tissue from the frontal sinus mucosa and viscous mucopus may occlude the frontal duct. CT scanning cannot accurately distinguish between mucosal swelling or the presence of viscous exudate when obstruction is present in the frontal duct and opacification observed in the frontal sinus.

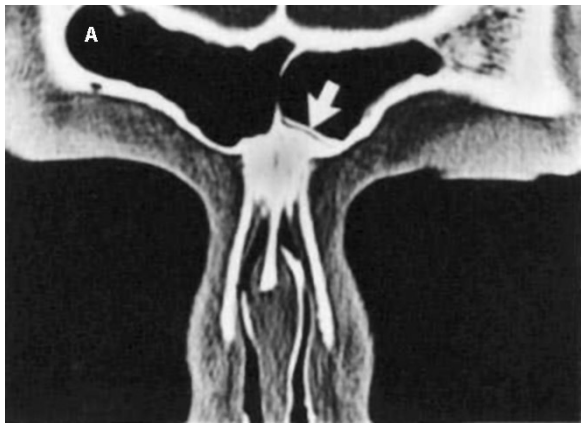

Fig. 6.1. Radiopaque contrast material (*arrow*) in the frontal sinus following noseblowing in healthy adult

Mucociliary Clearance in the Frontal Sinus

In the early 1930's Hilding [1] described the pattern of mucociliary clearance of the frontal sinus. Using fresh cadavers, ink was sprayed in a thin film over the mucosal surface of the sinus and was observed to be carried to the frontal ostium. Movements proceeded in a spiral pattern, and the velocity of flow increased as fluid approached the ostium.

Experimental Bacterial Infection of Canine Frontal Sinuses

In early work Arnold and coworkers [3] failed to produce experimental bacterial rhinitis by spraying bacteria into the nasal cavity in 42 healthy adults. They noted that 90%–95% of viable bacteria had disappeared within 5 to 10 min. Hilding [1] injected a bacterial suspension directly into the frontal sinus and also failed to produce infection; however, infection of the sinus was achieved by inoculation with a suspension of bacteria in warm milk. The milk coagulated after injection into the sinus and served to keep the bacteria in the sinus. No bacterial invasion of the frontal sinus mucosa was noted. During viral respiratory infection fibrin clots may be formed on the epithelial lining of the sinus [19], and similar to coagulated milk may provide substrate material to keep bacteria in the sinus.

Viral Rhino/Frontal Sinusitis

Viral respiratory tract infection produces a viscous exudate in the sinuses [9] and decreases the mucociliary clearance in the nose for several weeks [14]. Frontal sinus abnormalities with acute viral rhinosinusitis were demonstrated by CT scanning in 32% of 31 patients with acute viral rhinosinusitis [9]. It is unclear whether the frontal ostium also was occluded in those instances. The nasopharynx is believed to be the primary site of acute viral infection of the upper respiratory tract [20], but the nasal passage, paranasal sinuses, laryngeal and bronchial mucosa are also frequently involved. It is not clear how frequently respiratory viruses replicate in those secondary sites, but respiratory viruses have been recovered in cultures and identified by RT-PCR from sinus aspirates in patients with acute sinusitis [12, 15].

Chronic Bacterial Frontal Sinusitis

Specimens for bacterial cultures cannot be obtained from the frontal sinus cavity by way of the nasal passages. Frontal sinus mini-trephination is a technique that provides uncontaminated specimens from the sinus cavity for culture. Antila and co-workers recovered *H. influenza* and/or *S. pneumoniae* from 30% of 103 samples obtained by trephination of the frontal sinus in patients with acute frontal and maxillary sinusitis. Specimens were collected 24 h after initiation of antibiotic treatment [2]. In a study by Schlosser and co-workers [17] of 30 patients undergoing endoscopic surgery for chronic frontal sinus disease, 46 samples were obtained by trephination from the frontal sinus. Approximately one-third of the samples were negative for aerobic and anaerobic bacteria and fungi. There was a trend towards a different pattern of bacteria in patients with prior functional endoscopic sinus surgery (FESS) without frontal surgery versus patients with prior FESS with frontal "drill out" surgery, and compared to patients without any prior sinus surgery (Table 6.1). *H. influenzae* was isolated in two of eight samples from patients without prior FESS, but none of 21 samples from FESS patients without prior frontal sinus surgery (intact frontal sinus). *Staphylococcus aureus* and coagulase-negative Staphylococcus were isolated more frequently from patients with prior FESS with and with-

Table 6.1. Culture results of frontal sinus aspirates (46 trephines)

	No prior sinonasal surgery	Prior FESS[a] without frontal surgery	Prior surgery of frontal recess/sinus
No aerobic growth	37% (3/8)	38% (8/21)	33% (2/6)
Staphylococcus aureus	12% (1/8)	24% (5/21)	17% (1/6)
Coagulase-negative *Staphylococcus*	12% (1/8)	19% (4/21)	33% (2/6)
Haemophilus influenzae	25% (2/8)	0% (0/21)	17% (1/6)
Mixed oropharyngeal flora	12% (1/8)	5% (1/21)	17% (1/6)
Escherichia coli	0% (0/8)	5% (1/21)	0% (0/6)
Xanthamona	0% (0/8)	5% (1/21)	0% (0/6)
Group A *Streptococcus*	0% (0/8)	0% (0/21)	17% (1/6)
Serratia sp	0% (0/8)	0% (0/21)	17% (1/6)
Gram-negative rods-not specified	12% (1/8)	0% (0/21)	0% (0/6)
S. pneumonia	0% (0/8)	5% (1/21)	0% (0/6)
Anaerobic bacteria (Gram-Positive cocci)	0% (0/7)	0% (0/21)	25% (1/4)
Fungi (*Penicillium*)	0% (0/6)	7% (1/14)	0% (0/5)

[a] FESS, functional endoscopic sinus surgery.

With permission from The Laryngoscope [17].

out frontal surgery [43% (9/21) and 50% (3/6), respectively] compared to patients without FESS (25%; 2/8). One of six samples from patients with prior FESS with frontal sinus surgery had *H. influenzae* recovered. An array of other bacteria were also cultured, including mixed oropharyngeal flora, Group A Streptococcus, *S. pneumoniae*, *Escherichia coli*, *Serratia spp.*, and *Xanthomonas* (Table 6.1). Only one sample was positive for anaerobic bacteria, from a patient with prior FESS including frontal sinus surgery. This is in contrast to the studies by Brook, who reported frequent recovery of anaerobic bacteria when cultures were obtained through osteoplastic flaps from 13 patients with chronic frontal sinusitis [6]. The discrepancy between the studies is unexplained but may relate to the different sampling methods used.

Chronic Fungal Frontal Sinusitis

The frequency of isolation of fungi from the paranasal sinus of patients with chronic sinus disease has varied hugely. Ponikau and co-workers found at least one fungus in 96% of 210 patients with chronic sinusitis and from 100% of 14 healthy controls [16]. The fungal species recovered were similar in both groups. Fungal cultures from specimens obtained by mini trephination of the frontal sinus have recently been reported by Schlosser and co-workers [17]. They found penicillium in 4% of 24 samples from patients who had had prior FESS without frontal sinus surgery.

Chronic Inflammatory Sinus Disease in Postsurgery Patients

Bacteria are present in the nasopharynx at all times, while the mucosa of the intact sinus with normal mucociliary clearance is thought to be sterile [5].

Chronic sinus disease in patients with previous sinus surgery is characterized by:

- Decreased clearance of mucus from the paranasal sinuses
- Prolonged presence of gram-positive and/or gram-negative bacteria in the sinuses

Very little is known about the pathogenesis of chronic sinus disease in either the pre- or postsurgical state. The bacteria present in postsurgical patients do not appear to be the original cause of the disease. It is not clear to what extent the bacteria are responsible for the ongoing disease in patients who remain symptomatic after surgery, but they are believed to play a major role in the exudates and crusting which characterize the process. New information suggests their role may depend on the PAMP (pathogen-associated molecular pattern) of a given flora [10]. Toll-like receptors (TLRs) of the innate immune system are essential for shaping the adaptive immune response. The TLRs provide a signal that increases the antigenic function of immature dendritic cells, which influence the differentiation into Th1 or Th2 cytokine-producing T-lymphocytes [11]. A concurrent viral upper respiratory tract infection or allergen exposure may temporally change the existing balance of PAMP and the immune response in patients with chronic sinus disease. The degree of activation of pro-inflammatory signal cascades in response to bacterial flora in chronic sinus disease needs further investigation.

Conclusion

Microbiology of chronic frontal rhinosinusitis remains a controversial topic. Difficulty in specimen acquisition and occurrence of previous surgery are but two variables that may impact data. While the frontal sinuses are usually presumably sterile, in the setting of frontal outflow obstruction accompanied by bacterial inoculation of the sinus, infection may arise. However, the exact mechanism resulting in the development of chronic inflammation remains elusive.

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Chapter 7

Orbital Complications of Frontal Sinusitis

7

Robert T. Adelson, Bradley F. Marple

Core Messages

- The most common cause of orbital infections is sinusitis, most often seen in the second to third decades of life
- The propagation of orbital infection is facilitated by the valveless veins of the orbit that allow free communication between facial, sinus, and surrounding venous networks
- Orbital complications most often arise from the ethmoid sinuses; however, frontal sinusitis complications may progress rapidly and result in worse outcomes
- The orbital septum is the key feature in the classification of orbital infections
- Ophthalmological consultation is critical when physical exam findings suggest postseptal spread of orbital infection
- The bacteriology of orbital complications of sinusitis is similar to that of the sinusitis itself
- Contrast CT scans can distinguish cellulitis or abscess and assist in the planning of surgery when it is indicated
- The most common orbital complication of sinusitis is orbital cellulitis, which most often responds rapidly to intravenous antibiotics. Progression of symptoms or failure to respond to antibiotic treatment is an indication for surgical therapy
- Surgical intervention in postseptal orbital complications of sinusitis is frequently required (12%–66%)

Contents

Introduction

Sinusitis, in the antibiotic era, is a disease process for which infectious complications have become increasingly uncommon. It is estimated that a maximum of 1%–3% of all sinus infections result in intraorbital or intracranial complications [22]. The preantibiotic era was witness to a 17% incidence of death and 20% incidence of blindness in postseptal infections, declining in the modern era to 1%–2% and 1%–8%, respectively [6, 22]. The persistence of such morbidities demands further study of the complications of sinusitis.

Frontal sinusitis and orbital complications thereof is a narrow clinical window that demands both a high

level of diagnostic acumen and technical ability to engender a successful outcome. A thorough understanding of the pathogenesis, diagnosis, and current treatment recommendations for orbital complications of frontal sinusitis will allow physicians to decrease the morbidity and mortality associated with this condition.

Demographics

The overwhelming majority of orbital infections are a result of sinusitis, representing greater than 70% of cases in most series [8, 10, 11].

The most common complications of sinusitis in order of frequency are [1, 19, 20, 28]:

- Orbital involvement
- Intracranial complications
- Frontal bone osteomyelitis
- Soft tissue abscesses

Several case series have characterized further the population of patients affected by orbital complications of sinusitis, particularly in those patients with frontal sinusitis. Eighty-five percent of patients with orbital complications of paranasal sinusitis are within the pediatric age group, and within this group 68% are less than 15 years old [15, 24]. As the frontal sinus does not begin to pneumatize significantly until six - years of age, the population experiencing complications related to the frontal sinus is correspondingly narrowed [1, 11]. Orbital complications of frontal sinusitis are most common in patients in the second to third decades of life (average age of 25 years), in males more so than in females (ratio of 2.6:1 to 3.3:1), and involve the left eye more frequently than the right [19, 20, 24, 28]. The discrepant age, sex, and laterality trends have been noted by multiple authors, yet convincing explanations are lacking.

Relevant Orbital and Sinus Anatomy

The intimate relationship between the paranasal sinuses and the vital surrounding organs is foremost in the mind of surgeons whose routine operative approaches demand expert navigation of this compact, complex anatomy. In the context of acute sinusitis with orbital complications, anatomic landmarks are further obscured and surgery made cumbersome by the bleeding tendencies of inflamed sinonasal mucosa.

The orbit is separated from the ethmoid sinuses medially by a thin and often dehiscent lamina papyracea, from the maxillary sinus by a similarly thin orbital floor, and from the frontal sinus by a portion of the orbital roof. The bony orbit is vulnerable to spread of infection, directly or by thrombophlebitic spread, via the numerous fissures and foramina that transmit vessels and nerves through the sinuses, orbit, and intracranial space [15]. The periosteal lining of the orbital bones, the periorbita, is an additional layer of separation between the orbital contents and the sinuses. This fibrous tissue is firmly adherent to underlying bone at the orbital rims, suture lines, orbital fissures, and lacrimal crest but loosely adherent elsewhere, allowing infection to dissect into these potential subperiosteal spaces [3]. The orbital septum, a key feature of the classification of orbital infections, arises from the union of the periorbita with the periosteum of the forehead and cheekbones at the orbital rim (the *arcus marginalis*) [3, 21]. The orbital septa of the upper and lower eyelids form an anatomic barrier to infection and define the preseptal and postseptal spaces [4].

The valveless veins of the orbit play a key role in propagation of orbital infections, as they allow free communication between the facial, sinus, orbital, and intracranial venous network [25]. The superior ophthalmic vein is a well-defined vessel formed by the union of the angular and supraorbital veins, which receives multiple tributaries as it travels posterolaterally through the orbit to exit via the superior orbital fissure to enter the cavernous sinus [3, 13]. The inferior ophthalmic vein is a less well-defined structure, originating near the anterior orbital floor and terminating by sending one branch to the pterygoid plexus via the inferior orbital fissure and a second, larger contribution to the superior ophthalmic vein; both will ultimately drain into the cavernous sinus [3].

Although previously it had been widely accepted that lymphatics are absent within the orbit, orbital lymphangiomas have been reported and recent histochemical studies have confirmed the presence of lymphatics within the lacrimal gland and in the dura

mater of the optic nerve [3, 6, 21, 22, 26]. The anatomy of the orbital lymphatic system is still under active investigation, and while its role in orbital complications of sinusitis is not likely to be of any real clinical significance, a definitive answer is not yet available. In contradistinction, the upper and lower eyelids have well-described lymphatic networks, and these preseptal tissues drain into preauricular and submandibular nodes [21].

The anatomy of the frontal sinus foreshadows its potential for development of orbital and intracranial complications of sinusitis. The horizontal orbital plate of the frontal bone, the thinnest wall of the frontal sinus, forms the roof of the orbit and articulates with the ethmoid bone to contribute to both the roof of the nasal cavity and the floor of the anterior cranial fossa [16]. Venous drainage from the frontal sinus begins in diploic veins which pass through the multiple anterior and posterior table foramina (Breschet's canals), coalescing in sequentially larger diploic veins, developing into the frontal diploic vein that joins at the supraorbital notch with the supraorbital vein to create the superior ophthalmic vein described above [16]. Although not specifically addressed in this chapter, the diploic veins of Breschet contribute significantly to frontal bone osteomyelitis and intracranial complications of sinusitis via their communications with dural sinuses and the marrow cavity of the frontal bone [6, 15, 16].

Pathogenesis of Orbital Complications of Sinusitis

Orbital complications of sinusitis are most often attributable to the ethmoid sinuses, though 84% of cases have radiographic evidence of disease involving two or more sinuses, and some series establish a minimum pattern of concomitant maxillary, ethmoid, and frontal sinusitis in 79% of those cases with orbital complications [6, 22, 10, 19, 29].

It is generally accepted that orbital infections arising from a sinonasal source can arise by two mechanisms [5, 6, 10, 15, 18, 23, 27, 28, 29]:

- Direct extension
- Retrograde thrombophlebitis

The bony limits of the orbit are not perfect barriers to direct extension of infection into the orbit. Congenital or acquired bony dehiscences, neurovascular foramina, and open suture lines all constitute mechanisms by which direct extension can occur [5, 6, 23, 11, 18, 28]. The valveless veins of the sinonasal cavity and orbit provide a more circuitous route by which a septic thrombophlebitis can extend to involve the orbit [5, 6, 11, 18, 23, 28].

Classification of Orbital Complications of Sinusitis

An understanding of the relevant sinonasal and orbital anatomy as well as the mechanisms by which orbital complications develop is required to classify the disease state so that treatment recommendations can be made and outcomes studied. Hubert proposed the earliest well-documented classification scheme based on his experience with 114 patients in the pre-antibiotic era [14]. The classification of patients into five groups based on the anatomy involved, perceived progression of infection, responsiveness to treatment, and general prognosis is a convention that is still in use today, though as the widely accepted schema proposed by Chandler [5]. Chandler's work solidified the utility of this classification system, and his therapeutic principles characterize the modern approach to managing orbital complications of sinusitis (Table 7.1) [5, 13, 25].

Table 7.1. Chandler classification systems for orbital complications of sinusitis

Group 1	Inflammatory edema (preseptal cellulitis)
Group 2	Orbital cellulitis
Group 3	Subperiosteal abscess
Group 4	Orbital abscess
Group 5	Cavernous sinus thrombosis

From [5]

- Group I – Inflammatory edema (preseptal cellulitis) represents swelling of the eyelids anterior to the orbital septum thought to be

secondary to restricted venous drainage. The eyelids are usually not tender and, as inflammation does not involve the postseptal structures, chemosis, extraocular muscle movement limitations, and vision impairment should be absent [5, 6, 11, 18]. Authors disagree regarding the absence [5, 10, 27] or presence of mild proptosis at this stage [6, 22]. The degree of preseptal inflammation may hamper accurate assessment of proptosis, especially when examining pediatric patients.

- Group II – Orbital cellulitis results in a pronounced edema and inflammation of the orbital soft tissue without frank abscess formation [5, 6, 22]. It is vital to detect the signs of proptosis and decreased extraocular motility, as these are considered reliable signs of orbital soft tissue involvement [10, 19, 23]. Chemosis is almost always present to varying degrees, yet vision loss is very unusual in this stage, but should be monitored carefully [6, 18, 22].
- Group III – Subperiosteal abscess develops in the potential space between periorbita and bone [5]. The orbital contents are displaced by the mass effect of a collection of subperiosteal pus, frequently in an inferolateral direction. Chemosis and proptosis are reliably present, although decreased ocular mobility and vision loss may take some time to develop and are not always present early in the course of this stage [10, 15, 22, 24, 25, 27].
- Group IV – Orbital abscess, a collection of purulent, necrotic material within the orbital tissue, can develop as a result of a progressive orbital cellulitis or from the rupture of a subperiosteal abscess [5, 6, 15]. Severe proptosis and near complete ophthalmoplegia are noted, and visual loss is increasingly common within this group [10, 22, 27, 29].
- Group V – Cavernous sinus thrombosis may include such nonspecific signs and symptoms as fever, headache, periorbital edema, and photophobia in addition to more specific findings of proptosis, chemosis, ophthalmoplegia, and decreased visual acuity; however, the development of *bilateral* ocular symptoms is the classic finding in this condition [6, 15, 10, 23]. A more expeditious diagnosis is possible when patients demonstrate palsies of those cranial nerves transmitted through the cavernous sinus (III, IV, V1, V2, VI) or develop meningitic symptoms in the presence of a unilateral orbital infection [15, 24, 25].

Despite the clarity and near-ubiquitous application of Chandler's classification system, several other authors have modified his work, and their contributions are useful in highlighting focal changes in our concepts of orbital infections as well as advances in diagnostic technology over the last 34 years.

Schramm's large series of orbital cellulitis allowed him to identify periorbital (preseptal) cellulitis with chemosis as a distinct grouping intermediate in prognosis between Chandler's group I and group III (Table 7.2) [24]. Those patients with periorbital cellulitis with chemosis did not always respond to parenteral antibiotic therapy alone, and therefore frequent serial examinations and a lower threshold for surgical intervention are warranted [11, 24].

Moloney modified Chandler's classification to assign lower priority to orbital infections anterior to the septum, and then delineated the progression of postseptal, intraorbital infections (Table 7.3) [17]. Mortimore and Wormald applied advanced computed tomography (CT) imaging to Moloney's concept of dividing preseptal and postseptal infections, relying upon further radiologic differentiation to be made between cellulitis and an abscess [19, 20]. It is not clear that further, more stringent classifications of orbital infections have altered therapeutic strategies.

Table 7.2. Orbital cellulitis

Periorbital cellulitis
Periorbital cellulitis with chemosis
Orbital cellulitis
Subperiosteal abscess
Orbital abscess
Cavernous sinus thrombosis

From [24]

Table 7.3. Comparison of Moloney classification and the Groote Shuur modification of Moloney

Moloney	Groote Schuur modification
Pre-septal cellulitis	Pre-septal a. Cellulitis b Abscess
Subperiosteal abscess	Post-septal (subperiosteal) a. Phlegmon/cellulitis b. Abscess
Orbital cellulitis	Post-septal (intraconal) a. Cellulitis b. Abscess
Orbital abscess	I. Localized II. Diffuse
Cavernous sinus thrombosis	Considered intracranial

From [19]

Bacteriology

Orbital complications do not have a bacterial profile different from that of acute rhinosinusitis [6, 10, 11, 15, 22].

The most commonly cultured organisms in orbital infections are [1, 6, 10, 15]:

- *Streptococcus pneumoniae*
- *Haemophilus influenzae*
- *Moraxella catarrhalis*
- *Staphylococcus aureus*
- *Streptococcus pyogenes*

A study of patients with simultaneous frontal and maxillary sinusitis found *H. influenzae* and *S. pneumoniae* to be the most commonly isolated organisms [2].

The existing literature does not support a substantial difference in the bacterial populations implicated in frontal sinusitis from that of ethmoid sinusitis. The frontal sinus is the most frequent culprit for intracranial complications of sinusitis, and in these instances, *S. aureus* and polymicrobial infections are found at a slightly increased frequency [11]. The incidence of bacteremia in patients with orbital complications is greatest in children and declines steadily with age [6]. Schramm et al. reported bacteremia in 33% of children under 4 years old, yet demonstrated positive blood cultures in only 5% of the adult patients in a large case series [24].

Diagnostic Evaluation

The various systems for classifying orbital infections emphasize the importance of accurately differentiating between preseptal and postseptal involvement.

Patients typically present with:

- A history of recent upper respiratory infection or symptoms of acute bacterial rhinosinusitis
- And demonstrate:
- Fever
- Edematous eyelids
- Conjunctival injection
- Varying degrees of discomfort

Preseptal cellulitis is the most commonly encountered orbital complication of sinusitis, with multiple large studies documenting a frequency of 48% of such complications seen at tertiary referral centers and nearly 80% of the orbital complications seen overall [6, 10, 24, 28, 29]. Preseptal infections do not require imaging studies [6, 7, 10, 22, 23, 29].

Physical exam findings can be suggestive of a postseptal process, particularly the development of gaze restriction and proptosis [5, 15, 18, 27].

Signs of postseptal involvement include:

- Proptosis
- Gaze restriction
- Decreased visual acuity
- Color vision changes
- Afferent pupillary defect

Ophthalmologic examination is critical in measuring proptosis, evaluating extraocular motility, and, if

necessary, determining intraocular pressure. Traditionally, imaging studies are obtained when the history and physical exam are consistent with postseptal disease [7, 15, 19, 28, 29]. To further clarify those signs of postseptal infection, Younis suggested that the indications for obtaining a CT scan are identical to the indications for surgery, as addressed below [9, 29].

Contrast-enhanced CT scans of the sinuses in axial and coronal planes are essential to surgical planning, as the modality accurately distinguishes between cellulitis and abscesses and identifies which sinuses will need surgical drainage [6, 15, 20, 23, 25]. Magnetic resonance imaging (MRI) offers superior soft-tissue resolution and is most appropriate in the context of intracranial complications, while CT remains the standard initial, and often definitive, modality in the diagnosis of sinusitis and its orbital extension [29]. In one well-controlled study, clinical examination correctly diagnosed 81% of the cases of orbital complications of sinusitis, while 91% accuracy was achieved on the basis of CT findings alone [29]. Despite the advances in technology, CT findings are not absolute. Patt and Manning attribute four cases of blindness in a series of 159 patients with complicated acute sinusitis to negative or equivocal CT findings that delayed surgical therapy [23]. Radiographic imaging is integral to the diagnosis, staging, and surgical therapy for postseptal infections, but does not substitute for therapeutic decision-making.

Frontal sinus disease can be well-delineated only on CT imaging. Preoperative recognition of a frontal sinus etiology or an abscess in proximity to the frontal sinus is essential to proper surgical planning [7, 9]. There is some indication that frontal sinusitis complications may progress rapidly and result in worse outcomes than those infections arising from other paranasal sinuses [1]. Owing to the proximity and intimate connections of the frontal sinus to both the intracranial and orbital anatomy, response to therapy and progression of symptoms are especially important in patients with complicated frontal sinusitis.

Treatment of Orbital Complications of Sinusitis

Therapeutic options for the orbital complications of sinusitis generally correlate with the classification of infections. In general, treatment options will be based on the presence or absence of orbital signs (gaze restriction and proptosis), location of infection with regard to the orbital septum, progression of symptoms, responsiveness to medical therapy, and additional patient characteristics such as immune status and status of the contralateral eye [22, 23, 28].

Medical Therapy for Orbital Complications

Preseptal cellulitis, the most common orbital complication, is treated empirically with broad-spectrum intravenous antibiotics that cover the organisms listed above, have meaningful cerebrospinal fluid (CSF) penetration, and possesses activity against β-lactamase producing strains [6, 22]. Adjunctive topical and parenteral decongestants are often added, though steroids are not thought to be helpful [19, 24]. Patients who lack signs of postseptal involvement, such as proptosis, gaze restriction, decreased visual acuity, color vision changes, or afferent pupillary defect may be observed with serial ophthalmologic exams while receiving intravenous antibiotic therapy, deferring a CT scan for 24–48 h [6, 8, 10, 15, 19, 22, 28]. Progression of symptoms or failure to respond to antibiotics within 48 h of treatment necessitates a CT scan and is, in itself, an indication for surgical therapy.

Surgical Therapy for Orbital Infections

True preseptal cellulitis responds rapidly to intravenous antibiotics, and only in the rare case will surgery be required; typically the incision and drainage of a coalescing lid abscess [22]. In contrast, surgical intervention in postseptal disease is required in 12% to 66% of orbital complications of acute sinusitis [12, 24]. The indications for surgical therapy in postseptal infections comprise an evolving consensus of opinions from a number of large case series.

Surgery is recommended if any one of the following four indications is met [6, 23, 24, 28]:

- CT evidence of abscess formation
- Decreased visual acuity on presentation (20/60 or worse)

- Severe orbital complications on initial presentation with ipsilateral sinusitis (blindness, afferent papillary reflex, ophthalmoplegia)
- Progression of symptoms or failure to improve during the first 48 h of appropriate medical treatment

Immunocompromised patients (diabetes, chemotherapy, HIV) should be approached with a lower threshold for surgical intervention [23].

Though the above recommendations are widely accepted, dissenting opinions do exist. Souliere reported successful treatment with decongestants and intravenous antibiotics in five pediatric patients with subperiosteal abscesses and anterior ethmoiditis (Chandler Group III) [26]. In contrasting the risks of death or blindness resulting from progression of postseptal infection with the risks of endoscopic surgical techniques, our practice has been to favor operative exploration with regard to the indications listed above.

A number of different surgical techniques are applicable to the treatment of orbital complications of orbital sinusitis, though it is universally agreed that operative intervention should address the orbit and the paranasal sinuses simultaneously [6]. The advent of endoscopic surgical techniques has greatly reduced the morbidity of operative treatment. Chandler groups II (orbital cellulitis) and III (subperiosteal abscess) are routinely treated endoscopically; however, when inflammation precludes adequate drainage of the orbital infection, or ventilation of the involved sinuses, external techniques may be employed [20, 22, 25]. Chandler group IV usually requires an external ethmoidectomy and orbitotomy, though endoscopic techniques are gaining favor [6]. Cavernous sinus thrombosis, Chandler group V, is increasingly considered an intracranial complication of sinusitis, and as such its management should include neurosurgical consultation. Intravenous antibiotics are the primary therapeutic measure, though endoscopic surgery directed toward the involved sinuses (usually the ethmoid and sphenoid) is always recommended [6, 15, 19, 20, 22, 28]. Less clear is the utility of adjunctive steroids and heparin. Recent literature supports the use of steroids for cases of pituitary insufficiency; however, systemic anticoagulation remains controversial, balancing the bleeding risks with a potential decrease in thrombus propagation [6, 22].

Treatment of Orbital Complications of Frontal Sinusitis

The role of surgery in treating the orbital complications of frontal sinusitis is highlighted by the technical difficulties of operating on the acutely inflamed frontal sinus. Though the frontal sinus is only the third most frequently involved sinus in orbital infections, Hawkins' series found surgery to be required in every case of complicated frontal sinusitis [12]. Again, authors intimate that although frontal sinusitis is a less common source of orbital complications, those that take their origin from this sinus tend to be more aggressive in nature and portend more difficult clinical courses.

External frontoethmoidectomy had been an effective, commonly performed technique in the acute setting; however, complications including stenosis of the frontal sinus drainage tract (30%), CSF leak (5%), and diplopia (2%) have allowed endoscopic techniques to supplant this approach [20]. Frontal sinus trephine is an older technique that still has clinical value in the era of endoscopic sinus surgery. This simple and safe procedure can be employed acutely to treat complicated frontal sinusitis, allowing the surgeon to defer an endoscopic frontal sinusotomy until a time at which the operative field surrounding the frontal recess is less obscured by inflammation [20].

Conclusion

Orbital complications of sinusitis, though less frequent in the antibiotic era, are a source of morbidity and mortality that can be reduced further by attentive physical examination, prompt medical therapy, and strict adherence to the recommendations for surgical intervention. Orbital infections resulting from frontal sinusitis may be associated with a more

aggressive course, require surgery at a higher rate, and require external procedures if the challenging frontal recess anatomy is sufficiently obscured by inflammation. The role of intraoperative CT guidance in specifically treating orbital complications of sinusitis may have particular utility in allowing a wholly endoscopic approach to treating infections arising from acute frontal sinusitis.

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Chapter 8

CNS Complications of Frontal Sinus Disease

8

Andrew P. Lane

Core Messages

- **(Overview)** Although less common since the advent of antibiotics, CNS complications of frontal sinusitis still occur and warrant a high index of suspicion to permit timely diagnosis and management
- CNS complications of frontal sinusitis include meningitis, epidural abscess, subdural empyema, intracerebral abscess, and thrombosis of the cavernous sinus or superior sagittal sinus
- The frontal sinus is the most common sinus source of CNS complications
- Infection spreads to the CNS through vascular communications between the frontal sinus diploic veins and the dural venous plexus
- Progressive headache and fever are the most common presenting signs of CNS complications, although some may present silently
- The single most important study to obtain in the diagnosis of CNS complications of frontal sinusitis is a CT scan with and without contrast
- CNS complications of frontal sinusitis have a high incidence of long-term morbidity and mortality even with antibiotic therapy
- Treatment of CNS complications generally includes medical management with intravenous antibiotics, as well as surgical drainage of the frontal sinus and intracranial collections as indicated

Contents

Introduction

In the antibiotic era, intracranial complications of sinusitis have become less commonplace, but nevertheless continue to occur and be associated with significant morbidity and mortality. The frontal sinus is the most common source of intracranial complications of sinusitis, followed by the ethmoid, sphenoid, and maxillary sinuses [1]. Spread of infection from the frontal sinus to the intracranial space typically occurs by hematogenous spread through a communicating venous system. The small, valveless diploic veins (veins of Breschet) that extend through the posterior table of the sinus directly contribute to the venous plexi of the dura and periosteum [26]. Bacterial thrombi can travel throughout this network and seed intracranial sites remote from the frontal sinus, leading to meningitis, epidural or intracerebral abscesses, or subdural empyema. In some instances, a retrograde thrombophlebitis can develop and cause the further complications of cavernous or superior sagittal sinus thrombosis. Such life-threatening conditions must be recognized promptly and treated aggressively.

Epidemiology

Frontal sinusitis occurs most commonly in adolescent and young men, correlating with the time of peak development of the vascularity and pneumatization of the frontal sinus [19, 20, 32, 33]. The true incidence of frontal sinusitis complications today is unknown. Although the incidence of frontal sinusitis has not changed, it is clear that complications of sinusitis have become much less common, as antibiotic use has increased. More than a decade ago, a study of patients hospitalized for sinusitis showed an incidence of intracranial complications of 3.7% in that group [8]. Another study from the 1960's reported a 10% incidence of intracranial complications among patients admitted to the hospital for frontal sinusitis [2]. Regardless of how often it occurs, there continues to be a significant degree of morbidity and mortality associated with intracranial complications of acute frontal sinusitis, particularly if intervention is delayed.

Signs and Symptoms

The typical presentation of CNS complications of frontal sinusitis is characterized by:

- Acute or progressive headache
- Fever

The process may be silent until serious neurological symptoms and signs develop such as:

- Focal neurological deficits
- Change in mental status
- Lethargy
- Seizure
- Coma

The presentation depends in part on the location of the infection; for example, with frontal lobe involvement, the only manifestation may be a subtle change in personality. Superior sagittal sinus thrombosis is frequently associated with nausea and vomiting, in addition to severe headache. Patients do not necessarily complain of rhinosinusitis symptoms such as nasal congestion and rhinorrhea at the time of presentation, but may give a history of sinusitis symptoms and localizing frontal pressure or discomfort. In a small number of cases, there may be osteomyelitis of the anterior frontal sinus table, causing overlying edema of the forehead or even a pericranial abscess (Pott's Puffy Tumor).

Clinical Features and Diagnostic Evaluation

Patients with suspected intracranial complications of frontal sinusitis should undergo high-resolution computed tomography (CT) with and without contrast as the primary diagnostic test [8]. Input from otolaryngology, neurosurgery, ophthalmology, and infectious diseases services are important in creating a multidisciplinary approach to the care of the patient [21]. The need for lumbar puncture to rule out meningitis must be weighed against the risk of precipitating brain herniation, as determined by the imaging studies and signs of increased intracranial pressure. If elevated intracranial pressure has been excluded, lumbar puncture should be performed, with cytological, microbiological, and laboratory analysis of the cerebrospinal fluid [15].

Patients with sinusitis and the following signs should be presumed to have meningitis until proven otherwise:

- Persistent high fever
- Severe headache
- Meningismus
- Photophobia
- Irritability
- Altered mental status

However, meningitis is seldom caused by isolated frontal sinusitis, and it is more likely to result from ethmoid or sphenoid sinusitis or intracranial abscesses, which may occur in the epidural space, the subdural space, or intraparenchymally [9].

Epidural abscesses most commonly occur directly behind an intact posterior table of the frontal sinus. The dura is loosely attached in this region, allowing pus to collect and expand [1]. Symptoms may be very mild until the collection becomes large enough to increase intracranial pressure. Because of the proximity to the orbit, orbital swelling is common, together with forehead edema and tenderness. Other than the increased pressure, lumbar punctures are usually normal with epidural abscesses [25, 26].

Infections in the subdural space also do not yield diagnostic lumbar punctures, but may be associated with increased pressure, elevated protein, and pleocytosis, with normal glucose and lack of organisms [1, 20]. The subdural space is a potential space between the arachnoid matter and the dura. The arachnoid prevents extension of the infection to the leptomeninges, but allows transmission of local inflammation through to the underlying cortex [6]. Pus in the subdural space also precipitates vasculitis and septic venous thrombosis. The inflammatory edema and venous obstruction tends to lead to a cycle of increasing edema and infarction, creating a far greater degree of intracranial hypertension than the mass effect of the empyema itself [27]. The infection may spread freely in the subdural space, posteriorly over the cerebral hemisphere and inferiorly into the interhemispheric fissure. The infection may then spread to the contralateral side of the brain under or through the falx cerebri [26].

Subdural empyema usually presents with:

- Increasing headache
- Fever
- Elevated white blood cell count
- Meningeal signs

As the process progresses, cortical signs and symptoms develop such as:

- Hemiparesis
- Hemiplegia
- Cranial neuropathies
- Seizure

Ultimately, the increase in intracranial pressure causes [1, 26]:

- Nausea
- Vomiting
- Slowed heart rate
- Hypertension
- Decreased level of consciousness

Death may occur from transtentorial herniation, which may be precipitated by lumbar puncture in the setting of markedly elevated intracranial pressures [20].

Dural sinus thrombosis can result directly from septic emboli from the frontal sinus, or secondary to epidural, subdural, or brain abscesses. Patients with thrombosis of the superior sagittal sinus or the cavernous sinus are generally very ill appearing [15]. Meningeal signs and/or focal neurologic deficits are almost always evident at presentation.

In cavernous sinus thrombosis, the key findings are:

- Proptosis
- Chemosis
- Ophthalmoplegia
- Cranial nerves II and III palsies
- Visual loss develops as the disease process worsens
- Contralateral involvement is pathognomic

In addition to the physical exam findings, dural sinus thrombosis is usually evident on contrast CT, MRI, and MR venogram [11]. Venous engorgement, particularly of the superior ophthalmic vein in cavernous sinus thrombosis, is an important diagnostic finding. Lumbar puncture is not diagnostic.

Brain abscesses due to frontal sinusitis most commonly derive from septic emboli that travel to the frontal lobe via retrograde venous communications. Typically, there will be liquefaction necrosis of the brain surrounding the infected vein, with surrounding edema [32]. Because the blood supply is less robust, abscesses tend to form in the white matter rather than the gray matter, and they become encapsulat-

ed over weeks [24]. The initial symptoms of brain abscess may be very mild or nonexistent. Only with significant edema can focal neurologic signs or signs of increased intracranial pressure be seen. Unfortunately, brain abscesses may not be apparent until they rupture into the ventricular system, causing rapid death. In other cases, rapid growth of the abscess and reactive edema may cause uncal herniation through mass effect (Figs. 1–3).

Treatment

The organisms most commonly cultured either from the frontal sinus or from intracranial collections are staphylococcus and streptococcus species [18, 19]. Other gram-positive bacteria may be found, as well as anaerobes, and gram negatives such as *H. influenzae* [4]. Patients with intracranial complications of frontal sinusitis should be admitted to the hospital for aggressive intravenous antibiotic therapy with broad-spectrum agents that penetrate the blood-brain barrier. Culture results will ultimately direct the choice of antibiotic, but agents such as penicillinase-resistant penicillins, vancomycin, and third-generation cephalosporins provide appropriate initial coverage [15]. The roles of mannitol and corticosteroids for brain edema, and anticoagulants for dural sinus thrombosis, are controversial, but may be indicated in certain situations [29, 30]. Currently, anticoagulation is favored in superior sagittal sinus thrombosis (SSST) but not cavernous sinus thrombosis, as long as there is no gross blood on CT or lumbar puncture [31]. After neurological consultation, anticonvulsants may also be administered because of the significant association of seizures with intracranial complications.

Management principles of frontal sinus-related intracranial complications:

- In most cases, management of intracranial complications requires surgery in addition to medical therapy
- Ideally, when indicated, both the intracranial process and the sinus infection should be addressed at the same surgical procedure [8, 18,

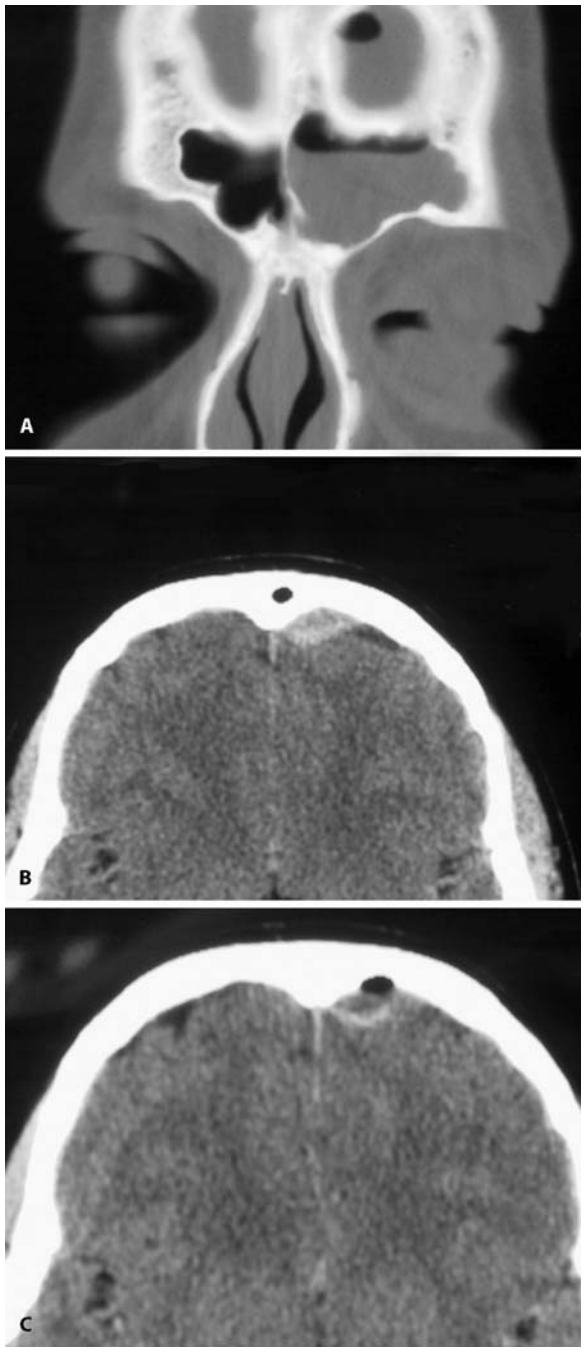

Fig. 8.1A–C. Frontal lobe pneumococcal abscess secondary to frontal sinusitis. **A** Coronal CT showing opacification of left frontal sinus. **B** Axial CT demonstrating abscess of frontal lobe

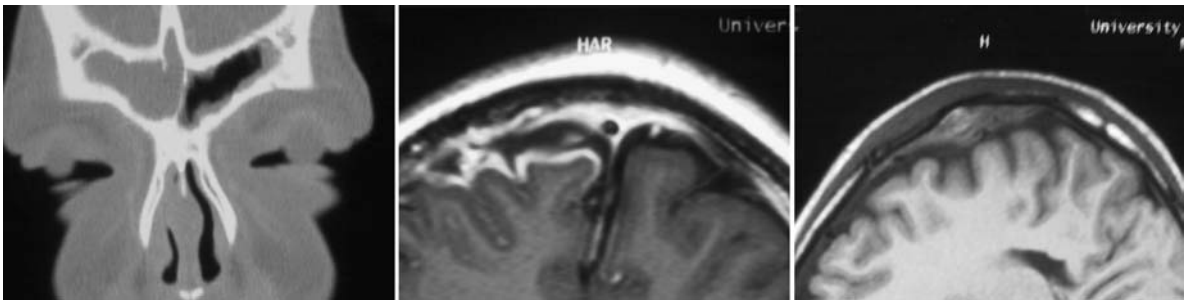

Fig. 8.2. Frontal sinusitis causing meningitis and frontal lobe abscess. Cultures of CSF and the abscess revealed staphylococcus A

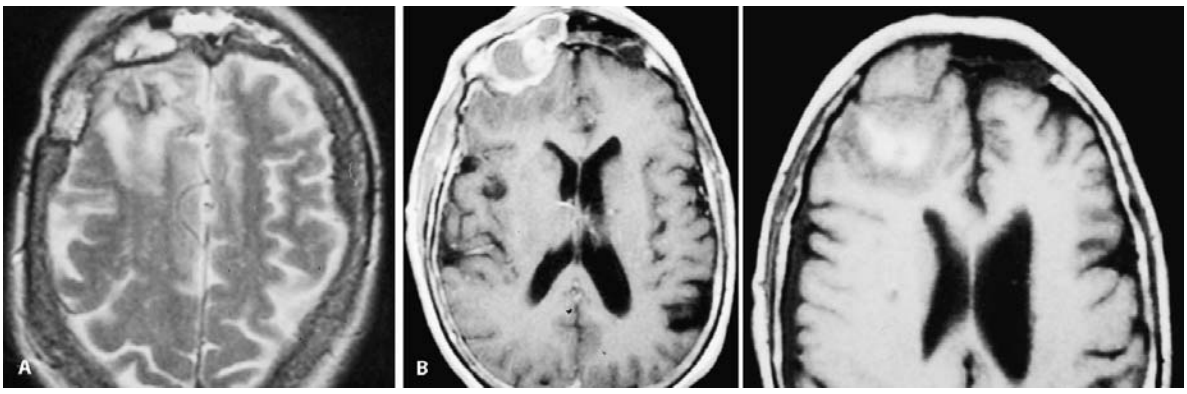

Fig. 8.3A,B. Frontal sinusitis causing septic thrombophlebitis and hemorrhagic brain infarction. **A** T2-weighted MRI demonstrating abscess. **B** T1-weighted image with higher signal intensity in the area of brain infarction

21, 26]. This theoretically prevents further seeding of the intracranial space from the infected sinus and has been shown to decrease the incidence of neurosurgical and sinus re-exploration.

- In the acute setting, drainage of the frontal sinus takes precedence over establishing improved intranasal outflow. Typically, the surgical intervention of choice is a frontal sinus trephination with drainage of the infected material and irrigation of the sinus [12, 21].

The trephination may be combined with an endoscopic frontal sinusotomy if the conditions are favorable [13], or a catheter may be brought out through the brow incision to allow for postoperative irrigation and to prevent re-accumulation of purulence. If the frontal table of the sinus is necrotic or eroded by osteomyelitis, wide surgical debridement of the bone is necessary, along with prolonged intravenous antibiotic therapy. Reconstruction of the defect is delayed until the infection is resolved, as demonstrated by gallium-67 citrate scan [12].

Surgical treatment of uncomplicated epidural abscess involves creation of burr holes without opening

the dura [35]. In the pediatric age group, there is evidence that this type of neurosurgery may not always be necessary, provided that adequate sinus drainage is achieved, there is minimal mass effect from the abscess, and the patient is given appropriate antibiotic therapy [16]. Subdural empyema may be managed by either burr holes or craniotomy, with opening of the dura to drain the collection [8]. Craniotomy provides wider access and may allow recognition of extensions of the empyema that would be missed with burr holes alone. On the other hand, with improved radiologic studies to localize the abscess, burr holes are sufficient in most cases [3]. When there is a brain abscess, the need for surgery depends largely on the extent of the abscess. Small or multiple abscesses, particularly in a stable patient or when located in an inaccessible area, are often managed medically with close observation [34]. Larger abscesses need to be drained to relieve the mass effect, which can be accomplished via aspiration or excision. Aspiration, or repeated aspiration, has the advantage of being less traumatic and is associated with fewer long-term sequelae [23]. Aspiration allows identification of the infecting organism to guide antibiotic therapy. Surgical excision of the abscess through a craniotomy is more definitive and may be desirable in a stable patient when the abscess is large, well-encapsulated, and not involving primary cortical areas. Excision may also be necessary when aspirations are unsuccessful [1].

The role of surgery in the management of dural sinus thrombosis is not completely defined, other than drainage of the frontal sinus source. Exploration of the cavernous sinus is generally not recommended, although it has been reported. Similarly, superior sagittal sinus thromboses are usually not explored, except in rare instances when thrombectomy is performed for very extensive thrombi [10]. Another interventional approach in this situation is the local infusion of thrombolytic agent into the dural sinus system [7, 14].

Prognosis

With the availability of antibiotic therapy, the incidence of intracranial complications of frontal sinusitis has decreased considerably. However, the morbidity and mortality of intracranial complications, once they occur, remains high.

A large series from 1991 reported a 33% incidence of long-term morbidity following intracranial complications of sinusitis, with the following sequelae being the most common [8]:

- Hemiparesis
- Hypesthesia
- Seizure disorder

Delay in surgical intervention was shown to correlate with increased long-term morbidity. In general, neurologic morbidities from meningitis are common, and systemic postinfection sequelae may also occur in the pediatric population [17]. Subdural empyema and brain abscess have greater mortality rates than meningitis, and survivors frequently suffer from the morbidities mentioned above, as well as variable cognitive deficits or focal cranial neuropathies [23]. Of all the CNS complications, the mortality from dural sinus thrombosis is the greatest, perhaps as high as 50%–80% [30]. Prior to antibiotics, these complications were virtually uniformly fatal.

Conclusion

Potent antibiotics and modern advancements in radiology have made intracranial complications of acute frontal sinusitis far less common than they once were. Nevertheless, such complications continue to occur and can result in long-term morbidities, particularly if diagnosis is delayed. It is therefore essential for the otolaryngologist to be cognizant of the potential for CNS complications, in order to initiate prompt, aggressive medical and surgical therapy. With early recognition and a multidisciplinary approach to management, improved outcomes may be possible for these serious disease processes.

CNS Complications of Frontal Sinusitis

- Meningitis
- Epidural abscess
- Subdural empyema
- Brain abscess
- Cavernous sinus thrombosis
- Superior sagittal sinus thrombosis
- Frontal bone osteomyelitis

Management of Suspected CNS Complications of Frontal Sinusitis

- Admit to hospital
- High-resolution CT scan with contrast of the head and paranasal sinuses
- Consider head MRI or MR venogram for dural sinus thrombosis
- Lumbar puncture if no evidence of increased intracranial pressure
- Neurosurgery, ophthalmology, infectious diseases consultations
- Broad-spectrum antibiotics that cross blood-brain barrier
- Drainage of affected frontal sinus via trephination
- Consider intranasal frontal sinusotomy if conditions favorable
- Coordinate with neurosurgery if drainage of intracranial abscess indicated
- Focus antibiotic coverage once cultures available
- Monitor for clinical and radiographic improvement

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Chapter 9

Frontal-Orbital-Ethmoid Mucoceles

9

James Palmer, Ioana Schipor

Core Messages

- Mucoceles are the most common benign tumor of the paranasal sinuses, and have a predilection for the anterior ethmoid cavity, most likely due to the labyrinthine nature of the anatomic region
- Treatment of mucoceles is surgical, with emphasis on the newer, less invasive endoscopic techniques.
- Evaluation is best carried out by CT scanning, with MRI and nasal endoscopy as adjuncts
- Great care must be taken in the postoperative period to keep the opening of a drained mucocele patent until normal mucociliary clearance is able to be re-established

Contents

Introduction

Mucoceles are slow-growing, benign expansile lesions found in the paranasal sinuses. On histopathology, they are cyst-like structures lined with respiratory epithelium and filled with mucus. Infected mucoceles are known as mucopyoceles. Mucoceles are locally destructive lesions causing bony resorption and displacement of adjacent structures, most notably the orbital contents. Treatment is surgical, and originally involved removal/resection of the entire lesion. As surgical instrumentation has improved and the pathophysiology is better understood, surgical treatment of mucoceles has evolved into procedures that are less invasive and emphasize surgical drainage over ablation.

Epidemiology

Mucoceles are uncommon in adults [16, 25, 28, 32]. These lesions can form in any of the paranasal sinuses. The first series of 14 patients [15] reported the frontal sinus as their most common location. Subsequent series have shown that approximately 60%–89% occur in the frontal sinus, followed by 8%–30% in the ethmoid sinuses, and less than 5% in the maxillary sinus. Sphenoid sinus mucoceles are rare [1, 21]. There are several case reports of mucoceles occurring in unusual locations, such as the pterygomaxillary space, orbital floor, and middle turbinate.

Mucoceles can form at any age, but the majority are diagnosed in patients 40 to 60 years old [1]. Males and females are equally affected. The incidence of skull base bony destruction and intracranial extension has been reported to be between 10% and 55% [10, 19].

Paransal sinus mucoceles are extremely rare in children, although several case reports [13, 18] and a small series of pediatric mucoceles [13] have been published. Some authors have noted an association between mucoceles and cystic fibrosis patients [8]; however, this is not always the case, and most pediatric frontal sinus mucoceles appear to be idiopathic.

Pathophysiology

Mucoceles develop after obstruction of the sinus ostium. They enlarge slowly and fill the affected sinus cavity, expanding and eroding the adjacent bony structures. Secondary infection can lead to a period of rapid expansion with a resultant increased risk of complications, especially in the periorbital area [30].

One proposed mechanism for mucocele formation is cystic degeneration of a seromucinous gland, resulting in a retention cyst [3]. However, detailed histopathologic studies have shown little evidence for this mechanism and instead have pointed to the dynamic interface between bone and mucocele lining as being responsible for mucocele expansion. It is generally thought that following obstruction of the frontal recess and subsequent infection within the frontal sinus cavity, continued stimulation of lymphocytes and monocytes leads to the production of cytokines by the lining fibroblasts. These cytokines, in turn, promote bone resorption and remodeling and result in expansion of the mucocele [25]. Bone erosion results from mass effect as well as from the presence of cytokines such as IL-1 and IL-6 [24]. Cultured fibroblasts derived from frontoethmoidal mucoceles have been shown to produce significantly elevated levels of prostaglandin E2 and collagenase, compared with normal frontal sinus mucosa fibroblasts. This suggests that the lining fibroblasts represent a major source of bone-resorbing factors [23].

Common etiologic factors related to frontoethmoid mucocele formations include: a known history of sinusitis, previous sinus surgery, allergy, and trauma (Table 9.1). Surgery can lead to mucocele formation either by directly blocking the sinus ostium with scar tissue or by entrapping sinus mucosa. Postsurgical sinus mucoceles can occur up to several years after the initial operation. Frontal sinus mucoceles were reported in 9.3% of cases after osteoplastic flaps [9]. Mucoceles have been described after both external and endoscopic sinus surgery [5, 12, 26, 28].

Table 9.1. Paranasal sinus mucoceles: common etiologies

Chronic rhinosinusitis
Previous sinus surgery
Previous maxillofacial trauma
Allergies
Tumors
Idiopathic

Uncommonly, mucoceles form as result of an ostial occlusion caused by a benign neoplasm (osteoma, fibrous dysplasia), or a malignant tumor [14, 30]. In as many as one-third of cases, however, the history is noncontributory and no demonstrable cause can be found [21].

Culture of the aspirated mucocele contents can sometimes confirm the presence of infection. A study demonstrated that the most common isolates were *Staphylococcus aureus*, alpha-hemolytic streptococci, *Haemophilus* species, and gram-negative bacilli. The predominant anaerobic isolates were *Propionibacterium acnes*, *Peptostreptococcus*, *Prevotella*, and *Fusobacterium* species [4].

Presentation

The expanding mucocele often compresses the orbit and, not surprisingly, many patients present initially to the ophthalmologist with orbital symptoms, such as pain, proptosis, diplopia, exophthalmos, globe dis-

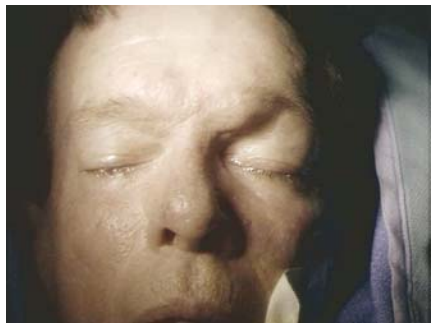

Fig. 9.1. Frontal sinus mucocele: left orbital proptosis

Table 9.2. Paranasal sinus mucoceles: common clinical presentations

Orbital symptoms: proptosis, globe displacement, diplopia, blurred vision, epiphora
Nasal symptoms: obstruction, mucopurulent rhinorrhea
Headaches
Facial or frontal swelling

placement, decreased visual acuity, or epiphora [2] (Fig. 9.1). Orbital expansion of the mucocele can lead to globe displacement, leading to exposure keratitis and central retinal block in more severe cases [7]. Other common presentations include headaches, facial pressure or swelling, nasal drainage, and obstruction (Table 9.2).

Intracranial extension through erosion of the posterior wall of the frontal sinus can lead to meningitis or CSF fistula [27, 31]. The posterior sinus wall is particularly prone to erosion because it is inherently thin. The tendency for bony erosion and intracranial extension is seen more often in the presence of infection.

Diagnosis

The diagnosis of a mucocele is based on the history, physical examination, and radiologic findings. Apart from the presenting features described above, often a palpable mass in the frontal region or in the area of the medial canthus accompany the proptosis and globe displacement. Office nasal endoscopy should assess other possible intranasal findings, such as polyposis, nasal septal deviation, etc., that may be addressed at the time of surgery.

Imaging plays a key role in the diagnosis of most mucoceles. Frontal sinus mucoceles can be seen on plain X-rays; however, lesions in the anterior ethmoids, sphenoid, and maxillary sinuses are difficult to diagnose using this modality.

The imaging of choice is CT scanning in both axial and direct coronal planes [21]. It clearly delineates the mucocele as a well-delineated, cyst-like, homogeneous lesion originating in a paranasal sinus and compressing surrounding structures. The bony changes surrounding the lesion can easily be seen (Fig. 9.2). The mucocele content demonstrates homogeneous mucoid attenuation (10–18 HU). Longstand-

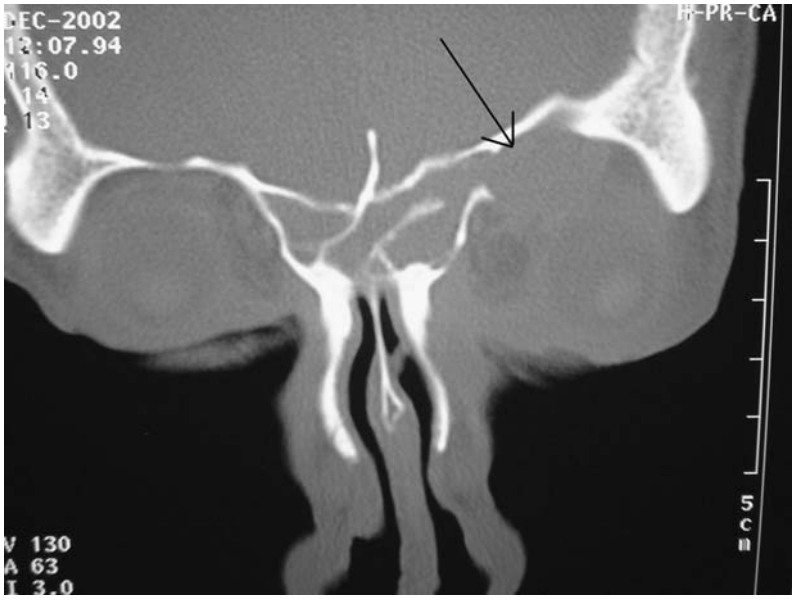

Fig. 9.2.
Coronal CT (bone windows) demonstrating opacification of the left frontal sinus with erosion of the orbital roof (*arrow*)

ing lesions have higher protein content and attenuate more (20–40 HU). Contrast enhancement is rarely necessary; however, after intravenous contrast medium injection the lesion shows rim enhancement.

Magnetic resonance imaging is useful when the diagnosis is uncertain and it is necessary to differentiate between different types of soft tissues within the sinonasal cavities, especially if the mucocele formed secondary to a neoplasm. Additionally, when the mucocele extends intracranially, MRI offers superior imaging of the surrounding brain. The usual signal characteristics for a mucocele are low T1 and high T2, but variations commonly occur depending on the presence of blood and the water content of the mucocele. Post-gadolinium images confirm the presence of fluid within the mucocele by showing absent signal [21]. Contrast-enhanced MRI is especially useful for delineating secondary mucocele formation: the nonenhancing mucocele is differentiated from the causative lesion (e.g. an obstructing tumor). It should be remembered that MRI does not provide the surgeon with the same bony detail that is available from CT.

Classification

Frontal sinus mucoceles can have various sizes and configurations. The degree of intraorbital involvement is not used to differentiate between the different types of lesions.

The following classification system was devised in order to standardize frontal sinus mucocele evaluation and management [11]:

- Type 1. Limited to frontal sinus (with or without orbital extension)
- Type 2. Frontoethmoid mucocele (with or without orbital extension)
- Type 3. Erosion of the posterior sinus wall
  - A. Minimal or no intracranial extension
  - B. Major intracranial extension
- Type 4. Erosion of the anterior wall
- Type 5. Erosion of both anterior and posterior wall
  - A. Minimal or no intracranial extension
  - B. Major intracranial extension

Treatment

The treatment of mucoceles is surgical. The goals of surgery are eradication of the mucocele with minimal morbidity and prevention of recurrences. Surgical approaches are based on the size, location, and extent of the mucocele. In the presence of infection, adjuvant antibiotic treatment is indicated. Since many of these lesions have an intracranial or intraorbital component, ideally the surgery should not be performed in the setting of an infection. The exception is an acute symptomatic mucopyocele.

Traditional teaching in the United States emphasized that the entire lining of a sinus mucocele must be completely removed. Historically, surgical therapy involved an external approach (Lynch-Howarth frontoethmoidectomy) or osteoplastic flaps with sinus cavity obliteration. These procedures carried significant morbidity and cosmetic deformity, as well as a significant rate of recurrence [29]. Additionally, postoperative radiographic follow-up became difficult after obliteration.

- More recent reports have shown that complete removal of the sinus lining is not necessary, and marsupialization is sufficient as long as ventilation of the sinus cavity is maintained [11]

Endoscopic drainage has been advocated in the belief that preservation of the frontal sinus mucosa and maintenance of a patent frontal recess result in a better clinical outcome [20].

In 1989 Kennedy et al. published the first series of 18 mucoceles treated by endoscopic marsupialization. Their study reported zero percent recurrence rate after follow-up averaging 18 months [18]. Another study, with longer follow-up, examined the recurrence rate in two groups of patients with sinus mucoceles: the first group was treated endoscopically (20 patients) and the second treated using a combined external and endoscopic approach (28 patients) [22]. The combined approach was used in the more severe cases where the anatomy, extent of disease, or previous surgery restricted endoscopic visu-

alization and access to the frontal sinus, as well as in cases where a fistulous tract was present. There were no recurrences in the group managed exclusively via a transnasal endoscopic approach after a mean follow-up of 34 months. There were three recurrences (11%) in the combined endoscopic/external drainage group after a mean follow-up of 44 months. Although it is difficult to directly compare these recurrence rates given the difference in severity of disease in the two patient groups, the endoscopic approach was clearly shown to be safe and efficacious, with minimum associated morbidity (Figs. 9.3 and 9.4).

Har-El has published the largest series of patients with mucoceles in the English literature [10]. One hundred and three patients with 108 paranasal sinus mucoceles were treated by wide endoscopic marsupialization. Postoperative stents were used in frontal mucoceles. His recurrence rate was 0.9% (one patient) after a mean follow-up of 4.6 years. The rate of major complications was also very low, with only one patient experiencing an intraoperative CSF leak, which resolved after immediate repair and postoperative bedrest. The author concluded that the endoscopic drainage should be considered the procedure of choice for management of paranasal sinus mucoceles.

The endoscopic approach is particularly useful when an extensive frontal mucocele has eroded the posterior frontal sinus wall. In these cases sinus obliteration is problematic given the difficulty of completely removing the lining mucosa from exposed dura [11].

No complications were reported in the small pediatric series reported by Hartley and Lund [13]. Seven children underwent endoscopic drainage of ethmoid and sphenoid mucoceles, and there were no recurrences after one-year follow-up.

Complex cases with extensive intracranial extension have been managed in a number of different ways. Neurosurgeons tend to use an open approach (craniotomy) and to remove the entire cyst lining [6]. Other authors have advocated wide marsupialization via an endoscopic transnasal approach [17]. Alternatively, mucoceles with intracranial extension are approached with a combined craniofacial and endoscopic approach [22].

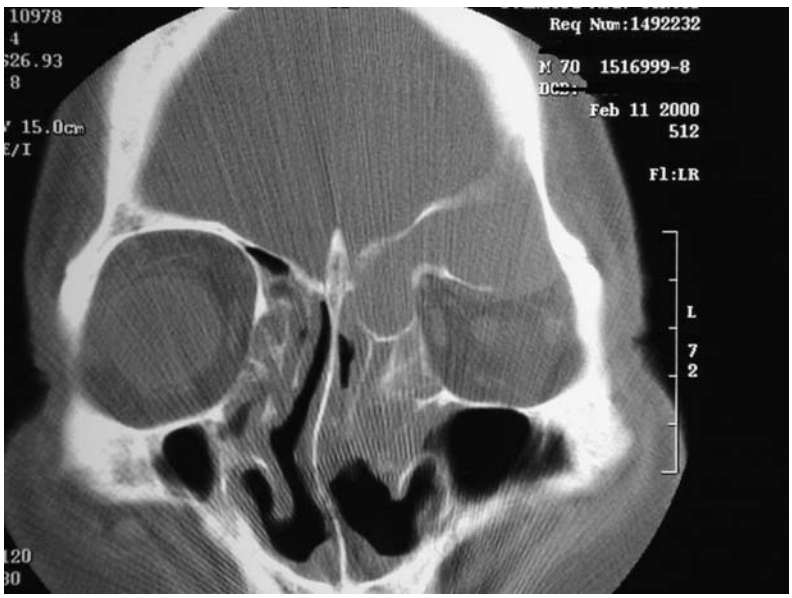

Fig. 9.3.
Preoperative CT of left frontal orbit mucocele eroding into the orbit

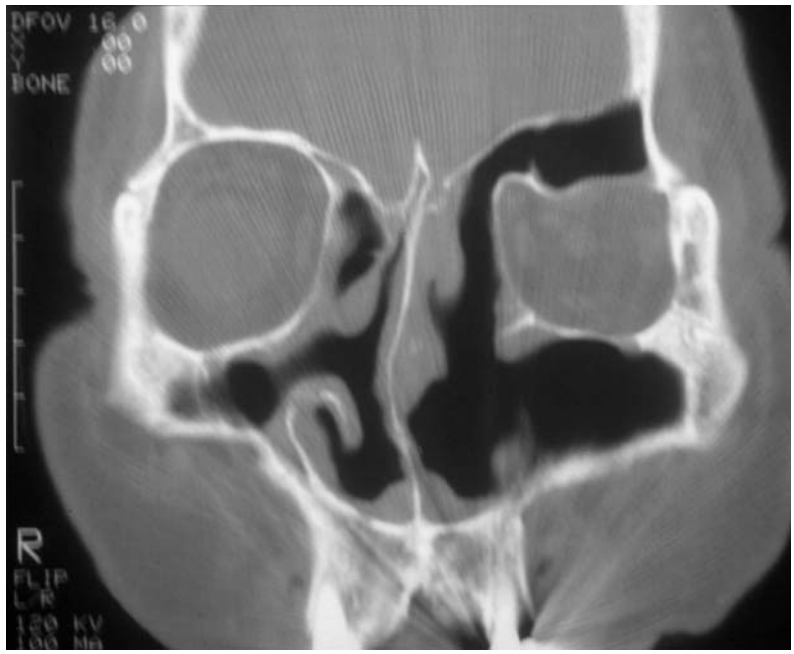

Fig. 9.4.
Postoperative CT after endoscopic drainage of mucocele

Surgical Technique

All patients should undergo preoperative CT scanning. The benefits of computer-aided, CT-based stereotactic navigation techniques have not yet been fully evaluated. In theory, however, stereotactic guidance may offer some advantages and may reduce the risk of surgical complications by being able to localize small mucoceles and by improving surgical orientation, especially in revision cases where anatomical landmarks may be distorted or missing.

The procedure can be performed either under local or, more commonly, under general anesthesia. The nose is topically decongested. Once the surgical landmarks are identified endoscopically, the mucocele is opened into the nasal cavity. The bone overlying the mucocele is usually thin and may be dehiscent [13]. Specimens should be sent for microbiology analysis. After entering the sac, the mucocele is then widely marsupialized in order to prevent reaccumulation. Occasionally the mucocele is filled with thin, clear fluid, raising suspicion of a CSF leak intraoperatively [22]. The medial orbital wall is often eroded in the case of ethmoid mucoceles, and the globe is obviously at risk in these cases during the drainage procedure. Postoperative packing is not routinely used. Attention to postoperative nasal hygiene, including nasal irrigation and topical steroids is critical. If the contents of the mucocele are purulent or if the microbiological cultures are positive, oral antibiotics are used. Close endoscopic follow-up postoperatively should be continued until the cavity heals and mucociliary clearance re-establishes.

Postoperatively, temporary diplopia after globe repositioning can occur. Recurrences are possible, although not common.

Conclusion

Mucoceles are the most common benign lesions of the paranasal sinuses. Ninety percent occur in the frontal and ethmoid sinuses and frequently cause destruction of the surrounding bone, including the

orbit. Diagnosis is made by CT scan. Over the past fifteen years the increasing use of endoscopic sinus surgery has resulted in safe and successful drainage of a large proportion of anatomically suitable lesions with minimal rates of recurrence and morbidity. Complex or revision cases may necessitate a combined endoscopic and external drainage procedure in order to prevent recurrence.

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Chapter 10

Pott's Puffy Tumor

10

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Core Messages

- Pott's puffy tumor was defined by the 18th century surgeon Percival Pott as a subperiosteal abscess of the frontal bone
- While originally described as a complication of trauma, this condition typically results from acute frontal sinusitis
- Spread of disease can occur by direct infection of the bone or by thrombophlebitis of the veins that perforate the anterior and posterior tables of the frontal sinus
- Intracranial infection commonly complicates Pott's puffy tumor. Headache and forehead swelling may be the only presenting symptoms so that radiologic evaluation of the brain is mandatory
- Broad-spectrum antibiotics must be instituted upon diagnosis and should include coverage of microaerophilic streptococcus species
- Surgical treatment includes drainage of the frontal sinus and the subperiosteal abscess, as well as neurosurgical intervention for any intracranial complications. Inspection of the frontal bone should be performed, either radiologically or directly, followed by debridement of necrotic foci

Contents

Introduction

Sir Percival Pott (1714–1788) was a surgeon of St. Bartholomew's Hospital in London who wrote a large number of treatises on subjects as varied as orthopedics, urology, and neurosurgery [6]. In 1760, he produced his *Observations on the Nature and Consequences of Wounds and Contusions of the Head, Fractures of the Skull, Concussions of the Brain, etc.* In this work he described "a puffy, circumscribed, indolent tumor of the scalp, and a spontaneous separation of the pericranium from the scull (sic.) under such a tumor" [3]. Hence was born the alliterative appellation, Pott's Puffy Tumor.

While originally described as a consequence of head trauma, this entity has become more commonly associated with complications of frontal sinusitis. The classic use of the Greek term "tumor" for swelling is rarely used today, instead having a modern connotation of a neoplasm. As defined by Pott, this "tumor" or swelling of the forehead is formed by a subperiosteal abscess. Pott termed this infectious collection as "matter" and went on to observe that it often appeared with "inflammation of the dura mater and the formation of matter between it and the

skull" [2]. Patients with subperiosteal abscesses of the frontal bone typically demonstrate focal necrosis of the frontal bone as well. Thus intracranial and osteomyelitic complications of frontal sinusitis are often associated with what Pott originally described as a "puffy tumor."

Anatomy and Pathogenesis

The frontal sinuses form as pneumatic extensions of the anterior ethmoid complex that project into the diploic space of the frontal bone. This process begins in infancy but progresses slowly, only becoming radiologically evident at 6 years of age [5, 9]. For this reason, complications of frontal sinusitis, including Pott's puffy tumor, are relatively rare in younger children.

Infection from the frontal sinus may progress beyond the confines of the sinus by direct extension from either [1, 8]:

- Focal osteitis or osteomyelitis or
- Through infectious thrombophlebitis

The posterior table of the frontal sinus is almost completely composed of compact bone, whereas the anterior table contains both compact and cancellous bone. Aggressive infection of the frontal sinus mucosa can invade directly into the underlying bone. Progressive infection leads to the development and expansion of poorly vascularized or necrotic sequestra of bone. Osteitis can continue through the full thickness of the posterior table to the dura and epidural space, whereas transmural osteomyelitis of the anterior table can directly extend to the pericranium.

Progressive thrombophlebitis without overt bone infection is another potential source of Pott's puffy tumor and its frequently associated intracranial complications. Venous drainage of the frontal sinus mucosa passes through valveless diploic veins that extend posteriorly to the dura and anteriorly to the pericranium. Infectious thrombophlebitis can therefore extend posteriorly, causing epidural abscess or meningitis. More rarely, septic thromboemboli can lead to frontal lobe abscess. Thrombophlebitis of the anterior table can similarly lead to infection of the frontal pericranium and development of Pott's puffy tumor. As the pericranium is elevated off of the underlying frontal bone by expansion of the abscess, the vascular supply to the bone is further compromised, promoting necrosis and osteomyelitis.

Clinical Presentation

Pott's 18th century description of frontal subpericranial abscess still remains pertinent over 200 years later [2]:

- Patients typically do not have a history of chronic or recurrent acute frontal sinusitis
- Pott's puffy tumor can rarely complicate chronic frontal disease
- Symptoms of frontal sinusitis can be present for a variable amount of time prior to development of forehead swelling, ranging from just a few days to months
- Previous treatment with antibiotics is common

Focal doughy or pitting forehead swelling heralds the presence of a subpericranial abscess. Often significant tissue edema surrounds and overlies the abscess and may extend into the preseptal orbital tissues.

Associated symptoms include:

- Headache
- Fever
- Nasal drainage
- Frontal sinus tenderness

Males appear to be more commonly affected than females [1, 8].

As Pott noted in his 1760 description, intracranial complications are frequently associated with Pott's puffy tumor.

Pott's described an epidural abscess ("matter"), but conditions that can also complicate this disease include:

- Meningitis
- Venous sinus thrombosis
- Subdural abscess
- Brain abscess

Despite the presence of such serious intracranial sequelae, headache and doughy edema of the forehead may be the only presenting symptoms. For this reason, any patient presenting with Pott's puffy tumor should be evaluated radiographically for intracranial infection (Fig. 10.1) [2].

In addition to imaging the brain itself, imaging can also be helpful in delineating areas of chronic osteomyelitis and in defining the size of the subpericranial abscess. Imaging of the orbit is also indicated in the presence of preseptal cellulitis or when vision or extraocular muscle movements are compromised. A contrast-enhanced computed tomographic (CT) study is the most effective imaging modality. as it allows for soft tissue and bone evaluation [3]. In order to further delineate the degree of bone infection and necrosis, nuclear medicine imaging may be useful. Merging nuclear medicine and CT imaging can yield precise localization of osteomyelitis [10].

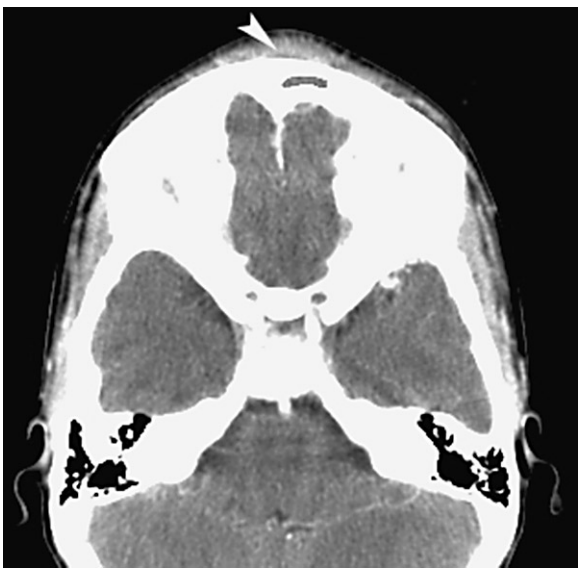

Fig. 10.1. Axial CT image demonstrating a small subperiosteal collection anterior to the frontal bone (*arrowhead*) with an associated intracranial abscess. Image courtesy of Albert Park, MD

Treatment

Once the extent of disease is defined, effective treatment can be initiated. The source of the infection, the frontal sinus, must be addressed as well as the subpericranial abscess and any bone or intracranial infection. Appropriate antibiotics must also be initiated.

Treatment of the frontal sinus is most easily accomplished through a trephine, although endoscopic treatment of the frontal sinusitis may also be effective [4]. Similarly, a limited subpericranial abscess can be drained through a small incision. The drawback of this minimally invasive approach is the inability to directly inspect the frontal bone for any necrotic areas.

When intracranial complications are present, simple drainage of the frontal sinus and the extracranial abscess will likely be insufficient. Because patients may deteriorate quickly from expansion of intracranial abscesses, prompt neurosurgical intervention is mandatory. Intracranial complications are typically treated with a bifrontal craniotomy, with thorough inspection of the frontal bone for necrotic areas and debridement of these areas when discovered [2]. This may necessitate a complete removal of posterior table of the frontal bone with cranialization of the frontal sinus or removal of the anterior table and collapse of the forehead skin onto the posterior table, known as a Riedel procedure (Fig. 10.2). The Riedel procedure carries with it significant aesthetic consequences which can be corrected with alloplastic or autogenous materials after sufficient time has passed to eradicate the original infectious process.

Materials used to reconstruct forehead contour after the Reidel procedure include:

- Split calvarial bone grafts
- Polymethyl-methacrylate
- Hydroxyapatite
- Titanium mesh

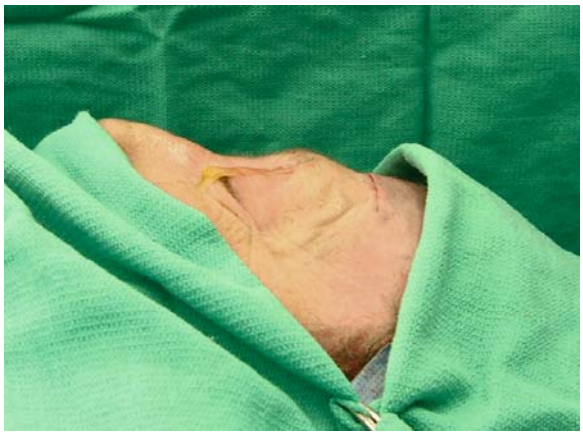

Fig. 10.2. Removal of the anterior table of the frontal bone (Riedel procedure) leaves a significant aesthetic defect

All these materials have been used successfully, and each has its inherent advantages and disadvantages [7].

In addition to prompt surgical intervention, intravenous antibiotics must be initiated early and continued for sufficient time, usually six weeks.

Organisms cultured from Pott's puffy tumor tend to be:

- Microaerophilic streptococci, including alpha-hemolytic streptococcus and peptostreptococcus
- Anaerobic bacteria

Obstruction of the frontal sinus by inflammatory edema likely leads to lower oxygen tension within the sinus, favoring the growth of microaerophilic and anaerobic bacteria. Empiric antimicrobial coverage started upon the diagnosis of Pott's puffy tumor must therefore include these organisms.

Conclusion

Pott's puffy tumor, described nearly 250 years ago, remains a rare complication of frontal sinusitis. Defined as a subpericranial abscess with surrounding edema, this entity is commonly accompanied by intracranial infectious complications. While rare in the post-antibiotic era, it may nevertheless develop despite previous antibiotics. Its associated intracranial complications and frontal bone infection and necrosis mandate quick diagnosis and treatment. Despite the presence of such complications, patients treated with drainage of abscesses, debridement of bone sequestra, and long-term intravenous antibiotics will most likely experience a favorable outcome.

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