Stilianos E. Kountakis Brent A. Senior Wolfgang Draf *Editors* 

# The Frontal Sinus

# **Second Edition**





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Stilianos E. Kountakis • Brent A. Senior Wolfgang Draf Editors

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Second Edition



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Videos to this book can also be accessed at http://www.springerimages.com/videos/ 978-3-662-48521-7

ISBN 978-3-662-48521-7 ISBN 978-3-662-48523-1 (eBook) DOI 10.1007/978-3-662-48523-1

Library of Congress Control Number: 2016941183

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Printed on acid-free paper

This Springer imprint is published by Springer Nature The registered company is Springer-Verlag GmbH Berlin Heidelberg Dedicated to the memory of my mother Eftihia who with her loving devotion inspired my pursuit of a medical career and to my father Emmanuel whose hard work since his teenage years allowed us to pursue our dreams.

To my loving wife Eleni and our children Eftihia, Emmanuel, Nikoleta and especially Alexandra who has taught me about the real meaning of courage, patience and fighting spirit. I pray that God gives them grace that they may be temperate, industrious, diligent, devout and charitable.

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

# In Memoriam

# Wolfgang Draf, MD, Hon MD, PhD, FRCS (Ed) 1941–2011



Dear Readers,

It is through this book that we are celebrating the memory of our colleague, Professor Dr. med. Wolfgang Draf, MD, Hon MD, PhD, FRCS (Ed) and acknowledge his contributions to the field of otolaryngology-head and neck surgery. Professor Draf was one of the editors of the first edition of *The Frontal Sinus*, and he was instrumental in its design, editing and final delivery.

Professor Draf completed his training at the Universities of Würzburg and Mainz and was Chairman of the Department of Otolaryngology-Head and Neck Surgery at the Hospital for ENT Diseases, Head, Neck and Facial Plastic Surgery, Fulda, Germany, from 1979 to 2005. After his retirement in 2005, he continued to practice medicine at the International Neuroscience Institute of the University of Magdeburg until 2011.

Professor Draf was a very prolific academician publishing more than 215 refereed manuscripts while also participating in the editing/publication of 17 textbooks. He lectured extensively all over the world and served as president of several German and European ENT societies, including the German Society of Otorhinolaryngology-Head and Neck Surgery from 1995 to 1996. Wolfgang was an exemplary teacher, directing the famous Sinus Course in Fulda, Germany, for over 20 years that helped train more than 2000 participants in endoscopic, microscopic and open sinus surgery techniques. Perhaps his most famous contribution to rhinology, however, was his eponymous classification of different transnasal approaches to the frontal sinus, a system that is now used worldwide.

Professor Draf was a patient advocate with a very welcoming personality to all who approached. He was a constant figure in international congresses with his familiar infectious smile and positive demeanor. One of these editors will remember the way he befriended his teenage son, introducing him to the joys the snorkeling in a quiet bay in the Philippines. While the other will always remember his warm greeting at meetings: "Stilianos, my young and energetic friend! How are you?" With such simple admonition and encouragement, jetlag would melt away, and the business of running around in the conference checking the latest technologies or planning the first edition of *The Frontal Sinus* would return! He was a motivator and an effective mentor, a fatherly international leader who always evoked the best out of anyone who approached him.

In remembrance, we chose to preserve Chap. 24 of the first edition of *The Frontal Sinus* titled "Endonasal Frontal Sinus Drainage Type I-III According to Draf" in the same format. It appears as Chap. 25 in this edition of the book.

We thank Wolfgang for his contributions to our specialty and we will always remember him.

May his memory be eternal. Stil Kountakis, MD, PhD Brent A. Senior, MD

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## **Chapter 1 The Evolution of Frontal Sinus Surgery from Antiquity to the 21<sup>st</sup> Century**

Adil A. Fatakia, Alla Y. Solyar, and Donald C. Lanza

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#### **Core Messages**

- Over the past 140 years, a rapid progression in the advancements of visualization and instrumentation has allowed for an evolution from open to endonasal techniques for the treatment of frontal sinus pathology.
- Currently, endoscopic endonasal procedures have supplanted many open approaches given the low morbidity and comparable outcomes, but some advanced cases may require a combination of open and endonasal techniques as well as solely open approaches.
- One lesson history has taught us is that re-establishing the natural drainage pathway of the frontal sinus into the ethmoid is a critical step in the management of most medically recalcitrant frontal sinus inflammatory disease

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#### Introduction

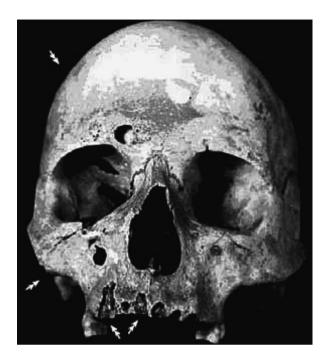
Surgery of the frontal bone has existed for many millennia [1-5] yet surgery of the frontal sinus was not described until 1750 CE [6, 7] long after the frontal sinus was first anatomically depicted (circa 1489) [8]. As with civilization, modern day frontal sinus surgery evolved rapidly over the last 140 years [9, 10]. Many events, inventions and individuals have shaped our current techniques. To this point, in 2013 there were over 2600 articles identified through PubMed search at the National Library of Medicine for the expression: "frontal sinus" surgery. Technologies that have made "state-of-the-art" frontal sinus surgery possible arose from improvements in: understanding of sinus physiology (1660), inhalant anesthesia (1849), artificial illumination (1879), x-ray imaging (1895), operating microscope (1921), antimicrobials (1940), instrument miniaturization (e.g. endoscopes 1950s) and the development of high speed endonasal drills. External approaches to the frontal sinus through trephinations and facial incisions dominated surgery from the eighteenth through the twentieth century and these still have a role today [9-11]. Although the importance of restoring the natural drainage into the ethmoid sinuses was acknowledged early in the evolution of frontal sinus surgery, technical challenges resulted in a substantial failure rate for this goal [9–11]. Since 1985, endoscopic endonasal approaches have gained popularity because of their relatively high success rate in restoring normal frontal sinus ventilation, lack of facial incisions, lower morbidity, improved monitoring of residual disease and faster patient recovery [12, 13]. However, occasionally both endonasal and external techniques are used in conjunction to help patients with the most challenging of frontal sinus disease [14-17].

#### Antiquity – 1760 CE

Paleontologists and archeologists have demonstrated that otolaryngology, as well as neurosurgery, have their roots in what is believed amongst the earliest surgical procedure known to man called – trepanation or trephination [1-5, 18]. Derived from the Greek *trypanon*, which means to bore, trepanation is the removal of bone from the skull – which in antiquity was performed to relieve evil spirits [3]. Prehistoric cave paintings from 25,000 years ago depict skull trepanations performed with archaic stone tools [1-5]. Trepanations through the ages alleviated "demons" that may have manifested themselves as head pressure/pains, seizures, and mental illness. Albeit less common than trepanations of the parietal bone, the procedure was also performed in the occipital and frontal bones [1, 2]. Opium, cocaine (Peru), and alcohol are among the earlier anesthetics available to aid in performing this procedure.

Examples of trepanation not only span time through to the present day but also span the globe [4, 5]. Anthropological evidence demonstrates disease and treatments specific to the frontal bone/sinus have existed for at least 5.5 millennia (Fig. 1.1) [2]. A "Bronze Age" man (circa 3500 BCE) had evidence of three trepanations of the frontal bone, but succumbed to persistent frontal sinus infection that had spread intracranially

**Fig. 1.1** Bronze age skull circa 3500 BCE with subacute osteomyelitis of the right maxillary and frontal sinus. Materials from excavation of burial ground Lchashen, (burial 52, Q 30–35 years old). Consistent with the later description of "Pott's puffy tumor" in 1760 CE [18]



[2]. Circa 400 BCE, Hippocrates, referred to as one of the "Father(s) of Rhinology" for his work with nasal polyps, also gave a technical description of trepanations [19]. Trepanations of the frontal sinus were also known to be applied to management of frontal sinus tumors as in the circumstance a 50 year old medieval man, from the region of the Czech Republic who had trephinations to manage a frontal bone meningioma [20]. In Peru, during 1400s CE, nearly 15 % of human remains had evidence of skull trephination [4, 5]. The practice of "stone cutting" or removing a portion of the frontal bone- which at the time was thought to alleviate maladies such as headache, mental illness and seizures was depicted in 16th century Renaissance painting [17]. The "stonecutters" surprisingly were not educated physicians, but rather apprenticed "barber-surgeons". One such prominent barber-surgeon was Ambroise Paré from the sixteenth century [21].

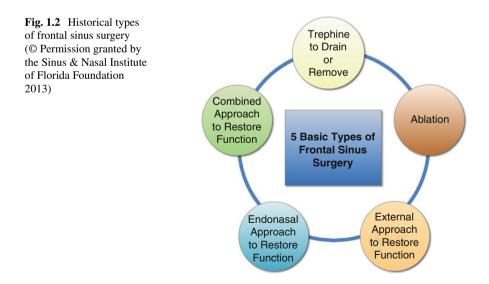
Procedures involving the frontal sinuses per se were not formally described until long after they were first anatomically illustrated by Leonardo da Vinci in 1489 CE [8]. In 1543 CE, Andreas Vesalius, a Flemish anatomist working in Padua, Italy, also considered the founder of modern human anatomy wrote the first detailed description of the pneumatized frontal, maxillary and sphenoid sinuses. In 1660 CE, Victor Schneider, a German anatomist and another perceived "Father of Rhinology", recognized for the first time that the lining of the nose and sinuses produced its own mucus [22]. This was the first time that nasal discharge was acknowledged not to arise from the cranial cavity and thus the mucosa became known as the "Schneiderian membrane". In 1760, Sir Percivall Pott described a case of forehead swelling characterized by a sub-periosteal abscess associated with osteomyelitis of the frontal bone [23].

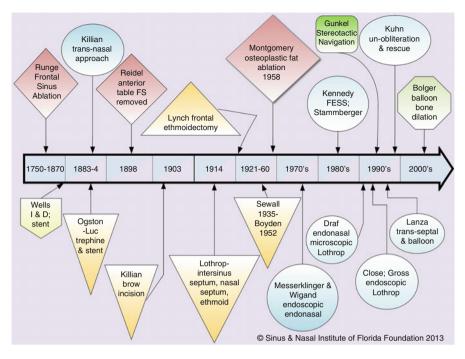
#### Frontal Sinus Surgery 1750: Present

Over the last 140 years many procedures have been described to manage the unique challenges associated with an individualized patient approach and available technologies. The historical description that follows is divided into the varied surgical approaches that have shaped our current day frontal sinus surgery. These are: trephination, ablation, external approaches to restore function, endonasal approaches, endonasal balloon dilation, and sometimes combinations of external and endonasal approaches are applied to this day (See Figs. 1.2 and 1.3).

#### Trephination and Drainage

As described earlier, trephination has been performed for many millennia. However, the first medical journal report of frontal sinus surgery appeared in the 1870 Lancet and described the work of Dr. Seolberg Wells in a man with a mucopyocele [24]. Dr. Wells created a forehead incision over a pointing brow infection and introduced a tube from the nasal passage into the frontal sinus and out the incision. The tube was removed 3 months later and the patient was restored to previous health. In 1884, Alexander Ogston evacuated the frontal sinus through a trephination the size of a "six-penny piece" [25]. The communication between the frontal and ethmoid sinuses was dilated, and mucosa was curetted, and a drainage tube was placed into the nose. Luc described a similar procedure 2 years later in the procedure became known as Ogston-Luc technique [9]. There was a





**Fig. 1.3** History of frontal sinus timeline. *Red diamonds* = ablative procedures. *Bone colored pentagon* = trephination procedures. *Blue ovals* = endonasal approaches. *Yellow triangles* = external approaches with intention of restoring drainage. *Grey-green octagon* = balloon dilation without tissue removal. *Green rectangle* = technology introduction (© Permission granted by the Sinus & Nasal Institute of Florida Foundation 2013) (Color figure online)

high failure rate caused by frontal recess stenosis leading to abandonment of this procedure.

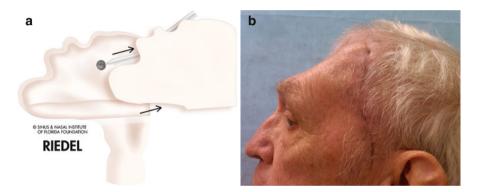
• Trephination of the frontal sinus is still a valued approach in the twenty-first century for acute management of frontal sinus infection and for the introduction of telescopes as in the above and below approach [26].

#### Ablation With and Without Reconstruction

Ablation of the frontal sinus, whereby the mucosa is completely removed from within, is described from both an anterior and posterior table approach. The posterior table approach is typically performed during craniotomy for management of infection or malignancy. Although, Runge is said to have performed the first anterior frontal ablation in 1750 [6], Hermann Kuhnt, a German ophthalmologist, was first to report a case series of frontal sinus obliterations in 1895 [27]. The technique described complete removal of the anterior wall of the frontal sinus, curettage of

frontal mucosa and re-draping of forehead skin. While functionally more successful than earlier procedures to establish drainage (Ogston), the resulting cosmetic deformity was extreme. In 1898, Riedel promoted not only removal of the anterior table of the frontal sinus but also removal of the inferior walls [9] (Fig. 1.4a, b). The procedure allowed additional infected bone to be removed, but resulted in severe cosmetic deformity. In 1903, in an attempt to improve cosmesis, Gustav Killian emphasized preserving the supraorbital ridge. In 1910, Marx had transplanted abdominal fat and a secondary procedure for reconstruction of the deformity [27]. Eventually these anterior ablative procedures were for the most part abandoned after numerous reports of morbidity, including late restenosis, supraorbital rim necrosis, mucocele formation and postoperative meningitis [6].

In an effort to minimize deformity, Hajek in 1903 proposed utilizing an osteoplastic flap whereby a hinged flap of anterior table frontal sinus bone was elevated with is periosteal blood supply attached [9, 11] (Fig. 1.5). The hinged flap allowed infection to be cleared, mucosa to be removed on all surfaces of the sinus, and the



**Fig. 1.4** (a) Schematic depiction of the "Reidel procedure" whereby the anterior table of the frontal is removed to gain access to ablate the frontal sinus (© Sinus & Nasal Institute of Florida Foundation 2013). (b) Later view of the deformity created by Reidel procedure employed in combination with neurosurgery for Postoperative infection in previously radiated patient with adenocarcinoma (© Permission granted by the Sinus & Nasal Institute of Florida Foundation 2013)

Fig. 1.5 Schematic depiction of the hinged osteoplastic flap with the sinus mucosa ablated from the lumen (© Permission granted by the Sinus & Nasal Institute of Florida Foundation 2013)



bone flap to be re-approximated. William Montgomery popularized the technique utilizing autologous fat grafts to obliterate the sinus cavity in 1958 [28, 29].

• When compared to fronto-ethmoidectomy procedures such as the Lynch procedure (see below), osteoplastic flaps with obliteration resulted in a decreased number of failures requiring re-operation.

On the other hand, complications such as CSF leak and forehead paresthesia were seen more commonly. Additionally, delayed failures at 8–20 years with mucocele formation are not uncommon even today in the most experienced hands [30, 31]. Although the osteoplastic flap had gained popularity for its aesthetic improvements in ablative surgery of the frontal sinus, it has also been used without ablation as a surgical approach for endoscopically inaccessible disease [12].

#### External Fronto-Ethmoidectomy to Restore Drainage

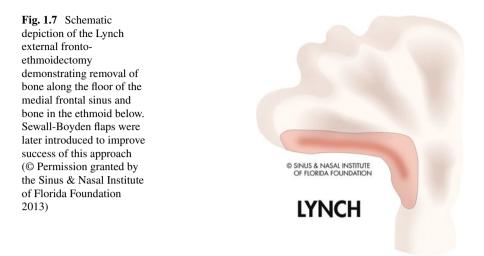
In 1908, Dr. Knapp described performing extensive external ethmoidectomy through a medial orbital incision while enlarging the nasal frontal recess [32]. In 1914, through a combination of intranasal and external approaches Lothrop described an aggressive resection of bilateral ethmoid cavities, frontal floors, superior nasal septum and the intersinus septum [33]. The goal was to create the largest frontal outflow tract possible, theorizing that this would prevent stenosis and re-accumulation of disease (Fig. 1.6).

• Given the cumbersome and technically challenging surgery, Lothrop's procedure did not gain widespread acceptance until it was reintroduced by Wolfgang Draf in 1990 (see below).

In 1921, Lynch introduced a medial periorbital incision. Excision of the ethmoid complex, lamina papyracea and frontal process of the maxilla was attained through this relatively well-hidden incision and a portion of the floor of the frontal sinus was removed as well (Fig. 1.7). Stents were placed for up to 10 days to encourage frontal

Fig. 1.6 Schematic depiction of the "Lothrop procedure" indicating the removal of the frontal sinus intersinus septum, the nasal septum and creating one common opening to the paired frontal sinuses from medial orbit to orbit (© Permission granted by the Sinus & Nasal Institute of Florida Foundation 2013)





recess maturation [9, 10]. The Lynch procedure provided a relatively straightforward and cosmetically acceptable approach to frontal sinus disease and gained favor due to its initial success. It was modified by introduction of a local septal flap by Sewell in 1935 and then revived as a technique by Boyden in the late 1950s [16]. Long-term results with the Sewall-Boyden modified Lynch procedure resulted in a frontal sinus patency rates of 85 % [16]. Besides scarring, medialization of orbital contents after removal of the lamina papyracea posed a particular concern associated with frontal recess stenosis [16].

#### Intranasal Restoration of Drainage Pathways

In 1883, Killian attempted a trans-nasal approach for drainage of the frontal sinus through the ethmoid with removal of the uncinate process [9, 10, 27]. In 1890, Schaeffer proposed entry into the frontal sinus via a nasal puncture technique to reestablish drainage and ventilation of the frontal sinus [9]. Unfortunately, the procedure was fraught with complications. One notable case was an autopsy that revealed absent frontal sinuses and two puncture wounds in the cribriform plate [6].

• Harvard Professor Harris P. Mosher proclaimed in the early half of the twentieth century that the trans-nasal approach to the ethmoid sinus was the easiest way to kill a patient [35].

The current day rigid, optical nasal endoscope was first developed in England by Professor H.H. Hopkins in the 1950s. The endoscopic techniques for sinus surgery arose out of Germany and Austria, with the work of Profs. Malte Wigand (DK) and Walter Messerklinger (AU) in the 1970s and 1980s [36]. In 1985, Prof. David Kennedy began advancing endonasal endoscopic sinus surgery with the Austrian technique, which he termed "functional endoscopic sinus surgery" [37]. Both Stammberger and Kennedy separately developed equipment and techniques, which helped to popularize endoscopic sinus surgery internationally [38, 39].

Endoscopic anatomical landmarks to the frontal ostia were described by Wigand, which included the anterior ethmoidal artery, medial lamella of the middle turbinate and the orbital wall. Wigand also described an endoscopic two portal technique useful in particularly difficult or recalcitrant cases. As described, a small trephination in the anterior wall of the frontal sinus allowed an endoscope or instruments to manipulate and visualize tissue within the sinus ostium from above or below [40, 41].

Along with Heinz Stammberger, Frederick Kuhn was instrumental in advancing knowledge of frontal anatomy and miniaturizing instrumentation to gain access to the frontal sinus endoscopically [42]. Kuhn developed specialized techniques to access the frontal sinus which enabled the evolution towards the endoscopic Lothrop procedure. Additionally, he described the "frontal sinus rescue procedure" [30] to manage frontal recess stenosis with a mucoperiosteal flap advancement and the "unobliteration procedure" [43]. In 2009, Kuhn reported on the patency rates of 294 frontal sinuses after primary endoscopic sinus surgery for chronic rhinosinusitis over a 45 month follow up period and showed 88 % were patent after a mean follow up of 45 months [13].

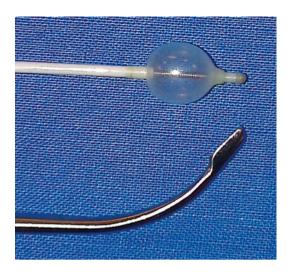
Throughout the 1990s Draf published multiple articles describing his unique microscopic technique and results on frontal sinus surgery. Draf developed a series of graded procedures providing sequentially larger frontal sinus access.

#### **Draf Procedures**

- Draf I was characterized as an anterior ethmoidectomy and opening of the frontal recess.
- Draf IIa called for removal of the frontal sinus floor lateral to the middle turbinate.
- Draf IIb was categorized by the removal of the frontal sinus floor from lamina papyrecea to the nasal septum unilaterally.
- Draf III also known as the "microscopically performed modified Lothrop" resulted in removal of bilateral frontal sinus floors, the superior aspect of the nasal septum and the inferior aspect of the intersinus septum, creating a common C- shaped cavity [24, 37].

Adding to this body of work, Close et al. reported on the first endoscopic Lothrop in 1993. In their small series of eight patients, there was one cerebrospinal fluid leak reported [44]. Gross et al. in 1995, reported an experience with ten patients using endonasal drills without any complications [45]. Around this time, image guided surgery and high speed curved drills became commercially available [45, 46]. The eventually widespread use of the technology would advance endoscopic frontal sinus surgery and popularize endoscopic Lothrop surgery as a viable alternative to ablative surgery. In 1997, Lanza et al. described an alternative technique to access





the frontal sinus termed the trans-septal frontal sinusotomy (TSFS) to approach the fontal sinus that was inaccessible through the frontal recess [47]. In this approach the floor of the frontal sinus is identified using intraoperative landmarks as well as computer-aided or image-guided surgery at the midline. Once the floor is entered with a drill and angled instrumentation, the dissection is carried anteriorly and then posterio-laterally to include the natural ostia in a safe direction away from the cribriform plate and skull base. In 2013, Wormald et al. reported a 95 % success rate with 45 month follow-up in 229 patients who had undergone an endoscopic modified Lothrop [48].

In 1993, Lanza first presented endonasal balloon dilation of the postoperative frontal recess with a five French Fogarty Biliary Balloon Probe as an alternative to rigid instrumentation to gently reduce frontal recess mucosal swelling [49] (Fig. 1.8). In 2005, Bolger et al. introduced a new balloon technology that provided enough force to displace bone in the frontal recess, allowing dilation of the fontal sinus without tissue removal and thus minimizing the disruption of the natural anatomy [50, 51].

#### **Summary**

Although, the advanced technology and instrumentation has facilitated this progression, the surgery of the frontal sinus remains the most difficult to master.

• Frontal sinus surgery has evolved from radical open and ablative procedures to minimally invasive endoscopic procedures that include balloon dilation.

One lesson history has provided is that re-establishing the natural drainage pathway of the frontal sinus into the ethmoid is a critical step in the management of most

 Table 1.1 Five premises of frontal sinus surgery [52]

1. Restoring frontal	sinus function is preferred to ablation/obliteration
2. Minimally invasi	ve techniques are typically associated with shorter recovery periods
3. Ability to post-op	peratively monitor residual/recurrent disease is best when function is restored
4. Post-operative, w	yound care is more labor intensive when function is restored than after

5. Complication rates with ablation procedures

medically recalcitrant frontal sinus inflammatory disease [34]. It is worth noting, that despite patency rates of 88 % for primary endonasal surgery [13], some age old techniques still find utility in the twenty-first century.

The newest techniques, balloon dilation and the modified Lothrop, are opportunities to depart further from the use of antiquated cures. As reported in persistent frontal recess stenosis after prior endoscopic surgery, balloon dilation and endoscopic modified Lothrop procedures had an 86 % and 95 % patency rates, respectively [48]. This suggests that the older external procedures will become even less common in the future allowing an opportunity for additional improvements in the surgical management of frontal sinus pathology.

In general, five premises are asserted in establishing the best paradigm for the surgical management of frontal sinus disease (Table 1.1). They are: (1) Restoring frontal sinus function is preferred to ablation/obliteration; (2) Minimally invasive techniques are typically associated with shorter recovery periods; (3) Ability to post-operatively monitor residual or recurrent disease is greatest (endoscopically or via imaging) when frontal sinus function is restored; (4) The need for post-operative, endoscopic, wound care can be more labor intensive when function is restored; and (5) Even in experienced hands, complication rates with ablation procedures are higher than those associated with minimally invasive techniques [52].

Acknowledgements We would like to thank the Sinus & Nasal Institute of Florida Foundation for its financial support in producing illustrations for this chapter. We thank Rick Mugavero for his help with Figs. 1.4a, 1.5, 1.6, and 1.7. We thank the Editors of Dental Archeology, The Prado Museum, and Royal Collection of Queen Elizabeth the II for their copyright permission to reproduce work in their possession.

#### References

- 1. Mogliazza S. An example of cranial trepanation dating to the Middle Bronze Age from Ebla, Syria. J Anthropol Sci. 2009;87:187–92.
- Zias J, Pomeranz S. Serial craniectomies for intracranial infection 5.5 millennia ago. Int J Osteoarchaeol. 1992;2(2):183–6.
- 3. Gross CG. A hole in the head. Neuroscientist. 1999;5(4):63-9.
- Clower WT, Finger S. Discovering trepanation: the contribution of Paul Broca. Neurosurgery. 2001;49(6):1417–25; discussion 1425–6.
- 5. Burton FA. Prehistoric trephining of the frontal sinus. Calif State J Med. 1920;18(9):321-4.
- Donald PJ. Surgical management of frontal sinus infections. In: Donald PJ, Gluckman JL, Rice DH, editors. The sinuses. New York: Raven Press; 1995. p. 201–32.

- 7. Runge, Ludolph Heinrich Title(s):Diss. med.-chir. ... Sur les principales maladies des sinus frontaux & maxillaires. In: Haller, Coll. de theses i; 1757 p. 127–48.
- 8. Pevsner J. Leonardo da Vinci's contributions to neuroscience. Trends Neurosci. 2002;25(4):217–20.
- 9. McLaughlin Jr RB. History of surgical approaches to the frontal sinus. Otolaryngol Clin North Am. 2001;34(1):49–58. Review.
- 10. Jacobs JB. 100 years of frontal sinus surgery. Laryngoscope. 1997;1077:1-36.
- Hajek M. Pathology and treatment of the inflammatory diseases of the nasal accessory sinuses. St Louis: CV Mosby; 1903.
- 12. Orlandi RR, Kennedy DW. Revision endoscopic frontal sinus surgery. Otolaryngol Clin North Am. 2001;34(1):77–90. Review.
- 13. Chan Y, Melroy CT, Kuhn CA, Kuhn FL, Daniel WT, Kuhn FA. Long-term frontal sinus patency after endoscopic frontal sinusotomy. Laryngoscope. 2009;119(6):1229–32.
- 14. Isa AY, Mennie J, McGarry GW. The frontal osteoplastic flap: does it still have a place in rhinological surgery? J Laryngol Otol. 2011;125(2):162–8.
- 15. Raghavan U, Jones NS. The place of Riedel's procedure in contemporary sinus surgery. J Laryngol Otol. 2004;118(9):700–5.
- Murr AH, Dedo HH. Frontoethmoidectomy with Sewall-Boyden reconstruction: indications, technique, and philosophy. Otolaryngol Clin North Am. 2001;34(1):153–65. Review.
- Hwang PH, Han JK, Bilstrom EJ, Kingdom TT, Fong KJ. Surgical revision of the failed obliterated frontal sinus. Am J Rhinol. 2005;19(5):425–9.
- 18. Yu A. Khudaverdyan the anthropology of infectious diseases of Bronze Age and Early Iron Age from Armenia. Dent Anthropol. 2011;24(2–3):42–54.
- Lathi A, Syed MMA, Kalakoti P, Qutb D, Kishve. Clinico-pathological profile of sinonasal masses: a study from a tertiary care hospital of India. Acta Otorhinolaryngol Ital. 2011;31(6):372–7.
- Smrcka V, Kuzelka V, Melkova J. Meningioma probable reason for trepanation. Int J Osteoarchaeol. 2003;13:325–30.
- 21. St. John V. Ambroise Paré, the Barber-Surgeon. Can Med Assoc J. 1955;72(8):612-5.
- 22. Ersner MS. Hay-fever. Laryngoscope. 1921;32:856.
- 23. Flamm ES. Percivall Pott: an 18th century neurosurgeon. J Neurosurg. 1992;76(2):319-26.
- 24. Wells S. Abscess of the frontal sinus; operation; cure. Lancet. 1870;I:694-5.
- 25. Ogston A. Trephinating the frontal sinus for catarrhal diseases. The Medical Chronicle No. 3. 1884;3:235–8.
- Batra PS, Citardi MJ, Lanza DC. Combined endoscopic trephination and endoscopic frontal sinusotomy for management of complex frontal sinus pathology. Am J Rhinol. 2005;19(5):435–41.
- 27. Draf W, Weber R, Keerl R, et al. Chapter 20. Endonasal & external micro- endoscopic surgery of the frontal sinus. In: Stamm AC, Wolfgang Draf, editors. Micro-endoscopic surgery of the paranasal sinuses and the skull base. Springer, Great Britain; 2000. p. 257.
- Goodale RL, Montgomery WW. Experiences with the osteoplastic anterior wall approach to the frontal sinus; case histories and recommendations. AMA Arch Otolaryngol. 1958;68(3):271–83.
- Montgomery WW. State-of-the-art for osteoplastic frontal sinus operation. Otolaryngol Clin North Am. 2001;34(1):167–77. Review.
- Javer AR, Sillers MJ, Kuhn FA. The frontal sinus unobliteration procedure. Otolaryngol Clin North Am. 2001;34(1):193–210. Review.
- Schenck NL. Frontal sinus disease. III. Experimental and clinical factors in failure of the frontal osteoplastic operation. Laryngoscope. 1975;85(1):76–92.
- Knapp A. The surgical treatment of orbital complications in disease of the nasal accessory sinuses. JAMA. 1908;LI(4):299–301.
- Lothrop HA. XIV. Frontal sinus suppuration: the establishment of permanent nasal drainage; the closure of external fistulae; epidermization of sinus. Ann Surg. 1914;59(6):937–57. Ann Otol Rhinol Laryngol. 1994 Dec;103(12):952–8.

- 34. Draf W, Weber R, Keerl R, Constantinidis J. Current aspects of frontal sinus surgery. I: endonasal frontal sinus drainage in inflammatory diseases of the paranasal sinuses. HNO. 1995;43(6):352–7.
- 35. Lawson W. The intranasal ethmoidectomy: evolution and an assessment of the procedure. Laryngoscope. 1994;104(6 Pt 2):1–49. Review.
- 36. Vining EM, Kennedy DW. The transmigration of endoscopic sinus surgery from Europe to the United States. Ear Nose Throat J. 1994;73(7):456–8. 460.
- Kennedy DW, Zinreich SJ, Rosenbaum AE, Johns ME. Functional endoscopic sinus surgery. Theory and diagnostic evaluation. Arch Otolaryngol. 1985;111(9):576–82.
- 38. Stammberger H, Posawetz W. Functional endoscopic sinus surgery. Concept, indications and results of the Messerklinger technique. Eur Arch Otorhinolaryngol. 1990;247:63–76.
- Stammberger H. FESS- "Uncapping the Egg" -the endoscopic approach to frontal recess and sinuses. A surgical technique of the Graz University Medical School. Tuttlingen: Endo-Press; 2000.
- 40. Wigand ME. Transnasal ethmoidectomy under endoscopical control. Rhinology. 1981;19(1):7–15.
- Hoseman W, Wigand ME. Indications, technique and results of endonasal endoscopic ethmoidectomy. Acta Otorhinolaryngol Belg. 1993;47:73–83.
- 42. Kuhn FA. Chronic frontal sinusitis: the endoscopic frontal recess approach. Oper Tech Otolaryngol Head Neck Surg. 1996;7:222–9.
- 43. Citardi MJ, Javer AR, Kuhn FA. Revision endoscopic frontal sinusotomy with mucoperiosteal flap advancement: the frontal sinus rescue procedure. Otolaryngol Clin North Am. 2001;34(1):123–32. Review.
- Close LG, Lee NK, Leach JL, Manning SC. Endoscopic resection of the intranasal frontal sinus floor. Ann Otol Rhinol Laryngol. 1994;103(12):952–8.
- 45. Gross WE, Gross CW, Becker D, Moore D, Phillips D. Modified transnasal endoscopic Lothrop procedure as an alternative to frontal sinus obliteration. Otolaryngol Head Neck Surg. 1995;113(4):427–34.
- 46. Gunkel AR, Freysinger W, Thumfart WF, Pototschnig C. Complete sphenoethmoidectomy and computer-assisted surgery. Acta Otorhinolaryngol Belg. 1995;49(3):257–61.
- 47. McLaughlin RB, Hwang PH, Lanza DC. Endoscopic trans-septal frontal sinusotomy: the rationale and results of an alternative technique. Am J Rhinol. 1999;13(4):279–87.
- Naidoo Y, Bassiouni A, Keen M, Wormald PJ. Long-term outcomes for the endoscopic modified lothrop/draf III procedure: a 10-year review. Laryngoscope. 2014;124(1);43–9.
- 49. Lanza DC. Postoperative care and avoiding frontal recess stenosis. In: Abstracts of the international advanced sinus symposium, Philadelphia, Jul 1993.
- Brown CL, Bolger WE. Safety and feasibility of balloon catheter dilation of paranasal sinus ostia: a preliminary investigation. Ann Otol Rhinol Laryngol. 2006;115(4):293–9; discussion 300–1.
- Wycherly BJ, Manes RP, Mikula SK. Initial clinical experience with balloon dilation in revision frontal sinus surgery. Ann Otol Rhinol Laryngol. 2010;119(7):468–71.
- 52. Lanza DC. Frontal sinus obliteration is rarely indicated. Arch Otolaryngol Head Neck Surg. 2005;131(6):531–2.

## **Chapter 2 Surgical Anatomy and Embryology of the Frontal Sinus**

Mohammad H. Al-Bar, Seth M. Lieberman, and Roy R. Casiano

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Mohammad H. Al-Bar, Seth M. Lieberman, and Roy R. Casiano have nothing to disclose.

**Electronic supplementary material** The online version of this chapter (doi:10.1007/978-3-662-48523-1\_2) contains supplementary material, which is available to authorized users.

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_2

#### **Core Messages**

- A thorough knowledge of frontal sinus anatomy is critical when performing 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 and/or the relationship to the 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.

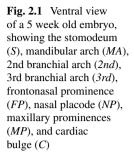
#### Introduction

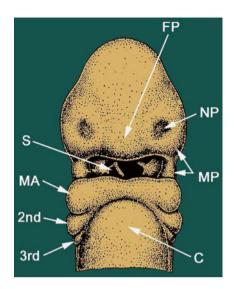
As with any surgical procedure, a thorough knowledge of anatomy is the one most important factor in minimizing complications and maximizing one's chances of a good surgical outcome. This is particularly important in performing endoscopic sinus surgery, as each paranasal sinus is in close proximity to 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 scan or MRI. However, despite these technologies 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. The frontal draining, 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**

All of the development of the head and neck, along with the face, nose, and paranasal sinuses, take 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 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. At this point the embryo has its first appearance of an identifiable head and face. An orifice in its middle, called the stomodeum appears (Fig. 2.1), surrounded by more than one prominence. Superiorly the stomodeum is limited by the frontonasal prominence and separated from it by the oronasal membrane which eventually becomes the hard palate by the end of the fifth week of gestation. The mandibular and maxillary arches (prominences) surround the stomodeum bilaterally, and are derivatives of the first branchial arch. The first branchial arch will ultimately give rise to all of the vascular and neural structures supplying this area [1-6].

The frontonasal prominence differentiates inferiorly with two nasal projections and one caudal mesodermic projection. The two nasal projections, or nasal placodes, later form the nasal cavity and primitive choana. The caudal mesodermic projection will form the nasal septum dividing the nasal cavity into two chambers by 5th–12th week of gestation. The primitive choana will be the point of development for the posterior pharyngeal wall as well as the different sinuses. As the embryo grows, the maxillary processes and the nasal placodes come

The three medial projections include anterior, inferior and superior projections.

- · Anterior projection will form the agger nasi.
- Inferior projection (maxillo-turbinate) will form the maxillary sinus.
- Superior projection (ethmoido-turbinate), will form the middle and superior turbinates and the small ethmoidal cells between the septum and lateral wall of the nose. The middle meatus develops between the formed inferior and middle turbinates [1, 3, 4].

together in the midline, to form the maxillary bone and the beginning of the external nose [1, 3-5].

Simultaneously, the cranial and facial bones are forming as well. The skeletal system develops from the mesoderm, 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. At birth, all cranial bones are separated by layers of connective tissue that later become fused and ossified in the post-natal 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 [1, 4].

Around the 25th–28th week of development, three medially directed projections arise from the lateral wall of the nose. Between these three medial projections, small lateral diverticula will invaginate into the lateral wall of the primitive choana to eventually form the nasal meati (Fig. 2.2).

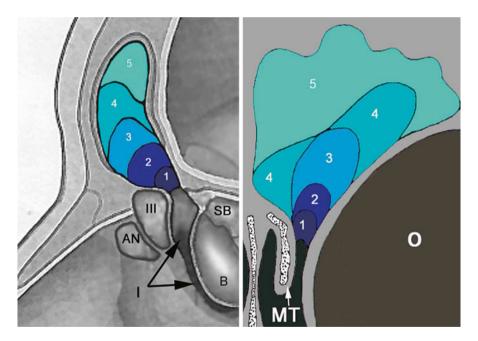
The middle meatus invaginates laterally giving shape to the embryonic infundibulum, along with the uncinate process. During the 13th week of development the infundibulum continues expanding superiorly, giving rise to the frontonasal recess as a primitive frontal sinus. It has been proposed that the frontal sinus might develop during the sixteenth 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.

Fig. 2.2 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



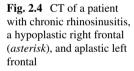
Frontal sinus development is variable as noted in cadaveric and radiological studies only identifiable in less than 1.5 % of infants less than 1 year of age [1, 2, 7, 8]. During this period, the frontal sinus remains as a potential pocket and has been referred to as a "cellulae ethmoidalis", since the findings point clearly to its close embryological and anatomical relationship with anterior ethmoid air cells.

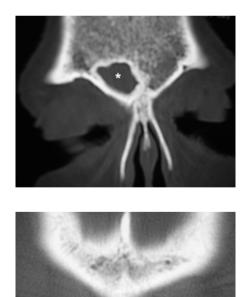
Primary pneumatization of the frontal bone occurs as a slow process up to the end of the first year of life. At this point, the frontal sinus remains as a small, smooth, blind pocket, for approximately the first 2 years of life, until the process of secondary pneumatization begins. From 2 years of age until adolescence, the frontal sinus progressively grows and fully pneumatizes (Fig. 2.3). Between 1 and 4 years of age, the frontal sinus begins 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 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 [1, 3, 5, 9–12].



**Fig. 2.3** Sagittal and coronal views of the frontal sinus noting it's progressive secondary pneumatization between the ages of 3 and 18 years of age. Between 1 and 4 years of age (I), 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 agar nasi air cell (AN), type III frontal infundibular cell (III), ethmoid bulla (B), suprabullar cell (SB), middle turbinate (MT), and orbit (O) are marked

The frontal sinuses develop within the frontal bones. Each bone remains separated by a vertical (sagittal) suture line that becomes ossified and eventually forms the frontal intersinus septum. Factors have not been elucidated in the formation of the frontal sinuses. Some authors have speculated that the adolescent growth phase may be stimulated by the process of mastication, different hormonal changes or even by climate and race. 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, divided by various septae. Occasionally, frontal sinuses may develop asymmetrically, or even fail to develop at all. Frontal sinuses may be more "dominant" on one side, while hypoplastic, or even aplastic, on the other side (Figs. 2.4 and 2.5). Aplasia of both frontal sinuses has been reported in 3-5 % of patients. 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 temporal bone. Race, geography, and climate, are just a few factors that have been implicated in the abnormal development of the frontal sinus. 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 [10-17].





**Fig. 2.5** CT of bilaterally aplastic frontal sinuses

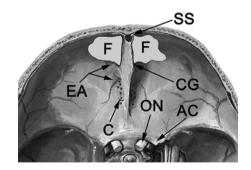
#### Surgical Anatomy of the Frontal Sinuses

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 [1, 3, 9].

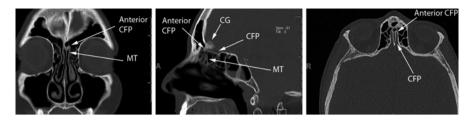
In an adult, the two frontal sinuses take on the shape of a pyramid. Anteriorly, the frontal bone is noted to be twice as thick as the posterior table [18-20].

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 [1, 3]. The anterior table is made up of thick cortical bone and averages about 4–12 mm in thickness. Pericranium is adherent to the bone, followed more superficially by the frontalis muscle, subcutaneous fat, and skin. The vascularized pericranium is frequently used for reconstruction of large anterior skull base defects or for frontal sinus obliteration [21, 22].

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 [1, 9, 10, 21–23]. It has a superior vertical, and a smaller inferior horizontal, portion. The horizontal portion forms part of the orbital roof. The posterior walls on each side join inferiorly to form the internal frontal crest, to which the falx cerebri inserts (Fig. 2.6). 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 from chronic inflammatory conditions [14]. The absence of bony walls cannot be address through a physical or endoscopic exam. However, with today's imaging studies this type of abnormality should be easily detected preoperatively.



**Fig. 2.6** 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 sagittal 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*)



**Fig. 2.7** CT scan at the same plane level show the relation of the cribriform plate (*CFP*) to the posterior wall of the frontal sinus and the vertical attachment of the middle turbinate (*MT*). *CG* Crista galli

During extended frontal procedures (Draf IIb and III), care should be taken to save the anterior cribriform plate fibrils posteriomedially and the orbit laterally. The anterior border of the cribriform plate can be identified at the level of the posterior wall of the frontal infundibulum (Figs. 2.7 and 2.16) [24]. The falx cerebri inserts into the posterior table of the frontal sinus, at a point corresponding to the posterior edge of the intersinus septum. The intersinus septum, thought to be a continuation of the fused ossified embryologic suture line, separates the frontal sinuses into distinct draining sinus cavities. Although the intersinus septum may vary in direction and thickness, the base of the intersinus septum approximates midline at the level of the infundibulum as it is continuous with the crista galli posteriorly, the perpendicular plate of the ethmoid inferiorly, and the nasal spine of the frontal bone anteriorly (Fig. 2.8). Pneumatization of the intersinus cells may occasionally extend into the crista galli [1, 8]. 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 forms the roof of the orbit through which the superior oblique muscle inserts and the supraorbital neurovascular pedicle courses towards the forehead skin via the supraorbital foramen. 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 of the frontal sinus extends itself as far as the angular prominence of the frontal bone. Supraorbital pneumatization may extend as far as the lesser wing of the sphenoid. The superior border of the frontal sinus is the nonpneumatized cancellous bone of the frontal bone.

One of the many interesting parts of the frontal sinus anatomy is the relationship of the frontal sinus outflow tract to the surrounding structures and the variety of pneumatization patterns in that area. 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 [2, 7, 23]. 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. 2.9).

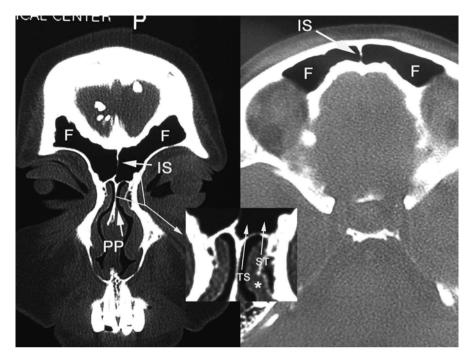
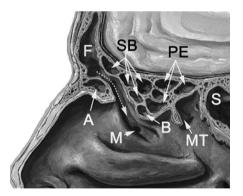


Fig. 2.8 CT of a normal well pneumatized frontal sinus in an adult. The intersinus septum (IS) of the frontal sinus (F) is continuous with the crista galli posteriorly, the perpendicular plate of the ethmoid (PP) inferiorly, and the nasal spine of the frontal bone anteriorly. In well-pneumatized frontal 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. 2.9** 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 hour-glass 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

The frontal sinus infundibulum is formed by the most inferior aspect of the frontal sinus and is affected largely by: (Fig. 2.10)

- · The size of the agger nasi
- The insertion of the uncinate process.
- And the type and site of ethmoidal frontal cells if present.
- The frontal sinus infundibulum is generally bounded by: (Fig. 2.11)
- The lamina papyracea laterally in its superior portion.
- The middle turbinate anterior vertical lamella medially.
- Anteroinferiorly by the agger nasi.
- And posteriorly by the ethmoid suprabullar air cells [1, 2, 5, 15, 18, 20, 25–33].

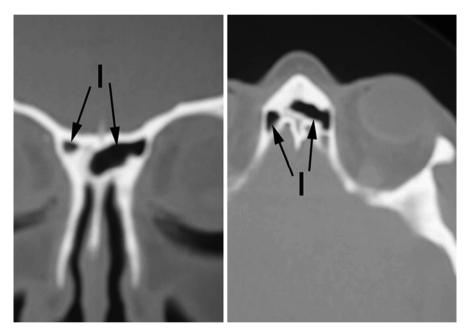
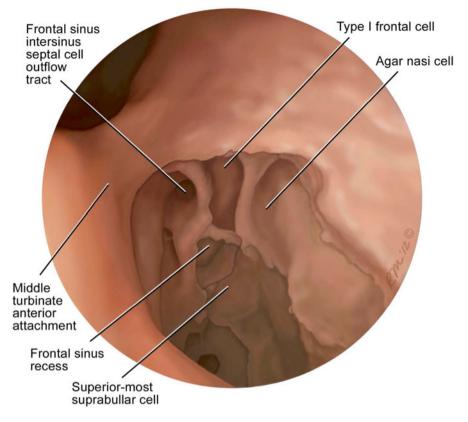


Fig. 2.10 The right frontal sinus infundibulum is narrowed and surrounded by thick bone. Unlike the left frontal infundibulum (which is very wide and accessible through a transnasal or supraturbinal approach), this right frontal infundibulum may be more prone to easy obstruction due to persistent inflammatory disease or from inadvertent surgical trauma with subsequent fibrosis or osteoneogenesis

A series of 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, and the frontal cells. It is important to know that these cells might be present in any given patient, not only



**Fig. 2.11** View of the frontal recess left side after Draf I frontal sinusotomy procedure with Type I Frontal cell, middle turbinate, suprabullar and agar nasi cells (From Casiano RR. Endoscopic Sinonasal Dissection Guide. New York: Thieme; 2012 with permission)

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 [15, 25–27, 30].

# The Uncinate Process

The uncinate process is one of the important landmarks to identify the frontal sinus. It has a crescent shape with a vertical, transitional and horizontal portion. The anterior superior end fuses with the posteromedial wall of the agger nasi cell and nasolacrimal duct. The attachment of this superior end varies thereby affecting the drainage of the frontal recess. Most commonly, the uncinate process is attached

to the lamina papyracea where the frontal sinus drains medial to the uncinate adjacent to the middle turbinate. In other cases where the uncinate is attached to the skull base or the middle turbinate, then the frontal sinus drains into the infundibulum, laterally. The uncinate process could be also pneumatized in around 2.5 % of individuals and may be the cause of significant obstruction [5, 18–20, 26, 32, 34].

# The Agger Nasi

The agger nasi is the most anterior of the ethmoid cells. It can sometimes be difficult to differentiate on coronal CT scan imaging and even during surgery. But with experience, its presence can be documented on CT scans in around 98 % of cases. It is intimately related to the uncinate process and the anterior head of the middle turbinate, along the ascending intranasal portion of the maxillofrontal suture line, and adjacent posteriorly to the lacrimal sac. As noted above, the uncinate process has an interesting relation to the agger nasi as it is attached usually to the posterior half of the agger nasi and commonly forms its posterior and medial wall [2, 15, 19, 25, 26, 30, 33].

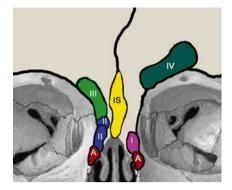
# The Frontal Cells

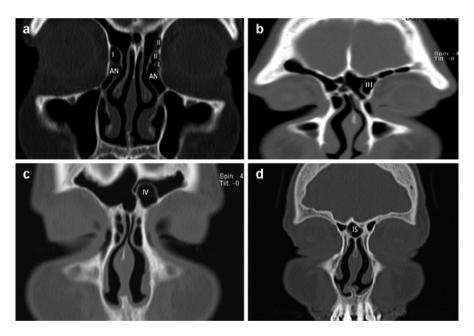
The frontal sinus can also be confused with "frontal infundibular cells", which represent a series of anterior ethmoidal cells directly superior to the agar nasi cell. The type, number and the location of these frontal cells along the anterior wall of the frontal outflow tract will affect the frontal sinus drainage and cause a shift either medially or laterally to these cells (Table 2.1). Bent and Kuhn divided frontal infundibulum cells into four categories, based on their relationship to the agger nasi cell and the orbital roof (Fig. 2.12). A type I frontal cell represents a single air cell above

Cell	Description			
Agger nasi	The most anterior ethmoid cell			
Suprabullar cells	The cell located superior to the bulla ethmoidalis and inferior to the skull ba It is pneumatized along the skull base and the posterior wall of the frontal sinus. It can extend into the supraorbital area along the roof of the orbit			
Frontal cells				
Type 1	Single air cell above the agger nasi			
Type 2	Series of cells above the agger nasi, but below the orbital roof			
Туре 3	Series of cells extend into the frontal sinus, but remain contiguous with the agger nasi cell			
Type 4	Isolated cell in the frontal cell			
Intersinus cell	Aerated intersinus septum			

Table 2.1 Frontal cells

**Fig. 2.12** Bent and Kuhn's classification of frontal infundibular air cells based on it's proximity to the agger nasi (*A*) and orbital roof. Types I (*I*), II (*II*), III (*III*), and IV (*IV*), are shown. In addition, one or more intersinus septal cell (*IS*) may also exist





**Fig. 2.13** Coronal CT scan illustrating the frontal cells types. (**a**) Shows the Agger nasi (AN) with a type I frontal cell (I) which is single and superior to agger nasi, and type II frontal cells (II) which are multiple and superior to agger nasi but below the orbital roof. (**b**) Shows a type III frontal cell (III) which is a single cell extending from agger nasi into the frontal sinus. (**c**) Shows a Type IV frontal cell (IV) which is isolated within the frontal sinus. (**d**) Intersinus frontal cell (IS) medially located

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. A completely isolated frontal cell (not contiguous with the agger nasi cell) within the frontal sinus cavity corresponds to a type IV cell (Fig. 2.13) [2, 7, 15, 25, 30, 35].

### The Suprabullar Cells

The size of the frontal recess is not only affected by the agger nasi pneumatization anteriorly but also affected posteriorly by the pneumatization of the suprabullar cells. These cells are located between the ethmoid bulla and the skull base and can communicate with the frontal recess. Supraorbital cells may also disturb the normal frontal sinus outflow tract in diseased states. On CT scans these supraorbital cells are essentially suprabullar cells with significant pneumatization over the orbital roof (Figs. 2.14 and 2.15) [26–28].

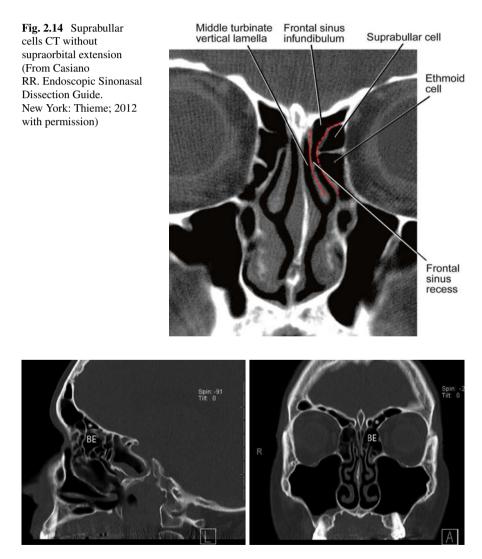
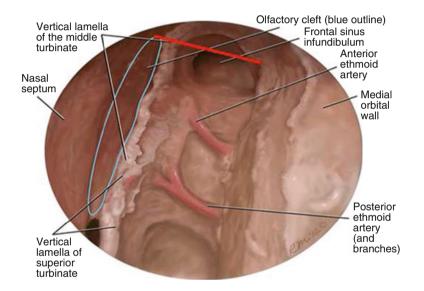


Fig. 2.15 Suprabullar cells with supraorbital extension. BE bulla ethmoidalis

The ethmoid bulla and the suprabullar cells have an important relationship to the anterior ethmoid artery, as it is commonly encountered during frontal sinus surgery. The anterior ethmoid artery arises in the orbit as a branch of the ophthalmic artery and passes through the anterior ethmoidal foramen to enter the anterior ethmoidal sinus (Figs. 2.16 and 2.17). It courses anteriorly from lateral to medial near the skull base at the junction of the ethmoid roof and the posterior border of the frontal recess. If the frontal sinus is absent, then the anterior ethmoid artery runs behind the first anterior ethmoidal cell [18, 20, 34].

The importance of landmarks in revision frontal surgery cannot be overstated. One such landmark is the natural ostium of the maxillary sinus., typically located in revision cases at the point where the orbital floor and lacrimal bone meet. This trajectory is followed superiorly in a line parallel to the convexity of the nasolacrimal



**Fig. 2.16** The anterior edge of the cribriform plate lies at the coronal plane of the posterior frontal sinus infundibulum (*red line*), adjacent to the nasal septum (From Casiano RR. Endoscopic Sinonasal Dissection Guide. New York: Thieme; 2012 with permission)

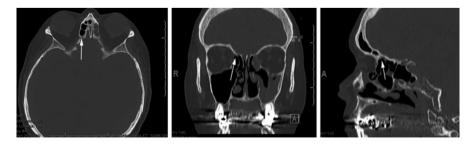
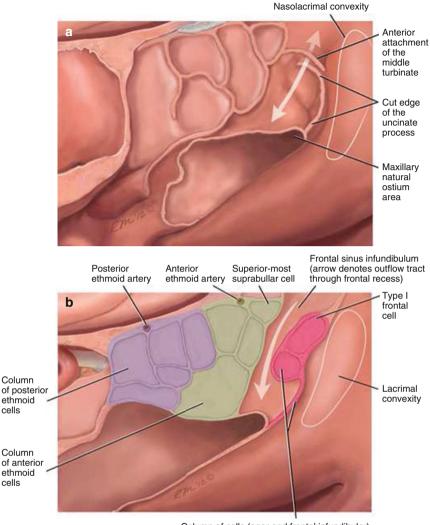


Fig. 2.17 Sinus CT showing the anterior ethmoid artery (*white arrow*) as it exit the orbit in the ethmoid sinus



Column of cells (agar and frontal infundibular) at the coronal plane of the uncinate process

**Fig. 2.18** The relation of frontal recess to the surrounding structures (From Casiano RR. Endoscopic Sinonasal Dissection Guide. New York: Thieme; 2012 with permission)

duct, leading to the frontal recess which opens a few millimeters behind the attachment of the middle turbinate (Fig. 2.18) [31, 36].

The vascular supply of the frontal sinus is derived from the 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 allows communication intracranially, intraorbitally, and with the midfacial and forehead skin. The posterior wall drains into the superior sagittal sinus, intracranially [9, 13].

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 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 [1, 15, 23]. 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. May or all of these physiological changes may culminate in chronic rhinosinusitis [15].

# Conclusions

Frontal sinus anatomy can be challenging even for the most experience surgeon. A thorough knowledge of the common 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 out-flow tract with well-established anatomical relationships to neighboring vital structures and ethmoidal air cells.

#### References

- Peynegre R, Rouvier P. Diseases of the sinuses a comprehensive textbook of diagnosis and treatment: anatomy and anatomical variations of the paranasal sinuses. Influence on sinus dysfunction. In: Gershwin M, Incaudo GA, editors. Diseases of the sinuses a comprehensive textbook of diagnosis and treatment. Totowa: Humana Press; 1996. p. 3–32.
- Kuhn FA. Surgery of the frontal sinus. In: Kennedy DW et al., editors. Diseases of the sinuses diagnosis and management. London: B.C Decker Hamilton; 2001. p. 281–301.
- Navarro JA. Cavidade do Nariz e Seios Paranasales Anatomia Cirurgica 1, All Dent Brazil; 1997. p. 3–24.

- Sadler TW. Langman's medical embryology. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2004. p. 363–401.
- 5. Chan R, Astor FC, Younis RT. Embryology and anatomy of the nose and paranasal sinuses. In: Younis RT, editor. Pediatric sinusitis and sinus surgery. New York: Taylor & Francis; 2006.
- Lang J. Clinical anatomy of the nose, nasal cavity and paranasal sinuses. New York: Thieme Medical Publishers; 1989. p. 1–3.
- 7. Friedman M, et al. Frontal sinus surgery 2004: update of clinical anatomy and surgical techniques. Oper Tech Otolaryngol Head Neck Surg. 2004;15(1):23–31.
- Hajek M. Pathology and treatment of the inflammatory diseases of the nasal accessory sinuses. 5th ed. St. Louis: CB Mosby; 1926. p. 34–52.
- 9. Schaeffer JP. The genesis, development, and adult anatomy of the nasofrontal region in man. Am J Anat. 1916;20(1):125–45.
- 10. Shah R, et al. Paranasal sinus development : a radiographic study. Laryngoscope. 2003;113(2):205-9.
- 11. Spaeth J, et al. The paranasal sinuses in CT-imaging: development from birth to age 25. Int J Pediatr Otorhinolarynol. 1997;39(1):25–40.
- 12. Wolf G, et al. Development of the paranasal sinuses in children: implications for paranasal sinus surgery. Ann Otol Rhinol Laryngol. 1993;102(9):705–11.
- 13. Aydinhoglu A, et al. Absence of frontal sinus in Turkish individuals. Yonsei Med J. 2003;44(2):215-8.
- 14. Cryer MH. Some variations in the frontal sinus. JAMA. 1907;26:284-9.
- McLaughlin RB Jr, et al. Clinically relevant frontal sinus anatomy and physiology. In: Kennedy DW, Lanza DC, editors. Current concepts in the surgical management of frontal sinus disease. Otolaryngol Clin North Am. 2001;34(1):1–21.
- 16. Tilley H. An investigation of the frontal sinus in 120 skulls from a surgical aspect, with case illustrating methods of treatment of disease in this situation. Lancet. 1896;2:866–70.
- 17. Vidic B. Extreme development of the paranasal sinuses. Ann Otol Rhinol Laryngol. 1969;78(6):1291-8.
- Walsh WE, Kern RC. Sinonasal anatomy, function, and evaluation. In: Bailey BJ, Johnson JT, Newlands SD, editors. Head & neck surgery – otolaryngology. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2006
- 19. Nafi Aygun S, Zinreich J. Radiology of the nasal cavity and paranasal sinus. In: Cummings otolaryngology head & neck surgery. 4th ed. Philadelphia: Elsevier Mosby; 2005
- 20. Sargi ZB and Casiano RR (2007). Surgical anatomy of the paranasal sinuses. In: Rhinologic and Sleep Apnea Surgical Techniques. Chapter 2, Editors: Kountakis SE, and Önerci M, Berlin Heidelberg and New York: Springer.
- 21. Mosher HP. The applied anatomy of the frontal sinus. Laryngoscope. 1904;14:830-55.
- 22. Williams PW. Rhinology a text book of diseases of the nose and nasal accessory sinuses. London: Longmans Green and Co.; 1910. p. 34–54.
- 23. Lothrop HA. The anatomy and surgery of the frontal sinus and anterior ethmoidal cells. Ann Surg. 1898;28:611–38.
- 24. Casiano RR. Endoscopic sinonasal dissection guide. New York: Thieme; 2011.
- 25. Bent J, et al. The frontal cell in frontal recess obstruction. Am J Rhinol. 1994;8:185-91.
- Bolger WE, et al. Paranasal sinus bony anatomic variations and mucosal abnormalities: CT analysis for endoscopic sinus surgery. Laryngoscope. 1991;101(1pt 1):56–64.
- Bolger WE, Mawn. CB Analysis of the suprabullar recess for endoscopic sinus surgery. Ann Otol Rhinol Laryngol Suppl. 2001;186:3–14.
- Loury MC. Endoscopic frontal recess and frontal sinus ostium dissection. Laryngoscope. 1993;103(4 pt 1):455–8.
- 29. Van Alyea QE. Frontal cells an anatomic study of these cells in consideration of their clinical significance. Arch Otolaryngol. 1941;34:11–23.
- Wormald P. The agger nasi cell: the key to understanding the anatomy of the frontal recess. Otolaryngol Head Neck Surg. 2003;129(5):497–507.

- 2 Surgical Anatomy and Embryology of the Frontal Sinus
- Folbe AJ, Casiano RR. Surgical anatomy in revision sinus surgery. In: Kountakis SE, Jacobs JB, Gosepath J, editors. Revision sinus surgery. Springer, Verlag, Berlin, Heidelberg; 2008;pp 53–61.
- Devyani Lal, Stankiewicz J. Primary sinus surgery in Cummings otolaryngology: head & neck surgery. 5th ed. Philadelphia: Elsevier Mosby; 2010.
- 33. Wormald P. Anatomy of the frontal recess and frontal sinus with three dimensional reconstruction. In: Worldmald, editor. Endoscopic sinus surgery, anatomy, three dimensional reconstruction, and surgical technique. 2nd ed. New York, NY: Thieme; 2008.
- Stammberger H. Endoscopic anatomy of lateral wall and ethmoidal sinuses. In: Stammberger H, Hawke M, editors. Essentials of functional endoscopic sinus surgery. St. Louis: Mosby-Year Book; 1993. p. 13–42.
- Kuhn FA. Chronic frontal sinusitis: the endoscopic frontal recess approach, in operative technigues. Otolaryngol Head Neck Surg. 1996;7(3):222–9.
- 36. Wigand ME. Endoscopic anatomy of the nose and paranasal sinus. From endoscopic surgery of the paranasal sinuses and anterior skull base. 2nd ed. New York: Thieme; 2008.

# Chapter 3 Radiologic Anatomy of the Frontal Sinus

Ramon E. Figueroa

#### Contents

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#### **Core Messages**

- Multidetector computed tomography (MDCT) is the primary imaging tool for a thorough evaluation of this complex anatomy, taking advantage of its capability to obtain multiplanar high quality reformatted images and volume rendered surface images in the computer workstation.
- Multiplanar capability of CT scanners has impacted the evaluation of the frontal sinus drainage pathways the most.
- 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.

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- The agger nasi cells and the uncinate process dictate the floor and the pattern of drainage of the frontal recess.
- Important anatomic variants impact on the anatomy of the frontal sinus drainage pathways and the anterior skull base. Familiarity with the frontal bulla cells, supraorbital ethmoid cells and the depth of the olfactory fossa, is required for safe anterior skull base and frontal recess surgical considerations.

# Introduction

The frontal sinus and its drainage pathway is one of the most complex anatomic areas of the anterior skull base. Its complexity is magnified by the frequency of anatomic variations which impact on the direction of drainage, efficiency of mucociliary clearance and morphology of the frontal recess. Multidetector computed tomography (MDCT) is the primary imaging tool for a thorough evaluation of this complex anatomy, taking advantage of its capability to obtain multiplanar high quality reformatted images and volume rendered surface images in the computer workstation. 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 most superior remnant of the first ethmoturbinal becomes the agger nasi mound, while 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, while 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 along 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**

Computed tomography (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]. Modern multidetector CT scanners with the corresponding high capacity workstations are now widely available in most hospitals and imaging centers, providing high resolution processed images 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 sagittal plane has become routine.

Typical high resolution multi detector scanning is performed in the axial plane (Fig. 3.1a) following the long axis of the hard palate, using low MA technique, a small field of view (18–20 cm) and 0.625 mm slice profile dictated by the thickness of the individual channels in the CT detector array, with data 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 coronal and

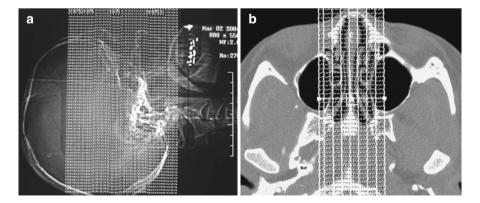


Fig. 3.1 High-resolution sinus MDCT protocol: (a) Lateral scout view shows the typical prescription of axial thin section slices. (b) An axial image at the level of the nasal cavity helps prescribe the sagittal reformatted images

sagittal planes perpendicular to the hard palate. Figure 3.1b shows the corresponding prescription for a set of sequential sagittal sections to encompass both frontal sinuses and their corresponding drainage pathways.

# **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. 3.2a, b), extending between the anterior and posterior frontal sinus walls at the skull base level. This

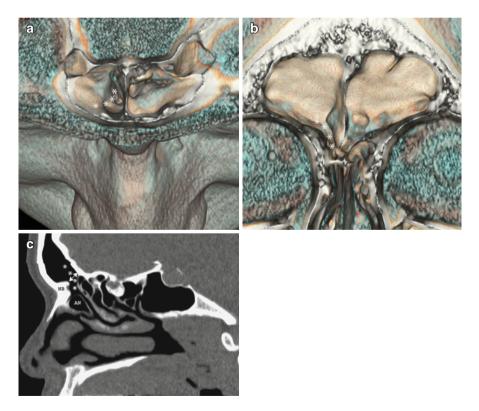
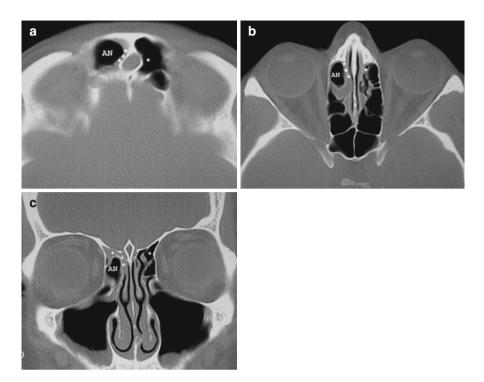


Fig. 3.2 The frontal sinus ostium: axial 3D volume rendered image (**a**), coronal 3D volume rendered image (**b**) and sagittal reformatted image (**c**) at the level of the frontal sinus illustrate the frontal sinus ostium (*arrows*), the frontal recess (\*\*\*\*), the nasal beak (*NB*) and the agger nasi (*AN*) cells

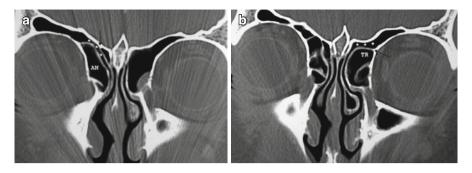


**Fig. 3.3** The frontal recess: a large right agger nasi cell (AN) is stenosing the right frontal recess (\*\*\*), which is opacified by congested mucosa and can be followed on coronal and sequential axial images. The left frontal recess (\*) is well aerated

point is typically demarcated along its anterior wall by the variably shaped bone ridge of the nasofrontal buttress, frequently called the "nasal beak" (Fig. 3.2c). 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. 3.2c) 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. 3.3). The medial wall is defined by the vertical attachment 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 anterior-inferior direction by hyper-pneumatized agger nasi cells (Fig. 3.3). Its inferior drainage is dictated by the insertion of the vertical attachment of the uncinate process, a



**Fig. 3.4** 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 ending in a blind end or "terminal recess" (*TR*)



**Fig. 3.5** 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 inferioris (\*) are shown better as sagittally oriented landmarks

sagittally oriented hook-like bony leaflet (Fig. 3.4). 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. 3.4a). If the uncinate process attaches laterally into the lamina papyracea of the orbit (Fig. 3.4b), the frontal recess opens directly into the superior aspect of the middle meatus, and the ethmoidal infundibulum ends superiorly into a blind "terminal recess".

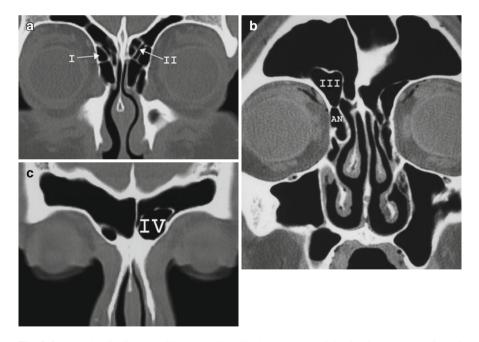
The ethmoidal infundibulum is a true three-dimensional space defined laterally by the lamina papyracea, anteromedially by the uncinate process and posteriorly by the bulla ethmoidalis (Fig. 3.5a). It opens medially into the middle meatus across the hiatus semilunaris inferioris, a cleft-like opening between the free posterior margin of the uncinate process and the corresponding anterior face of the bulla ethmoidalis (Fig. 3.5b). It is the functional common pathway of mucociliary drainage for

the anterior ethmoid, agger nasi and maxillary sinus mucus. The frontal sinus secretions can also drain through the ethmoidal infundibulum if the uncinate process does not attach to the lamina papyracea of the orbit.

# **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. 3.6). 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 sinusitis. Type I frontal cells are described as a single frontal recess cell above the agger nasi cell (Fig. 3.6a). Type II frontal cells are 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



**Fig. 3.6** 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)

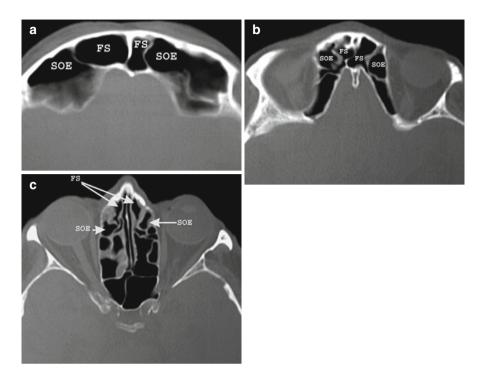
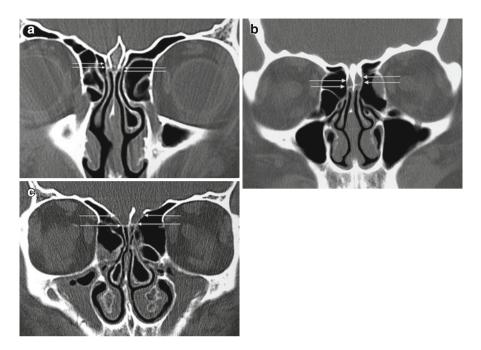


Fig. 3.7 Supraorbital ethmoid cells: in the sequential axial images (a-c) the supraorbital ethmoid cells (*SOE*) expand and pneumatize anteriorly into the orbital plate of the frontal bone, not to be confused with the frontal sinus (*FS*)

agger nasi, pneumatizing cephalad into the frontal sinus (Fig. 3.6b). Type IV frontal cell is a single isolated cell within the frontal sinus, frequently difficult to visualize due to its thin walls (Fig. 3.6c).

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. 3.7), 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 on axial images 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. 3.8). 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. Keros described the anatomic variations



**Fig. 3.8** 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)

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. This 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 constitute 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 problems in the frontal sinus drainage pathway.

# References

- 1. Bent JP, Cuilty-Siller C, Kuhn FH. The frontal cell as a cause of frontal sinus obstruction. Am J Rhinol. 1994;8(4):185–91.
- Bolger WE, Mawn CB. Analysis of the suprabullar and retrobullar recesses for endoscopic sinus surgery. Ann Otol Rhinol Laryngol. 2001;110:3–14.
- Daniels DL, Mafee MF, Smith MM, et al. The frontal sinus drainage pathway and related structures. AJNR Am J Neuroradiol. 2003;24:1618–26.
- Keros P. Uber die praktische bedeutung der niveauunterschiede der lamina cribosa des ethmoids. Laryngol Rhinol Otol (Stuttgart). 1965;41:808–13.
- Melhelm ER, Oliverio PJ, Benson ML, et al. Optimal CT evaluation for functional endoscopic sinus surgery. AJNR Am J Neuroradiol. 1996;17:181–8.
- 6. Stammberger HR. Functional endoscopic sinus surgery. Philadelphia: BC Decker; 1991.
- 7. Stammberger HR, Kennedy DW, Bolger WE, et al. Paranasal sinuses: anatomic terminology and nomenclature. Ann Rhinol Otol Laryngol Suppl. 1995;167:7–16.

# Chapter 4 Microbiology of Chronic Frontal Rhinosinusitis

#### **Subinoy Das**

#### Contents

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#### **Core Messages**

- The role of microbiology in chronic frontal rhinosinusitis remains poorly understood. Cultures poorly detect all types of microbiology present in the frontal sinus. The value of antimicrobial therapy is unclear in chronic forms of frontal rhinosinusitis.
- Viruses infect frontal sinus mucosa and may contribute to the chronicity of frontal sinus infections.
- Fungal disease manifests in several forms in the frontal sinus. Diagnosis is often difficult and can mimic malignancy and other types of diseases.

# Introduction

The microbiology and immunology underlying chronic frontal rhinosinusitis remain poorly described. Traditionally, our knowledge of the pathophysiology resulting in acute bacterial sinusitis and chronic rhinosinusitis has been, in part, determined by

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S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_4

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culture-based studies. However, cultures provide a reductionist view of the microbiota from the surfaces being sampled, sometimes identifying less than 2 % of the bacteria from any particular site. Cultures also do a poor job at identifying bacterial biofilms, the preferred bacterial phenotype within an epithelial surface, and are subject to contamination from ecologically distinct environmental niches, such as the nasopharynx, anterior nasal cavity, and frontal sinus mucosa. Furthermore, commensal bacterial species from the nasopharynx are possibly the predominant pathogens in the frontal sinus, at least in acute infections. As a result, it has been difficult to elucidate the true role that bacteria play in the pathophysiology of chronic frontal rhinosinusitis.

However, the growing use of high-throughput and molecular-based assays has increased our understanding into the pathophysiology of chronic rhinosinusitis. This chapter will discuss our current state of knowledge and theories on the role of various microbiologic agents in the pathogenesis of chronic frontal rhinosinusitis.

#### **Chronic Viral Frontal Rhinosinusitis**

While paranasal sinus mucosa is known to be frequently infected acutely with common upper respiratory viruses, little is known about the chronicity of such infections. However, viral infections are theorized to be causally related to chronic bacterial infections. For example, early historical work by Arnold et al. [2] failed to produce experimental bacterial rhinitis by spraying bacteria into the nasal cavities of 42 healthy adults. However, Hilding [4] was able to induce an experimental frontal sinus infection after suspending bacteria in warm milk. Viruses are known to halt mucociliary clearance and increase mucus formation which may serve a similar function. Furthermore, the most common experimental model for acute and subacute otitis media in the chinchilla [10] requires the use of a viral co-infection prior to inoculation with bacteria. Also, Buchman et al. [3] in an experimental influenza study, demonstrated the progressive increase in *Streptococcus pneumoniae* titers in nasal secretions following inoculation with the influenzae virus alone. The microbial interface and the interplay between viruses and commensal bacteria is an area of significant interest in the study of the pathophysiology of chronic rhinosinusitis.

#### **Chronic Bacterial Frontal Rhinosinusitis**

Chronic frontal sinus infections are of enormous importance to the otolaryngologist, particularly due to the fact that the foramina of Breschet, trans osseous venous channels in the posterior table of the frontal bone, provide a direct conduit for infectious agents to the intracranial contents. Much of what is known about chronic frontal rhinosinusitis has been gleaned from studies throughout the paranasal sinus contents, since it remains unclear if chronic frontal rhinosinusitis is

	No prior	Prior FESS <sup>a</sup>	
	sinonasal	without frontal	Prior surgery of
	surgery	surgery	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	12 % (1/8)	19 % (4/21)	33 % (2/6)
Staphylococcus			
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)
Xanthamonas	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. pneumoniae	0 % (0/8)	5 % (1/21)	0 % (0/6)
Anaerobic bacteria	0 % (0/7)	0 % (0/21)	25 % (1/4)
(Gram-Positive cocci)			
Fungi (Penicillium)	0 % (0/6)	7 % (1/14)	0 % (0/5)

 Table 4.1
 Culture results of frontal sinus aspirates (46 trephines)

With permission from *The Laryngoscope* [7]

<sup>a</sup>FESS functional endoscopic sinus surgery

pathophysiologically distinct from other types of chronic rhinosinusitis, particularly chronic ethmoiditis.

Schlosser et al. [7] examined bacterial and fungal cultures taken from 30 consecutive patients undergoing trephinations for chronic frontal sinusitis. Nearly 40 % of cultures demonstrated no growth; *Staphylococcus aureus* and coagulase-negative *Staphylococcus* species were the most common organisms detected (See Table 4.1).

Sanderson et al. [6] examined chronic rhinosinusitis samples taken at the time of surgery and analyzed these samples with confocal microscopy and fluorescent in situ hybridization techniques. They found the presence of bacterial biofilms in 14 of 18 CRS samples with non-typeable *Haemophilus influenzae* as the most predominant bacteria detected.

Stephenson et al. [8] performed a study examining both culture results and 16S rRNA sequencing of sinus samples taken during surgery. The use of molecular detection methods significantly increased the sensitivity of bacterial detection, but there were no significant differences in the microbiology of the samples between controls and patients with chronic rhinosinusitis.

Similarly, Abreu et al. [1] utilized a 16S rRNA microarray to compare differences between maxillary sinus samples from seven healthy control patients and seven patients with chronic rhinosinusitis, as confirmed by up-regulation in their mucin secretion and up-regulation of the Muc5A gene. Their analysis demonstrated no significant difference in sinus bacterial burden between patients with CRS and healthy controls, and they found the presence of suspected pathogenic bacteria in both groups. Therefore, neither the presence of bacteria nor the detection of certain strains of bacteria signified chronic rhinosinusitis. Rather, patients with CRS were less likely to demonstrate microbiological diversity, and were more likely to contain certain *Corynebacterium* strains. They were also less likely to contain bacteria associated with probiosis such as certain *Lactobacillus* species.

• While these studies have confirmed the presence of bacteria in surgical samples taken from patients with chronic rhinosinusitis, no studies to date have conclusively proven Koch's postulates for the causative role of any microbiologic agent in the pathophysiology of chronic frontal rhinosinusitis. As a result, current theories on the role of bacteria in chronic rhinosinusitis have been gleaned from studies on similar organ systems.

Stoltz et al. [9] examined newborn pigs containing a genetic knockout for the chloride transporter gene known to cause cystic fibrosis. They reported that these newborn pigs had no evidence of airway inflammation, but that these newborn pigs demonstrated defective bacterial clearance and developed hallmark evidence of cystic fibrosis only after a few months of life. *Staphylococcus aureus* was a common bacteria found in the lungs of these CF pigs, along with a multitude of other pathogens, similar to findings by Stephenson et al. and Abreu et al. This study suggested that in cystic fibrosis, the innate immune defect induced by a defective chloride transporter was responsible for defective bacterial clearance which led to airway inflammation. Neither the genetic defect alone nor any specific bacterial species such as *Pseudomonas aeruginosa* were solely responsible for the lung disease found in cystic fibrosis.

Hooper et al. [5] has also described the complex and intricate symbiotic relationship of commensal microbiota and the development and proper function of the host immune system, particularly within the gastrointestinal system. The lack of exposure to microbiota in early mammalian development has been demonstrated to causally create subsequent defects in host-bacterial homeostasis on epithelial surfaces. Furthermore, specific defects in the innate and adaptive arms of immunity manifest in derangements of the commensal microbiota.

• While the pathophysiologic causative agent remains unidentified for most subsets of chronic frontal rhinosinusitis, it is clear that proper homeostasis of the host immune system and the commensal microbiota residing on the surface of sinus epithelium is required to maintain normal sinus health.

Further research into the role of bacteria in the etiology of chronic frontal sinusitis will lead to more rational therapies that can fundamentally cure and prevent the formation of chronic frontal rhinosinusitis.

# **Chronic Fungal Frontal Rhinosinusitis**

Fungal disease of the paranasal sinuses can manifest in varied forms, depending in part on the type of fungus involved and the type of immune reaction to such fungus. Most commonly these manifestations are classified into invasive and non-invasive

types. An important requirement in diagnosing fungal disease of the frontal sinus is to rule out acute fulminant invasive fungal sinusitis, which is often lethal, particularly if care is delayed due to misdiagnosis. Chronic forms of fungal frontal rhinosinusitis include chronic invasive fungal rhinosinusitis, sinus fungal balls, saprophytic fungal infestation, and allergic fungal rhinosinusitis.

Chronic invasive frontal fungal rhinosinusitis has been differentiated into two distinct types: granulomatous type and chronic invasive type. Chronic granulomatous fungal rhinosinusitis is typified by non-caseating granulomas associated with *Aspergillus flavus* and is most often seen in Sudan, Saudi Arabia, India, and Pakistan. Chronic invasive (non-granulomatous) types have less fibrosis, however both forms often have significant orbital involvement.

 Sinus fungal balls are more commonly found in maxillary and sphenoid sinuses, BUT occasionally are found in the frontal sinus. These are non-invasive masses most commonly associated with Aspergillus.

Saprophytic fungal infestation is likely a localized fungal colonization, similar to oral thrush, however localized to the frontal sinus. It is very rare to manifest solely in the frontal sinus, though may signify poor mucociliary function.

Allergic fungal rhinosinusitis is a distinct clinical entity and often encompasses the frontal sinus. It is discussed in depth in a separate chapter. The microbiology of AFRS is varied, though dematiaceous (dark-colored) molds are often involved.

# Conclusion

The role of infectious agents in the pathophysiology of chronic frontal rhinosinusitis continues to remain poorly understood. While the importance of antimicrobial therapy is undisputed in acute and subacute forms, the role and value of antimicrobials still remains unclear in most chronic forms of frontal disease. Further research is critically necessary to elucidate the optimal role of antimicrobial therapy in the management of chronic frontal rhinosinusitis.

# References

- 1. Abreu NA, Nagalingam NA, Song Y, Roediger FC, Pletcher SD, Goldberg AN, Lynch SV. Sci Transl Med. 2012;4:1–9.
- 2. Arnold L, Ostrom ML, Singer C. Autosterilizing power of the nasal mucous membrane. Proc Soc Exp Biol Med. 1928;25:624.
- 3. Buchman CA, Doyle WJ, Skoner DP, Post C, Alper CM, Seroky JT, Anderson K, Preston RA, Hayden FG, Fireman P, Ehrlich GD. J Infect Dis. 1995;172:1348–51.
- 4. Hilding A. Studies of common cold and nasal physiology. Trans Am Laryngol Assoc. 1934; 56:253–71.

- 5. Hooper LV, Littman DR, Macpherson AJ. Interactions between the microbiota and the immune system. Science. 2012;8:1268–73.
- Sanderson AR, Leid JG, Hunsaker D. Bacterial biofilms on the sinus mucosa of human subjects with chronic rhinosinusitis. Laryngoscope. 2006;116:1121–6.
- Schlosser RJ, London SD, Gwaltney Jr JM, Gross CW. Microbiology of chronic frontal sinusitis. Laryngoscope. 2001;111:1330–2.
- Stephenson MF, Mfuna L, Dowd SE, Wolcott RD, Barbeau J, Poisson M, James G, Desrosiers M. Molecular characterization of the polymicrobial flora in chronic rhinosinusitis. J Otolaryngol Head Neck Surg. 2010;39:182–7.
- 9. Stolz DA, Meyerholz DK, Pezzulo AA, et al. Cystic fibrosis pigs develop lung disease and exhibit defective bacterial eradication at birth. Sci Transl Med. 2010;2:29–31.
- Suzuki K, Bakaletz LO. Synergistic effect of adenovirus type 1 and nontypeable Haemophilus influenzae in a chinchilla model of experimental otitis media. Infect Immun. 1994;62:1710–8.

# **Further Reading**

- Antila J, Suonpaa J, Lehtonen O. Bacteriological evaluation of 194 adult patients with acute frontal sinusitis and finding of simultaneous maxillary sinusitis. Acta Otolaryngol (Stock) Suppl. 1997;529:162–4.
- Berger G, Kattan A, Bernheim J, Ophir D, Finkelsein Y. Acute sinusitis: a histopathological and immuno-histochemical study. Laryngoscope. 2000;110:2089–94.
- Bjorkwall T. Bacteriological examination in maxillary sinusitis: bacterial flora of the maxillary antrum. Acta Otolaryngol Suppl (Stockh). 1950;83:1–58.
- Brook I. Bacteriology of acute and chronic frontal sinusitis. Arch Otolaryngol Head Neck Surg. 2002;128:583–5.
- Forsgren K, Fukami M, Penttila M, Kumlien J, Stierna P. Endoscopic and Caldwell-Luc approaches in chronic maxillary sinusitis: a comparative histopathologic study on preoperative and postoperative mucosal morphology. Ann Otol Rhinol Laryngol. 1996;104:350–7.
- Gwaltney Jr JM, Hendley JO, Phillips CD, Bass CR, Mygind N, Winther B. Nose blowing propels nasal fluid into the paranasal sinuses. Clin Inf Dis. 2000;30:387–91.
- Gwaltney Jr JM, Phillips CD, Miller DR, Riker DK. Computed tomographic study of the common cold. N Engl J Med. 1994;330:25–302.
- Hamory BH, Sande MA, Sydnor Jr A, Seale DL, Gwaltney JM. Etiology and antimicrobial therapy of acute maxillary sinusitis. J Infect Dis. 1979;139:197–202.
- Medzhitov R. Toll-like receptors and innate immunity. Nat Rev Immunol. 2001;1:135-45.
- Medzhitov R, Janeway Jr C. Innate immunity. N Engl J Med. 2000;343:338-44.
- Ohashi Y, Nakai Y. Functional and morphologic pathology of chronic sinusitis mucous membrane. Acta Otolaryngol Suppl. 1983;397:11–48.
- Pedersen M, Sakakura Y, Winther B, Brofeldt S, Mygind N. Nasal mucociliary transport, number of ciliated cells, and beating pattern in naturally acquired common colds. Eur J Resp Dis. 1983;64 Suppl 128:355–64.
- Pitkaranta A, Arruda E, Malmberg H, Hayden FG. Detection of rhinovirus in sinus brushing of patients with acute community-acquired sinusitis by reverse transcription-PCR. J Clin Microbiol. 1997;35:1791–3.
- Ponikau JU, Sherris DA, Kern EB, Holmburger HA, Frigas E, Gaffey TA, Roberts GD. The diagnosis and incidence of allergic fungal sinusitis. Mayo Clin Proc. 1999;74:877–84.
- Winther B. Effects on the nasal mucosa of upper respiratory viruses (common cold). Dan Med Bull. 1994;41:193–204.
- Winther B, Gwaltney JM, Humphries, Hendley JO. Cross-linked fibrin in nasal fluid of patients with the common cold. CID. 2002;34:708–10.
- Winther B, Gwaltney Jr JM, Mygind N, Turner RB, Hendley JO. Intranasal spread of rhinovirus during point-inoculation of the nasal mucosa. J Am Med Assoc. 1986;256:1763–7.

# **Chapter 5 Instruments for Frontal Sinus Surgery**

Vijay R. Ramakrishnan and Todd T. Kingdom

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#### **Core Messages**

- Frontal sinus surgery is the most challenging of the sinus procedures.
- All aspects of the operation must be optimized to afford the highest chances of success. This includes the availability of necessary instrumentation and familiarity with their use.
- Preoperative surgical goals should be established, and the procedure can be modified based on intraoperative findings to meet these goals. Patient-specific factors, such as etiology and extent of disease, response to medical

**Electronic supplementary material** The online version of this chapter (doi:10.1007/978-3-662-48523-1\_5) contains supplementary material, which is available to authorized users.

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therapies, and comorbid conditions, often factor into this decision-making process.

- Mucosal preservation in the frontal dissection is critical; therefore, sharp dissection and avoidance of trauma are baseline principles and should be applied where possible. Current innovation in frontal sinus instrumentation design helps meet these critical surgical objectives.
- Frontal sinus instrumentation is highly specialized, potentially costly, and delicate. Many technical aspects are driven by surgeon-preference and may rely more on certain instruments than others.
- Balloon technology may be used as a tool to facilitate delineation of the frontal outflow tract, but may not be sufficient in of itself in most cases.

# Introduction

Frontal sinus surgical anatomy is complex and the dissection is often technically demanding; as such, experience is requisite. Success rates are variable because of the many potential factors leading to surgical failure. Each subsequent attempt at frontal surgery on a patient may become progressively more difficult, so appropriate decision-making and early success is ideal.

Thorough examination of the computed tomography (CT) scan preoperatively is performed to examine and understand the patient's unique anatomic features. This review is accomplished in axial, sagittal, and coronal views and the surgeon can begin to develop the operative plan. The planned surgical steps and anticipated maneuvers will drive selection of instrumentation. The CT scan should be available in the operating room; in general, digital images are simpler to scroll through than printed images (Video 5.1). Use of image-guided surgery (IGS) can facilitate a more rapid scrolling through of the CT scan, and is helpful to assess the endoscopic anatomy in real-time. Although the use of IGS certainly can be helpful, and in fact is endorsed by the AAO-HNS for frontal surgery [1], its use has not thoroughly been demonstrated to improve surgical outcomes or decrease the rate of complications for frontal sinus surgery [2].

• Experience has demonstrated that mucosal preservation is most critical in the frontal dissection. As a result, there has been a gradual shift towards meticulous sharp dissection utilizing through-cutting instrumentation and microdebriders, allowing for mucosal-sparing approaches.

A number of different techniques are taught, but generally begin with bluntly identifying the frontal sinus outflow pathway in a gentle manner, and following this with sharp dissection of neighboring bony partitions and mucosa. The initial identification of the outflow tract can be achieved with probes or curettes, or perhaps balloon dilation, and subsequent sharp dissection follows. The introduction of new frontal sinus instrumentation over the years as had a large impact in the application of these surgical concepts.

One key to mucosal preservation is appropriate visualization. Angled endoscopy is mandatory for frontal dissection, typically requiring 45- and/or 70- degree

endoscopes. During no other aspect of endoscopic sinus surgery does hemostasis play as critical a role as in visualization during frontal sinus dissection. Anesthetic technique, patient positioning in a slight reverse Trendelenburg position, injection of lidocaine with epinephrine, placement of topical epinephrine-soaked cottonoids, and patience, are the main facilitators of excellent hemostasis.

If advanced procedures are planned, such as the Draf IIb or Draf III dissections, a drill may be required. Angled burs attaching to the microdebrider platform are available, and keep the operating room equipment setup fairly simple. These drills operate at low-speed, and can take some time when significant bone removal is required or in the setting of bony sclerosis. Cutting burs are available that can facilitate quicker bone removal, however more care is required with their use. When open or adjunct procedures are entertained, the required equipment must also be available. Finally, although complications are rare, the surgeon must be prepared to address and manage these without delay. In this chapter, we will describe current instrumentation for use in frontal sinus surgery.

## **CT Scan and IGS**

The CT scan must be available in the operating room for review during the surgical case. The IGS platform offers an additional benefit of simple scrolling through the multi-planar images aiding the surgeon in surgical planning. Several IGS platforms exist, and are described in detail in Chap. 20. Once the image-guidance apparatus is applied and registered, its accuracy is confirmed on fixed intranasal landmarks and rechecked often throughout the case.

Advantages of surgical navigation in frontal sinus surgery:

- Allows the surgeon to view the relevant surgical anatomy in multiple planes.
- Development of a surgical plan based on the complex anatomy present.
- · Confirms that complete and thorough dissection has been achieved.

In the frontal recess dissection, navigating with a curved suction or probe is performed to confirm anatomic understanding (Fig. 5.1). Certain manufacturers allow for the ability to navigate with the surgeon's choice of instruments, either by attaching an array to that particular instrument (such as a microdebrider or curette), or by sending the instrument to the company for custom creation.

#### **Endoscopy and Visualization**

• The rigid nasal endoscope is perhaps the single most important tool to consider and angled endoscopes are critical to achieving a complete frontal recess dissection in a safe manner while preserving underlying mucosa.

Specific techniques to remove bone of the frontal process of the maxilla neighboring the agger nasi cell may be utilized to allow for frontal dissection with 0- or 30-degree telescopes. However, most surgeons perform a retrograde dissection after



**Fig. 5.1** The image guidance system is applied and registered, and its accuracy is tested on intranasal landmarks such as the nasal floor. Scrolling through the anatomy is helpful for surgical planning. The *inset* shows commonly used instruments for frontal sinus navigation, including a curved suction and probe

identification of the skull base within the ethmoid sinus. This requires the use of 45- and/or 70-degree endoscopes. The 70-degree is perhaps initially more awkward, but allows for a more complete view, especially if the drainage pathway is located anteriorly (Fig. 5.2, Video 5.2). Though challenging at first, it is imperative that the surgeon becomes comfortable and facile with these more angled scopes in order to optimize patient outcomes.

Hemostasis is critical to visualization in the frontal recess dissection. Instruments that can aid in hemostasis include endoscopic bipolar forceps and the malleable suction bovie, although these are rarely used. Cottonoids soaked in hemostatic medications are the most useful; pressure and patience will provide a clean and dry operative field to optimize the chances of surgical success. Our preference is to place  $\frac{1}{2}'' \times 3''$  cottonoids soaked in 1:1000 epinephrine in the anterior ethmoid region prior to frontal recess dissection (Video 5.2). This technique has been described elsewhere, and is generally safe, although precautions should be taken [3].

# **Instrumentation by Technique**

# **Blunt Dissection**

The ultimate objective is removal of accessory cell partitions, marsupialization of these cells into the frontal recess, and removal of disease in the frontal recess while preserving the underlying mucosa. Surgeon preference guides the technical aspects

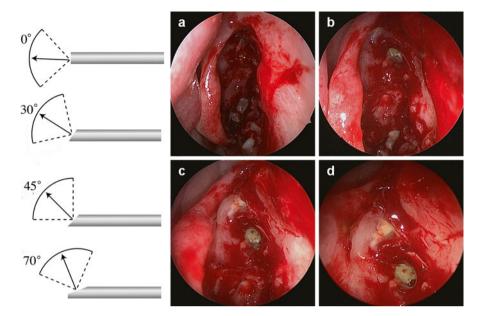
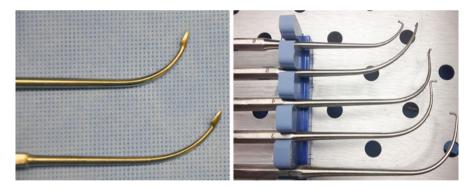


Fig. 5.2 Endoscopic appearance of left frontal dissection with angled endoscopes, which allow for more complete view of the anterior superior aspect of the frontal recess. (a) 0-degree, (b) 30-degree, (c) 45-degree, (d) 70-degree



**Fig. 5.3** Blunt dissection of the frontal recess can be performed with curettes (*left*) used to fracture partitions and probe pathways, or with Kuhn frontal sinus probes (*right*) which have a narrower profile and curved tips to mobilize bone fragments or finely adjust mucosal edges

of how this is done. These goals can be initially met with "blunt" dissection used to identify the frontal sinus outflow pathway and dilate this tract to facilitate the use of subsequent instruments. Gentle passage of probes and/or curettes can be used in this manner (Figs. 5.3 and 5.4). Curved suctions, and malleable suctions or curettes, can be used according to surgeon preference. More recently, balloon dilation has been used in this manner and is further discussed in Chap. 17 (Video 5.3). Giraffe-style cup forceps are used to pick out bone chips, carefully leaving the underlying mucosa undisturbed (Fig. 5.5).



**Fig. 5.4** Curved olive-tip suctions of varying diameters are available and will accommodate most frontal anatomy. Occasionally, specialized frontal suctions such as the van Alyea suction (*bottom*) or a malleable suction is preferable



**Fig. 5.5** Giraffe-type frontal cups (*right*) and through-cutting instruments (*left*) are available. 60- and 90-degree, and front-to-back and side-to-side varieties are shown



Fig. 5.6 *From left to right*, the Hosemann punch, mushroom punch, and Bachert punch, are shown. The Hosemann and Bachert punches are useful for aggressive bone removal, but may also denude mucosa as a result

# Sharp Dissection

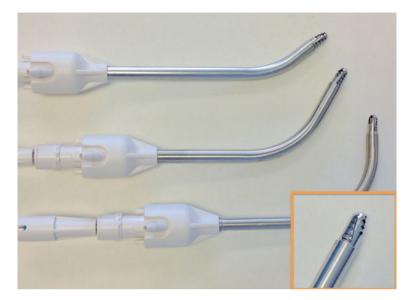
Once the outflow pathway has been identified and partially dissected, "sharp" dissection of cell partitions, bony fragments, and redundant mucosa is achieved with giraffe-style through-cutting forceps. Upturned punches are helpful to widen the frontal ostium and remove excess bone of the frontal sinus floor and nasofrontal beak, if desired (Figs. 5.5 and 5.6, Video 5.2). A wide selection of frontal sinus punches is currently available designed to meet different needs. Some will target removal of thinner bone while more robust punches are specifically designed to for thicker bone in this region.

• The microdebrider has become the workhorse for sinus dissection, and this includes the frontal recess and within the frontal sinus, if appropriate.

The combination of suction and mucosal-preservation makes the microdebrider a valuable tool; additionally, the angled debrider (Fig. 5.7) can be used in a similar fashion to a curette and navigation can be applied if desired. Caution, however, must be exercised when using powered-instrumentation in the frontal recess. The potential for inadvertent mucosal trauma is higher when using this technique.

# **Advanced Procedures**

Advanced frontal sinus procedures, particularly the Draf IIb and Draf III, dissections, require certain instrumentation as well. These procedures are thoroughly described in Chap. 26, and begin with standard frontal recess dissection.



**Fig. 5.7** Microdebriders (40-, 60-, and 90-degree) have a rotating tip, which can be used with caution under direct visualization for selective resection of polyps, mucosa, and bony partitions

Removal of the anterior portion of the middle turbinate and superior septum can be performed with through-cutting Blakesley forceps and the frontal giraffestyle through-cutting forceps. In the setting of bony thickening along the superior septum, the surgeon may proceed to using the drill. Angled drills are utilized to remove the floor of the frontal sinus and to thin the nasofrontal beak (Fig. 5.8). Removal of this bone is augmented by intermittent use of the punches shown in Fig. 5.6.

# **Adjunct Open Procedures**

Trephination or "mini"-trephination of the frontal sinus can be utilized to irrigate the sinus, aid in identification of outflow, or assist in performance of a combined open-endoscopic procedure. Rapid accomplishment of the "mini"-trephination is achieved with the appropriate instruments (Fig. 5.9), or a larger opening into the sinus can be performed with standard soft tissue sets, a drill, and a craniofacial plating system. The same instrument sets can be used for coronal approaches to the frontal sinus. The use of current navigation platforms with accompanying cranial posts or anchors for the reference frame has replaced the traditional 6-ft Caldwell plain film x-ray in planning for the osteoplastic flap (Fig. 5.10).



**Fig. 5.8** Diamond and cutting burs attach to the microdebrider platform. The inset shows a 15-degree diamond bur, a frontal finesse bur, and a 70-degree diamond bur. Continued innovation in drill technology will result in additional options and more rapid bone removal



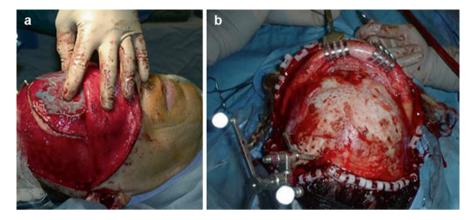
**Fig. 5.9** The mini-trephine kit contains a drill guide (*top*), a drill (*center*), and a cannula (*bottom*). Image guidance or anatomic landmarks can be used to decide on the location of trephination, and irrigation of saline or dilute fluorescein through the cannula can be seen transnasally to help identify the outflow tract

# Complications

Although rare, appropriate instrumentation should be available to address potential complications in a timely manner. Bleeding is the most common complication.

• The surgeon must be comfortable dealing with branches of the sphenopalatine artery or anterior ethmoid artery in a precise and efficient manner.

Endoscopic suction bipolar forceps are preferred for management when this is encountered. Bayonet bipolar forceps may also be used but often the anterior angle



**Fig. 5.10** Creation of the osteoplastic flap can be performed in the traditional manner using a 6-foot Caldwell view xray ( $\mathbf{a}$ ), or by using IGS with a post attached directly to the cranium rather than with fiducials ( $\mathbf{b}$ )

is challenging to achieve with this instrument and if the bleeding is profuse, the inability to concurrently suction is problematic. Careful focal treatment with a suction bovie has been used in this setting, but energy transmission to the skull base or orbit is a theoretical concern. Materials should be available for CSF leak repair in the event a leak or defect is encountered. This may necessitate the availability of synthetic dural substitute, tissue glue, dressing or packing, according to surgeon preference.

# **Postoperative Care**

Ideally, the surgeon would have everything available in the clinic setting that he/she has access to in the operating room. However, practically, a few basic instruments are required. Angled endoscopes, curved suctions, and probes are the basic required equipment. Cup forceps are useful to remove bone fragments, frontal sinus cannulas for irrigation or medication instillation, and through-cutting giraffe forceps and mushroom punches for managing stenosis and soft tissue disease.

# Conclusion

There has been tremendous expansion in our understanding of the surgical anatomy of the frontal recess and recent developments in more sophisticated surgical techniques to access frontal sinus disease. Concurrent to this has been a great deal of innovation in frontal sinus instrument design to aid the surgeon in applying this new knowledge and skill. Currently available instruments allow for mucosal-sparing techniques, precise soft tissue dissection, delicate bone removal, and access to regions formerly accessible only via external techniques. Understanding the available instruments, and their proper application, is very important to achieving optimal outcomes.

# References

- 1. http://www.entnet.org/Practice/policyIntraOperativeSurgery.cfm.
- Ramakrishnan VR, Orlandi RR, Citardi MJ, et al. The use of image-guided surgery in endoscopic sinus surgery: an evidence-based review with recommendations. Int Forum Allergy Rhinol. 2013;3(3):236–41.
- 3. Higgins TS, Hwang PH, Kingdom TT, et al. Systematic review of topical vasoconstrictors in endoscopic sinus surgery. Laryngoscope. 2011;121(2):422–32.

# Chapter 6 Acute Frontal Sinusitis

Ethan Soudry and Peter H. Hwang

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#### **Core Messages**

- Uncomplicated acute frontal sinusitis (AFS) is most often associated with an antecedent 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 diagnostic criteria for acute sinusitis and have symptoms referable to the forehead region.
- The predominant organisms cultured from patients with uncomplicated AFS are *Hemophilus influenzae*, *Streptococcus pneumoniae* and *Moraxella catarrhalis*.

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- When oral antibiotics are indicated, uncomplicated AFS should be treated for 10–14 days with amoxicillin-clavulanate (in patients without penicillin allergy).
- Although uncomplicated AFS is a self-limited disease, complicated acute frontal sinusitis can progress rapidly with catastrophic sequelae.
- Complicated AFS is suspected when symptoms are protracted and severe or when neurological deficits, frontal headache and fever are present.
- Work up of complicated AFS should include CT scan with IV contrast and MRI for inconclusive cases.
- Epidural and subdural abscesses are the most common intracranial complications of AFS.
- Patients with complicated AFS should be admitted for intravenous antibiotic therapy and intravenous hydration. Endoscopic sinus surgery or frontal trephination may be necessary to drain the frontal sinus. Craniotomy may be indicated for management of intracranial abscess.

# Introduction

The reported prevalence rates of acute rhinosinusitis (ARS) observed in primary care practice varies between 6 and 12 % [1]. Between 2000 and 2009 there was an average of 4.3 million outpatient visits annually for ARS. Antibiotics were prescribed in 83 % of these visits [2]. The National Ambulatory Medical Care Survey indicates that sinusitis (acute and chronic) is the fifth most common disease for which antibiotics are prescribed [3].

The primary predisposing factor for ARS is an antecedent upper respiratory viral infection. Approximately 0.5-2 % of viral upper respiratory tract infections are complicated by acute bacterial infection. The incidence of ARS is higher in winter months, in damp climates, and in cities with significant air pollution.

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

### **Etiology and Pathophysiology of Acute Frontal Sinusitis**

- Acute frontal sinusitis is most commonly preceded by a viral upper respiratory tract infection.
- Human rhinovirus is implicated in 50 % of cases, but other viruses may include coronavirus, influenza, parainfluenza, respiratory syncytial virus, adenovirus, and enterovirus.

#### 6 Acute Frontal Sinusitis

The peak prevalence of these viruses occurs in early fall and spring, which parallels the peak incidence of acute bacterial rhinosinusitis (ABRS). Viral infection leads to an inflammatory cascade in which T-helper type 1 cytokine polarization is associated with a high level of tumor necrosis factor- $\beta$  and interferon- $\gamma$ . There is also an associated release of proinflammatory cytokines such as interleukin (IL)-1 $\beta$ , IL-6, and IL-8. These cytokines are considered very potent chemoattractants for neutrophils [4]. The viral induction of the inflammatory cascade results in acute mucosal edema, occlusion of sinus ostia, and impairment of mucociliary clearance. The resulting mucus stasis can contribute to a milieu that favors the proliferation of pathogenic micro-organisms, resulting in acute bacterial sinusitis.

Risk factors for acute sinusitis may include a variety of host factors, including anatomic, inflammatory, immunologic, and environmental. Structural concerns, such as concha bullosa or septal deviation, may be clinically significant. Inflammatory conditions such as nasal polyposis may predispose to acute sinusitis by gross obstruction of sinus drainage by polyps, as well as by generalized mucosal edema. Environmental exposures should be considered, although the evidence for their associations can be variable. For example smoking is thought to be a risk factor for ARS by disrupting ciliary function [1], but the evidence for passive smoke exposure as a significant risk factor is less compelling [5]. Host immune factors such immunodeficiency or immunosuppression can be important risk factors, whereas the role of allergy in ARS is the subject of considerable debate, with studies both supporting and challenging its role [6, 7].

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 superior aspect of the ethmoid sinus that may affect frontal sinus drainage. 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 and suprabullar/frontal bullar cells posteriorly; supraorbital ethmoid cells laterally; and type I–IV frontal cells comprising variable spatial orientations within the frontal recess [8]. A recent study found that the presence of frontoethmoid cells in the posterior and posterolateral aspects of the frontal recess (suprabullar cells, frontal bullar cells, and supraorbital ethmoid cells) may have a more significant association with the development of frontal sinusitis than those cells in the anterior aspect of the frontal recess [9].

# **Uncomplicated Acute Frontal Sinusitis**

# Diagnosis

Historically recommended diagnostic algorithms based on combinations of major and minor symptoms have been abandoned in favor of more recent literature which focuses on three cardinal symptoms: purulent nasal discharge, nasal obstruction, and facial pain/

pressure/fullness [10]. According to the most recent guidelines from the American Academy of Otolaryngology (AAO) [10], ABRS is defined by cardinal symptoms of purulent nasal discharge, nasal obstruction and facial pain/pressure/fullness that are present 10 days or more beyond the onset of upper respiratory symptoms, or that worsen after initial improvement within the first 10 days (double worsening). The 10 day time point is selected in part because of the difficulty in discerning viral versus bacterial etiologies in the first 7–10 days of an acute upper respiratory tract infection [11].

The Infectious Diseases Society of America (IDSA) guidelines [12] define ABRS as either persistent symptoms or signs compatible with acute rhinosinusitis, lasting for 10 days without any evidence of clinical improvement; or onset with severe symptoms or signs of high fever 39 °C (102 °F) and purulent nasal discharge or facial pain lasting for at least three to four consecutive days at the beginning of illness; or onset with worsening symptoms or signs characterized by the new onset of fever, headache, or increase in nasal discharge following a typical viral upper respiratory infection (URI) that lasted 5–6 days and were initially improving ("double sickening").

The European Position Paper on Rhinosinusitis (EPOS) guidelines from 2012 define ARS in adults as sudden onset of two or more symptoms, one of which should be either nasal blockage/obstruction/congestion or nasal discharge (anterior/ posterior nasal drip) and the other being facial pain/pressure or reduction or loss of smell [1]. ABRS is suggested by the presence of at least three of any of the following symptoms and signs- discolored nasal discharge, severe local pain, fever >38 °C, elevated ESR/CRP or double sickening. Endoscopic evidence of middle meatal purulence supports the diagnosis.

Both the AAO and EPOS recommend against plain x-rays for patients already meeting the clinical diagnostic criteria. CT scan or MRI of the sinuses is recommended only when a complication is suspected or when the patient is immunocompromised.

There are no site-specific criteria for the diagnosis of acute frontal sinusitis. Generally acute frontal sinus symptoms are referable to the brow, temple, and frontal bone region. Frontal headache is the most prevalent symptom of acute frontal sinusitis [13].

• Thus, a diagnosis of acute frontal sinusitis should be considered in patients who meet the diagnostic 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 acute frontal sinusitis. This is especially true in those patients without a prior history of chronic headache.

# **Bacteriology**

The most common bacteria isolated from patients with ABRS are Streptococcus pneumoniae (20–43 %), Haemophilus influenzae (22–35 %) and Moraxella catarrhalis (2–10 %). Staphylococcus aureus, Streptococcus pyogenes, and anaerobic

bacteria may also be involved to a lesser extent, with anaerobic bacteria being classically associated with odontogenic infections. Pseudomonas aeruginosa and other gram-negative rods may be recovered in patients with nosocomial sinusitis (e.g., associated with nasal tubes or catheters), immunocompromised patients, and those with cystic fibrosis [14]. Although regional geographic variations exist, about 15–20 % of Strep. pneumoniae are resistant to penicillin, and about 80 % of M. catarrhalis and 30 % of H influenza are beta-lactamase producing [10].

In children, the pathogen profile of acute sinusitis in the US has undergone significant shifts since the introduction of the seven valent pneumococcal vaccine. The incidence of Strep pneumonia isolates has dropped from 44 to 27 %, along with reported increases in H influenzae from 37 to 44 %, Strep pyogenes from 7 to 12 %, and Staph aureus from 4 to 8 %, with no change in Moraxella catarrhalis.

Changing patterns of resistance rates deserve attention and should be taken in consideration in patients not responding to first line treatment. Endoscopic cultures of the middle meatus may be appropriate in these cases.

• Middle meatal cultures correlate well with maxillary sinus puncture cultures, with an of 87 % concordance rate [15].

Culture data specific to acute frontal sinusitis are scarce owing to the difficulty of obtaining frontal sinus cultures. Given that acute frontal sinusitis typically occurs in conjunction with acute maxillary and ethmoid sinusitis, it would be reasonable to expect that the same pathogens observed in acute maxillary and ethmoid sinusitis would also be found in acute frontal sinusitis. Although the literature is sparse, the few studies that have examined this indeed support this notion [16–18].

# Treatment

In light of the fact that some cases of acute bacterial sinusitis may spontaneously resolve without antibiotic therapy, the AAO recognizes that observation is an option for selected patients with uncomplicated ABRS who have mild pain and temperature <38.3 °C. Patients who are observed without antibiotic therapy must be reliable and compliant with follow up examination.

• Antibiotics should be started if the patient's condition fails to improve within 7 days or worsens at any time.

Conversely, in patients with more severe symptoms or multiple comorbidities, or in those that cannot be followed up, antibiotics should be prescribed at the outset. Antibiotic therapy should be selected for coverage of the primary organisms associated with acute rhinosinusitis: *Strep pneumoniae*, *H. influenzae*, and *M. catarrhalis*. Resistance patterns as indicated above should be taken in consideration as well. Risk factors for antibiotic resistance include: age <2 or age >65, prior antibiotics received within the previous month, prior hospitalization in the past 5 days, multiple co-morbidities, or immunocompromised status.

IDSA 2012 guidelines for antibiotics in acute sinusitis:

- Amoxicillin-clavulanate as empirical first line therapy in adults and children with severe or worsening symptom of acute sinusitis.
- Macrolides are not recommended due to high rates of resistance among S. pneumonia (30 %).
- TMP/SMX is also not recommended due to high rates of resistance among both S pneumonia and H influenza (30–40 %).
- Second generation oral cephalosporins are not recommended for monotherapy due to variable rates of resistance among S pneumoniae [12].
- In adult patients allergic to penicillin, either doxycycline or a respiratory fluoroquinolone (levofloxacin or moxifloxacin) may be used.
- In children, combination therapy of oral third generation cephalosporin (cefixime or cefpodoxime) and clindamycin is recommended.
- Routine coverage of MRSA is not recommended.
- Recommended treatment duration in uncomplicated ABRS is 5–7 days in adults and 10–14 days in children.

In patients who fail to improve with antimicrobial treatment within 3–5 days or whose symptoms actually worsen after 48–72 h, antimicrobial coverage should be broadened. Endoscopic culture should be pursued to direct more specific antibiotic coverage. Depending on the severity of symptoms and level of clinical suspicion, radiologic imaging should also be considered to rule out suppurative complications.

### **Additional Therapies**

There is level Ia evidence to support treatment of acute rhinosinusitis with intranasal corticosteroids as monotherapy in moderate disease, and as an adjunct to oral antibiotics in severe disease [19]. A recent Cochrane analysis suggests that oral corticosteroids are effective for short term relief of symptoms as an adjunct therapy to oral antibiotics in ARS [20]. A recent Cochrane review found that nasal irrigation with saline has limited benefit in shortening the duration of illness in adults with ARS, although it may be considered for symptomatic relief (level 1a) [21]. There is no evidence to support the use of antihistamines, either oral or intra-nasal, in the treatment ABRS, except in patients with co-existing allergic rhinitis. Also, there is no evidence that the use of nasal or oral decongestants alters the course of ARS, although they may be indicated for alleviating acute symptoms [1, 10].

#### Surgery

There is a limited role for surgery in uncomplicated acute frontal sinusitis. It should be considered only in those patients with severe symptoms not responding to aggressive oral or IV antibiotic therapy, or in whom there is concern for an imminent complication. Endoscopic frontal sinusotomy can be considered, either by traditional frontal recess dissection, or balloon dilation [22]. Frontal recess dissection in the face of acute infection may be especially challenging with extensive mucosal edema, inflammation and bleeding, necessitating advanced skills and experience in these procedures. External drainage via frontal sinus trephination is an alternative option and may be more facile for the less experienced surgeon. Trephination, however, only evacuates the frontal sinus and does not directly address restoration or widening of the natural drainage path of the frontal sinus.

## **Complicated Acute Frontal Sinusitis**

Extrasinus complications from acute bacterial rhinosinusitis are uncommon. The estimated incidence of complications, per one study from the Netherlands, is 1:12,000 for pediatric ABRS and 1:32,000 for adult ABRS [23]. Adolescent and young adult males are significantly more affected than females [24], with a seasonal pattern favoring the winter months [1, 25]. Whereas orbital complications are the most common complications from all forms of ABRS, the vast majority of intracranial complications result from acute frontal sinusitis [23, 26–35]. An epidemiologic study of intracranial complications of ABRS in US children recorded between 2.7 and 4.3 cases per million per year.

Infections can spread from the frontal sinus to intracranial structures, or less commonly to the orbits, by hematogenous or direct routes.

• 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.

Septic thrombophlebitis of the sinus submucosal venous net spreads through the valveless veins into the frontal bone dipole and then to the meningeal veins. These venous channels may be more porous in the developing sinus, and thus adolescents and young adults (especially male) are at increased risk for complications of acute frontal sinusitis. Alternatively, infection can reach the intracranial or orbital structures by erosion of the frontal sinus posterior table or floor, respectively, or through congenital or acquired bony dehiscences.

The workup of the patient with a suspected complication of acute frontal sinusitis includes carefully directed history and exam with specific attention to neurologic and ophthalmologic symptoms and signs. Nasal endoscopy should be performed to culture purulent material that can guide antimicrobial therapy. Lumbar puncture may also be indicated to obtain CSF cultures and to rule out meningitis, but only after exclusion of an abscess using imaging. Consultations with an ophthalmologist, neurosurgeon, neurologist, or infectious disease specialist should be considered.

Whereas radiologic imaging is usually unnecessary in uncomplicated acute frontal sinusitis, radiologic studies play an important role in confirming and characterizing

Table 6.1         Intracranial           complications	Epidural abscess
	Subdural abscesses
	Intraparenchymal brain abscess
	Meningitis
	Encephalitis
	Superior sagittal thrombosis
	Cerebral infarcts
	Cavernous sinus thrombosis

the extent of disease in patients with extrasinus complications. CT scan with intravenous contrast is the imaging modality of choice in evaluating intracranial or orbital complications of acute frontal sinusitis. CT scans can characterize bony erosions of the frontal sinus as well as phlegmons or rim-enhancing fluid collections in adjacent orbital and intracranial soft tissue. Serial imaging studies should be considered in patients who appear clinically unresponsive to initial treatment. MRI may also be useful, being more sensitive than CT in evaluating intracranial pathology, particularly when CT scans are negative or inconclusive in the setting of high suspicion for intracranial complication [36].

## Intracranial Complications

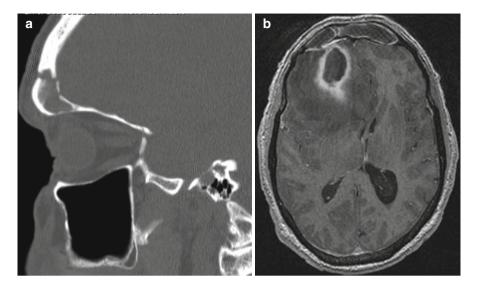
The most common intracranial complications caused by acute frontal sinusitis are epidural and subdural abscesses [23, 26–35]. Table 6.1 lists the range of intracranial complications from acute frontal sinusitis. Figure 6.1 depicts CT and MRI scans of a patient with frontal sinusitis complicated with intracerebral abscess.

Intracranial complications should be suspected when symptoms are protracted or more severe than would be expected for a typical case of acute sinusitis. The most common symptoms are severe frontal headache and fever. Other common warning signs are depicted in Table 6.2. Surprisingly, however, only 50 % of patients who manifest with complicated acute frontal sinusitis experience symptoms of acute sinusitis during the 1–2 weeks prior to presentation. Thirty to 40 % of patients with complicated AFS receive antibiotics in the weeks prior to presentation. The majority do not have a history of previous sinus problems.

Sinus cultures in patients with intracranial complications of acute frontal sinusitis may reveal no growth in up to 25 %. Nonetheless the most common cultured bacteria reported in these cases are Streptococcal species, Staphylococcal species and anaerobes. Gram-negative infections occur less frequently [23, 27, 28, 30, 33, 34]. Table 6.3 lists the most common pathogens.

Because complicated frontal sinusitis can progress rapidly with high morbidity, a high degree of clinical suspicion for potential complications should be maintained during the workup of patients with severe or persistent presentations of acute

#### 6 Acute Frontal Sinusitis



**Fig. 6.1** (a) Non contrast CT scan of a patient with complicated frontal sinusitis showing erosion of both anterior and posterior tables of the frontal sinus. (b) MRI brain T 1 post contrast of the same patient showing right frontal lobe intraparenchymal abscess associated with right frontal lobe epidural enhancement and bilateral frontal sinus mucosal thickening

Severe frontal headache
Altered mental status
Fever >39 °C
Cranial nerve palsy
Hemiparesis
Seizures
Nausea, vomiting
Photophobia
Nuchal rigidity
Forehead swelling
Focal neurologic signs
New onset seizures

**Table 6.2** Warning signs forintracranial complication

rhinosinusitis. Those patients with a confirmed diagnosis of complicated acute frontal sinusitis should be admitted emergently for intravenous antibiotic therapy, intravenous hydration, serial neurologic examination, and consideration for surgical treatment. If cultures can be obtained, these should be performed expeditiously so as to not interfere with the initiation of intravenous antibiotics. If cultures are not possible, empiric antibiotic therapy should be initiated immediately, choosing broad spectrum agents that have favorable penetration of the blood-brain barrier. As mentioned previously, a significant percentage of cultures from patients with intracranial

Table 6.3 Common         pathogens cultured in         intracranial complications         of acute frontal sinusitis	Aerobic bacteria Strep pneumoniae
	Strep milleri/anginosus
	Strep intermedius
	Staphylococcus aureus
	Staph coagulase negative
	Anaerobic bacteria
	Fusobacterium sp.
	Peptostreptococcus
	Prevotella
	Porphyromonas sp.
	Bacteroides sp.
	Propionibacterium acnes

complications are negative. This may perhaps occur because antibiotic therapy is often initiated emergently before cultures can be obtained. The duration of antimicrobial treatment varies with the nature and severity of the complication, as well as the response to initial therapy. Depending on the degree of morbidity, many patients with complicated acute frontal sinusitis 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.

The use of intravenous corticosteroids in patients with complicated AFS is controversial. Some studies 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 [37]. 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.

Surgical treatment should include craniotomy to evacuate any intracranial abscess, and concurrent drainage of the frontal sinus. Methods of draining the frontal sinus include trephination and endoscopic frontal sinusotomy (Draf 2a/Draf 2b). The advantages of trephination include technical simplicity, good efficacy of decompressing and draining the sinus, and provision of a portal to the sinus lumen for irrigation. Disadvantages of trephination include potential scar from the external incision, potential injury to the supraorbital nerve, and failure to address the critical area of impaired outflow of the sinus.

In experienced hands, endoscopic frontal sinusotomy is a satisfactory alternative technique for surgical management of complicated AFS. The endoscopic approach provides a minimally invasive means of improving frontal sinus drainage through its natural outflow tract. Disadvantages of the endoscopic approach include its technical complexity as well as the potential difficulty of obtaining adequate visualization in the acutely infected milieu. In addition, there is a higher risk of post-operative synechia and stenosis of the frontal sinus ostium. Use of silicone stents and creation of Draf 2b cavities has been reported in one study [28] to achieve a low rate of re-stenosis. Balloon dilation techniques may be an appropriate alternative for surgical enhancement of frontal sinus drainage.

#### 6 Acute Frontal Sinusitis

• In recent series, the mortality rate from intracranial complications of frontal sinusitis has been found to have decreased from earlier reports, but remains a notable 5 %.

Furthermore, 15–40 % of patients are reported to have residual neurological sequelae. These include cognitive defects in visual and verbal memory, new onset seizure disorder, cranial nerve palsies, hemiparesis, frontal syndrome and blindness. Patients with neurological deficits at the time of clinical presentation are at much higher risk for late or persistent sequelae compared to patients presenting without neurological symptoms.

## **Orbital Complications**

• Isolated acute frontal sinusitis infrequently causes orbital complications. However, acute frontal sinusitis in the context of pansinusitis is associated with 60–80 % of orbital complications [38, 39].

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. Potential orbital complications include orbital cellulitis, subperiosteal abscess, orbital abscess and cavernous sinus thrombosis. A subperiosteal abscess that is directly associated with frontal sinusitis is typically located supero-laterally within the orbit, displacing the globe medially and inferiorly.

Signs of an orbital complication include periorbital edema/erythema, chemosis, proptosis/globe displacement, double vision and ophthalmoplegia. Diminished visual acuity is a sign of advanced disease. Cranial neuropathies involving 3, 4, V1 and V2 and/or 6 may be associated with cavernous sinus thrombosis. Ophthalmological consultation is a critical part of the workup. CT scan of the sinus and orbits with IV contrast should be obtained to make the diagnosis.

Surgical treatment is indicated in patients not responding to 24–48 h of IV antibiotics or in patients with evidence of reduced visual acuity. Surgical drainage may be performed endoscopically in experienced hands [40], or through an external approach via Lynch incision with or without frontal trephination.

### Frontal Bone Osteomyelitis

Osseous complications of AFS occur in 5-10 % of the cases. Osteomyelitis of the frontal sinus may be caused by direct extension of infection or by thrombophlebitis of the diploic veins. The resulting vascular necrosis caused by frontal sinus osteitis leads to erosion of the anterior table of the frontal sinus, with possible progression to osteomyelitis.

Of the paranasal sinuses, the frontal sinus is most commonly associated with osteomyelitis.

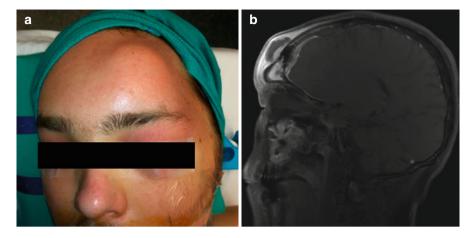


Fig. 6.2 (a) Patient with left forehead Pott's Puffy tumor. (b) MRI brain T 1 post contrast of the same patient showing a subgaleal abscess, left frontal sinus mucosal thickening and inflammation and left frontal lobe epidural enhancement

When osteomyelitis involves the anterior table, a subperiosteal abscess may develop, presenting as a subcutaneous fluctuant protuberance over the brow or forehead (Fig. 6.2). This abscess is known as Pott's Puffy tumor, which was first described by Sir Percival Pott in 1775. Strictly an infectious complication and not neoplastic in any way, Pott's Puffy Tumor may present with severe headache, fever, and photophobia.

Frontal bone osteomyelitis is predominantly observed in adolescents and young adults and is a risk factor for intracranial complications such as subdural empyema and brain abscess, which have been observed in 60-100 % of cases [41]. The most common organisms are streptococci, staphylococci and anaerobic bacteria.

Treatment should include administration of broad spectrum IV antibiotics and early surgical drainage. At a minimum, surgical drainage should include percutaneous drainage of the subperiosteal abscess, as well as drainage of the frontal sinus by either trephination or endoscopic frontal sinusotomy. Debridement of the infected bone may be indicated as well, although studies have shown that percutaneous drainage and repeated antibiotic irrigations through an externally placed drain may be effective and may substitute debridement [42]. In general, intravenous antibiotics are recommended for 4–6 weeks.

# References

- 1. Fokkens WJ, Lund VJ, Mullol J, Bachert C, Alobid I, Baroody F, et al. European position paper on rhinosinusitis and nasal polyps 2012. Rhinol Suppl. 2012;50:1–12, 3 p preceding table of contents, 1–298.
- 2. Fairlie T, Shapiro DJ, Hersh AL, Hicks LA. National trends in visit rates and antibiotic prescribing for adults with acute sinusitis. Arch Intern Med. 2012;172(19):1513–4.

#### 6 Acute Frontal Sinusitis

- Mattos JL, Woodard CR, Payne SC. Trends in common rhinologic illnesses: analysis of U.S. healthcare surveys 1995–2007. Int Forum Allergy Rhinol. 2011;1(1):3–12.
- 4. Eloy P, Poirrier AL, De Dorlodot C, Van Zele T, Watelet JB, Bertrand B. Actual concepts in rhinosinusitis: a review of clinical presentations, inflammatory pathways, cytokine profiles, remodeling, and management. Curr Allergy Asthma Rep. 2011;11(2):146–62.
- 5. Lieu JE, Feinstein AR. Confirmations and surprises in the association of tobacco use with sinusitis. Arch Otolaryngol Head Neck Surg. 2000;126(8):940–6.
- 6. Savolainen S. Allergy in patients with acute maxillary sinusitis. Allergy. 1989;44(2):116-22.
- 7. Schatz M, Zeiger RS, Chen W, Yang SJ, Corrao MA, Quinn VP. The burden of rhinitis in a managed care organization. Ann Allergy Asthma Immunol. 2008;101(3):240–7.
- Kuhn FA. Surgery of the frontal sinus. In: Kennedy DW, Bolger WE, Zinreich SJ, editors. Diseases of the sinuses: Diagnosis and Management. Hamilton, Ontario: BC Decker Inc.; 2001. p. 281–301.
- Lien CF, Weng HH, Chang YC, Lin YC, Wang WH. Computed tomographic analysis of frontal recess anatomy and its effect on the development of frontal sinusitis. Laryngoscope. 2010;120(12):2521–7.
- Rosenfeld RM, Andes D, Bhattacharyya N, Cheung D, Eisenberg S, Ganiats TG, et al. Clinical practice guideline: adult sinusitis. Otolaryngol Head Neck Surg. 2007;137(3 Suppl):S1–31.
- 11. Hwang PH. A 51-year-old woman with acute onset of facial pressure, rhinorrhea, and tooth pain: review of acute rhinosinusitis. JAMA. 2009;301(17):1798–807.
- Chow AW, Benninger MS, Brook I, Brozek JL, Goldstein EJ, Hicks LA, et al. IDSA clinical practice guideline for acute bacterial rhinosinusitis in children and adults. Clin Infect Dis. 2012;54(8):e72–112.
- 13. Seiden AM, Martin VT. Headache and the frontal sinus. Otolaryngol Clin North Am. 2001;34(1):227–41.
- 14. Brook I. Microbiology of sinusitis. Proc Am Thorac Soc. 2011;8(1):90-100.
- Benninger MS, Payne SC, Ferguson BJ, Hadley JA, Ahmad N. Endoscopically directed middle meatal cultures versus maxillary sinus taps in acute bacterial maxillary rhinosinusitis: a meta-analysis. Otolaryngol Head Neck Surg. 2006;134(1):3–9.
- Antila J, Suonpaa J, Lehtonen OP. Bacteriological evaluation of 194 adult patients with acute frontal sinusitis and findings of simultaneous maxillary sinusitis. Acta Otolaryngol Suppl. 1997;529:162–4.
- 17. Brook I. Bacteriology of acute and chronic frontal sinusitis. Arch Otolaryngol Head Neck Surg. 2002;128(5):583–5.
- 18. Brook I. Acute and chronic frontal sinusitis. Curr Opin Pulm Med. 2003;9(3):171-4.
- Zalmanovici A, Yaphe J. Intranasal steroids for acute sinusitis. Cochrane Database Syst Rev. 2009;4:CD005149.
- Venekamp RP, Thompson MJ, Hayward G, Heneghan CJ, Del Mar CB, Perera R, et al. Systemic corticosteroids for acute sinusitis. Cochrane Database Syst Rev. 2011;12:CD008115.
- Kassel JC, King D, Spurling GK. Saline nasal irrigation for acute upper respiratory tract infections. Cochrane Database Syst Rev. 2010;3:CD006821.
- Hopkins C, Noon E, Roberts D. Balloon sinuplasty in acute frontal sinusitis. Rhinology. 2009;47(4):375–8.
- Hansen FS, Hoffmans R, Georgalas C, Fokkens WJ. Complications of acute rhinosinusitis in The Netherlands. Fam Pract. 2012;29(2):147–53.
- 24. Piatt Jr JH. Intracranial suppuration complicating sinusitis among children: an epidemiological and clinical study. J Neurosurg Pediatr. 2011;7(6):567–74.
- Oxford LE, McClay J. Complications of acute sinusitis in children. Otolaryngol Head Neck Surg. 2005;133(1):32–7.
- Bair-Merritt MH, Shah SS, Zaoutis TE, Bell LM, Feudtner C. Suppurative intracranial complications of sinusitis in previously healthy children. Pediatr Infect Dis J. 2005;24(4):384–6.
- Bayonne E, Kania R, Tran P, Huy B, Herman P. Intracranial complications of rhinosinusitis. A review, typical imaging data and algorithm of management. Rhinology. 2009;47(1):59–65.

- Betz CS, Issing W, Matschke J, Kremer A, Uhl E, Leunig A. Complications of acute frontal sinusitis: a retrospective study. Eur Arch Otorhinolaryngol. 2008;265(1):63–72.
- 29. DelGaudio JM, Evans SH, Sobol SE, Parikh SL. Intracranial complications of sinusitis: what is the role of endoscopic sinus surgery in the acute setting. Am J Otolaryngol. 2010; 31(1):25–8.
- Germiller JA, Monin DL, Sparano AM, Tom LW. Intracranial complications of sinusitis in children and adolescents and their outcomes. Arch Otolaryngol Head Neck Surg. 2006; 132(9):969–76.
- Glickstein JS, Chandra RK, Thompson JW. Intracranial complications of pediatric sinusitis. Otolaryngol Head Neck Surg. 2006;134(5):733–6.
- 32. Hakim HE, Malik AC, Aronyk K, Ledi E, Bhargava R. The prevalence of intracranial complications in pediatric frontal sinusitis. Int J Pediatr Otorhinolaryngol. 2006;70(8):1383–7.
- Hicks CW, Weber JG, Reid JR, Moodley M. Identifying and managing intracranial complications of sinusitis in children: a retrospective series. Pediatr Infect Dis J. 2011;30(3):222–6.
- Kombogiorgas D, Seth R, Athwal R, Modha J, Singh J. Suppurative intracranial complications of sinusitis in adolescence. Single institute experience and review of literature. Br J Neurosurg. 2007;21(6):603–9.
- Leotta N, Chaseling R, Duncan G, Isaacs D. Intracranial suppuration. J Paediatr Child Health. 2005;41(9–10):508–12.
- 36. Blumfield E, Misra M. Pott's puffy tumor, intracranial, and orbital complications as the initial presentation of sinusitis in healthy adolescents, a case series. Emerg Radiol. 2011; 18(3):203–10.
- Clayman GL, Adams GL, Paugh DR, Koopmann Jr CF. Intracranial complications of paranasal sinusitis: a combined institutional review. Laryngoscope. 1991;101(3):234–9.
- Jackson K, Baker SR. Clinical implications of orbital cellulitis. Laryngoscope. 1986; 96(5):568–74.
- Schramm Jr VL, Curtin HD, Kennerdell JS. Evaluation of orbital cellulitis and results of treatment. Laryngoscope. 1982;92(7 Pt 1):732–8.
- Roithmann R, Uren B, Pater J, Wormald PJ. Endoscopic drainage of a superiorly based subperiosteal orbital abscess. Laryngoscope. 2008;118(1):162–4.
- 41. Ketenci I, Unlu Y, Tucer B, Vural A. The Pott's puffy tumor: a dangerous sign for intracranial complications. Eur Arch Otorhinolaryngol. 2011;268(12):1755–63.
- 42. Akiyama K, Karaki M, Mori N. Evaluation of adult Pott's puffy tumor: our five cases and 27 literature cases. Laryngoscope. 2012;122(11):2382–8.

# **Chapter 7 Chronic Frontal Rhinosinusitis: Diagnosis and Management**

Artur Gevorgyan and Wytske J. Fokkens

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<sup>©</sup> Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_7

#### **Core Messages**

- Frontal rhinosinusitis is the most challenging to treat given its dependence on the health of other sinuses and need for demanding surgery.
- Rhinosinusitis, including frontal sinusitis, is diagnosed based on a combination of symptoms, endoscopic and radiographic features. It is classified into acute or chronic depending on duration. Chronic rhinosinusitis is further subclassified into with nasal polyps (CRSwNP) and without nasal polyps (CRSsNP).
- Medical treatment pre- and post-operatively has to decrease the inflammatory reaction. Surgery is an integral part of rhinosinusitis management. Functional drainage of the frontal sinus relies on preservation of the mucosa of the frontal recess. Knowledge of frontal sinus anatomy is paramount before attempting to surgically treat frontal rhinosinusitis. Iatrogenic damage to the frontal recess is a key factor to avoid in frontal sinus surgery.
- An integrated surgical approach is recommended, with escalation of extent of surgery depending on disease amount, failure of previous procedures and surgeon's comfort.

# Introduction

Chronic frontal rhinosinusitis represents the presence of inflammatory disease in the frontal sinus. Given the location and anatomic variations of the frontal recess, frontal rhinosinusitis can frequently be a result of iatrogenic or traumatic closure of the frontal recess. The frontal sinus remains the most difficult sinus to treat given the difficulty of its examination and its dependence on ostiomeatal complex health. Nevertheless, many concepts around diagnosis and management of chronic frontal rhinosinusitis are common to all sinuses.

In this chapter, we will discuss the definitions, classification, diagnosis and management of chronic rhinosinusitis (CRS) in general, where necessary emphasizing the peculiarities specific to the frontal sinuses.

# **Classification and Definitions**

The last decade has seen the appearance of at least three major clinical practice guidelines on rhinosinusitis [8, 13, 32]. These guidelines have been useful in systematizing the approach to classification and management of CRS, significantly improving the quality of research.

For the purposes of this chapter, we will guide ourselves by the European Position Paper on Rhinosinusitis and Nasal Polyps 2012 [13]. This international effort has attempted to summarize the current knowledge on all aspects of epidemiology, pathogenesis, diagnosis and management of acute and chronic rhinosinusitis. A more concise version is available for the daily use by the practicing otolaryngologist [12, 13].

In *adults*, rhinosinusitis (with or without nasal polyps) is defined as inflammation of the nose and the paranasal sinuses, characterized by *two or more symptoms*, one of which should be either:

- Nasal blockage/obstruction/congestion or nasal discharge (anterior/posterior nasal drip)
- ± Facial pain/pressure
- ± Reduction or loss of smell

#### and either

- Endoscopic signs of:
  - Nasal polyps, and/or
  - Mucopurulent discharge primarily from middle meatus, and/or
  - Edema/mucosal obstruction primarily in middle meatus

and/or

- CT changes:
  - Mucosal changes within the ostiomeatal complex and/or sinuses

In *children*, rhinosinusitis is defined similarly, with a small difference in symptom presentation: instead of *reduction or loss of smell* children more frequently report *cough* (Chap. 16).

Based on duration of symptoms, rhinosinusitis can be classified into:

- *Acute*: lasting for <12 weeks with complete resolution of symptoms (Chap. 6)
- *Chronic*: lasting for  $\geq 12$  weeks without complete resolution of symptoms.

Chronic rhinosinusitis may also be subject to exacerbations.

It is important to document disease severity, for which the use of a 10 cm visual analogue scale (VAS) is recommended.

Measurement of disease severity on a VAS scale:

- *Mild* (0–3)
- *Moderate* (>3–7)
- Severe (>7–10)

A VAS score of >5 affects the patient's QOL.

Based on presence or absence of nasal polyps, CRS is further subclassified.

Classification based on presence of polyps:

- *CRS with nasal polyps* (*CRSwNP*): bilateral polyps, endoscopically visualized in middle meatus
- *CRS without nasal polyps (CRSsNP)*: no visible polyps in middle meatus, if necessary following a decongestant

Special types of chronic rhinosinusitis include:

- Aspirin exacerbated respiratory disease
- Ciliary dysmotility disorders
- CRS due to immune deficiencies
- CRS in cystic fibrosis
- Fungal rhinosinusitis (Chap. 11)
  - Allergic fungal rhinosinusitis
  - Mycetoma (fungal ball)
  - Acute invasive fungal rhinosinusitis
  - Chronic invasive fungal rhinosinusitis

# Epidemiology

The exact prevalence of CRS with and without polyps remains unknown in many countries. General practitioners are most likely to see most of the cases of mild and moderate severity CRS. The reported rates of CRS are within the range of 5-15% [5, 28, 38], whereas that of physician-diagnosed CRS between 1.01 and 9.6% of the general population [2, 7, 14], and the rates of physician-diagnosed CRSwNP between 2 and 4% [13, 16].

Confirmed factors	Presumed factors
Aspirin sensitivity	Allergy
Asthma	Anatomic factors (e.g. septal deviation, concha bullosa, displaced uncinate process, etc.)
Biofilms	Genetic factors
Ciliary dysmotility disorders	H. pylori
Cystic fibrosis (CF)	Pregnancy
Iatrogenic damage	Reflux
Immune deficiency	Environmental factors
Smoking	
Trauma	

Table 7.1 Factors implicated in CRS etiopathogenesis

CRSwNP has been reported in all races with a prevalence of 0.5–4.2 % of the population [17, 26]. Nasal polyps are rare under the age of 20 and appear at an average age of 42 years, lagging behind adult onset asthma by 7 years [13].

## **Etiology and Pathogenesis**

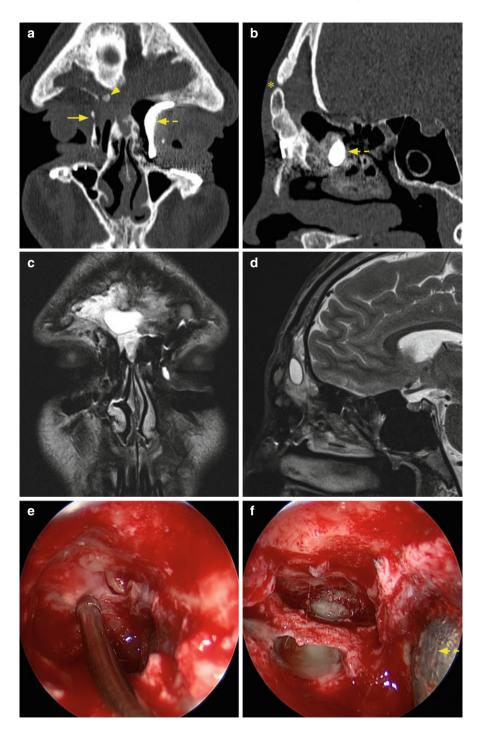
Multiple factors have been associated with the development of CRS. Despite individual correlations in clinical practice seem to be obvious, epidemiologic evidence lacks for several of the suggested factors (Table 7.1) [13]. For a complete review CRS etiopathogenesis, the reader is referred to the EPOS 2012 document [13].

Introgenic damage to frontal recess during previous surgery plays an important role in the pathogenesis of frontal rhinosinusitis in some cases. This will be further elucidated in the treatment section.

Trauma is another important consideration as an etiologic factor specifically in *frontal sinuses* (Fig. 7.1). Isolated frontal or panfacial trauma with obstruction of the frontal recess can lead to isolated frontal rhinosinusitis soon after the trauma, or mucocele formation many years after the injury.

## Diagnosis

The diagnosis of CRS is based on thorough history of symptoms, endoscopic examination and appropriate diagnostic imaging.



# History

#### Symptoms

The most common symptoms in CRS are:

- Nasal blockage, congestion or stuffiness;
- Nasal discharge or postnasal drip, often mucopurulent;
- Facial pain or pressure, headache, and
- Reduction/loss of smell.

CRSsNP is more frequently associated with facial pain, pressure or fullness, whereas CRSwNP is associated with hyposmia or anosmia.

Frontal rhinosinusitis is commonly associated with unilateral, bifrontal or periorbital pressure or pain. Of importance is distinguishing symptoms of frontal rhinosinusitis from headaches and migraine (Chap. 12).

It is important to ask the patient which of the symptoms are considered the most bothersome, and to elicit their onset, severity, character, duration and frequency, as well as any precipitating or palliating factors, including any previous treatment.

A useful way of documenting patient's complaints is the use of patient-reported outcome measures (PROMs). Sinonasal outcome test-22 (SNOT-22) and rhinosinusitis outcome measure (RSOM-31) are especially useful in this setting [9]. When completed by the patient prior to seeing the physician, these questionnaires provide a quick overview of symptoms and allow focusing on particularly troublesome ones. It also allows tracing patient progress over time, especially after initiating medical therapy or after surgery [19]. Documentation of individual symptom severity with a VAS score can also be useful both clinically and for research purposes [13].

Distant symptoms of CRS include pharyngeal, laryngeal and tracheal irritation causing sore throat, halitosis, dysphonia and cough. Patients with CRSwNP commonly mouth-breathe if polyps are obstructive. They may also complain of otologic

**Fig. 7.1** An example of severe traumatic frontal sinusitis. This patient sustained a panfacial trauma as a result of a motorcycle accident. He underwent an open reduction and internal fixation of facial fractures, and left medical orbital wall reconstruction with a titanium plate. Within a year, he developed frontal sinusitis and underwent external drainage at a peripheral hospital. The patient later developed a frontal mucocele due to scaring and obstruction of the frontal recess bilaterally by displaced bone and reconstruction plate, necessitating a Draf 3 approach to mucocele drainage. Coronal (**a**) and sagittal (**b**) CT scan images demonstrating displaced right lamina papyracea (*arrow*), titanium plate (*broken arrow*) along the left medial orbital wall and bony particles (*arrow head*) within the frontal sinus cavity. On sagittal images, the path (*asterisk*) of the previous external approach to frontal sinus drainage can be seen. Coronal (**c**) and sagittal (**d**) T2-weighted MRI images of the same patient demonstrating a frontal mucocele. (**e**, **f**) Intraoperative images of this patient before (**e**) and after (**f**) opening of the frontal mucocele. The titanium plate (*broken arrow*) can be seen, along with significant scarring of the entire frontal sinus

symptoms due to concurrent Eustachian tube dysfunction with ear fullness, otalgia and hearing loss. Occasionally, disease of maxillary teeth can lead to unilateral CRS, therefore questions about loose teeth and dental disease should be within the scope of history. CRS may also be associated with general symptoms of drowsiness, malaise, fever and sleep disturbance.

When suspecting CRS complications, one must inquire about ophthalmic (vision change, diplopia, visual field loss, epiphora, external swelling) and neurologic (headache, seizures, motor and sensory disturbance, especially of cranial nerves) complaints.

#### **Risk Factors**

During history taking, it is important to ask about factors that can predispose to CRS development (Table 7.1). For the frontal sinuses, this especially relates to previous surgery and trauma.

#### **Medical History**

Key questions about past and current health are important. It is paramount to inquiry about *respiratory health*, specifically cystic fibrosis (CF), asthma, COPD, bronchiectasis, recurrent pneumonias and lung arteriovenous malformations, as dictated by history, because lower respiratory problems often accompany CRS. Concurrent asthma and CRS, and especially aspirin exacerbated respiratory disease, are especially challenging to treat, and require expertise and close collaboration with a pulmonologist.

Other important aspects of general health inquiry include *autoimmune disorders*, including granulomatosis with polyangiitis (Wegener's granulomatosis), eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome), sarcoidosis, and *immune abnormalities* (acquired or inherited immune deficiencies, hematogenous malignancies, treatment with corticosteroids or chemotherapy, severe diabetes).

Finally, in patients considered for surgery, it is critical to document *cardiac health* and *bleeding status* (known hematologic disorders, previous severe bleeding during surgery, easy bruising, use of anticoagulants or antiplatelets agents).

#### **Surgical History**

Clear documentation of previous endonasal surgery is critical, as it may allow avoiding and anticipating complications. Surgery of the sinuses, septum, turbinates, tumors, as well as external nasal and nasal valve surgeries are important to know about as these may point to misdiagnosis, be the sole cause of iatrogenic rhinosinusitis, or warn about difficult to treat disease. A note of previous tolerance of general anesthesia by patient and family is also important.

#### Medications

Specific attention should be paid to previous medical treatment of rhinosinusitis, including antibiotic type, duration and success, intranasal or systemic corticosteroids, and nasal lavage. In patients with coexistent allergy, documentation of allergy treatment with antihistamines, decongestants, antileukotrienes and anti-IgE monoclonal antibodies is important.

Known allergies to medications and other healthcare products, as well as environmental allergies should be documented.

#### **Social History**

*Smoking* is known to exacerbate respiratory disease, thus smoking history and current habits and exposure to second hand smoke is important to elucidate. The patient should be encouraged to quit and provided resources to assist in this.

Significant *alcohol* intake, even though not directly associated with CRS, may be associated with generally poor health and is important to know prior to surgery.

*Cocaine* abuse may cause significant nasal dysfunction, leading to septal perforation. If possible, surgery should be delayed until the patient has stopped any illicit drug abuse.

Chemical irritants, encountered by patients in certain *occupations*, can be a cause of non-allergic rhinitis, which requires concurrent treatment with CRS. In addition, occupational exposure to irritants may be a risk factor for the occurrence of CRS, as evidenced by increased need for revision sinus surgery [20].

## Examination

#### **Anterior Rhinoscopy**

Anterior rhinoscopy is useful in the primary practice, and may reveal nasal polyps, mucosal edema, mucopurulent discharge, turbinate enlargement or septal deviations.

#### Nasal Endoscopy

In a specialty clinic, nasal endoscopy is the mainstay of rhinologic examination. We use a  $30^{\circ}$  2.7 mm, 18-mm-long endoscope. The small diameter of this endoscope allows for examination without the use of topical anesthetic, which may irritate the

mucosa and cause clear discharge, sneezing and decrease compliance with the examination. Despite the small size, this endoscope provides enough light for thorough examination of all sinuses. A three-pass technique is used to examine the inferior meatus towards the nasopharynx, above the inferior turbinate towards the sphenoethmoidal recess, and into the middle meatus.

The  $30^{\circ}$  angulation of the endoscope allows for ease of examination of the middle meatus and maxillary sinus after surgery, as well as the frontal recess and frontal neo-ostium after frontal sinus surgery. It also does not impede examination of the inferior turbinates and the nasal floor.

Examination of the frontal sinus is probably the most difficult. It requires the body of the sinus scope being positioned at the sill of the nostril, while aiming the tip of it superiorly towards and if possible into the middle meatus. A careful angulation superiorly will allow the examiner to thoroughly visualize the anterior ethmoid cells and the frontal sinus recess. Employing a similar approach, the Draf 3 neoostium can be visualized, though this is much easier accomplished, given its more anterior location.

Endoscopic confirmation of CRS is supported by findings of:

- Nasal polyps, and/or
- · Mucopurulent discharge primarily from middle meatus, and/or
- Edema/mucosal obstruction primarily in the middle meatus.

Other points of examination:

- · Rest of otolaryngologic regions, as necessary
- · Cranial nerves, if deficits are suspected
- Visual acuity, extraocular muscle movement, gross visual fields, color perception, as required.

# **Diagnostic Imaging**

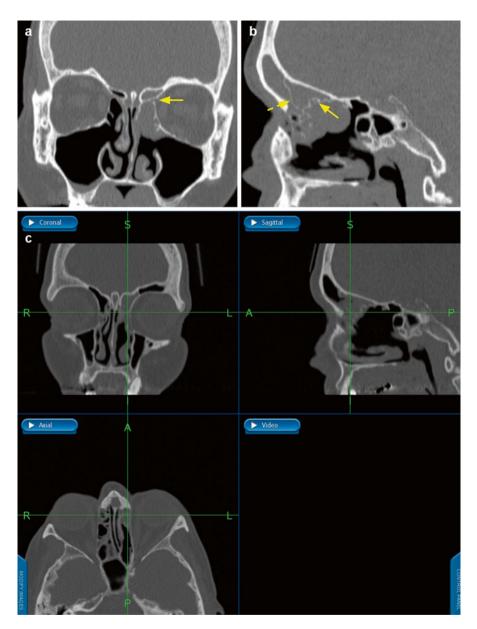
#### **Computer Tomography (CT)**

Sinus CT scan with reconstructions in coronal and sagittal planes is the gold standard in radiologic CRS confirmation. It is especially useful if the format of the diagnostic CT scan allows its direct use in image-guided equipment. Standard 0.7 or 1 mm sinus CT cuts at our institution have proven ideal in this setting.

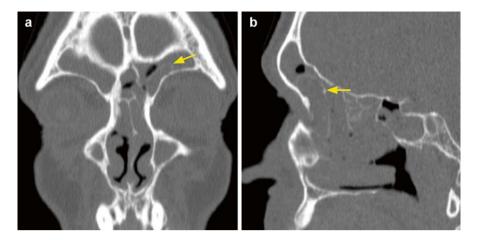
The CT changes, qualifying for CRS, include mucosal changes within the ostiomeatal complex and/or sinuses. Disease severity can be graded with the Lund-Mackay score [25]. Full opacification usually denotes lack of aeration of the sinus. Incomplete opacification implies a frontal recess, which is still functioning in the presence of significant disease in the frontal sinus or recess itself. The combination of patient complaints with documented endoscopic or CT abnormalities will lead to the choice of correct surgical procedure.

<b>Table 7.2</b> Anatomic features         on CT important in frontal         sinus surgery	Agger nasi position and size (coronal and sagittal images)
	Presence of frontoethmoidal Kuhn cells (coronal and sagittal images):
	Type I: single frontal recess cell above agger cell, below frontal sinus
	Type II: more than 1 cells in frontal recess, above agger cell, below frontal sinus
	Type III: large single cell pneumatizing cephalad into frontal recess
	Type IV: single isolated cell within the frontal
	Presence and variation of bullar cells (sagittal images)
	Suprabullar cell
	Frontal bullar cell
	Presence of midline pneumatization (coronal and sagittal images):
	Frontal intersinus septal cell
	Pneumatized crista galli
	Pathway of frontal recess drainage (axial images followed in a superior to inferior direction)
	Posterior table dehiscence (sagittal)
	Nasal beak size (sagittal)
	Anterior-posterior diameter of frontal sinus (mm) (sagittal):
	With beak intact (in mm)
	Anticipated size after Draf 3 (in mm)
	Lamina papyracea dehiscence (coronal)
	Orbital roof dehiscence (coronal)
	Anterior ethmoid artery (pedicled or in skull base) (coronal)
	Supraorbital ethmoid cells in relation to anterior ethmoid artery (coronal)
	Skull base (intact or dehiscent) (coronal)
	Keros classification of lateral lamella height (depth of olfactory fossa) (coronal):
	Type I – olfactory fossa 1–3 mm deep
	Type II – 4–7 mm
	Type III – 8–16 mm
	Type IV – >16 mm or asymmetric

Frontal sinus surgery is never a routine. Close examination of the bony windows allows for appreciation of general characteristics, anatomic abnormalities and extent of disease. Of vital importance is the knowledge of the frontal recess anatomy (Table 7.2). Multiple configurations of cells around the recess make frontal sinus surgery most challenging and at the same time most exciting [23, 36]. A particular mistake can happen in the presence of a large frontal bullar cell



**Fig. 7.2** An example of a large frontal bullar cell and a low course of anterior ethmoid artery (AEA). This patient had a previous attempt of Draf 2a frontal sinusotomy, which was abandoned due to the proximity of a hanging left AEA. This patient was treated with revision Draf 2a frontal sinusotomy, and the frontal bullar cell was safely removed with no compromise of the AEA. (a) Coronal CT image demonstrating the low course of the AEA (*arrow*) from the skull base. (b) Sagittal CT image demonstrating the AEA (*arrow*), as well as a large frontal bullar cell (*broken arrow*), which is obstructing the frontal recess anteriorly and forms a part of the bony canal of the AEA. (c) Image guided navigation images demonstrate the location of the safe entry into the frontal bullar cell



**Fig. 7.3** An example of a large type III Kuhn cell pneumatizing into the left frontal sinus. Coronal (a) and sagittal (b) CT images are shown with the arrow pointing at the type III Kuhn cell

(Fig. 7.2) or a large Kuhn cell pneumatizing cephalad (Fig. 7.3). Once the cell is opened, it may trick the surgeon to believe that one has opened the frontal sinus itself. A thorough understanding of anatomy, aided with three-dimensional reconstruction (e.g. the building block concept of Wormald) will aid the surgeon to be confident when opening frontal recess cells and directing the dissection along the correct path [41].

When there is a suspicion for sinonasal, orbital or intracranial tumors with spread towards the nose, a sinus CT with contrast can be obtained, though MRI is more useful. It is also useful in case of intraorbital or intracranial complications, in which case a contrasted CT scan is required to differentiate an abscess (hypodense collection with a surrounding enhancing ring) from cellulitis.

### Magnetic Resonance Imaging (MRI)

MRI of sinuses can be useful in several pathologies. Benign and malignant tumors of the nose are the most frequent reasons to request a sinus MRI. In CRS, MRI may help differentiate benign polyps from inverted papilloma in cases when the latter is bilateral and suspicious. Inverted papilloma is characterized by intermediate intensity on T2-weighted images, with characteristic cerebriform appearance, while the surrounding inflammation, edema and retained secretions have high signal intensity (Fig. 7.4). On T1-weighted MRI with contrast, the tumor will also have intermediate intensity, while secretions will not enhance, but the inflamed mucosa will have a high signal.

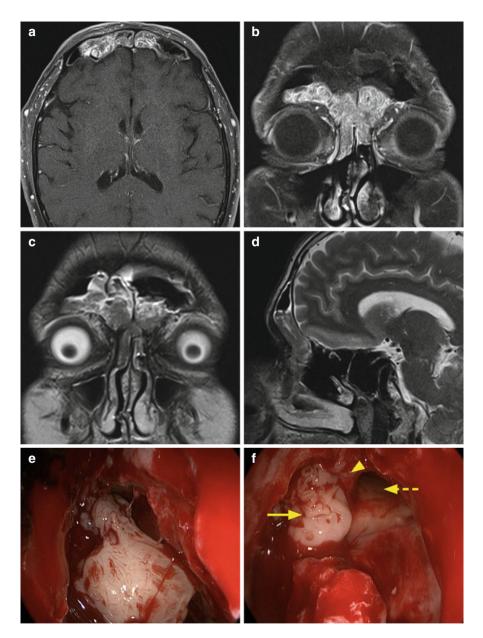


Fig. 7.4 MRI images of a large bilateral frontal inverted papilloma with characteristic cerebriform appearance: (a) T1-weighted axial images with contrast. (b) T1-weighted coronal images with contrast. (c) T2-weighted coronal images without contrast. (d) T2-weighted sagittal images without contrast. (e) Intraoperative image of a portion of inverted papilloma removed from the left frontal sinus. (f) Intraoperative image of a remainder of the inverted papilloma filling the right frontal sinus (*arrow*), while the left one is free of disease (*broken arrow*), where the tumor extended, but did not attach. The frontal intersinus septum is preserved (*arrow head*)

MRI is also useful in assessing mucoceles (Fig. 7.5). These can also be visualized on CT imaging in the form of opacification with characteristic rounded pushing borders and frequent bony dehiscence. On T2-weighted MRI, mucoceles, most of which have low protein content, have a bright intensity, while they appear hypointense on T1-weighted images. However, if the content of protein in the mucocele increases, they can also appear hypointense on T2-weighted images.

### **Functional Investigations**

#### **Olfactory Testing**

Olfaction is very frequently affected in CRS patients, especially in those with polyps. Routine testing of olfaction in CRS patients allows for documentation of baseline and improvement with treatment.

Psychophysical olfactory tests (e.g. University of Pennsylvania Smell Identification Test (UPSIT) and Sniffin' Sticks) are self-administered: the patient smells an odorant and chooses an answer from a multiple-choice list [10, 21]. They allow for testing the olfactory threshold and comparing it with established age and gender norms, as well as detection of malingering.

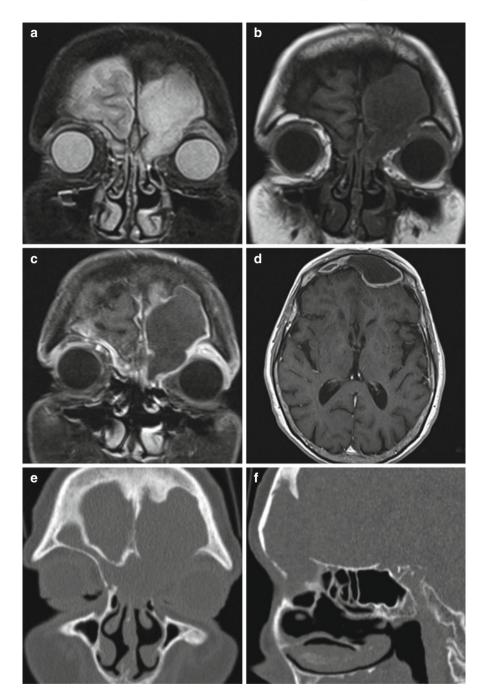
Electrophysiological tests are still considered research tools. Odor event-related potentials measure brain waves in response to odors, while electro-olfactogram measures the action potential directly on the olfactory mucosa.

#### Nasal Airflow and Resistance

Several methods are available to test nasal airflow, resistance, and site and size of obstruction. These include rhinomanometry (anterior and posterior), acoustic rhinometry and peak nasal inspiratory flow (PNIF) testing [33]. Most of these are still considered research tools, as correlation between these objective measures and patient reported outcomes measures has been difficult to establish [9]. However, we find PNIF an excellent clinical tool to objectify patients' complains, especially in situations where history and findings do not correlate [27].

#### Allergy Testing

Despite the role of allergy as a factor in CRS remains to be further investigated, allergy is certainly found in many patients with CRS. Up to 54 % of outpatients with CRS have been found to have a positive skin prick test (SPT) [3]. The prevalence of allergy in patients with CRSwNP has been reported been 10–64 % [13].



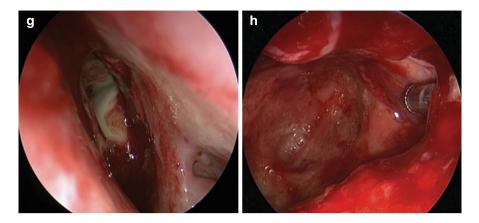


Fig. 7.5 (continued)

SPT to a panel of aeroallergens is the most widely available method for allergy testing, using purified allergens, and positive (histamine) and negative (saline) controls. Scoring of positive reactions is carried out according to Global Allergy and Asthma European Network (GA<sup>2</sup>LEN) and Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines [6].

Detection of allergen-specific serum immunoglobulin E (IgE) can be performed with radioallergosorbent test (RAST) and enzyme-linked immunosorbent assays (ELISA) [33]. IgE testing is usually as sensitive as SPT testing, and can be employed when the latter testing is negative.

Once positive reactions are identified, they are correlated with history and symptoms. A clinically important positive SPT can guide the physician to treat the allergic component along with co-existing CRS.

**Fig. 7.5** MRI images of a large left frontal mucocele displacing the orbit: (**a**) T2-weighted coronal images: the mucocele appears as a hyperintense collection with left orbital displacement, and is white due to high fluid content. (**b**) T1-weighted coronal images: iso- to hypointense appearing mucocele. On T1-weighted coronal (**c**) and axial (**d**) images with contrast the mucocele has enhancing borders. (**e**) Coronal CT images of the same patient demonstrating destruction of the lamina papyracea and the orbital roof. (**f**) Sagittal CT images demonstrate destruction of the posterior table of the frontal sinus by the expanding mucocele. (**g**) Intraoperative image of pus draining from the opened frontal mucocele cavity. (**h**) Auto-Draf 3 cavity, produced by an enlarging mucocele: most of the bone, usually removed by the Draf 3 procedure, was destroyed by the mucocele

#### **Evaluation of Mucociliary Clearance**

Mucociliary clearance is evaluated when there is a suspicion for ciliary disorders resulting in CRS, e.g. primary ciliary dyskinesia (PCD) and CF [33]. *Saccharine test* is an example of *mucociliary clearance time test*, which evaluates the speed with which the particles of saccharine, placed on the inferior turbinate, reach to the pharynx resulting in sweet sensation. The same test can be carried out with a dye (e.g. Evan's blue) or radiolabeled substances. Normal mucociliary clearance time is under 20 min.

*Electronic microscopy (EM)* is used to evaluate the structure of cilia and abnormalities of dynein arms. Mucosal cells for EM are obtained by scraping along inferior and middle turbinates.

*Ciliary beat frequency* can be evaluated when the harvested cells are investigated under polarized light microscope with the use of digital high-speed video imaging and computer software.

Harvested mucosa can also be cultured to investigate *ciliogenesis* in vitro. This technique, however, requires several weeks before the culture can be examined under EM.

#### **Other Nasal Tests**

Several other tests are currently being extensively studied, however have not yet found broad clinical applicability. These include nasal provocation tests, sampling of nasal secretions, cytology and histology, as well as nasal nitric oxide measurement.

# Bacteriology (See Chap. 4: Microbiology of Chronic Sinusitis)

#### **Biopsy**

Biopsy of the nasal mucosa is usually employed when there is a suspicion for benign or malignant tumors, or when the diagnosis of rhinosinusitis is not straightforward. It is imperative to obtain imaging prior to biopsy of intranasal lesions, to rule out communication with the brain (meningoceles, meningoencephaloceles) or a vascular lesion (angiofibroma, angiosarcoma, hemangiopericytoma, vascular malformation). A biopsy in the case of resistant to treat rhinosinusitis and especially destructive processes can help narrow the differential (Table 7.3).

#### **Blood and Other Tests**

When there is a suspicion for unorthodox cause of rhinosinusitis, blood and other tests can be used to investigate the plausible cause. Table 7.4 below presents a list of differential diagnoses in rhinosinusitis and respective tests.

Table 7.3       Differential         diagnoses in rhinosinusitis,         which can be diagnosed with         biopsy	Granulomatosis with polyangiitis (Wegener's granulomatosis)
	Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)
	Sarcoidosis
	Benign and malignant sinonasal tumors
	Lymphoma

Table 7.4 Blood and other tests important in investigating differential diagnoses in CRS

Test	Diagnostic association
ESR, CRP	Systemic, infectious and autoimmune disorders
ANA	Autoimmune disorders
Anti-Ro (SS-A), Anti-La (SS-B)	Sjogren disease
c-ANCA	Granulomatosis with polyangiitis (Wegener's granulomatosis)
p-ANCA	Eosinophilic granulomatosis with polyangiitis (Churg- Strauss syndrome)
ACE	Sarcoidosis
Calcium	Sarcoidosis, cancer
Sweat chloride	CF
CFTR gene mutation	CF
β2-transferrin	CSF leak
β trace protein	CSF leak
Eosinophilia	Allergy, eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)
βhCG	Pregnancy
TSH, T3, T4	Hypothyroidism
CBC	Infection, hematologic malignancies
Immune panel: total Ig, IgG subclasses, HIV	Immune deficiency
Aspirin challenge	Aspirin sensitivity
FTA-ABS	Syphilis
Mantoux tuberculin skin test	Tuberculosis
Biopsy, serology	Leprosy
Urine and hair testing	Cocaine abuse

ANA antinuclear antibody, ACE angiotensin-converting enzyme,  $\beta hCG$  beta human chorionic gonadotropin, *c*-ANCA cytoplasmic antineutrophil cytoplasmic antibodies, CBC complete blood count, CF cystic fibrosis, CFTR cystic fibrosis transmembrane conductance regulator, CRP C-reactive protein, CRS chronic rhinosinusitis, CSF cerebrospinal fluid, ESR erythrocyte sedimentation rate, FTA-Abs fluorescent treponemal antibody absorption, HIV human immunodeficiency virus, Ig immunoglobulin, p-ANCA Perinuclear Anti-Neutrophil Cytoplasmic Antibodies, TSH thyroid-stimulating hormone, T3 triiodothyronine, T4 thyroxine

Allergic rhinitis	
Non-allergic rhinitis	
Anatomic abnormalities of the septum and turbinates	
Degenerative disorders, e.g. in Parkinson's disease and Alzheimer's disease, when h he presenting symptom	yposmia is
Congenital (antrochoanal polyp, meningocele, meningoencephalocele)	
Foreign body	

Table 7.5 Other differential diagnoses in CRS

# **Consultations**

Treatment of rhinosinusitis may need to be coordinated with other specialties, which treat concurrent disorders or whose area of expertise is required when complications develop. Commonly, an opinion of an allergy and clinical immunology specialist is sought. Other colleagues with whom a rhinologist must establish a close cooperation are ophthalmologists, neurosurgeons, neurologists, pulmonologists, rheumatologists, dentists, radiologists, as well as radiation and medical oncologists.

# **Differential Diagnoses**

Other differential diagnoses, not highlighted previously, are presented in Table 7.5.

# Management

The recent decades of research have clearly established that CRS management is founded on a combination of medical and surgical management, often combined in a "sandwich" approach: optimal medical therapy, followed by surgery, followed by aggressive postoperative medical management.

# Evidence-Based Medical Management of CRSwNP and CRSsNP

Current evidence supports the treatment of CRS with intranasal corticosteroids (INCS) and nasal saline irrigations. Oral corticosteroids are primarily used in CRSwNP, while antibiotics (short or long-term) may have more effect in CRSsNP. A variety of other medical therapies have been proposed and tried, however there is no high level of evidence suggesting their benefit in CRS with or without polyps.

#### **Intranasal Corticosteroids**

INCS are an established first line treatment of CRSwNP and CRSsNP. INCS act locally on the nasal mucosa eliciting an anti-inflammatory and immunosuppressant effects, while mostly avoiding the systemic side effects of corticosteroids [4]. The local side effects include epistaxis, irritation, dryness, and septal perforation. The potential, but rare systemic side effects of INCS include development of cataracts, glaucoma, immune suppression, and effects on the hypothalamic-pituitary-adrenal axis, including growth reduction.

A Cochrane systematic review and meta-analysis comprehensively assessed the effectiveness of *INCS for CRSwNP* [22]. The pooled results favored INCS over placebo for symptoms (overall symptom scores, nasal obstruction, and proportion of responders for these symptoms), as well as objective and semi-objective measures (polyp score, change in polyp score, polyp recurrence after surgery, PNIF, olfaction score, and responders for reduction in polyp size and nasal airflow). No effect was found for low against high dose INCS.

A companion Cochrane review was also carried out to assess the effect of *INCS* for *CRSsNP* [34]. In comparison of topical steroids against placebo, the pooled results favored the former for symptom scores and proportion of responders, and there was no difference in relation to quality of life, endoscopic scores, radiologic changes or adverse events. There were no sufficient studies to perform a meta-analysis comparing INCS to no treatment, or two regimens of INCS therapy. The surgical state did to influence the results in this meta-analysis.

INCS can be delivered in the form of sprays, drops or irrigation. Aukema et al. have demonstrated that in patients who have failed optimal medical management with intranasal corticosteroid sprays and are indicated to have surgery, further treatment with intranasal corticosteroid drops can eliminate the need for surgery in almost half of patients, while improving their symptoms and PNIF values [1]. Patient position is said to have a role, however clinical evidence for this is not strong. One study compared the delivery of 1 % prednisolone acetate drops to mometasone furoate spray in a postoperative setting in patients meeting the symptomatic criteria for rhinosinusitis, most of whom had preoperative polyps (90 % and 79 %, respectively) [18]. Drops were applied with the patient lying supine, with the head extended 45° and slightly turned to the side of application (Mygind position). The study demonstrated no difference in polypoid change, edema and scar in the middle meatus and frontal recess 3 months postoperatively, but revealed a higher rate of *frontal ostia patency* with drops vs. spray (92.3 % vs. 76.3 %, respectively, p < 0.05).

Despite evidence for better penetration with high-volume devices, there are no randomized controlled studies comparing long term efficacy of steroid irrigation postoperatively (e.g. with budesonide) to other methods of delivery. Subgroup analysis of existing studies shows that sinus delivery methods (direct sinus cannulation or postoperative sinonasal irrigation) could achieve better symptom improvement compared with nasal delivery (simple sprays or low volume devices) and nasal aerosol or Turbuhaler [35].

#### **Nasal Saline Irrigation**

Nasal saline irrigation has long been used as an adjunctive measure in treating CRS. The evidence supporting the use of saline is moderate [15]. However, given the mostly benign nature of this treatment, it is recommended in CRS patients both with and without nasal polyps.

A variety of devices for saline and drug delivery have been developed: from low volume sprays, atomizers, nebulizers and larger volume sprays to large volume devices, including squeeze bottles, neti pots, bulb syringes and powered irrigation decides. The latter using at least 100 ml of fluid volume result in reliable distribution to the paranasal sinuses, especially after surgery [37]. No clear evidence of superiority between neti pots and squeeze bottles can be found. Delivery is significantly increased in the postoperative setting, when access to the sinuses is open. Head position may also affect delivery of saline or medication to sinuses, however only in the postoperative setting. Head down and forward position improves sinus delivery regardless of device. It is especially beneficial with low-volume devices and has less impact with high-volume ones.

#### Systemic Corticosteroids

There is considerable evidence for the use of *oral steroids in CRSwNP* [29]. The benefits include a significant short-term improvement in subjective and objective measures lasting 8–12 weeks, when combined with INCS use. At our clinic, we have been successfully using Prednisone 30 mg for 2 weeks without the need for tapering the dose. Using larger starting doses may require tapering. Predictably, systemic steroids bare more risk of harm.

Use of *oral steroids for CRSsNP* is not supported by studies of high level of evidence [29]. Some of the level 4 studies report subjective improvement in patient symptoms and objective improvement on imaging. Oral steroids in CRSsNP are considered optional, and their use must be balanced with the risk of potential side effects.

Systemic corticosteroids have also been used in the *perioperative setting*. Considerable debate exists about the timing of treatment. Our choice is to treat the patient in the immediate postoperative setting with the scheme described above. When used prior to surgery, steroids are shown to improve surgical visualization and may decrease operative time [29]. In a randomized, double-blind, placebo controlled study of 5 days preoperative and 9 days postoperative treatment with 30 md prednisone compared to placebo, Write and Agrawal found no significant differences in operative time or blood loss. However, they noted a higher incidence of surgery rated as "more than average difficulty" when compared to the steroid group. The study also reported improved olfaction in the steroid group at 2 weeks postoperatively, as well as improved endoscopic appearance of sinus cavities at 2 and 4 weeks, and 6 months postoperatively.

#### Antibiotics

The use of *systemic antibiotics for CRSwNP* remains an area requiring further research. Both short and long-term use of antibiotics in CRSwNP has resulted in a small effect on polyp size [13]. Doxycycline use in CRSwNP has garnered significant interest for its potential effect on polyps. In a randomized controlled study of patients with CRSwNP, van Zele et al. compared oral methylprednisolone (starting with 32 mg per day with a taper for 20 days total), doxycycline (200 mg on day 1 and 100 mg per day on days 2–20), and placebo [39]. Both methylprednisolone and doxycycline decreased nasal polyp size, with the steroid effect being more pronounced initially (between 2 and 3 weeks), while the antibiotic's effect lasting longer, up to 12 weeks. This study, however, did not compare doxycycline or systemic steroids effects directly.

Short-term treatment of CRSwNP with antibiotics, dictated by positive cultures, is recommended during exacerbations only. We commonly use 1–2 week courses of Augmentin for these purposes.

Long-term antibiotic use in patients with CRSsNP should be reserved to those who have failed treatment with INCS and nasal saline irrigation [13]. Given their effect in the lower airways as anti-inflammatory agents, macrolides have attracted much interest in the management of upper airway inflammation. In a meta-analysis of macrolide therapy for CRS, Pynnonen et al. included three studies, mostly of patients with CRSsNP (one study including some patients with CRSwNP, excluding only those with massive polyposis) [30]. Even though this study identified statistically significant changes in SNOT-20 score at 24 weeks, this result was clinically insignificant. There is also little evidence for the recommendation to suggest that patients with high serum IgE would be less likely to respond to macrolides than those with normal IgE [13]. The sub-analysis of a data obtained from the study by Wallwork et al. demonstrates a clinically insignificant effect of macrolides against placebo on SNOT-20 score change [30].

Most of the studies regarding long-term antibiotic use in CRS have small patient populations, as it is difficult to recruit patients into the placebo arms, and therefore, most studies are either prospective cohorts or retrospective analyses. For example, a retrospective study of 76 patients with recalcitrant CRS, both macrolides and trimethoprim-sulfamethoxazole were found effective in improving symptoms, with no significant differences between the antibiotic groups [40].

Rageb et al. carried out an interesting study comparing medical (erythromycin, nasal lavage and INCS) against surgical treatment of CRS after initial failure of medical treatment [31]. This study found no differences between the treatment groups at 6 or 12 months for Sinonasal Outcome Test-20 (SNOT-20) or the Short Form-36 Health Survey (SF-36), highlighting the importance of maximal medical therapy first with reservation of surgery for failures.

Topical antibiotics are not recommended for the management of CRS [13].

#### Therapies with No or Weak Evidence of Effect in CRSwNP and CRSsNP

There is insufficient evidence to recommend the following therapies in the routine treatment of *CRSwNP* and *CRSsNP*: anti-IgE, anti-IL5, antihistamines in non-allergic patients, topical and systemic antifungals, furosemide, immunosuppressants, leukotriene antagonists, aspirin desensitization, capsaicin, nasal decongestants, mucolytics, expectorants, surfactants including baby shampoo, probiotics homeopathic remedies, herbal medicines, manuka honey, proton pump inhibitors or phytopreparations [13].

# Surgical Management of Frontal Rhinosinusitis

Frontal sinus surgery will be discussed in detail in subsequent chapters of this book. Here, we would like to highlight several points about sinus surgery in general and frontal sinus surgery in particular.

Surgery for CRS with and without nasal polyps is an inseparable part of its management. Explaining possible need for surgery is important early on in the consultation. Similarly, it is important to underline that one time surgery may not be curative, and that multiple procedures might be required, especially in patients with polyps. Furthermore, it is imperative to stress once again the "sandwich" approach to CRS management, i.e. maximal medical therapy, followed by surgery, and again by aggressive postoperative medical management.

There are fewer randomized controlled trials in surgical than in medical treatment of CRS. Surgical trials are often unethical or impossible to carry out, and blinding is often compromised.

The goal of sinus surgery is to enhance drug delivery into the nasal cavity and sinuses. In a meta-analysis comparing the effects of INCS with placebo, a subgroup analysis in patients with sinus surgery compared to those without demonstrated similar symptom improvement irrespective of surgical status, but a significantly greater reduction in polyp size in patients with sinus surgery [35]. This is especially true for frontal sinuses, whose drainage is dependent on the health of the ostiome-atal complex.

When considering surgical treatment of frontal rhinosinusitis, the surgeon should consider whether the case is primary or revision, the anatomic peculiarities of the frontal recess (Table 7.2), presence of polyps, and most importantly the comfort of the operating surgeon. Even primary frontal sinus surgery can be at times challenging. The surgeons should weigh their skills and previous experience in frontal sinus procedures prior to proceeding with surgery or referring to a colleague.

In frontal rhinosinusitis, iatrogenic damage during previous surgery plays an especially important role. Disrespect for the natural draining pathway of the frontal sinus when it is free of disease can lead to iatrogenic rhinosinusitis by significant scarring and obstruction of the frontal recess [24]. This perpetuates a vicious cycle

Study anatomy in detail, especially in the frontal recess
Don't displace disease or bony septations, but remove by cutting
Don't strip mucosa
Don't shave circumferentially
Adequate frontal surgery should be the goal:
Avoid surgery when not indicated
Less than indicated surgery will lead to early recurrence
More than indicated surgery may lead to unnecessary scarring in healthy areas

of inflammation necessitating further surgery. Iatrogenic damage can result from unrecognized anatomy of the configuration of cells making the frontal recess. Displacement of bony walls of cells surrounding the frontal recess, e.g. the agger nasi, suprabullar or Kuhn cells, can result in frontal recess obstruction. When frontal recess surgery is required, use of incorrect technique, e.g. grasping and stripping the mucosal layers of or circumferential shaving around the recess, can also lead to scarring.

Several considerations can avoid iatrogenic damage, including careful study of preoperative sinus CT scans to understand frontal recess anatomy, using cutting instruments instead of grasping ones, avoidance of septation displacement and their complete removal when necessary, and avoidance of circumferential shaving around the frontal recess (Table 7.6).

Despite each case requires unique consideration, an integrated step-wise approach to frontal sinus surgery would ensure that the surgeon approaches each case with contemplation of benefit against morbidity inflicted by surgery (Table 7.7) [11]. The more advanced the procedures, the more potential damage they may carry.

In our practice setting, we rarely employ frontal sinus trephination, unless for topical delivery of medications in severe cases of acute or chronic invasive fungal rhinosinusitis. We also do not employ balloon technology.

# Conclusions

Frontal sinuses are challenging to treat. A thorough history, endoscopic examination, aided by diagnostic imaging and additional tests can help define the type of CRS and possible associated disorders, which make its treatment especially challenging.

Management of CRS includes an integrated approach with topical and systemic agents, as well as disease directed surgery. More so than in other sinuses, avoidance of iatrogenic damage is important, as it can perpetuate the inflammation and anatomic obstruction. However, a confident surgeon must not hesitate to proceed to advanced procedures, if previous rigorous attempts have failed, or current disease extent and complications dictate it.

Procedure	Indication
Ethmoidectomy/Draf I frontal sinusotomy	Primary frontal rhinosinusitis
Draf 2a frontal sinusotomy	Revision frontal rhinosinusitis, cystic fibrosis
	Frontal mucoceles after trauma or previous surgery
Draf 2b frontal sinusotomy	Exceptional cases of revision frontal sinusotomy with osteitic bone around the frontal recess
	Very large frontal beak
	Unilateral benign tumors
Draf 3 (modified endoscopic Lothrop procedure)	Recurrent frontal rhinosinusitis with failure of (repeated) bilateral Draf 2a procedures
	Extensive frontal sinus benign tumor
	Frontal mucoceles after trauma or previous surgery unreachable via unilateral approach
	Repair of CSF leak from posterior table of the frontal sinus
External frontal sinusotomy $\pm$ obliteration with osteoplastic flap or fat	Failure of (repeated) Draf 3 procedure for inflammatory disease
	Extensive benign tumors (osteoma, inverted papilloma) not amenable to Draf 3 approach
	Trauma with posterior table displacement or obstruction of frontal recess not amenable to Draf III
	Lateral mucoceles unreachable via Draf 3 approach (though in our experience these are rare)

Table 7.7 Modified integrated approach to frontal sinus surgery

## References

- 1. Aukema AA, Mulder PG, et al. Treatment of nasal polyposis and chronic rhinosinusitis with fluticasone propionate nasal drops reduces need for sinus surgery. J Allergy Clin Immunol. 2005;115(5):1017–23.
- Ahsan SF, Jumans S, et al. Chronic rhinosinusitis: a comparative study of disease occurrence in North of Scotland and Southern Caribbean otolaryngology outpatient clinics over a two month period. Scott Med J. 2004;49(4):130–3.
- 3. Benninger MS. Rhinitis, sinusitis, and their relationships to allergies. Am J Rhinol. 1992;6(2):37–43.
- 4. Benninger MS, Ahmad N, et al. The safety of intranasal steroids. Otolaryngol Head Neck Surg. 2003;129(6):739–50.
- Bhattacharyya N. Clinical and symptom criteria for the accurate diagnosis of chronic rhinosinusitis. Laryngoscope. 2006;116(7 Pt 2 Suppl 110):1–22.
- 6. Bousquet J, Heinzerling L, et al. Practical guide to skin prick tests in allergy to aeroallergens. Allergy. 2012;67(1):18–24.
- Chen Y, Dales R, et al. The epidemiology of chronic rhinosinusitis in Canadians. Laryngoscope. 2003;113(7):1199–205.
- Desrosiers M, Evans GA, et al. Canadian clinical practice guidelines for acute and chronic rhinosinusitis. J Otolaryngol Head Neck Surg. 2011;40 Suppl 2:S99–193.
- 9. Dietz de Loos DA, Segboer CL, et al. Disease-specific quality-of-life questionnaires in rhinitis and rhinosinusitis: review and evaluation. Curr Allergy Asthma Rep. 2013;13(2):162–70.

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- Doty RL, Shaman P, et al. Development of the University of Pennsylvania smell identification test: a standardized microencapsulated test of olfactory function. Physiol Behav. 1984;32(3):489–502.
- 11. Draf W, Weber R, et al. Current aspects of frontal sinus surgery. I: endonasal frontal sinus drainage in inflammatory diseases of the paranasal sinuses. HNO. 1995;43(6):352–7.
- 12. Fokkens WJ, Lund VJ, et al. EPOS 2012: European position paper on rhinosinusitis and nasal polyps. A summary for otorhinolaryngologists. Rhinology. 2012;50(1):1–12.
- Fokkens WJ, Lund VJ, et al. European position paper on rhinosinusitis and nasal polyps 2012. Rhinol Suppl. 2012;(23):3 p preceding table of contents, 1–298.
- Gordts F, Clement PA, et al. Prevalence of sinusitis signs in a non-ENT population. ORL J Otorhinolaryngol Relat Spec. 1996;58(6):315–9.
- Harvey R, Hannan SA, et al. Nasal saline irrigations for the symptoms of chronic rhinosinusitis. Cochrane Database Syst Rev. 2007;3:CD006394.
- Hastan D, Fokkens WJ, et al. Chronic rhinosinusitis in Europe an underestimated disease. A GA(2)LEN study. Allergy. 2011;66(9):1216–23.
- 17. Hedman J, Kaprio J, et al. Prevalence of asthma, aspirin intolerance, nasal polyposis and chronic obstructive pulmonary disease in a population-based study. Int J Epidemiol. 1999;28(4):717–22.
- Hong SD, Jang JY, et al. The effect of anatomically directed topical steroid drops on frontal recess patency after endoscopic sinus surgery: a prospective randomized single blind study. Am J Rhinol Allergy. 2012;26(3):209–12.
- Hopkins C, Slack R, et al. Long-term outcomes from the English national comparative audit of surgery for nasal polyposis and chronic rhinosinusitis. Laryngoscope. 2009;119(12):2459–65.
- 20. Hox V, Delrue S, et al. Negative impact of occupational exposure on surgical outcome in patients with rhinosinusitis. Allergy. 2012;67(4):560–5.
- Hummel T, Sekinger B, et al. 'Sniffin' sticks': olfactory performance assessed by the combined testing of odor identification, odor discrimination and olfactory threshold. Chem Senses. 1997;22(1):39–52.
- 22. Kalish L, Snidvongs K, et al. Topical steroids for nasal polyps. Cochrane Database Syst Rev. 2012;12:CD006549.
- 23. Kuhn FA. Chronic frontal sinusitis: the endoscopic frontal recess approach. Oper Tech Otolaryngol Head Neck Surg. 1996;7(3):222–9.
- 24. Kuhn FA. An integrated approach to frontal sinus surgery. Otolaryngol Clin North Am. 2006;39(3):437-61. viii.
- 25. Lund VJ, Mackay IS. Staging in rhinosinusitis. Rhinology. 1993;31(4):183-4.
- Min YG, Jung HW, et al. Prevalence and risk factors of chronic sinusitis in Korea: results of a nationwide survey. Eur Arch Otorhinolaryngol. 1996;253(7):435–9.
- Ottaviano G, Scadding GK, et al. Peak nasal inspiratory flow; normal range in adult population. Rhinology. 2006;44(1):32–5.
- Pilan RR, Pinna FR, et al. Prevalence of chronic rhinosinusitis in Sao Paulo. Rhinology. 2012;50(2):129–38.
- Poetker DM, Jakubowski LA, et al. Oral corticosteroids in the management of adult chronic rhinosinusitis with and without nasal polyps: an evidence-based review with recommendations. Int Forum Allergy Rhinol. 2013;3(2):104–20.
- Pynnonen MA, Venkatraman G, et al. Macrolide therapy for chronic rhinosinusitis: a metaanalysis. Otolaryngol Head Neck Surg. 2013;148(3):366–73.
- Ragab SM, Lund VJ, et al. Impact of chronic rhinosinusitis therapy on quality of life: a prospective randomized controlled trial. Rhinology. 2010;48(3):305–11.
- 32. Rosenfeld RM, Piccirillo JF, et al. Clinical practice guideline (update): adult sinusitis. Otolaryngol Head Neck Surg. 2015;152(2 Suppl):S1–39.
- 33. Scadding G, Hellings P, et al. Diagnostic tools in Rhinology EAACI position paper. Clin Transl Allergy. 2011;1(1):2.
- Snidvongs K, Kalish L, et al. Topical steroid for chronic rhinosinusitis without polyps. Cochrane Database Syst Rev. 2011;8:CD009274.

- 35. Snidvongs K, Kalish L, et al. Sinus surgery and delivery method influence the effectiveness of topical corticosteroids for chronic rhinosinusitis: systematic review and meta-analysis. Am J Rhinol Allergy. 2013;27(3):221–33.
- 36. Stammberger HR, Kennedy DW. Paranasal sinuses: anatomic terminology and nomenclature. Ann Otol Rhinol Laryngol Suppl. 1995;167:7–16.
- 37. Thomas WW, Harvey RJ, et al. Distribution of topical agents to the paranasal sinuses: an evidence-based review with recommendations. Int Forum Allergy Rhinol. 2013;3:691–703.
- 38. Tomassen P, Newson RB, et al. Reliability of EP3OS symptom criteria and nasal endoscopy in the assessment of chronic rhinosinusitis a GA(2) LEN study. Allergy. 2011;66(4):556–61.
- 39. Van Zele T, Gevaert P, et al. Oral steroids and doxycycline: two different approaches to treat nasal polyps. J Allergy Clin Immunol. 2010;125(5):1069–76. e4.
- 40. Videler WJ, van Hee K, et al. Long-term low-dose antibiotics in recalcitrant chronic rhinosinusitis: a retrospective analysis. Rhinology. 2012;50(1):45–55.
- Wormald PJ. Endoscopic sinus surgery: anatomy, three-dimensional reconstruction, and surgical technique. New York: Thieme Medical Publishers; 2013.

# **Chapter 8 Orbital Complications of Frontal Sinusitis**

Richard P. Manes, Bradley F. Marple, and Pete S. Batra

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Financial Disclosures Manes: consultant (Medtronic) Marple: consultant (Teva, Sunovian) Batra: Research grants (ARS, Medtronic), consultant (Medtronic)

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_8

#### **Core Messages**

- The most common cause of orbital infections is sinusitis, most commonly arising from the ethmoid sinuses. However, frontal sinusitis complications may progress rapidly and result in worse outcomes.
- The bacteriology of the orbital infection is similar to the sinusitis itself.
- Orbital infections exist on a clinical spectrum, and determining the correct diagnosis is of significant importance, as treatment may vary along the spectrum.
- The orbital septum is the key anatomic feature in the classification of orbital infections.
- Contrast computed tomography (CT) scans can distinguish cellulitis or abscess and assist in diagnosis and treatment.
- While preseptal and orbital cellulitis often respond to intravenous antibiotics, postseptal orbital complications of sinusitis often require surgical intervention.

#### 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 [33]. The pre-antibiotic 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 [9, 33]. Despite the advances in early diagnosis and aggressive antimicrobial therapy, spread of orbital infection to the cavernous sinus and intracranial compartment, although infrequent, is associated with morbidity and mortality rate of 10-20 % [20, 31]. The potential of such morbidities warrants careful study of these complications of sinusitis.

Frontal sinusitis and subsequent orbital complications is a narrow clinical window that demands both a high level of diagnostic acumen and technical ability to achieve a successful outcome. A thorough understanding of the anatomy, 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 [11, 12, 16, 17]. The most common complications of frontal sinusitis in order of frequency are [1, 28, 29, 43]:

- 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, particular in those patients with frontal sinusitis. Overall, 85 % of patients with orbital complications from sinusitis are within the pediatric age group and within this group 68 % are less than 15 years old [22, 39]. As the frontal sinus does not begin to pneumatize significantly until 6 years of age, the population experiencing complications related to the frontal sinus is correspondingly narrowed [1, 17]. 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 than in females (ratio of 2.6:1–3.3:1), and involve the left eye more frequently than the right [28–30, 39, 43]. The discrepant age, sex, and laterality trends have been noted by multiple authors, yet convincing explanations are lacking. There is a suggestion of increased incidence in the late fall through early spring months, thought to be secondary to increased incidence of sinusitis [30].

#### **Relevant Orbital and Sinus Anatomy**

The intimate relationship between the paranasal sinuses and the vital surrounding structures merits thorough understanding of this compact, complex anatomy. In the context of acute sinusitis with orbital complications, anatomic landmarks are often obscured and surgery made increasingly difficult by the bleeding propensity of inflamed sinonasal mucosa (Figs. 8.1, 8.2, and 8.3).



Fig. 8.1 A patient with left maxillary, ethmoid and frontal sinusitis, nasal polyps and a left medial subperiosteal abscess

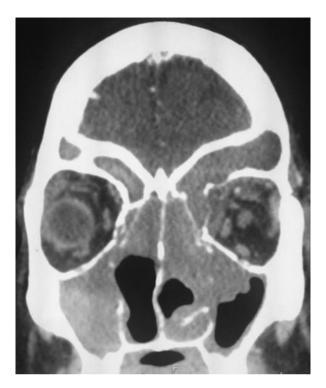


Fig. 8.2 Coronal CT scan with frontal sinusitis and erosion of the orbital roof and lamina paprycea



Fig. 8.3 Coronal CT scan of a large supraorbital ethmoid mucocele with erosion of the orbital roof

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 [22]. 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 [4]. 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*) [4, 32]. The orbital septa of the upper and lower eyelids form an anatomic barrier to infection and define the preseptal and postseptal spaces [5].

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 [40]. 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 [4, 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 [4].

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 [4, 9, 32, 33, 41]. Furthermore, the upper and lower eyelids have well described lymphatic networks and these preseptal tissues drain into preauricular and submandibular nodes [32].

The anatomy and the location of the frontal sinus predisposes to 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 [24]. 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 [24]. 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 [9, 22, 24].

#### 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 [9, 16, 28, 33, 44].

It is generally accepted that orbital infections arising from a sinonasal source can arise by two mechanisms [8, 9, 16, 22, 27, 35, 42–44]:

- 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 pathways by which direct extension can occur [8, 9, 17, 27, 35, 43]. This is more accentuated in children, because of the thinner bony septa and sinus wall, greater porosity of bones, open suture lines and larger vascular foramina [6]. 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 [8, 9, 17, 27, 35, 43]. In the absence of valves, communication between the sinuses and orbit may flow in either direction, enabling retrograde thrombophlebitis and the spread of infection.

#### **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 [21]. The classification of patients into five groups based on the involved anatomy, 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 [8]. Chandler's work codified the utility of this classification system, and his therapeutic principles characterize the modern approach to managing orbital complications of sinusitis (Table 8.1) [8, 19, 40].

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 are typically absent [8, 9, 17, 27]. Authors disagree regarding the absence [8, 16, 42] or presence of mild proptosis at this

Table 8.1       Chandler         classification systems for       orbital complications of         sinusitis       invisition	Group 1 – inflammatory edema (preseptal cellulitis)
	Group 2 – orbital cellulitis
	Group 3 – subperiosteal abscess
	Group 4 – orbital abscess
	Group 5 – cavernous sinus thrombosis
	Chandler et al. [8]

stage [9, 33]. The degree of preseptal inflammation may hamper accurate assessment of proptosis, especially when examining pediatric patients.

- Group II Orbital cellulitis results in pronounced edema and inflammation of the orbital soft tissue without frank abscess formation [8, 9, 33]. It is vital to detect the signs of proptosis and decreased extraocular motility, as these are considered reliable signs of orbital soft tissue involvement [16, 28, 35]. Chemosis is almost always present to varying degrees yet vision loss is very unusual in this stage, but should be monitored for carefully [9, 27, 33].
- Group III Subperiosteal abscess develops in the potential space between periorbita and bone [8]. 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 not always present early in the course of this stage [16, 22, 33, 39, 40, 42].
- 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 [8, 9, 22]. Severe proptosis and near complete restrictive ophthalmoplegia are noted and visual loss is increasingly common within this group [16, 33, 42, 44].
- Group V Cavernous sinus thrombosis may include such non-specific signs and symptoms as fever, headache, periorbital edema, and photophobia in addition to more specific findings of proptosis, chemosis, ophthalmoplegia and decreased visual acuity. The development of *bilateral* ocular symptoms is the classic finding in this stage [9, 16, 22, 35]. 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 [22, 39, 40].

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 the 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 8.2) [39]. 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 is warranted [17, 39].

Table 8.2       Schramm's         classification for orbital       complications of sinusitis	Orbital cellulitis Periorbital cellulitis
	Periorbital cellulitis with chemosis
	Orbital cellulitis
	Subperiosteal abscess
	Orbital abscess
	Cavernous sinus thrombosis
	Shramm et al. [45]

Table 8.3 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)
Orbital abscess	(a) Cellulitis
	I. Localized (orbital apex syndrome)
	II. Diffuse
	(b) Abscess
Cavernous sinus thrombosis	Not included. Considered intracranial

Mortimore and Wormald [28]

 Table 8.4
 Chadha classification for orbital complications of sinusitis

Stage	Classification	Treatment
Ι	Orbital cellulitis, no abscess	iv or oral antibiotics
IIa	Medial, small subperiosteal abscess (<1 cm)	iv antibiotics, close observation
IIb	Medial, large subperiosteal abscess (>1 cm)	Surgical drainage
IIc	Medial subperiosteal abscess with extension superiorly and/or laterally	Surgical drainage
III	Orbital abscess, peripheral or central	Surgical drainage

Chadha [7]

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 8.3) [26]. Mortimore and Wormald applied advanced computed tomography imaging to Moloney's concept of dividing preseptal and postseptal infections, relying upon further radiologic differentiation between cellulitis and abscess [28, 29]. Chadha performed a systematic review of the literature, attempting to divide orbital infections into categories with implications for management (Table 8.4) [7]. It is not clear that further, more stringent classifications of orbital infections have altered therapeutic strategies.

## Bacteriology

Orbital complications do not have a bacterial profile different from that of acute rhinosinusitis [9, 16, 17, 22, 33]. The most commonly cultured organisms in orbital infections are [1, 9, 16, 22, 30]:

- Streptococcus pneumoniae
- Haemophilus influenzae
- Moraxella catarrhalis
- Staphylococcus aureus
- Streptococcus pyogenes
- Anaerobic bacteria (*Prevotella*, *Porphyromonas*. *Fusobacterium* and *Peptostreptococcus* spp.)

Previous analysis of patients with simultaneous frontal and maxillary sinusitis found *H. influenzae* and *S. pneumoniae* to be the most commonly isolated organisms [2]. However, recent studies have evaluated the bacteriology of orbital complications of acute sinusitis in the post-vaccination era. The incidence of *H. influenza*-related disease has diminished as a result of immunization, though untypeable *Haemophilus* may still cause disease [3]. Another recent study evaluated bacteriology in the post-pneumococcal vaccine era [36]. A significant decrease in *S. pneumonia* and *Streptococcus viridans* was identified in the post-pneumococcal vaccine era, while an increase in *S. aureus* was noted.

The existing literature does not support a substantial difference in the bacterial populations implicated in frontal sinusitis from 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 [17]. The incidence of bacteremia in patients with orbital complications is greatest in children and declines steadily with age [9]. 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 [39].

#### **Diagnostic Evaluation**

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

Patients with preseptal cellulitis typically present with:

- History of recent upper respiratory infection or symptoms of acute bacterial rhinosinusitis
- · 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 [9, 16, 39, 43, 44]. Preseptal infections do not routinely require imaging studies [9, 10, 16, 33, 35, 44].

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 is consistent with postseptal disease [10, 22, 28, 43, 44]. To further clarify the utility of diagnostic imaging in this setting, Eustis and colleagues suggested the following parameters: (1) visual acuity changes; (2) proptosis; (3) limitation of motility; (4) uncertainty of diagnosis; and (5) deterioration of overall condition despite treatment [38].

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 [9, 22, 29, 35, 40]. Magnetic resonance imaging 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 with orbital extension [44]. 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 [44]. 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 [35]. Radiographic imaging is integral to the diagnosis, staging, and surgical therapy for postseptal infections, but should 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 [10, 15]. 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 is 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 (i.e. gaze restriction, 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 [33, 35, 43].

#### **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 CSF penetration, and possesses activity against beta-lactamase producing strains [9, 33]. Adjunctive topical and parenteral decongestants are often added, though steroids are not thought to be helpful [28, 39]. 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 [9, 11, 16, 22, 28, 30, 34, 38, 43]. Progression of symptoms or failure to respond to antibiotics within 48 h of treatment necessitates a CT scan and may constitute an indication for surgical therapy.

#### Surgical Therapy for Orbital Infections

True preseptal cellulitis responds rapidly to intravenous antibiotics and only in the exceptional cases will surgery be required; typically the incision and drainage of a coalescing lid abscess [33]. In contrast, surgical intervention in postseptal disease is required in 12–66 % of orbital complications of acute sinusitis [18, 39]. The indications for surgical therapy in postseptal infections comprise an evolving consensus of opinions from a number of large case series.

Surgery is recommended for one of the following four indications [9, 35, 39, 43]:

- 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 [35].

Though the above recommendations are widely accepted, dissenting opinions do exist. Oxford reported successful treatment of 18 pediatric patients with medial subperiosteal orbital abscesses (SPOA) with intravenous antibiotics, nasal saline irrigations and topical decongestants [34]. In evaluating patients successfully managed with medical therapy, Oxford identified five criteria for medical management of medial SPOA. These include: (1) normal vision, pupil and retina; (2) no ophthalmoplegia in one or more directions of gaze; (3) intraocular pressure less than 20 mmHg; (4) proptosis of 5 mm or less; and (5) abscess width of 4 mm or less on CT. Souliere reported successful treatment with decongestants and intravenous antibiotics in five pediatric patients with SPOA and anterior ethmoiditis (Chandler Group III) [41]. Ryan reported on 68 patients with SPOA, with 47 being treated medically [38]. Those requiring surgery had larger abscesses (>10 mm), were older (8.3 vs. 6.2 years) and had higher temperatures on admission (38.0° vs. 37.3°). There continues to be an increasing body of literature describing successful medical management of SPOA in select patients.

#### **Surgical Techniques**

A number of different surgical techniques are applicable to the treatment of orbital complications of sinusitis, though it is universally agreed that operative intervention should address the orbit and the paranasal sinuses simultaneously [9]. The advent of endoscopic surgical techniques has greatly reduced the morbidity of operative treatment. Chandler groups II (orbital cellulitis) and III (subperiosteal abscess) are now routinely treated endoscopically. Multiple studies have demonstrated that the endoscopic technique offers similar success rates when compared to traditional open approaches, with less hospitalization and less postoperative edema [25]. However, when bleeding secondary to inflammation precludes adequate drainage of the orbital infection, or ventilation of the involved sinuses, and measures to improve hemostasis fail, external techniques may need to be employed [29, 33, 40].

Multiple reports describe the endoscopic technique for SPOA in great detail [14, 37] Preoperatively, the CT scan should be evaluated carefully to identify the specific location of the infection, as well as any areas of bony dehiscence of the orbit or skull base. The procedure is performed under general anesthesia. The eye should be left visible to the operating surgeon with adequate corneal protection. The nose should be decongested with 1 % oxymetazoline hydrochloride on cotton pledgets, placed carefully in the nasal cavity. Avoiding mucosal disruption is paramount throughout the procedure, as the inflamed mucosa has a tendency to bleed and obscure visualization. The mucosa of the lateral nasal wall is then injected with local anesthesia. The authors typically use 1 % lidocaine with 1:100,000 epinephrine. If there are any cardiac concerns, 1:200,000 epinephrine may be substituted.

If any purulence is visualized in the middle meatus, it should be collected using sterile technique and sent for culture and sensitivity. Utilizing 0 and 30-degree telescopes, a maxillary antrostomy is performed. Once the maxillary antrostomy is complete, the bulla ethmoidalis is entered and removed with through-cutting instruments and a microdebrider. Posterior ethmoidectomy may be needed depending on the extent of disease. The lamina papyracea is skeletonized with through-cutting instruments. At this point, the surgeon may see pus emanating from the orbit. This should also be collected and sent for culture and sensitivity. If no drainage is seen, the lamina papyracea is then fractured with a Cottle elevator. Bone from the lamina papyracea is then elevated and removed to achieve adequate drainage of the abscess. Wide resection of the lamina papyracea is not always necessary, as minimal resection may provide adequate drainage [23]. The periorbita rarely needs to be incised in cases of SPOA. Subsequent antibiotic therapy is guided by intraoperative cultures.

Chandler group IV may also be managed endoscopically. Complete ethmoidectomy, medical orbital wall decompression and incision of the periorbita usually affords adequate drainage of most extraconal abscesses [9, 14]. Drainage of intraconal abscesses is best achieved through a combined open and endoscopic approach and should be managed concurrently with active participation of ophthalmology. 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 almost always recommended [9, 22, 28, 29, 33, 43]. 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 [9, 33].

#### 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.

## References

- 1. Altman KW, Austin MB, Tom LWC, et al. Complications of frontal sinusitis in adolescents: case presentations and treatment options. Int J Pediatr Otorhinolaryngol. 1997;41:9–20.
- Antila J, Suonpaa J, Lehtonen O. Bacteriological evaluation of 194 patients with acute frontal sinusitis and findings of simultaneous maxillary sinusitis. Acta Otolaryngol Suppl. 1997; 529:162–9.

- Barone S, Aiuto L. Periorbital and orbital cellulitis in the Haemophilus influenza vaccine era. J Pediatr Ophthalmol Strabismus. 1997;34:293–6.
- 4. Bedrossian EH. Surgical anatomy of the orbit. In: Della Rocca RC, Bedrossian EH, Arthurs BP, editors. Ophthalmic plastic surgery: decision making and techniques. New York: McGraw-Hill; 2002. p. 207–27.
- 5. Bedrossian EH. Surgical anatomy of the eyelids. In: Della Rocca RC, Bedrossian EH, Arthurs BP, editors. Ophthalmic plastic surgery: decision making and techniques. New York: McGraw-Hill; 2002. p. 25–41.
- Brook I. Microbiology and antimicrobial treatment of orbital and intracranial complications of sinusitis in children and their management. Int J Pediatr Otorhinolaryngol. 2009;73:1183–6.
- 7. Chadha NK. An evidence-based staging system for orbital injections from acute rhinosinusitis. Laryngoscope. 2012;122:S95–6.
- Chandler JR, Langenbrunner DJ, Stevens ER. The pathogenesis of orbital complications in acute sinusitis. Laryngoscope. 1970;80:1414–28.
- Choi SS, Grundfast KM. Complications in sinus disease. In: Kennedy DW, Bolger WE, Zinreich SJ, editors. Diseases of the sinuses: diagnosis and management. Ontario: B.C. Decker; 2001. p. 169–77.
- Clary RA, Cunningham MJ, Eavey RD. Orbital complications of acute sinusitis: comparison of computed tomography scan and surgical findings. Ann Otol Rhinol Laryngol. 1992;101:598–600.
- Davis JP, Stearns MP. Orbital complications of sinusitis: avoid delays in diagnosis. Postgrad Med J. 1994;70:108–10.
- Dolan RW, Chowdhury K. Diagnosis and treatment of intracranial complications of paranasal sinus infection. J Oral Maxillofac Surg. 1995;53:1080–7.
- Eustis H, Armstrong D, Buncic J, et al. Staging of orbital cellulitis in children: computerized tomography characteristics and treatment guidelines. J Pediatr Ophthalmol Strabismus. 1986;23:246–51.
- Fakhri S, Pereira K. Endoscopic management of orbital abscesses. Otolaryngol Clin N Am. 2006;39:1037–47.
- Garcia CE, Cunningham MJ, Clary RA, et al. the etiologic role of frontal sinusitis in pediatric orbital abscesses. Am J Otolaryngol. 1993;14:449–52.
- Goodwin WJ. Orbital complications of ethmoiditis. Otolaryngol Clin N Am. 1995; 18:139–47.
- Goldberg AN, Oroszlan G, Anderson TD. Complications of frontal sinusitis and their management. Otolaryngol Clin N Am. 2001;34:211–25.
- Hawkins DB, Clark RW. Orbital involvement in acute sinusitis. Clin Pediatr. 1977; 16:464–71.
- Healy GB. The pathogenesis of orbital complications in acute sinusitis. Laryngoscope. 1997; 107:441–6.
- Herrmann BW, Forsen JW. Simultaneous intracranial and orbital complications of acute rhinosinusitis in children. Int J Pediatr Otorhinolaryngol. 2004;68:619–25.
- 21. Hubert L. Orbital infections due to nasal sinusitis. NY Med J. 1937;37:1559-64.
- 22. Kendall KA, Senders CW. Orbital and intracranial complications of sinusitis in children and adults. In: Gershwin ME, Incaudo GA, editors. Diseases of the sinuses: a comprehensive textbook of diagnosis and treatment. Totowa: Humana Press; 1996. p. 247–72.
- 23. Khalifa BC. Extent of resection of the lamina papyracea in medial subperiosteal abscess. Otolaryngol Head Neck Surg. 2011;145:161–4.
- McLaughlin RB, Rehl RM, Lanza DC. Clinically relevant frontal sinus anatomy and physiology. Otolaryngol Clin N Am. 2001;34:1–22.
- Migirov L, Yakirevitch A, Bedron L, et al. Endoscopic sinus surgery for medial orbital subperiosteal abscess in children. J Otolaryngol Head Neck Surg. 2009;38:504–8.
- Moloney JR, Badham NJ, McRae A. The acute orbit, preseptal (periorbital) cellulitis, subperiosteal abscess and orbital cellulitis due to sinusitis. J Laryngol Otol. 1987;101(12supp):1–18.
- 27. Morgan PR, Morrison WV. Complications of frontal and ethmoid sinusitis. Laryngoscope. 1980;90:661–6.

- Mortimore S, Wormald PJ. The Goote-Schuur hospital classification of the orbital complications of sinusitis. J Laryngol Otol. 1997;111:719–23.
- 29. Mortimore S, Wormald PJ. Management of acute complicated sinusitis: a five year review. Otolaryngol Head Neck. 1999;121:639–42.
- Nageswaran S, Woods C, Benjamin D, et al. Orbital cellulitis in children. Pediatr Infect Dis J. 2006;25:695–9.
- Nathoo N, Nadvi SS, van Dellen JR, et al. Intracranial subdural empyemas in the era of computed tomography: a review of 699 cases. Neurosurgery. 1999;44:529–36.
- Nerad JA. Clinical anatomy. In: Nerad JA, Krachmer JH, editors. Occuloplastic surgery: the requisites in ophthalmology. St. Louis: Mosby; 2001. p. 25–70.
- 33. Osguthorpe JD, Hochman M. Inflammatory sinus diseases affecting the orbit. Otolaryngol Clin N Am. 1993;26:657–71.
- Oxford LE, McClay J. Medical and surgical management of subperiosteal orbital abscess secondary to acute sinusitis in children. Int J Pediatr Otorhinolaryngol. 2006;70:1853–61.
- Patt BS, Manning SC. Blindness resulting from orbital complications of sinusitis. Otolaryngol Head Neck Surg. 1991;104:789–95.
- 36. Pena MP, Preciado DA, Orestes M, et al. Orbital complications of acute sinusitis: changes in the post-pneumococcal vaccine era. JAMA Otolaryngol Head Neck Surg. 2013;139:223–7.
- Roithmann R, Uren B, Pater J, et al. Endoscopic drainage of a superiorly bases subperiosteal abscess. Laryngoscope. 2008;118:162–4.
- Ryan JT, Preciado DA, Bauman N. Management of pediatric orbital cellulitis in patients with radiographic findings of subperiosteal abscess. Otolaryngol Head Neck Surg. 2009;140:907–11.
- Schramm VL, Curtin HD, Kennerdell JS. Evaluation of orbital cellulites and results of treatment. Laryngoscope. 1982;92:732–8.
- 40. Shahin J, Gullane PJ, Dayal VS. Orbital complications of acute sinusitis. J Otolaryngol. 1987;16:23–7.
- Souliere CR, Antoine GA, Martin MP, et al. Selective non-surgical management of subperiosteal abscess of the orbit: computerized tomography and clinical course as indication for surgical drainage. Int J Pediatr Otorhinolaryngol. 1990;19:109–19.
- 42. Wagenmann M, Naclerio RM. Complications of sinusitis. J Allergy Clin Immunol. 1992;90:552–4.
- 43. Younis RT, Lazar RH, Bustillo A, et al. Orbital infection as a complication of sinusitis: are diagnostic and treatment trends changing? Ear Nose Throat J. 2001;81:771–5.
- 44. Younis RT, Anand VK, Davidson B. The role of computed tomography and magnetic resonance imaging in patients with sinusitis with complications. Laryngoscope. 2002;112:224–9.
- 45. Shramm VL, Curtin HD, Kennerdell JS. Evaluation of orbital cellulitis and results of treatment. Laryngoscope. 1982;92:732–8.

# Chapter 9 CNS Complications of Frontal Sinus Disease

Jonathan Liang and Andrew P. Lane

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#### **Core Messages**

- 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 commonly spreads to the CNS through vascular communications between the frontal sinus diploic veins and the dural venous plexus.

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- Progressive headache and fever are the most common presenting signs of CNS complications, although some may present silently.
- Pediatric patients with CNS complications of frontal sinusitis often have less classic presentations and associated extracranial complications.
- The single most important study to obtain in the diagnosis 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.

# 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 [3]. Suppurative complications may develop either via a direct or indirect pathway. Direct spread can occur though infection of the frontal bone (osteitis, osteomyelitis) or along preformed routes (encephaloceles, fractures, CSF fistula, tumors) [2]. An indirect pathway, which is more commonly seen, involves 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 [32]. 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 [22, 24, 36, 37]. 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 [10]. Another study from the 1960s reported a 10 % incidence of intracranial

complications among patients admitted to the hospital for frontal sinusitis [4]. 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 (Figs. 9.1, 9.2, and 9.3).

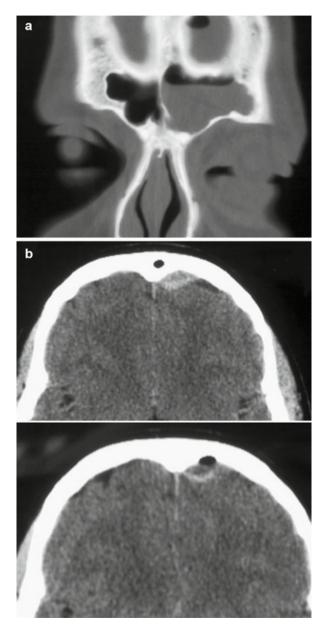


Fig. 9.1 Frontal lobe pneumococcal abscess secondary to frontal sinusitis. (a) Coronal CT showing opacification of left frontal sinus. (b) Axial CT demonstrating abscess of frontal lobe

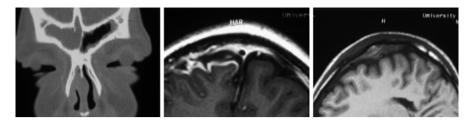
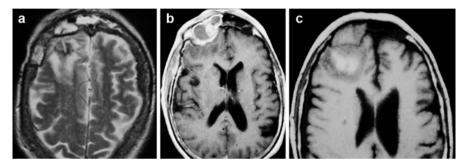


Fig. 9.2 Frontal sinusitis causing meningitis and frontal lobe abscess. Cultures of CSF and the abscess revealed staphylococcus A



**Fig. 9.3** Frontal sinusitis causing septic thrombophlebitis and hemorrhagic brain infarction. (a) T2 weighted MRI demonstrating abscess. (b, c) T1 weighted images with higher signal intensity in the area of brain infarction

# 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 (Pott's Puffy Tumor) or even a pericranial abscess. Pott's Puffy tumor is osteomyelitis of the frontal bone, and up to 40 % of these patients present with intracranial complications [28]. Pott's Puffy Tumor is often an indicator of intracranial complication because the infection may spread to the intracranial cavity through bony erosions, preformed pathways, or septic thrombosis [25].

#### **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 [10]. Magnetic resonance imaging (MRI) is an useful adjunctive study when suspicion for intracranial exam is high based on history of CT findings [2]. Input from otolaryngology, neurosurgery, ophthalmology, and infectious diseases services are important in creating a multidisciplinary approach to the care of the patient [26]. 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 [18].

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 [7].

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 [3]. 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 [30, 32].

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 [3, 24]. 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 [8]. 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 [33]. 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 [32].

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 [3, 32]:

- 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 [24].

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 [18]. 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 [13]. 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 [36]. Because the blood supply is less robust, abscesses tend to form in the white matter rather than the grey matter, and they become encapsulated over weeks [29]. 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.

In the pediatric population, intracranial complications from frontal sinusitis deserve special consideration. The risk of developing intracranial complications have been shown to be significantly higher in pediatric patients with acute frontal sinusitis versus acute sinusitis not involving the frontal sinus [19]. In the pediatric population, intracranial complications from sinusitis are more challenging to diagnose and treat. These patients frequently lack a significant sinus history and often present with vague and non-localizing signs and symptoms [16]. In contrast, adult patients presenting with intracranial complications from sinusitis usually have a history of chronic sinusitis. A large pediatric case series found a high incidence of concurrent orbital complications especially with epidural abscesses, suggesting that children presenting with orbital and forehead swelling and sinusitis should be evaluated for a concurrent intracranial infection even in the absence of neurologic findings [16].

#### Treatment

The organisms most commonly cultured either from the frontal sinus or from intracranial collections are staphylococcus and streptococcus species [2, 22, 23]. Other gram positive bacteria may be found, as well as anaerobes, and gram negatives such as H. influenza [6]. 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 [18]. The roles of mannitol and corticosteroids for brain edema, and anti-coagulants for dural sinus thrombosis, are controversial, but may be indicated in certain situations [34, 35]. 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. Neurosurgical drainage is often emergently indicated for patients with large intracranial collections. The decision regarding the timing of medical and surgical therapy is more controversial for patients with small intracranial collections (<1 cm). Initial medical management with intravenous antibiotics and serial radiologic evaluation has been advocated [11].
- Ideally, when indicated, both the intracranial process and the sinus infection should be addressed at the same surgical procedure [1, 10, 23, 26, 32]. 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. The role of neurosurgical drainage of intracranial complications is fairly well established, but the role of acute surgical intervention for the sinus disease is unclear. In one series, endoscopic sinus surgery did not appear to alter the need for neurosurgical intervention [11].
- 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 [14, 26].

The trephination may be combined with an endoscopic frontal sinusotomy if the conditions are favorable [15], or a catheter may be brought out through the brow incision to allow for post-operative irrigation and to prevent re-accumulation of purulence. The use of frontal sinus stents in the acute setting is controversial. The rationale for stenting is to minimize postoperative stenosis and improve mucosalization of the neo-ostium. The rationale against stenting is that it can lead to functional blockage and be a nidus for perpetual infection [2]. If the frontal table of the sinus is necrotic or eroded by osteomyelitis, wide surgical debridement of the bone is necessary often through a coronal incision, along with prolonged intravenous antibiotic therapy. Reconstruction of the defect is delayed until the infection is resolved, as demonstrated by gallium-67 citrate scan [14].

Surgical treatment of uncomplicated epidural abscess involves creation of burr holes without opening the dura [39]. 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 [20]. Subdural empyema may be managed by either burr holes or craniotomy, with opening of the dura to drain the collection [10]. 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 [5]. 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 [38]. 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 [27]. 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 [3].

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 [12]. Another interventional approach in this situation is the local infusion of thrombolytic agent into the dural sinus system [9, 17].

#### 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 [10]:

- · Hemiparesis
- · Hypoesthesia
- 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 post-infection sequelae may also occur in the pediatric population [21]. 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 [27]. Of all the CNS complications, the mortality from dural sinus thrombosis is the greatest, perhaps as high as 50–80 % [35]. 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 multi-disciplinary 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

## References

- 1. Bayonne E, Kania R, Tran P, Huy B, Herman P. Intracranial complications of rhinosinusitis. A review, typical imaging data and algorithm of management. Rhinology. 2009;47(1):59–65.
- 2. Betz CS, Issing W, Matschke J, Kremer A, Uhl E, Leunig A. Complications of acute frontal sinusitis: a retrospective study. Eur Arch Otorhinolaryngol. 2008;265(1):63–72.
- Blitzer ACP. Intracranial complications of the disease of the paranasal sinuses. In: Blitzer ALW, Friendman WH, editors. Surgery of the paranasal sinuses. Philadelphia: WB Saunders Co.; 1985. p. 328–37.
- 4. Bluestone CD, Steiner RE. Intracranial complications of acute frontal sinusitis. South Med J. 1965;58:1–10.
- 5. Bok AP, Peter JC. Subdural empyema: burr holes or craniotomy? A retrospective computerized tomography-era analysis of treatment in 90 cases. J Neurosurg. 1993;78(4):574–8.
- 6. Brook I. Bacteriology of acute and chronic frontal sinusitis. Arch Otolaryngol Head Neck Surg. 2002;128(5):583–5.
- Cb C. Subdural empyema secondary to purulent frontal sinusitis. Arch Otolaryngol Head Neck Surg. 1843;39:211–30.
- Choi SSGK. Complications in sinus disease. In: Kennedy DWBW, Zinreich SJ, editors. Disease of the sinuses: diagnosis and management. Hamilton: BC Decker Inc; 2001. p. 172–7.
- Cipri S, Gangemi A, Campolo C, Cafarelli F, Gambardella G. High-dose heparin plus warfarin administration in non-traumatic dural sinuses thrombosis. A clinical and neuroradiological study. J Neurosurg Sci. 1998;42(1):23–32.
- Clayman GL, Adams GL, Paugh DR, Koopmann Jr CF. Intracranial complications of paranasal sinusitis: a combined institutional review. Laryngoscope. 1991;101(3):234–9.
- 11. DelGaudio JM, Evans SH, Sobol SE, Parikh SL. Intracranial complications of sinusitis: what is the role of endoscopic sinus surgery in the acute setting. Am J Otolaryngol. 2010;31(1):25–8.
- Ekseth K, Bostrom S, Vegfors M. Reversibility of severe sagittal sinus thrombosis with open surgical thrombectomy combined with local infusion of tissue plasminogen activator: technical case report. Neurosurgery. 1998;43(4):960–5.
- 13. Eustis HS, Mafee MF, Walton C, Mondonca J. MR imaging and CT of orbital infections and complications in acute rhinosinusitis. Radiol Clin North Am. 1998;36(6):1165–83, xi.
- Gardiner LJ. Complicated frontal sinusitis: evaluation and management. Otolaryngol Head Neck Surg. 1986;95(3 Pt 1):333–43.
- Gerber ME, Myer 3rd CM, Prenger EC. Transcutaneous frontal sinus trephination with endoscopic visualization of the nasofrontal communication. Am J Otolaryngol. 1993;14(1):55–9.
- Germiller JA, Monin DL, Sparano AM, Tom LW. Intracranial complications of sinusitis in children and adolescents and their outcomes. Arch Otolaryngol Head Neck Surg. 2006;132(9):969–76.
- Gerszten PC, Welch WC, Spearman MP, Jungreis CA, Redner RL. Isolated deep cerebral venous thrombosis treated by direct endovascular thrombolysis. Surg Neurol. 1997;48(3): 261–6.
- Goldberg AN, Oroszlan G, Anderson TD. Complications of frontal sinusitis and their management. Otolaryngol Clin North Am. 2001;34(1):211–25.
- Hakim HE, Malik AC, Aronyk K, Ledi E, Bhargava R. The prevalence of intracranial complications in pediatric frontal sinusitis. Int J Pediatr Otorhinolaryngol. 2006;70(8):1383–7.

- 20. Heran NS, Steinbok P, Cochrane DD. Conservative neurosurgical management of intracranial epidural abscesses in children. Neurosurgery. 2003;53(4):893–7; discussion 7–8.
- Idriss ZH, Gutman LT, Kronfol NM. Brain abscesses in infants and children: current status of clinical findings, management and prognosis. Clin Pediatr (Phila). 1978;17(10):738–40, 45–6.
- 22. Kaplan RJ. Neurological complications of infections of head and neck. Otolaryngol Clin North Am. 1976;9(3):729–49.
- Kaufman DM, Litman N, Miller MH. Sinusitis: induced subdural empyema. Neurology. 1983;33(2):123–32.
- 24. Kaufman DM, Miller MH, Steigbigel NH. Subdural empyema: analysis of 17 recent cases and review of the literature. Medicine (Baltimore). 1975;54(6):485–98.
- 25. Ketenci I, Unlu Y, Tucer B, Vural A. The Pott's puffy tumor: a dangerous sign for intracranial complications. Eur Arch Otorhinolaryngol. 2011;268(12):1755–63.
- Lang EE, Curran AJ, Patil N, Walsh RM, Rawluk D, Walsh MA. Intracranial complications of acute frontal sinusitis. Clin Otolaryngol Allied Sci. 2001;26(6):452–7.
- 27. Maniglia AJ, Goodwin WJ, Arnold JE, Ganz E. Intracranial abscesses secondary to nasal, sinus, and orbital infections in adults and children. Arch Otolaryngol Head Neck Surg. 1989;115(12):1424–9.
- Masterson L, Leong P. Pott's puffy tumour: a forgotten complication of frontal sinus disease. Oral Maxillofac Surg. 2009;13(2):115–7.
- 29. Mohr RM, Nelson LR. Frontal sinus ablation for frontal osteomyelitis. Laryngoscope. 1982;92(9 Pt 1):1006–15.
- Morgan PR, Morrison WV. Complications of frontal and ethmoid sinusitis. Laryngoscope. 1980;90(4):661–6.
- 31. Recommendations on stroke prevention, diagnosis, and therapy. Report of the WHO Task Force on Stroke and other Cerebrovascular Disorders. Stroke. 1989;20(10):1407–31.
- Remmler D, Boles R. Intracranial complications of frontal sinusitis. Laryngoscope. 1980;90(11 Pt 1):1814–24.
- Renaudin JW, Frazee J. Subdural empyema importance of early diagnosis. Neurosurgery. 1980;7(5):477–9.
- 34. Soleau SW, Schmidt R, Stevens S, Osborn A, MacDonald JD. Extensive experience with dural sinus thrombosis. Neurosurgery. 2003;52(3):534–44; discussion 42–4.
- Southwick FS, Richardson Jr EP, Swartz MN. Septic thrombosis of the dural venous sinuses. Medicine (Baltimore). 1986;65(2):82–106.
- Wald ER, Pang D, Milmoe GJ, Schramm Jr VL. Sinusitis and its complications in the pediatric patient. Pediatr Clin North Am. 1981;28(4):777–96.
- Wenig BL, Goldstein MN, Abramson AL. Frontal sinusitis and its intracranial complications. Int J Pediatr Otorhinolaryngol. 1983;5(3):285–302.
- 38. Yang SY, Zhao CS. Review of 140 patients with brain abscess. Surg Neurol. 1993;39(4):290–6.
- Younis RT, Lazar RH, Anand VK. Intracranial complications of sinusitis: a 15-year review of 39 cases. Ear Nose Throat J. 2002;81(9):636–8, 40–2, 44.

# **Chapter 10 Allergy and the Frontal Sinus**

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#### **Core Messages**

- Allergic sensitivities have been identified in patients with CRS, most notably to perennial allergens such as dust mites, molds, cockroach, and pet dander.
- Allergy testing is integral to the workup of patients with chronic nasal symptoms and chronic non-infectious frontal sinusitis. Allergy testing can identify positive reactions to causative allergens and serve as the basis for environmental control measures and/or formulation of treatment vials for immunotherapy.

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_10

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- Most evidence does not support a direct connection between inhalant allergic rhinitis and CRS in general. However, several studies support the association between food allergies and CRS especially with nasal polyps. These food allergies may require and elimination challenge diet to identify. In adults in America, the most common foods identified are wheat and dairy.
- Over the past 15 years, the role of fungal elements and their immunologic effect in CRS have been debated. Allergic fungal rhinosinusitis (AFRS) is a clinicopathologic entity that has traditionally had a strong association with inhalant allergy, although the exact mechanisms of this have recently been called into question. AFRS often has significant involvement of the frontal sinus, demonstrating erosion and expansion of the paranasal sinus bony walls, along with heterogeneous intrasinus densities identified on imaging.
- Surgical goals for AFRS include:
  - Complete extirpation of all allergic mucin and fungal debris, thereby decreased the immunologic burden in atopic patients.
  - Establishment of permanent drainage and ventilation of the affected sinuses while preserving the integrity of sinonasal mucosa.
  - Allowing for post-operative access to previously diseased areas to improve access for nasal irrigations and post-operative debridement.

# Introduction

As the entry point to the respiratory tract, the nasal cavity and paranasal sinuses are repeatedly subjected to inhaled allergens and pathogens. The nasal mucosa plays a critical role in the host defense and is an immune responsive organ. Inflammation of nasal mucosa can be caused by a variety of antigens, irritants, and exposures.

• Allergic rhinitis (AR) is characterized as a Gell and Coombs Type I IgE-mediated hypersensitivity reaction in response to allergens such as pollen, dust mites, grasses, and animal dander.

The allergen is presented to a T-helper (Th) 2 cell by a resident antigenpresenting cell. Th2 cells produce cytokines, such as interleukin-4, and also interact with B cells. This interaction of the Th2 cell and B cell results in the formation and release of IgE antibody locally. IgE then goes on to bind to and activate mast cells and basophils. Activated mast cells and basophils degranulate and release inflammatory chemical mediators, including histamine, cytokines, leukotrienes, prostaglandins, and interleukins. Subsequent mucus secretion, vasodilation, smooth muscle contraction, and inhibition of mucociliary clearance results in symptoms. The classic nasal symptoms of seasonal allergic rhinitis are:

- Congestion
- Rhinorrhea
- Sneezing
- Itching.

Chronic exposure to an allergen is characterized less by the sneezing and rhinorrhea and more by thickened mucus and congestion. AR affects between ten and thirty percent of the population worldwide [1]. Food allergies are not always dependent upon IgE-mediated mechanisms, and in up to 30 % of patients with nasal polyps, hypersensitivity to foods such as dairy and wheat can be demonstrated by elimination challenge food diets, and improvement in nasal symptoms with the elimination of the food [2].

Allergic fungal rhinosinusitis (AFRS) represents a separate, yet related, clinicopathologic entity. AFRS affects 6–26 % of patients with chronic rhinosinusitis (CRS) that require surgery and 9–12 % of patients with nasal polyps [3–6]. This wide range of reported AFRS prevalence may be attributed to variations in diagnostic criteria, along with geographic variations. Though widely quoted, the diagnostic criteria proposed by Bent and Kuhn in 1994 has been the subject of debate since its debut [7]. While the pathophysiology of AR is relatively well understood, the underlying mechanism of disease of AFRS is less clearly understood, and continued controversy surrounds both the pathophysiology and diagnosis of AFRS. Both AR and AFRS affect the entirety of the nasal mucosa, and appear to influence outcomes in patients treated for frontal sinusitis.

In this chapter, the diagnosis, radiographic findings, treatment modalities, and impact on the frontal sinus for both AR and AFRS will be discussed.

## **Allergic Rhinitis**

AR is an IgE-mediated Type I hypersensitivity response that manifests clinically with sneezing, rhinorrhea, itching, and nasal congestion. A 2000 study of CRS patients undergoing endoscopic sinus surgery revealed that 84 % had positive reactions on allergy testing, and 60 % had significant allergic sensitivities, most notably to perennial allergens, such as dust mites, pet dander, cockroaches, and molds [8]. In a retrospective review of 91 patients with acute frontal sinusitis, Ruoppi et al reported that 24 % had AR [9]. Wide and colleagues also reported on a series of 456 patients who were treated for acute frontal sinusitis [10]. In this series, 85 (21 %) required surgical intervention, either trephination or endoscopic sinus surgery. The prevalence of AR was statistically significant between the surgical group (16 %) and medically-managed group (7 %).

• In the evaluation and management of patients with chronic rhinosinusitis, especially patients whose symptoms are refractory to medical management and surgical intervention is being considered, allergy testing is recommended, with either in vitro blood tests or allergy skin testing [11, 12].

The diagnosis of AR is supported by a clinical history of typical symptoms: sneezing, nasal congestion, rhinorrhea, post-nasal drainage, facial pressure, and fatigue; and is confirmed by a skin or blood test for allergies. The symptoms of AR overlap with symptoms of CRS; however, it is the onset and duration of symptoms and exposure-related symptoms that aid in diagnosing AR.

Perennial allergic rhinitis (PAR) can be difficult to diagnose because symptoms may be subclinical – moderate to severe nasal congestion, increased post-nasal drainage unrelated to exposure. These patients may develop acute or seasonal flares. Patients with acute or seasonal AR often self-report having "hay fever," with classic symptoms of sneezing, eye itching, and rhinorrhea upon allergen exposure.

Allergy testing is an important part of the workup in patients with chronic nasal symptoms and chronic non-infectious frontal sinusitis. If a patient has a true allergy, allergy testing can identify the causative allergen(s) and exposure to the allergen can be, in most cases, controlled. Allergy testing can also provide a basis for formulation of allergen vials for immunotherapy.

Avoidance of causative allergens is a mainstay of treatment for AR. Appropriate measures can be taken to minimize exposure to inciting allergens, such as eliminating exposure to domestic pets for patients with pet dander allergies, placing protective covers on pillows and mattresses for those patients with dust allergies, and exterminating the home to eliminate cockroaches.

• Immunotherapy can be effective in patients who fail to respond to environmental control measures and targeted pharmacotherapy or who have symptoms for over half of the year.

When AR symptoms are present for over half the year, immunotherapy is a cost effective alternative to targeted pharmacotherapy that may or may not be providing symptomatic relief. Further, immunotherapy is the only allergy treatment that has the potential to effect alterations in the immune system and 'cure' the disease.

Two major forms of allergy testing exist:

- In vitro testing (blood)
- Skin testing.

There are several forms of in vitro testing. The earliest forms of in vitro assessment of specific IgE levels were performed using radioallergosorbent testing (RAST) or a modified RAST. This term is still used by many to refer to blood tests for allergy, though almost universally the RAST methodology has been replaced by enzyme linked techniques without the radioactivity risk factors and disposal issues.

• While patients with allergies likely have allergen-specific IgE that is elevated during exposure, total IgE is not a good screen for allergy since it is often within normal limits.

Nevertheless, an elevated total IgE can give you an idea of the total allergy load of the patient and if very elevated, such as levels of 500 to greater than 1,000, can raise the likelihood that AFRS or the pulmonary corollary to AFRS, Allergic Bronchopulmonary Aspergillosis (ABPA) is present. Occasionally a patient may have elevated total IgE without commercially demonstrable specific allergen elevation. It is thought that in some situations this may represent superantigen stimulation from concomitant bacterial pathogens such as *Staphylococcus aureus*.

## In Vitro Testing for Allergy

A mini allergy screen of six antigens using in vitro batteries of one grass (Timothy), one weed (common ragweed), one tree (oak), two molds (*Alternaria* and *Helmithosporium*), and one dust mite (*Dermatophagoides pteronyssinus*), with epidermals (i.e. cat, horse, etc.) added if indicated by history, has a predictive value of 75 %. If the battery is expanded to a total of nine antigens by including a second grass (Bermuda), an additional tree (mountain cedar), and an additional mold (*Cladosporium*) then the predictive value increases to 95 % compared to a 13-antigen screen. This study was performed in patients living in Southwest Texas [13]. Practitioners in other parts of the country would need to tailor the antigens to the most prevalent and likely allergens in their particular region. Pollen maps available from many of the testing companies can help guide the selection of these antigens.

In vitro testing with modified RAST or CAP shows significant association with intradermal dilutional test (IDT) results however CAP appears to be better than modified RAST in confirming mold (Alternaria) allergy [14].

# Skin Testing for Allergy

Skin testing for environmental allergy typically occurs by two techniques: intradermal or prick testing. Intradermal dilutional testing (IDT), previously known as skin endpoint titration (SET), is the most time-consuming and sensitive allergy test and is able to indicate a safe starting dose for immunotherapy. A common practice is to perform a screen using dust mite, cat, dog, mold mix, tree mix and grass mix initially and only perform additional IDT within the individual pollen or mold component antigens if the respective mix is positive.

There are a wide variety of *prick testing* devices. One of the most popular, most reproducible and fastest to apply is the Multitest II device that can apply up to eight antigens at one time. A negative Multitest using 14 antigens plus histamine and glycerin controls indicates that significant inhalant allergy is unlikely. A positive Multitest may require additional in vitro or skin testing [15].

The simplest screen for allergies includes either an in vitro allergen screen of six to nine allergens (which would include perennials such as dust mite and molds) or a Multitest II prick test. The focus of the screen test should be on the following perennial allergens since they are most often associated with CRS:

- Dust mite
- Cockroach
- Cat (if applicable)
- Molds

If the screen is negative, then the patient probably does not have inhalant allergy. If the screen is positive, then the patient may well be allergic to multiple other allergens and further, more detailed investigation is warranted.

While imaging is not typically part of the diagnostic workup of AR, patients with chronic sinonasal symptoms often undergo computed tomography of the sinuses.

• Allergy can be found in up to 84 % of patients with refractory CRS.

In a study of 339 consecutive patients diagnosed with CRS, Tezer and colleagues found that 62.8 % had at least one positive skin prick tests (allergic patients) [16]. Review of computed tomography of the sinuses demonstrated that allergic patients were more likely to have maxillary mucosal thickening and frontal sinus hypoplasia. Inflammation of the nasal mucosa can impair outflow through the sinus ostia, which allows for stasis of secretions within the paranasal sinuses. As the last paranasal sinus to develop, the frontal sinus continues to pneumatize until after puberty. The inflammatory changes that a patient with allergies undergoes may interfere with the pneumatization process of the frontal sinus, resulting in a relatively underpneumatized sinus, as is seen in patients with cystic fibrosis.

While the preponderance of evidence does not support an association between inhalant allergic rhinitis and CRS except for AFS, there are several studies that support the association between food allergies and CRS especially with nasal polyps [17]. These food allergies may not be present on either skin or blood tests and may instead require and elimination challenge diet to identify. This is performed by having the patient eliminate all of the food to be tested from their diet for a period of 5-10 days, and then re-expose or challenge themselves with the food and monitor for exacerbation of symptoms over the next 24 h. In adults in America, the most common foods identified are wheat and dairy. Less frequently one is able to detect sensitivities to corn, soy or egg. Abstinence from that food for months may resolve or reduce nasal polyps in selected patients.

## **Medical Therapy for Allergic Rhinitis**

The cornerstone for the treatment of AR is avoidance of the allergen that provokes symptoms. When environmental controls are impractical or incompletely effective, then pharmacotherapy is instituted. A wide variety of medications are available for the treatment of allergic rhinitis. Medications selected should be targeted toward the patient symptoms.

Medications effective for allergic rhinitis include:

- Topical and oral antihistamines
- · Topical and oral decongestants

- · Topical and systemic steroids
- Topical steroid/antihistamine combination
- Mast cells stabilizers (cromolyn)
- · Leukotriene receptor antagonists
- Anticholinergics
- Saline nasal rinses
- Immunotherapy

Intranasal glucocorticoids (INGs) provide the most efficacious effect, with the least morbidity, in the treatment of AR and are considered first-line therapy [18]. They have been shown to be equally or more effective than oral H1-antihistamines in AR [19]. INGs provide targeted therapeutic effects to the nasal mucosa and provide relief from nasal-related symptoms. In patients with frontal sinus obstruction or narrowing, topical steroids can be directed to the frontal recess with the neck hyperextended or flexed to maximize exposure of the frontal recess to the topical steroid as it is applied. Patients should always be educated in directing the steroid spray away from the septum and toward the lateral wall of the nose or up toward the frontal recess in order to minimize septal excoriation and bleeding and the very rare complication of septal perforation. The onset of action of nasal steroid sprays is approximately 7 h and reach maximal efficacy after 2 weeks [18]. They may also be more effective if initiated a few days to a week prior to the patient's pollen allergy season. Systemic effects of INGs are minimal; patients using INGs are at low risk of developing hypothalamic-pituitary-adrenal (HPA) suppression, skin thinning, glaucoma, or cataract formation. Side effects of INGs tend to occur locally. The most common side effects are headache, throat irritation, burning sensation, crusting, dryness, and/or minor epistaxis [20].

Oral antihistamines can be divided into sedating and non-sedating medications. Fexofenadine, loratadine, desloratadine, and cetirizine at recommended doses cause no sedation, with the exception of cetirizine, which is mildly sedating. All are effective for sneezing and itching symptoms; however, they have little impact on nasal congestion. For this reason, antihistamines are often paired with an intranasal corticosteroid and are considered a first-line management option for AR. Sedating antihistamines are available over-the-counter and have anticholinergic properties, which thicken sinus and nasal secretions and over dry the nose in some patients. Onset of action of oral antihistamines is relatively rapid, ranging from 20 min to 2 h with effects lasting up to 24 h.

Azelastine and olopatadine are FDA-approved topical antihistamine nasal sprays that have a symptom relief profile similar to that of nasal steroid sprays. The onset of action for topical nasal antihistamine sprays is within 15 min and the effects can last up to 4 h. Side effects include nasal irritation, bitter taste, headache, epistaxis, and a slight sedation potential. They are less efficacious that intra-nasal corticosteroids; however, they have fewer systemic side effects than oral antihistamines.

In 2012, The Food and Drug Administration approved a combination nasal steroid/antihistamine spray, azelastine hydrochloride/fluticasone propionate (Dymista<sup>™</sup>), for the treatment of seasonal AR. Three clinical trials demonstrated that the combination of the antihistamine and steroid provided greater symptomatic relief than did either medication individually [21].

Topical decongestants can be utilized for short periods of time, no longer than 3–5 days, to decongest the nose and to optimize drainage of the frontal recess. Topical decongestants cause vasoconstriction resulting in decreased inflammation by acting on adrenergic receptors. Onset of action is relatively rapid, with symptom relief in 5–10 min. Prolonged use, however, can lead to rebound swelling and rhinitis medicamentosa. This may be minimized with concurrent use of a topical nasal steroid spray [22]. Most practitioners do not recommend long-term use of oral decongestants because of associated adverse events.

Leukotriene receptor antagonists (montelukast, zafirlukast) are FDA-approved for the treatment of seasonal AR. Approved for use in asthma in 1996, leukotriene receptor antagonists also show efficacy in seasonal AR [23]. A meta-analysis of 11 studies evaluated the efficacy of LTRAs either alone or in combination with other treatments when compared to placebo. Overall, LTRA's were found to produce a small but statistically significant improvement in nasal symptoms and quality of life. There was no statistical significance between LTRA's and antihistamines, and LTRA's were found to be less effective than INGs [24].

## **Allergic Fungal Rhinosinusitis**

AFRS is a form of CRS. AFRS was first recognized in 1976 by Safirstein, noting aspergillus-positive sinus cultures, nasal polyposis, and crusting, a presentation similar to ABPA [25]. Subsequent investigation further described, pathologically, the eosinophilic mucin found in these patients, characterized by "clumps of necrotic eosinophils and other cellular debris within a background of pale, eosinophilic-to-basophilic, amorphous mucin. The necrotic cellular debris is frequently arranged in multilayered rows. Charcot-Leyden crystals, which appear hexagonal in cross section and bipyramidal in longitudinal section, were a consistent finding within the allergic mucin."[26]

The most commonly cited diagnostic criteria for AFRS [7], from Bent and Kuhn in 1994, are the following five findings:

- Type I (IgE) mediated hypersensitivity
- Nasal polyposis
- · Characteristic computed tomography findings
- · Eosinophilic mucin without fungal invasion into mucosa
- Positive fungal stain

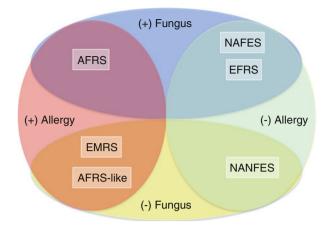
Patients with AFRS are most commonly young immunocompetent adults, living in warm or humid climates, with a history of IgE mediated allergy, nasal polyps on examination, and characteristic findings on CT, unilateral or bilateral. Orbital proptosis and telecanthus are characteristics of advanced disease. CT radiologic findings in the frontal sinus and other paranasal sinuses include bony erosion, opacified sinuses with central hyperattenuation thought to be due to the proteinaceous viscous nature of the inspissated secretions and sinus mucocele formation. Bone erosion can be along the skull base, periorbital regions, or involving other aspects of the bony paranasal sinus walls. The most common site of bony erosion is in the ethmoid sinuses and the lamina papyracea. MRI demonstrates central areas of low signal on T1 and T2 that correspond to areas of proteinaceous eosinophilic mucus. Mucosal inflammation has the appearance of a peripheral high-signal intensity. Research by Wise and colleagues demonstrate a gender and race predilection for AFRS bone erosion. In a retrospective review of 111 consecutive patients with AFRS from two southeastern tertiary care rhinology practices in the US, CT imaging was analyzed using a novel staging system to account for the characteristic bony erosion see in AFRS. These authors found that males and African-Americans with AFRS demonstrated significantly more bone erosion and therefore higher scores [27].

While CT imaging with characteristic bony erosion and physical findings of nasal polyps in an atopic patient may raise clinical suspicion for AFRS, the diagnosis can be supported with the histologic identification of allergic mucin, which grossly has been described as "peanut butter" or "axle grease."

Over the past 15 years, the role of fungal elements and its immunologic effect in CRS has been debated. It has been theorized that certain fungal proteins directly stimulate a Th2 response, causing release of interleukins 4 and 13 in patients with CRS. Shin and colleagues found that patients with CRS demonstrated an exaggerated humoral and cellular response, both Th1 and Th2, to common fungi, notably *Alternaria*, when compared to healthy controls [28]. These findings however have not been entirely replicated. While AFRS has classically been thought of as a type I hypersensitivity reaction, there is evidence that a type III hypersensitivity reaction also contributes to the pathophysiology. Shin et al also found that levels of IgG were elevated in patients with CRS in response to exposure to *Alternaria*, when compared to healthy controls.

AFRS constitutes a small subset of CRS patients. Questions have been raised regarding the presence of fungal elements in allergic mucin found in patients with AFRS. In 2000, a new clinical entity was proposed by BJ Ferguson, eosinophilic mucin rhinosinusitis (EMRS) [29]. Not all patients who were diagnosed with AFRS were found to have fungal elements on histologic evaluation of their allergic mucin. Patients that did not have fungal elements (i.e. EMRS) tended to be older, asthmatic, aspirin-sensitive, and have bilateral disease. They tended not to have AR and to have lower, though elevated, total IgE when compared to classically defined AFRS patients. These clinical differences led to the proposition that there are variations in patients who have classically been described as having AFRS.

Fungal elements are historically difficult to isolate and identify on histologic staining in the setting of eosinophilic mucin. The gold-standard Grocott-Gomori's (or Gömöri) methenamine silver (GMS) stain is not 100 % sensitive. A novel, more sensitive staining technique has recently been described by Guo and colleagues [30]. Eosinophilic mucin collected from allergic and non-allergic CRS patients were pre-digested with trypsin. The standard GMS stain detected fungi in 9 of 34 (27 %) specimens. GMS with trypsin digestion detected fungi in 31 of 34 (91 %) specimens. While this study provides evidence that with better staining techniques identification of fungal elements is improved, it also raises questions about the role



**Fig. 10.1** Diagram illustrating commonalities and differences in *AFRS* allergic fungal rhinosinusitis, *NAFES* non-allergic fungal eosinophilic sinusitis, *EFRS* eosinophilic fungal rhinosinusitis, *EMRS* eosinophilic mucin rhinosinusitis, *AFRS-like* similar to AFRS but without the presence of fungus, *NANFES* non-allergic non-fungal eosinophilic sinusitis

of IgE mediated pathophysiology. In this study, CRS patients without allergy were also found to have fungal elements in their eosinophilic mucin, which would support the creation of another clinical entity – eosinophilic fungal rhinosinusitis (EFRS). One must also wonder whether these more sensitive techniques are accomplishing the equivalent detection rates of saline lavage of the nose, in which 100 % of normal volunteers had recoverable fungus from the nose [31].

A consensus meeting was held in 2008 and the distinction between EMRS, AFRS, and EFRS was discussed. While sufficient evidence to clarify these entities is lacking, a consensus diagram was created [32]. Characterizing these various disease processes using a Venn diagram illustrates the similarities and differences of these clinical entities (Fig. 10.1).

## Treatment

Without a clear understanding of the pathophysiology of AFRS, appropriate and effective medical treatment remains controversial. However, once AFRS was determined to be a separate and unrelated entity from invasive fungal sinusitis, more refined, directed endoscopic sinus surgery has proven to be a mainstay of treatment.

Goals of surgical treatment for AFRS are as follows. Surgery should:

- Result in complete extirpation of all allergic mucin and fungal debris, thereby decreased the immunologic burden in atopic patients.
- Produce means of permanent drainage and ventilation of the affected sinuses while preserving the integrity of sinonasal mucosa.

• Allow for post-operative access to previously diseased areas to improve access for nasal irrigations and post-operative debridement.

It is worth mentioning that while the nasal anatomy can be distorted secondary to diffuse polyp growth, sinus ostia are subject to the same distortion and are often dilated, allowing for relatively easy access into the sinuses; one essentially follows the polyps to the mucin. The use of intraoperative image guidance can aid in surgery, improve completeness of surgery, and identify regions of bony dehiscence along the orbit and dura.

Surgical treatment for frontal sinus in AFRS typically includes thorough endoscopic sinus surgery, opening the frontal sinus ostium as widely as possible. If necessary for adequate ventilation, extended endoscopic frontal sinus procedures, such as a Draf IIb, can be considered. In this era of increased use of topical nasal medications and rinses for control of postoperative paranasal sinus edema and inflammation, widely patent paranasal sinus ostia are paramount for appropriate topical drug delivery.

• The recidivism of AFRS is high and patients should be followed closely, especially over the first year. If the frontal sinus cannot be adequately visualized endoscopically in the postoperative period despite appropriate topical therapy compliance or a burst of oral steroids for acute exacerbation, a total IgE or CT scan should be obtained to evaluate for potential recurrence.

Several factors have been found to be associated with failure of frontal sinusotomies in the setting of chronic inflammatory frontal sinus disease, but AFRS does not appear to be one of them. In a retrospective study of 66 consecutive patients who underwent frontal sinusotomies, Chandra and colleagues found that those patients who had advanced pre-operative disease and who had had prior surgery had a higher rate of failure from endoscopic frontal sinusotomies. Of note, nasal polyps, asthma, aspirin sensitivity, and AFRS was not associated with failure of endoscopic frontal sinusotomies [33]. These findings were corroborated by Gupta et al. who found that, in 34 patients with AFRS studied prospectively, preoperative frontal sinus opacification and sphenoid sinus opacification were predictors of increased chance of disease recurrence after surgery, done either endoscopically or externally [34]. In 2012, Naidoo et al. performed a retrospective chart review of patients who underwent endoscopic frontal sinusotomies [35]. Stenosis of the frontal sinus ostium correlated with persistence of symptoms; the presence of eosinophilic mucin, chronic rhinosinusitis, asthma, and allergy did not.

Adjunctive medical therapies for AFRS include

- Systemic and topical steroids
- · Antifungal therapy
- · Leukotriene modulators
- · Saline rinses
- · Immunotherapy
- Environmental controls that limit fungal exposure

With frontal AFRS, it is unlikely that nasal steroid sprays will reach the frontal sinus. Therefore, alternate topical nasal steroid preparations or systemic steroids are often recommended. For AFRS of the remaining sinuses, some investigators recommend three times the standard dose [36]. Budesonide respules, applied topically to the nasal cavity with greater distribution than standard topical spray, either by drops, atomizer, or rinse, have also been found to result in moderate to significant improvement in symptoms resulting in lower doses of oral steroids needed in postoperative patients with CRS, however atomized budesonide has not been directly studied in patients with AFRS [37].

Preoperative systemic corticosteroid therapy may be initiated approximately 1 week prior to surgery (0/5–1.0 mg/kg prednisone per day) with the aim of decreasing the size of nasal polyps and decrease bleeding [38]. Several placebo-controlled trials have demonstrated that systemic anti-inflammatory agents appear to be an effective medical therapy [39]. Because of the possible serious side effects of long-term steroid use, it is recommended that steroids be given during the perioperative period and subsequent post-operative period in bursts to help control nasal polyposis. In general, no additional benefit is achieved with prednisone dosage equivalents in excess of 60 mg per day, which approximate the maximal natural steroid surge in a stress response. Descending tapers over 10–30 days are frequently employed and some advocate a year of prednisone tapered down to 5 mg every other day [40]. Steroids should be dosed in the morning to minimize hypothalamic/pitu-itary suppression.

Short-term consequences of steroid usage include:

- Personality changes
- Hyperglycemia
- · Increased risk for gastric ulcer
- Slight increase in risk for avascular necrosis of the hip

Long-term consequences of systemic steroid usage include:

- Growth retardation in children
- Osteoporosis
- Glaucoma
- Cataracts

The role of systemic and topical antifungal therapy in AFRS is controversial. In the pulmonary form of the disease, allergic bronchopulmonary aspergillosis, (ABPA) systemic oral itraconazole resulted in statistically significant reductions in medication usage and total IgE in a randomized placebo controlled trial [41]. In AFRS, systemic itraconazole was found to trend toward better outcomes postoperatively, though was not statistically significantly different than topical steroids or nasal saline irrigations [34].

Topical antifungal therapy in chronic rhinosinusitis with Amphotericin B was shown to result in a 70 % improvement in symptoms in a non controlled trial [42]. A subsequent randomized, blinded controlled trial with a smaller quantity of antifungal irrigation showed no significant differences in the antifungal or placebo groups [43].

Immunotherapy to fungal antigens in surgically treated AFRS reduces recurrence in the initial few years, however long term follow up of these patients reveals that whether or not they receive immunotherapy, most patients improve after 4–10 years [44]. In a 2012 review of available literature to date, Hall and deShazo report on several small studies that taken together lend promising results [45]. They also found that high-dose immunotherapy used in patients with AFRS was well tolerated and unlikely to cause adverse reactions other than those occurring in patients with pollen immunotherapy. High-dose immunotherapy is typically initiated 1 month postoperatively and is continued for 3–5 years. Treatment consists of weekly injections increased to reach a maximally tolerated dose. The mechanism of action of immunotherapy is thought to decrease production of allergen-specific IgE and the production of IgG4 blocking antibodies that interfere with IgE antigen reaction [46].

Case reports of remarkable improvement of ABPA with omaluzumab (Xolair), a humanized anti IgE, have led to its use in some cases of AFRS with improvement [47]. Omaluzumab requires monthly bi- month injections in a physician's office and may cost up to \$10,000 or more a year.

## Conclusions

AR may be seen in patients with recurrent acute frontal sinusitis and chronic frontal sinusitis. The diagnosis of AR depends on history, skin and in vitro allergy testing. Optimal therapy for AR includes the identification and elimination of the allergen exposure. This is facilitated by allergy testing and elimination challenge food diets. Pharmacotherapy should be targeted toward the allergic symptoms. Immunotherapy can be utilized in patients who failed to achieve adequate symptom relief with environmental controls and pharmacotherapy or who have symptoms for the larger part of the year. The role of allergy in AFRS continues to be the subject of some debate. Surgery, with possible revision, remains a mainstay of treatment. Systemic steroids can be used during the perioperative period and in short bursts in the post-operative period to shrink polyps. Immunotherapy may reduce recurrence of AFRS in the first several years following surgical extirpation.

## References

- 1. World Allergy Organization. WAO White book on allergy 2011–2012: Executive summary. Pawankar R, Canonica GW, Holgate ST, Lockey RF.
- Lill C, Loader B, Seemann R, Zumtobel M, Brunner M, Heiduschka G, Thurnher D. Milk allergy is frequent in patients with chronic sinusitis and nasal polyposis. Am J Rhinol Allergy. 2011;25(6):e221–4.
- Cody DT, Neel HB, Ferreiro JA, Roberts GD. Allergic fungal sinusitis: the mayo clinic experience. Laryngoscope. 2004;104:1074–9.
- 4. Goh BS, Gendeh BS, Rose IM, Pit S, Samad SA. Prevalence of allergic fungal sinusitis in refractory chronic rhinosinusitis in adult Malaysians. Otolaryngol Head Neck Surg. 2005;133(1):27–31.

- Bakhshaee M, Fereidouni M, Mohajer MN, et al. Prevalence of allergic fungal rhinosiusitis in sinonasal polyposis. Eur Arch Otorhinolaryngol. 2013;270:3095–8 [Epub ahead of print, accessed March 30, 2013].
- 6. Laila M. Telmesani prevalence of allergic fungal sinusitis among patients with nasal polyps. Ann Saudi Med. 2009;29(3):212–4.
- Bent 3rd JP, Kuhn FA. Diagnosis of allergic fungal sinusitis. Otolaryngol Head Neck Surg. 1994;111(5):580–8.
- Emanuel IA, Shah SB. Chronic rhinosinusitis: allergy and sinus computed tomography relationships. Otolaryngol Head Neck Surg. 2000;123(6):687–91.
- Ruoppi P, Seppa J, Nuutinen J. Acute frontal sinusitis: etiological factors and treatment outcome. Acta Otolaryngol. 1993;113(2):201–5.
- Wide K, Suonpaa J, Paippala P. Recurrent and prolonged frontal sinusitis. Clin Otolaryngol. 2004;29:59–65.
- 11. Slavin RG, Spector SL, Bernstein IL, et al. American Academy of allergy, asthma and immunology, the American College of allergy, asthma and immunology, and the joint council of allergy, asthma and immunology. The diagnosis and management of sinusitis: a practice parameter update. J Allergy Clin Immunol. 2005;116(6,suppl):S13–47.
- Rosenfeld RM, Andes D, Bhattacharyya N, et al. Clinical practice guideline: adult sinusitis. Otolaryngol Head Neck Surg. 2007;137(3, suppl):S1–31.
- Lehr AJ, Mabry RL, Mabry CS. The screening RAST: is it a valid concept? Otolaryngol Head Neck Surg. 1997;117:54–5.
- 14. Chambers DW, Cook PR, Nishioka GJ, Erhart P. Comparison of mRAST and CAP with skin endpoint titration for *Alternaria tenius* and *Dermatophagoides pteronyssinus*. Otolaryngol Head Neck Surg. 1997;117:471–4.
- Levine JL, Mabry RL, Mabry CS. Comparison of multitest device skin testing and modified RAST results. Otolaryngol Head Neck Surg. 1998;118:797–9.
- Tezer M, Tahamiler R, Canakcioglu S. Computed tomography findings in chronic rhinosinusitis patients with and without allergy. Asian Pac J Allergy Immunol. 2006;24(2–3):123–7.
- Fokkens WJ, Lund VJ, Mullol J, et al. EPOS 2012: European position paper on rhinosinusitis and nasal polyps 2012. A summary for otorhinolaryngologists. Rhinology. 2012;50(1):1–12.
- Bousquet J, Van Cauwenberge P, Bachert C. Requirements for medications commonly used in the treatment of allergic rhinitis. European Academy of Allergy and Clinical Immunology (EAACI), Allergic Rhinitis and its Impact on Asthma (ARIA). Allergy. 2003;58(3):192–7.
- Weiner JM, Abramson MJ, Puy RM. Intranasal corticosteroids versus oral H1 receptor antagonists in allergic rhinitis: systemic review of randomized controlled trials. BMJ. 1998;317(7173):1624–9.
- Demoly P. Safety of intranasal corticosteroids in acute rhinosinusitis. Am J Otolaryngol. 2008;29(6):403–13.
- Azelastine/Fluticasone Propionate (Dymista) for seasonal allergic rhinitis. The medical letter on drugs and therapeutics. 29 Oct 2012 (Issue 1402) p. 85 (Accessed on 31 Mar 2013 at http:// secure.medicalletter.org/TML-article-1402a).
- 22. Ferguson BJ, Paramaesvaran S, Rubinstein E. A study of the effect of nasal steroid sprays in perennial allergic rhinitis patients with rhinitis medicamentosa. Otolaryngol Head Neck Surg. 2001;125(3):253–60.
- Suonpaa J, Antila J. Increase of acute frontal sinusitis in Southwestern Finland. Scand J Infect Dis. 1990;22:563–8.
- Wilson AM, O'Byrne PM, Parameswaran K. Leukotriene receptor antagonish for allergic rhinitis: a systemic review and meta-analysis. Am J Med. 2004;116:338–44.
- Safirstein BH. Allergic bronchopulmonary aspergillosis with obstruction of the upper respiratory tract. Chest. 1976;70(6):788–90.
- Katzenstein AL, Sale SR, Greenberger PA. Allergic Aspergillus sinusitis: a newly recognized form of sinusitis. J Allergy Clin Immunol. 1983;72(1):89–93.
- Wise SK, Rogers GA, Ghegan MD, et al. Radiologic staging system for allergic fungal rhinosinusitis (AFRS). Otolaryngol Head Neck Surg. 2009;140(5):735–40.

- Shin SH, Ponkiau JU, Sherris DA, et al. Chronic rhinosinusitis: an enhanced immune response to ubiquitous airborne fungi. J Allergy Clin Immunol. 2004;114:1369–75.
- 29. Ferguson BJ. Eosinphilic Mucin Rhinosinusitis: a distinct clinicopathological entity. Laryngoscope. 2000;110:799–813.
- 30. Guo G, Ghadersohi S, Kephart G, et al. Improving the detection of fungi in eosinophilic mucin: seeing what we could not see before. Otolaryngol Head Neck Surg. 2012;147(5):943–9.
- Ponikau JU, Sherris DA, Kern EB, et al. The diagnosis and incidence of allergic fungal sinusitis. Mayo Clin Proc. 1999;74:877–84.
- Chakrabarti A, Denning DW, Ferguson BJ, et al. Fungal rhinosinusitis: a categorization and definitional schema addressing current controversies. Laryngoscope. 2009;119(9):1809–18.
- Chandra RK, Palmer JN, Tangsujarittham T, Kennedy DW. Factors associated with failure of frontal sinusotomy in the early follow-up period. Otolaryngol Head Neck Surg. 2004;131:514–8.
- Gupta RP, Bahadur S, Thakar A, Handa KK, Sarkaar C. Management protocols of allergic fungal sinusitis. Indian J Otolaryngol Head Neck Surg. 2007;59(1):35–40.
- Naidoo Y, Weh D, Bassiouni A, Keen M, Wormald PJ. Long-term results after primary frontal sinus surgery. Int Forum Allergy Rhinol. 2012;2(3):185–90.
- 36. Kuhn FA, Javer AR. Allergic fungal sinusitis: a four year follow-up. Am J Rhinol. 2000;14:49–56.
- Kanowitz SJ, Batra PS, Citardi MJ. Topical budesonide via mucosa atomization device in refractory postoperative chronic rhinosinusitis. Otolaryngol Head Neck Surg. 2008;139:131–6.
- 38. Marple BR. Allergic fungal rhinosinusitis: current theories and management strategies. Laryngoscope. 2001;111:1006–19.
- Hissaria P, Smith W, Wormald PJ, et al. A short course of systemic steroids in sinonasal polyposis: a double-blind, randomized, placebo controlled trial with evaluation of outcome measures. J Allergy Clin Immunol. 2006;118:128–33.
- 40. Schubert MS. Allergic fungal sinusitis: pathogenesis and management strategies. Drugs. 2004;64(4):363–74.
- 41. Stevens DA, Schwartz HJ, Lee JY, Moskovitz BL, Jerome DC, Catanzaro A, Bamberger DM, Weinmann AJ, Tuazon CU, Judson MA, Platts-Mills TAE, DeGraff AC. A randomized trial of itraconazole in allergic bronchopulmonary aspergillosis. N Engl J Med. 2000;342:756–62.
- 42. Ponikau J, Sherris D, Kita H, Kern E. Intranasal antifungal treatment in 51 patients with chronic rhinosinusitis. Allergy Clin Immunol. 2002;110:862–6.
- Weschta M, Rimek D, Formanek M, Polzehl D, Podbielski A, Riechelmann H. Topical antifungal treatment of chronic rhinosinusitis with nasal polyps: a randomized, double-blind clinical trial. J Allergy Clin Immunol. 2001;113:1122–8.
- Marple B, Newcomer M, Schwade N, Mabry R. Natural history of allergic fungal rhinosinusitis: a 4- to 10-year follow-up. Otolaryngol Head Neck Surg. 2002;127(5):361–6.
- Hall AG, deShazo RD. Immunotherapy for allergic fungal sinusitis. Curr Opin Allergy Clin Immunol. 2012;12:629–34.
- 46. Ferguson BJ. What role do systemic corticosteroids, immunotherapy, and antifungal drugs play in the therapy of allergic fungal rhinosinusitis? Arch Otolaryngol Head Neck Surg. 1998;14:1174–8.
- Collins J, Devos G, Hudes G, Rosenstreich D. Allergic bronchopulmonary aspergillosis treated successfully for one year with omalizumab. J Asthma Allergy. 2012;5:65–70. doi:10.2147/ JAA.S34579. Epub 2012 Nov 8.

# Chapter 11 The Role of Fungus in Diseases of the Frontal Sinus

Nathan A. Deckard, Bradley F. Marple, and Pete S. Batra

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Financial Disclosures Deckard: None Marple: Batra: Research grants (ARS, Medtronic), consultant (Medtronic)

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_11

# Introduction

The nose and paranasal sinuses can be hosts to a variety of disease states, of which fungal species are an increasingly well-understood etiologic agent. Over the past 35 years, enhanced understanding of the role of fungus in sinus disease and the complex interactions between host and pathogen have allowed for a logical classification of fungal rhinosinusitis facilitating proper prognostic information and therapeutic intervention. Coincident with this same time period is the introduction and popularization of minimally invasive endoscopic techniques to better understand frontal sinus anatomy and address pathologic conditions. As such, fungal rhinosinusitis involving the frontal sinus is now more amenable to appropriate treatment with endoscopic approaches.

## **Basic Mycology**

Fungi are eukaryotic organisms ubiquitous to our environment and the human body. Scientists estimate the total number of different fungal species ranges between 20,000 and 1.5 million, of which approximately 400 are responsible for human illnesses, perhaps with only a few dozen species responsible for over 90 % of infections [26, 39, 48]. Fungi can exist either as yeast or molds.

Characteristically, molds produce *hyphae*, multicellular, branching tubular extensions (2–10  $\mu$ m in diameter), which coalesce as a colony known as a *mycelium* [40]. Yeasts are unicellular, from 3 to 15  $\mu$ m in diameter, and reproduce asexually via budding; though failure of buds to detach can result in a characteristic chain of fungal cells known as *pseudohyphae* [40]. The spore is fungi's evolutionary solution to the survival problems posed by unfavorable conditions. These derivatives of sexual or asexual fungal reproduction disperse readily into the environment, can withstand adverse surroundings, and retain their germinative abilities until more receptive surroundings are encountered. Inhalation of spores is the most common route by which fungal rhinosinusitis is initiated. Once the nasal mucosa has been accessed, development of a pathologic condition is determined not only by the inherent characteristics of the fungus, but by the host's immune system and the complex interplay between the two.

## **Classification of Fungal Rhinosinusitis**

Fungal disease of the nose and paranasal sinuses can be classified based on the clinical, radiologic, and histologic manifestations of the host-pathogen relationship. Most commonly accepted classification schemes divide fungal rhinosinusitis into invasive and non-invasive diseases based solely on histopathologic evidence of fungus penetrating host tissue (Table 11.1) [11].

Table 11.1       Classification of fungal rhinosinusitis	Invasive fungal sinusitis
	Acute fulminant invasive fungal sinusitis
	Granulomatous invasive fungal sinusitis
	Chronic invasive fungal sinusitis
	Non-invasive fungal sinusitis
	Saprophytic fugal infestation
	Fungal ball
	Allergic fungal rhinosinusitis
	Ferguson [13]

# Acute Fulminant Invasive Fungal Sinusitis

The characteristics of acute fulminant invasive fungal sinusitis (AFIFS) are as follows:

- A clinical time course of less than 4 weeks duration.
- Prominent pathologic evidence of vascular invasion, which may include hyphal invasion of blood vessels, such as the carotid artery and cavernous sinus, vasculitis with thrombosis, and tissue infarction [6, 13].
- The genus *Aspergillus* and the class zygomycetes are responsible for most cases of AFIFS [6].
- AFIFS is almost always seen in immunocompromised patients, though it has been occasionally been reported in patients with normal immune function [4].
- Conditions associated with impaired neutrophil function or neutropenia, such as hemochromatosis, uncontrolled diabetes mellitus, AIDS, hematologic malignancies, or those undergoing iatrogenic immunosuppression from anti-neoplastic chemotherapy or following transplantation, are particularly prone to development of AFIFS [10, 18].
- A high index of suspicion for invasive disease must be maintained in the immunocompromised patient with symptoms of rhinosinusitis, as early findings are often subtle.

# **Clinical Presentation**

Patients may present with:

- Facial swelling is the most commonly reported finding according to a recent systematic review [54].
- Fever of unknown origin, present in 50–90 % of patients in the 3 days prior to diagnosis [18, 57].
- Rhinorrhea
- Double vision
- Ophthalmoplegia

- · Headache or facial pain
- Hypoesthesia or anesthesia of the face or oral cavity. This is a particularly concerning sign for early invasive disease and can precede mucosal changes. Patients should be questioned specifically and facial sensation must be tested accurately to identify neurologic deficits [14].

Timely endoscopic exam and directed biopsies are indicated in any immunocompromised patient with facial anesthesia or above signs and symptoms that fail to improve despite appropriate medical therapy [14, 18, 19]. Endoscopic findings will change drastically as the disease progresses. Alterations in the visualized nasal mucosa may be subtle early in the course of AFIFS; however, nasal mucosa changes are the most consistent physical finding and should always be investigated carefully with nasal endoscopy. Mucosal abnormalities are most commonly noted at the middle turbinate (67 %), followed by the nasal septum (24 %) [18]. Pale mucosa with evidence of decreased bleeding or sensation may be reflective of tissue ischemia and incipient fungal angioinvasion [9, 18, 19]. The natural history of AFIFS leads to extrasinus involvement and more obvious findings in later stages of the disease.

Findings seen in later stages of the disease include:

- · Necrotic nasal and/or palate mucosa
- · Densely anesthetic regions of the face
- Proptosis
- · Ophthalmoplegia
- · Decreased vision
- Mental status changes

#### Radiology

Diagnostic imaging of the paranasal sinuses is often performed in the work-up of patients with presumed or proven AFIFS. High-resolution, non-contrasted CT scan of the sinuses in axial and coronal planes is required to adequately evaluate sinus anatomy and the extent of disease. MRI is recommended in patients who present with signs or symptoms of orbital or intracranial involvement, or in those with skull base or lamina papyracea erosion noted on CT scan. Although bone erosion and extrasinus extension are historically cited as classic findings of AFIFS, recent investigations have shown severe unilateral thickening of nasal cavity mucosa to be the most consistent CT finding suggestive of early IFS; yet this is a non-specific finding [9]. Others have suggested thickening of peri-antral fat planes as another early indicator of AFIFS; however, most authors have found this finding to be either non-specific or too uncommonly encountered in AFIFS to assist in providing diagnostic assistance [9].

#### **Treatment of Acute Fulminant Invasive Fungal Sinusitis**

The most important treatment for AFIFS is reversal of the patient's underlying immunocompromised state if possible. Otherwise, treatment of AFIFS relies on medical and surgical therapy directed against the offending fungal pathogen. Operative debridement decreases the fungal load and removes necrotic tissue. Endoscopic techniques directed to completely address the sinonasal disease process, are favored to aggressive radical resections of disease beyond the confines of the sinonasal cavity [18, 24]. Systemic antifungal therapy is routinely employed in AFIFS as an adjunct to surgery. Liposomal formulations of amphotericin-B, the mainstay of antifungal therapy for over 50 years, have improved safety profiles, less renal toxicity, and are effective in treating AFIFS [18, 55]. The topical route of administration via nasal irrigations or nebulizer may enhance delivery of drug within the sinonasal cavity and should be considered in AFIFS patients [14]. Azole antifungal medications, echinocandins, and iron chelating agents may be used as alternative medications in select patients [7].

The prognosis of AFIFS is heavily dependent on the patient's immune status, as those who recover neutrophil function have the greatest chance of survival [24]. Patients with hematologic malignancies have typically been thought to have lower survival (20–50 %), as their immune deficiency is not amenable to rapid improvement [14, 18]. However, in a recent systematic review, survival of patients with hematologic malignancies was virtually identical to that of the entire patient cohort, with overall survival for all AFIFS patients was 46.1 %. Diabetics, in general, did continue to do better than non-diabetics with a survival rate of 50.75 % (p<0.003, OR 0.492), presumably due to the potential reversibility of their underlying disorder, while the lowest survival rates were seen in patients with altered mental status (9.1 %), aplastic anemia (20 %), and renal/liver failure (23.8 %) [54].

#### Acute Fulminant Invasive Fungal Sinusitis and the Frontal Sinus

The frontal sinus is the most unlikely site of involvement in AFIFS, as only 4.8 % of cases in a large series demonstrated definitive histopathologic changes, and never in isolation from the other paranasal sinuses [19]. Though outcomes specifically for frontal sinus AFIFS are not reported in the literature, its proximity to the intracranial space would give AFIFS significant potential for untoward outcomes. Extended endoscopic techniques, such as the endoscopic modified Lothrop or Draf IIb, provide wide exposure of the frontal sinus to facilitate adequate biopsies and thorough debridement. Open frontal approaches, such as an osteoplastic flap, may be considered for wide exposure of the frontal sinus; however, this approach should be considered as a fallback option and the sinus must never be obliterated when addressing AFIFS. Wide access to the frontal sinus allows the surgeon clear access to both perform postoperative surveillance with routine office endoscopy as well as deliver topical antifungal medication via irrigations or nebulizer.

## Chronic Invasive Fungal Sinusitis

Chronic invasive fungal sinusitis (CIFS) is a slowly progressive fungal infection with a typical time course over 12 months. This is further subdivided into granulomatous invasive fungal sinusitis (GIFS) and chronic invasive fungal sinusitis (CIFS) based on histopathology [10]. GIFS is a rare entity that is largely reported in Sudan, Saudi Arabia, and the Indian subcontinent. *Aspergillus flavus* is the most common fungus isolated in these patients [6]. It typically presents with an enlarging mass in the cheek, orbit, nose, and paranasal sinuses in immunocompetent hosts, with proptosis being a prominent feature. Histopathologically, a granulomatous response is seen with considerable fibrosis.

In contrast, CIFS is a slowly destructive process that most commonly affects the ethmoid and sphenoid sinuses, but may involve any of the paranasal sinuses. Histologically, it is characterized by dense accumulation of hyphae, occasional presence of vascular invasion, and sparse inflammatory reaction. The process is usually seen in the context of AIDS, diabetes mellitus, and corticosteroid treatment. Tissue cultures are positive in >50 % of cases, and *Aspergillus fumigatus* is the most commonly isolated agent [11, 41]. Most authors regard GIFS and CIFS as identical with respect to the, diagnostic evaluation, treatment options, and clinical course [6, 11, 53].

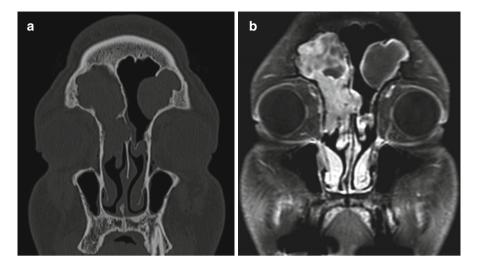
Typical patient presentation includes symptoms of chronic rhinosinusitis (CRS), made remarkable by their long duration, slow progression, and refractoriness to standard therapy. Patients are usually immunocompetent and, therefore, it is not until the development of associated ophthalmologic or neurologic findings, such as facial paresthesias, seizures, altered mental status, proptosis, or vision changes, that alternate diagnostic possibilities like GIFS or CIFS are explored [53].

Because of the chronicity of CIFS, coupled with concerning neurologic or ophthalmologic deficits, the differential diagnosis should include [47, 53]:

- Malignant processes
- · Benign neoplasms
- Autoimmune disease
- Intracranial pathology
- · Orbital neoplasms
- · Unusual sinonasal infectious agents

#### Diagnosis

Diagnostic evaluation should begin with a complete head and neck exam, including nasal endoscopy and biopsy, as well as careful neurologic evaluation with cranial nerve testing to determine the extent of imaging that will be required initially. Neurologic or ophthalmologic deficits warrant a contrast enhanced MRI of the brain, orbit, and sinuses to evaluate for intracranial and orbital extension in addition to high-resolution coronal and axial CT scan of the sinuses to delineate the extent of paranasal sinus disease (Fig. 11.1a, b). Mucosal thickening and bone erosion may be noted and can mimic neoplastic lesions. MRI is useful in assessing dural and intracranial extension [22, 53]. However, a diagnosis of invasive fungal disease can only be established on histopathologic grounds, though imaging may shorten the differential diagnosis and guide directed biopsies [53].



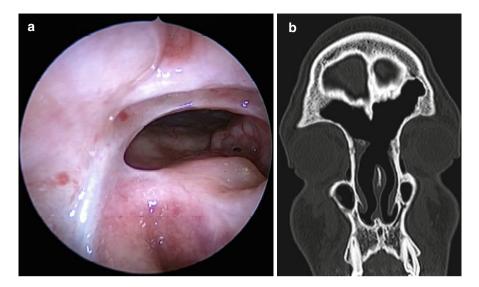
**Fig. 11.1** (a) Coronal bone window CT scan demonstrates complete right frontal opacification in patient with known chronic granulomatous fungal rhinosinusitis (b). T1- weighted MRI with contrast demonstrates enhancing lesion in the *right* frontal sinus. In contrast, the *left* frontal sinus has a mucous retention cyst

#### **Treatment of Chronic Invasive Fungal Sinusitis**

The extent of surgery necessary to control CIFS is a point of controversy, as is the need for and duration of concomitant antifungals. A minority of authors draw a distinction between granulomatous and non-granulomatous CIFS, treating the non-granulomatous variety with aggressive surgery and antifungals as for AFIFS, with surgery alone being reserved for GIFS [11, 41]. The majority opinion favors debridement of all non-viable sinus tissue, preservation of as much normal anatomy as possible, and allowing prolonged culture-guided systemic antifungal medications to eliminate the remaining fungal infection [53]. Though the literature lacks definitive recommendations for duration of systemic antifungal therapy in CIFS, it may be possible to transition some postoperative patients to topical antifungal irrigations in an effort to avoid the renal toxicity of long-term amphotericin B.

#### Chronic Invasive Fungal Sinusitis and the Frontal Sinus

CIFS of the frontal sinus is not a well-documented entity, thus it is not clear that diagnostic or treatment strategies would vary significantly from those described for the other paranasal sinuses. Patients with symptoms of CRS refractory to medical therapy, especially persistent headache, visual changes or development of neurologic deficits require expeditious physical evaluation and appropriate imaging. Invasive infections of the frontal sinus have a predilection for early involvement of the intracranial space, either directly via bone erosion or angioinvasion of vessels



**Fig. 11.2** (a, b) Endoscopic view at 1-year demonstrates patent frontal neo-ostium after Lothrop procedure. Corresponding coronal bone window CT demonstrates excellent frontal aeration

that traverse the posterior table. Aggressive surgical therapy is recommended to resect all visible frontal sinus disease and establish healthy tissue margins. An endoscopic approach is favored, with careful consideration of an osteoplastic flap to ensure clearance of all disease (Fig. 11.2a, b). Postoperative antifungal medication is initiated systemically, with conversion to topical irrigations as dictated by clinical response and follow-up endoscopy.

# Paranasal Sinus Fungal Ball

Fungal ball (FB) best typifies non-invasive fungal disease of the paranasal sinuses, a condition resulting from sequestration of densely tangled, concentrically arranged masses of fungal hyphal elements within a sinus in the absence of mucosal invasion [12]. FB (formerly, and inaccurately, referred to as "mycetoma") has been reported since the late nineteenth century, though most early case series have been small owing to the relative infrequency of this condition. One series estimates FB represents 3.7 % of inflammatory sinus conditions [17]. Patients with FB are typically females (2.97:1, female:male ratio) with mean age of 52.7 years (range 19–85 years). The maxillary sinus is the most frequently affected (84.4 %), followed by the sphenoid sinus (14.4 %) [42]. Ethmoid and, especially, frontal sinus involvement is rare.

Fig. 11.3 Coronal CT scan demonstrates right frontal fungal ball with multiple areas of hyperdensity. This was cleared via endoscopic frontal sinusotomy



#### **Clinical Presentation**

Medical attention is typically sought for symptoms suggestive of CRS, with symptoms including facial pain or headache, nasal airway obstruction, or purulent rhinorrhea localizing to the side of the fungal ball [13, 15]. Patients with maxillary FB may present with facial or dental pain, initially being misdiagnosed as an odontogenic process. Sphenoid FB may present with vertex headaches and non-specific postnasal drainage, highlighting the need for imaging to elucidate proper diagnosis. Nasal endoscopy may demonstrate polyp disease in only 10 % of patients, and is more likely to show normal to mild mucosal inflammation without evidence of fungus or other revealing characteristics [25].

#### Radiology

CT scan of the paranasal sinuses is the study of choice for diagnosis of FBs, though imaging is certainly not diagnostic. Single sinus involvement is reported in 59–94 % of FB cases, almost always with near complete opacification of the involved sinus, and frequently demonstrating hyperdensity within the opacification (41 %) (Fig. 11.3) [17, 25]. Bony sclerosis of the involved sinus is common, as radiographic

evidence of this bony thickening is noted in 33–62 % in different case series [17]. In contrast, bony erosion, commonly seen in AFRS, is noted in only 3.6–17 % of CT scans of FB patients [17, 25].

#### **Treatment of the Paranasal Sinus Fungal Ball**

Complete surgical removal of the FB, with thorough irrigation of involved sinus and establishment of sinus ventilation, constitutes treatment of choice for this non-invasive fungal disease. Endoscopic techniques are usually sufficient to achieve these surgical objectives. Recent studies report recurrence rates of 3.7–6.8 % in those patients treated endoscopically [17, 25]. Postoperative antifungal therapy is not necessary unless the patient suffers from comorbid conditions with predisposition to compromised immune function. Progression from FB to AFIFS has been reported in patients with blood dyscrasias, diabetes mellitus, systemic corticosteroids, or other similar conditions associated with immunodeficiency [15]. In these patients, antifungal selection should be guided by fungal histology and culture results to identify the least toxic, most cost-effective agent available. Amphotericin B formulations should be restricted to cases in which culture results suggest resistance to imidazole antifungals [15].

#### Paranasal Sinus Fungal Ball and the Frontal Sinus

Frontal sinus involvement with FB is distinctly unusual. The first case of FB isolated to the frontal sinus was reported in 1978, successfully treated solely by removal via an osteoplastic flap approach [52]. Other studies attest to the relative rarity of this condition. Ferreiro reported an incidence of 21 % for FB involving the frontal sinus, with only 7 % of patients having disease isolated to this site alone [17]. Klossek et al. noted frontal sinus location in only 1.8 % of 109 patients with FB [25]. Difficult locations within the frontal sinus were addressed via a complete endoscopic anterior ethmoidectomy combined with irrigations through the anterior wall of the frontal sinus, successfully treating both cases of frontal sinus FB [25]. Indeed, the frontal sinus poses a significant surgical challenge for successful evacuation of a FB. Endoscopic frontal sinusotomy may be sufficient for successful extirpation of frontal sinus FB. This can be extended to a Draf IIb or III procedure based on the amount of frontal access required to achieve the surgical goals. Endoscopic frontal trephination may also serve as an additional porthole for irrigation of fungus in a difficult to reach frontal location. Osteoplastic frontal flap should be used as an absolute last resort for frontal FB; obliteration is contraindicated given it precludes the ability to monitor recurrent disease.

## Allergic Fungal Rhinosinusitis

Allergic fungal rhinosinusitis (AFRS) was initially described by Safirstein in 1976 who reported on a 24-year-old woman with recurrent nasal obstruction, mucosal ulcerations, thick secretions within the nose, and culture evidence of *Aspergillus* that resembled the clinico-pathologic findings of allergic bronchopulmonary Aspergillosis (ABPA) [46]. Several early authors further reported on these findings helping clarify this as a distinct disease entity [23, 39, 45]. Millar and colleagues reported on similarities between material obtained from the maxillary sinuses of five patients and pathologically diagnosed specimens of ABPA [39]. Katzenstein et al. retrospectively reviewed 113 consecutive cases, identifying seven young adults with asthma and nasal polyposis with similar findings and termed the condition allergic *Aspergillus* sinusitis [23]. Though *Aspergillus* was almost exclusively associated with the disorder in early descriptions, later studies have demonstrated that the dematiaceous family of fungus is present in a majority of cases of AFRS, giving credence to a more generalized term [30].

#### Pathogenesis

Despite improved understanding of the disease process and advances in treatment of AFRS, no single unifying explanation exists for the pathogenesis of AFRS. A popular theory, referred to as "the AFRS cycle," offers a preliminary construct through which the multifactorial process can be better understood. The theory posits AFRS as the sinonasal correlate of ABPA and depicts a cascading inflammatory cycle resulting in the diagnostic characteristics of AFRS [33, 34, 36, 37]. Disease initiation requires fungal antigens inhaled by an atopic host to generate Gel and Coombs type I (IgE) and, possibly, type III (immune-complex) reactions, which induce an intense eosinophilic inflammatory response. Increased IgE levels can be seen both systemically and within the eosinophilic mucin [8]. Patency of sinus ostia is compromised and resultant stasis facilitates fungal proliferation and production of viscid fungal mucin. This mucin accumulates within sinuses producing further obstruction perpetuating the AFRS cycle [21, 33, 36, 37].

Sequestered collections of mucin, the hallmark of AFRS, provoke changes in the effected sinuses consistent with those usually attributed to mucoceles [5, 36, 44]:

- Bony remodeling
- Bony erosion
- Extension into contiguous anatomic spaces

Persistence of the disease state allows inflammatory mediators to slowly damage the sinonasal mucosa [26]. These inflammatory mediators are:

- · Major basic protein
- Eosinophil cationic protein

- Eosinophil peroxidase
- Eosinophil derived neurotoxin
- Tumor-necrosis factor-beta
- Interleukins 4, 5, 10, and 13

#### Epidemiology

AFRS is more commonly diagnosed in younger populations (average age 21.9–42.4 years) and may represent 5–10 % of all patients undergoing surgery for CRS [30, 36, 37]. Manning has suggested a slight male preponderance (1.6:1), though this is not borne out in other reviews [30]. AFRS also appears to disproportionately affect African Americans and patients of low socioeconomic class [56]. Multiple studies have depicted AFRS to have a geographic variability favoring temperate regions with relatively high humidity, especially Texas, the Mississippi River basin, and portions of the American southeast and southwest where AFRS may represent upwards of 20 % of all patients undergoing surgery for CRS [16].

### **Clinical Features**

The unrelenting inflammation of AFRS can result in a host of patient signs and symptoms. Typical presentation includes unilateral symptoms suggestive of underlying CRS. Unchecked AFRS may lead to [5, 32, 34, 44]:

- Diplopia
- Proptosis
- Blindness
- Facial dysmorphia (hypertelorism, malar flattening)
- Intracranial extension
- · Complete nasal airway obstruction

AFRS patients are atopic (>90 %) and frequently report history of allergic rhinitis and asthma; yet classic aspirin sensitive triad is not part of the disease constellation [36]. Typically, these patients have symptoms of sinusitis refractory to antibiotics, intranasal corticosteroids, immunotherapy, as well as attempts at prior surgery if eosinophilic mucin was not noted or collected at the time of operation; thereby failing to establish the correct diagnosis [21, 34, 36].

## Diagnosis

The Bent and Kuhn criteria are generally regarded as the most well accepted diagnostic criteria for AFRS (Table 11.2) [2]. However, a positive fungal stain suffices for their requirement of a positive fungal culture. Fungal morphology is sufficient to establish the presence of fungi, and often specific enough to identify the responsible

Table 11.2       Bent and Kuhn         diagnostic criteria for allergic         fungal rhinosinusitis	1. Gel and Coombs type I (IgE-mediated) hypersensitivity
	2. Nasal polyposis
	3. Characteristic radiologic findings
	4. Positive fungal stain and/or fungal culture
	5. Eosinophilic mucin without fungal invasion into sinus tissue
	Bent and Kuhn [2]

organism at the genus level [48]. Reliance on fungal cultures for diagnosis is hindered by the variable yield of such cultures (64–100 %) as well as techniques which may merely identify a saprophytic organism within the nose and not the fungus responsible for the patient's clinical findings [30, 36].

Eosinophilic mucin, a diagnostic criterion of AFRS, is perhaps the most specific finding of the disease and occupies a central role in the understanding of the pathogenesis, histology, diagnosis and treatment of the disease process. Eosinophilic mucin is thick, highly viscous, tan to dark green or brown material that may be removed from the sinuses with some difficulty. Extra-mucosal fungi are identified microscopically with various silver stains, while hematoxylin and eosin stains illustrate the sheets of eosinophils and Charcot-Leyden crystals within a mucinous background [21, 36].

#### Radiology

Diagnostic imaging findings in AFRS have been delineated in a number of retrospective reviews including both CT and MRI modalities. AFRS patients demonstrate bilateral disease in 51 % of cases, with asymmetric involvement in 78 % of reviewed cases [41]. Complete opacification of at least one sinus was noted in 98 % of reviewed cases.

Complete sinus cavity opacification is associated with the following signs that have become suggestive of AFRS (Fig. 11.4):

- Sinus expansion (98 %)
- Remodeling of the sinus walls (95 %)
- Bony erosion (91 %)

AFRS can also be characterized by the nature of CT scan attenuation and MRI signal intensities. Opacified paranasal sinuses have increased central signal attenuation on non-contrast CT, which correspond with hypointense areas on T1-weighted MRI and signal voids on T2-weighted MRI [31, 41].

These heterogeneous areas of signal intensity within opacified sinuses on softtissue CT algorithms are thought to result from heavy metal accumulations and calcium salt precipitation within inspissated mucin and debris [41]. The presence of hyperdensities on CT, corresponding to areas of signal dropout on T2-weighted MRI, can be highly suggestive, though not confirmatory, for the diagnosis of AFRS.



**Fig. 11.4** Coronal CT scan with AFRS demonstrates expansion of the left frontal sinus with bowing of the intersinus septum. Complex pneumatization pattern, including an expansile type III cell, is noted in the left frontal recess

## **Surgical Treatment**

Though the ideal treatment strategy for AFRS remains open for debate, comprehensive endoscopic sinus surgery forms the basic foundation for any successful intervention in this disease process. Functional endoscopic sinus surgery (FESS) techniques are employed to interrupt the "AFRS cycle" and set the stage for postoperative immune modulation.

The goals of sinus surgery are [35, 36]

- Complete removal of all eosinophilic mucin and fungal debris.
- Achievement of permanent drainage and ventilation of the affected sinuses while preserving underlying mucosa.
- Provide postoperative access to the diseased areas, such that adequate adjunctive topical care can be performed.

Preoperative antibiotics and corticosteroids (equivalent to 0.5–1.0 mg/kg/day of prednisone) are utilized to decrease generalized sinonasal inflammation and polyp volume, thereby improving visualization and decreasing bleeding at the time of surgery [36]. Meticulous postoperative care with serial endoscopic debridement is

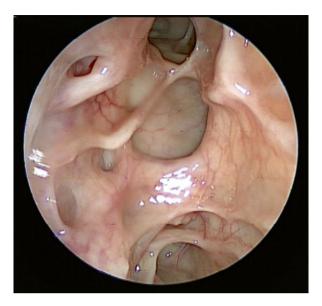


Fig. 11.5 Postoperative endoscopy demonstrates healed frontal internal ostium after comprehensive FESS in patient demonstrated in Fig. 11.4

imperative to achieve functional sinonasal cavities (Fig. 11.5). Patients are tapered from oral steroids over the ensuing weeks and transitioned to innovative topical therapies to minimize risk of relapse of AFRS.

## **Medical Treatment**

The similarities between ABPA and AFRS play a large role in much of the current concepts of medical therapy for AFRS. Successful application of steroids in ABPA patients led to their introduction in AFRS cases. Decreased recurrence rates in those treated with steroids, and marked recidivism in those who discontinue treatment, have made systemic steroids an integral therapy for AFRS, though no consensus has been reached on the ideal dose or duration [3, 26, 49]. The addition of topical steroids within the newly ventilated sinonasal cavity is expected to assist in alleviating local inflammation, whereas preoperatively, this route is limited by obstructive nasal polyps [36]. A pilot study of CRS patients in 2009 suggested that the addition of budesonide suspension to nasal saline irrigations produces significant improvement in subjective patient symptoms based on a visual analog scale, as well as objective findings on CT and endoscopy [51]. This was followed by a trial of 111 patients who were randomized to receive daily irrigations of budesonide (1 mg) or betamethasone (1 mg) diluted in 240 mL saline. Improvements were noted in patient symptom scores, SNOT-22 scores, and endoscopy scores when compared to baseline (p < 0.001). In addition, patients with high tissue eosinophilia or nasal polyps

had greater improvement [50]. This technique allows for improved steroid contact with sinus mucosa but with less than 5 % residual of the total drug within the sinus, which is equivalent to that of standard nasal steroid sprays [20].

Institution of immunotherapy directed against fungal antigens should be considered in the postoperative period in order to modulate the patient's exuberant inflammatory reaction to fungi [28]. Retrospective data has shown that patients receiving immunotherapy have significantly better overall outcomes than those postoperative patients who declined or discontinued immunotherapy. Potential benefits include symptom control, decrease in the use of topical and systemic steroid use, reduction in revision surgery, and improvement in both subjective quality of life scores and objective assessments of the postoperative inflammatory state of the sinuses [1, 27, 29]. However, immunotherapy failed to show a significant impact on long term control of disease when patients are followed beyond the first 5 years as the disease may enter a quiescent state after successful initial control of the disease [38].

Additional adjunctive measures in the management of AFRS directly target the fungi that initiate the "AFRS cycle." Systemic antifungals have not clearly demonstrated their value in treating AFRS, and all are fraught with poor therapeutic indices, risks of serious medical complications, increased costs and uncertain duration of drug therapy [36]. Generally, systemic antifungal therapy is reserved for cases that are refractory to traditional treatment. Given that patients may inhale up to  $5.7 \times 10^7$  spores of various fungi each day, it seems more efficacious to alter the host's immune response rather than expose the patient to chronic antifungal therapy [43]. Topical antifungals likely have lower risks of complications; however, their efficacy, as in systemic therapy, is limited to conjecture.

#### **AFRS and the Frontal Sinus**

The exact frequency with which the frontal sinus is involved in cases of AFRS is unknown, though one radiographic study puts the estimate as high as 71 % [41]. Proximity of the frontal sinus to both the anterior cranial fossa and orbit increases the precision required to address disease in this location. Accumulation of dense eosinophilic mucin, in a manner very similar to the pressure necrosis exerted by mucoceles, can cause dissolution and erosion of already delicate bone and extension of the process into the orbit or intracranial space [36]. Complete evacuation of eosinophilic mucin and fungal debris from the frontal sinus coupled with establishment of permanent ventilation and drainage is a requisite to successfully manage AFRS involving the frontal sinus. Preservation of the mucosa at the internal ostium is key to achieving long-term frontal recess patency. Typically, the fungal process will widen the frontal outflow drainage pathway, thus endoscopic frontal sinusotomy should be sufficient to achieve the surgical objectives [26, 36]. However, in cases with extensive fungal involvement or complex pneumatization patterns, Draf IIb or III may be required. If a frontal osteoplastic flap is required, Kuhn and Swain caution against frontal sinus obliteration in treating fungal disease, especially in complicated cases with erosion through the posterior table or orbital roof, as frontal

sinus mucosa cannot be removed completely from the underlying periorbita or dura [26]. Surgery should allow for postoperative visualization of the frontal sinus though the frontal internal ostium during clinic endoscopy to evaluate for recurrence of disease (Fig. 11.5). If re-stenosis of the frontal ostium is noted or there is significant recurrence of polyp disease, CT imaging may be warranted in monitoring for recurrent disease or frontal ostial stenosis with mucocele formation.

## Conclusion

The accrued body of literature attests to the improved understanding of the role of fungus in paranasal sinus disease over the past 35 years. The frontal sinus is not a common location for fungal disease, and as such, most otorhinolaryngologists have limited experience in treating fungal pathology in this location. Indeed, the close proximity to critical structures and narrow confines of the frontal recess add to the surgical dilemma. Nonetheless, endoscopic frontal approaches, either through standard endoscopic frontal sinus of ungal disease involving the frontal sinus. Further, a careful understanding of fungal sinus disease states, appropriate diagnostic investigation, and perioperative medical therapy, coupled with sound knowledge of the surgical anatomy of the frontal sinus, will provide patients the best opportunity for an optimal outcome.

## References

- Bassichis BA, Marple BF, Mabry RL, Newcomer MT, Schwade ND. Use of immunotherapy in previously treated patients with allergic fungal sinusitis. Otolaryngol Head Neck Surg. 2001;125:487–90.
- Bent J, Kuhn F. Diagnosis of allergic fungal sinusitis. Otolaryngol Head Neck Surg. 1994;111:580–8.
- 3. Bent JP, Kuhn FA. Allergic fungal sinusitis/polyposis. Allergy Asthma Proc. 1996; 17:259–68.
- 4. Blitzer A, Lawson W. Fungal infections of the nose and paranasal sinuses, part I. Otolaryngol Clin N Am. 1993;26:1007–35.
- Carter KD, Graham SM, Carpenter KM. Ophthalmologic manifestations of allergic fungal sinusitis. Am J Ophthalmol. 2001;127:189–95.
- Chakrabarti A, Denning DW, Ferguson BJ, Ponikau J, Buzina W, Kita H, et al. Fungal rhinosinusitis: a categorization and definitional schema addressing current controversies. Laryngoscope. 2009;119:1809–18.
- Chirch L, Roche P, Fuhrer J. Successful treatment of invasive Aspergillus sinusitis with caspofungin and voriconazole. Ear Nose Throat J. 2008;87:30–3.
- Collins M, Nair S, Smith W, Kette F, Gillis D, Wormald PJ. Role of local immunoglobulin E production in the pathophysiology of noninvasive fungal sinusitis. Laryngoscope. 2004;114:1242–6.
- DelGaudio JM, Swain RE, Kingdom TT, Muller S, Hudgins PA. Computed tomographic findings in patients with invasive fungal sinusitis. Arch Otolaryngol Head Neck Surg. 2003;129: 236–40.

- 10. deShazo RD. Fungal sinusitis. Am J Med Sci. 1998;316:39-45.
- deShazo RD, O'Brien M, Chapin K, Soto-Aguilar M, Gardner L, Swain R. A new classification and diagnostic criteria for invasive fungal sinusitis. Arch Otolaryngol Head Neck Surg. 1997;123:1181–8.
- deShazo RD, O'Brien M, Chapin K, Soto-Aguilar M, Swain R, Lyons M, et al. Criteria for the diagnosis of sinus mycetoma. J Allergy Clin Immunol. 1997;99:475–85.
- 13. Ferguson BJ. Definitions of fungal rhinosinusitis. Otolaryngol Clin N Am. 2000;33:227-35.
- 14. Ferguson BJ. Mucormycosis of the nose and paranasal sinuses. Otolaryngol Clin N Am. 2000;33:349–65.
- 15. Ferguson BJ. Fungus balls of the paranasal sinuses. Otolaryngol Clin N Am. 2000;33: 389–98.
- Ferguson BJ, Barnes L, Bernstein JM, Brown D, Clark 3rd CE, Cook PR, et al. Geographic variation in allergic fungal rhinosinusitis. Otolaryngol Clin N Am. 2000;33:441–9.
- 17. Ferreiro JA, Carlson BA, Cody DT. Paranasal sinus fungus balls. Head Neck. 1997;19:481-6.
- Gillespie MB, O'Malley BW. An algorithmic approach to the diagnosis and management of invasive fungal rhinosinusitis in the immunocompromised patient. Otolaryngol Clin N Am. 2000;33:323–34.
- 19. Gillespie MB, O'Malley BW, Francis HW. An approach to fulminant invasive fungal sinusitis in the immunocompromised host. Arch Otolaryngol Head Neck Surg. 1998;124:520–6.
- Harvey RJ, Debnath N, Srubiski A, Bleier B, Schlosser RJ. Fluid residuals and drug exposure in nasal irrigation. Otolaryngol Head Neck Surg. 2009;141:757–61.
- Houser SM, Corey JP. Allergic fungal rhinosinusitis: pathophysiology, epidemiology and diagnosis. Otolaryngol Clin N Am. 2000;33:399–408.
- 22. Ilica AT, Mossa-Basha M, Maluf F, Izbudak I, Aygun N. Clinical and radiologic features of fungal diseases of the paranasal sinuses. J Comput Assist Tomogr. 2012;36:570–6.
- Katzenstein AA, Sale SR, Greenberger PA. Allergic Aspergillus sinusitis: a newly recognized form of sinusitis. J Allergy Clin Immunol. 1983;72:89–93.
- Kennedy CA, Adams GL, Neglia JP, Giebink GS. Impact of surgical treatment on paranasal fungal infections in bone marrow transplant patients. Otolaryngol Head Neck Surg. 1997;116:610–6.
- Klossek JM, Serrano E, Peloquin L, Percodani J, Fontanel JP, Pessey JJ. Functional endoscopic sinus surgery and 109 mycetomas of paranasal sinuses. Laryngoscope. 1997;107: 112–7.
- 26. Kuhn FA, Swan R. Allergic fungal sinusitis: diagnosis and treatment. Curr Opin Otolaryngol Head Neck Surg. 2003;11:1–5.
- Mabry RL, Mabry CS. Immunotherapy for allergic fungal sinusitis: the second year. Otolaryngol Head Neck Surg. 1997;117:367–71.
- Mabry RL, Manning SC, Mabry CS. Immunotherapy in the treatment of allergic fungal sinusitis. Otolaryngol Head Neck Surg. 1997;116:31–5.
- 29. Mabry RL, Marple BF, Folker RJ, Mabry CS. Immunotherapy for allergic fungal sinusitis: three years' experience. Otolaryngol Head Neck Surg. 1998;119:648–51.
- Manning SC, Holman M. Further evidence for allergic fungal sinusitis. Laryngoscope. 1998;108:1485–96.
- Manning SC, Merkel M, Kriesel K, Vuitch F, Marple BF. Computed tomography and magnetic resonance diagnosis of allergic fungal sinusitis. Laryngoscope. 1997;107:170–6.
- 32. Manning SC, Schaefer S, Close L. Culture-positive allergic fungal sinusitis. Arch Otolaryngol Head Neck Surg. 1991;117:174–8.
- Manning SC, Vuitch F, Weinberg A, Brown OE. Allergic aspergillosis: a newly recognized form of sinusitis in the pediatric population. Laryngoscope. 1989;108:1485–96.
- 34. Marple BF. Allergic fungal sinusitis. Curr Opin Otolaryngol. 1999;7:383-7.
- 35. Marple BF. Allergic fungal rhinosinusitis: surgical management. Otolaryngol Clin N Am. 2000;33:409–18.
- 36. Marple BF. Allergic fungal rhinosinusitis: current theories and management strategies. Laryngoscope. 2001;111:1006–19.

- Marple BF, Mabry RL. Comprehensive management of allergic fungal sinusitis. Am J Rhinol. 1998;12:263–8.
- Marple BF, Newcomer M, Schwade N, Mabry R. Natural history of allergic fungal rhinosinusitis: a 4- to 10-year follow-up. Otolaryngol Head Neck Surg. 2002;127:361–6.
- 39. Millar JW, Johnston A, Lamb D. Allergic aspergillosis of the maxillary sinus. Thorax. 1981;36:710.
- 40. Mitchell TG. Overview of basic medical mycology. Otolaryngol Clin N Am. 2001;33: 237–50.
- 41. Mukherji SK, Figueroa RE, Ginsberg LE, Zeifer BA, Marple BF, Alley JG, et al. Allergic fungal sinusitis: CT findings. Radiology. 1998;207:417–22.
- 42. Nicolai P, Lombardi D, Tomenzoli D, Villaret AB, Piccioni M, Mensi M, et al. Fungus ball of the paranasal sinuses: experience in 160 patients treated with endoscopic surgery. Laryngoscope. 2009;119:2275–9.
- Novey HS. Epidemiology of allergic bronchopulmonary aspergillosis. Immunol Allergy Clin N Am. 1998;18:641–53.
- 44. Nussenbaum B, Marple BF, Schwade ND. Characteristics of bony erosion in allergic fungal rhinosinusitis. Otolaryngol Head Neck Surg. 2001;124:150–4.
- Robson J, Hogan P, Benn R, Gatenby PA. Allergic fungal sinusitis presenting as a paranasal sinus tumor. Aust NZ J Med. 1989;19:351–3.
- Safirstein B. Allergic bronchopulmonary aspergillosis with obstruction of the upper respiratory tract. Chest. 1976;70:788–90.
- Sarti EJ, Blaugrund SM, Lin PT, Camins MB. Paranasal sinus disease with intracranial extension: aspergillosis versus malignancy. Laryngoscope. 2000;98:632–5.
- 48. Schell WA. Histopathology of fungal rhinosinusitis. Otolaryngol Clin N Am. 2000;33: 251–76.
- Schubert MS, Goetz DW. Evaluation and treatment of allergic fungal sinusitis II: treatment and follow-up. J Allergy Clin Immunol. 1998;103:717–23.
- Snidvongs K, Pratt E, Chin D, Sacks R, Earls P, Harvey RJ. Corticosteroid nasal irrigations after endoscopic sinus surgery in the management of chronic rhinosinusitis. Int Forum Allergy Rhinol. 2012;2:415–21.
- Steinke JW, Payne SC, Tessier ME, Borish LO, Han JK, Borish LC. Pilot study of budesonide inhalant suspension irrigations for chronic eosinophilic sinusitis. J Allergy Clin Immunol. 2009;124:1352–4.
- 52. Stevens MH. Aspergillosis of the frontal sinus. Arch Otolaryngol. 1978;104:153-6.
- Stringer SP, Ryan MW. Chronic invasive fungal rhinosinusitis. Otolaryngol Clin N Am. 2000;33:375–87.
- Turner JH, Soudry E, Nayak JV, Hwang PH. Survival outcomes in acute invasive fungal sinusitis: a systematic review and quantitative synthesis of published evidence. Laryngoscope. 2013;123:1112–8.
- 55. Wehl G, Hoegler W, Kropshofer G, Meister B, Fink FM, Heitger A. Rhinocerebral mucormycosis in a boy with recurrent acute lymphoblastic leukemia: long-term survival with systemic antifungal treatment. J Pediatr Hematol Oncol. 2002;24:492–4.
- Wise SK, Ghegan MD, Gorham E, Schlosser RJ. Socioeconomic factors in the diagnosis of allergic fungal rhinosinusitis. Otolaryngol Head Neck Surg. 2008;138:38–42.
- Yohai RA, Bullock JD, Aziz AA, Markert RJ. Survival factors in rhinoorbital cerebral mucormycosis. Surv Ophthalmol. 1994;39:3–22.

# **Chapter 12 Headache and the Frontal Sinus**

Charles A. Parker and Allen M. Seiden

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# Abbreviations

AAOHNS	American Academy of Otolaryngology-Head and Neck Surgery
IHS	International Headache Society
MOH	Medication overuse headache

### **Core Messages**

• Frontal headache frequently accompanies obstruction and inflammation of the frontal sinuses, but may also reflect other sources of head pain not related to sinus pathology.

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- "Sinus headache" is often misdiagnosed migraine headache.
- A thorough history that defines the pattern of headache is essential to help diagnose its cause.
- A diagnosis of sinus-related headache needs to be confirmed by a thorough nasal examination that should include nasal endoscopy and appropriate radiographs.
- Many of the primary and secondary headache disorders may cause headache in the frontal region, and therefore need to be considered in the differential diagnosis.

# Introduction

Headache is a remarkably common symptom that affects nearly half of the global population annually. According to the World Health Organization's report on the Global Burden of Disease, headache ranks among the top ten most disabling disorders [1]. The International Headache Society (IHS) has classified headaches as either primary or secondary and within these two categories are over 150 headache types.

- Primary headaches are not due to an identifiable cause and comprise 90 % of headache disorders.
  - The most common primary headache disorders are:
    - Migraine
    - Tension-type
    - Cluster headaches
- Secondary headache disorders are those in which a headache occurs in relation to another process and resolves or reduces when the underlying disease process is treated. Common secondary causes of headache include
  - Acute infections
  - Medication overuse
  - Cervicogenic
  - Post traumatic [2].

Patients with headache will often present to a variety of specialists looking for an answer to relieve their discomfort. Evaluation by their primary care physician or neurologist may result in a diagnosis of one of the primary headache syndromes, and an underlying sinus problem may be missed. Figure 12.1 shows the CT scan of a 16-year old girl who complained of headaches for over 1 year without associated nasal obstruction or nasal discharge. She was diagnosed with migraines, but had not responded to traditional therapy. The scan demonstrates complete opacification of both frontal sinuses, and endoscopic frontal sinusotomy drained inspissated mucus that relieved her head pain.

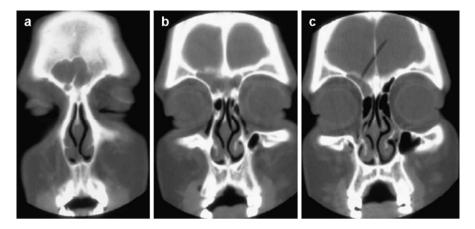


Fig. 12.1 A 16-year-old girl complaining of frontal headaches for over 1 year was diagnosed with migraine headaches. (a, b) On CT scan, the frontal sinuses are completely opacified. (c) Prominent agger nasi cells with obstruction of the right frontal recess

Likewise, patients will present to the otolaryngologist because they or their referring physician believe the headache to be related to underlying sinus pathology. The primary focus of the otolaryngologic evaluation is to exclude this possibility, but to do so requires not only an understanding of what can cause sinus-related pain, but also an ability to recognize other, more common headache syndromes.

## Pathophysiology

Clinicians and patients alike recognize a relationship between nasal/sinus pathology and head pain, but this relationship is highly variable and therefore controversial. There has been little data to document irrefutably when and why it exists.

The ophthalmic and maxillary divisions of the trigeminal nerve provide sensory innervation to the nose and paranasal sinuses. Stammberger and Wolf have postulated that free nerve endings respond to chemical, mechanical, and caloric stimuli to prompt the release of substance P. This produces an orthodromic impulse traveling along nociceptive C fibers that is interpreted centrally as pain, but may not be well localized by higher cortical centers. At the same time, an anti-dromic impulse results in the peripheral release of substance P, causing localized neurogenic edema and hypersecretion. This produces additional mucosal swelling and impaction, furthering the sensation of pain [3]. Based on this concept, areas of narrowing in the nose or ostiomeatal complex might be prone to impaction, causing mechanical stimulation of the trigeminal nerve and thereby producing headache pain.

Early studies by Sluder were some of the first to demonstrate that sinus inflammation can present with referred head pain [4]. His experiments revealed that closure of the infundibulum and frontonasal opening can lead to a vacuum or negative pressure within the frontal sinus that resulted in frontal headache. This phenomenon most often occurred in the frontal sinus rather than the other paranasal sinuses. Although confirmatory data is scant, several studies as cited by Stammberger and Wolf have demonstrated that hypoxia in the sinuses can give a sensation of pain [3].

A series of experiments performed by Wolff in the 1940s also supported the concept of referred pain due to sinus inflammation [5]. In a small series of human volunteers, noxious stimuli were placed at various sites within the paranasal sinuses, at the sinus ostia, and within the nasal cavity. Surprisingly, the sinus mucosa was not very sensitive. Rather, the mucosa surrounding the ostia and nasal turbinates was much more pain-sensitive. In addition, the pain was often not felt locally, but was referred to dermatomes of the first and second divisions of the trigeminal nerve. Thus, whereas stimulation applied to the walls of the frontal sinus led to a mild localized pain at that site, stimulation of the frontal recess and frontonasal area produced an intense local pain and pain over the medial canthus, zygoma, and upper molars.

Recently, Wolff's experiments have been repeated in a randomized single-blinded study. Ten volunteers without any nasal contact points were randomized on four separate visits to receive intranasal pressure, adrenaline, substance P and placebo. While the stimuli did produce variable local discomfort, itching or sneezing, none of the stimuli caused referred pain to the face as in Wolff's original experiment [6]. While Wolff's experiments are considered classic and are frequently quoted in support of sinus induced headache, there is a growing body of evidence that argues both for and against this phenomenon.

## **Patient Evaluation**

In evaluating the headache patient, much reliance is placed upon the history, as history alone will often differentiate primary from secondary headache disorders. Dodick describes a systematic approach in history taking to elicit the most vital information and prevent overlooking a potentially fatal secondary cause (Table 12.1) [7].

Key points in the headache history include:

- · Chronicity
- · Age of onset
- Frequency of episodes
- · Duration of episodes
- · Location of pain
- · Character and severity of pain
- Associated symptoms
- Aggravating and alleviating factors.

If along with frontal headache, patients present with active nasal symptoms such as congestion and drainage, this will usually alert the clinician to the possibility of an underlying sinus problem. However, it is not uncommon for patients with migraine headache to report symptoms typically associated with sinus disease such as nasal congestion [8], and patients may also have associated rhinitis

	History	Possible causes
Systemic symptoms/signs/	Fever, chills, night sweats,	Giant cell arteritis
disease	myalgias, weight loss	Infection
		Malignancy
	Prior malignancy, immunocompromised, HIV	Metastatic disease, opportunistic infection
<u>N</u> eurologic symptoms/signs	Focal or global neurologic changes – behavioral, visual loss, diplopia, pulsatile tinnitus	CNS disease – neoplastic, infectious, vascular, intracranial hypertension
<u>O</u> nset sudden	How quickly does pain go from 0 to 10 – thunder clap	Vascular – stroke, SAH, cerebral venous sinus thrombosis, arterial dissection
<u><i>O</i></u> nset after age 50 years	Primary headache is rare after 50 years	CNS disease – infection, inflammation, neoplastic; giant cell arteritis
<u><i>P</i></u> attern change (if prior headache history)	Progressive with loss of headache free periods	
-	Precipitated by Valsalva	Chiari malformation, brain tumor, CSF leak
_	Postural worsening	Worse with standing or lying – intracranial hypotension from CSF leak, intracranial hypertension; neck movements worsening – cervicogenic Worse with neck movements – cervicogenic headache
_	Papilledema	Intracranial hypertension

Table 12.1 SNOOP4: secondary causes of headache

that is unrelated to their head pain. On the other hand, patients may have no nasal complaints despite the presence of extensive inflammatory changes within the paranasal sinuses. Therefore, further workup is required to confirm the headache is indeed sinus-related.

The physical examination often provides little in the diagnosis of primary headache syndromes but is often diagnostic for secondary headache disorders. In contrast to that of the primary care physician or neurologist, the role of the otolaryngologist in evaluating the patient diagnosed with "sinus headache" is often heavily reliant on the physical examination and radiographic findings.

To detect evidence of occult inflammatory sinus disease, anterior rhinoscopy alone is generally not adequate. To visualize the middle meatus, frontal recess, superior meatus, and sphenoethmoidal recess properly, nasal endoscopy is indispensable. It is very important to correlate endoscopic findings with symptoms.

Fig. 12.2 Endoscopic view of a left middle meatus, with a purulent discharge from the upper middle meatus and frontal recess suggesting frontal sinus infection



**Fig. 12.3** Endoscopic view of a left middle meatus with mucosal edema over the agger nasi region and a polyp protruding from the upper middle meatus, suggesting frontal recess and frontal sinus disease



- In patients presenting with frontal headache, findings suggestive of frontal sinus disease include:
  - Purulent discharge from the frontal recess (Fig. 12.2)
  - Polypoid change in the upper middle meatus under the attachment of the middle turbinate
  - Enlarged and edematous agger nasi cell (Fig. 12.3).

These findings would certainly warrant further investigation. When no mucosal inflammation at all is present but anatomic variations can be seen, the relationship of such findings to chronic headache becomes much more tenuous and controversial [9].

If a nasal endoscopic examination is unremarkable, but the history strongly suggests nasal- or sinus-related pain, radiologic evaluation is still indicated. Plain sinus radiographs do not demonstrate the frontal recess and ethmoid sinus adequately and as such are rarely helpful. Computed axial tomography (CT) in the coronal plane with appropriate bone windows remains the procedure of choice [10]. In addition to frank opacification, it is important to look for areas of

Rhinogenic	Rhinosinusitis		
	Mucosal contact points		
	Anatomic variations: agger nasi cell, frontal cell, prominent ethmoid bulla		
	Prior sinus surgery		
	Frontal sinus trauma		
Primary headache	Migraine		
	Tension-type headache		
	Cluster headache		
Secondary headache	Medication over-use headache		
	Cervicogenic headache		
	Temporomandibular joint disorders		
Headache emergencies	Intracranial neoplasm		
	Giant cell arteritis		

Table 12.2 Differential diagnosis of frontal headache

mucosal contact and secondary mucosal thickening, particularly in association with anatomic variations.

## **Differential Diagnosis**

Frontal headache is a nonspecific symptom that may be associated with multiple disease processes (Table 12.2). Not only should one consider a rhinogenic etiology for the cause of frontal pain but also primary and secondary headache syndromes that can be confused as sinus pain.

## Rhinogenic

### **Rhinosinusitis Headache**

To properly diagnose a patient with a headache of sinonasal origin, one should be familiar with the current diagnostic criteria outlined by the IHS for diagnosis of headache attributed to rhinosinusitis and that of the American Academy of Otolaryngology-Head and Neck Surgery (AAOHNS) for diagnosis of rhinosinusitis (Tables 12.3 and 12.4) [2, 11]. The key in diagnosing the patient with head pain attributed to rhinosinusitis is exam and/or radiographic findings of inflammation and the temporal resolution of pain with the treatment of sinusitis. It should be noted that the IHS criteria state that chronic sinusitis is not a cause of headache unless relapsing into an acute stage; however, it is not uncommon for the otolaryngologist to evaluate patients with chronic rhinosinusitis that present with chronic head pain.

Both acute and chronic frontal sinusitis may present with frontal headaches. Acute frontal sinusitis almost always presents with severe frontal headache of relatively short duration and associated nasal purulence and obstruction. Chronic Table 12.3 IHS diagnostic criteria for headache attributed to rhinosinusitis

- A. Frontal headache accompanied by pain in one or more regions of the face, ears or teeth and fulfilling criteria C and D
- B. Clinical, nasal endoscopic, CT and/or MRI imaging and/or laboratory evidence of acute or acute-on-chronic rhinosinusitis
- C. Headache and facial pain develop simultaneously with onset or acute exacerbation of rhinosinusitis
- D. Headache and/or facial pain resolve within 7 days after remission or successful treatment of acute or acute-on-chronic rhinosinusitis

Acute bacterial rhinosinusitis (ABRS)	Symptoms/signs present for 10 days or more
	OR
	Worsening of symptoms/signs within 10 days after an initial period of improvement (double worsening)
Symptoms/signs of acute	Purulent nasal drainage
rhinosinusitis	Plus
	Nasal obstruction
	OR
	Facial pain-pressure-fullness
Chronic bacterial rhinosinusitis	2 or more of the following symptoms/signs for >12 weeks
	1. Mucopurulent drainage
	2. Nasal obstruction
	3. Facial pain-pressure-fullness
	AND
	Evidence of inflammation (purulence, edema or polyps) by nasal endoscopy or radiography
Recurrent acute bacterial	4 or more episodes of ABRS per year without symptoms/signs
rhinosinusitis	of rhinosinusitis between episodes

Table 12.4 Rhinosinusitis diagnostic criteria

Acute - symptoms less than 4 weeks

Subacute – symptoms from 4 to 12 weeks

Chronic - symptoms greater than 12 weeks

frontal sinusitis may also present with headache, described as a dull, constant pressure, but often in the absence of nasal symptoms and a normal nasal endoscopic exam. The diagnosis of headache attributed to acute rhinosinusitis is often relatively straight forward, however, that of chronic sinusitis can be challenging. Table 12.5 describes the common characteristics of head pain related to frontal sinus disease [3].

Alternatively, reducing nasal and sinus inflammation and observing a change in the patient's headache pattern may achieve some confirmation that the headache is rhinogenic in origin, although there is little data in this regard. Such therapy might include topical and systemic decongestants, topical and systemic steroids, antibiotics, or allergy medications as appropriate.

Pain localized around:	Glabella
	Inner canthus
	Between the eyes
	Above the eyebrow
Pain described as:	Dull
	Constant
	Sensation of pressure or fullness
	Worse in the morning, bending over or with the Valsalva maneuver

Table 12.5 Characteristics of head pain attributed to frontal/ethmoid sinus disease

<b>Table 12.6</b>	Common	findings in	n migraneurs	leading to	misdiagnosis of	"sinus headache"
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Migraine triggers	Weather changes (83 %)
	Seasonal variation (73 %)
	Exposure to allergens (62 %)
Location of pain	Bilateral forehead and maxillary pain (62 %)
	Pain in distribution of second division of the trigeminal nerve (76 %)
Associated symptoms	Nasal congestion (56 %)
	Eyelid edema (37 %)
	Rhinorrhea (25 %)
	Conjunctival injection (22 %)

The term "sinus headache" is commonly used by both patients and physicians to describe head pain that is thought to be due to sinus inflammation and/or infection.

• The Sinus Allergy and Migraine Headache Study looked at 100 patients who were thought to have "sinus headaches". Of this group, 86 % of patients thought to have "sinus headache" where found to have migraine or probable migraine based on IHS criteria. Only 3 % were diagnosed as having headache secondary to rhinosinusitis [12].

This common misdiagnosis was thought to be due to the association of specific triggers, location of pain and associated symptoms amongst migraneurs that are commonly seen in rhinosinusitis patients (Table 12.6). Unfortunately, the diagnosis of "sinus headache" is commonly given for what is misdiagnosed migraine without aura, resulting in unnecessary diagnostic studies, surgical interventions and delays in appropriate migraine therapy [13].

### Non-infectious Sinus Pathology

Any cause of frontal sinus obstruction may lead to the development of frontal headaches with or without frontal sinusitis. While frontal obstruction is most commonly due to an inflammatory or infectious process, other possible etiologies include an enlarged agger nasi cell [14], frontal cell [15], prominent ethmoid bullae, frontal

**Fig. 12.4** A large osteoma within the frontal sinus in a patient presenting with frontal headaches

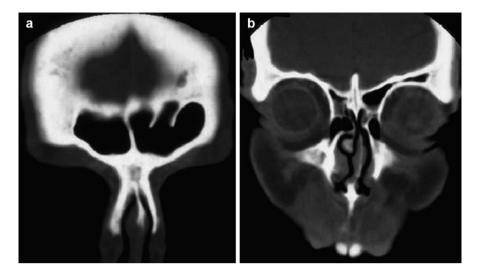


osteoma [16] (Fig. 12.4), postoperative scarring [17] and trauma [18]. Han et al. found among 102 patients that underwent frontal sinus surgery the cause of frontal obstruction included polyps (53 %), frontal recess synechia (21 %), agger nasi cell (12 %) and narrow ostiomeatal complex (5 %) [17].

For example, the patient whose radiograph is pictured in Fig. 12.5 presented with a 10-month history of persistent right frontal headaches. The CT scan demonstrates a large, obstructing agger nasi cell and secondary mucosal thickening within the frontal recess, although the frontal sinus seems to be well aerated. His headache was relieved by surgically opening the frontal recess.

### Mucosal Contact Point Headaches

Despite the many studies describing contact point induced headaches, it still remains an area of controversy due to the fact that most studies are retrospective case series or expert opinion articles on the topic. The few prospective studies generally lack significant patient numbers or have insufficient follow-up time [19]. A contact point is defined as two mucosal points that remain in contact despite topical decongestion [20]. Commonly this is due to a septal spur or a medialized turbinate, however, these findings are common variations seen in asymptomatic individuals as well. Identifying a contact point is done by nasal endoscopy or CT scan. Demonstrating its causal relationship to head pain is by showing improvement in pain after application of decongestant or anesthetic to the contact point. A recent systematic review



**Fig. 12.5** CT scan of a patient presenting with a 10-month history of right frontal headaches. (**a**) The frontal sinus appears aerated without disease. (**b**) A large, right agger nasi cell with secondary mucosal thickening within the frontal recess

concluded that contact points are a common finding among asymptomatic patients and are not related to headache or facial pain and that while most studies show improvement in headache after contact point surgery, this improvement is frequently partial and temporary [20].

• Patients that would potentially benefit the most from contact point surgery are those that have failed medical therapy for migraine or tension-type headache, have a normal nasal endoscopy and CT scan for rhinosinusitis, and have responded positively when a local anesthetic has been applied to their contact point.

Despite fulfilling these criteria a discussion should be had with each patient explaining the possibility of persistent or recurrent head pain after surgery [19].

# **IHS Primary Headache Syndromes**

Headaches are considered primary when the headache and associated features are not secondary to an exogenous cause. These make up nearly 90 % of headache disorders and include tension-type, migraine and cluster headaches.

### Migraine

Migraine is a common disorder that affects 18 % of women, 6 % of men and 4 % of children or nearly 30 million Americans [21].

 Although tension-type headache is the most prevalent primary headache disorder, of those patients that present to their physician for evaluation of headache, ~90 % will meet diagnostic criteria for migraine headache [22].

The IHS provides diagnostic criteria for six main subtypes of migraine. The two main subtypes are migraine without aura and migraine with aura. Of these, migraine without aura accounts for approximately 85 % of migraine headaches [23]. Migraine can be described as having four phases: the prodrome, the aura, the headache and the postdrome; however, not all four phases need be present for diagnosis [24]. The prodrome describes a period hours to days before the onset of headache. It commonly manifests as fatigue, poor concentration and/or stiff neck. The aura can be a visual, sensory or motor phenomenon that immediately precedes the onset of headache. The most frequent aura is visual derangements such as flashing lights, visual spots or lines, bilateral photophobia or loss of vision. Auras commonly develop gradually over 5–20 min and last for less than 60 min. The headache is often unilateral, throbbing in nature, moderate to severe in pain and worsens with physical activity. The headache will often last hours to days. It is not uncommon for patients with a migraine headache to experience pain in the frontal region. In a study of patients with migraine without aura, the initial headache was localized solely to the frontal region in 31 % and to the frontal region along with another region in an additional 25 % of patients [25]. In a study of patients suffering migraine with aura, the initial headache involved the frontal region in 59 % of patients [23]. Nausea often accompanies the headache in 90 % of migraneurs. Following the headache, the postdrome period, patients describe feeling tired and irritable. Headache can often be precipitated by various environmental or dietary triggers such as: menstruation, stress, fatigue, altered sleep, weather changes, alcohol or medications. Criteria for the diagnosis of migraine headache have been established by IHS and are listed in Table 12.7 [2].

- A screening questionnaire has been shown to have a 93 % positive predictive value for the diagnosis of migraine when two of the three questions are answered positively [26]:
  - Photophobia: Does light bother you when you have a headache?
  - Impairment: Do you experience headaches that impair your ability to function?
  - Nausea: Do you feel nauseated or sick to your stomach when you experience a headache?

This screening tool offers the clinician a quick and simplified approach to recognize migraine as a potential diagnosis and prompt further questioning.

### **Tension-Type Headache**

Tension-type headache is the most common of the primary headache disorders with a lifetime prevalence of 69 % in men and 88 % in women [27].

Migraine without aura	A. At least 5 attacks fulfilling criteria B–D
wigianie without auta	
	B. Headache attacks lasting 4–72 h (untreated or unsuccessfully treated)
	C. Headache has $\geq 2$ of the following characteristics:
	1. Unilateral location
	2. Pulsating quality
	3. Moderate or severe pain intensity
	4. Aggravation by or causing avoidance of routine physical activity (e.g., walking, climbing stairs)
	D. During headache $\geq 1$ of the following:
	1. Nausea and/or vomiting
	2. Photophobia and phonophobia
	E. Not attributed to another disorder
Migraine with typical	A. At least 2 attacks fulfilling criterion B
aura	B. Aura consisting of $\geq 1$ of the following, but no motor weakness:
	1. Fully reversible visual symptoms including positive and/or negative features
	<ol> <li>Fully reversible sensory symptoms including positive and/or negative features</li> </ol>
	3. Fully reversible dysphasic speech disturbance
	C. At least two of the following:
	1. Homonymous visual symptoms and/or unilateral sensory symptoms
	2. At least one aura symptom develops gradually over ≥5 min and/ or different aura symptoms occur in succession over ≥5 min
	3. Each symptom lasts $\geq 5$ and $\leq 60$ min
	D. Headache fulfilling criteria B–D for <i>Migraine without aura</i> begins during the aura or follows aura within 60 min
	E. Not attributed to another disorder
Chronic migraine with or without aura	Migraine headache on $\geq 15$ day per month for >3 months
Probable migraine with or without aura	Attacks fulfilling all but one of criteria A–D above

Table 12.7 International headache society diagnostic criteria for migraine

• Rarely do patients with tension – type headache present to their physician for evaluation and thus this should be a diagnosis of exclusion in the office setting.

The pain associated with a tension-type headache is often described as bilateral, a dull ache, nonpulsating and/or band like pressure along the frontotemporal region. One review found the frontal region to be the predominant region of pain in 40 % of patients [28]. Although the pain is usually bilateral, it may be unilateral in 10–20 % of patients. The pain of a tension-type headache often builds in intensity, may fluctuate in severity and persist for days. In contrast to migraines, the pain of a tension-type headache is mild to moderate, not aggravated by physical activity and not associated with nausea. However, 25 % of patients with tension-type headache

Episodic tension-type headache	A. Infrequent or frequent headache fulfilling criteria B–D
	B. Headache lasting from 30 min to 7 days
	C. Headache has $\geq 2$ of the following characteristics:
	1. Bilateral location
	2. Pressing/tightening (non-pulsating) quality
	3. Mild or moderate intensity
	4. Not aggravated by routine physical activity
	D. Both of the following:
	1. No nausea or vomiting (anorexia may occur)
	2. No more than one of photophobia or phonophobia
	E. Not attributed to another disorder
Infrequent episodic tension-type headache	Headache <1 day per month OR <12 days per year
Frequent episodic tension-type headache	Headache >1 but < 15 days per month OR >12 but <180 days per year
Chronic tension-type headache	Headache >15 days per month for 3 months OR >180 days per year
Probable tension-type headache	Episodes fulfilling all but one of criteria A-D above

Table 12.8 International headache society diagnostic criteria for tension-type headache

also have migraines [27]. The IHS diagnostic criteria for tension-type headache are listed in Table 12.8 [2].

### **Cluster Headache**

Cluster headaches are the rarest of the primary headaches discussed, with a prevalence of only 1.5 % [27]. The most common form of cluster headache is episodic, whereas only 10 % of cluster headache patients will have the chronic form. Headache episodes are characterized by recurrent bursts of short lasting but severe unilateral pain along the orbital or temporal area. Head pain is often described as deep, piercing or burning. Associated symptoms include conjunctival injection, lacrimation, nasal congestion, rhinorrhea, facial sweating, miosis and eyelid edema. Although some of these autonomic symptoms and signs may be seen in migraine headache, they tend to be subtle, whereas they are much more prominent in cluster headache. Headaches typically last from 15 to 180 min if left untreated. A typical pattern of attack is one to three episodes per day over a period of 6-8 weeks followed by a symptom-free interval of 9-12 months. In contrast to migraines, cluster headaches are more common among males and African-Americans. These men often display certain physical characteristics, such as a ruddy complexion, deep furrows of the forehead, and deep folds of the glabellar and nasolabial areas. They tend to be tall and trim, usually smoke, and are more likely to consume alcohol [29]. Obstructive sleep apnea has been seen in up to 50 % of patients with cluster headache [7]. The IHS diagnostic criteria for cluster headache are listed in Table 12.9 [2].

Cluster headache	A. At least 5 attacks fulfilling criteria B–D
	B. Severe or very severe unilateral orbital, supraorbital and/or temporal pain lasting 15–180 min if untreated
	C. Headache is accompanied by $\geq 1$ of the following:
	1. Ipsilateral conjunctival injection and/or lacrimation
	2. Ipsilateral nasal congestion and/or rhinorrhoea
	3. Ipsilateral eyelid oedema
	4. Ipsilateral forehead and facial sweating
	5. Ipsilateral miosis and/or ptosis
	6. A sense of restlessness or agitation
	D. Attacks have a frequency from 1 every 2 days to 8 per day
	E. Not attributed to another disorder
Episodic cluster headache	At least 2 cluster periods lasting 1 week to 1 year are separated by a remission period lasting $\geq 1$ month
Chronic cluster headache	Cluster periods occur for >1 year without remission periods or remission periods < 1 month

Table 12.9 International headache society diagnostic criteria for cluster headache

**Fig. 12.6** Endoscopic view of the right ethmoid cavity in a patient who had surgery several years previously, now presenting with severe right frontal headache



A 35-year-old white male was referred because of a severe intermittent right frontal headache for 1 month. He described this as following an upper respiratory infection, but had no residual congestion or drainage. However, he did describe intermittent tearing of the right eye. The headache was described as throbbing retro-orbital and frontal pain. This patient had a similar headache 3 years previously, and at that time endoscopic sinus surgery was performed and the headache resolved. Therefore, when this current episode began, he was placed on antibiotics and steroids, but did not respond. Figure 12.6 is an

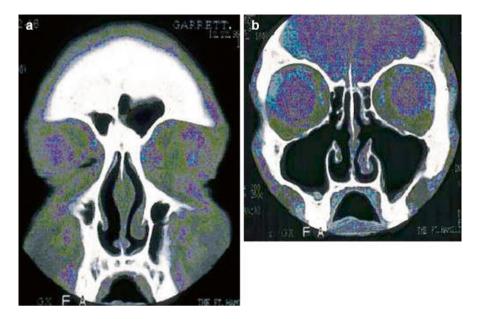


Fig. 12.7 The CT scan of the patient in Fig. 12.6, demonstrating postsurgical changes but no evidence of active sinus disease

endoscopic view of the right ethmoid cavity in this patient, demonstrating postsurgical changes with an open frontal recess. His sinus scan is shown in Fig. 12.7, and is clear of disease. This patient's headache was not related to sinus pathology, but rather was a cluster headache and did respond to appropriate medication. One might speculate that the headache he experienced 3 years previously also was cluster, but this demonstrates the confusion that might arise when evaluating these patients.

## IHS Secondary Headache Syndromes

Secondary headaches are attributed to an underlying cause. These headaches can be classified as acute or chronic (>15 days/month for at least 3 months). Key to diagnosing a secondary headache is demonstrating the temporal relationship of the headache with the underlying pathology. The most common secondary headaches are acute in nature and induced by alcohol, fever, trauma and infection. These are often easily identifiable. Chronic secondary headaches are rare, affecting 1-3 % of the population. These patients often pose a diagnostic challenge and present to multiple specialists for evaluation. Common causes of chronic secondary headaches that may present with frontal pain are medication-overuse headache, cervicogenic headache and temporomandibular pain [30].

### **Medication Overuse Headache**

Medication overuse headache (MOH) is becoming a growing problem that is estimated to affect about 1.5 % of the population worldwide [31]. MOH is typically seen in patients with a primary headache disorder, typically episodic migraine or tension-type headache, which then transforms into a chronic daily headache. It is characterized by a progressively worsening headache with the increasing use of analgesic or similar medications that demonstrate reduced efficacy. Susceptible individuals are those with daily or near daily use of any acute headache medication such as triptans, ergotamine and opioids. The headache commonly varies in location and laterality, is commonly present upon wakening and may follow a predictable pattern associated with timing of the last dose of medications and its withdrawal. MOH should be suspected in any individual with a prior headache diagnosis that presents with a new chronic daily headache or reduced medication efficacy.

#### **Cervicogenic Headache**

Although neck pain may be a common finding in primary headache disorders [32]; it may also be a source for head pain. Cooper et al. studied the referral patterns of patients with cervical joint pain and found that C1-2 and C2-3 joint pain was often associated with forehead and orbital pain [33]. Characteristics of cervicogenic headache include unilateral head pain that fluctuates with neck movement; associated neck, shoulder or arm pain; and pain that radiates from the occiput to forehead; however, none of these findings are diagnostic for cervicogenic headache. Diagnosis requires the demonstration of a cervical spine or soft tissue disorder and resolution of headache with anesthetic blockade of the pain source. Prior history of trauma should be elicited, as there is a 53 % prevalence of cervicogenic headache after whiplash [34].

### Headache Emergencies

The greatest underlying concern for the patient with a new onset headache is that it represents an underlying life threatening process [35]. Table 12.1 describes a systematic way in questioning headache patients to avoid missing a potential "red flag" which would warrant further investigation [7].

### **Intracranial Neoplasm**

Intracranial neoplasm is the most feared cause of new-onset headache, but fortunately it is an uncommon cause of headache. • A recent prospective study looking at headache attributed to intracranial tumors identified headache as the sole presenting symptom in 40 % of patients but at the time of diagnosis 96 % of patients had developed other neurologic symptoms or signs such as cognitive disturbance, motor or sensory signs, visual field defects, cranial nerve lesions, coordination disturbances and seizures [36]

Vazquez-Barquero et al. found that only 8 % of patients presented with isolated headache and that focal neurological symptoms were present in 57 % of patients, while seizures occurred in 9 % [37].

However, in the absence of focal neurological signs, presenting headache symptoms are usually nonspecific. While most headaches in these patients do not meet specific IHS diagnostic criteria, if classified the most common primary headache phenotype would be tension-type headache (23.5 %) followed by episodic migraine without aura (13.5 %) [36]. The frontal region was the most common site of headache, occurring in 68 %, and was usually bifrontal although worse ipsilateral to the tumor. The classically described presentation of nocturnal headache that awakens the patient from sleep, morning headaches, headaches worsened with the Valsalva maneuver, and associated vomiting is rarely seen. The pain is commonly bilaterally localized over the frontal area and described as a pressure or tightening quality. Only 30 % of patients will have unilateral frontal headaches [36].

Patients with pre-existing headache disorders that develop headache secondary to an intracranial neoplasm can be very difficult to diagnose, as very often the headache pattern may be similar [38]. Atypical features in those with pre-existing headache that warrant further investigation include progressive pattern, worsening with Valsalva or lying down, nocturnal occurrence and unresponsiveness to analgesics [36].

#### **Giant Cell Arteritis**

• A diagnosis of giant cell arteritis or temporal arteritis should be considered in any patient greater than 50 years of age with a new-onset headache, regardless of the location of that headache.

Temporal arteritis is a vasculitis involving small and medium-sized vessels, and typically produces headache as its presenting symptom. The temporal location is the most common site of pain, but the frontal region has been reported as the primary site in 33 % of patients [39]. Associated symptoms may include temporal artery tenderness (69 %), jaw claudication (67 %), weight loss (55 %), and visual symptoms (40 %). Polymyalgia rheumatica, an inflammatory rheumatic condition characterized by pain and morning stiffness in the shoulders, hips and neck, was seen in approximately 50 % of patients [40]. Complete visual loss can occur in up to 10 % of patients despite treatment. The erythrocyte sedimentation rate (ESR) is a good screening test, having been found to be greater than 50 mm/h in 89 % of patients and greater than 100 in 41 % [1]. Diagnosis requires three of the following five items: greater than or equal to 50 years of age, a new headache,

temporal artery abnormalities (tenderness, decreased pulsation), an ESR greater than 50 mm/h and a biopsy specimen showing vasculitis predominated by granulomatous infiltration [35].

## Conclusion

Otolaryngologists often see patients with frontal headache for evaluation of underlying sinonasal pathology. For successful diagnosis and appropriate management, the otolaryngologist must understand the presentation and differential diagnosis of primary and secondary headache disorders that may cause headache in the frontal region.

## References

- 1. Jensen R, Stovner LJ. Epidemiology and comorbidity of headache. Lancet Neurol. 2008;7(4):354-61.
- 2. Headache Classification Subcommittee of the International Headache Society. The international classification of headache disorders, 2nd edn. Cephalalgia. 2004;24 Suppl 1:1–160.
- Stammberger H, Wolf G. Headaches and sinus disease: the endoscopic approach. Ann Otol Rhinol Laryngol. 1988;97 suppl 134:3–23.
- 4. Sluder G. Nasal neurology, headaches and eye disorders. St. Louis: C.V. Mosby Co.; 1927.
- 5. Wolff HG, McAuliffe GW, Goodell H. Experimental studies on headache: pain reference from the nasal and paranasal cavities. Trans Am Neurol Assoc. 1942;68:82–3.
- Abu-Bakra M, Jones NS. Does stimulation of nasal mucosa cause referred pain to the face? Clin Otolaryngol Allied Sci. 2001;26(5):430–2.
- 7. Dodick DW. Pearls: headache. Semin Neurol. 2010;30(1):74-81.
- Schreiber CP, Hutchinson S, Webster CJ, et al. Prevalence of migraine in patients with a history of self-reported or physician-diagnosed "sinus" headache. Arch Intern Med. 2004;164(16):1769–72.
- 9. Cook PR, Nishioka GJ, Davis WE, et al. Functional endoscopic sinus surgery in patients with normal computed tomography scans. Otolaryngol Head Neck Surg. 1994;110:505–9.
- Zinreich SJ, et al. CT of nasal cavity and paranasal sinuses: imaging requirements for functional endoscopic sinus surgery. J Radiol. 1987;163:769–75.
- Rosenfield RM. Clinical practice guideline on adult sinusitis. Otolaryngol Head Neck Surg. 2007;137(3):365–77.
- 12. Eross E, Dodick D, Eross M. The sinus, allergy and migraine study (SAMS). Headache. 2007;47(2):213–24.
- Levine HL, Setzen M, Cady RK, et al. An otolaryngology, neurology, allergy, and primary care consensus on diagnosis and treatment of sinus headache. Otolaryngol Head Neck Surg. 2006;134(3):516–23.
- Kuhn FA, Bolger WE, Tisdal RG. The agger nasi cell in frontal recess obstruction: an anatomic, radiologic and clinical correlation. Oper Technol Otolaryngol Head Neck Surg. 1991;2(4):226–31.
- Bent III JP, Cuilty-Siller C, Kuhn FA. The frontal cell as a cause of frontal sinus obstruction. Am J Rhinol. 1994;8(4):185–91.
- Leiberman A, Tovi F. A small osteoma of the frontal sinus causing headaches. J Laryngol Otol. 1984;98:1147–9.

- 17. Han JK, Ghanem T, Lee B, et al. Various causes for frontal sinus obstruction. Am J Otolaryngol. 2009;30(2):80–2.
- Khan OA, Majumdar S, Jones NS. Facial pain following sinonasal surgery or facial trauma. Clin Otolaryngol Allied Sci. 2002;27(3):171–4.
- 19. Patel ZM, Kennedy DW, Setzen M, et al. "Sinus headache": rhinogenic headache or migraine? An evidence-based guide to diagnosis and treatment. Int Forum Allergy Rhinol. 2013;3(3):221–30.
- Harrison L, Jones NS. Intranasal contact points as a cause of facial pain or headache: a systematic review. Clin Otolaryngol. 2013;38(1):8–22.
- 21. Lipton RB, Bigal ME. Ten lessons on the epidemiology of migraine. Headache. 2007;47 Suppl 1:S2–9.
- Dowson A, Dahlof C, Tepper S, et al. Prevalence and diagnosis of migraine in a primary care setting. Cephalalgia. 2002;22:581–2.
- 23. Sjaastad O, Fredriksen T, Sand T. The localization of the initial pain of attack: a comparison between classic migraine and cervicogenic headache. Funct Neurol. 1989;4:73–8.
- 24. Sliberstein SD. Migraine. Lancet. 2004;363(9406):381-91.
- 25. Sjaastad O, Bovim G, Stovner LJ. Common migraine ("migraine without aura"): localization of the initial pain of attack. Funct Neurol. 1993;8:27–32.
- Lipton RB, Dodick D, Sadovsky R. A self-administered screener for migraine in primary care: the ID migraine validation study. Neurology. 2003;61(3):375–82.
- Silberstein SD, Young WB. Headache and facial pain. In: Goetz CG, editor. Textbook of clinical neurology. 3rd ed. Philadelphia: Saunders; 2007. p. 1245–66.
- Langemark M, Olesen J, Poulson DL, et al. Clinical characterization of patients with chronic tension headache. Headache. 1988;28:590–6.
- 29. Manzoni GC, Terzano MG, Bono G, et al. Cluster headache clinical findings in 180 patients. Cephalalgia. 1983;3:1–30.
- Aaseth K, Grande RB, Kvaerner KJ, et al. Prevalence of secondary chronic headaches in a population-based sample of 30-44-year-old persons. The Akerhus study of chronic headache. Cephalalgia. 2008;28(7):705–13.
- Dodick D, Freitag F. Evidence-based understanding of medication-overuse headache: clinical implications. Headache. 2006;46 Suppl 4:S202–11.
- 32. Calhoun AH, Ford S, Millen C, et al. The prevalence of neck pain in migraine. Headache. 2010;50(8):1273–7.
- Cooper G, Bailey B, Bogduk N. Cervical zygapophysial joint pain maps. Pain Med. 2007;8(4):344–5.
- Bogduk N, Govind J. Cervicogenic headache: an assessment of the evidence on clinical diagnosis, invasive tests and treatment. Lancet Neurol. 2009;8(10):959–68.
- Friedman BW, Lipton RB. Headache emergencies: diagnosis and management. Neurol Clin. 2012;30(1):43–59.
- Valentinis L, Tuniz F, Valent F. Headache attributed to intracranial tumours: a prospective cohort study. Cephalalgia. 2010;30(4):389–98.
- 37. Vazquez-Barquero P, Ibanez FJ, Herrera S, et al. Isolated headache as the presenting manifestation of intracranial tumors: a prospective study. Cephalalgia. 1994;14:270–2.
- Forsyth P, Posner J. Headaches in patients with brain tumors: a study of 111 patients. Neurology. 1993;43:1678–83.
- 39. Solomon S, Cappa KG. The headache of temporal arteritis. J Am Geriatr Soc. 1987;35:163–5.
- Huston KA, Hunder GG, Lie JT, et al. Temporal arteritis: a 25-year epidemiologic, clinical, and pathologic study. Ann Intern Med. 1978;88(2):162–7.

# Chapter 13 Frontal-Orbital-Ethmoid Mucoceles

Raewyn Campbell, Ameet Kamat, Ioana Schipor, and James Palmer

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### **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 endoscopic techniques.

Electronic supplementary material The online version of this chapter

 $<sup>(</sup>doi:10.1007/978-3-662-48523-1_13)$  contains supplementary material, which is available to authorized users.

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<sup>©</sup> Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_13

- 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 re-established.
- Acutely infected frontal sinus mucopyoceles associated with a complication are best treated by surgical drainage through a trephine, followed by weeks of intravenous antibiotics, and then subsequent endoscopic drainage of the frontal sinus.

## Introduction

Mucoceles are slowly growing, benign, expansile lesions found in the paranasal sinuses. On histopathology, they are cyst-like structures lined with a pseudostratified respiratory epithelium and filled with sterile 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 can form in any of the paranasal sinuses. Approximately 60–89 % occur in the frontal sinus, 8–30 % in the ethmoid sinuses, 5–10 % in the maxillary sinus and 2–3 % in the sphenoid sinus [1–5]. There are several case reports of mucoceles occurring in unusual locations, such as the pterygomaxillary space, orbital floor, and middle turbinate [6–8]. The incidence of skull base bony destruction and intracranial extension has been reported to be between 10 % and 55 % [9, 10].

Paranasal sinus mucoceles are uncommon lesions. They can form at any age, however, the majority are diagnosed in patients aged 40–60 years [1, 5]. Males and females are equally affected. Mucoceles are extremely rare in children, although several case reports and a small series of pediatric mucoceles, have been published [11–13]. Some authors have noted an association between mucoceles and cystic fibrosis [14] 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 due to infection, fibrosis, inflammation, trauma, surgery or tumors (Table 13.1). They enlarge slowly and fill the affected sinus cavity, expanding and eroding the adjacent bony structures. The mucocele expands in the direction of least resistance, which often includes the thin bone of the superior orbital wall. Secondary infection can lead to a period of rapid expansion with a resultant increased risk of complications, especially in the periorbital area [15].

One proposed mechanism for mucocele formation is cystic degeneration of a seromucinous gland, resulting in a retention cyst [16]. However, detailed histopathologic studies have shown little evidence for this mechanism and instead have suggested that the mechanism responsible for mucocele expansion is the dynamic interface between bone and the mucocele lining. 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 by bacterial lipopolysaccharides leads to the production of cytokines by the lining fibroblasts [17, 18]. These cytokines, in turn, promote bone resorption and remodeling and result in mucocele expansion [19]. Bone erosion results from positive pressure as well as from the presence of cytokines such as tumor necrosis factor, prostaglandins and interleukins such as IL-1, IL-12 and IL-6 [17, 20]. Cultured fibroblasts derived from frontoethmoidal mucoceles have been shown to produce significantly elevated levels of prostaglandin E2 and collagenase, compared with normal frontal sinus mucosal fibroblasts [21]. This suggests that the lining fibroblasts represent a major source of bone-resorbing factors [21].

Common etiologic factors related to frontoethmoid mucocele formation include: a history of sinusitis, previous sinus surgery, allergy, and trauma (Table 13.1). Surgery can lead to mucocele formation either by directly blocking the sinus ostium with scar tissue or by entrapping sinus mucosa. Post-surgical paranasal sinus mucoceles can occur several years after the initial operation. Frontal sinus mucoceles were reported in 9.3–19.3 % of cases after osteoplastic flaps or frontal sinus obliteration procedures [5, 22]. Mucoceles have been described after both external and endoscopic sinus surgery [23–26].

Uncommonly, mucoceles form as result of inflammatory conditions (cystic fibrosis, nasal polyposis, Wegener's granulomatosis) or an ostial occlusion caused by a benign neoplasm (osteoma, fibrous dysplasia, nasal polyposis), or a malignant

	Chronic rhinosinusitis with or without nasal polyposis
	Previous sinus surgery
	Previous maxillofacial trauma
	Allergies
Table 13.1Paranasal sinusmucoceles: commonetiologies	Tumors- benign and malignant
	Inflammatory conditions e.g. Wegener's granulomatosis
	Idiopathic

tumor [5, 15, 27, 28]. In up to one third of cases, however, the history is noncontributory and no demonstrable cause can be found [2].

Culture of the aspirated mucocele contents can sometimes confirm the presence of infection, which is generally polymicrobial [18]. In fact, the most common aerobic isolates cultured from mycopyoceles are *Staphyloccocus aureus*, alpha-hemolytic streptococci, *Haemophilus* species, and gram-negative bacilli [18]. The predominant anaerobic isolates are *Propionibacterium acnes*, *Peptostreptococcus*, *Prevotella*, and *Fusobacterium* species [18].

# 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 displacement, decreased visual accuity, or epiphora [29] (Fig. 13.1). Other common presentations include headaches, facial pressure or swelling, nasal drainage and obstruction (Table 13.2). Orbital expansion of the mucocele can lead to globe displacement resulting in exposure keratitis. Other orbital complications include: central retinal artery occlusion, superior ophthalmic vein thrombosis or cavernous sinus thrombosis in more severe cases [30, 31].



Fig. 13.1 Frontal sinus mucocele: left orbital proptosis

Table 13.2Paranasal sinusmucoceles: common clinicalpresentations

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

Intracranial extension through erosion of the posterior wall of the frontal sinus, or through the roof of the ethmoid sinus, can lead to meningitis, a CSF fistula or rarely, frontal lobe syndrome [32–34]. The posterior frontal 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. Other, less common, sequelae include intracranial abscess, seizures and osteomyelitis [31, 35].

### 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, accompanies 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 mucoceles. The imaging mode of choice is CT scanning [2].

CT scans clearly delineate the mucocele as a well circumscribed, cyst-like, homogeneous, isodense lesion originating in a paranasal sinus and compressing surrounding structures. The osteolytic or sclerotic bony changes surrounding the lesion can easily be seen (Fig. 13.2). The mucocele content demonstrates homogeneous mucoid attenuation (10–18 Hounsfield Units (HU)). Longstanding lesions have higher protein content and attenuate more (20–40 HU). Contrast enhancement is

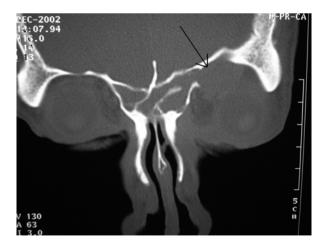
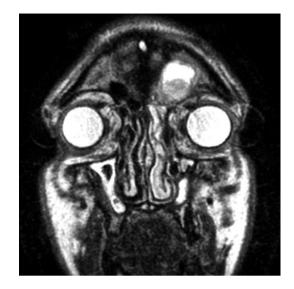
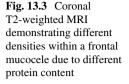


Fig. 13.2 Coronal CT (bone windows) demonstrating opacification of the left frontal sinus with erosion of the orbital roof (*arrow*)





rarely necessary; however, after intravenous contrast medium injection the lesion demonstrates rim enhancement.

Magnetic resonance imaging (MRI) is useful when the diagnosis is uncertain and it is necessary to differentiate between different types of soft tissue 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 intensity signal on T1-weighted sequences and high intensity signal on T2-weighted sequences. However, variations commonly occur depending on the age and protein content of the mucocele (Fig. 13.3). Post-gadolinium contrast images confirm the presence of fluid within the mucocele by showing either absent signal enhancement or a peripherally enhancing cystic structure (Fig. 13.4) [2]. Contrast enhanced MRI is especially useful for delineating a mucocele from a 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 scanning.

## 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 (Figs. 13.5 and 13.6).

The following classification system was devised in order to standardize frontal sinus mucocele evaluation and management [36]:

- Type 1. Limited to frontal sinus (with or without orbital extension)
- Type 2. Frontoethmoid mucocele (with or without orbital extension)

**Fig. 13.4** Coronal T1-weighted MRI with contrast demonstrating peripheral rim enhancement of the same left frontal mucocele as Fig. 13.3







- Type 3. Erosion of the posterior sinus wall
  - A. Minimal or no intracranial extension
  - B. Major intracranial extension
- Type 4. Erosion of the anterior sinus wall



Fig. 13.6 Postoperative CT after endoscopic drainage of mucocele

- Type 5. Erosion of both anterior and posterior sinus 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 recurrence. 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 surgery should not be performed in the setting of an acute infection. The exception is an acute, symptomatic mucopyocele. It is our experience that operative failure secondary to scar formation, stenosis and adhesions is more likely after operating on an acutely infected mucocele/mucopyocele. Treatment with antibiotics and systemic steroids for a period prior to, and after, surgery improves post-operative success.

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 (e.g. Lynch-Howarth frontoethmoidectomy) or osteoplastic flaps with sinus cavity obliteration. These procedures carried significant morbidity and cosmetic deformity, as well as significantly higher rates of recurrence and of complications when compared to the endoscopic approach [4, 37]. Additionally, post-operative radiographic follow-up becomes difficult after obliteration. Further, complete removal of the mucosa and obliteration of the sinus may be difficult in cases where the posterior wall of the frontal sinus or the orbital wall is dehiscent and mucosa is adherent to dura or orbital periosteum.

• 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 [5, 9, 12, 36, 38, 39].

Endoscopic drainage has been advocated as preservation of the frontal sinus mucosa and maintenance of a patent frontal recess results in better clinical outcomes [4, 40]. In fact, histological and physiological studies have demonstrated that the mucocele mucosa retains functional respiratory epithelium and regains normal mucociliary clearance after marsupialization [19, 41].

In 1989 Kennedy et al. published the first series of 18 mucoceles treated by endoscopic marsupialization. Their study reported a 0 % recurrence rate after an average follow-up of 18 months [12]. Another study, with longer follow-up, examined the recurrence rate in two groups of patients with paranasal sinus mucoceles: the first group was treated endoscopically (20 patients) and the second group was treated using a combined external and endoscopic approach (28 patients) [39]. The combined approach was used in the more severe cases where the anatomy, extent of disease or previous surgery, restricted endoscopic visualization and access to the frontal sinus, or where a fistulous tract was present [39]. There were no recurrences in the group managed exclusively via a transnasal endoscopic approach after a mean follow-up of 34 months [39]. There were three recurrences (11 %) in the combined endoscopic/external drainage group after a mean follow-up of 44 months [39]. 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.

Har-El has published the largest series of patients with mucoceles in the English literature [9]. One hundred and three patients with 108 paranasal sinus mucoceles were treated by wide endoscopic marsupialization [9]. Post-operative stents were used in frontal sinus mucoceles [9]. The recurrence rate was 0.9 % (one patient) after a mean follow up of 4.6 years [9]. The rate of major complications was also very low, with only one patient experiencing an intraoperative CSF leak, which resolved after immediate repair and post-operative bed rest [9]. The author concluded that the endoscopic drainage should be considered the procedure of choice for management of paranasal sinus mucoceles [9].

Sautter et al. endoscopically managed 57 patients with paranasal sinus mucoceles [5]. Revision surgery was required in 17.5 % of patients for restenosis or retained lateral frontal compartment mucocele [5]. The majority of these patients had undergone previous sinus surgery [5]. All ophthalmologic symptoms resolved or improved post-operatively and 98.2 % were still functionally patent at 15 months follow-up [5].

The endoscopic approach is particularly useful when an extensive frontoethmoidal mucocele has eroded the posterior frontal sinus wall and/or the lamina papyracea. In these cases, sinus obliteration is problematic given the difficulty of completely removing the lining mucosa from the exposed dura and/or the orbital periosteum [5, 36]. No complications were reported in the small pediatric series reported by Hartley and Lund [11]. Seven children underwent endoscopic drainage of ethmoid and sphenoid mucoceles, and there were no recurrences after 1 year of follow-up [11].

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 [42]. Other authors have advocated wide marsupialization via an endoscopic transnasal approach [43]. Alternatively, mucoceles with intracranial extension are approached with a combined craniofacial and endoscopic approach [39].

## Surgical Technique

All patients should undergo pre-operative CT scanning. Computer aided, CT-based stereotactic navigation techniques have expanded the number of lesions accessible via the endoscopic approach. 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 [11]. Specimens should be sent for microbiological and pathological 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 [39]. The medial orbital wall is often eroded in the case of ethmoid mucoceles and the globe is obviously at risk in these cases. A thin silastic sheet may be placed over the periorbita for 1-2 weeks to prevent crusting in this location. If it appears that prolapse of the orbital contents may occlude frontal sinus drainage then an endoscopic frontal sinus drillout procedure should be considered [38]. To enhance mucosal healing after a drillout procedure, free mucosal grafts taken from the septum may be used to cover exposed bone in the frontal sinus. Steroid-eluting frontal sinus implants may present another option for maintaining sinus patency post-operatively. Post-operative packing is not routinely used. Attention to post-operative nasal hygiene, including nasal irrigations and topical steroids are critical. If the contents of the mucocele were purulent or if the microbiological cultures were positive, oral antibiotics are used. Close endoscopic follow up post-operatively should be continued until the cavity heals and mucociliary clearance is re-established.

Post-operatively, temporary diplopia after globe repositioning can occur.

Recurrences may occur, on average, more than 4 years post-operatively, however, have been described up to 41 years post-operatively [44]. Therefore, patients require long-term follow-up and surveillance.

### Antibiotic Treatment

Whilst surgical management is of primary importance in the management of mucoceles, antimicrobial therapy forms part of the management paradigm in the treatment of mucopyoceles. Empirical antibiotic choice should focus on the predominant aerobic and anaerobic bacteria known to be present [18]. Antimicrobials effective against anaerobes and *S. aureus* include the combination of a penicillin (e.g. amoxicillin) with a  $\beta$ -lactamase inhibitor (e.g. clavulanate) or clindamycin. In addition, an antimicrobial effective against aerobic gram-negative rods, such as gentamicin, ceftazidime, a quinolone (in adults) or a carbapanem, may be added to empirical therapy until culture results are obtained [18].

## **Special Clinical Circumstances**

There are two clinical circumstances deserve specialized attention. The acutely infected mucopyocele that presents with a complication is best treated by open techniques. Endoscopic drainage, whether through a Draf II–III approach, is best not performed in the acute setting on frontal sinus mucopyoceles that are "hot". Invariably, the widely patent frontal sinus outflow tract will slowly scar down, most likely due to the inflammation associated with the bone infection. These clinical situations are best treated with acute drainage through a frontal sinus trephine approach, followed by intravenous antibiotics, and then definitive surgery. We prefer to treat the mucopyocele as an osteomyelitis with 6 weeks of culture directed intravenous antibiotics. Once the postoperative antibiotic course is finished, take the patient back at that time for endoscopic drainage, often through a Draf III approach. Please see clinical videos for delineation of this successful approach. The video takes each step, including preoperative CT, trephine drainage, subsequent separate trip to OR for Draf III, and 6 month follow up (Video 13.1).

A second special clinical scenario is long standing mucocele in a previously endoscopically operated frontal sinus, especially in a patient that has significant risks for undergoing general anesthesia. In these patients, office drainage of the mucocele can be quite successful. Begin with endoscopic placement of pledgets soaked in 1 % lidocaine, consider local injection with a spinal needle, and then using a sharp curved probe such as a 90° Kuhn-Bolger curette, a Van Alyea cannula, or 90° bent malleable dcr probe, enter the mucocele through the soft tissue scar. At that point, use suction to clear the contents and allow transillumination for safety. Once safe position is confirmed, increase the size of the drainage pathway by either frontal sinus dissection instruments or balloon sinuplasty. In a "cold" mucocele, great long-term success can be achieved using this local anesthetic treatment technique.

# 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 confirmed by CT scan. 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.

# References

- Arrue P, Thorn Kany M, Serrano E, et al. Mucoceles of the paranasal sinuses: uncommon location. J Laryngol Otol. 1998;112:840–4.
- Lloyd G, Lund VJ, Savy L, Howard D. Optimum imaging for mucoceles. J Laryngol Otol. 2000;114:233–6.
- 3. Iannetti G, Cascone P, Valentini V, et al. Paranasal sinus mucocele: diagnosis and treatment. J Craniofac Surg. 1997;8:391–8.
- 4. Obeso S, Llorente JL, Rodrigo JP, et al. Paranasal sinuses mucoceles: our experience in 72 patients. Acta Otorhinolaringol Esp. 2009;60(5):332–9.
- Sautter NB, Citardi MJ, Perry J, Batra PS. Paranasal sinus mucoceles with skull-base and/or orbital erosion is the endoscopic approach sufficient? Otolaryngol Head Neck Surg. 2008;139(4):570–4.
- Stack BC, Klotch DW. Mucocele of the pterygomaxillary space. Ann Otol Rhinol Laryngol. 1995;104(3):246–7.
- Nkenke E, Amann K, Maier T, Benz M, Kramer M, Haeusler G, Benz S, Wiltfang J, Vairaktaris EG, Neukam FW, Holbach L. Untreated 'blow-in' fracture of the orbital floor causing a mucocele report of an unusual late complication. J Craniomaxillofac Surg. 2005;33(4):255–9.
- Toledano A, Herraiz C, Mate A, Plaza G, Aparicio JM, De Los Santos G, Galindo AN. Mucocele of the middle turbinate: a case report. Otolaryngol Head Neck Surg. 2002;126(4):442–4.
- 9. Har-El G. Endoscopic management of 108 sinus mucoceles. Laryngoscope. 2000;111: 2131-4.
- Koike Y, Tokoro K, Chiba Y, et al. Intracranial extension of paranasal sinus mucoceles: two case reports. Surg Neurol. 1996;45:44–8.
- 11. Hartley BEJ, Lund VJ. Endoscopic drainage of pediatric paranasal sinus mucoceles. Int J Pediatr Otorhinolaryngol. 1999;50:109–11.
- Kennedy DW, Josephson JS, Zinreich SJ, et al. Endoscopic sinus surgery for mucoceles: a viable alternative. Laryngoscope. 1989;99:885–95.
- Serrano E, Klossek JM, Percodani J, Yardeni E, Dufour X. Surgical management of paranasal sinus mucoceles: a long-term study of 60 cases. Otolaryngol Head Neck Surg. 2004;131(1): 133–40.

- 14. Guttenplan MD, Wetmore RF. Paranasal sinus mucoceles in cystic fibrosis. Clin Pediatr. 1989;28:429–30.
- Stiernberg CM, Bailey BJ, Calhoun KH, et al. Management of invasive frontoethmoidal sinus mucoceles. Arch Otolaryngol Head Neck Surg. 1986;112:1060–3.
- 16. Batsakis JG. Tumours of the head and neck. Baltimore: Williams and Wilkins; 1980.
- Kariya S, Okano M, Hattori H, Sugata Y, Matsumoto R, Fukushima K, Akagi H, Nishizaki K. Expression of IL-12 and T helper cell cytokines in the fluid of paranasal sinus mucoceles. Am J Otolaryngol. 2007;28(2):83–6.
- 18. Brook I, Frazier EH. The microbiology of mucopyocele. Laryngoscope. 2001;111(10): 1771–3.
- Lund VJ, Milroy CM. Fronto-ethmoidal mucoceles: a histopathological analysis. J Laryngol Otol. 1991;105:921–3.
- Lund VJ, Henderson B, Song Y. Involvement of cytokines and vascular adhesion receptors in the pathology of fronto-ethmoidal mucoceles. Acta Otolaryngol. 1993;113:540–6.
- Lund VJ, Harvey W, Meghji S, Harris M. Prostaglandin synthesis in the pathogenesis of fronto-ethmoidal mucoceles. Acta Otolaryngol. 1988;106:145–51.
- 22. Hardy JM, Montgomery WW. Osteoplastic frontal sinusotomy: an analysis of 250 operations. Ann Otol Rhinol Laryngol. 1976;85:523–32.
- Busaba NY, Salman SD. Ethmoid mucocele as a late complication of endoscopic ethmoidectomy. Otolaryngol Head Neck Surg. 2003;128:517–22.
- 24. Har-El G, Balwally AN, Lucente FE. Sinus mucoceles; is marsupialization enough? Otolaryngol Head Neck Surg. 1997;117:633–40.
- Moriyama H, Nakajima T, Honda Y. Studies on mucoceles of the ethmoid and sphenoid sinuses. J Laryngol Otol. 1979;106:23–7.
- Natvig K, Larsen TE. Mucocoele of the paranasal sinuses: a retrospective clinical and histological study. J Laryngol Otol. 1978;92:1075–82.
- Hesselink JR, Weber AL, New PF, et al. Evaluation of mucoceles of the paranasal sinuses with computed tomography. Radiology. 1979;133:397–400.
- Koktekir BE, Karalezli A, Topal O, Erbek S. Strabismus secondary to frontal sinus mucocele associated with nasal polyposis. J Craniofac Surg. 2012;23(4):e340–1.
- 29. Avery G, Tang RA, Close LG. Ophthalmic manifestations of mucoceles. Ann Ophthalmol. 1983;15:734–7.
- 30. Garston JB. Frontal sinus mucocele. Proc R Soc Med. 1968;61:549-51.
- Flint PW, Haughey BH, Lund VJ, et al. Cummings otolaryngology head and neck surgery. 5th ed. Philadelphia: Mosby Elsevier; 2010.
- Nakayama T, Mori K, Maeda M. Giant pyocele in the anterior intracranial fossa case report. Neurol Med Chir (Tokyo). 1998;38:499–502.
- Voegels RL, Balbani AP, Santos Junior RC, et al. Frontoethmoidal mucocele with intracranial extension: a case report. Ear Nose Throat J. 1998;77:117–20.
- 34. Vissochi M, Esposito G, Della Pepa GM, Doglietto F, Nucci CG, Fontanella MM, Montano M. Giant frontal mucocele complicated by subdural empyema: treatment of a rare association. Acta Neurol Belg. 2012;112(1):85–90.
- Leventer DB, Linberg JV, Ellis B. Frontoethmoidal mucoceles causing bilateral chorioretinal folds. Arch Ophthalmol. 2001;119(6):922–3.
- Har-El G. Transnasal endoscopic management of frontal mucoceles. Otolaryngol Clin North Am. 2001;34:243–51.
- Rubin JS, Lund VJ, Salmon B. Frontoethmoidectomy in the treatment of mucoceles. A neglected operation. Arch Otolaryngol Head Neck Surg. 1986;112:434–6.
- Khong JJ, Malhotra R, Selva D, Wormald PJ. Endoscopic sinus surgery for paranasal sinus mucocele including modified endoscopic Lothrop procedure for frontal sinus mucocele. J Laryngol Otol. 2004;118(5):352–6.
- Lund VJ. Endoscopic management of paranasal sinus mucoceles. J Laryngol Otol. 1998; 112:36–40.

- Kuhn FA, Javer AR. Primary endoscopic management of the frontal sinus. Otolaryngol Clin North Am. 2001;34:59–75.
- 41. Har-El G, Dimaio T. Histologic physiologic studies of marsupialized sinus Mucoceles. J Otolaryngol. 2000;29(4):195–8.
- 42. Delfini R, Missori P, Iannetti G, et al. Mucoceles of the paranasal sinuses with intracranial and intraorbital extension: report of 28 cases. Neurosurgery. 1993;32:901–6.
- 43. Ikeda K, Takahashi C, Oshima T, et al. Endonasal endoscopic marsupialization of paranasal sinus mucoceles. Am J Rhinol. 2000;14:107–11.
- 44. du Mayne MD, Moya-Plana A, Malinvaud D, Laccourreye O, Bonfils P. Sinus mucocele: natural history and long-term recurrence rate. Eur Ann Otorhinolaryngol Head Neck Dis. 2012;129(3):125–30.

# Chapter 14 Pott's Puffy Tumor

### **Richard R. Orlandi**

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### **Core Messages**

- Pott's puffy tumor was defined by the eighteenth 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.

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<sup>©</sup> Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_14

• 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.

## 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" [2, 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 focal osteitis or osteomyelitis or through infectious thrombophlebitis [1, 8]. The posterior table of the frontal sinus is almost completely composed of compact bone while 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 while 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 thrombophlebit 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 eighteenth century description of frontal subpericranial abscess still remains pertinent over 200 years later. Patients typically do not have a history of chronic or recurrent acute frontal sinusitis, although 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 [2]. 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. Headache, fever, and nasal drainage are common associated symptoms and frontal sinus tenderness is typically present as well. 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 meningitis, venous sinus thrombosis, subdural abscess or brain abscess can also complicate this disease. 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. 14.1) [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.



**Fig. 14.1** 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)

• 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].

# 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 sub-pericranial 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



Fig. 14.2 Removal of the anterior table of the frontal bone (Reidel procedure) leaves a significant aesthetic defect

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 Reidel procedure (Fig. 14.2). The Reidel 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. Split calvarial bone grafts, polymethyl-methacrylate, hydroxyapatite, and titanium mesh have all 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 6 weeks.

Organisms cultured from Pott's puffy tumor tend to be microaerophilic streptococci, including alpha-hemolytic streptococcus and peptostreptococcus. Anaerobic bacteria may be isolated as well. 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.

# Conclusions

Pott's puffy tumor, described over 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.

Conflicts of Interest None

# References

- 1. Altman S, Austin MB, Tom LTC, Knox GW. Complications of frontal sinusitis in adolescents: case presentations and treatment options. Int J Pediatr Otorhinolaryngol. 1997;41:9–20.
- Bambidakis NC, Cohen AR. Intracranial complications of frontal sinusitis in children: Pott's puffy tumor revisited. Pediatr Neurosurg. 2001;35:82–9.
- 3. Blackshaw G, Thomson N. Pott's puffy tumour reviewed. J Laryngol Otol. 1990;104:574-7.
- Chandra RK, Schlosser R, Kennedy DW. Use of the 70-degree diamond burr in the management of complicated frontal sinus disease. Laryngoscope. 2004;114:188–92.
- 5. Davis W. Development and anatomy of the nasal accessory sinuses in man. Philadelphia: Saunders; 1914.
- 6. Flamm ES. Percival Pott: an 18th century neurosurgeon. J Neurosurg. 1992;76:319-26.
- Kuttenberger JJ, Hardt N. Long-term results following reconstruction of craniofacial defects with titanium micro-mesh systems. J Craniomaxillofac Surg. 2001;29:75–81.
- 8. Marshall AH, Jones NS. Osteomyelitis of the frontal bone secondary to frontal sinusitis. J Laryngol Otol. 2000;114:944–6.
- 9. Onodi A. Accessory sinuses of the nose in children. New York: William Wood; 1911.
- Strumas N, Antonyshyn O, Caldwell CB, Mainprize J. Multimodality imaging for precise localization of craniofacial osteomyelitis. J Craniofac Surg. 2003;14:215–9.

# Chapter 15 The Frontal Sinus and Nasal Polyps

# Dustin M. Dalgorf and Richard J. Harvey

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#### **Core Messages**

- Chronic rhinosinusitis with nasal polyposis is a chronic inflammatory condition of the upper airways and is not a simple disease of ostiomeatal complex occlusion.
- The presence of nasal polyposis in the frontal sinus most often does not cause frontal headaches or major symptoms localized to the frontal sinuses.
- Frontal sinus disease contributes to the total inflammatory burden of the sinuses and endoscopic sinus surgery aims to reduce the overall inflammatory load rather than address disease specific symptoms.

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- The goal of endoscopic sinus surgery is to create a functional wide-open common cavity to enable maximal delivery of topical anti-inflammatory therapy and removal of hypersecretory mucin.
- Topical corticosteroid therapy does not reach the sinus prior to surgery and the use of simple sprays is ineffective in any state of the sinus.
- There are three endoscopic surgical options to address the frontal sinus: (1) sphenoethmoidectomy with preservation of the frontal recess outflow tract (2) Draf 2a frontal sinusotomy and (3) Draf 3 modified endoscopic Lothrop procedure.
- The distinction between which procedures to choose is not based on a simple hierarchy of more extended surgery. The choice is based on both anatomical and disease factors that must be addressed in order to incorporate the frontal sinus into the new common cavity and provide good access to topical therapy.

### Introduction

Chronic rhinosinusitis with nasal polyps (CRSwNP) is a heterogeneous group of phenotypes and likely represents the end-point of multiple etiologies, rather than a single disease process. CRSwNP is a subtype of chronic rhinosinusitis (CRS), which generally presents with worse disease severity and poorer treatment outcomes compared to CRS without nasal polyps. Current concepts in the pathogenesis of CRSwNP focus on the inflammatory process secondary to dysregulation of local immune function, impaired epithelial barrier and a sustained inflammatory response to foreign antigens [1]. CRSwNP is characterized by an excessive T-helper 2 (TH<sub>2</sub>) inflammatory process, eosinophilic infiltration and decreased regulatory T-cell (Treg) function.

CRSwNP is a chronic inflammatory condition of the upper airways and is not a simple disease of ostiomeatal complex (OMC) occlusion [2, 3]. Radiographic studies demonstrate that OMC occlusion was correlated with sinus disease only for CRS patients without nasal polyps (CRSsNP) and not for CRSwNP [2, 3]. Patients with CRSwNP have diffuse sinus mucosal inflammation that is unlikely to be caused by local anatomical obstruction. Any interventions focused on ventilation of the OMC and correcting the drainage pathways of the anterior functional unit (comprised of the maxillary, anterior ethmoid and frontal sinuses) is unlikely to provide significant modification of the underlying chronic inflammatory process or maintain long-term symptom control.

• The goal of endoscopic sinus surgery is to create a wide-open common cavity to enable maximal delivery of topical anti-inflammatory therapy and removal of hypersecretory mucin.

It has been demonstrated in patients with CRS that local mucosal inflammation can be well controlled when corticosteroid solution is delivered with a high volume high pressure irrigation device [4]. When subgroup analysis was performed, both CRSwNP and the most challenging eosinophilic patients (>10/high power field [HPF]) had as good or better improvement in symptoms, Sino-Nasal Outcome Test 22 (SNOT-22) and endoscopy scores compared to CRS without nasal polyps or those with low tissue eosinophilia ( $\leq$ 10/HPF) [4].

A recent meta-analysis has shown that the use of topical corticosteroid after endoscopic sinus surgery is beneficial for the management of CRSwNP [5]. This study included 40 studies and 3,624 patients. When compared to placebo, topical corticosteroids improved overall symptom score, decreased polyp score, reduced polyp size and prevented polyp recurrence after sinus surgery. There was a greater reduction of polyp score when topical corticosteroid was administered any time after endoscopic sinus surgery compared to patients who never had surgery.

The final endpoint is to establish a functional sinonasal cavity. This involves complete removal of all sinus partitions to create a common sinus cavity. This will also avoid leaving behind disconnected cells, mucocele formation, mucus recirculation, overcome obstructive phenomenon and enable maximal delivery of topical corticosteroid therapy. There is little basis for surgery with the aim of correcting ventilation and drainage nor is there a role for giving patients pre-operative systemic steroids, then performing a sinus procedure without a long-term post-operative plan of controlling the inflammation.

### Diagnosis

The presence of nasal polyposis in the frontal sinus most often does not cause frontal headaches or major symptoms localized to the frontal sinuses. Among patients with radiographic evidence of frontal recess obstruction and/or frontal sinus disease, symptoms of frontal pain or headache was more common in CRS without polyposis compared to CRSwNP [6]. Within the CRSwNP group, only 29 % reported symptoms of frontal pain or headache regardless of the degree of sinus opacification [6].

• If the degree of inflammatory frontal disease does not cause disease specific symptoms, then why should a surgeon choose to operate on the frontal sinuses (especially in the setting of primary sinus surgery)?

The concept of the inflammatory burden of disease describes patients with the highest inflammatory load as those having eosinophilic CRS with nasal polyposis and concomitant asthma and/or aspirin intolerance [7]. These patients represent the extreme end of the "inflammatory spectrum" and experience worse subjective and objective post-operative outcomes, higher recurrence rates and need for revision surgery. It is hypothesized that more radical surgery achieves the goal of eradicating the presence of proinflammatory mediators such as eosinophils in mucosa,

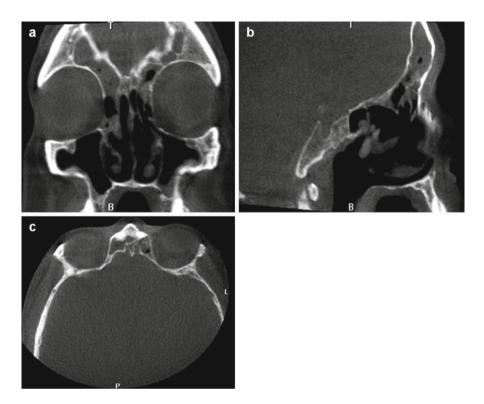


Fig. 15.1 Patient with chronic rhinosinusitis with nasal polyposis having undergone previous endoscopic sinus surgery. (a) Coronal (b) sagittal and (c) axial CT scan demonstrates extensive frontal sinus disease contributing to the overall inflammatory burden of disease

eosinophilic mucin, fungal and staphylococcal antigens and bacterial load that contribute to the local inflammatory burden [7]. Frontal sinus disease contributes to the total inflammatory burden of the sinuses (Fig. 15.1).

• The decision to operate on the frontal sinus is not to address disease specific symptoms, but in effort to reduce the overall inflammatory burden of disease.

Systemic corticosteroids are the mainstay of managing frontal sinus disease prior to surgery. However, the prolonged use of systemic corticosteroids can be associated with complications involving different organ systems. Many of these effects are either dose and/or duration-dependent. Among clinicians, there is great variability in prescribing practices because there is little known about the risks associated with multiple short courses of corticosteroids that are often used in the management of CRSwNP. It is believed that the duration of corticosteroid therapy should be 2–3 weeks in duration in order to reflect the life cycle of tissue eosinophils. It has been demonstrated that CRSwNP patients receiving more than three short courses of systemic corticosteroid treatment per year (more than 21 days per year of treatment, prednisolone 1 mg/kg/day for a 6–10 day course) had reduced bone mineral density [8]. In this study, 10.9 % and 43.5 % had osteoporosis and osteopenia respectively in the lumbar spine and 48.8 % had asymptomatic adrenal insufficiency [8]. Avascular necrosis of the femoral hip is a rare event and reported in doses over 40 mg and a total accumulative steroid dose of 290–1,000 mg [9]. Thus, if more than three courses of systemic steroid therapy are required within a 12 month period (with each course a maximal accumulative dose of 290–1,000 mg) to control patient symptoms then consideration for wide endoscopic sinus surgery with the goal of transitioning the patient from systemic therapy to topical corticosteroid therapy is indicated.

The complication profile associated with systemic corticosteroid therapy is the key aspect driving the need to transition therapy for chronic inflammatory airways disease from systemic to topical methods of drug delivery. A history of response to previous oral corticosteroids can provide important diagnostic information.

• Nasal polyposis patients that are steroid responsive are likely to have greater benefit from post-operative topical corticosteroid therapy.

There is good evidence to show that topical corticosteroid therapy does not reach the sinus prior to surgery and the use of simple sprays is ineffective in any state of the sinus [10]. Total sinus distribution of topical irrigation improved significantly to all sinuses after endoscopic sinus surgery [10]. In particular, distribution to the frontal sinuses was almost undetectable pre-operatively and significantly improved after surgery [10]. Distribution of nasal irrigation was also influenced by the type of delivery device. Delivery via neti pot and squeeze bottle techniques were significantly better than pressurized spray techniques both prior and after surgery [10].

# **Functional Frontal Sinus Surgery**

The goal of frontal sinus surgery is the same as surgery for all other sinuses. The objective is a wide-open frontal sinus that is incorporated with the other sinuses into a new common cavity. The creation of a single common cavity enables maximal delivery of topical corticosteroid therapy in order to achieve long-term inflammatory control. There are three options to attain this goal in the frontal sinuses: (1) sphenoethmoidectomy with preservation of the frontal recess outflow tract (2) Draf 2a frontal sinusotomy and (3) Draf 3 endoscopic modified Lothrop procedure (EMLP). The distinction between which procedures to choose is not based on a simple hierarchy of more extended surgery. It is not a case of "doing a bit more to the sinus as it is misbehaving", but rather it is important to have a scientific approach. The choice of procedure is based on both anatomical and disease factors that must be addressed in order to incorporate the frontal sinus into the new common cavity and provide good access to topical therapy.

Performing a sphenoethmoidectomy with preservation of the frontal sinus is most appropriate in patients with small or hypoplastic frontal sinuses, low surface area, low inflammatory disease burden and minimal contribution of the frontal

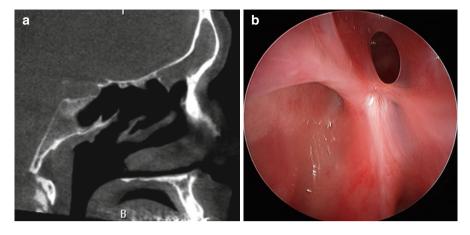


Fig. 15.2 Iatrogenic frontal sinus disease secondary to scarring between the uncinate process and bulla ethmoidalis. (a) Sagittal CT scan demonstrating frontal sinus opacification and (b) endoscopic view of mucosal adhesion secondary to scarring between the uncinate process and bulla ethmoidalis

sinuses to overall inflammatory control. In such cases when the frontal sinus is preserved and not addressed, it is critical to prevent iatrogenic frontal disease by staggering the height of the bulla and uncinate process. This will avoid scarring and adhesion of these structures post-operatively and maintain function of the frontal recess (Fig. 15.2).

A Draf 2a frontal sinusotomy is performed when it is the first procedure on the frontal sinus. The Draf 2a frontal sinusotomy must be of sufficient size in order to ensure patency and access to topical corticosteroid therapy postoperatively. It has been demonstrated that a minimum Draf 2a intra-operative diameter of 5 mm is necessary to prevent stenosis of the frontal recess [11]. Below an intra-operative diameter of 5 mm the ostia obstruction rate was greater than 30 % post-operatively [11]. In this study, the overall average minimum intra-operative diameter of the frontal neo-ostium was 5.6 mm, which was reduced to 3.5 mm after healing post-operatively [11]. It has also been shown that a minimum ostial dimension of 3.95 mm is required to enable penetration of topical irrigation into the sinus [12]. If the data from these two studies are extrapolated, in order to achieve a healed post-operative mucosa lined frontal sinus opening of at least 4–5 mm, a frontal recess bony opening must be a minimum of 10 mm intra-operatively.

A Draf 3 EMLP is indicated when performing a second procedure on the frontal sinus or in the setting of complex and multiple frontal cells. A Draf 3 can be indicated in primary frontal sinus surgery in certain high-risk patient populations. A recent study evaluated the success rate of Draf 2a frontal sinusotomy [13]. This study demonstrated that 14 % of patients undergoing either primary or revision Draf 2a procedures had persistent symptoms despite ongoing post-operative maximal medical therapy and required a Draf 3 procedure [13].

• Among the patients requiring Draf 3, those with multiple risk factors including asthma, nasal polyps, Lund-Mackay score >16 and frontal ostium size <4 mm were at particular risk of a poor surgical outcome from a Draf 2a procedure [13].

This patient group with accumulative risk factors should be considered for performance of a primary Draf 3 EMLP.

A Draf 2b procedure, described as removal of the frontal sinus floor from the lamina papyracea extending medially to the nasal septum, has little role in chronic inflammatory sinus disease.

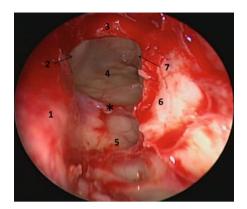
• Draf2b procedures are not commonly performed because it causes circumferential injury leading to a higher likelihood of restenosis compared to Draf 2a procedures 23 % and 3.6 % respectively [14].

Regardless of the type of frontal sinus procedure performed, the management of nasal polyposis must focus on controlling the underlying inflammatory process. Corticosteroids are the foundation of current anti-inflammatory therapy. Prolonged use of systemic corticosteroids places the patient at risk of potential side effects. As an alternative, topical delivery of corticosteroids provides an effective method of disease control with minimal risk of complications. The role of endoscopic sinus surgery in CRSwNP or eosinophilic CRS is to create wide access of the sinus mucosa for long-term symptom control with topical anti-inflammatory therapy [1]. Effective topical delivery depends on a wide-open common sinus cavity and the method of topical delivery system [1].

• High volume high-pressure delivery systems maximize delivery of topical corticosteroid therapy and facilitate mechanical lavage of the hypersecretory mucus, inflammatory products and disruption of bacterial biofilms [10].

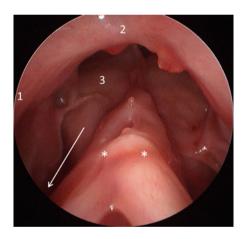
# Draf 2a Frontal Sinusotomy

Performance of a Draf 2a frontal sinusotomy is achieved by complete dissection of all cells within the anatomical limits of the frontal recess (Fig. 15.3). The defined anatomical limits of dissection establish the boundaries of the paranasal surgical box including the horizontal and vertical components of this box [15]. The boundaries of the horizontal component of the paranasal surgical box are defined during the complete sphenoethmoidectomy including the medial orbital wall laterally, middle turbinate medially and skull base superiorly [15]. These limits are extended superiorly to delineate the boundaries of the vertical component of the paranasal surgical box. The boundaries of the vertical paranasal surgical box define the frontal recess and include the middle turbinate and intersinus septum medially, lamina papyracea and supraorbital roof laterally, nasofrontal beak anteriorly, and skull base posteriorly [15] (Fig. 15.3). Identification of the anatomical limits of the surgical box and removal of all cells and partitions within its confines ensures complete dissection of the frontal recess.



**Fig. 15.3** Complete left Draf 2a frontal sinusotomy. The limits of dissection that define the vertical component of the paranasal surgical box are viewed with a single position of the endoscope. Medial landmarks include (1) middle turbinate and (2) frontal intersinus septum, anterior landmark (3) nasofrontal beak, posterior landmarks (4) posterior table frontal sinus and (5) skull base, lateral landmarks (6) lamina papyracea and (7) supraorbital roof. The asterisk identifies the anterior ethmoid artery

Fig. 15.4 Complete Draf 3 modified endoscopic Lothrop procedure. The limits of the Draf 3 cavity are defined as (1) orbital plates of frontal bone and periosteum of the skin over the frontal process of the maxilla, (2) anterior table of frontal sinus/nasofrontal beak, and (3) posterior table of frontal sinus, first olfactory fascicle (asterisk). The arrow indicates the frontal sinus outflow tract



# Draf 3 Endoscopic Modified Lothrop Procedure

A Draf 3 EMLP is achieved by removal of all bone along the floor of the frontal sinus from lamina papyracea to lamina papyracea (Fig. 15.4). The goal of Draf 3 is to create a frontal sinus opening wider than the normal anatomical limits of the frontal recess as described above. Traditionally, the bone of the frontal sinus floor is removed following identification of at least one frontal recess and often with the use of angled endoscopes. However, identification of the frontal recess can be difficult due to severe burden of disease, scarring and occupation of tumor. The outside-in Draf 3 is an alternative approach that avoids initial dissection in the frontal recess where the anatomy can be most challenging and begins drilling away from the

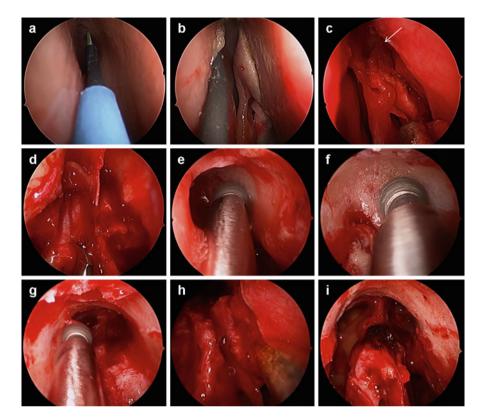


Fig. 15.5 Sequential steps of the outside-in Draf 3 modified endoscopic Lothrop procedure. (a) Marking the position of the nasofrontal beak with the assistance of image guidance, (b) mucosal cuts over the septum and lateral nasal wall, (c) identification of the first olfactory neuron (*arrow*), (d) creation of the septal window with the first olfactory neuron and orbital plates of the frontal bone on either side visible with a single position of the endoscope, (e) identification of periosteum of the skin over the frontal process of the maxilla, (f) after identification of all landmarks the nasofrontal beak is drilled from outside-in, (g) identification of the frontal sinus, (h) frontal recess dissection in order to connect the frontal and lower sinus cavities, (i) complete Draf 3 modified Lothrop cavity

frontal recess [16]. In this approach, the limits of the endoscopic Draf 3 cavity are established early on during the procedure allowing safe removal of bone within the confines of these limits (Fig. 15.5). The traditional Draf 3 approach identifies these limits at the end of the procedure and these limits are only used to define the end-point of dissection. Laterally, the limits of the Draf 3 cavity include the orbital plates of the frontal bone and periosteum of the skin over the frontal process of the maxilla on both sides [16]. Posteriorly, the first olfactory fascicle on each side demarcates the skull base at the forward projection of the of the frontal sinus [16]. Anteriorly, the dissection is taken to the plane of the anterior table of the frontal sinus [16]. The outside-in approach to Draf3 was demonstrated to be shorter and more predictable across various types of sinus pathology compared to the traditional approach [16].

### **Obliterative Frontal Sinus Surgery**

Frontal sinus surgery can be functional (Draf 3 EMLP) or obliterative (osteoplastic flap with frontal sinus obliteration and cranialization). External approaches to the frontal sinus are not necessarily obliterative in nature.

Indications for Osteoplastic flap with frontal sinus obliteration (usually with fat):

- When it is unlikely to achieve a functional connection of the frontal sinus back to the lower sinuses or nasal cavity.
- Post-radiation therapy
- · Loss of medial orbital wall and medialization of orbital contents
- Trauma and naso-orbitoethmoidal fractures resulting in narrowing or closure of the anteroposterior frontal recess distance.

Frontal sinus cranialization is rare and mainly indicated in tumor cases. Although the exact incidence is unknown, the major risk of external frontal sinus approaches is delayed mucocele formation and chronic forehead pain [17].

# Conclusion

Addressing the frontal sinuses in the setting of polyposis is a carefully considered undertaking. The era of performing 'ventilation' surgery on a polyposis patient who is in an operation under the influence of extensive systemic corticosteroid is no longer valid. The decision to address the frontal sinus is based on the degree of symptoms, burden of inflammation, anatomical limits of the frontal anatomy and the need to transition a patient away from reliance on systemic medication to treat their frontal sinus to local topical care. Consideration for the current disease, potential exacerbations and chronicity should be made when deciding on the appropriate intervention for a patient.

# References

- Chin D, Harvey RJ. Nasal polyposis: an inflammatory condition requiring effective antiinflammatory treatment. Curr Opin Otolaryngol Head Neck Surg. 2013;21(1):23–30.
- Leung RM, Kern RC, Conley DB, et al. Ostiomeatal complex obstruction is not associated with adjacent sinus disease in chronic rhinosinusitis with polyps. Am J Rhinol Allergy. 2011;25(6):401–3.
- Snidvongs K, Chin D, Sacks R, et al. Eosinophilic rhinosinusitis is not a disease of ostiomeatal occlusion. Laryngoscope. 2013;123(5):1070–4.
- Snidvongs K, Pratt E, Chin D, et al. Corticosteroid nasal irrigations after endoscopic sinus surgery in the management of chronic rhinosinusitis. Int Forum Allergy Rhinol. 2012;2(5):415–21.
- 5. Kalish L, Snidvongs K, Sivasubramaniam R, et al. Topical steroids for nasal polyps. Cochrane Database Syst Rev. 2012;12:CD006549.

- DelGaudio JM, Wise SK, Wise JC. Association of radiological evidence of frontal sinus disease with the presence of frontal pain. Am J Rhinol. 2005;19(2):167–73.
- 7. Bassiouni A, Naidoo Y, Wormald PJ. When FESS fails: the inflammatory load hypothesis in refractory chronic rhinosinusitis. Laryngoscope. 2012;122(2):460–6.
- Bonfils P, Halimi P, Malinvaud D. Adrenal suppression and osteoporosis after treatment of nasal polyposis. Acta Otolaryngol. 2006;126(11):1195–200.
- 9. Poetker DM, Smith TL. What rhinologists and allergists should know about the medico-legal implications of corticosteroid use: a review of the literature. Int Forum Allergy Rhinol. 2012;2(2):95–103.
- Harvey RJ, Goddard JC, Wise SK, et al. Effects of endoscopic sinus surgery and delivery device on cadaver sinus irrigation. Otolaryngol Head Neck Surgery Off J Am Acad Otolaryngol-Head Neck Surg. 2008;139(1):137–42.
- 11. Hosemann W, Kuhnel T, Held P, et al. Endonasal frontal sinusotomy in surgical management of chronic sinusitis: a critical evaluation. Am J Rhinol. 1997;11(1):1–9.
- Grobler A, Weitzel EK, Buele A, et al. Pre- and postoperative sinus penetration of nasal irrigation. Laryngoscope. 2008;118(11):2078–81.
- Naidoo Y, Bassiouni A, Keen M, et al. Risk factors and outcomes for primary, revision, and modified Lothrop (Draf III) frontal sinus surgery. Int Forum Allergy Rhinol. 2013;3(5):412–7.
- Dhepnorrarat RC, Subramaniam S, Sethi DS. Endoscopic surgery for fronto-ethmoidal mucoceles: a 15-year experience. Otolaryngol Head Neck Surgery Off J Am Acad Otolaryngol-Head Neck Surg. 2012;147(2):345–50.
- Dalgorf DM, Harvey RJ. Chapter 1: sinonasal anatomy and function. Am J Rhinol Allergy. 2013;27 Suppl 1:3–6.
- Chin D, Snidvongs K, Kalish L, et al. The outside-in approach to the modified endoscopic Lothrop procedure. Laryngoscope. 2012;122(8):1661–9.
- Lee JM, Chiu AG. Role of maximal endoscopic sinus surgery techniques in chronic rhinosinusitis. Otolaryngol Clin North Am. 2010;43(3):579–89, ix.

# Chapter 16 Pediatric Frontal Sinusitis

Kenneth D. Rodriguez and Charles S. Ebert Jr.

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# Introduction

Rhinosinusitis (RS) is a common diagnosis affecting more than 31 million adults annually [1].

• The development of rhinosinusitis is multifactorial and may involve the interaction of numerous factors such as viruses, bacteria, fungi, allergic disease, nonallergic inflammation, genetic and anatomical causes [2].

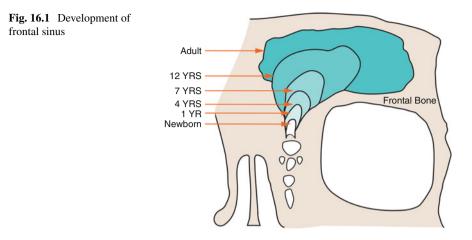
While there is clear evidence that rhinosinusitis adversely affects the quality of life in both adults and children, making the diagnosis in children is at times more

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difficult than in adults [3, 4]. The prevalence of chronic rhinosinusitis in children is higher in younger age groups likely due to higher incidence of viral upper respiratory tract infections [3]. Frontal rhinosinusitis is even a more uncommon finding and a challenging diagnostic entity since symptoms are often non-specific.

The frontal sinus is the last of the paranasal sinuses to develop and at birth is not aerated. Development typically begins at approximately 2 years of age and continues growth through puberty (Fig. 16.1) [5]. Nearly 3 % of children under 5 years of age have frontal sinuses while up to 50 % have aerated fontal sinuses by age 10 [6]. By adulthood, hypoplasia or poor pneumatization of the frontal sinus is found in up to 30 %, while aplasia is present unilaterally in 15 % and bilaterally in 5 % of adults [5, 7]. (A comprehensive review of paranasal sinus embryology and anatomy is found in Chap. 2).

This chapter will provide an overview of the pathogenesis of rhinosinusitis in children, as well as an overview on current classifications and diagnoses. In addition, this chapter will provide the reader with current medical and surgical treatment options as it relates to the frontal sinus in children.

### **Diagnosis and Definitions**

The guidelines for diagnosing pediatric rhinosinusitis were originally based on adult criteria [8]. In 2001 the American Academy of Otolaryngology and the American Academy of Pediatrics categorized childhood infections involving the upper respiratory tract and paranasal sinuses based on duration and severity of symptoms (See Table 16.1) [9]. Symptoms linked to sinusitis include: cough, purulent nasal drainage, nasal congestion, fatigue, hyposmia/anosmia, otalgia, maxillary tooth pain, fevers, and headache/facial pain. The duration of these symptoms allows for the classification of rhinosinusitis into the following categories: acute viral,

Туре	Duration	Symptoms
Acute viral	<10 days	Nasal drainage, cough, fever (if present) occurs within first 24 h and is typically low grade
Acute bacterial	10-30 days	Purulent nasal drainage, nasal obstruction, facial pain and pressure, cough, fever >102, headache, sore throat, halitosis, hyposmia, fatigue, ETD
Subacute bacterial	30–90 days	
Recurrent acute	10–30 days separated by 10 days of symptom resolution	
Chronic	>90 days	Purulent nasal drainage, nasal obstruction, facial pain and pressure, cough, headache, sore throat, halitosis hyposmia, fatigue, ETD

 Table 16.1
 Classification of pediatric sinusitis [2, 9, 13]

ETD Eustachian tube dysfunction

acute bacterial, subacute bacterial, recurrent acute bacterial, and chronic (See Table 16.1).

It can be difficult for the physician to distinguish between a URI, adenoid hypertrophy, allergic disease and sinusitis. Children typically have six to eight episodes of viral upper respiratory tract infections per year with only an estimated 5-13 % complicated by secondary bacterial sinusitis [10–12]. When eliciting a history emphasis should be placed on specific symptoms such as facial pain and pressure, cough, nasal drainage, sense of smell, nasal airway obstruction, or fevers. It cannot be overstated that sinusitis is a combination of these symptoms without any one symptom being pathognomonic for acute or chronic sinusitis. The duration of the condition should be determined as true bacterial acute sinusitis is more likely to be present if symptoms last more than 7–10 days, often with a sudden worsening of symptoms.

• Children's symptoms of rhinosinusitis are varied and there is no symptom that can differentiate viral from bacterial pathology.

Typically, fevers over 102 °F along with purulent nasal discharge over 3–4 days favor bacterial sinusitis over viral etiology of symptoms.

• Viral upper respiratory tract infections tend to last 5–7 days and resolve or at least significantly abate by 10 days [9].

Younger children may present with increased irritability instead of facial pain or headache complaints and cough seems to be a more common complaint of children with CRS [3]. Although parental input for younger children may help elicit symptoms and their duration, their interpretation of symptoms can be misleading. Older children and adolescents can be more helpful describing their symptoms of nasal congestion, hyposmia, or facial pain/pressure. In children, in addition to allergy, asthma, and reflux one must always keep entities such as cystic fibrosis (CF), immune deficiency, primary ciliary dyskinesia (PCD), foreign body, cleft palate, and adenoid hypertrophy as contributing or causative of the patient's symptoms. • Although challenging, nasal endoscopy is extremely valuable and should be attempted when feasible. If purulent material is noted, culture can be obtained and guide medical therapy.

While obtaining samples from the frontal sinus itself in unoperated sinuses is rare, in certain circumstances (Pott's Puffy Tumor) it can be accomplished by direct tap into the frontal sinus through the forehead. That being said, any patient with suspected osteomyelitis of the frontal bone should be admitted and comprehensively evaluated.

Imaging can be a valuable adjunct in assisting with the diagnosis but has inherent radiation exposure concerns for children. To assess this risk, a retrospective cohort study was performed where patients without previous cancer diagnoses under the age of 22 who were evaluated with CT between 1985 and 2002 were studied [14]. They determined that use of CT scans in children to deliver cumulative doses of about 50 mGy might almost triple the risk of leukemia (five to ten head CT's – accumulation of 50 mGy red bone marrow dose) and doses of about 60 mGy (two to three head CT's about 60 mGy cumulative brain dose) might triple the risk of brain cancer [14]. The cumulative absolute risk in the 10 years after the first scan for patients younger than 10 years equates to one excess case of leukemia and one excess case of brain tumor per 10,000 head CT scans [14]. For children with normal life expectancy, the lifetime excess risk of any incident cancer for a head CT scan is about 1 cancer per 2,000 scans for exposure at age 15 years [15].

The utility of CT, when clinically indicated is high (Figs. 16.2, 16.3, and 16.4). However, without a history indicative of CRS results can be confounding. A study



Fig. 16.2 Coronal non-contrast CT image of 6 year old female patient with chronic left sided frontal sinus mucosal thickening

Fig. 16.3 Sagittal non-contrast CT image of a 4 year old male patient with chronic sinusitis. The patient has total opacification of the frontal sinus with near total opacification of the ethmoid cavity and notable mucosal thickening with the sphenoid sinus



Fig. 16.4 Sagittal non-contrast CT image of a 6 year old female patient with chronic sinusitis. The patient has total opacification of the frontal sinus with mucosal thickening within the anterior ethmoid cavity. There is some mild mucosal thickening on the floor of the sphenoid sinus

31 W 2500 : L 500

was performed to determine the incidental Lund-MacKay scores for pediatric patients without chronic rhinosinusitis [16]. Of 191 patients with mean age of 9, the frontal sinuses were absent in 40.1 % and the mean scaled Lund score was 2.81 [16]. Only 37 (19.3 %) patients had completely radiographically normal sinuses [16]. The clinical practice guidelines from Pediatrics recommend against imaging studies to confirm a diagnosis of clinical sinusitis in children <6 years of age but CT scans of the paranasal sinuses should be performed in patients in whom surgery is being considered [9].

# Pathogenesis

It is critically important to understand the pathogenesis of RS to maximize treatment options. Although the etiology of RS is not fully understood, it seems that the interactions of numerous variables play a role in the development inflammation resulting in RS. Infectious microorganisms clearly play an important role in the inflammatory process.

• Viral infections are known to destroy the cilia of the epithelium and result in stasis of secretions providing a milieu for bacterial overgrowth [4].

This process causes mucosal edema, which can result in ostial obstruction. This phenomenon is of particular concern in the frontal sinus, where the natural ostia is narrow. If the acute infectious process does not clear, the edema persists and results in CRS [4].

In contrast to adults, there is evidence to suggest that CRS in children is more of a TH-1 (neutrophilic) dominated process than in adults who demonstrate frequent TH-2 (eosinophilic) predominance [3, 4, 17].

• The colonization of the adenoid pad in children by bacteria or biofilms of bacteria may predispose children to recurrent infections [3, 18]. In fact, similar bacteria isolates have been noted on the lateral nasal wall as in the adenoid pad [19].

In addition, the quantity of bacterial colonization correlates with nasal symptoms. While the frontal sinus is not intimately related to the nasopharynx, persistent bacterial infection resulting in inflammation in the sphenoid, posterior ethmoid, or maxillary sinuses could clearly affect mucosal edema in the frontal recess and subsequent frontal sinusitis (Fig. 16.3).

Allergies and gastroesophageal reflux (GERD) have also been implicated in predisposing children to CRS. Although these conditions do not have a causal relationship with CRS in children, evidence suggests a common link.

• It has been reported that up to 70 % of children with CRS have allergic rhinitis [20].

Phipps et al. noted that children with medically refractory CRS had a high incidence (63 %) of GERD diagnosed with nasopharyngeal and esophageal pH probes [21]. Other environmental factors such as exposure to tobacco smoke and pollutants may also contribute to disrupted ciliary function, thereby providing access for bacterial overgrowth [3].

CF and PCD represent a complex subset of patients with impaired mucociliary clearance. These patients are at high risk for development of CRS due to their underlying ciliary dysfunction and the refractory nature of the disease process. Their management typically involves multimodality therapy with earlier surgical intervention and sinonasal irrigation. Patients with CF are classically known to have underdeveloped para-nasal sinuses as well as near universal CT evidence of sinus disease [22]. Eggesbo et al. published on CT characteristics in patients with CF where 116 patients with ages 3–54 years and 136 control patients' ages 7–51 years were compared [22]. In patients with two CF mutations they found more frequent

frontal sinus aplasia, ethmoid and sphenoid hypoplasia, no concha bullosa or Haller cells, as well as low ethmoid roof in 30 % of children [22]. This CT data is especially relevant given that patients with CF, despite the appearance of their CT scan, often do not complain of sinonasal complaints and are often evaluated for deteriorating lung function [23]. Second, although the complication profile is similar between CF and non-CF patients with endoscopic sinus surgery (ESS) it is critical to understand that their sinus anatomy is distorted and thorough evaluation of the CT scan with the assistance of image guidance is indicated [24].

# **Medical and Surgical Therapy**

The current data on pediatric sinusitis microbiology is limited and antibiotic therapy is often empiric in children with symptoms consistent with sinusitis.

 A study of cultures taken from the middle meatus of 133 children demonstrated 72.4 % Streptococcus pneumoniae, 60.5 % Haemophilus influenza, and 58.3 % Moraxella catarrhalis isolated were resistant to first-line antibiotics [25].

This underscores the need for culture directed therapy where possible. Frontal sinus data is especially limited; therefore, some of the data must be extrapolated from studies that included adults. In a 2002 study in patients aged 11–72, aspirates of 15 acutely and 13 chronically infected frontal sinuses were studied [26].

- Microbiology of sinusitis [26]
  - Acute most common pathogens
    - · Haemophilus influenza
    - Streptococcus pneumoniae
    - Moraxella catarrhalis
  - Chronic sinusitis most common pathogens
    - Microaerophilic streptococci
    - H influenza
    - Staphylococcus aureus.

If there is no purulence noted, findings such as significant adenoid hypertrophy, turbinate hypertrophy, or nasal polyposis may help explain symptomatology.

There is at present no absolute consensus on first line antibiotic therapy for children with CRS. It is clear that resistance is becoming an ever increasing area of concern.

- Antibiotic therapy for CRS in children:
  - If a single medication is desired and no culture results are available amoxicillinclavulanic acid is a reasonable first line choice

- If MRSA is suspected clindamycin, trimethroprim-sulfamethoxazole, or doxycycline represent options either as single agents or in combination but clearly each medication has certain deficiencies
- For patients with CF, where Pseudomonas aeruginosa is routinely cultured, a fluoroquinolone is indicated but should be used with caution in the pediatric population given its risk of cartilage damage [27].

There is no clear standard for optimal treatment length in the pediatric population. Total antibiotic therapy for anywhere from 3 to 6 weeks is currently accepted for treatment of chronic rhinosinusitis [3, 28–30]. Topical intranasal steroids can also be utilized but there is no strong data to support this recommendation.

• A review of the literature in 2007 of topical treatments in pediatric rhinosinusitis was performed with the authors determining that with the exception of topical steroids (modest benefit) no significant evidence of effectiveness supports the use of topical treatments in pediatric patients [31].

CRS in children can warrant surgical intervention. Strong indications include complete nasal airway obstruction secondary to polyposis, mucocele, fungal sinusitis (allergic fungal sinusitis or fungus ball), as well as orbital complications (subperiosteal or orbital abscess) or intracranial abscess. For CRS, not responsive the medical therapy (3–6 weeks antibiotic therapy plus/minus topical steroid) surgical therapy should be considered.

• In patients with clinical evidence of adenoid hypertrophy, adenoidectomy should be considered prior to ESS.

In a meta-analysis performed in 2008 whose goal was to evaluate the currently available literature regarding the reported effectiveness of adenoidectomy alone in the management of medically refractory pediatric chronic rhinosinusitis, they determined that adenoidectomy reduces caregiver reported symptoms of chronic rhinosinusitis in a majority of pediatric patients [32]. They further conclude that given its simplicity, low risk profile, and effectiveness, adenoidectomy should be considered first line therapy for medically refractory, uncomplicated pediatric rhinosinusitis [32]. At the time of adenoidectomy, nasal endoscopy can be performed and if indicated cultures can be obtained. There are also reports of antral lavage adding significant benefit at the time of adenoidectomy for children with more severe sinus disease (based on CT) [33].

In a review of the role of sinus surgery in children by Ramadan et al. they note that the success of pediatric ESS ranged from 82 % to 100 % in selected patients and that children undergoing ESS for CRS usually have more severe disease compared to those who get medical treatment or adenoidectomy [34]. Although these results are promising, additional studies are needed to assess the best candidates for medical versus surgical therapy (ESS, adenoidectomy or both) in children. In addition, the extent of surgery should be considered. Clearly all of the diseased sinuses should be addressed but there have been questions as relates to growth of the facial skeleton

following ESS. Senior et al. published on eight patients following unilateral ESS for orbital complications with follow up averaging 6.9 years demonstrating minimal changes in facial volume measurements, specifically subtle enlargement of the orbit on the surgical side [35]. Thus, based on these findings, ESS does not dramatically change facial development in the pediatric population [35]. That being said, the senior author of this chapter recommends comprehensive surgery with the goal to open all air cells within the diseased regions. The risks of ESS in children have been shown to be comparable with the risk profile of adults with a major complication rate of 0.6 % in a 1998 meta-analysis [36].

Surgical approaches to the frontal sinus are covered in detail elsewhere in this text but will be briefly illustrated here. For operative disease of the frontal sinus the first surgical option should be endoscopic endonasal. Image guidance in this setting is often a helpful adjunct. Open approaches, with or without an endoscopic assist can be considered if an endonasal approach is difficult or not possible. Options include trephination versus brow or coronal approach with osteoplastic flap. The advantage of a coronal approach is preservation of forehead sensation by sparing the supraorbital nerves with the disadvantage being the large incision. That being said, the scar is often well camouflaged by the patient's hair. The brow incision can often be well camouflaged by the eyebrow or within a horizontal rhytid but can result in loss of sensation to the forehead.

### **Complications of Pediatric Frontal Rhinosinusitis**

• Complications of frontal sinusitis in children can be potentially life threatening and are due to the proximity of the frontal sinus to intracranial contents as well as the orbit.

The route of spread can be directly through bony dehiscence or retrograde flow through valve-less veins of Breschet (posterior table vessels). Multiple case reports are in the literature outlining Pott's Puffy Tumor in the pediatric population [37–39]. In a review published in 2006 of 16 pediatric patients with intracranial complications of frontal sinusitis, subdural (56 %), epidural (44 %), and cerebral abscesses (19 %) were the most common complications [40]. Meningitis was identified in 13 % as a solo entity and was associated with another intracranial complication in 6 % and polymicrobial cultures were obtained in 50 % of patients [40]. Headache, nasal congestion, and visual changes were the most common early symptoms with neurological findings indicating more advanced disease [40]. Another report identified pediatric orbital complications as a direct result of frontal sinus disease [41]. Once again, bony dehiscence and valveless veins are the likely mode of spread. The position of the abscess often can indicate whether the inflammation/abscess is of ethmoid (medial orbit) or frontal sinus (superior orbit) origin. Imaging is often needed to differentiate cellulitis from more serious orbital

abscess or cavernous sinus thrombosis. When possible, imaging with contrast should be utilized up front as this more clearly delineates inflammatory processes. Often CT is in the initial imaging modality utilized due to its availability, comfort level amongst Otolaryngologists, and superior view of the bony architecture. Coronal and sagittal reconstructions can be extremely helpful for surgical planning. MRI may serve to provide further information if clinically indicated especially of intracranial processes. Intravenous antibiotics with surgical therapy as dictated by exam and radiographical findings are warranted for treatment of these serious sequalae of frontal sinusitis. In regard to IV antibiotic choice, consideration should be given to penetration of the blood-brain barrier as it relates to intracranial infections.

# Conclusions

Frontal sinusitis in children is rare and represents a diagnostic dilemma. Untreated disease may lead to complications that require urgent treatment. Careful attention to clinical history that supports rhinosinusitis is necessary to make the diagnosis in children. A conservative approach of treatment should be utilized. Oral antibiotics are the first line therapy of bacterial frontal sinusitis. Surgical therapy (adenoidectomy/FESS) should be reserved for medical failures. For orbital or intracranial complications surgical therapy may be a first line option in conjunction with appropriate medical therapy.

# References

- 1. Slavin RG. Management of sinusitis. J Am Geriatr Soc. 1991;39:212-7.
- Chandran SK, Higgins TS. Pediatric rhinosinusitis: definitions, diagnosis and management an overview. Am J Rhinol Allergy. 2013;27(3):s16–9.
- Wu AW, Shapiro NL, Bhattacharyya N. Chronic rhinosinusitis in children: what are the treatment options. Immunol Allergy Clin N Am. 2009;29:705–17.
- Lusk R. Chronic rhinosinusitis: contrasts between children and adult patients. Clin Allergy Immunol. 2007;20:287–98.
- Scuderi AJ, Harnsberger HR, Boyer RS. Pneumatization of the paranasal sinuses: normal features of importance to the accurate interpretation of CT scans and MR images. AJR Am J Roentgenol. 1993;160(5):1101–4.
- Cannon CR, McCay B, Halton JR. Paranasal sinus development in children and its relationship to sinusitis. J Miss State Med Assoc. 1995;36:40–3.
- Chan R, Frank AC, Younis RT. Embryology and anatomy of the nose and paranasal sinuses. In: Pediatric sinusitis and sinus surgery. New York: Taylor & Francis; 2006. p. 1–14.
- Rosenfeld RM, Andes D, Bhattacharyya N, et al. Clinical practice guideline: adult sinusitis. Otolaryngol Head Neck Surg. 2007;137:S1–31.
- American Academy of Pediatrics, Subcommittee on Management of Sinusitis and Committee on Quality Improvement. Clinical management guideline: management of sinusitis. Pediatrics. 2001;108:798–808.

- 10. Aitken M, Taylor JA. Prevalence of clinical sinusitis in young children followed up by primary care pediatricians. Arch Pediatr Adolesc Med. 1998;152:244–8.
- 11. Ueda D, Yoto Y. The ten-day mark as a practical diagnostic approach for acute paranasal sinusitis in children. Pediatr Infect Dis J. 1996;15:576–9.
- Wald ER, Guerra N, Byers C. Upper respiratory tract infections in young children: duration of and frequency of complications. Pediatrics. 1991;87:129–33.
- 13. Wald ER, Milmoe GJ, Bowen A, et al. Acute maxillary sinusitis in children. N Engl J Med. 1981;304:749.
- 14. Pearce MS, Salotti JA, Little MP, McHugh K, Lee C, Kim KP, Howe NL, Ronckers CM, Rajaraman P, Sir Craft AW, Parker L, Berrington de González A. Radiation exposure from CT scans in childhood and subsequent risk of leukaemia and brain tumours: a retrospective cohort study. Lancet. 2012;380:499–505.
- Berrington de Gonzalez A, Mahesh M, Kim KP, et al. Projected cancer risks from computed tomographic scans performed in the United States in 2007. Arch Int Med. 2009; 169:2071–7.
- Hill M, Bhattacharyya N, Hall TA, Lufkin R, et al. Incidental paranasal sinus imaging abnormalities and the normal Lund score in children. Otolaryngol Head Neck Surg. 2004;130:171–5.
- 17. Berger G, Kogan T, Paker M, et al. Pediatric chronic rhinosinusitis histopathology: differences and similarities with the adult form. Otolaryngol Head Neck Surg. 2011;144(1):85–90.
- 18. Coticchia J, Zuliani G, Coleman C, et al. Biofilm surface area in the pediatric nasopharynx: chronic rhinosinusitis vs. obstructive sleep apnea. Arch Otolaryngol Head Neck Surg. 2007;133:110–4.
- Bernstein JM, Dryia D, Murphy TF. Molecular typing of paired bacterial isolates from the adenoid and lateral nasal wall of the nose of children undergoing adenoidectomy: implication in acute rhinosinusitis. Otolaryngol Head Neck Surg. 2001;125:593–7.
- 20. Furukawa CT. The role of allergy in sinusitis in children. J Allergy Clin Immunol. 1992;90:515–7.
- Phipps CD, Wood WE, Gibson WS, et al. Gastroesophageal reflux contributing chronic sinus disease in children. Arch Otolaryngol Head Neck Surg. 2000;126:831–6.
- Eggesbo HB, Sovik S, Dolvik S, et al. CT characterization of developmental variations of the paranasal sinuses in cystic fibrosis. Acta Radiol. 2001;42:482–93.
- Robertson JM, Friedman EM, Rubin BK. Nasal and sinus disease in cystic fibrosis. Paediatr Respir Rev. 2008;9(3):213–9.
- Schulte DL, Kasperbauer JL. Safety of paranasal sinus surgery in patients with cystic fibrosis. Laryngoscope. 1998;108:1813–5.
- Huang WH, Fang SY. High prevalence of antibiotic resistance in isolates from the middle meatus of children and adults with acute rhinosinusitis. Am J Rhinol. 2004;18(6):387–91.
- Brook I. Bacteriology of acute and chronic frontal sinusitis. Arch Otolaryngol Head Neck Surg. 2002;128:583–5.
- 27. Chalumeau M, Tonnelier S, D'Athis P, Tréluyer JM, Gendrel D, Bréart G, Pons G, Pediatric Fluoroquinolone Safety Study Investigators. Fluoroquinolone safety in pediatric patients: a prospective, multicenter, comparative cohort study in France. Pediatrics. 2003;111:e714.
- 28. Parsons DS. Chronic sinusitis: a medical or surgical disease? Otolaryngol Clin North Am. 1996;29:1–9.
- 29. Rosenfeld RM. Pilot study of outcomes in pediatric rhinosinusitis. Arch Otolaryngol Head Neck Surg. 1995;121:729–36.
- Clement PAR, Bluestone CD, Gordts F, et al. Management of rhinosinusitis in children. Int J Pediatr Otorhinolaryngol. 1999;49(S1):S95–100.
- Fiocchi A, Sarratud T, Bouygue GR, Ghiglioni D, Bernardo L, Terracciano L. Topical treatment of rhinosinusitis. Pediatr Allergy Immunol. 2007;18 Suppl 18:62–7.
- Brietzke SE, Brigger MT. Adenoidectomy outcomes in pediatric rhinosinusitis: a metaanalysis. Int J Pediatr Otorhinolaryngol. 2008;72(10):1541–5. doi:10.1016/j.ijporl.2008.07.008. Epub 2008 Aug 23.

- 33. Ramadan HH, Cost JL. Outcome of adenoidectomy versus adenoidectomy with maxillary sinus wash for chronic rhinosinusitis in children. Laryngoscope. 2008;118:871–3.
- 34. Makary CA, Ramadan HH. The role of sinus surgery in children. Laryngoscope. 2013;123:1348–52.
- Senior B, Wirtschafter A, Mai C, Becker C, Belenky W. Quantitative impact of pediatric sinus surgery on facial growth. Laryngoscope. 2000;110(11):1866–70.
- Hebert RL, Bent JP. Meta-analysis of outcomes of pediatric functional endoscopic sinus surgery. Laryngoscope. 1998;108:796–9.
- Haider HR, Mayatepek E, Schaper J, Vogel M. Pott's puffy tumor: a forgotten differential diagnosis of frontal swelling of the forehead. J Pediatr Surg. 2012;47:1919–21.
- Parida PK, Surianarayanan G, Ganeshan S, Saxena SK. Pott's puffy tumor in pediatric age group: a retrospective study. Int J Pediatr Otorhinolaryngol. 2012;76:1274–7.
- Blumfield E, Misra M. Pott's puffy tumor, intracranial, and orbital complications as the initial presentation of sinusitis in healthy adolescents, a case series. Emerg Radiol. 2011;18:203–10.
- Herrmann BW, Chung JC, Eisenbeis JF, Forsen Jr JW. Intracranial complications of pediatric frontal rhinosinusitis. Am J Rhinol. 2006;20:320–4.
- 41. Garcia CE, Cunningham MJ, Clary RA, Joseph MP. The etiologic role of frontal sinusitis in pediatric orbital abscesses. Am J Otolaryngol. 1993;14(6):449–52.

# **Chapter 17 Balloon Catheter Dilation of the Frontal Sinus Ostium**

#### **Michael Sillers**

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#### **Core Messages**

- The decision to operate on the frontal sinus is based on persistent symptoms that have been refractory to appropriate medical therapy with associated disease by computed tomography
- Many surgical options are available for the treatment of symptomatic, medically refractory frontal sinus disease.
- Careful consideration should be given to the patient's unique anatomy and underlying disease process in the thought process of procedure selection.
- Balloon catheter dilation of the frontal sinus outflow tract allows for sinus ostial dilation with the option to spare tissue as a stand-alone procedure.
- Balloon catheter dilation can be utilized to complement traditional instrumentation and techniques.
- Balloon catheter dilation can be performed in an office setting with safety and outcomes comparable to those in traditional operating room settings.

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# Introduction

The surgical treatment of frontal sinus disease offers a unique opportunity to significantly improve the quality of life of patients with medically refractory frontal sinus disease. While the majority of patients undergoing frontal sinus surgery improve, there is a subset of patients who demonstrate persistent or recurrent symptoms in spite of what was felt to be appropriate surgery [1]. The decision to operate on the frontal sinus outflow tract should not be made without great thought. Included in the thought process of recommending frontal sinus surgery should be an understanding of the underlying pathology (inflammatory vs. neoplastic), the unique anatomy of the frontal recess, the need for adequate exposure, the impact of the particular procedure on long term post-operative follow-up, and the unique features of a wide array of surgical instrumentation available. Innovation and technology have significantly increased the specific surgical options for medically refractory disease in the frontal sinus.

Kuhn is credited with the philosophy of applying an integrated approach to the surgical treatment of frontal sinus disease. The overriding principle is to restore frontal sinus function while preserving normal anatomic and supporting structures, such as the middle turbinate while applying techniques from less aggressive to more aggressive. This includes simply treating frontal sinus mucosal thickening felt secondary to anterior ethmoid disease by ethmoidectomy alone (Draf I) or may be as complex as employing an osteoplastic frontal sinusotomy for reversing obliteration and re-pneumatizing a previously obliterated frontal sinus [2]. For example, performing an osteoplastic frontal sinusotomy with fat obliteration for a small frontal recess inverted papilloma would make long-term endoscopic surveillance problematic versus a less aggressive, more physiologic Draf IIB procedure. While a modified Lothrop procedure arguably creates the largest opening possible for the frontal sinus via an endoscopic approach, perhaps simply dilating and displacing the medial and superior walls of a Type III frontal cell with a balloon and then removal of bone fragments with a giraffe forceps or an angled microdebrider blade would suffice as treatment for inflammatory disease. As rhinologic surgeons we should strive to avoid a "bigger is better" mentality and remember the "functional" aspect of "functional endoscopic sinus surgery" (FESS). A surgeon's inability to perform a less invasive, more physiologic procedure is not a reason to perform a more aggressive, less physiologic operation [2].

The purpose of this chapter is to discuss the indications, relative contraindications and techniques for using balloon catheter dilation (BCD) of the frontal sinus outflow tract. It is important to state at the onset that the decision to intervene surgically is not instrument or site of service dependent.

• Before consideration for surgery, patients should have failed appropriate medical therapy, have persistent symptoms, and demonstrate pathology on computed tomography (CT).

If these basic criteria are not met, sinus surgery, regardless of the instrumentation available, should generally not be considered.

In general BCD offers the unique opportunity to achieve durable sinus ostial and outflow tract dilation while sparing tissue in the process [3–8]. Specifically for the frontal sinus outflow tract this technology allows for fracturing and lateral displacement of the medial and superior wall of obstructing frontal cells, medial displacement of an obstructing intersinus septal cell wall, and/or dilating soft tissue stenosis in previously operated patients.

Relative contraindications for balloon catheter dilation as a stand-alone procedure include:

- Cases where the underlying histology is in question
- Dense neo-osteogenesis of the frontal sinus outflow tract where sufficient displacement of bony walls is unlikely
- · Extensive polyposis

In these cases traditional instrumentation should be utilized to either complement BCD technology or in its stead.

An additional consideration for BCD is the ability to utilize this technology in the office setting. This has the obvious advantages of the elimination of the risks and recovery of general anesthesia and avoidance of cost associated with it and with the hospital outpatient department or ambulatory surgical facility [9]. In-office balloon catheter dilation has been shown to be safe and well tolerated with outcomes similar to those achieved in traditional venues [10–12].

### Devices

There are currently two manufacturers of FDA approved devices for balloon catheter dilation of the frontal, maxillary and sphenoid sinuses. Acclarent, Inc.<sup>TM</sup> and Entellus<sup>TM</sup> each have devices specifically designed for some of the unique features of frontal sinus outflow tract anatomy. Path Assist<sup>TM</sup> (Fig. 17.1) and Scout<sup>TM</sup> (Fig. 17.2) are designed to mimic a frontal seeker and are quite helpful in patients who have undergone prior surgery. Having the ability to change the trajectory of the tip of the wire is essential to proper ostial cannulation and subsequent balloon advancement and dilation of the frontal sinus outflow tract. Spin<sup>TM</sup> (Fig. 17.3) has the unique feature of a guide wire that the surgeon can literally "spin" and change the wire trajectory which is quite helpful when multiple ostia are present in the frontal recess. Express<sup>TM</sup> (Fig. 17.4) has a malleable tip feature that can be very helpful in providing the proper angle for wire and balloon advancement and subsequent dilation.



Fig. 17.1 Path Assist<sup>TM</sup>, Entellus



Fig. 17.2 Scout<sup>TM</sup>, Acclarent, Inc

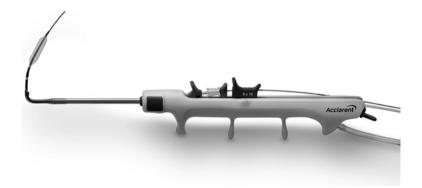


Fig. 17.3 Spin<sup>™</sup>, Acclarent



Fig. 17.4 Express<sup>™</sup>, Entellus

### Techniques

Techniques for successful balloon catheter advancement and frontal sinus outflow tract dilation vary depending on the patient' unique anatomy and or history of prior surgery.

• In general when dilating multiple sinuses, dilating the sphenoid sinus, frontal sinus and finally the maxillary sinus is preferred in that order

Balloon catheter dilation has been shown to be safe and tolerable in a traditional operating room setting with general anesthesia as well as in the office under topical anesthesia.

Successful and satisfying outcomes in the office are directly related to adequate anesthesia that requires patience on the part of the surgeon and patient alike. Combinations of 4 % lidocaine or 2 % tetracaine coupled with topical oxymetazoline or adrenalin on cotton pledgets have been described. Local infiltrative anesthesia may also be utilized. Staining 1:1,000 topical adrenalin with methylene blue or fluorescein will help minimize the catastrophic risk of inadvertent injection of concentrated adrenalin. Specifically for the frontal sinus placement of cotton pledgets both medial and lateral to the middle turbinate is helpful and allows for gentle medial displacement of the middle turbinate once anesthesia is achieved. Adequate anesthesia is often achieved when the patient reports dental numbness which generally occurs 10–15 min following proper placement of the anesthetic.

Beginning with a 0° endoscope the patient's nasal cavity is examined and the middle turbinate is gently medialized with a Cottle or Freer elevator. The frontal recess is inspected with a 30 or 45° angled endoscope (Fig. 17.5). In the previously operated patient a frontal sinus ostium seeker or the Path Assist<sup>TM</sup> with lighted tip can be used to palpate a stenosed outflow tract or gently break through a soft tissue scar. The added benefit of light assistance is transillumination the frontal sinus once Path Assist<sup>TM</sup> or Scout<sup>TM</sup> is properly advanced (Fig. 17.6). At this point the guide wire is advanced into the frontal sinus, once again using transillumination to confirm



Fig. 17.5 Previously operated frontal recess with symptomatic stenosis

**Fig. 17.6** Scout<sup>TM</sup> advanced into the frontal recess



**Fig. 17.7** Guide wire advancement into the frontal sinus



its location Figs. 17.7 and 17.8). The appropriate diameter and length balloon is advanced and dilated to 10–12 atm of pressure (Fig. 17.9).

• In the awake patient balloon dilation is the moment that intense pain may be reported. Communicating this in advance is essential.

Balloon inflation generally lasts a few seconds and patients have reported this as tolerable in the overwhelming majority of instances.

• In patients with simple narrowing or soft tissue stenosis a single dilation may be sufficient. However, in cases with a more lengthy obstruction, such as a Type III frontal cell medial wall, two or more dilations may be necessary to adequately re-establish a patent frontal sinus outflow tract.

Following dilation (Fig. 17.10), irrigation can be performed through a catheter advanced over the guide wire (Fig. 17.11). This can be a challenge in the office setting simply because of the volume of fluid necessary to wash and/or collect secretions for microbiologic studies (Fig. 17.12). The surgeon, staff and patient should be adequately gowned and provided with eye protection. Once the catheter is in place,

**Fig. 17.8** Transillumination of the left frontal sinus. Navigation tracking wire is in the midline

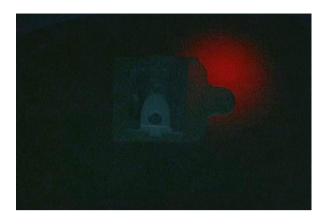


Fig. 17.9 Balloon dilation



**Fig. 17.10** Endoscopic view of the frontal sinus after dilation

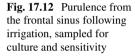


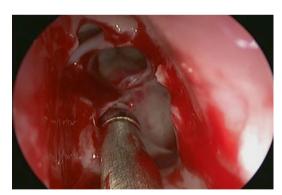
the patient leans forward, tucks their chin, and holds their breath during irrigation. A basin is placed under the chin and mouth to collect the run-off. At this point the adequacy of the dilation is assessed and the sinusotomy may be enlarged with traditional instrumentation (Figs. 17.13 and 17.14). Upon completion of the procedure, the patient is observed for bleeding and discharged. Packing is rarely required.

**Fig. 17.11** Irrigation catheter in the frontal sinus









**Fig. 17.13** Frontal sinus mushroom punch utilized to enlarge the frontal sinus ostium

# Conclusion

Balloon catheter dilatation represents a new technology that complements existing surgical instrumentation for the treatment of medically refractory chronic rhinosinusitis. As with any surgical procedure appropriate selection of technique, instrumentation, and even site of service is guided by the disease process, the extent of disease, and the impact on post-operative follow-up. Rhinologic surgeons should

**Fig. 17.14** Endoscopic view at the completion of the procedure



carefully appraise new technology and be willing to evolve thought processes in the treatment of an overwhelmingly benign disease process that may have a significant impact on patients' quality of life.

### References

- Naidoo Y, Keen M, Bassiouni A, Wormald P-J. Risk factors for outcomes for primary, revision, and modified Lothrop (Draf III) frontal sinus surgery. Int Forum Allergy Rhinol. 2013;3:412–7.
- Kuhn F. An integrated approach to frontal sinus surgery. Otolaryngol Clin N Am. 2006;39(3):437–61.
- 3. Bolger W, Brown C, Church C, et al. Safety and outcomes of balloon catheter sinusotomy: a multi-center 24 week analysis in 115 patients. Otolaryngol Head Neck Surg. 2007;137:10–20.
- Levine H, Sertich II A, Hoisington D, et al. A multicenter registry of balloon catheter sinusotomy outcomes for 1036 patients. Ann Otol Rhinol Laryngol. 2008;117:263–70.
- Weiss R, Church C, Kuhn F, et al. Long-term outcomes analysis of balloon catheter sinusotomy: two year follow-up. Otolaryngol Head Neck Surg. 2008;139:S38–46.
- Stankiewicz J, Truitt T, Atkins J. One-year results: transantral balloon dilation of the ethmoid infundibulum. Ear Nose Throat. 2010;89:72–7.
- Stankiewicz J, Truitt T, Winegar B, et al. Two year results: antral dilation of the ethmoid infundibulum. Int Forum Allergy Rhinol. 2012;2(3):199–206.
- Kutluhan A, Bozdemir K, Cetin H, et al. Endoscopic balloon dilation sinuplasty including ethmoidal air cells in chronic rhinosinusitis. Ann Otol Rhinol Laryngol. 2009;118(12):881–6.
- Prickett K, Wise S, DelGaudio J. Cost analysis of office-based and operating room procedures in rhinology. Int Forum Allergy Rhinol. 2012;2:207–11.
- Albritton F, Casiano R, Sillers M. Feasibility of in-office endoscopic sinus surgery with balloon sinus dilation. Am J Rhinol Allergy. 2012;26(3):243–8.
- 11. Karanfilov B, Silvers S, Pasha R, et al. Office-based balloon sinus dilation: a prospective multi-center study of 203 patients. Int Forum Allergy Rhinol. 2013;3:404–11.
- 12. Cutler J, Truitt T, Atkins J, et al. First clinic experience: patient selection and outcomes for ostial dilation for chronic rhinosinusitis. Int Forum Allergy Rhinol. 2011;1(6):460–5.

## **Chapter 18 Balloon Catheter Sinuplasty for Children** with Chronic Rhinosinusitis

Andrew Terrell and Hassan H. Ramadan

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Electronic supplementary material The online version of this chapter

(doi:10.1007/978-3-662-48523-1\_18) contains supplementary material, which is available to authorized users.

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_18

#### **Core Messages**

- Several surgical procedures are available for treatment of chronic rhinosinusitis in children
- Diagnosis can be difficult to distinguish from chronic adenoiditis
- Majority of children respond to medical treatment, surgery should be a last resort
- Coronal CT scan is the imaging modality of choice in children with chronic rhinosinusitis.
- Pathophysiology in these children is mainly blockage of the ostiomeatal complex area.
- · Balloon dilation of sinus ostia is a surgical option for these children

## Introduction

Pediatric chronic rhinosinusitis remains a common problem frequently encountered by the otolaryngologist. It can be a source of frustration and loss of work to parents. Quality of life studies have shown that children with chronic rhinosinusitis have more limitations in their activities and more bodily pain compared to children with other chronic diseases such as asthma, ADHD, and arthritis [1]. The practitioner should be comfortable with the diagnosis and management of chronic rhinosinusitis in children and have surgical options which have been shown to improve the quality of life of these patients.

Balloon catheter technology has provided improved treatment options in many areas of medicine including cardiac, vascular, and urologic conditions. More recently, balloons have been introduced for the treatment of chronic rhinosinusitis (CRS). Balloon catheter sinuplasty (BCS) has become an effective treatment of CRS in adults [2–5]. Results from adult studies demonstrate an excellent safety profile with a major complication rate of 0.0035 % per sinus or 0.01 % per patient [6]. It has also been shown that BCS can also be performed safely and effectively in children [7] (Table 18.1).

Surgery for children with CRS continues to evolve. Adenoidectomy is currently the mainstay of treatment for pediatric CRS, but is effective in a little over 50 % of cases [8, 9]. It has even less success in asthmatics and children 7 years old and younger. Also, 25 % of children undergoing adenoidectomy may need revision surgery [9]. Endoscopic sinus surgery (ESS) has been shown to be more effective than adenoidectomy alone [9–11], although revisions may still be required in 12 % of children [9]. The complication rate is low, but serious complications can occur including hemorrhage, meningitis,

2009- Sa	afety and feasibility study in children
2010-В	CS had success in children prior to sinus surgery
2011- B	CS was advantageous to adenoidectomy alone
2011- B	CS after adenoidectomy failure had similar outcome
to sinus	surgery
2012- B	CS was as successful to ESS in children with CRS

 Table 18.1
 Development of balloon catheter sinuplasty in children

and orbital complications [11]. BCS can become a tool in the surgeon's armamentarium which can increase the effectiveness of adenoidectomy, while avoiding some of the risks that are inherent to traditional endoscopic sinus surgery.

## **Indications and Patient Selection**

The vast majority of children with sinus symptoms do not require surgical intervention. This point cannot be overemphasized. Children suffer frequent upper respiratory infections, up to six to eight per year with 5–13 % of infections that can become secondarily infected by bacteria [12]. Parents should understand that this is normal. Abnormal CT scans can also be quite common in children. One study of scans in children taken for non-sinusitis reasons, reports a mean Lund-MacKay score of 2.8, with only 19 % of children having a completely normal scan [13]. Thus CT scans should be interpreted in the clinical context and a surgical recommendation should not be made based on the CT scan only (Table 18.2).

## Medical Management

Every patient should have a trial of maximal medical therapy over a 3–6 month period prior to consideration for surgery to include:

- Oral and sometimes i.v. antibiotics
- Nasal steroids
- Oral decongestants
- Systemic steroids
- Allergy management

## Work Up

Children should undergo the following work up:

- Allergy testing
- Asthma evaluation

Children who failed medical treatment and are considered for surgery Children who require a culture from maxillary sinus Children who are having an adenoidectomy/had an adenoidectomy Children who had CT scan with evidence of maxillary sinus disease and blockage of OMC

Table 18.2 Indications

- Immunoglobulin deficiency work up
- Sweat chloride test if necessary

## **Diagnostic Testing**

The mainstay of diagnostic testing is:

- CT scan to be performed at the end of 3 week course of appropriate antibiotic
- Rigid endoscopy if possible specifically in older child
- CT staging according to the Lund-Mackay system. As stated above
- Abnormal CT scans are common
- A score of 5 has been reported to be indicative of true disease [14].

#### Contraindications to Balloon Catheter Sinuplasty in Children

Children should not be considered for BCS if they have any of the following (Table 18.3):

- Had previous sinonasal surgery in target ostia
- Cystic fibrosis
- Extensive sinonasal osteoneogenesis
- Sinonasal tumors or obstructive lesions
- History of facial trauma that distorts sinus anatomy and precludes access to the sinus ostium
- Ciliary dysfunction
- Hypoplastic sinus as these have been shown to be more difficult to cannulate [7] (Figs. 18.1 and 18.2)
- It is also helpful to have experience with balloons in adults prior to working on children.

Children with no evidence of sinusitis on CT scan
Children with cystic fibrosis
Children with fungal disease
Children with cyst/polyp in sinus that requires removal
Children with hypoplastic/atelectatic maxillary sinus
Children who had prior sinus surgery
- Children with anatomical obstruction preventing visualization of OMC

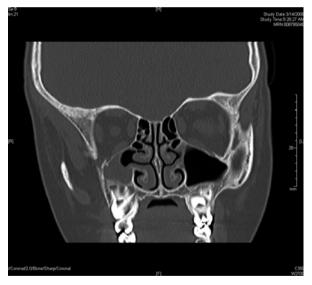
Table 18.3 Contraindications



**Fig. 18.2** Hypoplastic maxillary sinus. The retracted uncinate process makes sinus cannulation difficult

**Fig. 18.1** Hypoplastic maxillary sinus. The retracted uncinate process makes sinus cannulation

difficult



## **Surgical Technique**

## Equipment

Standard endoscopic sinus equipment should be available including:

- 4 mm scopes and 2.7 mm pediatric scopes
- Digital camera and tower

- Standard endoscopic sinus instruments
- Balloons, 5 mm Acclarent balloon or 6 mm Entellus balloon
- Topical and systemic decongestants
- Recent CT scan of sinuses

Cyclops (Acclarent, Menlo Park, California) can be useful to improve visualization around the uncinate and confirm dilation of the natural ostia. Drawbacks of the multi-angled scope is that the picture quality tends to be darker, the scope is heavier, and seems slightly wider which can make a significant difference in small noses.

Balloons are available from different suppliers and the surgeon should use a system with which he or she is comfortable. Early studies were performed using delivery systems that required confirmation of the balloon placement with fluoroscopy. Transillumination guide wires have obviated the need for fluoroscopy with associated radiation exposure. The one exception is the sphenoid sinus where transillumination is not useful. Balloons are available in different sizes. For pediatric cases a 5 mm balloon is typically sufficient although 7 mm balloons should be available and may be useful in older children.

• As with any operation it is imperative that the surgical staff is familiar with and educated regarding the surgical equipment.

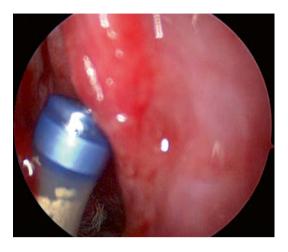
In particular, the scrub nurse should be comfortable with the balloon delivery system as he or she may need to help with advancement of the guide wire and insufflation of the balloon. Having the guide catheter in the right place and not being able to have the wire advanced properly or not having the balloon insufflate can be a source of surgical consternation.

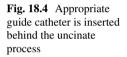
#### Technique

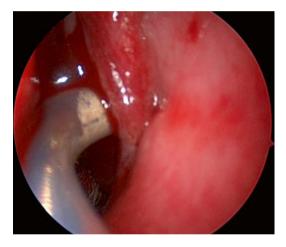
#### Acclarent Balloon

- Procedure performed under general anesthesia
- Patient placed in the supine position
- 4 mm endoscope is used for visualization
- The nose is decongested with oxymetazoline-moistened pledgets, placed in the nasal cavity as well as the middle meatus
- Injection of the middle turbinate and uncinate process with 1 % lidocaine with 1:100,000 epinephrine
- Appropriate guide catheter is inserted behind the uncinate process (Figs. 18.3 and 18.4)
- 110S guide for maxillary sinus dilation, 70 guide for frontal sinus and 0 for sphenoid
- Guide wire is passed through the catheter into the intended sinus
- Confirmation that the guide wire is in the sinus with transillumination
- The sinus balloon (5 mm balloon) is passed over the guide

**Fig. 18.3** Appropriate guide catheter is inserted behind the uncinate process





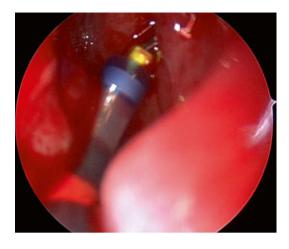


- Use markers on balloon to ensure proper placement (Figs. 18.5 and 18.6)
- Inflate the balloon to 12 atmospheric pressure (Fig. 18.7)
- Irrigation/wash of sinus
- Remove balloon and wire, then remove guide
- Confirmation dilation of ostium with direct visualization
- Merogel packing (Medtronic, Jacksonville, Florida) is typically sufficient (Video 1).

#### **Entellus Balloon**

The setup is exactly the same as the Acclarent balloon. The procedure is somewhat similar, except that the entellus apparatus is all one piece. The tip of the apparatus is similar to a seeker. Usually the tip is fashioned depending which sinus is being dilated. Afterwards the seeker tip is introduced behind the uncinate process and

**Fig. 18.5** Markers on balloon (*yellow*) ensure proper placement



**Fig. 18.6** Markers on balloon (*yellow*) ensure proper placement





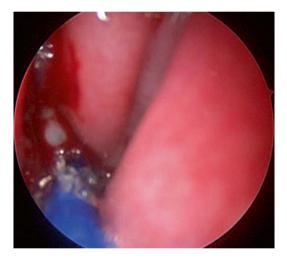
**Fig. 18.7** The balloon is inflated to 12 atmospheric pressure



seeker tip is positioned behind the uncinate process and then introduced into the sinus cavity

Fig. 18.8 The balloon

**Fig. 18.9** The balloon is introduced into the sinus under direct visualization



introduced into the sinus (Fig. 18.8). Confirmation can be done using transillumination. Once confirmed, the balloon is then introduced into the sinus under direct visualization (Fig. 18.9). It is then inflated using the syringe attached to the device (Fig. 18.10). Similarly the balloon is inflated at two different positions. We use the 6 mm balloon for all children. Confirmation of dilation is then done. Packing, if needed, is inserted as above (Video 2) (Table 18.4).

## Pitfalls, Tips, Advantages and Disadvantages

- Creating an accessory ostium
- Stripping of the mucosa posteriorly, thus collapsing the whole lining anteriorly

**Fig. 18.10** The balloon is inflated using the syringe attached to the Entellus device



Table 18.4 Advantages

No tissue removal
Minimal manipulation of sinus
Reduced pain and bleeding postoperatively
No need for packing

<b>Table 18.5</b>	Disadvantages
-------------------	---------------

Risk of creating a false (accessory) ostium Excessive trauma to the area with resultant scarring Use of inappropriate size balloon resulting in either trauma or inadequate dilation

- Significant injury to ostium and uncinate to the extent that an antrostomy may be needed
- Guide catheter is introduced tip up (personal preference for children)
- Hold tension on balloon while inflated to prevent inflated balloon from slipping inside/outside the sinus (Table 18.5)

## **Postoperative Care**

- Child discharged after a period of observation
- Oral antibiotics are given for 7 days postoperatively
- Avoid use of topical sprays for 7 days
- Avoid nose blowing
- Follow up in 2 weeks

#### **Results and Complications**

Outcomes in children's sinus literature are often based on 12 month SN-5 scores compared to the preoperative SN-5 score. The SN-5 is a quality of life questionnaire which has been shown to correlate with CT scans [15]. As described in Kay and Rosenfeld's original paper [16], a decrease of 0.5-0.9 is a mild change, 1.0-1.4 is a moderate change and >1.5 is a large change. Any child with a decrease in SN-5 score greater than or equal to 0.5 is considered a success. Scores with a decrease of <0.5 are considered failures. Any child with a worse score and any child needing revision surgery is considered a surgical failure.

In a study of 49 children, 30 had both BCS and adenoidectomy, while 19 underwent adenoidectomy alone. Twenty-four (80 %) of the children who underwent BCS had improvement of their symptoms compared to ten (52.6 %) of those who had an adenoidectomy alone (p=0.04) Two of the patients (6 %) in the BCS group failed and subsequently required ESS. Three children (15 %) in the adenoidectomy group that failed went on to have BCS [17].

The mean change in SN-5 scores for all 49 children improved from 4.1 preoperatively to 2.9 postoperatively (p < 0.0001). The mean change for SN-5 scores in children undergoing BCS improved from 4.2 preoperatively to 3.0 postoperatively (p < 0.0001). For the adenoidectomy group the preoperative SN-5 score improved from 3.8 to 2.9 postoperatively (p < 0.01) [17].

Balloon catheter sinuplasty can also be used in patients who fail adenoidectomy, as demonstrated in a separate study involving 26 children. The mean (SD) preoperative SN-5 score was 4.6 (0.9). The mean (SD) postoperative score was 3.0 (1.2), for a mean difference of 1.6 (P<.001). A decrease of at least 0.5 on the postoperative score was considered a surgical success. Twenty-one children were treated successfully with balloon dilation (81 %). The five children involved in the surgical failure cases did not require more surgery [18].

In a study from Michigan [19], 15 patients who underwent BCS with ethmoidectomy were compared to 16 patients who underwent traditional sinus surgery. At an average 37 week post-op, 62.5 % of those who underwent traditional ESS and 80.0 % of those who had BCS with ethmoidectomy had improvement in symptom scores. Antibiotic usage decreased in 73 % of the patients who underwent BCS as opposed to 38 % of patients who had ESS [19].

#### **Complications**

- Missing the natural ostium
- Nose bleed (minor)
- Orbital/CNS injury (no reports)
- Trauma to ostium with secondary scarring/granuloma formation

## Conclusion

While the majority of children with chronic rhinosinusitis can be controlled with proper medical management, a subset may be refractory to these efforts. These children can benefit from proper and judicious surgical intervention. As adenoidectomy is only effective in around 50 % of children, balloon catheter sinuplasty can be used to improve outcomes. Care and attention should focus on proper instrumentation and visualization to ensure dilation of the natural ostia. When used properly it has an exceptional safety profile. As such, we have found it to be a useful step in the surgical algorithm prior to traditional endoscopic sinus surgery.

#### References

- 1. Cunningham JM, Chiu EJ, Landgraf JM, Gliklich RE. The health impact of chronic recurrent rhinosinusitis in children. Arch Otolaryngol Head Neck Surg. 2000;126:1363–8.
- Bolger WE, Brown CL, Church CA, et al. Safety and outcomes of balloon catheter sinusotomy: a multicenter 24-week analysis in 115 patients. Otolaryngol Head Neck Surg. 2007;137:10–20.
- Kuhn FA, Church CA, Goldberg AN, et al. Balloon catheter sinusotomy: one-year follow-up outcomes and role in functional endoscopic sinus surgery. Otolaryngol Head Neck Surg. 2008;139:S27–37.
- Levine HL, Sertich 2nd AP, Hoisington DR, Weiss RL, Pritikin J. Multicenter registry of balloon catheter sinusotomy outcomes for 1,036 patients. Ann Otol Rhinol Laryngol. 2008;117:263–70.
- Weiss RL, Church CA, Kuhn FA, Levine HL, Sillers MJ, Vaughan WC. Long-term outcome analysis of balloon catheter sinusotomy: two-year follow-up. Otolaryngol Head Neck Surg. 2008;139:S38–46.
- Melroy CT. The balloon dilating catheter as an instrument in sinus surgery. Otolaryngol Head Neck Surg. 2008;139:S23–6.
- Ramadan HH. Safety and feasibility of balloon sinuplasty for treatment of chronic rhinosinusitis in children. Ann Otol Rhinol Laryngol. 2009;118:161–5.
- 8. Vandenberg SJ, Heatley DG. Efficacy of adenoidectomy in relieving symptoms of chronic sinusitis in children. Arch Otolaryngol Head Neck Surg. 1997;123:675–8.
- 9. Ramadan HH. Surgical management of chronic sinusitis in children. Laryngoscope. 2004;114:2103–9.
- Chang PH, Lee LA, Huang CC, Lai CH, Lee TJ. Functional endoscopic sinus surgery in children using a limited approach. Arch Otolaryngol Head Neck Surg. 2004;130:1033–6.
- Hebert 2nd RL, Bent 3rd JP. Meta-analysis of outcomes of pediatric functional endoscopic sinus surgery. Laryngoscope. 1998;108:796–9.
- 12. Choi SH, Han MY, Ahn YM, et al. Predisposing factors associated with chronic and recurrent rhinosinusitis in childhood. Allergy Asthma Immunol Res. 2012;4:80–4.
- Hill M, Bhattacharyya N, Hall TR, Lufkin R, Shapiro NL. Incidental paranasal sinus imaging abnormalities and the normal Lund score in children. Otolaryngol Head Neck Surg. 2004;130:171–5.
- Bhattacharyya N, Jones DT, Hill M, Shapiro NL. The diagnostic accuracy of computed tomography in pediatric chronic rhinosinusitis. Arch Otolaryngol Head Neck Surg. 2004;130:1029–32.
- 15. Terrell AM, Ramadan HH. Correlation between SN-5 and computed tomography in children with chronic rhinosinusitis. Laryngoscope. 2009;119:1394–8.

- Kay DJ, Rosenfeld RM. Quality of life for children with persistent sinonasal symptoms. Otolaryngol Head Neck Surg. 2003;128:17–26.
- Ramadan HH, Terrell AM. Balloon catheter sinuplasty and adenoidectomy in children with chronic rhinosinusitis. Ann Otol Rhinol Laryngol. 2010;119:578–82.
- Ramadan HH, Bueller H, Hester ST, Terrell AM. Sinus balloon catheter dilation after adenoidectomy failure for children with chronic rhinosinusitis. Arch Otolaryngol Head Neck Surg. 2012;138:635–7.
- Thottam PJ, Haupert M, Saraiya S, Dworkin J, Sirigiri R, Belenky WM. Functional endoscopic sinus surgery (FESS) alone versus balloon catheter sinuplasty (BCS) and ethmoidectomy: a comparative outcome analysis in pediatric chronic rhinosinusitis. Int J Pediatr Otorhinolaryngol. 2012;76:1355–60.

# Chapter 19 Primary Endoscopic Surgery

David W. Jang and Stilianos E. Kountakis

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**Electronic supplementary material** The online version of this chapter (doi:10.1007/978-3-662-48523-1\_19) contains supplementary material, which is available to authorized users.

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_19

#### **Core Messages**

- Primary endoscopic frontal sinusotomy focuses on a thorough dissection of frontal recess cells with minimal manipulation of the frontal ostium.
- Mucosal preservation during surgery is imperative in preventing postoperative stenosis.
- Understanding frontal recess anatomy requires having a three-dimensional understanding of the uncinate process and the various types of frontal recess cells
- The surgeon must formulate a stepwise plan based on careful analysis of preoperative CT images.
- Careful postoperative debridement and long-term follow up are necessary.

## Introduction

The advent of endonasal endoscopic frontal sinusotomy as the standard of care for chronic frontal sinusitis can be attributed to three major factors [1]:

- 1. High resolution video endoscopy
- 2. Development of specialized instrumentation
- 3. Better understanding of frontal sinus pathophysiology

Reports on long-term patency rates have also been published in the last 20 years, showing that the success rate for endoscopic frontal sinusotomy is comparable to the open procedures that were once commonly performed. However, endoscopic frontal sinusotomy continues to be technically challenging for many surgeons due to the complex anatomy and the relatively high re-stenosis rates.

The types of endonasal frontal sinusotomy have been classified by Draf as seen in Table 19.1 [2]. The Draf Type 1 frontal sinusotomy, which can be used

Type	Extent of surgery
Ι	Anterior ethmoidectomy with drainage of the frontal recess without touching the frontal sinus outflow tract
IIa	Removal of ethmoidal cells protruding into the frontal sinus creating an opening between the middle turbinate medially and the lamina papyracea laterally
IIb	Removal of the frontal sinus floor between the nasal septum medially and the lamina papyracea laterally
III	Type II drainage on both sides with removal of the superior nasal septum and lower portion of intersinus septum

Table 19.1 Endonasal frontal sinus drainage types I-III according to Draf

Adopted from Weber et al. [1]

interchangeably with "frontal recess surgery", consists of dissection of the frontal recess without manipulation of the frontal sinus ostium itself. This is often the initial procedure in the surgical management algorithm for frontal sinusitis. The Draf Type 2a and 2b procedures may be performed as primary surgery in cases of mucoceles or serious complications of acute sinusitis. The Draf 3, or the endoscopic modified Lothrop procedure, is typically reserved for patients who are undergoing revision surgery or endoscopic resection of anterior cranial base tumors [3].

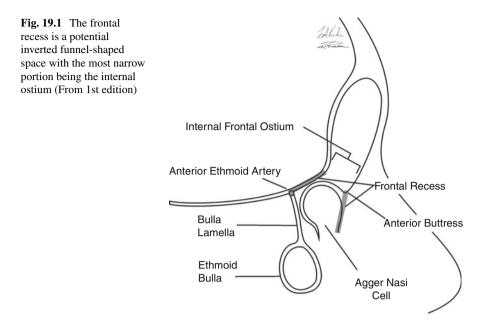
#### Outcomes

The literature regarding outcomes for endoscopic frontal sinus surgery usually includes all types of frontal sinusotomy, without distinguishing whether surgery was limited to the frontal recess or not. In addition, data for primary and revision surgeries are often presented together. Follow-up time and sample size vary widely, and patency rates range from 68 % to 100 % [1, 4]. One of the first large outcomes studies was by Wigand and Hosemann in 1991, who reported that more than twothirds of their patients had patent frontal ostia via visualization or probing after a mean follow-up of 3.5 years [5]. More recent studies have shown better long-term results. A retrospective review by Friedman et al. reported long-term results for 152 patients (255 frontal sinuses) undergoing frontal recess dissection, and found a 67.6 % and 71.1 % patency rates after initial and revision surgery, respectively, and a 78.3 % rate of significant symptom improvement at a mean follow-up of 72 months [6]. Similarly, Chan et al. reported an overall patency rate of 88 % in 161 patients (294 frontal sinuses), 42 of which were primary cases, at a mean follow-up of 45.9 months [7]. Naidoo et al. reported their results for primary Draf 2a frontal sinusotomy in 109 patients (210 frontal sinuses). Overall patency rate was 92 % with complete symptom resolution in 78 % [8].

One can conclude from this data that endoscopic frontal sinusotomy is an effective procedure with acceptable long-term results. However, it is clear that long-term follow-up in these patients is necessary, as re-stenosis and recurrence of symptoms can occur after several years.

There is little consensus on predictive factors for frontal re-stenosis, but the following have all been implicated [4, 7, 8]:

- Frontal ostium diameter
- Smoking
- Asthma
- Radiologic severity
- Number of prior surgeries
- Tissue eosinophilia
- Aspirin-sensitivity



#### **Frontal Recess Anatomy**

A thorough understanding of three-dimensional frontal recess anatomy is necessary in order to perform effective endoscopic surgery in this area. A key concept is that the frontal sinus ostium opens into an inverted funnel-shaped space called the frontal recess, rather than a "nasofrontal duct "as the older literature states [9] (Fig. 19.1).

The boundaries of the frontal recess are:

- · The skull base superiorly
- Middle turbinate medially
- Lamina papyracea laterally
- · Nasofrontal beak anteriorly
- The ethmoid bulla posteriorly.

Each of these boundaries needs to be clearly identified during dissection of the frontal recess. Because the frontal recess is technically part of the anterior ethmoid cavity, it contains many variations of ethmoid septations that determine the configuration of the frontal outflow tract into the middle meatus. Despite the fact that the anatomy is so highly variable, several authors have been able to describe frontal recess anatomy in a way that is useful and practical to the surgeon.

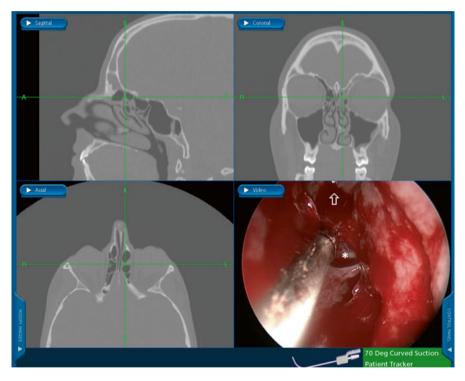
Based on the work of Van Aylea in the 1930s and 1940s, Kuhn described the different types of frontal recess cells as shown in Table 19.2 [10, 11]. The agger nasi cell (ANC), which refers to a single ethmoid cell anterior to the frontal outflow tract, is arguably the most prevalent frontal recess cell, and has been shown to correlate with frontal sinus obstruction [12, 13]. Cells that are found above the agger nasi are referred to as frontal cells (FC) types I-III. Any cell found completely within the

Table 19.2 Frontal recess cells as described by Kuhn

1.	Agger nasi cell
2.	Supraorbital ethmoid cells
3.	Frontal Cells
	(a) Type I
	(b) Type II
	(c) Type III
	(d) Type IV
4.	Frontal bulla cells
5.	Suprabullar cells

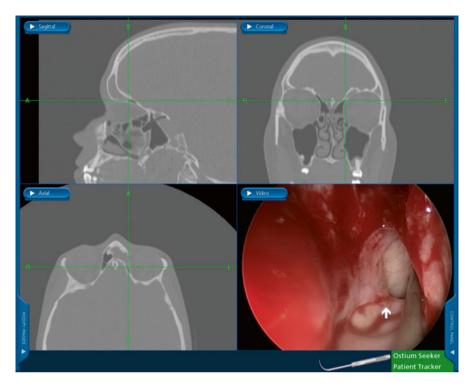
6. Interfrontal sinus septal cells

From Karanfilov and Kuhn [9]



**Fig. 19.2** View of left frontal recess with 45-degree endoscope showing agger nasi cell (*arrow*) and suprabullar cell (*asterisk*). Tip of suction navigator is placed at the location of the frontal outflow tract, which is obstructed by both cells

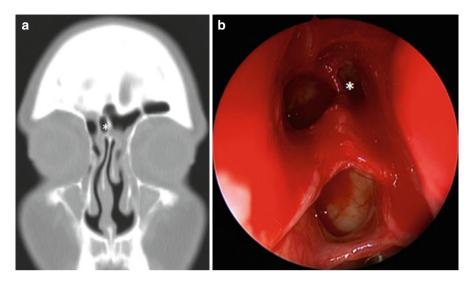
sinus itself is referred to as a type IV frontal cell. Posterior to the frontal outflow tract, virtually all patients will have an ethmoid bulla, which forms the posterior border of the frontal recess. However, cells above the bulla—suprabullar cell (SBC) or frontal bulla cell (FBC)—can extend into the frontal recess and cause obstruction of the frontal outflow tract. Figure 19.2 demonstrates obstruction of the frontal outflow tract by an ANC and SBC.



**Fig. 19.3** View of left frontal recess with 45-degree endoscope. Tip of ostium seeker is inside a supraorbital cell. The anterior ethmoidal artery (*arrow*) is located at the posterior margin of the SOEC opening. The frontal outflow tract is immediately anterior and medial to the SOEC

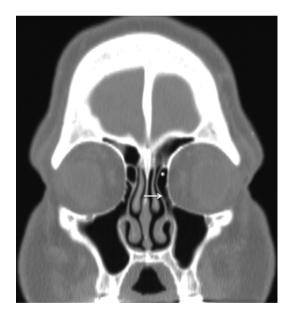
In some instances, a supraorbital ethmoid cell (SOEC) can form from pneumatization of the orbital plate of the frontal bone. Because the opening of the SOEC is directly adjacent to the frontal ostium, it can displace or narrow the frontal sinus ostium. More often, the SOEC can serve as a useful landmark for the anterior ethmoidal artery (AEA), since the posterior margin of the SOEC opening contains the AEA in the vast majority of cases [14] (Fig. 19.3). The interfrontal sinus septal cell (IFSSC) forms from pneumatization of the frontal sinus septum, and is located medial to the frontal sinuses. Despite its central location, it typically drains unilaterally (Fig. 19.4). Both SOECs and IFSSCs can easily be mistaken for the frontal sinus ostium. While the incidence of each of these frontal recess cells varies, they all have the potential to cause outflow tract obstruction and therefore need to be recognized [15].

Another key anatomic concept is recognizing the superior attachment of the uncinate process, which can guide dissection in the frontal recess [16]. The uncinate process will course laterally and attach to the lamina papyracea in the majority of cases [17]. In this situation, the frontal sinus will drain into the middle meatus *medial* to the uncinate process. The space formed by the junction of the uncinate process and the lamina papyracea is called the recessus terminalis, and can be

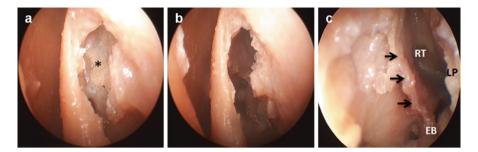


**Fig. 19.4** Coronal CT scan and 45-degree endoscopic view of interfrontal sinus septal cell (*aster-isk*) that opens into the right frontal recess. Frontal sinus ostium is seen adjacent to this

**Fig. 19.5** Coronal CT scan showing uncinate process (*arrow*) attaching to lamina papyracea. Recessus terminalis is indicated by the *asterisk* 



mistaken for the frontal recess (Fig. 19.5). In remaining cases, the uncinate process will attach to the skull base or the middle turbinate, producing a drainage pathway *lateral* to the uncinate process. The superior attachment in these cases must be carefully handled as to prevent fracture of the lateral lamella or fovea ethmoidalis. More recently, Wormald has suggested that understanding the anatomy of the frontal



**Fig. 19.6** Cadaveric dissection of left frontal recess. (a) Anterior aspect of the agger nasi cell is removed revealing the posterior wall of the agger nasi (*asterisk*) which is continuous with the uncinate process. (b) Posterior wall of the agger nasi (*anterior aspect of uncinate*) is removed. (c) 45-degree endoscopic view of the cut edge of the uncinate process (*arrows*). The uncinate attaches to the lamina papyracea (*LP*) laterally, forming the recessus terminalis (*RT*). Posterior to the uncinate, the lamella of the ethmoid bulla (*EB*) can be seen

recess is dependent on recognizing the close relationship between the uncinate process and the ANC. In this model, the pneumatization pattern of the ANC and FCs will determine the orientation and attachment site of the uncinate process and as a result, the location of the frontal ostium [18]. Figure 19.6 demonstrates the relationship of the ANC and the uncinate process.

#### Instrumentation

High-resolution video endoscopy with angled endoscopes has revolutionized the management of inflammatory diseases of the frontal sinuses. While the 0-degree endoscope is effective in performing the majority of the ethmoidectomy, a 45 or 70-degree endoscope is often necessary to fully visualize the frontal recess. A variety of angled instruments are available for removal of frontal recess cells. These instruments typically have a 90-degree curvature, or a less-angled 55-degree curvature. Non-cutting giraffe forceps as well as through-cutting forceps that open in an anterior-posterior orientation, as well as a medial-lateral orientation allow for precise removal of frontal cell fragments with preservation of mucosa. Curved frontal sinus curettes, suction catheters, rongeurs, mushroom punches, and frontal ostium seekers are also used when exploring the frontal recess and removing obstructing cells [9].

Powered instrumentation also plays an important role in frontal recess surgery. A 2.9 or 3.5 mm microdebrider blade with a 90° angle is commercially available. The blade is thin and long enough to insert into the frontal sinus and is capable of removing polyps while preserving underlying mucosa. At the same time, the mouth of the debrider is smaller than the standard straight 4 mm blade and can be rotated in the position desired by the surgeon. The microdebrider can be used to remove frontal recess cells with precision and minimal damage to surrounding mucosa. In patients

with extensive polypoid disease, removal of polyps in the frontal recess and within the sinus itself using forceps may prove to be time-consuming and may lead to mucosal stripping. The judicious use of an angled microdebrider is highly recommended in such cases (Video 19.1).

• Stereotactic navigation is recommended for primary frontal sinus surgery because of the complex anatomy. Even in cases with simple anatomy, image guidance is an invaluable teaching and training tool.

Virtually all commercially available image guidance systems have angled instruments that can be used for localization.

## Technique

A careful analysis of CT images should be performed prior to initiating surgery. The CT scan is ideally performed with 1 mm or less axial cuts, and high-resolution reformatted coronal and sagittal images should be available as well. It is recommended that the surgeon systematically reviews all three planes of the CT scan in order to create a three-dimensional concept of the patient's frontal recess anatomy.

Things to look for on the CT scan:

- Kuhn frontal recess cells
- Relationship of the uncinate process to the frontal ostium
- · Relationship of the agger nasi cell to the frontal ostium
- · Location of the anterior ethmoidal artery

In addition, the surgeon must formulate a stepwise plan based on this anatomy. Haphazard removal of septations in the frontal recess with the hope of finding the frontal ostium is not time-efficient, may lead to complications, and is generally not advisable. When reviewing the imaging, it is helpful to keep in mind some of the common causes of failure after primary frontal sinusotomy, namely incomplete dissection of frontal recess cells and incomplete removal of the superior attachment of the uncinate process.

Approaches to the frontal recess include the following:

- · Posterior to anterior ethmoidectomy
- · Anterior approach via agger nasi cell with or without "axillary flap"
- "Bulla intact" frontal sinusotomy

Because of the nature of mucosal inflammation in chronic rhinosinusitis, frontal sinus disease is usually found in conjunction with inflammation of the other paranasal sinuses. In our practice, endoscopic sinus surgery proceeds first with an ethmoidectomy in an anterior to posterior direction to the level of the sphenoid sinus. With the skull base defined by the level of the sphenoid roof, ethmoid cells along the skull base are then removed in a posterior to anterior direction, ending with dissection of the frontal recess. Therefore, frontal sinusotomy is typically performed last. Although the nuances of surgery of the ethmoid, maxillary, and sphenoid sinuses are beyond the scope of this chapter, having an understanding of the anterior ethmoidectomy is crucial to the success of frontal sinusotomy.

The anterior ethmoidectomy begins with careful medialization of the middle turbinate. Any compromise of turbinate mucosa during this step can produce synechiae in the region of the axilla of the middle turbinate, which can in turn scar the frontal recess. While the middle turbinate may need to be resected in some cases, care must be taken to prevent the cut end of the turbinate from lateralizing and scarring to the lamina papyracea. If a concha bullosa exists, the entire lateral aspect needs to be removed since the middle turbinate will serve as the medial limit of dissection in the frontal recess. Next, a thorough removal of the uncinate process up to its superior attachment must be performed. If the uncinate is not completely visualized with the 0-degree endoscope, it is important to re-address this area later in the procedure. Inferiorly, the natural os of the maxillary sinus is then identified, and a maxillary antrostomy may or may not be performed. The medial wall of the orbit, or the lamina papyracea, must then be visualized or palpated. The lamina papyracea needs to be skeletonized during the anterior ethmoidectomy in order to serve as the lateral extent of dissection in the frontal recess.

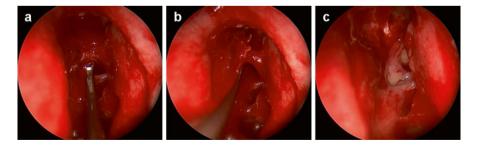
After the skull base is defined, ethmoidectomy proceeds in a posterior to anterior direction along the skull base. As the surgeon approaches the frontal recess, angled endoscopes are usually necessary for adequate visualization. The anterior ethmoidal artery (AEA), which is found in between the second and third lamellae, courses the skull base in an anteromedial direction as it exits the lamina papyracea. It is often found hanging below the level of the skull base, and is dehiscent in up to 66 % of cases [19]. Knowledge of its anatomy is important in preventing troublesome bleeding and in performing a thorough dissection of frontal recess cells. Because of its vulnerable yet variable location, several studies have attempted to define its endoscopic anatomy. As mentioned previously, a useful landmark for the AEA is the SOEC. Because the AEA can be coursing within the posterior opening of the SOEC, aggressive removal of the septation extending from the posterior margin of the supraorbital cell is not advised. Once the AEA is identified, the remaining septations of the ethmoid bulla, SBC, FBC, and anterior margin of the SOEC opening can be removed along the skull base posterior to the frontal ostium, keeping in mind that the ethmoid roof begins to curve superiorly as one approaches the ostium.

In the frontal recess anterior to the ostium, the uncinate process must first be removed up to its superior attachment site. The frontal recess cells, particularly the ANC must be addressed using angled instrumentation. The walls of these cells are removed without disturbing the surrounding mucosa, in a process described by Stammberger as "uncapping an egg" [20]. As these cells are removed, the frontal ostium will become more visible. It is important to note that the while the ANC and the FCs are typically anterior to the ostium, they can be either medial or lateral to the ostium. In addition, the dense bone of the nasofrontal beak can be gently palpated in order to confirm the location of the ostium and ensure that all frontal cells are removed (Video 19.2). If an IFSSC exists, the laterality of its drainage pathway

should be identified preoperatively on imaging. Removal of the septation separating the frontal sinus from the IFSSC will open the drainage pathway for both. With the Draf type I frontal sinusotomy, it should be noted that the frontal ostium itself is untouched, and circumferential mucosal preservation of the ostium should be achieved. Stenting of the frontal ostium may be considered in cases where mucosa is inadvertently injured or the frontal ostium is unusually narrow.

In contrast to the traditional posterior to anterior approach to the frontal recess, other approaches have been described. In cases of isolated frontal sinus disease, frontal sinusotomy alone can be performed without a complete anterior ethmoidectomy. After the middle turbinate is medialized, the superior portion of the uncinate process is removed up to its attachment. The agger nasi cell and frontal cells are then removed, leaving the ethmoid bulla intact. In most cases, this is sufficient to allow identification of the frontal ostium and restoration of the outflow tract. The "intact bulla sinusotomy", first described by Louri in 1993, essentially leaves the ethmoid bulla to be used as the posterior boundary of the frontal recess, thereby preventing injury to the anterior ethmoidal artery and minimizing trauma to healthy sinus cavities [21, 22].

In an approach described by Schaefer and Close in 1990, endoscopic frontal sinusotomy begins with removal of the anterior wall of the agger nasi cell at the axilla of the middle turbinate [23]. The posterior wall of the agger nasi and any existing Kuhn frontal cells are subsequently removed with up-biting forceps. A variation of this technique has been described as the agger nasi "punch-out procedure" [24]. The technique was elaborated by Wormald, who described the use of a posteriorly based mucosal flap to cover the bone along the cut edge of the middle turbinate axilla. The flap is raised and set aside initially, and the anterior wall of the agger nasi cell is entered with a Hajek sphenoid punch. This provides excellent exposure to the frontal recess with a 0-degree endoscope, facilitating removal of other frontal cells with straight instruments. Wormald reported a high rate of frontal ostium patency at a mean follow-up of 15.4 months [25] (Fig. 19.7).



**Fig. 19.7** Left frontal recess dissection with 45-degree endoscope. (a) 90-degree frontal sinus curette placed immediately posterior to a partition containing the anterior wall of the suprabullar cell and the posterior wall of the agger nasi cell. (b) This partition is gently fractured in an anterior direction (c) frontal sinus ostium visualized after removal of the agger nasi (\*See Video 19.2 for corresponding video of Fig. 19.7)

Because lateralization of the middle turbinate has been cited as a common cause of failure after frontal sinusotomy, it must be ensured that the middle turbinate remains in the medialized position during the healing period. Although non-absorbable nasal packing is rarely used in our practice, finger cots are generally better tolerated than petroleum jelly impregnated gauze. We prefer absorbable hemostatic materials, which help to stent open the middle meatus. Also available are absorbable medicated stents, which can be placed in the ethmoid cavity. Finally, surgical techniques for maintaining middle turbinate medialization include creation of controlled adhesions with the septum and a trans-septal middle turbinate suture [26, 27].

Techniques to maintain medialization of the middle turbinate:

- Use of spacer or stent
- Controlled adhesion with septum
- · Trans-septal suture

In difficult cases, an image guidance system is helpful in confirming entry into the frontal sinus. Some have reported the utility of trans illumination as a method for confirming patency of the frontal outflow tract [28]. Other techniques include intraoperative use of a balloon dilatation device with an illuminated guide wire. For cases in which excessive bleeding prevents adequate visualization, angled suction instruments are an option. However, most bleeding subsides with tamponade using oxymetazoline or epinephrine-soaked pledgets, in addition to optimization of the patient's blood pressure. In rare cases when bleeding and poor visualization persist, it is recommended that the procedure is staged.

Postoperatively, patients are prescribed oral antibiotics, nasal steroid spray, and high-pressure nasal saline irrigations.

• In our practice, patients with severe polyposis or history of sinus tissue eosinophilia are also given budesonide irrigations and a month-long prednisone taper if not contraindicated.

Postoperative debridements are performed twice during the first month using angled rigid endoscopes and instruments. Because of the long-term risk of frontal stenosis, these patients are routinely monitored for several years or more.

## Conclusion

Primary endoscopic frontal sinusotomy focuses on the removal of obstructing frontal recess cells with mucosal preservation and minimal manipulation of the frontal sinus ostium. This requires technical skill as well as a sound understanding of the three-dimensional anatomy of the frontal recess. Familiarizing oneself with the different types of frontal recess cells as described by Kuhn is most useful. In addition, knowing the variations of the superior attachment of the uncinate process, as well as the pneumatization patterns of the ANC and FCs, is crucial to successful frontal recess dissection. New instrumentation along with advances in the knowledge of sinonasal anatomy and physiology have allowed excellent long-term outcomes with this approach.

## References

- 1. Weber R, Draf W, Kratzsch B, Hoseman W, Schaefer SD. Modern concepts of frontal sinus surgery. Laryngoscope. 2001;111:137–46.
- Draf W. Endonasal micro-endoscopic frontal sinus surgery, the Fulda concept. Oper Tech Otolaryngol Head Neck Surg. 1991;2:234–40.
- 3. Draf W. Endonasal frontal sinus drainage type I-III according to Draf. In: Kountakis SE, Senior BE, Draf W, editors. The frontal sinus. Berlin: Springer; 2005. p. 219–32.
- Chandra RK, Palmer JN, Tangsujarittham T, Kennedy DW. Factors associated with failure of frontal sinusotomy in the early follow-up period. Otolaryngol Head Neck Surg. 2004;131(4):514–8.
- Wigand ME, Hosemann WG. Endoscopic surgery for frontal sinusitis and its complications. Am J Rhinol. 1991;5(3):85–9.
- Friedman M, Bliznikas D, Vidyasagar R, Joseph NJ, Landsberg R. Long-term results after endoscopic sinus surgery involving frontal recess dissection. Laryngoscope. 2006;116(4):573–9.
- 7. Chan Y, Melroy CT, Kuhn CA, Kuhn FL, Daniel WT, Kuhn FA. Long-term frontal sinus patency after endoscopic frontal sinusotomy. Laryngoscope. 2009;119(6):1229–32.
- Naidoo Y, Wen D, Bassiouni A, Keen M, Wormald PJ. Long-term results after primary frontal sinus surgery. Int Forum Allergy Rhinol. 2012;2(3):185–90.
- Karanfilov BI, Kuhn FA. The endoscopic frontal recess approach. In: Kountakis SE, Senior BE, Draf W, editors. The frontal sinus. Berlin: Springer; 2005. p. 219–32.
- 10. Van Alyea OE. Ethmoid labyrinth: anatomic study with clinical significance of its structural characteristics. Arch Otolaryngol. 1939;29:881–901.
- 11. Kuhn FA. Chronic frontal sinusitis: the endoscopic frontal recess approach. Oper Tech Otolaryngol Head Neck Surg. 1996;7:222–9.
- Bradley DT, Kountakis SE. The role of agger nasi air cells in patients requiring revision endoscopic frontal sinus surgery. Otolaryngol Head Neck Surg. 2004;131(4):525–7.
- Brunner E, Jacobs JB, Shpizner BA, Lebowitz RA, Holliday RA. Role of the agger nasi cell in chronic frontal sinusitis. Ann Otol Rhinol Laryngol. 1996;105(9):694–700.
- Jang DW, Lachanas VA, White LC, Kountakis SE. Supraorbital ethmoid cell: a constant landmark for endoscopic identification of the anterior ethmoidal artery. Submitted for publication. Otolaryngol Head Neck Surg. 2014;151(6):1073–7.
- Lien CF, Weng HH, Chang YC, Lin YC, Wang WH. Computed tomographic analysis of frontal recess anatomy and its effect on the development of frontal sinusitis. Laryngoscope. 2010;120(12):2521–7.
- Stammberger H, Kopp W, Dekornfeld TJ, et al. Special endoscopic anatomy. In: Stammberger H, Hawke M, editors. Functional endoscopic sinus surgery: the Messerklinger technique. Philadelphia: BC Decker Publishers; 1991. p. 61–90.
- Landsberg R, Friedman M. A computer-assisted anatomical study of the nasofrontal region. Laryngoscope. 2001;111(12):2125–30.
- Wormald PJ. The agger nasi cell: the key to understanding the anatomy of the frontal recess. Otolaryngol Head Neck Surg. 2003;129(5):497–507.
- Araujo Filho BC, Weber R, Pinheiro Neto CD, Lessa MM, Voegels RL, Butugan O. Endoscopic anatomy of the anterior ethmoidal artery: a cadaveric dissection study. Braz J Otorhinolaryngol. 2006;72(3):303–8.

- Stammberger H. FESS "Uncapping the Egg" the endoscopic approach to frontal recess and sinuses. A surgical technique of the Graz University Medical School. Tuttlingen: Endo-Press; 2000.
- 21. Louri MC. Endoscopic frontal recess and frontal sinus ostium dissection. Laryngoscope. 1993;103:455-8.
- 22. Landsberg R, Segev Y, Friedman M, Fliss DM, Derowe A. A targeted endoscopic approach to chronic isolated frontal sinusitis. Otolaryngol Head Neck Surg. 2006;134(1):28–32.
- 23. Schaefer SD, Close LG. Endoscopic management of frontal sinus disease. Laryngoscope. 1990;100:155-60.
- 24. Pletcher SD, Sindwani R, Metson R. The agger nasi punch-out procedure (POP): maximizing exposure of the frontal recess. Laryngoscope. 2006;116:1710–2.
- 25. Wormald PJ. The axillary flap approach to the frontal recess. Laryngoscope. 2002;112:494–9.
- 26. Thornton RS. Middle turbinate stabilization technique in endoscopic sinus surgery. Arch Otolaryngol Head Neck Surg. 1996;122(8):869–72.
- Bolger WE, Kuhn FA, Kennedy DW. Middle turbinate stabilization after functional endoscopic sinus surgery: the controlled synechiae technique. Laryngoscope. 1999;109(11): 1852–3.
- Friedman M, Landsberg R, Tanyeri H. Intraoperative and postoperative assessment of frontal sinus patency by transillumination. Laryngoscope. 2000;110(4):683–4.

# **Chapter 20 Image-Guidance in Frontal Sinus Surgery**

David Healy Jr. and Ralph Metson

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#### **Core Messages**

- The utilization of image-guidance systems continues to increase for sinus surgery in general and frontal sinus surgery in particular.
- Image-guidance systems can assist surgeons with identification and enlargement of the frontal sinus ostium.
- Image-guidance systems can be utilized to facilitate the design and execution of external approaches to the frontal sinus.
- Image-guidance systems appear to be most beneficial for frontal sinus surgery in which normal anatomic landmarks are obscured from extensive disease or prior surgery.
- Technology is no substitute for technique.

## Introduction

Stereotactic navigation, or image-guidance, is particularly well-suited to endoscopic sinus surgery which is performed within the stable bony framework of the face, sinuses, and skull. The ability of image-guidance systems to provide the surgeon with enhanced anatomic localization during frontal sinus surgery offers the potential for improved clinical outcome.

• Surgery of the frontal sinus is particularly well suited for surgical navigation systems because of the proximity of the sinus to the orbit and cranial cavities, which demands a high degree of precision and provides little room for misjudgments regarding anatomic relationships.

The variable anatomical development of the frontal sinus, as well as its anterior superior location within the nasal cavity, increases the possibility of disorientation during surgery. The loss of surgical landmarks can be particularly problematic in patients with extensive disease or a history of previous surgery [1].

## **Image-Guidance Systems**

Commercially available image-guidance systems use either an optical-based (infrared) or an electromagnetic-based (radiofrequency) signal to track the position of a surgical instrument relative to the patient's head and sinuses.

• During setup, a reference frame or receiver is registered to the topography of the patient's head and face, allowing the computer to calculate the stereotactic location of the patient's anatomy relative to the reference frame.

This information is processed by a computer workstation, so the location of image-guided instrument tips can be depicted on a three dimensional video display of the patient's preoperative CT scan. Both electromagnetic and optical-based technologies have been found to be highly accurate, providing anatomical localization within 2 mm at the start of surgery [2, 6] and deteriorating by less than 1 mm at the conclusion of surgery [6].

#### Equipment

Electromagnetic-based systems use a radiofrequency emitter mounted near the patient's head to generate an electromagnetic field at the surgical site (Fig. 20.1). A receiver is positioned on the patient, typically on the forehead, and is registered for stereotactic mapping (Fig. 20.2). Separate radiofrequency receivers are integrated into probes and surgical instruments that determine their location relative to the patient. Cables connect the transmitter and receivers to the central workstation, where the data is processed and displayed on a multiplanar video image of the patient's preoperative CT scan.

Optical-based image-guidance systems use an infrared camera array to determine instrument and head position (Fig. 20.3). The camera tracks the coordinate position of optical markers that are attached to probes or surgical instruments. A

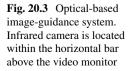


Fig. 20.1 Electromagnetic-based image-guidance system



**Fig. 20.2** Headset and registration probe used for the electromagnetic-based system. The transmitter (*black*) emits radiofrequency signals that are detected by the receivers (*white*) located in the headset and probe. These devices are attached by wires to the image-guidance system





**Fig. 20.4** Headset and probe used for the optical-based imageguidance system. The mirrored spheres reflect the infrared signal, enabling the camera to track the position of the patient's head and the probe tip



separate set of markers is mounted to a reference headset worn by the patient during surgery to monitor head position (Fig. 20.4). Optical-based systems use an infrared emitter in the camera array, which illuminates highly reflective spheres attached to the surgical instrument and patient headset. The camera tracks the infrared emissions reflected from the spheres, and this spatial information is processed by an optical digitizer and displayed in multiplanar format on a video monitor (Fig. 20.5).

## **Considerations**

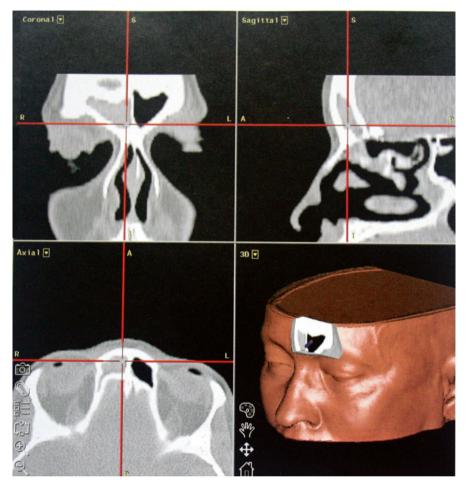
Although both types of Image-Guidance systems are relatively easy to use and have an equivalent degree of anatomic precision, these tracking technologies are associated with different disadvantages.

• For electromagnetic systems, metallic objects in the surgical field may cause signal distortion.

Instrument tables, anesthesia equipment, and other sizable metallic devices need to be kept an appropriate distance from the surgical field, or blocked with radiopaque shields. Another practical drawback is the need for wires to transmit the signal from the receivers attached to the patient and the surgical instruments back to the computer. These wires can become entangled in the surgical field or interfere with access around the operative site.

• When using an optical-based system, a primary disadvantage is the need to maintain a clear line of sight between the infrared camera and the optical markers mounted on the surgical instrument and patient headset.

The instrument must be held with the reflective spheres uncovered and pointed in the direction of the infrared camera, and the endoscope must be positioned so as not to interfere with this line of sight. Furthermore, operating room personnel and



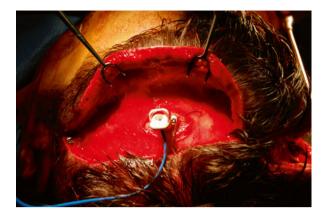
**Fig. 20.5** Video display of axial, coronal, sagittal and 3D views of preoperative CT scan in a patient with an opacified right frontal sinus. The location of the cross hairs corresponds to the position of the tip of the surgical instrument within the nasal and sinus cavities during endoscopic surgery

equipment cannot be located between the patient headset and the camera lens, which is generally positioned 4 ft above the head of the table.

## Instrumentation

For routine endoscopic surgery of the frontal sinus, conventional navigational instrumentation is may be used. However, for external approaches to the frontal sinus, such as an osteoplastic flap procedure, a forehead reference array would interfere with the placement of the coronal or brow incision. Specialized reference arrays which may be anchored directly to bone have been developed for such cases

Fig. 20.6 Bone-anchored reference array for electromagnetic imageguidance system. This array replaced the standard headset for external approaches to the frontal sinus and skull base



(Fig. 20.6). These posts can be percutaneously affixed to the skull at a site distant from the surgical incision.

# Image-Guidance for Endonasal Approaches to the Frontal Sinus

## **Pre-operative Planning**

Most image-guidance systems allow the surgeon to navigate ("scroll-through") the CT images in multi-planar modality, allowing critical evaluation of sinus anatomy and pathology immediately prior to beginning the surgery. By depicting three-dimensional information in a multiplanar format, synchronized viewing of all three orthogonal planes is possible. The ability to rapidly and simultaneously scroll through all three planes promotes a better sense of the three-dimensional relation-ships of the frontal sinus in regard to important surrounding structures. It is often possible to follow the entire course of the nasofrontal outflow and examine it for areas of pathology. In this regard, surgical navigation systems may be particularly helpful for preoperative planning.

## Frontal Sinusotomy

• Intraoperatively, image-guidance technology may be used to help identify the frontal ostium in an atraumatic manner during frontal sinusotomy.

In those patients with disease limited to the frontal recess, an anterior ethmoidectomy is performed and obstructing tissue removed from the recess. An imageguidance equipped instrument such as a ball-tipped probe or curved suction cannula



Fig. 20.7 Curved image-guided instrumentation used to assist in identification of the frontal sinus outflow tracts

(Fig. 20.7) is then passed to confirm ostial location and patency (Fig. 20.8). Proximity to the adjacent skull base and orbit can also be assessed. Surgical navigation systems may help to distinguish the frontal sinus ostium from an adjacent supraorbital ethmoid cell. When a supraorbital ethmoid cell is present, its opening is typically found in a posterolateral location compared to the more anteriomedial location of the true frontal sinus ostium. However, within the narrow confines of the frontal recess, these two openings can be easily confused and the employment of image-guided technology may clarify the surgical anatomy.

The frontal ostium can also be confused with a fronto-ethmoidal air cell, especially when it pneumatizes superiorly above the nasofrontal beak (i.e. a Type III frontoethmoidal cell using the Kuhn classification). Additionally, distinguishing the true frontal ostium from the interfrontal sinus cell can be critical to performing an adequate frontal sinusotomy, particularly when this cell is diseased. Image-guidance can be very useful in assessing these structures, which all exhibit high variability in patients.

By providing anatomical localization and preventing surgical disorientation, image-guidance technology has been shown to increase surgeon confidence [3]. In a review of 800 sinus procedures done at a community hospital, Reardon [4] noted a significant increase in the number of frontal sinuses entered after the introduction of a surgical navigation system. The incidence of maxillary, ethmoid, and sphenoid sinus entry did not change with image-guidance application.

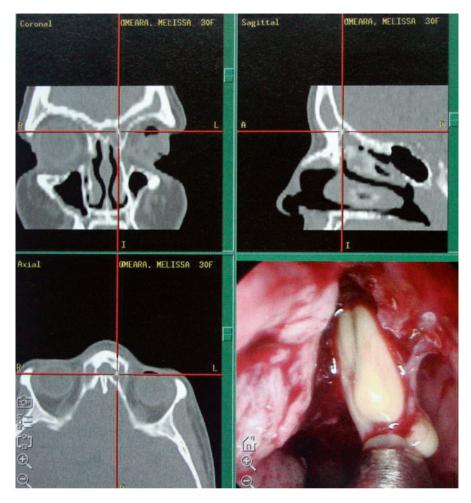


Fig. 20.8 Intraoperative view of image-guided frontal sinusotomy

## Endoscopic Modified Lothrop

Surgery on the frontal sinus remains a clinical challenge because of the high rate of ostial restenosis after frontal sinusotomy. In the past patients who failed frontal sinusotomy proceeded to frontal sinus obliteration. More recently, the frontal sinus drill out procedure, also known as the frontal drill out or Draf III procedure, has been described.

The endoscopic modified Lothrop procedure can be a technically demanding procedure because of the narrow anatomy of the frontal recess, the angled field of view at which the surgeon operates, and the paucity of landmarks from previous surgery. These factors increase the likelihood of surgical disorientation even for the experienced sinus surgeon. When an image-guidance system is utilized for drill out surgery, a calibrated curved probe can be used to assist in identification of the frontal

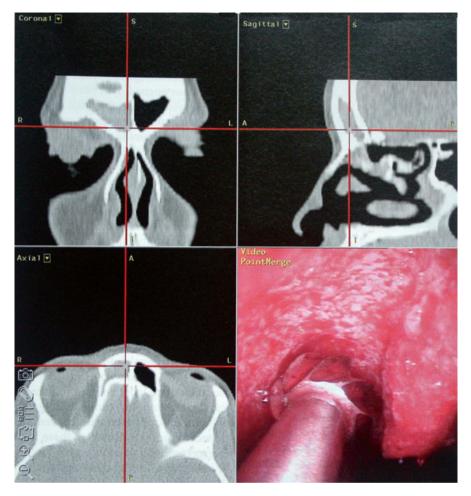


Fig. 20.9 Intraoperative view of image-guided endoscopic frontal sinus drill out (Modified Lothrop or Draf III procedure)

ostium and to ensure that drilling is performed in the direction of the frontal sinus floor. Without an image-guidance system, initial drilling can be essentially blind until the frontal sinus is entered. Once the frontal sinus interior has been identified, bone removal continues under direct endoscopic visualization.

• The surgical navigation system is used during bone removal to alert the surgeon to the proximity of the skull base, orbit and anterior nasal skin (Fig. 20.9).

At the conclusion of surgery, the image-guidance system is used to verify that all compartments of the frontal sinus, including supraorbital ethmoid cells, have been completely opened.

Success rates for endoscopic modified Lothrop surgery with and without imageguidance are comparable, although there appears to be a trend toward a higher surgical success rate when surgical navigation systems are employed [7, 8]. Even though image-guidance may not alter the overall long-term outcome of modified Lothrop surgery, the extent to which image-guidance systems enhances surgeon confidence, particularly when drilling in the vicinity of the orbit and skull base, cannot be overstated.

#### Image-Guidance in External Approaches to the Frontal Sinus

#### Frontal Trephination

External landmarks are typically used to determine the placement of an external trephination incision, which is positioned at the inferomedial aspect of the brow and/or the superomedial aspect of the orbit.

• Image-guidance can serve as a helpful adjunct to confirm that the trephination site is properly positioned for this approach, particularly when the frontal sinus is small.

In addition, prior to performance of the frontal osteotomy, an image-guidance probe can ensure the surgeon that bone is removed in the proper location to enter the frontal sinus safely.

### Osteoplastic Flap

When endoscopic approaches to the frontal sinus fail to control frontal sinusitis, or when anatomic and/or pathologic considerations obviate endoscopic approaches, an osteoplastic flap approach may be considered. With this approach, the frontal sinus cavity can be obliterated or maintained based on clinical considerations and surgeon judgment. Although frontal sinus obliteration is highly successful, its rate of major intraoperative complications remains high, occurring in over 20 % of patients [9]. These complications include dural exposure, dural injury with cerebrospinal fluid leak, and exposure of orbital fat [9]. Most complications are due to misdirected osteotomies that extend beyond the confines of the frontal sinus and result in an osteoplastic flap which is too large. Underestimation of the size of the frontal sinus can result in a bony flap that is too small, making complete removal of mucosa from the sinus interior difficult and increasing the risk of postoperative mucocele formation.

A critical practical consideration in using image-guidance with the osteoplastic flap approach is that the skull reference array must be anchored to parasagittal

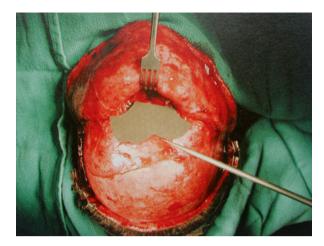


Fig. 20.10 Intraoperative view during image-guided frontal sinus obliteration surgery. The imageguidance probe is used to verify the location of the frontal sinus perimeter and direct bony cuts through the anterior table

calvarial bone rather than placed on the surface of the patient's forehead. Even if a direct soft tissue approach is employed, retraction can be expected to distort the position of a reference frame attached only to skin. If a coronal flap is utilized, then the skull reference array may be anchored and registered once the flap has been raised and the skull exposed. Use of the bone-anchored array affords unencumbered access to the frontal region throughout the procedure. Once the frontal bone has been exposed through a coronal or mid-forehead incision, a hand-held probe is used to outline the perimeter of the frontal sinus. This information, possibly in conjunction with the x-ray template, is used to direct bony cuts through the anterior table with a sagittal saw and expose the sinus interior (Fig. 20.10). Anatomic accuracy of the image-guidance system is verified once the frontal sinus has been opened. Frontal sinus surgery with possible obliteration may then commence, with image-guidance used for anatomic localization as needed.

Carrau et al. [5] were the first to report the use of image-guidance technology for the localization of the osteoplastic flap during frontal sinus obliteration surgery. Measuring the difference between the frontal sinus perimeter outlined by an image-guidance probe and that obtained with a traditional radiographic template in six cases, the authors found that the surgical navigation system was more accurate. A later study [10] compared four frontal sinus mapping methods: 6-ft Caldwell radiography, sinus transillumination, sinus trephination with probing, and image-guidance technology. The authors concluded that image-guided mapping of the frontal sinus was the most accurate method of delineating the limits of the frontal sinus and least likely to overestimate the real sinus margins. Since successful frontal sinus obliteration surgery is predicated upon the precise localization of osseous anatomy, the utilization of a surgical navigation system may enhance the safety of this procedure. One study demonstrated a significant reduction in

the rate of intraoperative complications during frontal sinus obliteration when this method of image-guided surgery was utilized [11].

#### **Endoscopic Frontal Sinus Obliteration**

The endoscopic approach to frontal sinus obliteration provides a minimally-invasive alternative to traditional frontal sinus obliteration. This technique combines a supraorbital incision, similar to that used for frontal sinus trephination, with endoscopic instrumentation. Standard image-guidance headsets that do not conceal the medial canthal region may be employed. A curvilinear incision is made along the inferior edge of the medial evebrow and carried down through the subcutaneous tissue and periosteum. The location of the frontal sinus is then verified with the surgical navigation system and the medial floor of the sinus opened. This bony opening is enlarged to permit passage of both a nasal endoscope and surgical instruments. Using both straight and angled endoscopes, the frontal sinus mucosa is elevated and removed. The entire interior of the frontal sinus is then drilled with a diamond burr under endoscopic visualization to remove any mucosal remnants. The surgical navigation system is used to assist with orientation while drilling within the sinus. It is particularly helpful when exenterating intrafrontal cells or removing septations within the frontal sinus. Once drilling is complete, the frontal sinus ostium is plugged with oxidized cellulose and the sinus is completely filled with abdominal fat. The incision is then closed in layers.

Thus far, the use of image-guidance technology in endoscopic frontal sinus obliteration has avoided complications associated with conventional frontal sinus obliteration such as dural exposure, dural tear with cerebrospinal fluid leak, and orbital entry. In addition, results indicate that operative time, blood loss, and length of hospital stay were all significantly reduced for those undergoing endoscopic obliteration when compared with conventional osteoplastic techniques [12]. However, these results should be interpreted with caution as the long-term outcome of endoscopic frontal sinus obliteration has yet to be determined.

# Conclusion

Image-guidance systems appear to be particularly well-suited to frontal sinus surgery. They can assist the surgeon with localization of the frontal ostium during endonasal procedures and the sinus perimeter during external procedures. Navigation technology has the potential to improve the efficacy and safety of frontal sinus surgery.

• Image-guidance use is no substitution for proper surgical training and technique.

# References

- Kennedy D, Shaman P, Han W, et al. Complications of ethmoidectomy: a survey of the American Academy of Otolaryngology-Head and Neck Surgery. Otolaryngol Head Neck Surg. 1994;111:589–99.
- Metson R, Gliklich RE, Cosenza M. A comparison of image-guidance systems for sinus surgery. Laryngoscope. 1998;108:1164–70.
- Metson R, Cosenza MJ, Cunningham MJ, et al. Physician experience with an optical imageguidance system for sinus surgery. Laryngoscope. 2000;110:972–6.
- Reardon EJ. Navigational risks associated with sinus surgery and the clinical effects of implementing a navigational system for sinus surgery. Laryngoscope. 2002;112(suppl):1–19.
- 5. Carrau RL, Snyderman CH, Curtin HB, et al. Computer-assisted frontal sinusotomy. Otolaryngol Head Neck Surg. 1994;111:727–32.
- 6. Metson R, Cosenza M, Gliklich RE, et al. The role of image-guidance systems for head and neck surgery. Arch Otolaryngol Head Neck Surg. 1999;125:1100–4.
- Javer AR, Kuhn FA. Stereotactic computer-assisted navigational (SCAN) sinus surgery: accuracy of an electromagnetic tracking system with the tissue debrider and when utilizing different headsets for the same patient. Am J Rhinol. 2000;14:361–5.
- Samaha M, Cosenza MJ, Metson R. Endoscopic frontal sinus drillout in 100 patients. Arch Otolaryngol Head Neck Surg. 2003;129:854–8.
- 9. Weber R, Draf W, Keerl R, et al. Osteoplastic frontal sinus surgery with fat obliteration: technique and long term results using MRI in 82 operations. Laryngoscope. 2000;110(6): 1037–44.
- 10. Ansari K, Seikaly H, Elford G. Assessment of the accuracy and safety of the different methods used in mapping the frontal sinus. J Otolaryngol. 2003;32:254–8.
- 11. Sindwani R, Metson R. Impact of image-guidance on complications during osteoplastic frontal sinus surgery. Otolaryngol Head Neck Surg. in press.
- 12. Ung F, Sindwani R, Metson R. Endoscopic frontal sinus obliteration: a new technique for the treatment of chronic frontal sinusitis. Otolaryngol Head Neck Surg. 2005;133:551–5.

# **Chapter 21 Office-Based Treatment and Management of the Frontal Sinus**

Praveen Duggal and John M. DelGaudio

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#### **Core Messages**

- Office-based frontal sinus procedures can decrease costs to the patient and insurance company, while preserving physician reimbursement.
- Patient selection is the most important consideration when performing in-office procedures.

Electronic supplementary material The online version of this chapter

(doi:10.1007/978-3-662-48523-1\_21) contains supplementary material, which is available to authorized users.

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_21

- The ideal in-office patient has a low anxiety level and is capable of fully understanding the procedure, its benefits, limitations, and risks.
- Local anesthetics and vasoconstrictive medications applied topically and by injection can provide analgesia and good hemostasis during the procedures.
- Advantages of in-office procedures include: cost advantage for patients and insurance companies, decreased time off from work, no need for general anesthesia, no operating room (OR), anesthesia, or recovery room fees. Physician reimbursements are equal to the OR.
- Anatomical factors that may make an office-based approach more difficult are an ipsilateral superior nasal septum deviation, concha bullosa, lateralized middle turbinate, and neo-osteogenesis.
- Balloon dilation is a useful tool with some primary frontal sinus disease and post-operative frontal outflow tract stenosis.
- Topical medical therapy including nasal steroid sprays, steroid irrigations, and topical steroid drops can be beneficial in the postoperative period and for frontal recess edema.

# Introduction

The endoscopic technique allows the Otolaryngologist to treat sinonasal disease through a minimally invasive approach. Improvements in visualization and instrumentation have expanded the boundaries of endoscopic treatment to include orbital, skull base, and intracranial pathologies. Endoscopic approaches provide excellent visualization of anatomy and pathology, and advanced techniques have reduced morbidity while equaling or improving outcomes compared to open procedures. While technological advances have allowed us to go further with the endoscope, pressures of the financial healthcare crisis are requiring physicians to try to reduce cost while improving patient outcomes and satisfaction. One way of achieving this is by the use of office-based procedures whenever possible. Office-based sinonasal procedures have been found to be cost-effective with high patient satisfaction [1]. The ability to perform office surgery eliminates the need for a general anesthetic, reduces patient downtime and lost productivity, and eliminates operating room, anesthesia, and recovery room expenses. This can reduce healthcare expenditures and decreases time to treatment and potential cure. A recent publication performed a cost analysis of office-based versus operating room sinonasal procedures and showed that office procedures could be performed with significantly lower costs to the patient and healthcare system than matched procedures in the operating room. There was no significant difference in physician reimbursement [1].

Rhinologic disease processes involving the frontal sinus can be challenging from a medical and surgical management perspective. The frontal outflow tract is complex and anatomically varied from patient to patient. While not for every patient, some frontal sinus disease can be handled in an office setting just as well as in an operating room. This chapter will discuss the patient and disease factors that are amenable to office-based treatment.

## **Indications and Patient Selection**

## Anatomic Considerations

Surgery of the frontal sinus is the most difficult and challenging of all sinus surgery due to the varied and complex anatomy of the frontal recess. The borders of the frontal recess are the agger nasi cell anteriorly, the ethmoid bulla posteriorly, the attachment of the middle turbinate medially, and the orbit laterally. The frontal recess can contain multiple cells including frontal cells [2], suprabullar, frontal bullar, and supraorbital cells. Imaging is of vital importance to every patient in order to gain an understanding of the patient's anatomy and disease process. Computerized tomography (CT) scan of the sinuses is indispensable in evaluating the anatomy. Thin section axial images (0.5–1 mm slice thickness) should be obtained. Reformatted images in the sagittal and coronal planes give additional information regarding the anatomy of the frontal sinus outflow tract.

Prior to any intervention the sinus surgeon should review the imaging of the patient's frontal recess anatomy. Patients with simpler frontal recess configurations are better candidates for office surgical procedures. A larger A-P diameter of the frontal recess will also make access and visualization easier in the office. Anatomical factors that may make an office-based approach more difficult are a superior nasal septal deviation, concha bullosa, and a lateralized middle turbinate.

 Middle turbinate lateralization and bony obstruction, especially neo-osteogenesis, may make an office frontal sinus procedure difficult (Fig. 21.1).

## **Patient Selection**

• The most important consideration in office-based rhinologic surgery is patient selection [3].

While the underlying disease process or patient anatomy may be amenable to office intervention, the importance of patient tolerance and compliance is paramount. A thorough explanation of the procedure, goals, expectations, and postprocedure care should occur prior to any intervention. Informed consent should discuss all risks associated with the procedure and ample time for questions or patient deliberation should be given. After a candid dialogue, the physician should assess the patient's receptiveness to the procedure as well as their associated anxiety level.

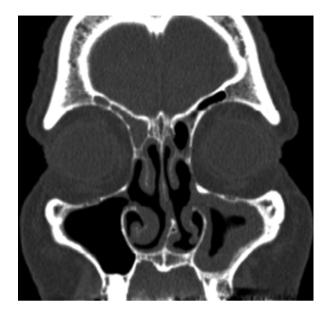


Fig. 21.1 Coronal CT scan showing a lateralized right middle turbinate and neo-osteogenesis of the right frontal sinus outflow tract

• Highly nervous patients are poor candidates for in-office procedures. However, apprehension associated with in-office procedures can mitigate after explaining the procedure with the patient.

Patients frequently become more receptive to the in-office procedure when faced with the alternative of going to the OR and having a general anesthesia and more postoperative downtime. One should not exclude those patients whose underlying health and co-morbidities may prohibit them from undergoing general anesthesia. These patients can be excellent candidates for a minimally invasive office-based approach. Even well-selected patients can have a vasovagal response, resulting in a self-limited episode of systemic hypotension often associated with bradycardia, peripheral vasodilation, and possibly a loss of consciousness. In a recent study by Radvansky et al., in-office rhinologic procedures had a vasovagal episode rate of 0.16 % [4]. The physician and staff should be prepared to manage a patient's symptoms if this occurs in the office.

## Anesthesia

• After appropriate patient selection, adequate anesthesia is the most important component in performing successful office surgery.

Sufficient anesthesia can be obtained with topical agents alone, but injected local anesthetics are frequently necessary, especially when dissection requires deeper penetration into the sinonasal cavities. As with any nasal endoscopic procedure, mucosal vasoconstriction is important in providing adequate access and visualization, thus reducing unnecessary tissue trauma and pain. Common office decongestants such as oxymetazoline, phenylephrine, or 1:1,000 epinephrine are used topically within the nose to achieve vasoconstriction as well as minimize bleeding. The former two medications can be applied either through a spray or delivered to the tissue on cotton applicators, while the epinephrine can only be applied by cotton applicators.

Topical anesthetic agents are routinely used in nasal endoscopic procedures and are readily available [5]. The most common medications are pontocaine and lidocaine. These medications provide surface anesthesia and are therefore only effective at the areas of mucosal contact. Each anesthetic has its associated toxicity levels and these should be reviewed prior to any use. Often these anesthetic and decongestant medications are combined to provide both anesthesia and vasoconstriction at the same time. For office procedures multiple applications of the topical medicine combination spray are necessary, as the distribution of mucosal contact improves with greater mucosal decongestion.

In addition to topical anesthetics, local anesthetic injections are frequently employed. These local blocks can provide some additional anesthesia to the sinonasal cavity. We use 1 % Lidocaine with 1:100,000 epinephrine. Longer lasting agents can be used but are not necessary, as there is not significant pain after the procedure. For frontal sinus procedures we will inject the attachment and leading edge of the middle turbinate as well as the superior lateral nasal wall.

 The topical anesthetic and decongestant combination and local anesthesia are usually sufficient to provide adequate anesthesia and a cooperative patient.

We do not use sedatives in any of our patients prior to or during the procedure, but this is an option for patients undergoing office surgery. Sedated patients should be monitored during and after the procedure, but this is not necessary when using only topical and local anesthetics. Patients receiving sedation need to be accompanied by another adult and should not be allowed to drive after the procedure.

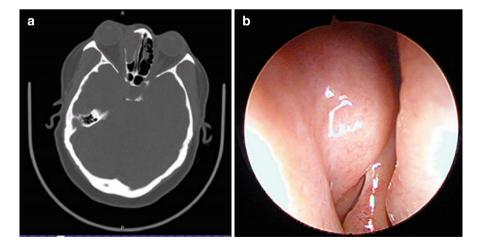
# **Conditions Amenable to Office Frontal Sinus Surgery**

## Frontal Sinusitis

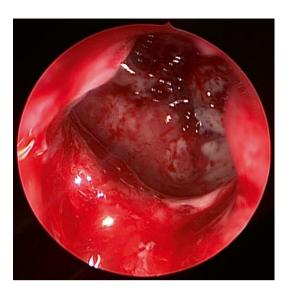
Frontal sinusitis is the result of acute or chronic inflammation and obstruction of the frontal sinus drainage pathway. Patients most likely to benefit from an in-office intervention are those with isolated acute frontal sinusitis with significant symptoms, and those with isolated chronic frontal sinusitis. For a primary frontal sinus procedure in the office the patient should have relatively simple frontal recess anatomy. A significant septal deviation to the side of the disease is a relative contraindication. Performing a primary frontal sinusotomy with cold instruments in the office is difficult and rarely done, but balloon technology can be an appropriate tool to open the frontal recess in these situations.

# Frontal Mucoceles

A mucocele is an expanded mucus-filled sinus that results from obstruction of the sinus outflow tract. This is often secondary to chronic inflammation, nasal polyposis, trauma, or a consequence of a prior surgery, such as with lateral scarring of the middle turbinate. Thinning and remodeling of bone, often with areas of dehiscence, can be seen on CT imaging as the mucocele expands. Contents of mucoceles can vary and depend on the chronicity of the disease process involved. Frontal sinus and frontoethmoidal mucoceles are the most frequent of all sinonasal mucoceles [6]. These are frequently asymptomatic and are noticed incidentally on imaging studies. However, they can present with headache, and when large can cause nasal obstruction and orbital symptoms such as proptosis and diplopia from erosion of the lamina papyracea. Wide marsupialization of the mucocele is the recommended treatment [7]. This can be performed in the office if the inferior aspect of the mucocele can be visualized bulging into the nasal cavity and ethmoid sinus, or it can be accessed with dissection through the uncinate process and anterior ethmoid cells (Fig. 21.2a, b). If the frontal recess has previously been opened then this is usually a very straightforward procedure, as there are not usually multiple obstructing ethmoid cells. If there has been no previous surgery on the sinuses then this is more difficult and can require more dissection through the anterior ethmoid cells. A recent study on in-office mucocele drainage reported that 35 of 36 were successfully drained in the office, with the one failure having significant neoosteogenesis. The long-term success rate was 91 %, with 3 requiring additional surgery due to recurrence from scarring or loculation causing incomplete drainage. This series included 51 % with bone erosion, with 46 % having orbital erosion and 20% skull base erosion. Patient satisfaction was extremely high, with only one patient reporting that they would prefer having the procedure in the OR rather than the office [8].



**Fig. 21.2** (a) Axial CT scan of a superficial mucoceles partially eroding the right lamina papyracea. (b) Endoscopic view of the same mucocele bulging in the right nasal cavity anterior to the attachment of the right middle turbinate



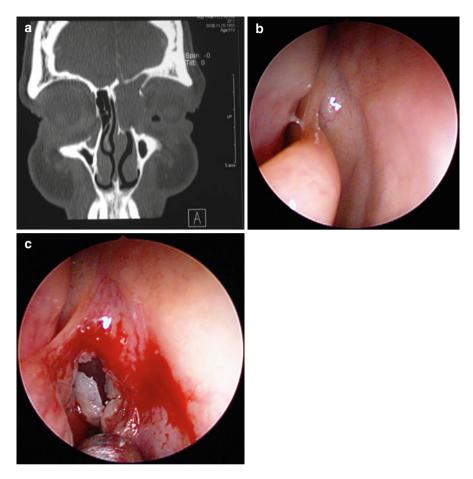
**Fig. 21.3** Endoscopic view after fully opening a left frontal mucocele in the office

In previously operated patients the mucocele is usually more accessible. After adequate anesthesia is obtained, the inferior wall of the mucocele cavity is perforated with a curette. This will usually result in mucus draining into the nasal cavity if it hasn't already begun with the local anesthetic injections. The angled curette and cutting forceps can be used to remove the entire inferior wall of the mucocele. The anterior and posterior walls should be removed as widely as possible with cutting instruments such as a frontal mushroom punch, frontal through-cutting forceps, or a frontal Kerrison rongeur. All bone chips and loose mucosa should be removed. Every attempt should be made to minimize trauma and prevent any stripping of frontal sinus or frontal recess mucosa. Even though this is being done in the office the goal is the same as if it is being done in the operating room, creating a large drainage pathway to decompress the mucocele and prevent recurrence, scarring or stenosis of the frontal recess (Fig. 21.3).

• Patients need to be counseled prior to the procedure that they will hear crunching noises as the bone is being removed. This can be very disconcerting to the patient if they are not told before the procedure.

The office drainage of frontal mucoceles in the patient that has not had previous sinus surgery is not as straightforward as in the previously operated patient. These patients have normal anatomic structures that need to be traversed to access the mucocele. The CT scan needs to be thoroughly assessed to determine if the mucocele can addressed in the office, and what is the best approach. If the mucocele extends all the way down to the uncinate process or the agger nasi cell then the ethmoid bulla does not need to be disturbed (Fig. 21.4a–d) (Video 21.1).

If the mucocele extends to the suprabullar recess or involves the ethmoid bulla then the ethmoid bulla needs to be opened. The upper portion of the vertical uncinate process and the middle turbinate need to be adequately anesthetized. The uncinate process is divided with back-biting forceps, and the superior portion removed

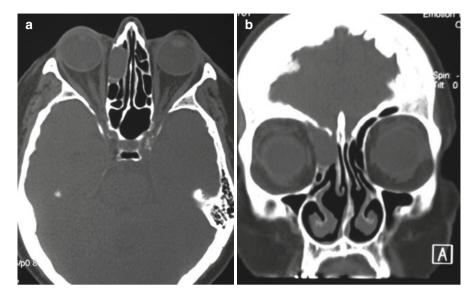


**Fig. 21.4** (a) Coronal CT scan of a left sided frontal mucocele in a previously un-operated patient. (b–c) Endoscopic view of the mucocele bulging into the nasal cavity, opened with frontal cutting instruments, and then drained

with 45-degree through-cutting forceps or a straight mushroom punch. The agger nasi cell can then be easily accessed through its inferior wall, which should be widely removed. This typically then provides wide access to the frontal recess and the bulging mucocele (Fig. 21.5a–b) (Video 21.2). Once the inferior wall of the mucocele is identified it can be opened up as mentioned above.

Bone expansion and erosion are common with mucoceles of the frontal sinus and are not a contraindication for office treatment. The most common areas of dehiscent bone are the orbit, followed by the posterior table of the frontal sinus and the ethmoid roof. These areas need to be recognized on the CT scan and the surgical approach needs to take this into account (Figs. 21.5a, b and 21.6a, b).

Since the periorbita is usually intact, drainage of the mucocele does not cause prolapse of orbital contents into the mucocele cavity. Lateral dissection in the frontal recess is to be avoided in these cases until the orbital contents are identified.



**Fig. 21.5** (a) Axial CT showing right mucocele obstructing frontal outflow tract. (b) Coronal CT shows agger nasi cell inferior to right frontal mucocele. Mucocele erodes the lamina papyracea

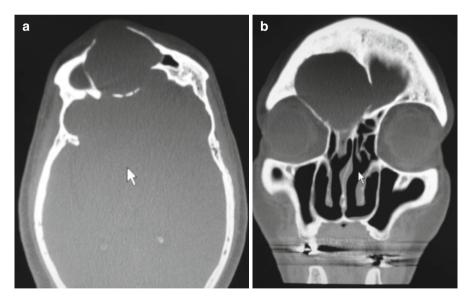


Fig. 21.6 (a, b) Axial and Coronal CT scan images with orbital wall expansion and both anterior and posterior table frontal sinus erosion

Blindly sticking a suction into the frontal sinus is to be avoided so as not to inadvertently injury the orbital contents or frontal lobe. Neo-osteogenesis is a common finding, and can lead to obstruction of the frontal recess in the previously operated patient. Neo-osteogenesis is a relative contraindication for an in-office procedure. Thickened bone can be difficult to remove without significant force to manipulate the bone, which is difficult or impossible for the awake patient to tolerate, and is not recommended in an office setting.

# Nasal Polyps

Nasal polyps are the end-stage of nasal mucosal inflammation and are secondary to chronic rhinosinusitis, allergic and metabolic conditions, and other rhinologic disorders. Nasal polyposis has been shown to be linked with chronic rhinosinusitis in 20–33 % of patients [9]. While the specific cause is still not completely understood, nasal polyposis is a problem often associated with chronic inflammation of the sinonasal mucosa. Recurrence of nasal polyps is a common occurrence and can lead to repeated obstruction of the frontal drainage pathway.

The treatment of nasal polyps often involves a combination of medical and surgical management. Medical treatment is the mainstay of treatment, with the goal of eliminating or reducing the inflammatory reaction. This generally consists of a combination of topical and systemic steroids. Systemic steroids have significant benefits in the short-term management of nasal polyposis [10]. However, the prolonged use or higher dosage of systemic steroids comes with serious adverse risks which can include physical and medicolegal implications [11]. In addition, extensive nasal polyposis is unlikely to improve with medical management alone.

 Patients with recurrent sinonasal polyposis after adequate endoscopic sinus surgery and failed medical therapy are good candidates for office polypectomy [12].

Frontal recess polyp disease can be addressed aggressively as long as the anatomy is well understood and only soft tissue obstruction of the previously opened frontal sinus is present. This can be performed alone or in combination with removal of polyps from the other sinuses. Unilateral sinonasal masses that mimic nasal polyps include such pathologies as meningoencephaloceles and inverted papilloma. However, with good pre-operative imaging and clinical suspicion, complications and improper diagnosis can be avoided.

The approach for removal of sinonasal polyps is generally the same as in the operating room. The technique involves angled endoscopes and instrumentation. Isolated nasal polyps in the ethmoids and frontal recess can be removed with through-cutting instruments at the attachment of the polyps. For more extensive nasal polyps that are common in patients with prior surgery, the 40 or 60-degree microdebrider blades can be used conservatively. The removal of all sinonasal polyp disease should be attempted. Only pedunculated polyps should be removed in the frontal recess and up into the frontal sinus, and mucosa along the bony frontal outflow tract should be avoided as circumferential scarring and stenosis can occur in the frontal recess (Video 21.3). As polyps are removed the reapplication of topical and local anesthetic may be necessary to anesthetize deeper tissue. Use of the microdebrider can lead to bleeding from the shearing of polypoid tissue, but this is

generally mild. Utilizing topical vasoconstrictive or hemostatic agents and keeping the patient sitting up or in reverse Trendelenburg position can reduce blood loss [13]. Postoperative treatment with topical steroid therapies is very important in postoperative and long term management of these patients.

#### **Balloon Dilation**

Balloon technology is a relatively recent tool in Otolaryngology to access and dilate the sinus outflow tract.

• Balloon technology can be used in the frontal sinus with the goal of dilating the frontal outflow tract while preserving mucosa.

There are multiple manufactures of sinus balloon technology including Acclarent (Menlo Park, CA), and Entellus Medical (Maple Grove, MN) which are the only current FDA-approved systems for the frontal sinus at of the time of this publication.

Balloon technology and its indications for use have been discussed and debated in the literature [14–16]. The CLEAR study (Clinical Evaluation to confirm safety and efficacy of sinuplasty in the paranasal sinuses) demonstrated clinical results of balloon dilation [17–19]. The study mainly focused on comparisons between hybrid procedures (balloon sinuplasty with endoscopic sinus surgery) and balloon sinuplasty alone. This multi-institutional study showed no difference in SNOT-20 scores at 2 years and an observable patency rate of 85 % at 1 year. The same percentage of patients showed improvement of their symptoms at 2 years and radiographic evidence confirmed resolution of disease at 2 years. However, the CLEAR study was not randomized and there was no control group in the study. Plaza et al. [20] evaluated balloon dilation of the frontal recess in a double-blind randomized clinical trial of hybrid FESS and balloon dilation versus conventional FESS in the treatment of chronic rhinosinusitis. They showed a statistically significant reduction in Lund-Mackay score in favor of the hybrid procedure versus standard FESS.

Recent studies by Albritton et al. [21, 22] showed clinical success as well as safety and patient tolerance with use of balloon dilation in the office setting. Ninety one percent of office dilations were successfully achieved and less than 6 % noted intense pain during the procedure. A recent prospective study performed on 203 patients with medically refractory chronic sinusitis showed almost 94 % technical success with 251 out of 268 successful dilations of the frontal sinus in the office [22].

Indications for balloon dilation in the office can vary among physicians.

 Most patients with uncomplicated frontal sinus anatomy and isolated frontal sinusitis are good candidates for the balloon.

Other possible candidates for a dilation procedure include those patients with narrowing of the non-bony post-operative frontal sinus outflow tract. Dilations could be performed on these patients that would minimize trauma and circumferential stenosis (Fig. 21.7a, b).

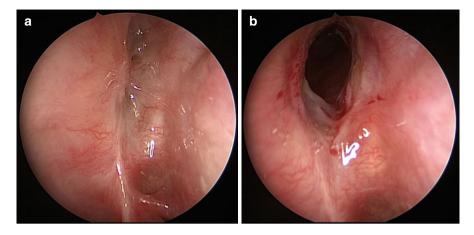


Fig. 21.7 (a) Stenosis and scarring of the frontal sinus outflow tract after endoscopic sinus surgery. (b) Frontal sinus outflow tract post in-office balloon dilation

The technique for office-based balloon dilation of the frontal sinus has been described elsewhere [23]. Balloon dilation of the frontal sinus is performed using an angled endoscope and a 70-degree sinus cannula. The cannula is placed between the uncinate process and the upper face of the ethmoid bulla. A lighted flexible guide-wire can then be placed under endoscopic visualization into the frontal recess until cannulation of the frontal ostium is achieved. The lighted guide-wire allows for trans illumination of the forehead skin overlying the frontal sinus to confirm entrance of the wire into the frontal sinus. The balloon-dilating catheter is then advanced over the guide-wire into the frontal recess. The balloon is then inflated to the recommended manufacturer guidelines for approximately 5 s. The balloon is then frontal outflow tract as needed (Video 21.4).

#### Post op Management and Procedures

Performing a properly indicated and technically proficient sinus surgery is only part of the treatment for a good clinical outcome. Long-term management of chronic rhinosinusitis involves controlling the mucosal inflammatory process, which requires routine surveillance. If needed, medical and/or surgical intervention is utilized to prevent a patient's underlying disease process from progressing.

Our routine post-operative care involves an office endoscopy and debridement one week after the initial surgery, followed by another additional debridement between week 2 and 4 depending on the disease process, patient compliance, and appearance of the sinus mucosa. Frequency of follow-up and endoscopic debridement is determined by the patient's clinical and endoscopic improvement. There is

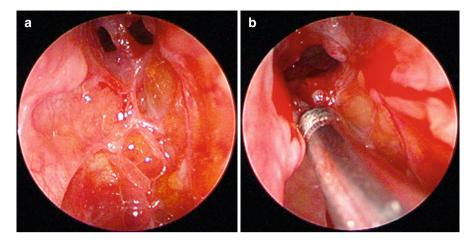


Fig. 21.8 (a) Endoscopic view of frontal sinus outflow tract adhesions in the early post-operative period. (b) Endoscopic view after in-office lysis of adhesion

level B evidence that in-office endoscopic sinus cavity debridement after ESS improves both short-term and long-term clinical outcomes [24].

After any frontal sinus procedure, the frontal sinus should again be visualized in the post-operative period. At the first post-operative visit the frontal recess should be visualized with an angled endoscope and suctioned with a curved suction. Generally mucus, crust, dissolvable nasal packing, or blood clot is found at the first visit and should be suctioned and removed. Attention is given to areas of denuded bone, as these areas will be more prone to crusting and scarring. Due to the narrow drainage pathway of the frontal recess cicatricial bands can form. If synechiae are noted at the early post-operative visits they should be lysed to avoid further stenosis (Fig. 21.8a, b). Circumferential scarring or stenosis that is seen at later appointments can still be addressed in the office. Often these areas of scar can be taken down with frontal cutting instruments while trying to avoid causing further trauma to the frontal drainage pathway.

• Balloon dilation of the frontal ostium may be a good tool to employ in these instances if the scar or stenosis is not from neo-osteogenesis or bony obstruction.

## Nasal Irrigations and Topical Medications

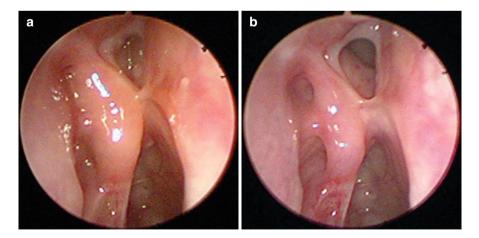
Nasal irrigations have been shown to be well tolerated and efficacious in improving symptoms of chronic rhinosinusitis [25]. A high-volume, low-pressure system in a squeeze bottle remains the optimal delivery device. The authors' post-operative management commonly includes nasal saline irrigations at least twice a day generally starting on the operative day.

To control mucosal inflammation and edema post-operatively topical medical therapy has been used to optimize healing. Advantages of topical medical therapy include direct drug delivery onto diseased tissue with the potential for delivering higher local drug concentrations while minimizing systemic absorption. Disadvantages of topical medical therapy include application challenges, local discomfort, epistaxis, and inconsistent sinus penetration [26].

Topical nasal steroid sprays are employed in those patients with open sinus cavities with post-operative inflammation and/or residual sinonasal polyposis. Topical nasal steroid sprays have been shown to improve clinical outcomes by minimizing mucosal inflammation when started within the first 2–6 weeks after sinus surgery [24, 26]. Unlike the use of systemic steroids, nasal steroid sprays have minimal systemic effects and therefore can be used as long-term therapy. In patients with nasal polyps it has been shown that polyp recurrence rate was reduced and time to polyp recurrence was lengthened [27, 28].

Another common post-operative topical treatment is nasal steroid irrigations. This is commonly employed in patients that have recurrence or persistence of nasal polyps or significant post-operative edema. A recent Cochrane review [29] of topical steroids used in patients with chronic rhinosinusitis with nasal polyposis showed improved symptoms, reduced polyp size, and decreased polyp recurrence after surgery. A commonly used preparation is budesonide saline irrigations twice daily (0.5 mg/2 mL or 1 mg/2 mL mixed into 240 mL of saline). Topical steroid rinses can be beneficial at any time, but are most effective in patients with residual mucosal inflammation after surgery or recurrent edema or polyps after the first or second post-operative visit.

Topical steroid drops can also be used to treat and prevent frontal ostium stenosis and edema. A retrospective study by DelGaudio et al. [30] evaluated three postop-



**Fig. 21.9** (a) Endoscopic view showing edema of a patient's left frontal outflow tract at 3 months post-operatively. (b) Endoscopic view of the same patient's left frontal outflow tract after 6 weeks of dexamethasone ophthalmic drops applied intranasally twice a day

erative off-label nasal steroid solutions (dexamethasone ophthalmic drops, prednisolone ophthalmic drops, and ciprofloxacin/dexamethasone otic drops) in patients after undergoing revision endoscopic sinus surgery. Steroid drops were used to treat frontal ostium stenosis or frontal recess edema. The results demonstrated 64 % percent of sinuses were treated successfully with topical steroid drops after endoscopic sinus surgery. The results also showed that off-label steroid drops may lower the risk of revision sinus surgery and outflow tract stenosis while reducing the number of oral steroid rescue episodes (Fig. 21.9a, b). Nasal sprays tend to provide more nasal cavity coverage, whereas sinonasal irrigations and drops tend to provide improved penetration to the sinuses and may be a better post-operative topical therapy delivery technique [24, 31].

## Conclusion

Otolaryngologists continue to make great strides in their evaluation and treatment of frontal sinus disease. Attempts to reduce healthcare expenditures and improve patient satisfaction are reasons to perform office-based surgery. Patient selection is crucial in deciding who will benefit most from an office-based frontal sinus intervention. Office procedures offer the advantage of decreased time off from work, cost advantages for the patient and payers, no risks of general anesthesia, and equal reimbursement rates for the physician. With the proper anesthesia, knowledge of the anatomy and surgical technique, there are multiple situations that are amenable to in-office frontal sinus procedures.

## References

- 1. Wise SK, DelGaudio JM. Cost analysis of office-based and operating room procedures in rhinology. Int Forum Allergy Rhinol. 2012;2(3):207–11.
- 2. Bent J, Cuilty-Siller C, Kuhn F. The frontal cell as a cause of frontal sinus obstruction. Am J Rhinol. 1994;8:185–91.
- Wise SK, Patel ZM. Chapter 5: patient selection and informed consent for office-based rhinology procedures. In: Patel Z, Wise SK, DelGaudio JM, editors. Office-based rhinology principles and techniques. 1st ed. San Diego: Plural Publishing; 2013.
- 4. Radvansky BM, Husain Q, Cherla DV, Choudhry OJ, Eloy JA. In-office vasovagal response after rhinologic manipulation. Int Forum Allergy Rhinol. 2013;3:510–14.
- Snidvongs K, Harvey RJ. Chapter 6: nasal and sinus anesthesia for office procedures. In: Patel Z, Wise SK, DelGaudio JM, editors. Office-based rhinology principles and techniques. 1st ed. San Diego: Plural Publishing; 2013.
- Scangas GA, Gudis DA, Kennedy DW. The natural history and clinical characteristics of paranasal sinus mucoceles: a clinical review. Int Forum Allergy Rhinol. 2013;3:712–7.
- Laury A, DelGaudio JM. Chapter 12: office-based management of mucoceles. In: Patel Z, Wise SK, DelGaudio JM, editors. Office-based rhinology principles and techniques. 1st ed. San Diego: Plural Publishing; 2013.
- Barrow EM, DelGaudio JM. In-office drainage of sinus mucoceles: an alternative to or drainage. Laryngoscope. 2015;125(5):1043–7. PMID 25418415.

- DeMarcantonio MA, Han JK. Nasal polyps: pathogenesis and treatment implications. Otolaryngol Clin N Am. 2011;44(3):685–95, ix.
- Poetker DM, Jakubowski LA, Lal D, Hwang PH, Wright ED, Smith TL. Oral corticosteroids in the management of adult chronic rhinosinusitis with and without nasal polyps: an evidencebased review with recommendations. Int Forum Allergy Rhinol. 2013;3(2):104–20.
- 11. Poetker DM, Smith TL. What rhinologists and allergists should know about the medico-legal implications of corticosteroid use: a review of the literature. Int Forum Allergy Rhinol. 2012;2(2):95–103.
- Henriquez OA, DelGaudio JM. Chapter 11: office-based nasal polypectomy. In: Patel Z, Wise SK, DelGaudio JM, editors. Office-based rhinology principles and techniques. 1st ed. San Diego: Plural Publishing; 2013.
- 13. Ko MT, Chuang KC, Su CY. Multiple analyses of factors related to intraoperative blood loss and the role of reverse Trendelenburg position in endoscopic sinus surgery. Laryngoscope. 2008;118(9):1687–91.
- 14. Batra PS, Ryan MW, Sindwani R, Marple BF. Balloon catheter technology in rhinology: reviewing the evidence. Laryngoscope. 2011;121(1):226–32.
- Kim E, Cutler JL. Balloon dilatation of the paranasal sinuses: a tool in sinus surgery. Otolaryngol Clin N Am. 2009;42(5):847–56, x.
- Batra PS. Evidence-based practice: balloon catheter dilation in rhinology. Otolaryngol Clin N Am. 2012;45(5):993–1004.
- Bolger WE, Brown CL, Church CA, Goldberg AN, Karanfilov B, Kuhn FA, Levine HL, Sillers MJ, Vaughan WC, Weiss RL. Safety and outcomes of balloon catheter sinusotomy: a multicenter 24-week analysis in 115 patients. Otolaryngol Head Neck Surg. 2007;137(1):10–20.
- Kuhn FA, Church CA, Goldberg AN, Levine HL, Sillers MJ, Vaughan WC, Weiss RL. Balloon catheter sinusotomy: one-year follow-up – outcomes and role in functional endoscopic sinus surgery. Otolaryngol Head Neck Surg. 2008;139(3 Suppl 3):S27–37.
- Weiss RL, Church CA, Kuhn FA, Levine HL, Sillers MJ, Vaughan WC. Long-term outcome analysis of balloon catheter sinusotomy: two-year follow-up. Otolaryngol Head Neck Surg. 2008;139(3 Suppl 3):S38–46.
- Plaza G, Eisenberg G, Montojo J, Onrubia T, Urbasos M, O'Connor C. Balloon dilation of the frontal recess: a randomized clinical trial. Ann Otol Rhinol Laryngol. 2011;120(8):511–8.
- Albritton FD, Casiano RR, Sillers MJ. Feasibility of in-office endoscopic sinus surgery with balloon sinus dilation. Am J Rhinol Allergy. 2012;26(3):243–8.
- Karanfilov B, Silvers S, Pasha R, Sikand A, Shikani A, Sillers M. Office-based balloon sinus dilation: a prospective, multicenter study of 203 patients. Int Forum Allergy Rhinol. 2013;3(5):404–11.
- Sillers MJ, Melroy CT. In-office functional endoscopic sinus surgery for chronic rhinosinusitis utilizing balloon catheter dilation technology. Curr Opin Otolaryngol Head Neck Surg. 2013;21(1):17–22.
- 24. Rudmik L, Schlosser RJ, Smith TL, Soler ZM. Impact of topical nasal steroid therapy on symptoms of nasal polyposis: a meta-analysis. Laryngoscope. 2012;122(7):1431–7.
- 25. Harvey R, Hannan SA, Badia L, Scadding G. Nasal saline irrigations for the symptoms of chronic rhinosinusitis. Cochrane Database Syst Rev. 2007(3):CD006394.
- Rudmik L, Hoy M, Schlosser RJ, Harvey RJ, Welch KC, Lund V, Smith TL. Topical therapies in the management of chronic rhinosinusitis: an evidence-based review with recommendations. Int Forum Allergy Rhinol. 2013;3(4):281–98.
- Jorissen M, Bachert C. Effect of corticosteroids on wound healing after endoscopic sinus surgery. Rhinology. 2009;47(3):280–6.
- Stjarne P, Olsson P, Alenius M. Use of mometasone furoate to prevent polyp relapse after endoscopic sinus surgery. Arch Otolaryngol Head Neck Surg. 2009;135(3):296–302.
- 29. Kalish L, Snidvongs K, Sivasubramaniam R, Cope D, Harvey RJ. Topical steroids for nasal polyps. Cochrane Database Syst Rev. 2012;12, CD006549.
- DelGaudio JM, Wise SK. Topical steroid drops for the treatment of sinus ostia stenosis in the postoperative period. Am J Rhinol. 2006;20(6):563–7.
- Harvey RJ, Debnath N, Srubiski A, Bleier B, Schlosser RJ. Fluid residuals and drug exposure in nasal irrigation. Otolaryngol Head Neck Surg. 2009;141(6):757–61.

# Chapter 22 Revision Endoscopic Frontal Sinus Surgery

Alexander G. Chiu, Gregg H. Goldstein, and David W. Kennedy

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_22

#### Core Messages

- Successful revision endoscopic frontal sinus surgery starts with proper patient selection and medical management of co-morbidities and environmental influences
- Pre-operative planning in at least two and preferably three CT planes is needed in order to plan the surgical approach and the frontal sinus drainage pathway should be clearly identified preoperatively
- Common anatomical causes for revision frontal surgery include a retained superior uncinate process, superior cap of the ethmoid bulla, agger nasi cells, lateralized middle turbinate remnants, frontal recess and supraorbital ethmoid cells
- Surgical approach is most safely done from a posterior to anterior direction along the skull base, where the skull base can first be identified in the posterior ethmoid or sphenoid sinus
- During surgery, the position of the frontal sinus drainage pathway should be reconfirmed with a small malleable probe
- All bony fragments must be removed from the frontal recess, and specialized through cutting instruments and a curved microdebrider should be used so as to spare frontal recess mucosa
- Nearly as important as a good technical surgery is meticulous long-term post-operative debridements and surveillance to insure frontal recess patency

# Introduction

Many will agree that revision endoscopic frontal sinus surgery is one of the most difficult operations for the endoscopic surgeon. The fact that there exists an abundance of different technical operations to treat frontal sinus disease underscores the complexity and nature of its difficulty. Over the years, there has been a progression from external, obliterative procedures to endoscopic management of recurrent or persistent frontal sinus disease and balloon dilatation procedures. Despite the change in techniques, the keys to successful revision frontal sinus surgery have remained proper patient selection, meticulous technique, a thorough knowledge and preoperative conceptualization of the anatomy, and a significant commitment to follow-up care from both the patient and physician.

# **Patient Selection**

When evaluating a patient for a revision endoscopic frontal procedure, it is important to review the patient's symptoms, associated co-morbidities, and radiographic studies. Before deciding on the necessity for any type of revision surgery, it is advisable to review the original CT scan, before any surgery was performed. This helps the surgeon to evaluate the indications for the original surgery and is particularly important for the frontal sinus where the primary surgical indication may be headache. In general, the symptom of headache correlates poorly with chronic rhinosinusitis and it is important to establish the presence or absence of disease in the frontal sinus prior to the first operation. If headache remains the primary symptom, a revision surgery on asymptomatic iatrogenic mucosal change may be avoided if a careful review of the scans prior to the 1st surgery is performed. This review of the prior scans becomes particularly important if the frontal sinus has been obliterated. It is this review that leads to the initial and most important decision to be made, whether or not the patient will benefit from a revision surgery.

As with other revision sinus surgeries, careful consideration should be given to the environmental and general host factors that predispose to recurrent disease. Underlying factors such as allergic rhinitis, underlying immune deficiencies and smoking should be investigated and where possible, managed before any revision surgery is undertaken.

Frontal sinusitis following functional endoscopic sinus surgery (FESS) can represent:

- · Persistent disease
- · Recurrent disease
- · Iatrogenic disease

Persistent disease may be the result of an incomplete initial surgery, poor postoperative care, or as a result of underlying factors predisposing to chronic inflammation. In order to evaluate whether the initial surgery(s) was inadequate, the initial pre-operative report should be reviewed along with an examination of the pre and post-surgical CT scans. An initial operative report which does not mention the dissection of superior ethmoid cells, agger nasi cells and/or frontal recess cells may mean that a proper frontal recess dissection was never performed. Reviewing postoperative CT scans is an appropriate next step, and is an objective aid in determining a cause for persistent disease.

Reviewing of CT scans is best done in multiple planes. In office consultation should result in a review of axial and coronal sections, at a maximum of 3 mm sections, through the paranasal sinuses. Many image guidance companies now offer work stations that allow for the review of CT scans in the sagittal plane, as well as the coronal and axial views and software which accomplishes this is also available from the Internet.

The coronal view is excellent in determining the presence of the following:

- · Remaining agger nasi
- Superior uncinate process
- · Frontal recess
- · Supraorbital ethmoid cells

Sagittal and axial views are important in determining the following:

- Anterior to posterior dimension of the frontal recess
- The identification of a supraorbital ethmoid cell
- · Frontal recess
- Interseptal frontal sinus cell

Using the combination of the triplanar views, the surgeon should build a 3-D concept of the frontal recess anatomy and of the frontal sinus drainage pathway. The dimensions of the frontal recess, particularly in the antero-posterior diameter, should be identified. The presence of neo-osteogenesis may make it impossible to work with the normal fine through cutting frontal recess instruments. The overall frontal sinus pneumatization should also be considered in deciding whether or not to proceed with a revision procedure. A poorly pneumatized frontal sinus, irrespective of the size of the frontal recess, appears less likely to remain patent.

## **Persistent Disease**

The two most common local obstructive causes of persistent frontal recess obstruction are either a medially displaced uncinate process or obstruction from a remnant agger nasi cell (Table 22.1). In a series of 67 patients undergoing revision endoscopic frontal sinus surgery, 79 % of patients had evidence of residual ethmoid bulla or agger nasi cells and 49 % had remnant uncinate processes [1]. A medially displaced uncinate process can result from disease within the terminal recess of the infundibulum, displacing the uncinate medially, where it can fuse to the middle turbinate. A frontal sinus drainage pathway that is medial to the displaced uncinate will be obstructed by this displacement.

The cap of a remnant agger nasi cell is a common finding in a dissection in which angled endoscopes were never used. A 45 or  $70^{\circ}$  endoscope is needed to visualize the top of the frontal sinus. When using a  $30^{\circ}$  or straight endoscope, true visualization of the entire frontal sinus is often unattainable. Entrance into an agger nasi or frontal recess cell can easily be mistaken for the frontal sinus, and the cap and offending frontal recess obstruction will remain.

# *Recurrent or Persistent Disease in the Presence of an Adequate Surgical Procedure*

If it is determined that the initial surgery was adequate, it increases the chances of a patient having recurrent or persistent frontal sinusitis as a result of either a general host or environmental problem. In these cases, revision surgery is not necessarily

Remnant	superior uncinate process	
Agger na	si cell	
Remnant	cap of ethmoid bulla	
Frontal re	cess cells	
Supraorb	tal ethmoid cells	
Iatrogeni	c scarring or neo-osteogenesis	
Polyps ar	d/or mucocele formation	

Table 22.1Commonanatomical causes forrevision frontal sinus surgery

the answer to the problem and treatment of the underlying condition should be more aggressively pursued, particularly in the symptomatic patient.

• The most common sign of recurrent disease is mucosal thickening within the frontal recess and sinus.

Some studies suggest that chronic frontal sinusitis reflects increased disease severity with generalized inflammation and an increased risk of recurrence [2, 3]. In a review of 549 patients with nasal polyposis, patients with asthma, Samter's triad or frontal sinus disease on initial presentation had an increased likelihood of needing revision surgery [4].

• If a surgeon is able to pass a curved 4 mm suction past the polyps or swollen mucosa into the frontal sinus, then further surgical therapy is unlikely to be of additional benefit, unless residual osteitic bony partitions are present. However, more recently some have suggested that a wider opening may facilitate topical steroid irrigation access, even in the frontal sinus.

If residual bony partitions are identified, they can frequently be removed in the office under local infiltrative anesthesia. Persistent frontal recess disease is often seen in patients with nasal polyposis, allergic fungal rhinosinusitis, and recurrent sinus infections. In these cases, appropriate medical therapy should be aggressively pursued. Oral and topical steroids, culture directed antibiotics and/or antifungals may be used to decrease the mucosal edema. In some cases, careful local infiltration with a small particle depot steroid (Kenalog 10) into the thickened frontal recess mucosa may help to control the edema. However, given the known complications of this procedure, great care should be exercised to avoid any intravascular injection or injection under pressure. Environmental allergies should also be controlled and an immune work-up may be warranted in a patient with recurrent, acute infections.

#### Iatrogenic Disease

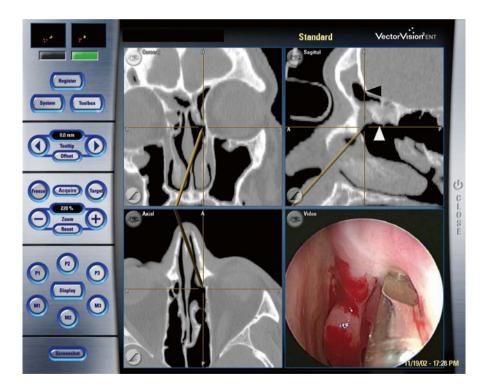
While persistent disease may be due to incomplete initial surgery, and often is corrected with a meticulous revision procedure, iatrogenic problems represent some of the most difficult cases to treat. The incidence of frontal sinusitis following FESS is unknown. Published reports over the last decade quote a 2-11 % rate of persistent frontal sinusitis symptoms with 1-5 % of patients requiring revision surgery [5]. However, this appears to be a significantly lower figure than expected, given the overall revision rate for endoscopic sinus surgery. In any case, the incidence of the iatrogenic frontal sinus may change with the increased rate of frontal sinus balloon dilation procedures now being performed [6]. While balloon technology should reduce the incidence of mucosal stripping and scarring, the limitations of balloon technology also being recognized. Studies have demonstrated at least a 12 % failure rate due to anatomical variations and the complexity of the frontal recess [7]. Cadaveric studies have also shown that misplaced guidewire insertions that lead to

false passages and mucosal stripping may be more prevalent than previously expected [8]. The long term incidence of mucocele formation from balloon dilatation is, at this point in time, unknown. This is important because studies have demonstrated that the average time between trauma or surgery and mucocele formation is approximately 17 years [9].

• Iatrogenic disease is often the result of circumferential stripping of frontal recess mucosa.

This can result in scarring and ultimately neo-osteogenesis. Neo-osteogenesis represents our most difficult challenge in revision frontal sinus surgery (Fig. 22.1). The inflamed and hardened bone is difficult to remove and often has to be drilled out to provide an adequate opening. Any procedure involving a drill creates the potential for a great amount of fibrin debris, neo-osteogenesis and stenosis, and requires more extensive post-operative debridements.

• If not meticulously addressed in the post-operative period, sinuses in which the drill is used are more likely to re-stenose.



**Fig. 22.1** Image-guided tri-planar CT scan of the frontal recess in a patient undergoing revision endoscopic frontal sinus surgery. The coronal view shows the pointer at a left lateralized middle turbinate remnant. On the sagittal view, the *white arrow* points to extensive neo-osteogenesis along the posterior frontal recess. The *black arrow* shows a type 3 frontal recess cell

A second manifestation of iatrogenic disease is mucocele formation. As noted above, mucoceles may form years after initial surgery, and can result in thinning or dehiscence of the anterior or posterior tables of the frontal sinus.

• Mucoceles are proof that long term follow-up is needed after any frontal sinus surgery, because the stenosis and obstruction that leads to the mucocele can be observed for years before the mucocele develops.

Neel et al. also clearly demonstrated the necessity of long-term follow-up in their patients undergoing a modified Lynch procedure. Their failure rate with that procedure grew from 7 % at a mean follow-up of 3.7 years to 30 % at 7 years [10].

# **Pre-operative Planning**

Once the decision has been made to perform a revision endoscopic procedure, it is imperative in the pre-operative period to review each patient's frontal sinus anatomy and determine the best procedure taking into account anatomy, amount of disease and underlying co-morbidities.

### Anatomy

From a surgical standpoint, the frontal recess can be thought of as a box with four surrounding walls. Creating a wide frontal sinusotomy requires a stepwise approach to evaluate each wall of the box.

• The best approach is to start with detailed pre-operative planning. Surgical navigation, using 1 mm axial sections reformatted into sagittal and coronal views, allows for three-dimensional analysis of the frontal recess. The surgeon should carefully scroll through the images in each of these planes until a three-dimensional concept of the regional anatomy, adjacent cells and locations of the natural drainage pathway is established and conceptualized by the surgeon.

The anterior wall of the frontal recess is addressed by the dissection of the superior uncinate and agger nasi cells. Posteriorly, the superior attachment of the ethmoid bulla and any supraorbital ethmoid cell must be opened to expose the box to its greatest anterior-posterior dimension. The anterior ethmoid artery is located along the skull base posterior to the frontal recess, typically where the dome of the ethmoid becomes horizontal. Most frequently, but not always, the anterior ethmoid artery lies posterior to the supraorbital ethmoid cell openings. Potential complications, related to a dehiscent anterior ethmoid artery, or an artery which travels in a bony mesentery below the skull base, can be evaluated prior to the operation and avoided during surgery (Fig. 22.2).

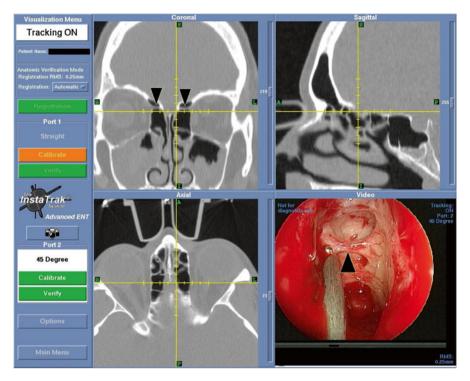


Fig. 22.2 Endoscopic and radiologic view of the anterior ethmoid artery (*black arrows*) as it courses below the skull base

Along with the anterior ethmoid artery, the skull base should be evaluated prior to revision surgery. CSF leak or injury to the skull base is more likely to occur in revision sinus surgery than in primary surgery as a result of distorted anatomy, possible dehiscence from prior surgeries, and more aggressive moves to eradicate disease and maximally enlarge the frontal recess. Adequate pre-operative planning may help to avoid these complications.

• One of the most useful pieces of information is the distance from the nasofrontal beak to the olfactory cleft. This can be evaluated on the axial image and can give the surgeon a sense of how much room he or she has in the anterior-posterior dimension.

# **Choice of Procedure**

Once the films have been reviewed, a decision should be made as to which procedure should be performed. Balloon dilatation procedures are being used with greater frequency both in the operating room and office setting, but there is predominantly level 4 evidence supporting its use [11]. In the office, it may serve a role during the postoperative period in preventing formal surgical revision in the operating room, or in the intensive care unit for critically ill patients that are poor candidates for operative procedures. In some cases, balloon dilatation may provide an alternative to a revision surgical procedure. At this time, however, the evidence supports endoscopic frontal sinus surgical procedure that has been classified by Draf into three types, based on the extent of surgery.

A Draf I procedure is an anterior ethmoidectomy with drainage of the frontal recess without touching the frontal sinus outflow tract [12]. This is best reserved for primary cases of chronic sinusitis without polyposis and without evidence of frontal sinus disease.

A Draf IIA procedure involves the removal of ethmoid cells protruding into the frontal sinus creating an opening between the middle turbinate medially and the lamina papyracea laterally. This incorporates the concept of "uncapping the egg" made popular by Stammberger, and is the most commonly utilized frontal sinus surgery [13]. The key to this procedure is the delicate removal of bony partitions with preservation of the mucosa. When done properly with the removal of small bony partitions, a Draf IIA is the adequate procedure for any frontal recess that is greater than 4 mm in the anterior-posterior dimension. As stated earlier, the majority of revision cases are secondary to remnant uncinate processes, agger nasi, and/ or frontal recess cells. Clearance of these remaining obstructions can successfully result in a patent frontal sinusotomy without the use of a drill or external incision.

A Draf IIB involves the removal of the frontal sinus floor between the nasal septum medially and the lamina papyracea laterally. In order to allow for this, the anterior portion of the middle turbinate is resected where it lies medial to the frontal sinus. Opening the sinus in this fashion involves the use of angled thru-cutting forceps and may require the use of an endoscopic drill. Although it is not usually performed as an initial procedure, the most common indications for this procedure are the presence of a narrow anterior-posterior or medial-lateral dimension, osteitic middle turbinate and/or intersinus septal cell.

The frontal intersinus septal cell occurs in the septum between the two frontal sinuses. In a review of 300 CT scans, the intersinus septal cell was present in 101 or 34 % of scans [14]. This cell may pneumatize only the lower intersinus septum or extend to the top of the frontal sinus. Utilizing the frontal sinus interseptal cell is another technique to widen the frontal recess. Removing the common wall that separate the cell from the frontal recess, and the floor of the sinus from the lamina papyracea to the middle turbinate, keeps the posterior and anterior mucosa of the frontal recess intact while enlarging the medial-lateral dimension.

A Draf III or trans-septal frontal sinusotomy involves the removal of the upper part of the nasal septum and the lower part of the frontal sinus septum, in addition to the Type IIB drainage of both frontal sinuses. Also known as a modified Lothrop procedure and median frontal sinus drainage procedure, this has been used an alternative to the frontal sinus obliteration in revision cases with significant neo-osteogenesis, narrow anterior-posterior dimension and/or significant polypoid thickening or debris. In CRS, this procedure should be generally reserved for patients who have already failed a more conservative procedure, but the long term patency rates for a well performed Draf III procedure are excellent. However, more recently, in a minority opinion, Wormald has suggested that this may be an appropriate primary procedure for patients with severe disease. In Wormald's review of 338 procedures, the Draf III procedure appeared to show a significantly reduced rate of revision surgery in patients with Samter's triad compared to less aggressive procedures [15].

# **Surgical Equipment**

Once the decision has been made to perform a revision procedure, specialized instruments should be used to maximize sound surgical technique. Each of the following aid in achieving the principles for successful frontal sinus surgery: sparing of frontal recess mucosa and accurate identification of frontal recess anatomy.

# Surgical Navigation Systems

With the advent of surgical navigation in the late 1980s, endoscopic surgeons have been increasingly utilizing this technology for intraoperative localization and preoperative planning. Fine-cut axial CT scans, often 1 mm in section, are reformatted into coronal and sagittal views and allow for greater understanding of anatomy that has been distorted by previous surgery, polypoid mucosa and/or anatomical variants. Image guidance has particular benefit in revision frontal sinus cases.

## Angled Endoscopes and Instruments

Angled instruments are essential in frontal sinus surgery. Forty five and seventy degree endoscopes allow direct visualization of the frontal recess and anterior skull base. The 45° telescope is particularly helpful because it is both easy to use because of its wide angle of view and has improved illumination.

Popularization of endoscopic techniques for frontal sinus surgery has brought about the development of specialized instruments. Powered instrument companies have devised angled drills, diamond and cutting, that may be attached to hand held microdebriders. The 70° diamond suction irrigation drill has, in particular, made a dramatic difference to this surgery. In particular, the drill reduces the amount of trauma and exposed bone during the approach, as well as decreasing the size of the septal perforation required [16]. More recently, 30,000 rpm curved diamond and cutting drills have been introduced that both carry the same advantages of mucosal preservation and dramatically reduce surgical time. There is a variety of 90° instruments designed to reach around the nasofrontal beak and into the frontal recess. Angled and malleable curettes have been devised to aid in the removal of the cap of obstructing ethmoid air cells. Revision procedures often become a methodic process of cut, remove, suction and re-examine. These specialized instruments allow for preservation of mucosa and removal of fine bony fragments that if left behind, can serve as a nidus for scarring and infection.

After the patient has been properly selected, associated disease factors have been controlled, pre-operative planning has been performed, and adequate specialized equipment has been prepared, the surgeon is finally ready for surgery.

# **Revision Frontal Sinusotomy: General Principles of Surgical Technique**

• In revision surgical procedures the anatomy is significantly distorted and landmarks such as the middle turbinate may be partially resected, making them unreliable for anatomic localization.

Accurate identification of both the medial orbital wall and the skull base is essential if the risk of complications is to be minimized (Table 22.2).

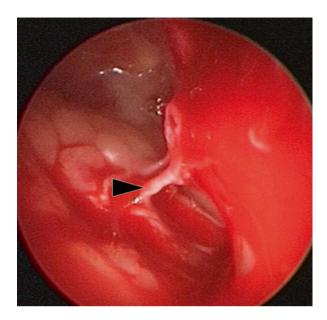
• As in all endoscopic surgical cases, it should be remembered that the skull base is usually most easily identified in the posterior ethmoid or sphenoid sinuses, where it is more horizontal and the cells are larger

Care always needs to be taken where the skull base slopes down medially towards the attachment of the middle turbinate in the region of the anterior ethmoid artery. The ethmoid roof is at its thinnest in this area, and may even be membranous in part, making it particularly vulnerable to injury. As this area is approached, it is important to stay close and parallel to the medial orbital wall while keeping in the mind the opening of the frontal sinus is most frequently medial, close to the attachment of the middle turbinate to the skull base.

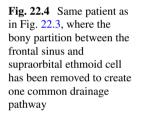
As the dissection along the skull base is carried forwards, the anterior ethmoid artery typically lies in a superior extension of the anterior wall of the bulla ethmoidalis at, or somewhat below, the skull base and courses anteriorly as it travels medially. The openings of one, or more frequently two, supraorbital ethmoid cells often lie anterior to the vessel and extend laterally and superiorly (Fig. 22.3).

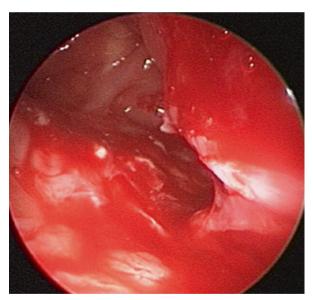
Table 22.2 Ochera	i principies of surgical technique
Accurate identificat	tion of medial orbital wall and skull base
•	tull base posteriorly in the sphenoid sinus, and then dissect from a posterior a along the skull base
Use a 45 or 70° end	loscope
Identify the anterio	r ethmoid artery as it crosses the ethmoid roof
Stay close to the m	edial orbital wall keeping in mind the opening of the frontal is often medial
Identify supraorbita	al ethmoid and frontal recess cell openings
Make sure all remn	ant osteitic bony fragments are removed from the frontal recess

	<b>Table 22.2</b>	General	principles	of surgical	technique
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**Fig. 22.3** View of the frontal sinus and supraorbital ethmoid cell from a  $70^{\circ}$  endoscope. The *arrow* points to the bony partition separating the two drainage pathways





The opening to the frontal sinus is frequently not immediately evident. Very fine malleable probes have been developed which can be utilized to gently probe the openings and help determine which of these recesses truly pass superiorly into the frontal sinus. Once the opening has been clearly identified, adjacent bony partitions may be fractured with specialized frontal sinus instruments to open the frontal sinus. Bony fragments are then teased out and redundant mucosa is trimmed with a curved microdebrider or through cutting instruments (Fig. 22.4). It is extremely important in revision surgery to not end the case until all bony partitions have been removed completely. In general, frontal sinus stents are not utilized. However, the recent development of drug eluting biodegradable implants makes the use of a small stent (Propel Mini, Intersect Ent, Menlo Park, Ca) a realistic consideration to control postoperative mucosal edema. Although drug-eluting implants have proven effective within the ethmoid cavity, their use within the frontal sinus warrants additional study. In patients where there is a significant mucosal loss, consideration can be given to placing a layer of Silastic within the frontal sinus. Woodworth originally described the use of a free mucosal graft to cover the exposed bone following a Draf III procedure and in recent years this has become more widely accepted [17].

#### **Post-operative Care**

The actual surgery to open a frontal sinus is often the easy part in the management of frontal sinus disease. The post-operative period is where much of the difficulty lies.

 Revision frontal sinusotomies must be carefully and diligently examined following surgery

A failure to actively debride the recess, ensure its patency and suction contaminated blood and mucus from the sinus is a recipe for re-stenosis and failure. To do this in a setting of an awake, often anxious patient, with topical analgesia alone, makes this portion of the process very challenging, but can be aided by the careful application of topical cocaine solution to the site. Where local debridements are necessary, the region of the frontal recess can be infiltrated with 1 % xylocaine with 1:100,000 adrenaline using a bent 2" 27 gauge needle and a small syringe.

The timing of the first post-operative debridement varies with individual surgeon's preference. Some debride on post-operative day 1, while others wait for an additional 3–7 days. It is advantageous to have a full set of frontal sinus instruments available in the clinic. This is coupled with angled suctions that are long and curved enough to reach into the frontal sinus. Debridements should be aimed at clearing away fibrin debris and any loose bony fragments, while keeping trauma to the surrounding mucosa to a minimum. If a drug eluting implant is placed, the remnants are usually removed at 3 or 4 weeks postoperatively.

While the mechanical care of the frontal sinusotomy is important to prevent restenosis, medical management of the disease state is essential to long-term success. In a patient with significant polypoid edema, post-operative oral steroids can be used to keep the edema to a minimum. Intranasal steroids sprayed in the Moffit or head down position, can help with delivery to the frontal recess. Post-operative antibiotics should also be given in an infectious setting, and antibiotics with good bone penetration should be used in patients with evidence of neo-osteogenesis.

This routine of mechanical debridement and post-operative medication should be continued on a weekly basis until the mucosa of the frontal recess is healed. Once the sinusotomy is secure, routine surveillance by nasal endoscopy should continue for the life of the patient.

# Conclusion

Revision endoscopic frontal sinus surgery remains a great challenge to all who practice sinus surgery. The last few decades have brought about a specialization of instruments and techniques aimed at treating frontal sinus disease endoscopically and avoiding frontal sinus obliteration. Continued investigations will further elucidate the long term utility of balloon dilatation procedures. Surgical technique aside, the most important decisions are still made in the office. These entail assessing whether or not the patient is a good surgical candidate, the appropriate choice of endoscopic procedure given the individual patient's anatomy and disease process, and the institution of aggressive adjuvant medical therapy.

# References

- 1. Chiu AG, Vaughan WC. Revision endoscopic frontal sinus surgery with surgical navigation. Otolaryngol Head Neck Surg. 2004;130(3):312–8.
- 2. Anderson TD, Kennedy DW. Surgical intervention for sinusitis in adults. Curr Allergy Asthma Rep. 2001;1:282–8.
- 3. Bhattacharyya N. The completely opacified frontal or sphenoid sinus: a marker of more severe disease in chronic rhinosinusitis? Laryngoscope. 2005;115(12):2123–6.
- 4. Mendelsohn D, Jeremic G, Wright ED, et al. Revision rates after endoscopic sinus surgery: a recurrence analysis. Ann Otol Rhinol Laryngol. 2011;120(3):162–6.
- Orlandi RR, Kennedy DW. Revision endoscopic frontal sinus surgery. Otolaryngol Clin N Am. 2001;34(1):77–90. Review.
- 6. Psaltis AJ, Soler ZM, Nguyen SA, et al. Changing trends in sinus and septal surgery, 2007 to 2009. Int Forum Allergy Rhinol. 2012;2(5):357–61.
- Heimgartner S, Eckardt J, Simmen D. Limitations of balloon sinuplasty in frontal sinus surgery. Eur Arch Otorhinolaryngol. 2011;268(10):1463–7.
- Brenner PS, Abadie WM, Weitzel EK, et al. Unexpected consequences of transnasal balloon dilation of the maxillary ostium. Int Forum Allergy Rhinol. 2011;1(6):466–70.
- 9. Scangas GA, Gudis DA, Kennedy DW. The natural history and clinical characteristics of paranasal sinus mucoceles: a clinical review. Int Forum Allergy Rhinol. 2013;0:1–6.
- Neel 3rd HB, McDonald TJ, Facer GW. Modified Lynch procedure for chronic frontal sinus diseases: rationale, technique and long-term results. Laryngoscope. 1987;97(11):1274–9.
- Batra PS. Evidence-based practice: balloon catheter dilation in rhinology. Otolaryngol Clin N Am. 2012;45(5):993–1004.
- 12. Weber R, Draf W, Kratzsch B, et al. Modern concepts of frontal sinus surgery. Laryngoscope. 2001;111(1):137–46.
- 13. Stammberger HR. Functional endoscopic sinus surgery: the Messerklinger technique. Philadelphia: BC Decker; 1991. p. 60–87.
- 14. Merrit R, Bent JP, Kuhn FA. The intersinus septal cell. Am J Rhinol. 1996;10(5):299-302.
- 15. Bassiouni A, Wormald PJ. Role of frontal sinus surgery in nasal polyp recurrence. Laryngoscope. 2013;123(1):36–41.
- Chandra RK, Schlosser R, Kennedy DW. Use of the 70-degree diamond burr in the management of complicated frontal sinus disease. Laryngoscope. 2004;114(2):188–92.
- 17. Conger Jr BT, Riley K, Woodworth BA. Otolaryngol Head Neck Surg. 2012;146(4):664-8.

# Chapter 23 The Supraorbital Ethmoid Cell

#### Brett T. Comer and Stilianos E. Kountakis

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#### **Core Messages**

- The supraorbital ethmoid cell lies posterior and lateral to the frontal sinus, anterior to the anterior ethmoid artery, and drains lateral or posterior to the true frontal ostium
- Misidentification or lack of identification of the supraorbital ethmoid cell may lead to iatrogenic injuries intraoperatively or long-term failure from functional endoscopic sinus surgery
- Image-guided navigation may be beneficial not only for verification of supraorbital ethmoid cell location during the course of dissection, but also for intraoperative teaching of anatomical relationships to residents and fellows
- The "nipple sign" or "orbital beak" can help identify anterior ethmoid artery location, even in patients with distorted anatomy or significant inflammatory changes

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_23

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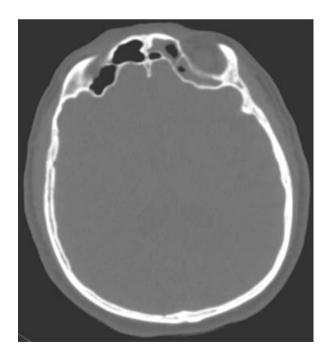
# Introduction

The frontal sinus grows as an extension from the anterior ethmoid air cells, between the first and third ethmoturbinals [11]. Variations of frontal sinus anatomy within patients are common [1], as are variations among patients. The frontal sinus outflow region also has a multitude of possible anatomic pneumatization patterns, as previously described [8].

• The supraorbital ethmoid cell (SOEC) is defined as pneumatization of the orbital plate of the frontal bone and its ostium drains in the frontal recess.

Anatomically, the SOEC lies posterior and lateral to the frontal sinus, anterior to the anterior ethmoidal artery (AEA), and drains lateral or posterior to the true frontal ostium (Fig. 23.1). Radiographically, the SOEC may be associated with septations of the frontal sinuses [5] (Fig. 23.2), may be associated with mucocele formation [2], or may contribute to orbital proptosis [4] (Figs. 23.3 and 23.4).

During surgical dissection, the SOEC ostium can easily be confused with the frontal sinus ostium, and therein lies the crux of the challenges this anatomic variation can present for even the most experienced rhinologic surgeons: misidentification of the SOEC ostium may lead to intraoperative complications such as penetration of the anterior skull base with cerebrospinal fluid leak or hemorrhage due to damage to the AEA. Conversely, lack of recognition of the SOEC intraoperatively can lead to long-term FESS failure because of failure to open and connect the SOEC ostium to the frontal recess [2] (Fig. 23.5).



**Fig. 23.1** Axial CT image showing a no chronic sinus disease in the right frontal sinus and SOEC, and partial opacification of the left frontal sinus and left SOEC. The patient is slightly canted in this CT scan, but note the position of the SOECs relative to the orbit in this case

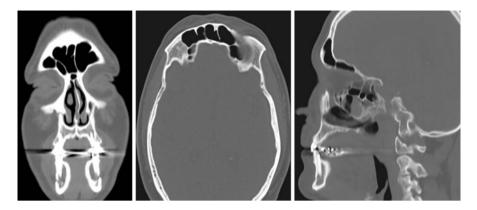


Fig. 23.2 Coronal and triplanar CT imaging demonstrating the association of frontal septations with SOECs

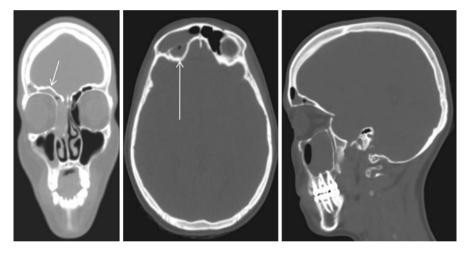
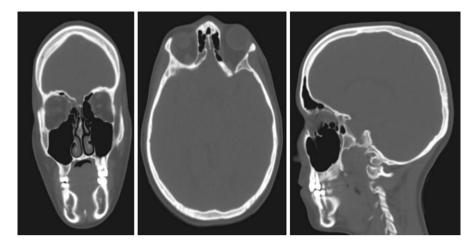
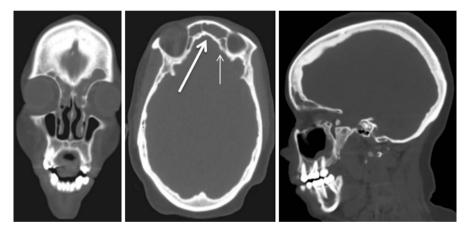


Fig. 23.3 Triplanar Imaging of a 21-year-old patient who presented with acute right periorbital edema and proptosis, showing an opacified right SOEC (*arrow*)

Prior studies estimating the prevalence of SOECs have noted wide variations in ethnicity. For example, Asian individuals were found to have lower relative prevalence, as just 2.6 % of Korean subjects [3] and 5.4 % of Chinese subjects [15] had SOECs. Caucasians were found to have a much higher prevalence, as different studies found SOECs rates of 62 % [8], 65 % [3], and 69 % [5]. In one study, African Americans were found to have a 36 % rate of SOECs [5]. Finally, older cadaveric studies noted a relatively low rate of SOECs [6, 13, 14]. Because of the everincreasing use of triplanar CT imaging for sinus anatomy surgery, it is anticipated that further research will better define the rates of SOECs among different ethnic groups.



**Fig. 23.4** Triplanar Imaging of the same 21-year-old patient after 6 weeks of medical therapy, including nasal saline rinses, flunisolide, PO steroid taper, and oral antibiotics. Note complete resolution of disease except for the isolated right SOEC



**Fig. 23.5** Coronal and sagittal CT cuts of a 49-year-old patient with CRS who failed three prior FESS, showing the left frontal sinus (*large arrow*) in relation to a SOEC (*small arrow*)

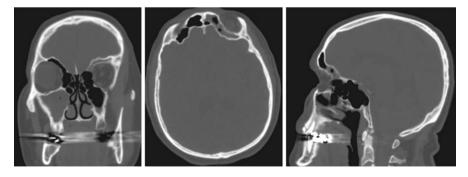
#### Key Points Related to Preoperative Imaging for Patients with SOECs are:

- The use of image-guidance protocol for the preoperative imaging
- Studying post-surgical anatomic changes from prior surgeries
- Identifying relationship of the cribriform plates to the skull base and to the middle turbinate
- Identifying the location of the anterior ethmoid artery and its relationship to the skull base

#### **Radiographic Presentation**

Fine-cut triplanar CT imaging is standard imaging for any patient with suspected chronic rhinosinusitis. Our institution's protocol is to use 1.0 mm cuts, as this results in an excellent 3D model of the patient should the CT imaging subsequently be used for image-guided surgery. Because surgical dissection occurs in the frontal outflow region and in the frontal sinus when dealing with SOECs, the IGS-compatible imaging serves multiple purposes. First, it allows for triplanar imaging both preoperatively and intraoperatively. When working with fellows and residents, triplanar imaging can be advantageous for teaching anatomy and its variations prior to initiation of the procedure. Specifically, nuances of the frontal outflow relationships can be identified (Fig. 23.6). Secondly, the intraoperative use of IGS has the obvious advantage of serving to verify visualized anatomy during the course of dissection. In patients with considerable edema and polyps near the skull base, the IGS system can serve as one additional safety measure to help verify anatomic locations in especially challenging operative cases.

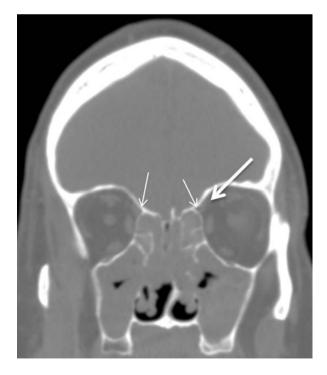
Several different nomenclature systems exist for the anatomic variations of the frontal outflow region [8]. The SOEC is but one of these variations and it behooves any sinus surgeon to study extensively the frontal outflow region on preoperative imaging in order to reduce the risk of intraoperative complications, such as skull base penetration. The SOEC may initially appear as a septated frontal sinus [5], but examination of triplanar CT imaging will help to delineate this. With regards to the frontal outflow region, it is this rhinologist's preference to first study the coronal images serially from anterior-to-posterior, and then posterior-to-anterior, taking care to note (1) the bulla ethmoidalis and any potential suprabullar cells, (2) the uncinate process attachment, (3) the depth and symmetry of the cribriform plate, (4) the course of the frontal outflow tract, and finally (5) the indication of the presence of any SOEC(s).



**Fig. 23.6** Triplanar CT imaging of a 65-year-old male with left frontal sinus and SOEC chronic rhinosinusitis. Note that on coronal imaging, it is possible to see the relationship between the SOEC, the AEA, and the skull base

Additionally, one should make note of the location of the AEA, which typically can be identified by looking for the "nipple sign," or "orbital beak" on coronal imaging. The AEA foramen is usually at the junction of the superior and medial orbital walls and also represents the level of the frontoethmoidal suture line. The superior oblique muscle also tends to be in close proximity to this location. These structures tend to be fairly constant, even in previously operated sinonasal passages or in the presence of significant sinonasal pathology [7] (Fig. 23.7). After identifying the AEA foramen, which typically can be done in >95 % of coronal CT sinus images [7, 9, 10], one then looks to identify and trace the route of the AEA itself. This task is typically more difficult to do, especially in the presence of considerable inflammatory disease. Note whether the AEA appears to be encased in bone or whether it appears to hang in a mesentery below the skull base. Some have noted that the presence of suprabullar cells tends to be associated with the AEA being located further from the skull base [7]. Others have noted that the presence of supraorbital cells is associated with the AEA being further from the skull base. Based on the data available, it can generally be concluded that the greater the amount of pneumatization of the ethmoid and frontal region, the greater the likelihood that the AEA will be positioned away from the skull base: thus, the immense importance of proper preoperative evaluation of CT imaging in order to reduce the risk of iatrogenic complications [12]. Once the AEA is properly identified, the relationship of a SOEC, if present, becomes clear, as does the relationship of the SOEC to the frontal outflow tract.

Fig. 23.7 Coronal CT image of a patient with Samter's triad showing the bilateral locations of the AEC foramen. Note the relationship of the superior oblique muscle (large arrow) to the foramen (small arrow) on each side. Note also that despite anatomic changes from multiple prior surgeries, as well as extensive inflammatory disease, the AEA foramen location and its relationship to the skull base is easily identifiable on each side



During the Course of Dissection of SOECs, Key Points Include:

- · Sparing mucosa in the frontal outflow region as much as feasible
- Consider using an angled 30-degree or 45-degree scope for dissection
- Use image-guidance to verify, not identify, anatomy
- Be certain to take down any boney septation between the SOEC and true frontal outflow tract to facilitate patency and topical medication delivery postoperatively

# **Surgical Considerations**

Patients who have frontal sinus disease or SOECs often times have had prior FESS. The frontal outflow region challenges even the most experienced surgeon at times, and the presence of SOECs may further complicate the situation.

• The SOEC ostium drains in the frontal recess, posterior and lateral to the frontal sinus ostium and anterior to the anterior ethmoidal artery.

On triplanar CT imaging such as that used during image-guided surgery, the SOEC may appear as a septation of the frontal sinus. Preoperatively, it is imperative to identify the presence of the SOEC on imaging, so that intraoperatively the boney medial-anterior wall of the SOEC can be removed, resulting in better patency of the frontal outflow region.

Nasal preparation for dissection involving the SOECs is performed by placement of oxymetazoline-soaked pledgets in the nasal passages. The IGS system is then set up and calibrated, and the patient is draped in a standard fashion for sinus surgery. After approximately 15 min, the pledgets are removed, and the standard steps of FESS, including uncinectomy, maxillary antrostomy, and ethmoidectomy are performed as necessary. Working from a general posterior-to-anterior direction, the frontal outflow region is approached. When working at the skull base, it is this author's preference to first change to a 45-degree 4.0 mm reverse-post rigid endoscope, and also to verify anatomic locations using an image-guided curved suction. The true frontal sinus is cannulated with the suction, if possible. The suction is then used to locate the most superior aspect of the lamina papyracea and the medial portion of the SOEC. In many cases, the location of the anterior ethmoid artery can be identified visually; occasionally, an image-guided probe must be used to verify location. The inferior medial wall of the SOEC is gently punctured with a probe, curved suction, or J-curette. A J-curette is then used to gently palpate the skull base and remove the anterior medial wall of the SOEC by sweeping in a posterior-toanterior direction. Multiple sweeps may be necessary to completely remove the party wall, and the surgeon should remember not to sweep too far medially in those patients with low-lying cribriform plates. As with any surgery in the frontal sinus or frontal outflow region, it is important to reduce the risk of circumferential stenosis by sparing mucosa as much as possible during the dissection. With some SOECs, it

is necessary to remove the anterior wall relatively far laterally, such that the surgeon is working well lateral and superior to the sagittal plane of the lamina papyracea. Because space for dissection in this region may be limited, this author has found that sweeping a 90-degree ball probe or a frontal sinus seeker may be helpful in removing the more lateral portions of the common boney septation between the SOEC and frontal sinus. An alternative option is to use a giraffe forceps for dissection. In rare cases, an anterior/superior septectomy may be used in order to facilitate far lateral dissection by placing dissection instruments contralateral to the SOEC and working across the midline. Given the multiple variations of angled endoscopes and angled dissection instruments now available, this situation is relatively uncommon. Additionally, care is taken not to dissect too far posteriorly, as the AEA is immediately posterior to the SOEC and can be easily injured, especially if it hangs in a mesentery below the skull base. Should the AEA be damaged during surgery, an endoscopic bipolar cautery may be used to achieve hemostasis in most cases. Occasionally, when a larger-diameter AEA retracts into the orbit, a Lynch incision must be made to place a surgical clip on the transected artery. Finally, intraoperative penetration of the skull base resulting in CSF leak may be repaired with mucosal grafting and/or multilayer repair.

#### Keys to Appropriate Postoperative Care Include:

- · At least TID nasal saline irrigations, aiming superiorly in the nasal passages
- A burst-and-taper of oral steroids
- · Meticulous postoperative debridement
- · Consider adjunctive therapies in patients with asthma

#### **Postoperative Care**

Postoperatively, any patient who has extensive dissection in the frontal outflow region or who has extensive polyp disease is placed on an oral steroid burst and taper. This author prefers to use a 27-day taper, starting at 40 mg daily of prednisone. Patients are counseled on the theoretical risks of long-term oral steroid use, particularly avascular necrosis of the hips. Patients are started on TID nasal saline rinses starting on postoperative day 1, with particular attention to aiming the rinses superiorly in the nasal cavity. Debridement is performed 1 week after surgery, and, based on pathology results, simple steroid nasal spray is started at this point for those patients with low eosinophil counts vs. budesonide rinses (1 respule in 3 oz of water) in the head-down position for those patients with higher eosinophil counts. Any patient with asthma is maintained perioperatively on montelukast or zifirleukast. Approximately 1 week after surgery, meticulous debridement of the sinonasal passage is performed as necessary in order to reduce the risk of postoperative synechiae formation and also to provide better patency for the frontal outflow region.

# Conclusion

Rhinologic surgeons should be aware that the presence of multiseptations within the frontal sinus on sinus CT may indicate the presence of supraorbital ethmoid air cells. Identification of these cells in patients with frontal recess disease may prevent complications and ensure that the appropriate disease sinus cells will be targeted.

# References

- Cakur B, Sumbullu MA, Durna NB. Aplasia and agenesis of the frontal sinus in Turkish individuals: a retrospective study using dental volumetric tomography. Int J Med Sci. 2011;8:278–82.
- 2. Chiu AG, Vaughan WC. Management of the lateral frontal sinus lesion and the supraorbital cell mucocele. Am J Rhinol. 2004;18(2):83–6.
- 3. Cho JH, Citardi MJ, Lee WT, et al. Comparison of frontal pneumatization patterns between Koreans and Caucasians. Otolaryngol Head Neck Surg. 2006;135(5):780–6.
- Comer BT, Kincaid NW, Kountakis SE. The association between supraorbital ethmoid air cells and orbital proptosis in patients with chronic rhinosinusitis. Int Forum Allergy Rhinol. 2013;3:147–9.
- Comer BT, Kincaid NW, Smith NJ, et al. Frontal sinus septations predict the presence of supraorbital ethmoid cells. Laryngoscope. 2013;123(9):2090–3.
- Dixon FW. The clinical significance of the anatomical arrangement of the paranasal sinuses. Ann Otol Rhinol Laryngol. 1958;67:736–41.
- Joshi AA, Shah KD, Bradoo RA. Radiological correlation between the anterior ethmoidal artery and the supraorbital cell. Indian J Otolaryngol Head Neck Surg. 2010;62(3):299–303.
- 8. Lee WT, Kuhn FA, Citardi MJ. 3D computed tomographic analysis of frontal recess anatomy in patients without frontal sinusitis. Otolaryngol Head Neck Surg. 2004;131:164–73.
- 9. McDonald SE, Robinson PJ, Nunez DA. Radiological anatomy of the anterior ethmoidal artery for functional endoscopic sinus surgery. J Laryngol Otol. 2008;122:264–7.
- Monjas-Canovas I, Garcia-Garrigos E, Arenas-Jimenez JJ, et al. Radiological anatomy of the ethmoidal arteries: CT cadaver study. Acta Otorrinolaringol Esp. 2011;62(5):367–74.
- Schaeffer JP. The genesis, development, and adult anatomy of the nasofrontal region in man. Am J Anat. 1916;20:125–45.
- 12. Simmen D, Raghavan U, Briner HR, et al. The surgeon's view of the anterior ethmoid artery. Clin Otolaryngol. 2006;31:187–91.
- Van Alyea OE. Ethmoid labyrinth: anatomic study, with consideration of the clinical significance of its structural characteristics. Arch Otolaryngol Head Neck Surg. 1939;29:881–902.
- Van Alyea OE. Frontal cells: an anatomic study of these cells with consideration of their clinical significance. Arch Otolaryngol Head Neck Surg. 1941;34:11–23.
- Zhang L, Han D, Wentong G, Tao J, Wang X, Li Y, Zhou B. Computed tomographic and endoscopic analysis of supraorbital ethmoid cells. Otolaryngol Head Neck Surg. 2007;137:562–8.

# **Chapter 24 "Above and Below" FESS: Simple Trephine with Endoscopic Sinus Surgery**

Ankit M. Patel and Winston C. Vaughan

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#### **Core Messages**

- In most cases of frontal sinus disease, endoscopic approaches are favored; however, in some situations where an endoscopic approach is insufficient, an "above and below" approach may be suitable, serving as an alternative to more invasive procedures
- Situations where this may be considered include large or laterally-based frontal sinus cells, lesions of the frontal sinus lateral to the plane of the lamina papyracea, trauma, revision surgery, and complicated infection
- Endoscopic frontal sinusotomy is performed first, followed by trephination

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# Background

Historically, frontal sinus disease was treated using external approaches, with the first written reports of frontal trephination dating back to the late 1800s. In 1921, Lynch reported on his experience and technique of external frontoethmoidectomy. In the 1950s and 1960s, Montgomery popularized the osteoplastic flap approach with obliteration of the frontal sinus.

In the late 1970s, Messerklinger and Wigand introduced endoscopic sinus surgery. Since that time, increased emphasis has been placed on atraumatic, mucosal-sparing endoscopic techniques that incorporate the natural drainage pathways of the paranasal sinuses–"functional" endoscopic sinus surgery (FESS). This led to improved healing, preservation of the mucociliary transport, and better results. In the mid-1980s, image-guided surgery was introduced.

Over the last two decades, there have also been tremendous advances in imaging. With these advances in imaging, knowledge of endonasal anatomy, instrumentation, and image-guided surgery, there has been an overwhelming move away from external approaches toward minimally invasive endoscopic approaches for frontal sinus surgery [1–6]. Functional endoscopic sinus surgery is now considered the first-line approach for frontal sinus disease. Table 24.1 summarizes the major approaches to the frontal sinus most often used.

However, there are cases when the endoscopic technique itself is insufficient. In these cases, an external approach with frontal sinus trephination (above), along with endoscopic sinus approach (below) can provide improved visualization and allow for more precise surgery. This technique is especially useful for cases where endoscopic surgery is insufficient, but the osteoplastic flap approach is too aggressive. These situations may include cases where there are large or laterally-based frontal cells that cannot be approached safely endoscopically. A lesion in the frontal sinus that is lateral to the plane of the lamina papyracea on preoperative coronal CT scan may suggest the need for an "above and below" approach. Potential applications for this combined approach are listed in Table 24.2.

Anterior ethmoidectomy Frontal sinusotomy
Frontal sinus rescue procedure
"Above and below FESS" (trephine+endoscopic surgery) Unilateral "frontal sinus drillout"
Endoscopic modified Lothrop, Transseptal frontal sinusotomy
External ethmoidectomy/Lynch approach
Osteoplastic flap without obliteration
Osteoplastic flap with obliteration

Table 24.2 Relative         indications for "Above         and Below" FESS	Electively, for visualization to facilitate endoscopic frontal sinusotomy
	Inability to completely address disease endoscopically:
	Laterally-based frontal sinus lesions
	Type III or IV frontal cell, which cannot be addressed endoscopically
	Large tumors or inflammatory lesions involving frontal sinus, including:
	Osteoma
	Inverted papilloma
	Fibrous dysplasia
	Trauma with distorted frontal recess or need to evaluate posterior frontal wall
	Revision cases with extensive scarring or neo-osteogenesis
	Distorted anatomy in the frontal recess
	Pott's puffy tumor

#### Technique

Decongestant-soaked pledgets are placed in the nasal cavity. The image-guidance system, if being used, is calibrated and verified using known landmarks. Image-guided systems can also provide a guide for the initial brow incision and external entry site. If image-guidance is not being used, the position and size of the frontal sinus is confirmed on preoperative CT scan in relation to the supraorbital rim or with 6-foot Caldwell templates. Typically, incision and trephination location will be through the medial eyebrow at the supraorbital rim without shaving this region.

The endoscopic portion of the surgery is done first. A complete uncinectomy is performed. Superiorly, a complete uncinectomy will create additional space for endoscopic work as well as help to create a larger frontal sinus outflow drainage pathway. The superior uncinate process may attach to the middle turbinate, lamina papyracea, or skull base. Review of preoperative CT scan films will identify its attachment point.

Maxillary antrostomy is then performed to serve as a landmark. The ethmoid bulla may then be removed via the retrobullar recess. Superiorly, this is traced to the skull base. The lamina papyracea should be identified and preserved. The anterior ethmoid artery may often be identified at the skull base at this point as well. Preoperative review of coronal CT scans will reveal a medial dimpling of the lamina papyracea at the location of the anterior ethmoid artery. The artery may be dehiscent or coursing from medial to lateral at a position inferior to the skull base. In both these instances, the artery is at risk for injury.

If complete sphenoethmoidectomy is planned, it may be performed at this time, with removal of posterior ethmoid cells and sphenoidotomy. The skull base should

be identified posteriorly, at the sphenoid face. It then is traced from posterior to anterior with removal of ethmoid cells along the skull base. If complete sphenoethmoidectomy is not necessary, then dissection may stop at the basal lamella, which is traced to the skull base.

*Key landmarks should always be reconfirmed for frontal recess dissection.* These are:

- · Lamina papyracea medially
- Skull base superiorly
- Anterior ethmoid artery superiorly and posteriorly, which marks the start of the frontal recess
- The middle turbinate and its attachment to the skull base
- The nasofrontal bone/beak

The agger nasi cell, which is present in a majority of patients, should be identified. Endoscopically, it will appear as a bulge of the lateral nasal wall at the junction of the lateral nasal wall and the middle turbinate. This must be removed downward (uncapping the egg) in its entirety. Next, the frontal recess is opened with mucosal preservation. Any frontal recess cells, supraorbital cells, and intersinus cells are opened endoscopically. Review of sagittal preoperative CT scans or image-guided scans is critical to maximize the diameter of the frontal sinus drainage pathway. The frontal recess can then be enlarged using a combination of curved mushroom punches, giraffe forceps, seekers and limited use of microdebriders. The mucosa of the frontal sinus should be preserved as much as possible to maintain the functional nature of FESS.

Once the endonasal frontal sinusotomy has been completed to its full extent, then the external approach is begun. Sometimes, due to tumor, trauma, previous surgery, or the patient's anatomy, endoscopic frontal sinusotomy cannot be completed endoscopically. In these cases, as much as possible of the previously described dissection is performed in a safe fashion. Trephination and endoscopic visualization through the trephine may also facilitate further dissection from below.

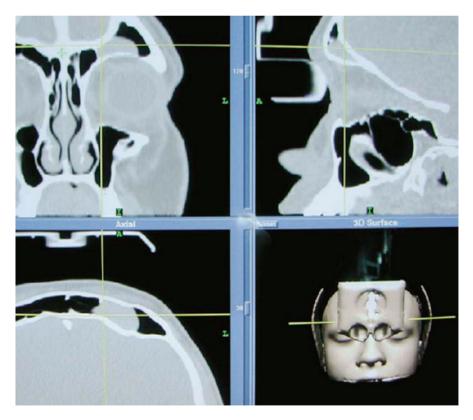
The external approach field is now prepped. If image guidance is being used, it is used to confirm the optimal eyebrow incision and frontal sinus entry point. Lidocaine with epinephrine is used to infiltrate the eyebrow incision. A 1–2-cm incision is carried through the medial eyebrow. The incision should be beveled to parallel the hair shafts of the eyebrow. No electrocautery should be used in the superficial dermis, to prevent injury to hair follicles. Bipolar cautery or pressure is less traumatic.

A self-retaining retractor is placed into the incision. The incision is carried down to bone. Deeper hemostasis is carefully achieved with bipolar cautery. Next, a 4-mm drill bit is used to perform the external trephine. The trephine may be enlarged using Kerrison rongeurs. Angled endoscopes (adult or pediatric) are used to visualize the frontal sinus through the trephine. The remaining pathology of the frontal sinus may then be addressed via the trephine, with the trephine enlarged (max: 6–8 mm) to accommodate both endoscope as well as instrumentation.

If the frontal sinus outflow tract is still not seen endonasally, the frontal sinus can be irrigated through the trephine. The endoscope is used within the middle meatus to visualize the draining irrigation fluid (this can be colored with methylene blue). This will facilitate further dissection. The endoscope is now placed back through the trephine, and angled instruments from within the nose are used to complete frontal sinusotomy. If necessary, a stent may be placed upon completion of the above-and-below procedure from below and visualized from above. The external incision is closed in layers using absorbable suture for deep tissues and permanent 5–0 sutures for the skin.

#### **Illustrative Case**

This patient has a laterally-based frontal sinus mucocele with left forehead pain, and has failed medical treatment. There is a large obstructing type III frontal cell. Because of the large size of the frontal cell, the patient was counseled regarding the possible need for trephination in conjunction with FESS. Intraoperatively, the lateral wall of the type III frontal cell could not be sufficiently opened endoscopically from below. "Above and below" FESS with the addition of a simple trephine was performed, to remove more of the lateral border of the type III frontal cell and drain the mucocele. Figures 24.1, 24.2, 24.3, 24.4, 24.5, 24.6, 24.7, 24.8, and 24.9 illustrate the anatomy and technique.



**Fig. 24.1** A laterally-based mucocele, symptomatic and persistent despite medical management. A large, obstructing type III frontal cell is present

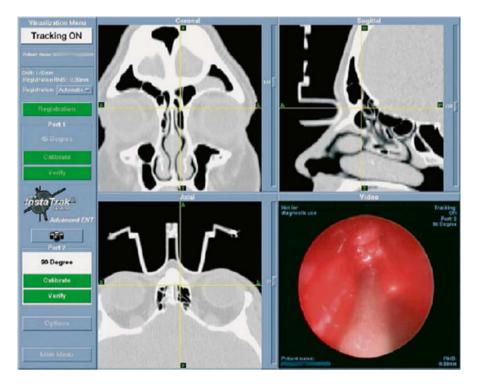


Fig. 24.2 Endoscopic approach, at the base of the type III frontal cell, above agger nasi and at the 'beak'

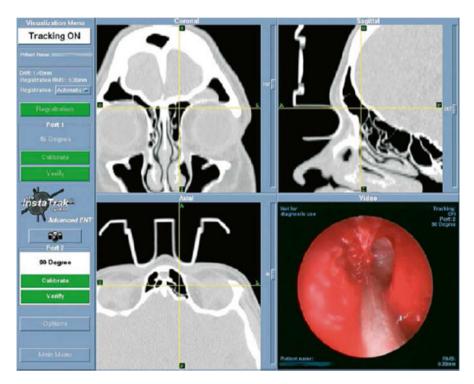
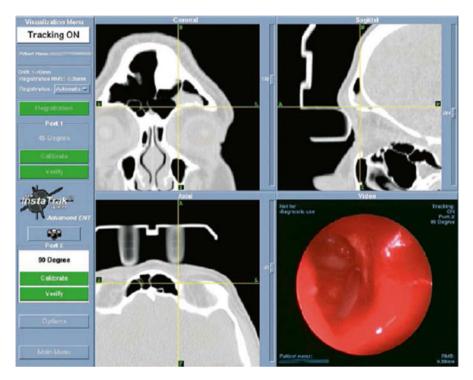


Fig. 24.3 Cross-hairs depict dissection of the type III frontal cell medially



**Fig. 24.4** Laterally-based mucocele, endoscopic view. Endonasal instrumentation is insufficient to take down the lateral septation sufficiently and drain the mucocele. At this point, trephination is needed to help facilitate more complete surgery

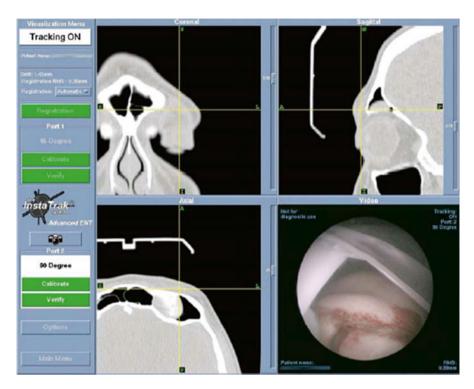
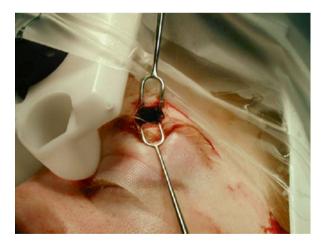


Fig. 24.5 External approach. Image-guidance is used to confirm incision site and entry point of trephination through the medial eyebrow



**Fig. 24.6** Trephination incision. Alcohol-prepped image-guidance headset is in place. This may be retracted and put back into position as needed



Fig. 24.7 Reverse  $70^{\circ}$  endoscope through trephine site. Curved ( $90^{\circ}$ ) giraffe forceps used endonasally under endoscopic visualization from above



**Fig. 24.8** Angled endoscope through trephine looking down into recess from above. Angled image-guidance suction from below, guided by endoscope from above



**Fig. 24.9** Additional instrumentation for frontal sinus trephination

# Conclusion

While endoscopic approaches are preferred for management of disease of the frontal sinus, in some situations, transnasal techniques alone will not allow sufficient access to the frontal sinus. One alternative in these cases to more aggressive open approaches is the "above and below" approach utilizing a small trephination to assist the dissection. This technique allows access to lesions located cephalad and laterally in the frontal sinus, and may also be beneficial in the setting of trauma, revision surgery, and complicated acute sinusitis. This simple technique is well-tolerated by patients and may be easily incorporated into the rhinologist's practice.

# References

- 1. Benoit CM, Duncavage JA. Combined external and endoscopic frontal sinusotomy with stent placement: a retrospective review. Laryngoscope. 2001;111(7):1246–9.
- 2. Bent 3rd JP, et al. Combined endoscopic intranasal and external frontal sinusotomy. Am J Rhinol. 1997;11(5):348–54.
- 3. Chiu AG, Vaughan WC. Revision endoscopic frontal sinus surgery with surgical navigation. Otolaryngol Head Neck Surg. 2004;130(3):312–8.
- 4. El-Silimy O. Combined endonasal and percutaneous endoscopic approach to Pott's puffy tumour. Rhinology. 1996;34(2):119–22.
- Gallagher RM, Gross CW. The role of mini-trephination in the management of frontal sinusitis. Am J Rhinol. 1999;13(4):289–93.
- Gerber ME, Myer 3rd CM, Prenger EC. Transcutaneous frontal sinus trephination with endoscopic visualization of the nasofrontal communication. Am J Otolaryngol. 1993;14(1):55–9.

# Chapter 25 Endonasal Frontal Sinus Drainage Type I–III According to Draf

## **Wolfgang Draf**

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_25

#### **Core Messages**

- The Endonasal Type I–III Drainages allow the surgeon to adapt the frontal sinus surgery to the underlying pathology
- From type I-III upwards, surgery is increasingly invasive
- The type III median drainage [2] is identical to the endoscopic modified Lothrop procedure [3]
- The concept of endonasal drainage of the frontal sinus implicates preservation of bony boundaries of frontal sinus outlet, in contrast to the classic external frontoorbital procedure [9, 11, 16, 23]. This means less danger of shrinking and reclosure with development of mucocele. It is a surgical strategy, not just a technique. The frontoorbital external operation should not be used anymore for treatment of inflammatory diseases
- When the type III drainage is technically not possible (anterior-posterior diameter of the frontal sinus less than 0.8 cm) or has failed, osteoplastic frontal sinus obliteration must be considered

# Introduction

Endonasal surgery of the paranasal sinuses began, apart from a couple of earlier reports, some 100 years ago [4, 5, 10, 24, 25]. Only a few skilled surgeons have been able to perform endonasal ethmoidectomy and adequate drainage of the frontal sinus using just a headlight and the naked eye, whereas others created serious complications such as CSF leak, meningitis, brain abscess, and encephalitis ending in the pre-antibiotic era mostly with the death of the patient. This is why for decades, until the 1970s, endonasal sinus surgery was not accepted in most leading institutions.

The renaissance of endonasal surgery was due to several advances:

- · New optical aids such as the microscope and endoscope
- Improved understanding of the physiology and pathophysiology of nasal and paranasal sinus mucosa
- Patients no longer accepting the sometimes serious sequelae of external operations in addition to an unsatisfactory outcome Remarkable progress in anesthesiology providing the endonasal surgeon with an almost bloodless field

Between 1980 and 1984, an endonasal surgical concept with different degrees of frontal sinus opening was worked out and intensively tested before being published [2].

With increasing experience and referrals of difficult frontal sinus cases, it became obvious that not all problems can be solved via an endonasal route. Therefore the osteoplastic obliterative frontal sinus operation [27] was included in the concept, in order to deal with all different kinds of frontal sinus problems. In difficult revision cases, the endonasal operation sometimes has to be combined with the osteoplastic, mostly obliterative procedure [2].

#### **Operative Technique, Indications**

For the endonasal frontal sinus, an operation using some type of general anesthesia is required. In addition, topical decongestion helps to provide a dry field.

Surgery on the frontal recess is usually preceded at least by an anterior, more often than not by a complete ethmoidectomy. Exceptions are those cases where a complete ethmoidectomy has already been performed. It is important to remove agger nasi cells and to visualize the attachment of the middle turbinate medially, the lamina papyracea laterally, and the anterior skull base with the anterior ethmoidal artery superiorly.

#### *Type I: Simple Drainage (Fig. 25.1a)*

*The type I drainage* is established by ethmoidectomy including the cell septa in the region of the frontal recess. The inferior part of Killian's infundibulum and its mucosa is not touched. This approach is indicated when there is only minor pathology in the frontal sinus and the patient does not suffer from 'prognostic risk factors' like aspirin intolerance and asthma, which are associated with poor quality of mucosa and possible problems in outcome (Table 25.1). In the majority of cases the frontal sinus heals because of the improved drainage via the ethmoid cavity [10].

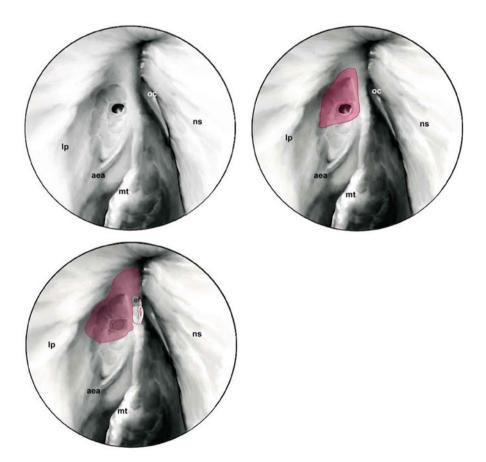
#### *Type II a/b: Extended Drainage (Fig. 25.1b–d)*

Extended drainage is achieved after ethmoidectomy by resecting the floor of the frontal sinus between the lamina papyracea and the middle turbinate (type II a) or the nasal septum (type II b) anterior to the ventral margin of the olfactory fossa.

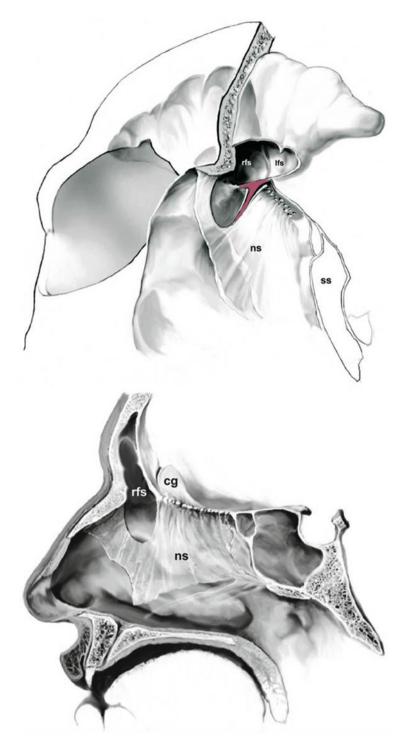
In the classification of May and Schaitkin [17] type IIa corresponds with NFA II (nasofrontal approach) and type IIb with NFA III. Hosemann et al. [6–8] showed in a detailed anatomical study that the maximum diameter of a neo-ostium of the frontal sinus (type IIa), which could be gained using a spoon or a curette, was 11 mm with an average of 5.6 mm. They also presented an excellent critical evaluation and results [8].

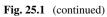
If one needs to achieve a larger drainage opening like type II-b, a drill is used because of the increasing thickness of the bone medially towards the nasal septum. During drilling with the diamond burr, bone dust fogs the endoscope, demanding repeated cleaning. At this point the microscope is useful, allowing one to work with two hands, while an assistant holds a simple self-retracting speculum according to Cholewa [1]. The endoscopic four-hand technique, introduced by May [16], is also a useful alternative, allowing the surgeon to work with two hands while an assistant holds the endoscope.

In revision cases after incomplete ethmoidectomy, it is recommended that a wide approach to the ethmoid sinuses is created using a microscope and drill or punch when possible. Punches and through-cutting instruments [18] help preserve the mucosa, whereas the drill is more destructive in this respect. The wide approach to the ethmoid is obtained by exposing the lacrimal bone and reducing it, as well as parts of the agger nasi and part of the frontal process of the maxilla, until the lamina papyracea is clearly seen through the microscope. This facilitates better visualization of the frontal recess to allow further work on the frontal sinus floor, but also



**Fig. 25.1** (**a**–**f**). Endonasal frontal sinus drainage 2. (**a**) Type I drainage (Simple drainage, *right side*). *aea* anterior ethmoidal artery, *lp* lamina papyracea, *mt* middle turbinate, *ns* nasal septum, *oc* olfactory cleft. (**b**) Type II a drainage (enlarged drainage, **a**, *right side*). Opening of frontal sinus between lamina papyracea and middle turbinate. Mostly possible without drill. (**c**) Type IIb drainage (enlarged drainage, **b**, *right side*). Drainage of the frontal sinus between lamina papyracea and middle turbinate. (**d**) Type IIb drainage (enlarged drainage, **b**, *right side*). Drainage of the frontal sinus between lamina papyracea and nasal septum. Usually medially drill necessary. (**d**) Type IIb drainage (median drainage) with "Frontal T" (*red*) and first olfactory fiber on both sides (View from *left inferior*). (**f**) Type III drainage (median drainage) sagittal view: removal of the frontal sinus floor in front of the olfactory cleft





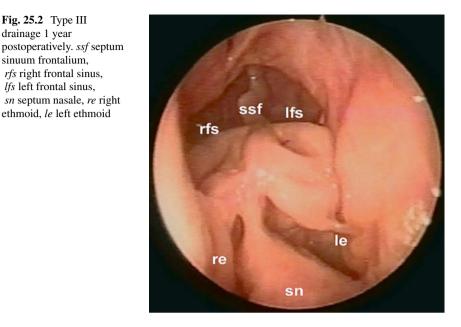
(A)	Indications type I drainage	
	Acute sinusitis	Failure of conservative surgery
		Orbital and endocranial complications
	Chronic sinusitis	First time surgery
		No risk factors (aspirin intolerance, asthma, triad)
		Revision after incomplete ethmoidectomy
( <i>B</i> )	Indications type IIa drainage	Serious complications of acute sinusitis
		Medial muco-pyocele
		Tumor surgery (benign tumors)
		Good quality mucosa
	Indications type IIb drainage	All indications of type IIa, if the resulting IIa is smaller
		than 5×7 mm. For type II b drill necessary
(C)	Indications type III drainage	Difficult revision surgery
		Primarily in patients with prognostic risk factors and
		severe polyposis particularly patients with triad
		mucoviscidosis
		Mucoviscidosis
		Kartagener's syndrome
		Ciliary immotility syndrome
		Benign and malignant tumors (see text)

Table 25.1 Indications for endonasal frontal sinus drainage Type I-III

makes the postoperative treatment less painful. As soon the frontal recess is identified using the middle turbinate and where identifiable, the anterior ethmoidal artery as landmarks, the frontal infundibulum is exposed and the anterior ethmoidal cells are resected. During surgery, repeated considerations of the pre-operative CT scans will establish the presence of so-called frontal cells [13] (Fig. 25.2, see also Chap. 2) which can develop far into the frontal sinus, giving the surgeon the erroneous impression that the frontal sinus has been properly opened. Sagittal CT slices and navigation may be helpful in difficult situations. When frontal cells are present, a procedure called "uncapping the egg" by Stammberger [26] uses a 45° telescope and results in a type IIa drainage.

An alternative when the middle turbinate has been retracted laterally after previous surgery and is obstructing the frontal sinus drainage is the so-called "frontal sinus rescue procedure" [12]. The decision should be left to the patient as to whether or not he desires a more conservative procedure like this, which has a relatively higher frequency of recurrence and need for re-operation.

If, after a type IIa drainage has been performed, further widening to produce a type IIb is required, the diamond burr is introduced into the clearly visible gap in the infundibulum and drawn across the bone in a medial direction. Care is taken to ensure that the frontal sinus opening is bordered by bone on all sides and that mucosa is preserved at least on one part of the circumference. To also safely create medially the widest possible opening of the frontal sinus floor, one should identify the first ipsilateral olfactory fiber (for details see type III drainage, Fig 25.2b, c). At the end a rubber finger stall can be introduced into the frontal sinus for about 5 days.



The indications for one or the other Type II drainage in general are listed in Table 25.1b. If the surgeon feels that the type IIa drainage is too small in regard to the underlying pathology, it is better to perform the type II b drainage procedure.

Some authors [9, 22, 32] advocated the use of soft, flexible silicone stents in cases of a frontal sinus neo-ostium less than 5 mm in diameter, since more rigid silicone tubes have not given satisfying results [19, 23]. So far the techniques using soft silicone drainage devices showed promising results although long term observation is still lacking.

# Type III: Endonasal Median Drainage (Fig. 25.1e, f)

*Endonasal median drainage or type III* The extended IIb opening is enlarged by resecting portions of the superior nasal septum in the neighborhood of the frontal sinus floor. The diameter of this opening should be about 1.5 cm. This is followed by resection of the frontal sinus septum or septa, if more than one are present. Starting on one side of the patient, one crosses the midline until the contralateral lamina papyracea is reached.

To achieve the maximum possible opening of the frontal sinus, it is very helpful to *identify the first olfactory fibers* on both sides: the middle turbinate is exposed and is resected in very small pieces from an anterior to posterior direction, along its origin at the skull base. After about 5 mm one will see the first olfactory fiber coming out of a small bony hole, slightly medial to the origin of the middle turbinate. The

same is done on the contralateral side. Finally the so-called "*Frontal T*" (Draf W, unpublished data) (Fig. 25.1e) results. Its long crus is represented by the posterior border of the perpendicular ethmoid lamina resection, and the shorter wings on both sides are provided by the posterior margins of the frontal sinus floor resection.

After that, the ethmoidectomy on the left side is performed exactly as it was on the right.

To perform the type III drainage in the technically most efficient way, it is helpful to interchange the use of the endoscope and microscope. Alternatively, this procedure can be done with the endoscope alone, though it is more time-consuming. Curved drills of different angles used with the shaver motor are helpful [34]. They allow a more superior reach into the frontal sinus and resection of the interfrontal sinus septum or septa, if more than one are present. They also allow removal of superiorly located frontal cells when present, and thus they help achieve a more complete operation. These measures help to create excellent landmarks for the anterior border of the olfactory fossa on both sides, which allow for an easier and safer complete resection of the frontal sinus floor as far posteriorly as the location of the first olfactory fiber.

Finally, a rubber finger stall is placed into each frontal sinus, and two more are placed in the ethmoid cavity and the inferior nasal meatus bilaterally. This packing is left for 7 days (!) under prophylactic antibiotic treatment. The rubber finger stalls do not stick to the surrounding tissue and are therefore easily and painlessly removed. The packing allows re-epithelialization of a major portion of the surgical cavity, which simplifies the postoperative treatment.

In difficult revision cases, one can begin the type III drainage primarily from two starting points, either from the lateral side as already described or from medially. The *primary lateral approach* is recommended if the previous ethmoidal work was incomplete and the middle turbinate is still present as a landmark. One should adopt the primary medial approach, if the ethmoid has been cleared and/or if the middle turbinate is absent.

The *medial approach* begins with the partial resection of the perpendicular plate of the nasal septum, followed by identification of the first olfactory fiber on each side as already described (Fig. 25.3).

The endonasal median drainage is identical with the NFA IV [16] and the "modified Lothrop procedure" [3]. Lothrop [14, 15] himself warned against using the endonasal route, judging it as too dangerous during his time; he performed the median drainage via an external approach. Halle [5] in 1906 created a large drainage from the frontal sinus directly to the nose using the endonasal approach, and using only a headlight and the naked eye.

The principle difference between the endonasal median frontal sinus drainage and the classic external Jansen, Lothrop, Ritter, Lynch, and Howarth operations is that the bony borders around the frontal sinus drainage are preserved. This makes it more stable in the long term and reduces the likelihood of reclosure by scarring, which may lead to recurrent frontal sinusitis or a mucocele, not to mention the avoidance of external scar.

The endonasal median drainage (type III) is indicated (Table 25.1c) after one or several previous sinus operations have not resolved the frontal sinus problem,

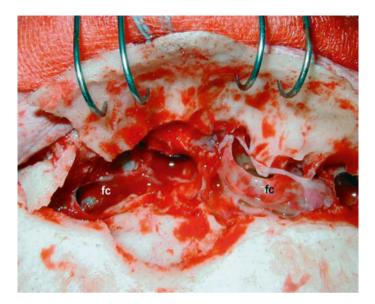


Fig. 25.3 Frontal sinus view from above after coronal osteoplastic revision. Several frontal cells of different sizes narrow the drainage into the nose (*fc* frontal cell)

including an external frontoethmoidectomy. It is also justified as a primary procedure in patients with severe polyposis and other prognostic "risk factors" affecting outcome, such as aspirin intolerance, asthma, Samter's triad (aspirin hypersensitivity, asthma, and allergy), Kartagener's syndrome, mucoviscidosis, and ciliary dyskinesia syndrome (Table 25.1c). Its use in patients with severe polyposis without these risk factors is undetermined and needs to be evaluated. It seems that patients with generalized polyposis but who still show air on coronal CT ("halo sign") in the periphery of the sinuses along the skull base have a comparatively better prognosis than those without, and can be managed by a more conservative technique. The procedure is also useful for removal of benign tumors in the frontal and ethmoid sinuses, as long as the main portion of the tumor in the frontal sinus is medial to a vertical line through the lamina papyracea. In addition, the use of the type III drainage makes the removal of malignant tumors which are just reaching the frontal sinus safer (Table 25.2).

#### **Results of Endonasal Frontal Sinus Surgery**

Judging the *results* of endonasal frontal sinus surgery requires a postoperative follow-up of ten or more years [11, 20, 21]. The failure rate of Neel et al. [11] with a modified Lynch procedure grew from 7 % at a mean follow-up of 3.7 years to 30 % at 7 years.

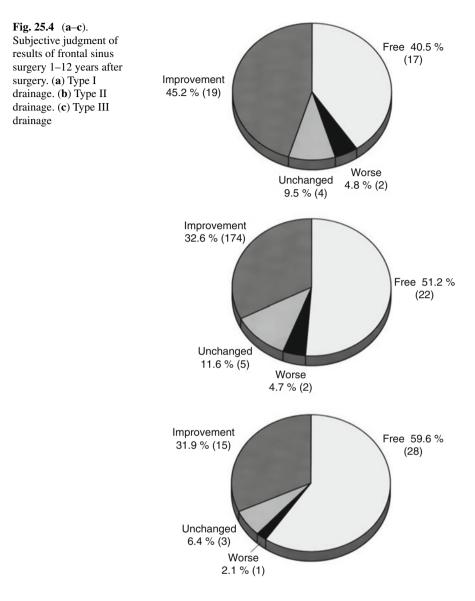
1.	Correctly performed Type III drainage failed		
2.	Patients after many endonasal and various external frontal sinus operations, so-called problem frontal sinus, sometimes in combination with complete endonasal ethmoidectomy		
3.	Type III drainage technically not possible (anterior-posterior diameter less than 8 mm)		
4.	Laterally located muco-pyocele		
5.	Major destruction of posterior wall		
6.	Inflammatory complications after trauma (e.g. alloplastic material, without or with obliteration)		
7.	Aesthetic correction of pneumatosinus dilatans frontalis (mostly without obliteration)		
8.	Major benign tumors (e.g. osteoma) without or with obliteration		

Table 25.2 Indications for the osteoplastic flap procedure

Kikawada et al. [12] presented a retrospective review of 22 consecutive cases of extended frontal sinus surgery (Draf type III) in patients with obstructive frontal sinusitis caused by postoperative scarring, with a follow-up time of at least 12 months after surgery. Of the 16 patients who underwent the type III procedure, in 14 (88 %) the patency of the newly created frontal sinus drainage and an aerated sinus were confirmed. Of 12 sides in nine patients who underwent Draf type II procedure, five sides (42 %) were also confirmed as cured. In the opinion of the authors, the median drainage operation (type III) on the frontal sinus showed excellent long-term results compared with the type II procedure.

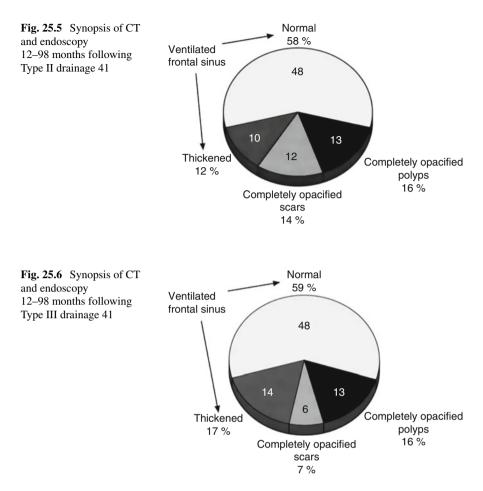
Weber et al. [29, 33] carried out two studies in 1995 and 1996. In the first retrospective study, patients who underwent endonasal frontal sinus drainage (471 type I drainages, 125 type II drainages, and 52 type III drainages) between 1979 and 1992 were evaluated. From these groups, random patients were examined: 42 patients with type I drainage, 43 with type II drainage, and 47 with type III drainage were included in the study. In each patient, the indication for surgery was chronic polypoid rhinosinusitis, except in five patients with type III drainage, in whom an orbital complication presented associated with acute sinusitis. The follow-up period was between 1 year and 12 years with a median of 5 years. The subjective estimation of operative results by the patients is shown in Fig. 25.4a-c. Applying subjective and objective criteria to evaluate the success of endonasal frontal sinus drainage (Grade 1 = endoscopically normal mucosa, independent of the subjective complaints; Grade 2=subjectively free of symptoms, but with endoscopically visible inflammatory mucosal changes; Grade 3 = no subjective improvement and pathologic mucosa = failure), it was possible to achieve a success rate of 85.7% with type I drainage, 83.8%with Type II drainage, and 91.5 % with type III drainage. This means that, despite the choice of prognostically unfavorable cases, type III drainages appeared to show the best results, though this was not statistically significant among the three groups.

In the second study [32], endoscopic and CT examinations were systematically carried out (Figs. 25.5 and 25.6). After 12–98 months follow-up of patients with type II drainage, 58 % of 83 frontal sinuses were ventilated and normal. A ventilated frontal sinus with hyperplastic mucosa was seen in 12 %. Scar tissue occlusion with



total opacification on CT was evident in 14 %. In 16 %, total opacification was due to recurrent polyposis. Seventy-nine percent of the patients were free of symptoms or had only minor problems.

Twelve to 89 months following type III drainage, 59 % of 81 frontal sinuses were ventilated and normal. A ventilated frontal sinus with hyperplastic mucosa was seen in 17 %. Scar tissue occlusion with total opacification on CT was obvious in 7 % and, in 16 %, there was total opacification due to recurrent polyposis. Ninety-five percent of the patients were free of symptoms or had only minor problems. Already



this first series of re-evaluation of long-term results demonstrates the value of the endonasal frontal sinus surgery.

In a retrospective study Mertens et al. [17] compared the results of 236 patients operated on between 1985 and 1993 using different techniques. After follow-up of 3-10 years only 8 % of patients needed revision. The lowest revision rate was seen after endonasal technique according Draf's classification (5.9 %) compared to the osteoplastic techniques according to Jansen-Ritter (Lynch) and Riedel (10.6 %).

#### **Postoperative Care**

There are different ways of providing *postoperative care*. Within the years the following standards proved to be efficient:

#### Packing

Rubber finger stalls (Rhinotamp; Vostra Aachen) filled with sponge have stood the test of time. They provide safe hemostasis, are a stimulator of re-epithelialization of bare bone, are cost-effective and painless to remove. In cases of type I-type IIb drainage, the packing is left between 2 and 5 days maximum without antibiotic prophylaxis. It is of utmost importance to fix the rubber finger stalls with threads at the nasal dorsum to avoid aspiration. The more stable the middle turbinate at the end of operation is, the shorter the time of uncomfortable packing can be. The risk of adhesions and synechiae is low because this type of packing suppresses the development of granulations.

After a type III drainage, we leave the packing in place for 7 days postoperatively as recommended by Toffel [28].

Leaving rubber finger stalls for 1 week carries the following advantages [33]:

- 1. The fibrinoid phase of wound healing is somehow overcome. Reclosure of the large drainage by scars is remarkably reduced, since bare bone is reepithelialized almost completely.
- 2. Sedation and general anesthesia are not necessary for packing removal. Rubber finger packs do not bind to the wound. Removal of the tamponade does not lead to renewed tissue trauma. The patients are prepared preoperatively for a somewhat uncomfortable postoperative time. This is by far compensated by the optimal wound healing and easy postoperative care with less crusting.

#### **Postoperative Therapy**

The question of whether postoperative intensive mechanical cleansing is necessary or the wound cavity is self-cleaning without external measures is very controversial.

In an obstructed nose or sinus, when the patient has complaints that can be explained with occlusion of the sinus ostial region by crusts, mechanical cleaning must be done. However, since each cleaning leads to injury, freshly granulating tissue, and partial removal of new epithelium, a rather controlled and conservative approach to instrument cleaning seems appropriate.

The patients are given the following instructions to ensure proper healing:

- 1. Irrigate the nasal cavities with saline solution at least once a day, sometimes more frequently.
- 2. Use one of the corticosteroid sprays 1–3 times/day.
- 3. The recommendation is made to use peanut oil 1 h after the use of corticosteroid spray, for general care of the mucosa.

In patients with extreme crusting, the physician should inquire about the use of medications because as a side effect, they cause mucosal desiccation. These include psychotropic medications or beta-blockers. Spectacular improvement is possible once these medications are changed.

#### **General Postoperative Medication**

- 1. *Antibiotics*: They are indicated in the postoperative period for 1–2 weeks in cases of acute or purulent sinusitis. In type III drainage, we recommend prophylactic antibiotic use, as long as the tamponade is in place.
- 2. Antiallergic medical therapy: This is recommended for 6 weeks postoperatively if allergy is diagnosed by history or specific tests. The presence of a large number of eosinophils in the inflamed tissue may provide additional guidance in the decision-making process. In less severe cases we prescribe day antihistamines. In severe allergy patients (e.g. Samter's triad), the combination of antihistamines with low-dose corticosteroid medication for 6 weeks is helpful to prevent early recurrence of polyps.

#### Failures

#### Postoperative Frontal Sinusitis After Type I and Type II Drainage

Sometimes after ethmoidectomy and type I as well as type II drainage, the patients may develop more problems in the frontal sinus than before surgery. Postoperative sinus CT will provide information if frontal sinusitis has developed.

The pathogenesis of recurrent frontal sinusitis after surgery can involve various mechanisms. Either remnant ethmoidal cells developed recurrent sinusitis or mechanical irritations of the mucosa in the frontal recess can result in a severe scar around Killian's infundibulum. Both pathologies may result in blockage of the frontal sinus drainage.

This can be avoided by performing at least a complete anterior ethmoidectomy and using extremely atraumatic handling of the frontal recess mucosa. For treatment we recommend the following procedures: a type IIa drainage if a type I procedure was performed previously, a type IIb drainage if a type IIa procedure was performed previously, and a type III drainage after a previous type IIb.

#### **Reclosure** After Type III Drainage

Several technical details can lead to this problem:

- (a) The "chimney" between the anterior ethmoid and the frontal sinus has not been opened well. It is important that after the anterior ethmoidal artery is identified, the surgeon proceeds along the skull base medial to the lamina papyracea to enter into the frontal sinus.
- (b) The anterior-posterior opening of the frontal sinus floor, particularly in the midline, is too small. The identification of the first olfactory fiber bilaterally and the creation of the "Frontal T" are very helpful to avoid this problem.
- (c) The resection of the septum/a sinuum frontalium has been missed or was not performed to a satisfying degree. The new curved drills between 15° and 60° angle are ideal for this purpose.
- (d) The resection of the superior nasal septum was too small. The diameter of resection must be 1.5 cm just in front of the "Frontal T" and below the frontal sinus floor.
- (e) The packing between the ethmoid and the frontal sinus was not left long enough. 7 days proved to be the best time frame for using rubber finger packings.

# Complications

In principle, the complication rate of endonasal frontal sinus drainage procedures is low and similar to the frequency of complications of endonasal pansinus operations.

An evaluation of complications with special respect to endonasal frontal sinus surgery was not performed. The operation can be classified as very safe, even with identification of the first olfactory fibers, when optical aids such as microscope and/ or endoscope are used and the techniques described are followed.

We have analyzed the complications of our endonasal micro-endoscopic pansinus operations in two studies [30, 31].

The significant complications were:

- 1. Injury to the periorbit in 14 % of cases. This had no further consequences except in one patient, who developed periorbital hematoma. No cases of blindness or other orbital lesions like muscle injury with double vision or lacrimal drainage obstruction occurred.
- 2. Dural injury occurred in 2.3 % of cases. The subsequent course was uneventful and free of complications after immediate plastic closure of the defect with preserved fascia and fibrin glue. Persistent CSF leakage or meningitis was not observed.
- 3. A postoperative disturbance of the sense of smell was confirmed by a smell test in only one patient.

# General Guidelines for Surgical Therapy of Frontal Sinus Inflammatory Diseases Not Responding to Conservative Measures

# How to Treat Frontal Sinusitis, Which Has Not Responded to Conservative Measures Nor Has Been Operated Before?

Depending on the individual situation, in most of the cases, the endonasal type I or type II drainage will be sufficient, whereas in severe polyposis with Samter's triad or other risk factors, a primary type III operation may be indicated.

# What to Do in Cases of Chronic Postoperative Frontal Sinusitis After One or Even Several Previous Operations, Otherwise Referred to as "Iatrogenic" Sinusitis [10, 22, 26]?

The term "iatrogenic sinusitis" suggests that previous surgeons have made a mistake. Since many other factors may have contributed to an unsatisfactory surgical outcome, such as a particular anatomic variant, it seems more appropriate to use the term "*postoperative sinusitis*"(*see* also above) indicative that such a patient has already undergone surgery, an important fact in further decision-making.

In many patients one can prove with CT that the ethmoidectomy was incomplete. Inflamed residual anterior ethmoid cells often cause symptoms of chronic frontal sinusitis, whereas the more posteriorly located, well-drained parts of the sinus system are aerated. In this situation completion of the ethmoidectomy in combination with a type IIa/b procedure is indicated. In cases of Samter's triad and other prognostic risk factors, the type III drainage procedure is the best choice. If the patient had numerous prior operations and wishes to have only one more "final" sinus surgery, the surgeon has to choose between the type III drainage and the osteoplastic flap procedure with obliteration. If the frontal sinus is large enough and has an anterior-posterior diameter of at least 0.8 mm, the type III drainage may be attempted. If the frontal sinus has a smaller diameter, the frontal sinus fat obliteration is the safer technique, although more extensive.

# How "Radical" the Extent of Primary Surgery Should Be in Patients with Extensive Polyposis of the Frontal Sinus?

In this situation, particularly in the presence of aspirin hypersensitivity or/and asthma, experience has shown that the primary type III drainage is most likely to be successful. In cases of recurrence and severe frontal sinus symptoms, the osteoplastic operation is indicated

# Should Patients with So-Called Spontaneous or Postoperative Mucoceles of the Frontal Sinus, Be Operated Endonasally or via an External Approach?

As long as the endonasal route using a type II or type III drainage procedures provides a sufficient opening and not a bottleneck situation, the *endonasal marsupialization* is reliable and the least traumatic measure. However, if the medial border of the mucocele is laterally to a vertical line through the lamina papyracea, the endonasal approach is rarely possible. This is also the case if, usually after several previous operations, multiple mucoceles are diagnosed. In this situation the frontal sinus obliteration is usually indicated. The final decision is made on the basis of a multi-planar sinus CT, often in combination with MR, since MR gives the best diagnostic information of a mucopyocele. A previous Jansen-Ritter, Howarth, or Lynch operation is not a contraindication to endonasal drainage.

# Is a Pott's Puffy Tumor Always an Indication for an External Procedure?

If the anterior-posterior diameter of the frontal sinus is 0.8 mm or greater, the likelihood of a successful type III drainage is high enough to be tried. Long term postoperative antibiotic therapy is mandatory.

However, these general guidelines cannot replace personal experience!

#### Conclusion

The endonasal frontal sinus type I–III drainage procedures provide suitable surgical options for the treatment of frontal sinus disease. In cases where the endonasal approach is not possible or is unsuccessful, the osteoplastic flap procedure with or without obliteration may provide a solution. The chance of complete reepitheliazation of eventually bare bone is very likely with the endonasal frontal sinus operations, since they respect the outer osseous borders of the newly created frontal sinus drainage and minimize the danger of frontal sinus outlet shrinking, thus preventing mucocele formation. This concept has revolutionized frontal sinus surgery, so that the classic external frontoorbital frontal sinus operations according to Jansen-Ritter or Lynch or Howarth are considered obsolete for the treatment of chronic inflammatory diseases of the frontal sinus.

## References

- 1. Cholewa ER. Cited after Tange RA, Pirsig W Het neusspeculum. Door de eeuwen heen. Universiteitsmuseum van de Universiteit van Utrecht. Glaxo BV; 1888.
- Draf W. Endonasal micro-endoscopic frontal sinus surgery. The Fulda concept. Oper Tech Otolaryngol Head Neck Surg. 1991;2:234–40.
- Gross WE, Gross CW, Becker D, Moore D, Phillips D. Modified transnasal endoscopic Lothrop procedure as an alternative to frontal sinus obliteration. Otolaryngol Head Neck Surg. 1995;113:427–34.
- 4. Halle M. Die intranasalen Operationen bei eitrigen Erkrankungen der Nebenhoehlen der Nase. Arch Laryngol Rhinol. 1915;29:73–112.
- 5. Halle M. Externe und interne Operation der Nebenhoehleneiterungen. Berl klin Wschr. 1906;43(1369–1372):1404–7.
- 6. Hosemann W, Gross R, Goede U, Kuehnel T. Clinical anatomy of the nasal process of the frontal bone (spina nasalis interna). Otolaryngol Head Neck Surg. 2001;125:60–5.
- Hosemann W, Kuehnel T, Held P, Wagner W, Felderhoff A. Endonasal frontal sinusotomy in surgical management of chronic sinusitis : a critical evaluation. Am J Rhinol. 1997;11:1–9.
- Hosemann WG, Weber RK, Keerl RE, Lund VJ. Minimally invasive endonasal sinus surgery. Stuttgart: Thieme; 2000. p. 54–9.
- Hoyt III WH. Endoscopic stenting of nasofrontal communication in frontal sinus disease. Ear Nose Throat J. 1993;72:596–7.
- 10. Ingals EF. New operation and instruments for draining the frontal sinus. Ann Otol Rhinol Laryngol. 1905;14:513–9.
- 11. Kennedy DW, Senior BA. Endoscopic sinus surgery. A review. Otolaryngol Clin North Am. 1997;30:313–29.
- Kuhn FA, Javer AR, Nagpal K, Citardi MJ. The frontal sinus rescue procedure: early experience and three - year follow-up. Am J Rhinol. 2000;14:211–6.
- Lang J. Clinical anatomy of the nose nasal cavity and paranasal sinuses. Stuttgart: Thieme; 1989. p. 58–9.
- 14. Lothrop HA. Frontal sinus suppuration. Ann Surg. 1914;59:937-57.
- Lothrop HA. The anatomy and surgery of the frontal sinus and anterior ethmoidal cells. Ann Surg. 1899;29:175–215.
- May M, Schaitkin B. Frontal sinus surgery: endonasal drainage instead of an external osteoplastic approach. Oper Tech Otolaryngol Head Neck Surg. 1995;6:184–92.
- 17. Mertens J, Eggers S, Maune S. Langzeitergebnisse nach Stirnhoehlenoperationen: Vergleich extranasaler und endonasaler Operationstechniken. Laryngorhinootol. 2000;79:396–9.
- Moriyama H, Fukami M, Yanagi K, Ohtori N, Kaneta K. Endoscopic endonasal treatment of ostium of the frontal sinus and the results of endoscopic surgery. Am J Rhinol. 1994;8:67–70.
- 19. Neel III HB, Whitaker JH, Lake CF. Thin rubber sheeting in frontal sinus surgery: animal and clinical studies. Laryngoscope. 1976;86:524–36.
- 20. Neel HB, McDonald TJ, Facer GW. Modified Lynch procedure for chronic frontal sinus diseases: rationale, technique, and long term results. Laryngoscope. 1987;97:1274.
- Orlandi RR, Kennedy DW. Revision endoscopic frontal sinus surgery. Otolaryngol Clin North Am. 2001;34:77–90.
- 22. Rains III BM. Frontal sinus stenting. Otolaryngol Clin North Am. 2001;34:101-10.
- 23. Schaefer SD. Close LG endoscopic management of frontal sinus disease. Laryngoscope. 1990;100:155–60.
- 24. Schaeffer M. Zur Diagnose und Therapie der Erkrankungen der Nebenhoehlen der Nase mit Ausnahme des Sinus maxillaris. Dtsch Med Wschr. 1890;19:905–7.
- 25. Spiess G. Die endonasale Chirurgie des Sinus frontalis. Arch Laryngol. 1899;9:285–91.
- 26. Stammberger H. F.E.S.S., "uncapping the egg". The endoscopic approach to frontal recess and sinuses. Storz Company Prints; 2000.

- Tato JM, Bergaglio OE. Surgery of frontal sinus. Fat grafts: a new technique. Otolaryngologica. 1949;3:1.
- Toffel PH. Secure endoscopic sinus surgery with middle meatal stenting. Oper Tech Otolaryngol Head Neck Surg. 1995;6:157–62.
- Weber R, Draf W, Keerl R, Schick B, Saha A. Micro-endoscopic pansinusoperation in chronic sinusitis. Results and complications. Am J Otolaryngol. 1997;18:247–53.
- Weber R, Draf W. Die endonasale mikro-endoskopische Pansinusoperation bei chronischer Sinusitis. II. Ergebnisse und Komplikationen. Otorhinolaryngol Nova. 1992;2:63–9.
- Weber R, Draf W. Komplikationen der endonasalen mikro-endoskopischen Siebbeinoperation. HNO. 1992;40:170–5.
- Weber R, Hosemann W, Draf W, Keerl R, Schick B, Schinzel S. Endonasal frontal sinus surgery with long-term stenting of the nasofrontal duct. Laryngorhinootologie. 1997;76:728–34.
- 33. Weber R, Keerl R, Huppmann A, Draf W, Saha A. Wound healing after endonasal sinus surgery in time-lapse video: a new way of continuous in vivo observation and documentation in rhinology. In: Stamm A, Draf W, editors. Micro-endoscopic surgery of the paranasal sinuses and skull base. Berlin: Springer; 2000. p. 329–45. Chapter 26.
- Wormald PJ, Ananda A, Nair S. Modified endoscopic Lothrop as a salvage for the failed osteoplastic flap with obliteration. Laryngoscope. 2003;11:1988–92.

### **Further Reading**

Howarth WG. Operations on the frontal sinus. J Laryngol Otol. 1921;36:417-21.

- Jansen A. Zur Eroeffnung der Nebenhoehlen der Nase bei chronischer Eiterung. Arch Laryng Rhinol (Berl). 1894;1:135–57.
- Kikawada T, Fujigaki M, Kikura M, Matsumoto M, Kikawada K. Extended endoscopic frontal sinus surgery to interrupted nasofrontal communication caused by scarring of the anterior ethmoid. Arch Otolaryngol Head Neck Surg. 1999;125:92–6.
- Lynch RC. The technique of a radical frontal sinus operation which has given me the best results. Laryngoscope. 1921;31:1–5.
- Ritter G. Eine neue Methode zur Erhaltung der vorderen Stirnhoehlenwand bei Radikaloperationen chronischer Stirnhoehleneiterungen. Dtsch Med Wochenschr. 1906;32:1294–6.
- Stammberger H. Functional endoscopic sinus surgery, the Messerklinger technique. Philadelphia: BC Decker; 1991.

## Chapter 26 Endoscopic Modified Lothrop Procedure

Jastin L. Antisdel and Stilianos E. Kountakis

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#### **Core Messages**

- The endoscopic modified Lothrop procedure (EMLP) is recommended as a surgical option when unable to keep frontal sinuses openings patent with previous endoscopic approaches
- The success of the EMLP depends on the underlying mucosal pathology and its effective management
- The EMLP is the preferred approach for benign tumors of the frontal sinus such as inverted papilloma, since it allows for direct endoscopic postoperative surveillance for tumor recurrence

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_26

- In order to perform the EMLP the total anterior-posterior dimension at the cephalad margin of the frontal recess between the nasal bones at the root of the nose and the anterior skull base should allow adequate space for insertion of instruments and drills without risking injury to the skull base
- While performing the EMLP avoid drilling on the posterior margin of the frontal recess and posterior frontal sinus wall to prevent injury to the skull base with cerebrospinal fluid rhinorrhea and postoperative circumferential stenosis of the frontal opening
- Absolute contraindications in performing the EMLP include inadequate surgical training and the lack of proper instrumentation

## Introduction

Frontal sinus surgery has remained a challenging procedure despite continued advances in instrumentation and surgical techniques.

Partly to blame is the relatively inaccessible location of the frontal recess, posterior and cephalad to the anterior insertion of the middle turbinate, hiding away from the surgeon's direct line of vision. Moreover, multiple anterior ethmoid cells may occupy the frontal recess complicating the approach to the frontal sinus [13]. The variable size and location of these air cells contribute to the numerous patterns of the frontal sinus outflow pathway that is actually a potential space amongst the surface of these frontal recess cells, leading to the internal frontal sinus ostium. The remote location and anatomic complexity of the frontal recess along with its close proximity to the lamina papyracea and anterior skull base, led Lothrop [14] to state that an intranasal approach for frontal sinus drainage was too dangerous to perform. Instead, he described an external approach, which consisted of external ethmoidectomy to enlarge the nasofrontal drainage pathway. This included removal of the frontal sinus floors that were connected through a large nasal septectomy and bilateral removal of the lacrimal bone and portion of the lamina papyracea. This caused medial orbital fat collapse with later stenosis of the newly created nasofrontal outflow communication.

The development of the external osteoplastic flap procedure [10] with or without frontal sinus obliteration in the 1940s–1960s eliminated the need for a nasofrontal communication and quickly became the standard of care. However, failure rates averaged 10 % in early reports [2, 10] and more recently, Weber et al. [17] reported frontal mucoceles seen by magnetic resonance imaging in 9.4 % of the patients approximately 2 years after osteoplastic frontal sinus obliteration. Further time has shown that many, if not most obliterated frontal sinuses will eventually develop

1914	Lothrop procedure
1960s	Open osteoplastic flap frontal surgery becomes standard of care
1970s	Linear tomography and improved preoperative anatomic evaluation
1980s	Computed tomography
	Popularizing of endoscopic sinus surgery
1990s	Powered instrumentation
	Computer image-guided endoscopic surgery
1991	Extended frontal sinusotomy by Draf
1994	Endoscopic resection of the intranasal frontal sinus floor by Close
1995	Endoscopic modified Lothrop procedure by Gross
2003	Endoscopic modified Lothrop for salvage of frontal obliteration by Wormald

Table 26.1 Chronological advances leading to the endoscopic modified Lothrop procedure

mucoceles. This is usually in a delayed fashion, with one study showing an average of 11 years until failure [18].

The introduction of the nasal endoscope and endoscopic sinus surgery techniques allowed for better visualization of the frontal recess during surgery and provided an alternative to the open techniques for the surgical treatment of frontal sinus disease. Furthermore, endoscopic frontal surgery precisely addresses the exact location of chronic frontal sinus pathology that is obstructing the frontal sinus outflow pathway. In comparison, the open techniques (obliteration or cranialization) destroys the outflow tracts and mucosa. Despite all the endoscopic technique and instrumentation advances, the frontal sinus continues to remain challenging for many otolaryngologists. The necessary extent of surgery performed in the frontal recess being continues to be debated in the literature. Excessive mucosal damage during endoscopic surgery can lead to scarring with obstruction of frontal drainage, and resection of the middle turbinate can lead to lateralization of the turbinate and obstruction of the frontal recess, as reported by Kuhn et al. [12]. As endoscopic advances continued, the Lothrop procedure was revisited as an alternative to the open destructive techniques. Draf in 1991 described removal of the frontal sinus floor bilaterally using endoscopic and microscopic techniques, and classified the extent of surgery in the frontal recess [5]. Close et al. in 1994 reported their results with 11 patients, and soon thereafter a series of reports established the legitimacy of the procedure with successful long-term surgical outcomes [4] (Table 26.1). In the process, the procedure was renamed to accurately reflect the location and extent of surgery. The EMLPoffers several distinct advantages over open techniques and in the hands of modern rhinologists has displaced the osteoplastic flap approach as the procedure of choice in persistent frontal disease after failure of medical therapy and more conservative endoscopic surgery.

#### The Advantages of the EMLP Are

- No external incision with improved cosmesis
- Decreased morbidity
- No hospital stay
- No drains
- No abdominal wound
- · Avoidance of supra-orbital and supra-trochlear nerve injury
- Reduced blood loss
- No burying of mucosa
- Less pain
- · Lower total cost
- · Can still convert to open approaches if necessary
- Allows for endoscopic postoperative evaluation for persistent or recurrent disease
- · Avoids long term risk of mucocele and the associated complications

## **Indications and Patient Selection**

The success of the EMLP depends on the anatomy of the frontal recess and the underlying mucosal pathology. Sinus surgery in patients with chronic rhinosinusitis in general is indicated when maximum medical therapy fails to control the symptoms of the disease. Initial surgical intervention in primary cases should avoid overly excessive manipulation in the frontal recess unless absolutely necessary. More extensive frontal surgery is performed in revision cases when scarring or persistent disease in the frontal recess and internal frontal ostium interferes with frontal sinus drainage. The EMLP is recommended as a surgical option when other endoscopic techniques have failed and prior to considering open osteoplastic flap surgery [6–9, 11, 16]. Table 26.2 lists all frontal sinus procedures available for the otolaryngologist as part of a protocol in the surgical management of frontal sinus disease. Patients with underlying mucosal disease such as hyperplastic rhinosinusitis with nasal polyposis, sarcoidosis, Wegener's granulomatosis, and Samter's triad require

**Table 26.2**Frontal sinusprocedure protocol

<ol> <li>Endoscopic uncinectomy and anterior ethmoidectomy without surgery in the frontal recess</li> </ol>
2. Frontal recess surgery
3. Minitrephination of the frontal sinus as an aid in endoscopic sinus surgery
4. Surgical manipulation of the internal frontal sinus ostium
5. Unilateral resection of frontal sinus floor (Draf II procedure)

- 6. EMLP
- 7. Osteoplastic flap surgery

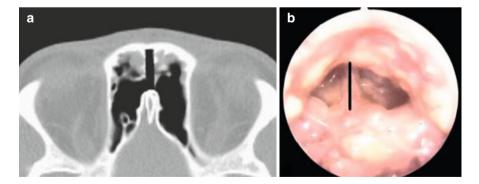
aggressive postoperative care to control mucosal inflammation and prevent restenosis of the nasofrontal drainage pathway with symptom recurrence. One of the advantages of the EMLP is that once performed, it does not prevent a surgeon from resorting to open osteoplastic flap techniques if the EMLP fails and frontal sinus obstruction recurs.

The anatomy of the frontal sinus and the cephalad margin of the frontal recess at the level of the internal frontal ostium are critical in the selection of patients for the EMLP.

# As Part of the Preoperative Evaluation, the Surgeon Should Do the Following

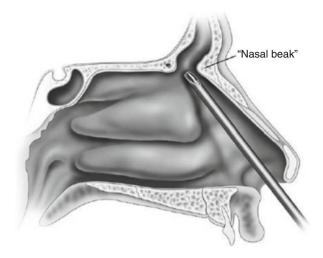
- Carefully review the anatomy on high-resolution CT to identify the number, size, and location of air cells present in the frontal recess.
- Measure and calculate distances on the CT to ensure that there is enough space between the skull base and the root of the nose for drilling (Fig. 26.1). Fig. 26.2 shows the think nasal beak that should be drilled.

The number of air cells in the frontal recess is important, since it indicates the number of sinus cell walls that should be removed in order to reach the internal frontal sinus ostium. Review of the CT determines whether the patient is a candidate for the EMLP. There should be enough space between the skull base and the root of the nose to allow introduction of instruments and drills for removal of the nasal beak to create a large frontal drainage space, while protecting the skull base. Thick nasal beaks narrow the space between the nasal beak and the anterior skull base, making



**Fig. 26.1** The total anterior-posterior dimension at the cephalad margin of the frontal recess, between the nasal bones at the root of the nose and the anterior skull base (*solid black line*) should allow the insertion of drills for nasal beak removal while minimizing injury to the skull base. (**a**) Axial sinus CT through the cephalad margin of the frontal recess. (**b**) Endoscopic picture 3 weeks after the EMLP

Fig. 26.2 Nasal beak



introduction of instruments into the frontal sinus very difficult which increases the chances for skull base injury with cerebrospinal fluid (CSF) rhinorrhea.

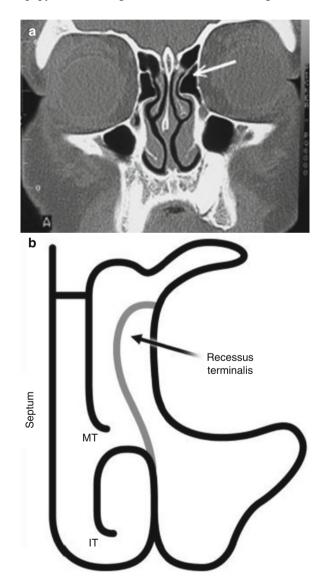
As experience with the technique has grown, the procedure has been used successfully not only for chronic frontal sinus obstruction but also for resection of osteomas and benign tumors such as inverted papillomas. A main advantage of using the EMLP to remove frontal sinus inverted papillomas is the ability to directly inspect the sinus postoperatively for recurrence of the tumor.

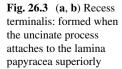
#### Indications and Contraindications for the EMLP Are as Follows

- Indications
  - Persistent chronic frontal sinusitis with failure of appropriate medical therapy and after unsuccessful primary endoscopic frontal sinusotomy
  - Frontal sinus mucoceles
  - Inverted papilloma
  - Osteoma
  - Trauma
  - Last-resort procedure prior to osteoplastic frontal sinus obliteration
- Contraindications
  - Hypoplastic frontal sinus and frontal recess
  - Lack of experience by the surgeon
  - Lack of proper instrumentation
  - Sinus disease located in a supra-orbital ethmoid air cell and not in the frontal sinus

### **Surgical Technique**

The EMLP requires general anesthesia with meticulous intraoperative hemostasis. The nasal cavities are first decongested using topical decongestant. Local anesthetic (per surgeons preference) can then be used. The extent and type of local decongestant applied depends on the medical condition of each individual patient. Both preand intraoperative review of the CT scan, combined with rigid nasal endoscopy allows for choice of the most approachable frontal recess to start the procedure. The middle turbinate or remnant is medialized and the superior attachment of the uncinate process remnant is resected. In the most common configuration, the uncinate process attaches to the lamina papyracea forming the recessus terminalis (Fig. 26.3).





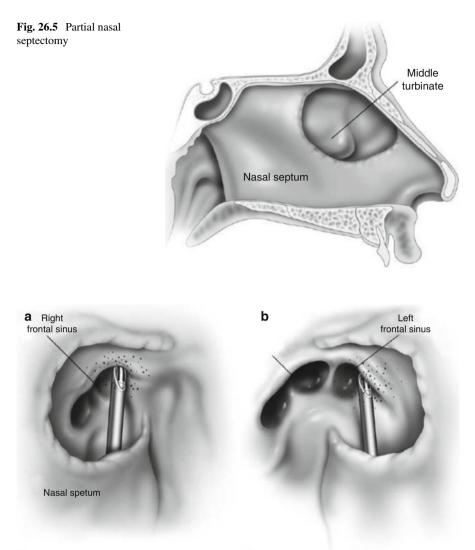
**Fig. 26.4** Unilateral frontal stenosis after circumferential scar formation

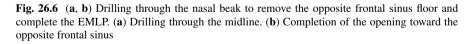


The frontal ostium is identified posterior and medial to the recessus terminalis. Computer image guidance may be used to help with the identification of the internal frontal sinus ostium. Drilling is initiated in an anterior direction through the anterior insertion of the middle turbinate to enlarge the frontal sinus ostium until the level of the nasal bone is reached. Similarly, drilling is performed in a lateral direction until the level of the plane of the lamina papyracea is reached. This is essentially a Draf IIb. Care is taken not to remove the mucosa over the lateral wall of the frontal recess at the plane of the lamina papyracea to preserve the ciliated epithelium responsible for transporting secretions out of the frontal sinus. Care is also taken to prevent mucosal injury at the posterior margin of the frontal sinus and ostium to prevent circumferential mucosal injury with possible postoperative ostial stenosis (Fig. 26.4) as well as to avoid injury to the skull base with possible CSF rhinorrhea.

Once the level of the nasal bones is reached anteriorly, drilling is directed medially until the plane of the nasal septum is reached. In the process, part of the nasal beak is removed (Fig. 26.2). To avoid going through the nasal bones and causing soft tissue injury over the nasal root at the glabella, two fingertips are placed over the nasal root to feel and sense the closeness of the drill to the nasal bones. Once the nasal septum is reached, a partial septectomy is necessary for the creation of a large common nasofrontal drainage pathway. The center of the surgical septal perforation is located right under the floors of the frontal sinuses (Fig. 26.5). The size of the septectomy should be approximately 2 cm. Smaller septal perforations accumulate crusting despite aggressive nasal saline irrigation and thus cause inflammation and delayed healing. Drilling then is continued though the nasal beak, removing the frontal sinus floor on the opposite side and continued until the opposite lamina papyracea is reached (Fig. 26.6).

In the process, the posterior wall of the frontal sinus is protected to prevent injury to the skull base and to avoid circumferential mucosal scarring with stenosis. There





is no need to remove the frontal sinus septum all the way posteriorly to the skull base. Drilling in this area may lead to violation of an anteriorly displaced skull base. With successful surgery, the common frontal sinus cavities can be easily inspected using a nasal endoscope (Fig. 26.7). Additional techniques have been described, such as an "outside-in" approach where the limits of the sinusotomy are first defined and the frontal recess is addressed last [19]. This is of utility when the frontal recess is involved by tumor, inflammatory disease, or scarring.

**Fig. 26.7** Endoscopic view of the common frontal sinus cavities 2 years after the EMLP



## **Postoperative Care**

Aggressive medical management to remove crusting and control mucosal disease is vital for successful outcomes. All patients are placed on postoperative antibiotics, with high-dose mucolytics and intranasal steroid sprays. Patients with nasal polyposis benefit from short-term tapering doses of oral systemic steroids. Patients are instructed to perform nasal saline irrigations with high volume, high flow device at least twice daily throughout healing process. Endoscopic debridement is performed in the office setting one week after surgery and is repeated every 2 weeks thereafter until crusting in the common nasofrontal pathway is not an issue. During these visits, debris present in the open frontal sinus is removed. This is very important in cases of allergic fungal sinusitis in order to reduce the fungal load. Any persistent polyps in patients with eosinophilic nasal polyposis may be removed. Higher dose of topical steroid, such as budesonide irrigations are also a consideration in patients with nasal polyposis.

### **Results and Complications**

Initial reports by Draf [5], Close [4], and Becker [1] indicated high rates of frontal drainage pathway patency after surgery for chronic sinusitis, but it was quickly realized that the operative site after the EMLP requires at least 12–18 months after surgery for stabilization. Casiano [3] reported that 12 of 21 patients (57 %) had patent common frontal opening by flexible fiber optic examination with a mean followup of 6.5 months. Gross et al. [9] found a 95 % frontal drainage patency rate with a mean follow-up of 12 months, but as experience with the procedure accumulated and patients were followed for longer periods of time, the overall patency success rate was reduced. When 44 patients were followed for an average of 40 months after EMLP, 9 (20 %) required revision EMLP and 8 of 44 (18 %) patients eventually required osteoplastic flap with frontal sinus obliteration, with an overall frontal drainage patency rate of 82 % [16]. This number is more realistic when patients are followed long-term, especially if one considers that as we gain more experience with the procedure, EMLP is more often performed in patients with more aggressive mucosal disease, which probably contributes to the long-term failure rate of this surgery. In their report however, Gross and colleagues reported that surgical outcomes were not influenced by comorbidities such as eosinophilic CRS with nasal polyposis and aspirin-sensitive asthma [7]. Close follow-up and management of early polyps prevented frontal sinus obstruction in these patients.

A primary concern in concentric scarring over time with narrowing of the Lothrop cavity. Metson et al. found that frontal drainage occlusion occurred in 12.5 % of patients treated with EMLP [15]. Subsequent larger study found that 28.6 % of patients had more than 60 % decrease in size of Lothrop cavity by 1 year post-op. Revision EMLP was required in 40.0 % of those patients. Average narrowing in the group was 33 % [20]. In answer to this stenosis it is vitally important to maximize the size of the cavity. Some have employed techniques to attempt to minimalize the stenosis. Hildenbrand et al. reported their experience using free septal mucosal grafts to epithelialize the nasofrontal communication in attempt to prevent stenosis [21]. These patients had average decrease of 36.9 %, which is not significantly different than previously reported rates of stenosis.

Instrumentation advances also has played a role in the higher success rates observed over time compared to initial reports. Powered microdebriders and finesse drill-bits are available with appropriate angulation and decreased size, allowing for more maneuverability in the frontal recess with reduced mucosal trauma and post-operative stenosis. Meta-analysis on all studies from 1990 to 2008 showed that 86.1 % of EMLP was successful. 80 % of those failures underwent revision EMLP with only 20 % undergoing osteoplastic flap. Symptoms improved in 82.2 % of patients [22]. The longest term follow-up study to date (10 years) shows a patency rate 95 %, with the 5 % having patency after single revision EMLP [23].

Major complications with the EMLP are infrequent but can be devastating, since the operative field is between the orbits and just anterior to a very thin skull base. Lack of proper surgical training and instrumentation should preclude attempts to perform this procedure. Skull base violations with CSF leaks and pneumocephalus has been reported even in the most experience hands [16], but none of the studies have reported more than one patient having skull base violation per study. Serious eye injury has not been reported in any of the studies. Meta-analysis shows major complications less than 1 % and minor at 4 % (Anderson/Sindwani).

#### Conclusion

The EMLP has been shown to be an effective alternative to osteoplastic flap frontal surgery. With present instrumentation, proper training, and appropriate patient selection, the EMLP is a proven part of the rhinologist's armamentarium. This

procedure, combined with other advancement has nearly made osteoplastic flap surgery a procedure of the past. In selected cases such as inverted papilloma of the frontal sinus, it has become the procedure of choice since it allows for endoscopic frontal sinus inspection and postoperative surveillance.

## References

- 1. Becker DG, Moore D, Lindsey WH, Gross WE, Gross CW. Modified transnasal endoscopic Lothrop procedure: further considerations. Laryngoscope. 1995;105(11):1161–6.
- 2. Bosley WR. Osteoplastic obliteration of the frontal sinuses. A review of 100 patients. Laryngoscope. 1972;82:1463–76.
- Casiano RR, Livingston JA. Endoscopic Lothrop procedure: the University of Miami experience. Am J Rhinol. 1998;12:335–9.
- Close LG, Lee NK, Leach LJ, Manning SC. Endoscopic resection of the intranasal frontal sinus floor. Ann Otol Rhinol Laryngol. 1994;103:952–8.
- 5. Draf W. Endonasal micro-endoscopic frontal sinus surgery, the Fulda concept. Oper Tech Otolaryngol Head Neck Surg. 1991;2:234–40.
- 6. Gross CW. Surgical treatments for symptomatic chronic frontal sinusitis. Arch Otolaryngol Head Neck Surg. 1999;126:101–2.
- 7. Gross CW, Schlosser RJ. The modified Lothrop procedure: lessons learned. Laryngoscope. 2001;111:1302–5.
- 8. Gross CW, Zachmann GC, Becker DG, et al. Follow-up of University of Virginia experience with the modified Lothrop procedure. Am J Rhinol. 1997;11:49–54.
- 9. Gross WE, Gross CW, Becker D, et al. Modified transnasal endoscopic Lothrop procedure as an alternative to frontal sinus obliteration. Otolaryngol Head Neck Surg. 1995;113: 427–34.
- Hardy JM, Montgomery WW. Osteoplastic frontal sinusotomy: an analysis of 250 operations. Ann Otol Rhinol Laryngol. 1976;85:523–32.
- 11. Kountakis SE, Gross CW. Long-term results of the Lothrop operation. Curr Opin Otolaryngol Head Neck Surg. 2003;11:37–40.
- 12. Kuhn FA, Javer AR, Nagpal K, Citardi MJ. The frontal sinus rescue procedure: early experience and three year follow-up. Am J Rhinol. 2000;14:211–6.
- 13. Libersa C, Laude M, Libersa JC. The pneumatization of the accessory cavities of the nasal fossae during growth. Anat Clin. 1981;2:265–73.
- 14. Lothrop HA. Frontal sinus suppuration. Ann Surg. 1914;59:937-57.
- Metson R, Gliklich RE. Clinical outcome of endoscopic surgery for frontal sinusitis. Arch Otolaryngol Head Neck Surg. 1998;124:478–9.
- 16. Schlosser RJ, Zachmann G, Harrison S, et al. The endoscopic modified Lothrop: long-term follow-up on 44 patients. Am J Rhinol. 2002;16:103–8.
- 17. Weber R, Draf W, Kratzsch B, et al. Modern concepts of frontal sinus surgery. Laryngoscope. 2001;111:137–46.
- Chandra RK, Kennedy DW, Palmer JN. Endoscopic management of failed frontal sinus obliteration. Am J Rhinol. 2004;18(5):279–84.
- Chin D, Snidvongs K, Kalish L, Sacks R, Harvey RJ. The outside-in approach to the modified endoscopic Lothrop procedure. Laryngoscope. 2012;122(8):1661–9.
- Tran KN, Beule AG, Singal D, Wormald PJ. Frontal ostium restenosis after the endoscopic modified Lothrop procedure. Laryngoscope. 2007;117(8):1457–62.

- Hildenbrand T, Wormald PJ, Weber RK. Endoscopic frontal sinus drainage Draf type III with mucosal transplants. Am J Rhinol Allergy. 2012;26(2):148–51. doi:10.2500/ajra.2012.26.3731.
- 22. Anderson P, Sindwani R. Safety and efficacy of the endoscopic modified Lothrop procedure: a systematic review and meta-analysis. Laryngoscope. 2009;119(9):1828–33. doi:10.1002/lary.20565.
- Naidoo Y, Bassiouni A, Keen M, Wormald PJ. Long-term outcomes for the endoscopic modified Lothrop/Draf III procedure: a 10-year review. Laryngoscope. 2014;124(1):43–9.

## Chapter 27 Frontal Sinus Rescue

Martin J. Citardi

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#### **Core Messages**

- The frontal sinus rescue procedure, more formally known as revision endoscopic frontal sinusotomy with mucoperiosteal flap advancement, is a technique for the management of frontal sinus obstruction after middle turbinate resection.
- In this procedure, the frontal stenosis is addressed, and a small mucoperiosteal flap is advanced over the denuded region of the frontal neo-ostium.
- In this way, the normal frontal mucociliary clearance is restored to the frontal sinus that has been obstructed by previous middle turbinate resection.

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Texas Sinus Institute, University of Texas McGovern Medical School at Houston, 6431 Fannin Street, MSB 5.036, Houston, TX 77030, USA e-mail: Martin, J.Citardi@uth.tmc.edu • The procedure offers several distinct advantages in the setting of frontal recess stenosis related to previous middle turbinate resection, including preservation that lateral frontal recess mucosa and less trauma to the frontal recess than alternative procedures (such as the modified endoscopic Lothrop procedure and/or frontal sinus obliteration).

## Introduction

Since the late 1980s, endoscopic frontal sinusotomy has emerged as the preferred technique for the surgical management of chronic frontal sinusitis that is refractory to routine medical treatment [9]. During endoscopic frontal sinusotomy, the partitions of the cells that pneumatize the frontal recess are carefully identified and removed under endoscopic visualization. Throughout the procedure, mucosa is preserved, and the boundaries of the frontal recess are not disturbed. Thus, after endoscopic frontal sinusotomy, the frontal recess should be a widely patent, mucosa-lined structure with rigid walls.

If a frontal recess boundary is not fixed, then it may collapse into the frontal recess and cause secondary frontal recess/ostium stenosis. Today, this most commonly occurs after resection of the middle turbinate (which forms the medial boundary of the frontal recess). Because the middle turbinate remnant that remains after middle turbinate resection is often destabilized, it may fall laterally and compromise frontal recess patency. Simply stated, standard endoscopic frontal sinusotomy is often inadequate for the surgical treatment of chronic frontal sinusitis after middle turbinate resection, since the technique cannot compensate for frontal recess stenosis due to collapse of the middle turbinate. Standard endoscopic frontal sinusotomy requires that borders of the frontal recess are rigid and fixed; the technique removes various partitions from within the confines of the irregularly-shaped rigid box, known as the frontal recess. In the setting of previous medical turbinate resection, the medial wall of the frontal recess often collapses laterally compromising the volume of the frontal recess and thus limited the role of standard endoscopic techniques. In contrast, revision endoscopic frontal sinusotomy with mucoperiosteal flap advancement, termed the frontal sinus rescue procedure (FSR), has been developed as a modification of the standard endoscopic frontal sinusotomy technique [5, 10].

The important principles of FSR are as follows:

- FSR is not the creation of a simple hole to drain an obstructed frontal sinus.
- In FSR, bony and soft obstruction caused by the destabilized middle turbinate remnant is removed, and then a mucosal flap is advanced across the denuded bone at the medial aspect of the frontal ostium.
- Critical lateral frontal recess mucosa is preserved; thus mucociliary clearance is restored, since the normal drainage pattern is down the lateral aspect of the frontal recess.
- Of course, the entire FSR procedure is performed under endoscopic visualization.

#### **Historical Perspective**

In 1921, Lynch described the external frontoethmoidectomy procedure for management of frontal sinus disease [12]. It soon became apparent that frontal recess stenosis was the most important cause of failure of frontoethmoidectomy, and the failure rates were unacceptably high. This high failure rate was due in part to medial prolapse of the orbital contents as well as scaring at the frontal ostium/recess—a situation that is quite analogous to the scenario that may develop in the frontal recess after middle turbinate resection. In attempted to minimize this problem, Sewall, in 1935, developed a medially-based mucoperiosteal flap, which he used to reline the frontal opening [16]. In 1936, McNaught reported a variation on Sewall's strategy when he introduced a laterally-based mucoperiosteal flap, which was also used to reline the frontal opening [13]. In 1952, Boyden described his experience in 57 operations in which he had successfully employed the Sewall procedure [2]. The success of the mucoperiosteal flap for frontal sinus surgery has also been corroborated by the work of Ogura [14] and Baron [1].

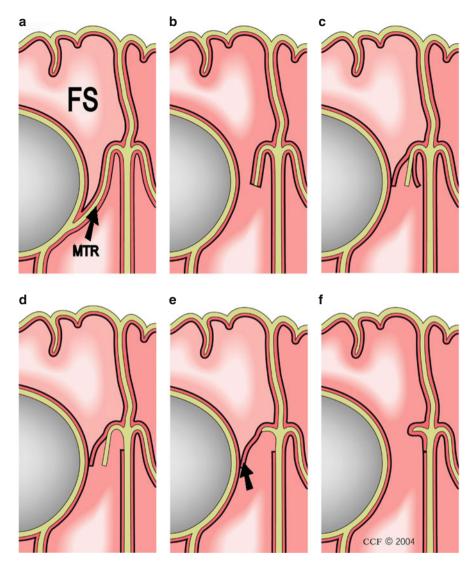
In the 1996–1997, Kuhn developed the FSR, and initial experiences were reported later [5, 10]. FSR builds upon the concepts outlined by Sewall, McNaught, Boyden and others who were able to use mucoperiosteal flaps to cover denuded areas of a surgically-enlarged frontal neo-ostium. Thus, the importance of avoid-ing exposed bony in the frontal recess has been recognized for many decades. Similarly, the ability of a mucoperiosteal flap to minimize secondary stenosis is not a new idea.

#### Technique

Most FSR procedures are performed with intraoperative surgical navigation [3, 4]. Review of the preoperative high-resolution sinus CT at the computer workstation greatly facilitates the comprehension of complex three-dimensional frontal recess anatomy, and this knowledge can be directly applied through intraoperative surgical navigation. Of course, the surgeon may rely upon CT scan images without surgical navigation; but this approach may be more difficult.

The procedure may be performed under general or local anesthesia with intravenous sedation. Achieving adequate levels of anesthesia with local blocks may be problematic; therefore, almost all patients require general anesthesia.

At the beginning of the surgery, detailed endoscopic examination of the nasal cavity with particular attention to the frontal recess region must be performed. Topical 0.05 % oxymetazoline provides significant mucosal decongestion with a relatively long-duration. Intraoperative surgical navigation may be invaluable during the initial diagnostic nasal endoscopy. Often gentle palpation may help to delineate critical anatomic features. The relationship of the middle turbinate remnant to the medial orbital wall must be established (Fig. 27.1a). In addition, the relative position of the skull base, including the cribriform plate should be determined.



**Fig. 27.1** This series of illustrations demonstrates the fundamental principles of the frontal sinus rescue procedure. In panel (**a**), the middle turbinate remnant (*MTR*) has scarred across the outflow tract frontal sinus (*FS*). In panel (**b**), the lateral attachment/adhesion of the middle turbinate remnant has been released. In panel (**c**), mucosa from the medial and lateral aspects of the middle turbinate remnant has been partially elevated. In panel (**d**), the mucosa from the medial aspect of the middle turbinate remnant has been resected, and the remaining mucosa from its lateral aspect has been preserved. This mucosa, indicated by the *arrow*, forms the mucoperiosteal flap. In panel (**f**), the mucosal flap has been advanced across the former middle turbinate attachment point

Because the frontal recess is far anterior along the skull base, the  $30^{\circ}$ ,  $45^{\circ}$  and  $70^{\circ}$  telescopes must be used to provide adequate visualization. A 0-degree telescope will not provide an adequate view of the area. After this initial examination is complete, the middle turbinate stub and adjacent medial orbital wall should be infiltrated with 1 % lidocaine with 1:100,000 epinephrine, which is used for its vasoconstrictive effect. Over infiltration of local anesthetic will tend to distort the soft-tissue anatomy, and suboptimal infiltration will be associated with greater mucosal oozing.

The initial step is a parasagittal incision along the most anterior aspect of the middle turbinate stub (Fig. 27.1b). A sickle knife may be passed above the  $30^{\circ}$ ,  $45^{\circ}$ or  $70^{\circ}$  telescope to achieve this objective. Alternatively, a frontal recess curette may be used to create a controlled tear, but this approach often induces unacceptable collateral tissue damage. Small, through-cutting giraffe forceps, which were introduced several years ago, provide a direct means to create this incision. The parasagittal incision releases the scar band that has pulled the middle turbinate laterally across the frontal recess outflow track. At this point, the middle turbinate stub should be apparent. This bony remnant may be directly attached to the skull base, or it may simply be encased in thickened, scarred mucosa. The mucosa along the medial and lateral aspects of the bony middle turbinate remnant is then gently elevated from the underlying bone (Fig. 27.1c). The medial mucosa, as well as a very small area of adjacent mucosa along the nasal roof (frontal sinus floor), is removed and discarded (Fig. 27.1d). The lateral mucosa flap is preserved; this mucosa is the mucoperiosteal flap for which FSR is formally named. After elevation, the mucoperiosteal flap mucosa is gently pushed superiorly so as to avoid inadvertent trauma and damage. Next, the bony middle turbinate stub must be removed (Fig. 27.1e). If it is merely a free fragment, then a non-cutting giraffe forceps may be used to grasp and take it from the operative field. If the bony middle turbinate stub is attached to the skull base, then it must be freed of that attachment and removed. Today, the throughcutting frontal giraffe forceps are ideally suited for this function. Alternatively, a frontal recess curette may be used to fracture the middle turbinate stub. Finally, the mucoperiosteal flap, which had been displaced superiorly, is repositioned over the former middle turbinate site (Fig. 27.1f). The raw surface of the underside of the flap faces the denuded bony of the middle turbinate removal site; these two surfaces are likely to stick together throughout the healing process.

In some patients, the mucosa on the medial aspect of the middle turbinate remnant is relatively thick and robust while the mucosa on the corresponding medial aspect is thinner. In this situation, the lateral mucosa may be too fragile for mobilization and manipulation. As a result, the lateral mucosa may be sacrificed and the medial mucosa can be preserved as flap. After preservation of the medial mucosa with sacrifice of the lateral mucosa, the bony middle turbinate remnant must be addressed in the same fashion as in standard FSR. This procedure, termed a reverse FSR (rFSR), addresses this limitation while preserving the critical mucosa of the lateral frontal recess. In most instances, a frontal recess stent should not be used, since the stent may displace the delicate flap and thus undo what the procedure aims to accomplish. Of course, in certain instances, a soft, low-caliber frontal recess stent will be appropriate.

It must be emphasized that the mucosa of the lateral frontal recess is not disturbed during a standard frontal sinus rescue procedure.

The natural mucociliary clearance process for the frontal sinus is along the lateral frontal recess; preservation of this mucosa helps to achieve restoration of frontal sinus function.

After FSR, thorough and comprehensive postoperative care must be performed [8]. Serial nasal endoscopy provides a simple means for monitoring the frontal neoostium as well as a platform for early intervention for the release of early fibrinous adhesions. Gentle debridement under endoscopic visualization is necessary. Acute suppurative exacerbations of chronic rhinosinusitis may be cultured and appropriate culture-directed antibiotics should be instituted. All patients should perform irrigations with isotonic or hypertonic saline solution. Some patients will also receive systemic corticosteroids for a few weeks. (Full discussion of the strategy for postoperative care is beyond the scope of this chapter.)

The steps for FSR are schematically illustrated in Fig. 27.1.

## Indications

Frontal sinus rescue was designed for the surgical treatment of chronic frontal sinusitis due to frontal recess obstruction after middle turbinate resection. Frontal sinus rescue compensates for the destabilized middle turbinate and secondary bony frontal ostium stenosis, while standard endoscopic frontal sinusotomy inadequately addresses these issues.

Concomitant issues may include frontal sinus/recess osteitis/osteoneogenesis, frontal bone osteomyelitis and mucocele with or without bony erosion. Since acute infection is associated with greater bleeding which may obscure visualization, FSR may not be feasible for the surgical management of acute frontal sinusitis requiring surgical drainage; however, consideration for FSR in this scenario may be appropriate. In addition, FSR may play a significant role in the surgical management of allergic fungal sinusitis involving the frontal sinus after middle turbinate resection, since FSR creates a patent functional tract that permits long-term endoscopic monitoring and debridement. Finally FSR may be incorporated into a surgical procedure that reverses prior frontal sinus obliteration.

In the situation of frontal sinusitis after middle turbinate resection, the central problem is obstruction of the frontal sinus outflow tract by residual bony and soft tissue scar. Both the modified endoscopic Lothrop procedure and frontal sinus obliteration have been presented as alternatives for surgical management of refractory

frontal sinusitis, due to frontal recess/ostium stenosis after middle turbinate amputation. It must be emphasized that both of these procedures carry significant morbidity, and the long-term prognosis after these procedures is often suboptimal.

Frontal sinus obliteration with autologous fat [6] may be complicated by perioperative morbidity, chronic pain and delayed mucocele formation [15]. Furthermore, the evaluation of the frontal sinus in a patient with persistent symptoms after fat obliteration is typically impossible, since MRI signal characteristics of the fat graft is inconsistent and mixed, even in the asymptomatic patient [11]. Frontal sinus obliteration focuses on the frontal sinus contents, but the real issue in frontal sinusitis is the frontal recess. As a result, frontal sinus obliteration is a misdirected procedure that destroys a potentially healthy frontal sinus.

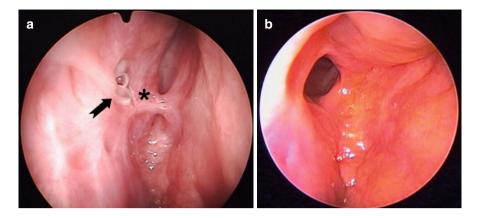
The modified endoscopic Lothrop procedure, a technique for the resection of frontal sinus floor under endoscopic visualization, has gained some popularity [7]. Although this is an endoscopic procedure, it is not minimally invasive. The frontal drillout inevitably causes significant frontal recess trauma, including destruction of mucosa, which leads to soft tissue and bony stenosis. The long-term impact of this procedure is unknown. Because frontal recess mucosa is inevitably disrupted by the modified endoscopic Lothrop procedure, normal frontal mucociliary clearance is often irreversibly altered. Thus, even a frontal sinus with a patent frontal neo-ostium after drillout may not clear its mucus appropriately.

FSR should be considered in the context of frontal sinus obliteration and modified endoscopic Lothrop procedure. FSR re-establishes normal frontal sinus/recess mucociliary clearance, while these other procedures tend to disrupt this physiology. In particular, frontal sinus obliteration destroys the frontal sinus, and the modified endoscopic Lothrop procedure destroys the frontal recess. FSR seeks to preserve these structures and promote normal sinus function.

### FSR Outcomes

The published surgical results demonstrate that FSR achieves frontal recess patency and function. In a preliminary report, relief from frontal recess scar and frontal ostium stenosis was achieved in 14 of 16 sides (87.5 %) in 12 patients with average follow-up of 8.5 months [5]. In an update of the initial publication, Kuhn noted frontal recess patency (confirmed by nasal endoscopy) and complete resolution of symptoms in 29 of 32 operative sides in 24 patients [10]. It should be noted that 18 sides were successfully treated with FSR on the first attempt, 7 sides required a revision FSR procedure, and 4 sides required 2 revision FSR procedures. Mean follow-up was 9.6 months, and one patient had long-term patency at 37 months.

Representative endoscopic images of the healed frontal recess after FSR are shown in Figs. 27.2 and 27.3.



**Fig. 27.2** (a) After middle turbinate resection, the remnant middle turbinate may scar the frontal recess, leading to formal frontal recess/ostium stenosis. In this endoscopic view of the right frontal recess, the frontal ostium (*arrow*) is stenotic, and middle turbinate remnant (indicated by the "\*") is simply encased in scar. Purulent secretions drain slowly from the narrowed frontal ostium. (b) This endoscopic image of the right frontal recess was obtained 8 years after FSR. The frontal neo-ostium is clearly patent and functional. The preoperative view of this frontal recess is shown in (a)

**Fig. 27.3** The FSR mucoperiosteal flap heals across the former insertion point for the destabilized middle turbinate stub. This endoscopic image of the left frontal recess shows a patent left frontal neo-ostium, 7 years after revision FSR. The flap (indicated by the "\*") has healed well, and the mucosa is quite healthy



### Advantages

FSR offers several distinct advantages:

- Because the lateral frontal recess mucosa is not disturbed by the procedure, mucociliary clearance is restored.
- FSR compensates for both bony and soft tissue stenosis induced by middle turbinate resection.

#### 27 Frontal Sinus Rescue

- FSR builds upon established techniques for endoscopic frontal sinusotomy, and FSR incorporates mucoperiosteal flaps that were first used as means to re-line surgically created frontal neo-ostia created via an external ethmoidectomy.
- The FSR mucoperiosteal flap minimizes the tendency for granulation tissue and stenosis.
- FSR is truly minimally invasive; in contrast, the alternative procedure of frontal sinus obliteration is quite extensive with significant morbidity.
- FSR is also a truly functional procedure; in contrast, the alternative procedure of modified endoscopic Lothrop causes significant frontal recess trauma that may ultimately compromise the final surgical result.
- Because FSR is not destructive to the frontal recess, surgical revision under endoscopic visualization is reasonably easy to pursue.

## Disadvantages

The potential disadvantages of FSR must be considered as well:

- The mucoperiosteal flap, which is the key feature of FSR, is extremely delicate. If the flap is disrupted, the procedure cannot be completed.
- FSR is a difficult technique. The entire procedure is performed with curved frontal recess instruments under the visualization provided by the angled telescopes.
- Endoscopic frontal recess instrumentation is required; in particular, fine throughcutting giraffe forceps greatly facilitate the procedure.
- The cribriform plate is just behind the site of the surgical manipulations. Hence, FSR carries the risk of skull based injury with concomitant cerebrospinal fluid leak.
- In some instances, revision FSR will be required.

## Conclusion

Frontal sinusitis after middle turbinate resection is a serious surgical challenge, for which standard endoscopic frontal sinusotomy is poorly suited. The frontal sinus rescue procedure, more formally known as revision endoscopic frontal sinusotomy with mucoperiosteal flap advancement, is uniquely designed for the correction of frontal stenosis due to middle turbinate resection. In this procedure, the bony and soft-tissue is removed, and the surgically enlarged frontal neo-ostium is relined with a small mucoperiosteal flap. Mucosa of the lateral frontal recess is preserved. In this way, frontal sinus rescue procedure may restore appropriate mucociliary clearance to a frontal sinus obstructed due to previous middle turbinate resection.

## References

1. Baron SH, Dedo HH, Henry CR. The mucoperiosteal flap in frontal sinus surgery (The Sewall-Boyden-McNaught operation). Laryngoscope. 1973;83:1266–80.

- Boyden GL. Surgical treatment of chronic frontal sinusitis. Ann Otol Rhinol Laryngol. 1952; 61:558–66.
- 3. Citardi MJ. Computer-aided frontal sinus surgery. Otolaryngol Clin N Am. 2001;34:111-22.
- 4. Citardi MJ. Computer-aided otorhinolarngology-head and neck surgery. New York: Marcel Dekker; 2002.
- Citardi MJ, Javer AR, Kuhn FA. Revision endoscopic frontal sinusotomy with mucoperiosteal flap advancement: the frontal sinus rescue procedure. Otolaryngol Clin N Am. 2001;34: 123–32.
- Goodale RL, Montgomery WW. Experience with osteoplastic anterior wall approach to frontal sinuses. Arch Otolaryngol. 1958;68:271–83.
- 7. Gross WE, Gross CW, Becker DG, et al. Modified transnasal endoscopic Lothrop procedure as an alternative to frontal sinus obliteration. Otolaryngol Head Neck Surg. 1996;113:427–34.
- Kuhn FA, Citardi MJ. Advances in postoperative care following functional endoscopic sinus surgery. Otolaryngol Clin N Am. 1997;30:479–90.
- Kuhn FA, Javer AR. Primary endoscopic management of the frontal sinus. Otolaryngol Clin N Am. 2001;34:59–75.
- Kuhn FA, Javer AR, Nagpal K, Citardi MJ. The frontal sinus rescue procedure: early experience and three-year follow-up. Am J Rhinol. 2000;14:211–6.
- 11. Loevner LA, Yousem DM, Lanza DC, et al. MR evaluation of frontal sinus osteoplastic flaps with autogenous fat grafts. Am J Neuroradiol. 1995;16:1721–6.
- 12. Lynch RC. The technique of radical frontal sinus operation that has give me the best results. Laryngoscope. 1921;31:1–5.
- 13. McNaught RC. A refinement of the external frontoehtmosphenoid operation: a new nasofrontal pedicle flap. Arch Otolaryngol. 1936;23:544–9.
- 14. Ogura JH, Watson RK, Jurema AA. Frontal sinus surgery: the use of a mucoperiostal flap for reconstruction of a nasofrontal duct. Laryngoscope. 1960;70:1229–43.
- 15. Sessions RB, Alford BR, Stratton C, et al. Current concepts of frontal sinus surgery: an appraisal of the osteoplastic flap fat obliteration operation. Laryngoscope. 1972;82:918–30.
- 16. Sewall EC. The operative technique of nasal sinus disease. Ann Otol Rhinol Laryngol. 1935;44:307-16.

## Chapter 28 The Frontal Sinus "Box": A Simple Anatomic Concept with Implications for Surgical Approaches

Brent A. Senior, Adam Campbell, Anthony Del Signore, and Mohamed Tomoum

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Envisioning the Frontal Sinus and the Frontal Recess as a Box Conclusions	387 390

#### **Core Messages**

- Dissection of the Frontal Recess may be challenging and detailed study of the anatomy should be undertaken prior to surgical dissection
- There is no one 'key' anatomic process in the frontal recess, but each cells contribution should be considered
- The concept of the frontal recess as a box with the opening of the frontal sinus occupying the top of the box allows the surgeon to envision the frontal recess and drainage pathway from every direction to allow both complete and safe dissection

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<sup>©</sup> Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_28

## Introduction

Completely addressing the frontal recess is considered one of the most difficult aspects of functional endoscopic sinus surgery. The challenge lies in the proximity of the frontal recess to the skull base, the lamina papyracea and the wide variation in anatomical configurations in addition to the technical limitations in addressing such an anteriorly located region. A lack of preparation and inadequate surgical planning and understanding can lead to injury of the skull base, orbit and/or anterior ethmoid arteries. Similarly nuances of anatomical variation between frontal sinus outflow tract and the surrounding pneumatized cells greatly change the approach and the degree of surgical difficulty.

• The inherent difficulty of frontal sinus surgery lies in the acute angle necessary to reach the frontal sinus outflow tract.

The presence and degree of pneumatization of surrounding structures, for example the agger nasi anteriorly, ethmoid bulla posteriorly and the intersinus septal cell medially, can further complicate exposure and potential access. Furthermore, it has been noted that the most common reason for frontal recess obstruction in the surgically naive patient or those with non traumatic obstruction are medial displacement of the uncinate process and enlargement of the agger nasi cell [5].

## **Embryological Origins**

Given the complexities associated with the frontal sinus a firm understanding of paranasal sinus anatomy and its embryologic origins is required. The sinonasal development begins during the eighth week of development, where five to six lateral nasal ridges (ethmoturbinals) initially appear with three to four ridges ultimately persisting.

The first ethmoturbinal regresses leaving its ascending portion as the agger nasi and the descending portion forms the uncinate process. The second and third ethmoturbinals form the middle and superior turbinate respectively. The fourth and fifth ethmoturbinals fuse to form the supreme turbinate. Meanwhile, between the first and second ethmoturbinals a furrow appears, contributing to the ascending aspect of the frontal sinus recess while the descending aspect forms that ethmoid infundibulum, hiatus semilunaris and the region of the middle meatus. The ethmoid bulla then forms from a secondary prominence from the lateral nasal wall with the associated suprabullar and retrobullar recesses creating the sinus lateralis.

The frontal sinus and frontal recess formation is highly complex and therefore multiple theories regarding its origins exist. One such theory proposed by Stammberger stated the frontal recess is formed from the continuation of the ascending pneumatization of the furrow between the first and second ethmoturbinals. It goes on further stating that the frontal sinus proper is due to anterior pneumatization of the recess into the frontal bone [7].

In contrast, Schaeffer proposed that multiple furrows form within the middle meatus ultimately forming the frontal recess, frontal sinus and anterior ethmoid cells [6]. Kasper expanded upon this theory suggesting that the first fold forms the agger nasi, the second fold forms the frontal sinus and the third and fourth furrows form the remaining anterior ethmoid cells [2]. In truth, any of these furrows could become the frontal sinus causing the highly variable anatomic relationship between the frontal sinus and nasal cavity.

#### **Pertinent Anatomy**

The first landmark identified upon entrance into the nose is the agger nasi lying anterior to the insertion of the middle turbinate along the lateral nasal wall. Typically the insertion is also along the superior aspect of the infundibulum or frontal recess [4]. The agger nasi region takes it origin from the embryologic first basal lamella, a flat plate of bone jutting off of the lateral nasal wall in the fetus. Frequently, this plate of bone becomes pneumatized leading to an agger nasi cell. When pneumatized, this cell's borders are formed anteriorly by the frontal process of the maxilla, superiorly by the frontal recess, laterally by the nasal bones and lacrimal bone and medially by the uncinate process. Occasionally if the cell's pneumatization pattern extends inferiorly, it can ultimately pneumatize the uncinate process. Conversely, if the cell pneumatizes superiorly or becomes stacked with multiple cells, these cells are termed "frontal cells" in the manner of Bent and Kuhn (Table 28.1). A Bent and Kuhn Type 1 Frontal cell involves a single cell sitting on the pneumatized agger nasi (Image 28.1). A Type 2 Frontal cell involves stacking of two or more cells on the agger nasi cell. A Type 3 Frontal cell involves a single large pneumatized cell over the agger nasi cell that pushes into the frontal sinus (Image 28.2). A Type 4 Frontal cell is an isolated cell pneumatizing within the frontal sinus without apparent connection to the agger nasi area below. Additionally, the frontal sinus septum can pneumatize creating an intersinus septal cell that typically drains unilaterally (Image 28.3).

Agger nasi cell
Supraorbital ethmoid cell
Type 1: Single frontal sinus cell above agger nasi
Type 2: Tier of frontal sinus cells above agger nasi
Type 3: Single ethmoidal cell extending superiorly into frontal sinus
Type 4: Isolated cell in the frontal sinus
Frontal bulla cells
Supra bulla cells
Intersinus septal cell

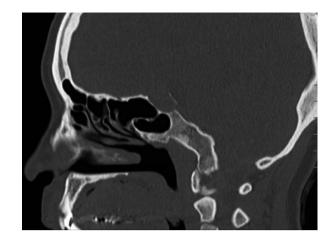
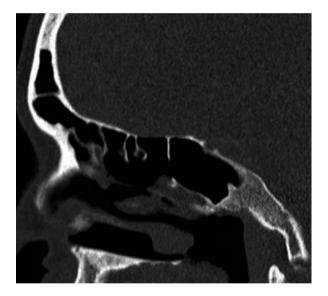


Image 28.2 Type 3 frontal cell

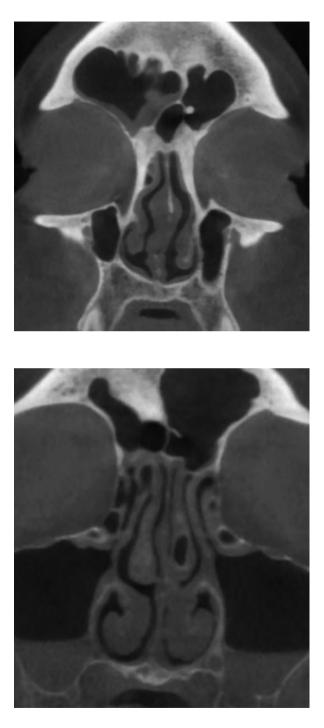


Moving posteriorly, the next structure that is encountered in coronal plane is the uncinate process. It is typically oriented in the sagittal plane and is a thin piece of bone along whose posterior border lies the hiatus semilunaris. Superiorly and posteriorly, it most often attaches to the lamina papyracea with the frontal sinus outflow typically lying superiomedially (Image 28.4). However, variations in the superior attachment exist, whereby it can also attach to either the skull base and/or the vertical aspect of the middle turbinate attachment near the cribriform plate, thus allowing for great variation in the position and diameter of the frontal outflow.

Next in the coronal plane is the ethmoid bulla. The ethmoid bulla takes its origin from the embryologic second basal lamella lying parallel to the first basal lamella and having its origin on the lamina papyracea. As this basal lamella pneumatizes, it forms

**Image 28.1** Agger nasi cell and type 1 frontal cell

Image 28.3 Intersinus septal cell



**Image 28.4** Uncinate attaching to lamina (patient left)

a ball of cells projecting medially into the middle meatus ("bulla"). Depending on the degree of pneumatization and cell development, it can project superiorly to the skull base delineating the posterior aspect of the frontal recess ("suprabullar cell"), or if pneumatizing anterior to the posterior wall of the frontal sinus along the skull base, a "frontal bullar cell."

Bordered in part by the ethmoid bulla, the space of the sinus lateralis, is defined as a region above and behind the bulla and below the skull base. Specifically, it consists of two discreet spaces, the suprabullar recess superiorly (between the bulla and the skull base) and retrobullar recess posteriorly (between the bulla and the basal lamella of the middle turbinate). In cases where the ethmoid bulla does not extend to the skull base to form the posterior aspect of the frontal recess, the sinus lateralis may then provide direct access to the frontal sinus. However, more commonly, the sinus lateralis and its suprabullar space may only be a potential space with the bulla having pneumatized cells extending to the skull base.

## **Popular Theories of Frontal Sinus Dissection**

Over the last several decades, various landmarks and different surgical approaches have been described in order to assist in the understanding of frontal sinus anatomy. For example, Stammberger described the removal of ethmoid cells squeezing up toward the frontal sinus in a posterior to anterior fashion as key to identification of the frontal sinus ostium, a technique that he described as "uncapping an egg" [7].

Significantly, Kuhn called into question the longstanding concept of the "nasofrontal duct" for the drainage of the frontal sinus, noting that the more appropriate understanding of the drainage area is a "recess" or region, rather than a defined tubular structure ("duct"). The primary tenet of his approach to the frontal sinus is that the mucus membrane of the recess be kept intact in order to prevent scarring, stenosis and osteoneogenesis. He asserts that the frontal sinusotomy should begin with an uncinectomy, ethmoid bullectomy and anterior or posterior ethmoidectomy to identify the skull base in a posterior to anterior approach. If a complete ethmoidectomy is to be performed due to ethmoid disease, then the procedure begins with identification of the sphenoid sinus and skull base posteriorly. The dissection is brought anteriorly to identify the anterior ethmoid artery, which marks the upward transition into the frontal recess. Curved frontal sinus curettes can be gently used to remove any frontal recess cells. If a large agger nasi cell is present, the posterior and superior aspects should be removed to allow frontal recess access. In the case of a supraorbital ethmoid cell, the common wall between this and the frontal recess must be removed as superiorly as safely possible in order to prevent future frontal sinus outflow restriction [3].

Wormald et al. proposed that the agger nasi cell was the basis on which to visualize the frontal recess, as it was present in approximately 98.5 % of patients [1, 8]. In the simplest case of frontal recess anatomy, a single large agger nasi cell, the anterior aspect (the axilla) of the cell is opened allowing further dissection of its medial, posterior and superior borders. As the agger nasi cell increases in size with further pneumatization, the uncinate is pushed medially to insert on the middle turbinate and the frontal sinus drainage pathway is pushed posteriorly. Yet if the agger nasi cell and associated ethmoidal cells have caused the uncinate to insert on the skull base, this should alert the surgeon that the olfactory fossa is just medial to the insertion of the uncinate. In combination with the knowledge of the frontal ethmoidal cells and the insertion of the uncinate, the authors feel that the location and size of the agger cell will lead to safe dissection of the frontal recess.

Wormald further explained a method of visualization of the anatomy of the frontal recess and frontal sinus by describing the anatomy with 'building blocks' [9]. In this explanation, the agger nasi cell, when present, is visualized first in the coronal plain and then its size and position confirmed using axial and parasagittal plains. As additional cells surrounding the agger nasi cell are identified, they are added atop and around the agger and numbered appropriately. Once a visual or mental 3D representation of the frontal recess cells has been developed, then the frontal sinus drainage pathway can be evaluated. The method for which to complete this involves evaluating the sinus in the axial plain and following the drainage pathway inferiorly through the scans. Much like the 3D representation, this pathway can be confirmed using the parasagittal and coronal scans. Intraoperatively, this should allow for the surgeon to have a surgical plan for each cell of the frontal recess and assist in avoiding iatrogenic injury.

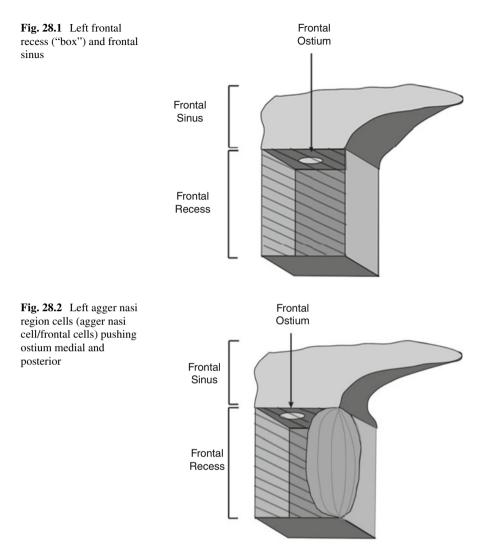
#### Envisioning the Frontal Sinus and the Frontal Recess as a Box

In sagittal view, the frontal sinus and its drainage region appears as an hourglass shaped structure. Superiorly the frontal sinus narrows down to its ostium, draining into the larger frontal recess below. All of the structures discussed above contribute to the frontal recess and it is the complexity of this frontal recess that ultimately results in the anatomic confusion associated with frontal sinus dissection.

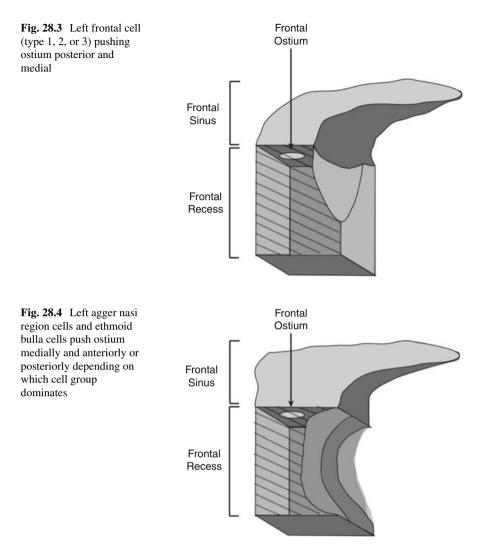
• The embryology of various structures in addition to the variability in the degree of pneumatization and position of these different cells and associated spaces will push the frontal outflow into different positions anteriorly, posteriorly, medially, and laterally. It is with this idea in mind that we developed the concept of the "frontal sinus box" in order to understand this complex anatomy of the frontal recess and thereby to facilitate effective and safe surgery in this region.

Specifically this concept provides the surgeon with a facile way to determine based on review of the preoperative CT images, how various pneumatization patterns will affect the predicted location of the frontal sinus outflow.

Imagine the frontal recess as a six-sided box with the frontal floor and ostium occupying the top side of the box. The lateral wall of the box is the lateral wall of the nose, while the medial wall of the box is the middle turbinate (Fig. 28.1). Anterior and posterior walls are made up of cells in these respective areas: agger

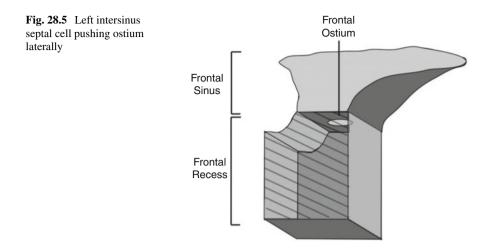


nasi region cells anteriorly (frontal cells); bullar cells posteriorly. Each of the corners of the box is going to be squeezed and pushed by cells occupying that area, thereby narrowing and pushing the opening of the frontal sinus above in different directions. How much each of the corners of the box is pushed is going to depend on the presence and degree of pneumatization of these different cells and spaces. For example, cell groups squeezing the lateral anterior corner of the box, pushing the opening of the frontal sinus posteriorly and medially, will arise from the agger nasi region (agger nasi cell and type 1, 2, or 3 frontal cells) (Figs. 28.2 and 28.3). In contrast, cells occupying the posterior lateral corner will arise from the ethmoid bulla and will push the frontal sinus opening anteriorly and medially. In effect these two corners will begin to push against one



another, and it is the degree of "pushing" from these two corners that will ultimately determine the exact anterior/posterior position of the frontal ostium (Fig. 28.4).

While this helps us to understand the anterior posterior position of the frontal ostium, it is important to remember that both of these cell groups will also tend to push the frontal ostium medially, and it is this common configuration that leads to commonly held adage that "the drainage of the frontal sinus is always medial." Presence of an inter-sinus septal cell in the midline, however, will tend to push the frontal ostium laterally and posteriorly (Fig. 28.5 and Image 28.3). In addition, the superior attachment of the uncinate is also important in accurately locating the medial/lateral opening of the frontal sinus. Commonly, the uncinate is found



attaching to the lamina papyracea, resulting in the frontal ostium being pushed medially (Image 28.4). Conversely the frontal recess will be displaced laterally if the uncinate process attaches to the middle turbinate.

## Conclusions

Over the past 30 years, endoscopic sinus surgery has proven to be highly successful with decreased morbidity in the treatment of medically refractory chronic rhinosinusitis. Yet, given the limitations of rigid endoscopes to properly visualize the frontal recess and frontal sinus ostia, this area continues to prove difficult to safely access. Therefore, proper treatment and surgical dissection necessitates a proper understanding of the individual patients surgical anatomy and the implications this has on the approach to each individual frontal recess. In this chapter, we have detailed an encompassing theory of frontal recess anatomy that will help the surgeon both understand how the frontal recess develops but how pneumatization patterns can effect the disease state, drainage pathway and surgical approach. It is our experience that visualization of the recess in this fashion can allow for a safe and thorough approach to the frontal recess.

## References

- Bolger WE, Butzin CA, Parsons DS. Paranasal sinus bony anatomic variations and mucosal abnormalities: CT analysis for endoscopic sinus surgery. Laryngoscope. 1991;101(1 Pt 1): 56–64.
- 2. Kasper KA. Nasofrontal connections: a study based on one hundred consecutive dissections. Arch Otolaryngol. 1936;23:322–44.

- Kuhn FA, Javer AR. Primary endoscopic management of the frontal sinus. Otolaryngol Clin N Am. 2001;34(1):59–75.
- 4. Loury MC. Endoscopic frontal recess and frontal sinus ostium dissection. Laryngoscope. 1993;103(4 Pt 1):455-8.
- Orlandi RR, Kennedy DW. Revision endoscopic frontal sinus surgery. Otolaryngol Clin N Am. 2001;34(1):77–90.
- Schaeffer JP. The nose, paranasal sinuses, nasolacrimal passageways and olfactory organ in man: a genetic, developmental, and anatomico-physiological consideration. Philadelphia: P. Blakiston's Son; 1920.
- 7. Stammberger H. Functional endoscopic sinus surgery. Philadelphia: BC Decker; 1991.
- 8. Wormald PJ. The agger nasi cell: the key to understanding the anatomy of the frontal recess. Otolaryngol Head Neck Surg. 2003;129(5):497–507.
- 9. Wormald PJ. Surgery of the frontal recess and frontal sinus. Rhinology. 2005;43(2):82-5.
- 10. Kuhn FA. Chronic frontal sinusitis: the endoscopic frontal recess approach. Oper Tech Otolaryngol Head Neck Surg. 1996;7:222–9.

## Chapter 29 Frontal Sinus Stenting

Calvin C. Wei, Seth J. Kanowitz, Richard A. Lebowitz, and Joseph B. Jacobs

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_29

#### **Core Messages**

- Post-operative stenting of the frontal sinus outflow tract has been demonstrated to improve long-term patency rates.
- Soft (Silicone) sheets or stents are superior to rigid stents.
- A minimum 6-week period of stenting is generally recommended.
- Routine care after stent placement includes appropriate antibiotic therapy, nasal irrigation, gentle debridement, and topical nasal steroid spray.

#### Introduction

The concept of frontal sinus stenting to minimize post-operative stenosis and improve mucosalization of the frontal sinus outflow tract (FSOT) following frontal sinus surgery has been reported in the literature for nearly 100 years. The external fronto-ethmoidectomy, as originally described by Lynch, involved post-operative stenting of the nasofrontal communication. Technological advances in sinus endoscopes, surgical instruments, high resolution computed tomographic (CT) scanning, and image guidance have allowed for improved visualization and intranasal surgical access to the nasofrontal region. However, despite these advances, post-operative stenosis of the FSOT with recurrent frontal sinus disease remains a significant problem.

Factors such as polyposis, osteitic bone, and lateralization of the middle turbinate/middle turbinate remnant may lead to FSOT stenosis, regardless of the surgical approach and the adequacy of the frontal sinusotomy. Failure rates of nearly 30 % have been reported in the literature – and because of this propensity for postoperative stenosis of the FSOT, stenting remains an important component in the surgical management of chronic frontal sinusitis.

#### **Stenting Materials**

The concept of frontal sinus stenting dates back to 1905 when Ingals reported the use of a gold tube, placed endonasally, to help stent the surgical bed until the naso-frontal duct was mucosalized [1]. In 1921, in the initial description of the fronto-ethmoidectomy procedure that now bears his name, Lynch placed a firm rubber tube in the nasofrontal duct to help maintain patency [2]. The stent remained in place for 5 days post-operatively. Lynch initially reported a 100 % success rate in 15 patients treated with this technique, and followed for a period of 2.5 years. Unfortunately, the long-term failure rate for this procedure was found to be approximately 30 % [3, 4].

In the 1940s and 50s, Goodale, Harris, and Scharfe described their experiences with the use of tantalum for frontal sinus stenting [5–8]. Originally discovered by Eckenberg in 1902, tantalum is an inert basic element. Goodale described the use of a thin sheet of tantalum sutured to the orbital periosteum, while Harris and Scharfe both employed tantalum tubes extending from the frontal sinus into the nose. In their

series, the authors reported success rates that were superior to the classic Lynch operation with decreased scarring of the nasofrontal duct, improved epithelialization, and decreased granulation tissue formation. Metson employed similar techniques and tantalum foil for drainage of frontal sinus mucoceles, but added an acrylic tube for mucoceles with intracranial extension [9]. In 1972, Barton described similar results in 34 patients implanted with a 6 or 8 millimeter (mm) Dacron<sup>©</sup> Woven Arterial Graft sutured into the frontal sinus floor and extending downward into the middle meatus [10]. None of the implants were removed during the 17-year study period and all of the patients were relieved of their frontal headache symptoms.

Initially, most surgeons used rigid frontal sinus stents. However, in animal and clinical trials published in 1976, Neel demonstrated the superiority of thin, pliable Silastic© sheeting [3, 4]. He reported a 29 % failure rate with rubber tubing and a 17 % failure rate with thin Silastic© sheeting, in patients followed for an average of 13.5 years postoperatively. In his canine model, Neel demonstrated significant fibrosis and osteoblastic activity, with little or no epithelialization, in nasofrontal ducts that had been stented with firm rubber stents. In contrast, a normal mucosal lining was observed on histological specimens in ducts stented with thin Silastic© sheeting. The difference was felt to be due to local ischemia, impaired drainage, and infection around the rigid tubes.

Schaefer and Close employed Silastic<sup>®</sup> tubing for small endoscopic frontal sinusotomies (4–6 mm) in four of 36 patients treated [11]. However, a 50 % stenosis rate resulted, which was attributed to a failure to maintain a post-operative communication between an air passage and the mucosa, thus resulting in massively hypertrophied mucosa and obstruction of the frontal sinus ostium. More recently, numerous authors have described the use of a variety of Silicone tubes, as well as rolled Silicone sheeting, placed either externally or endoscopically, to help maintain patency of the nasofrontal duct [12–22].

Bioabsorbable, steroid-eluting stents have recently been developed and have been found to be efficacious in published clinical trials. The Propel mometasoneeluting stent (Intersect ENT, Palo Alto, CA) is the first FDA-approved device for direct delivery of steroid medication into the ethmoid and frontal sinus cavity following surgery. It is composed of a biodegradable polymer in a lattice pattern which contains a total of 370 micrograms of mometasone furoate designed for gradual controlled release over 30 days. The stents are resorbed in a predictable fashion. Approximately 75 % is absorbed after 14 days and by day 30, an average of less than 10 % of the stent material remains in the sinus cavity [23]. The mometasone implant simultaneously stents open the sinus cavities mechanically and delivers topical steroids to the postoperative sinus cavities with a controlled rate of delivery. As a scaffold, the ethmoid stent maintains medialization of the middle turbinate and prevents the development of scarring between the middle turbinate and the lateral nasal wall. The frontal sinus stent prevents mucosal scarring and inflammation at the frontal sinus ostium which is prone to stenosis. Three major studies have demonstrated the effectiveness of the mometasone implant [23–25].

• A prospective, multicenter, randomized, double-blind pilot study was performed by Murr et al. which demonstrated that the mometasone-eluting stent demonstrated a statistically significant reduction in inflammation at days 21–45 after the stent was deployed, and decreased the frequency of polyp formation and the development of mucosal adhesions.

- Forwith et al. then performed a prospective, multicenter, single-cohort clinical trial (ADVANCE) [24]. The study demonstrated that the use of the mometasone implant provided minimal mean ethmoid sinus inflammation scores and low rates of polypoid tissue formation, adhesion formation and middle turbinate lateralization. This study also demonstrated statistically significant patient-reported outcome scores (Rhinosinusitis Disability Index and Sinonasal Outcome Test-22) with the use of the stent.
- A third study (ADVANCE 2), a prospective, multicenter, randomized, doubleblind trial, utilized used an intrapatient design to determine if the use of the mometasone stent would decrease the use of postoperative oral steroids and lysis of post-surgical mucous adhesions [25].
- The mometasone implant provided a 29.0 % relative reduction in postoperative interventions (P=0.028) and a 52 % (P=0.005) decrease in lysis of adhesions. The relative reduction in frank polyposis was 44.9 % (P=0.002).

These studies provide a high level of evidence that steroid-eluting implants improve surgical outcomes by decreasing mucosal adhesions, polyposis and the need for postoperative interventions.

#### **Indications for Stenting**

There are no standard, accepted indications for post-operative stenting of the frontal sinus outflow tract. Routine stenting is not advocated, and the decision to place a frontal sinus stent is based on the surgeon's assessment of the patient's risk for stenosis of the frontal sinus outflow tract. A number of conditions need to be considered as risk factors for FSOT stenosis, and thus, as potential indications for stenting.

Hosemann demonstrated a doubling (16 % vs. 33 %) of the rate of FSOT stenosis when the intraoperative diameter of the neo-ostium was <5 mm [16]. Therefore, a FSOT diameter of less than 5 mm is often considered an indication for stenting. Other indications include extensive demucosalization, particularly with circumferential exposure of bone, at the level of the frontal sinus ostium; osteitic bone (as determined by pre-operative CT) in the FSOT; extensive polyposis (as is often seen in patients with allergic fungal sinusitis (AFS)); flail middle turbinate, particularly in cases of partial middle turbinate resection; and revision frontal sinus surgery with pre-operative scarring or lateralization of the middle turbinate.

With the development of additional frontal sinus surgical techniques, including the Draf III/Endoscopic Modified Lothrop Procedure (EMLP), the insertion of stents into the frontal sinus outflow tract for up to 6 months in cases with a narrow drainage pathway may significantly improve postoperative patency of the frontal neo-ostium. Stents utilized include soft silicone (Vostra, Aachen, Germany), the Rains Frontal Sinus Stent (Smith & Nephew, Memphis, TN) or the Parell T-Stent (Xomed) [26, 27].

## **Indications for FSOT Stenting**

- · Frontal sinus neo-ostium diameter less than 5 mm
- · Extensive or circumferential exposure of bone in the FSOT
- Polyposis/AFS
- Flail/lateralized middle turbinate
- Revision frontal sinus surgery

## **External Versus Endoscopic Approach**

The initial works of Lynch, Goodale, Harris, and Scharfe predated the availability of fiberoptic nasal endoscopes, and endoscopic sinus instrumentation. Therefore, the techniques of those authors involved an external approach to the frontal sinus and placement of the stent. As the surgical management of frontal sinus disease shifts from external to endoscopic approaches, the techniques of frontal sinus stenting have changed as well (Figs. 29.1, 29.2, and 29.3).

However, some authors still report the use of an external approach for the placement of a frontal sinus stent. Barton employed a modified Lynch external frontal sinusotomy for the placement of a Dacron<sup>©</sup> graft with a reported 100 % success rate for relief of frontal headache symptoms [10]. Neel also employed a modified Lynch external approach (Neel-Lake) for the placement of thin Silastic<sup>©</sup> sheeting to stent the nasofrontal duct. In 13 patients (14 ducts), there was one (7 %) short-term failure at 4 months, which was treated with frontal sinus obliteration. After an additional 7 years of observation, the overall failure rate was 20 % (3 ducts), with both longterm failures being successfully treated with revision frontal sinusotomy [3, 4].



**Fig. 29.1** Endoscopic view of stenotic right frontal sinus neo-ostium

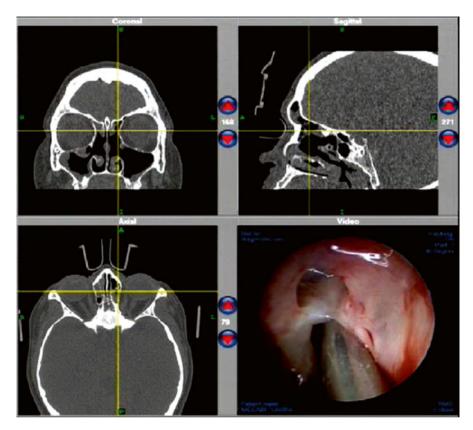


Fig. 29.2 Intraoperative image guidance with probe in stenotic left frontal sinus outflow tract

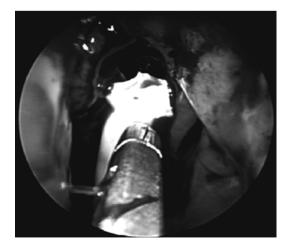


Fig. 29.3 Endoscopic placement of silastic stent in left frontal sinus outflow tract

Using a similar external approach in 18 patients who failed a previous transnasal widening of the nasofrontal communication, Yamasoba placed a Silicone T-tube in the frontal sinus outflow tract [22]. Complete epithelialization of the nasofrontal communication, and resolution of symptoms was reported in all patients after tube removal. Two patients subsequently suffered closure of the FSOT. More recently, Amble placed thin silicone rubber sheeting to reconstruct the nasofrontal drainage after a modified external Lynch procedure in which the frontal process of the superior maxilla was preserved [12]. Of the 164 patients studied, 96 % achieved resolution of their symptoms.

In 1990, Schaefer and Close first reported their experience with endoscopic placement of thin Silastic<sup>®</sup> tubing as a frontal sinus stent, resulting in a 50 % failure rate in the four patients studied [11]. Employing three different kinds of stents (Rains© self-retaining silicone tube, U-shaped silicone tube, and H-shaped silicone tube) and various Draf endoscopic frontal sinus drainage procedures in 12 patients, Weber reported complete resolution or significant improvement in ten patients' frontal sinus symptoms, and moderate improvement in two patients. However, while clinically significant stenosis of the FSOT did not occur, stenting could not prevent the recurrence of endoscopically or radiographically visible polypoid mucosal disease [21]. Hoyt reported similar results in 21 patients (32 stents) who had vented tubular plastic stents placed endoscopically [17]. Freeman placed a biflanged Silicone tube (Freeman<sup>©</sup> frontal sinus stent) endoscopically in 55 sinuses and externally in nine sinuses with follow-up of 12-45 months [14]. Six sinuses eventually required fat obliteration, four due to restenosis secondary to lateralization of the middle turbinate with scarring, and two due to the development of frontal sinus polyps. Rains also employed a soft Silicone tube with a tapered collapsible bulb placed endoscopically in 67 patients. With a total of 102 stents placed, and follow-up of 8-48 months, a failure rate of 6 % was reported. Allergic fungal sinusitis was present in all cases requiring revision [20].

Ultimately, the success of all non-obliterative frontal sinus surgery, whether external or endoscopic, is judged by the long-term functional patency of the FSOT. In many instances the FSOT may not be visibly patent, but can be endoscopically probed in asymptomatic patients [18].

#### **Preoperative Assessment for FSOT Stenting**

- Carefully review the sinus anatomy on CT to determine the potential surgical diameter of the frontal sinus neo-ostium, as limited by the frontal beak, anterior skull base, medial orbit, and cribriform plate.
- Evaluate the pre-operative CT for radiographic evidence of AFS, and/or osteitis of the bone of the FSOT.
- Perform a thorough nasal endoscopic examination with particular attention to polyposis in the frontal recess, scarring from prior surgery, and previous partial middle turbinectomy.

## **Duration of Stenting**

Currently no prospective controlled studies or definitive standards for the duration of frontal sinus stenting exist in the literature. Stenting duration ranges from as little as 5 days to as long as 17 years, however, most recommendations fall somewhere in-between [2, 10].

Neel demonstrated, histologically, that re-epithelialization of the nasofrontal drainage pathway of canines stented with thin Silicone rubber is complete within approximately 8 weeks. Based upon this work, Neel removed Silastic<sup>©</sup> sheeting stents in his patients beginning after a minimum of 6 weeks (mean 6 months). This resulted in a failure rate of 20 % with a 7-year follow-up period [3].

Employing a 6-week duration of stenting using 4 mm Silastic<sup>®</sup> tubing, Schaefer encountered a 50 % failure rate. However, this technique was only utilized in four patients, and failure was attributed to the extent of frontal sinus disease, lateral extent of the disease, and the difficulty in placing the catheter within the frontal sinus [11]. Benoit employed the Rains<sup>®</sup> frontal sinus stent for an average of 5 weeks with a FSOT patency rate of 79 % at 12 months follow-up [13]. Rains reported a 96 % patency rate at 48 months follow-up, with the same stent and the same average duration of stenting (5 weeks; range 6–130 days) [20]. Hoyt removed the plastic tubing stenting material at 8 weeks with a failure rate of 9.5 % in 21 patients, but follow-up was unspecified and limited [17]. Similarly, Amble removed the Silicone rubber sheeting between 6 and 8 weeks post-operatively in most patients with an 18 % revision rate at an average of 47 months follow-up.

Citing improved patency with a longer duration of stenting, Weber recommended removal of the Rains<sup>©</sup> frontal sinus stent, U-shaped Silicone tube, and H-shaped Silicone tube at 6 months. In the 15 sinuses available for evaluation at an average of 19.4 months after surgery, no relevant restenosis of the FSOT was appreciated with this longer period of stenting [21]. Freeman also described a period of stenting lasting between 6 and 12 months for patients' stented to correct FSOT stenosis, while a period of 4 weeks was employed for those used to prevent post-operative stenosis [14]. Whenever stent removal is deemed appropriate, all authors report successful removal of the stenting material in the office using endoscopes and endoscopic sinus instrumentation.

#### **Postoperative Stent Management**

Most authors agree that regular debridement and irrigation of the nasal cavity and stent, regardless of material and placement technique, are necessary to maintain stent patency, minimize scarring and adhesions, and improve long-term results. Even during the early days of frontal sinus stenting, Goodale and Harris routinely probed and cleaned the tantalum tubes with a curved suction [5, 6, 15]. While the

trials examining the efficacy of the bioabsorbable steroid-eluting stents have not dictated a specific postoperative management procedure, regular debridement of the sinus in which the stent has been deployed is advocated for optimal surgical results. Crusting and blood clot between the interstices of the stent in addition to the sinus cavities themselves should be meticulously removed to reduce fibrin deposition and the likelihood of scar formation. For a steroid-eluting stent placed within the middle meatus, the middle turbinate should be carefully monitored in the postoperative period to ensure that it heals without scarring to the lateral nasal wall, a common source of surgical failure. For a steroid-eluting stent placed in the frontal recess, thorough debridement of the frontal sinus outflow tract should be diligently performed to prevent mucosal scarring and possible frontal sinus stenosis.

Nasal irrigation usually begins within the first few post-operative days and is maintained for at least the duration of stenting. Amble employed a regimen of nasal irrigation two to three times daily, twice daily placement of petroleum jelly into the nasal cavity, broad-spectrum antibiotics for 10–21 days, and the application of a heating pad for 30 min two to three times daily [12]. Routine post-operative endoscopic removal of blood clots, debris, dried secretions, and granulation tissue from within the nasal cavity and within the stent itself, is performed in the office as needed.

The use of topical and/or oral steroids has been recommended to reduce postoperative inflammation and scar formation. Weber advocated saline nasal irrigation and a 6-month course of topical inhaled nasal steroids [21]. Rains initiated inhaled topical nasal steroids at 2–3 weeks after surgery, with oral steroids prescribed when marked polypoid disease is present [20].

Appropriate antibiotic therapy is also recommended, but not for the entire duration of longer stenting periods. However, if an episode of acute frontal sinusitis occurs it should be treated accordingly with antibiotics. If purulent drainage persists despite appropriate medical therapy, the stent may act as a foreign body and consideration should be given to removing it.

#### Conclusion

Frontal sinus stenting has demonstrated the ability to improve FSOT patency in specific cases; however, failure rates of approximately 30 % still persist. Long-term patency is improved with the use of soft Silicone sheets or stents as opposed to rigid stenting material. While duration of stenting varies widely in the literature, an average of approximately 6 weeks is generally accepted. Routine endoscopic debridement, nasal irrigation, appropriate antibiotic therapy, and topical nasal spray are important to help maintain stent patency. Acute episodes of frontal sinusitis during stenting should be treated appropriately, and if purulent discharge persists, consideration should be given to removing the stent.

## References

- 1. Ingals EE. New operation and instruments for draining the frontal sinus. Tr Am Laryng Rhin Otol Soc. 1905;11:183–9.
- Lynch RC. The technique of radical frontal sinus surgery operation which has given me the best results. Laryngoscope. 1921;31:1–5.
- Neel HB, McDonald TJ, Facer GW. Modified Lynch procedure for chronic frontal sinus diseases: rationale, technique, and long-term results. Laryngoscope. 1987;97:1274–9.
- 4. Neel HB, Whicker JH, Lake CF. Thin rubber sheeting in frontal sinus surgery: animal and clinical studies. Laryngoscope. 1976;86:524–36.
- 5. Goodale RL. Ten years' experience in the use of tantalum in frontal sinus surgery. Laryngoscope. 1954;64:65–72.
- Goodale RL. The use of tantalum in radical frontal sinus surgery. Ann Otol Rhinol Laryngol. 1945;45:757–62.
- 7. Harris HE. The use of tantalum tubes in frontal sinus surgery. Cleve Clin Q. 1948;15:129-33.
- 8. Scharfe ED. The use of tantalum in otolaryngology. Arch Otolaryngol. 1953;58:133-40.
- 9. Kaplan S, Schwartz A, Metson BF. Mucocele of the frontal and ethmoid sinuses. Arch Otolaryngol. 1950;51:172–87.
- 10. Barton RT. Dacron prosthesis in frontal sinus surgery. Laryngoscope. 1972;82:1795-802.
- 11. Schaefer SD, Close LG. Endoscopic management of frontal sinus disease. Laryngoscope. 1990;100:155–60.
- Amble FR, Kern EB, Neel B, et al. Nasofrontal duct reconstruction with silicone rubber sheeting for inflammatory frontal sinus disease: analysis of 164 cases. Laryngoscope. 1996;106: 809–15.
- 13. Benoit CM, Duncavage JA. Combined external and endoscopic frontal sinusotomy with stent placement: a retrospective review. Laryngoscope. 2001;111:1246–9.
- 14. Freeman SB, Blom ED. Frontal sinus stents. Laryngoscope. 2000;110:1179-82.
- 15. El Har G, Lucente FE. Endoscopic intranasal frontal sinusotomy. Laryngoscope. 1995;105: 440–3.
- 16. Hosemann W, Kuhnel TH, Held P, et al. Endonasal frontal sinusotomy in surgical management of chronic sinusitis: a critical evaluation. Am J Rhinol. 1997;11:1–19.
- Hoyt WH. Endoscopic stenting of nasofrontal communication in frontal sinus disease. Ear Nose Throat J. 1993;72:596–7.
- 18. Jacobs JB. 100 years of frontal sinus surgery. Laryngoscope. 1997;107:1-36.
- 19. Mirza S, Johnson AP. A simple and effective frontal sinus stent. J Laryngol Otol. 2000;114: 955–6.
- 20. Rains BM. Frontal sinus stenting. Otolaryngol Clin North Am. 2001;34:101-10.
- Weber R, Mai R, Hosemann W, et al. The success of 6-month stenting in endonasal frontal sinus surgery. Ear Nose Throat J. 2000;79:930–2,934, 937–8, 940–1.
- 22. Yamasoba T, Kikuchi S, Higo R. Transient positioning of a silicone T tube in frontal sinus surgery. Otolaryngol Head Neck Surg. 1994;111:776–80.
- Murr AH, Smith TL, Hwang PH, et al. Safety and efficacy of a novel bioabsorbable, steroideluting sinus stent. Int Forum Allergy Rhinol. 2001;1(1):23–32.
- Forwith KD, Chandra RK, Yun PT, et al. ADVANCE: a multisite trial of bioabsorbable steroideluting sinus implants. Laryngoscope. 2011;121(11):2473–80.
- Marple BF, Smith TL, Han JK, et al. Advance II: a prospective, randomized study assessing safety and efficacy of bioabsorbable steroid-releasing sinus implants. Otolaryngol Head Neck Surg. 2012;146(6):1004–11.
- 26. Weber R, Draf W, Kratzsch B, et al. Modern concepts of frontal sinus surgery. Laryngoscope. 2001;111(1):137–46.
- 27. Weber R, Hosemann W, Draf W, et al. Endonasal frontal sinus surgery with longterm stenting of the nasofrontal duct. Laryngorhinootologie. 1997;76:728–34.

## Chapter 30 Outcomes After Frontal Sinus Surgery

Michael G. Stewart and Aaron Pearlman

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_30

#### **Core Messages**

- Assessing results in the treatment of the frontal sinus is difficult in that objective and subjective outcomes must be considered, and often, do not correlate with one another.
- Subjective disease-specific quality of life instruments have been standardized and validated in assessing chronic rhinosinusitis, but none are specific to the frontal sinus.
- Objective outcomes such as CT scan findings, endoscopic examination, microbiologic data, and the need for further medical care may play a role in the outcomes of frontal sinus disease.
- The various disease processes that make frontal sinus surgery necessary and the type of surgery performed on the frontal sinus will affect the ability to predict outcomes.

## Introduction

Assessment of outcome after sinus surgery has not been uniformly defined or standardized, and assessment is best considered to be multifactorial. Objective outcomes, such as endoscopic examination findings, CT scan findings, or surgical revision rate, are important to assess. Subjective outcomes, such as symptoms and quality of life (QOL), are also important. Furthermore, there is good evidence that the objective and subjective data in rhinosinusitis do not usually correlate well. Therefore, the assessment of both subjective and objectives outcome is important. After frontal sinus surgery – and particularly some types of frontal sinus surgery – these issues are particularly relevant, since the anatomy is invariably altered, and may remain "abnormal" even with resolution of symptoms.

In addition to discussing *how* to assess outcome, we should consider *when* to assess outcome. In some diseases, long-term outcomes are invariably preferred over short-term outcomes. However in many chronic relapsing conditions, even if the long-term outcome is not to cure, improvements in short-term status are still worth-while and should be pursued.

## Outcomes Assessment in General Chronic Rhinosinusitis

There is no reason to believe that assessment of outcome in frontal sinusitis should be any different than outcome in rhinosinusitis involving different sinuses. Fortunately, much prior work has already been performed in this area, which will be reviewed herein. There has actually been little work specifically on frontal sinusitis, or outcomes assessment after frontal sinus surgery, but that is not a major issue. Symptoms and QOL should still be important outcomes to assess, and are likely a major driver of patient behavior after surgery – like they are before surgery. In addition, the impact of sinusitis on symptoms and severity of other diseases, such as asthma, is likely still important. However, objective outcome assessment, in particular CT scan findings, might require a modified interpretation after revision surgery. There are limited data on CT mucosal changes that should be expected after surgical intervention. Landmarks have been removed and anatomy altered, but in fact the degree of mucosal changes, and the response to medical treatment, may be different in post-surgical sinuses. For example, some degree of underlying mucosal thickening might be expected and should perhaps be graded as "normal." In addition, in some surgery such as obliteration, the sinus should be opacified with fatty/fibrous material and aeration would actually represent a potential treatment failure. So, the assessment of some objective outcomes will require interpretation.

In summary, outcomes assessed in rhinosinusitis can be divided into two general categories: subjective outcomes and objective outcomes. Both have been reported frequently in the literature, and clinicians also typically use both types of outcome in their everyday evaluations.

#### Subjective Outcomes

Symptoms are a key issue in rhinosinusitis, and are often the primary reason that patients seek initial medical attention and return for further treatment. There is currently no standardized, validated tool to measure symptom burden in rhinosinusitis, although some tools have been reported [9].

An additional assessment of subjective outcome is QOL, which is measured using validated instruments. QOL instruments are generally divided into two types, global and disease-specific, and both have advantages and disadvantages. Global instruments have the advantage of being comparable between diseases and can be used for "benchmarking" against known problems, but have the disadvantage of being less sensitive to the effects of a particular disease. Disease-specific instruments are designed to address the condition of interest, and are much more sensitive to changes in status, however they have the disadvantage of not being comparable across different diseases and therefore they can be difficult to interpret. For example, what does an increase of 21 points on scale X actually *mean* to a patient or interpreting physician?

In the assessment of symptoms or QOL, it is also important to keep in mind that patients without disease will usually not score 0 (or 100) on scales. As an example, in one study of the Sino-Nasal Outcome Tool – 16 items, patients with rhinosinusitis scored an average of 22.4 (on a 0–48 point scale), and patients with ear disease scored a mean of 10.5 [3]. So, the baseline or "normal" score should be considered when reviewing results in any population.

The popularity and use of QOL tools continues to grow, and in general the systematic assessment of QOL yields important information about what patients are feeling, and the actual treatment effects. However, the use of QOL instruments is often not fully understood.

• Most QOL instruments are validated to measure QOL in *populations*, not individual patients; the statistical criteria required to be able to discriminate between individual patients is more stringent.

In addition, many instruments are designed to assess QOL averaged over a recent period of time, not day-to-day changes. For example, items on the SF-36 global instrument ask about the previous 4 weeks, and items on the Chronic Sinusitis Survey ask about the previous 8 weeks. Therefore, QOL instruments might not be the best tools for assessment of short-term outcome changes.

Another option is to use presence or severity of specific symptoms. However, there needs to be some agreement on exactly which symptoms are important to measure. A simple listing of potential symptoms will not suffice because it will give equal "weight" to each symptom. For example, if there are ten possible symptoms listed, then each symptom counts for 1/10th (10 %) of the total "symptom score."

• Rhinologists would probably agree that purulent rhinorrhea is a more important and predictive symptom of sinusitis than headache.

If each were on the same list, however, then changes in each would be counted similarly. Some work is still needed to define and validate a symptom tool for use as an outcome measure in rhinosinusitis [17].

Despite the obvious importance of symptoms and subjective outcomes, there are problems with using them in isolation – a large problem being that some symptoms and QOL changes will be due to other diseases besides rhinosinusitis. Therefore, subjective outcomes are only part of the overall picture.

## **Objective Outcomes**

There are several objective outcomes which can be assessed after the treatment of frontal sinusitis. CT scan findings are very important as an assessment of mucosal thickening, ostia obstruction, fluid level, and aeration of the frontal sinus, as well as demonstrating the integrity of bony walls and extrasinus extension of disease, for example. There are several staging systems that have been proposed for sinus CT scans; among the most popular are the Lund-MacKay, Kennedy, and Harvard systems [19]. These staging systems were designed to be classificatory systems, and were not designed to be prognostic systems (i.e., predict outcome). However, it is possible that a classificatory system could still predict an outcome. Potential problems with any CT staging system include the effect of previous surgical sinus dissection, and difficulty in differentiating mucosal thickening from retained secretions [19]. Despite these potential issues however, the CT scan is widely available, and a very important tool for assessing the sinus mucosa and anatomy. In addition to CT findings, another important objective outcome is the endoscopic examination findings. There is of course some subjectivity since a clinician must review the examination, however there is a standardized staging system for endoscopic findings [20], and the inter-rater reliability seems to be fairly high, so this can be considered an objective measure of outcome.

• Since endoscopic examination and the CT scan are both assessing anatomy and mucosal status, not surprisingly there is a high correlation between endoscopic stage and CT stage [25].

This is likely particularly true after some types of frontal sinus surgery where the sinus has been opened, making endoscopic evaluation even easier.

Bacteriology, and in particular the presence of resistant bacteria, or fungus, can be an important outcome in frontal sinus surgery. However, there is always the possibility of sampling error and differences in lab techniques when reviewing culture results.

Another potential "objective" outcome is the need for oral or topical medications, with the concept being that successful surgery might reduce the need for extensive medical treatment. However, that outcome may be problematic because the use of medications to control persistent disease or prevent recurrent disease may be desirable, and medications are often taken for related problems such as allergies. In addition, patients may use or not take their medications for different reasons, making medication use not the ideal "objective" outcome.

#### Association Between Objective and Subjective Outcomes

It is well-established that symptoms and QOL do not correlate with CT scan findings in chronic rhinosinusitis [8, 10, 14, 16, 33, 34] – which does not mean that CT scan findings are not important, or that symptoms are not important. However it does mean that they are not measuring the same thing, and that you cannot predict one by knowing the other. Therefore, in evaluation of outcomes in the frontal sinus in particular, it is important to consider assessment of both objective and subjective outcome measures.

# Available Validated Health Status Instruments for Use in Chronic Rhinosinusitis

Validated Quality of Life (QOL) instrum	nents for chronic rhinosinusitis
Global	Disease-specific
Short Form 36- item health survey (SF-36)	Chronic Sinusitis Survey (CSS)
Short Form 12-item health survey (SF-12)	Rhinosinusitis Outcome Measure (RSOM-31)

Validated Quality of Life	(QOL) instruments for chronic rhinosinusitis
Global	Disease-specific
	Sinonasal Outcome Test (SNOT-20), (SNOT-16)
	Rhinosinusitis Disability Index (RSDI)
	Rhinosinusitis Quality of Life Survey (RhinoQOL)

#### Global QOL Instruments

There are hundreds of validated global QOL instruments, any of which could be potentially used as an outcome assessment in revision surgery [35]. The *Short Form 36-item Health Survey* (SF-36) has been used in studies of chronic rhinosinusitis and the effect of sinus surgery, and it is clearly sensitive to the impact of chronic rhinosinusitis; the SF-36 is scored into eight subscales. A shorter version, the SF-12, is also a global instrument, and is scored into only two subscales – physical health component and mental health component. Desirable characteristics of the SF-36 and SF-12 are that they have been used extensively, and there are good benchmark comparison data for healthy people and also several different diseases. There are, of course, many other validated global QOL instruments that could be used.

#### **Disease-Specific Instruments**

There are several validated disease-specific instruments for rhinosinusitis in adults, and all have been used by different investigators. Content, length, period of symptom recall, and scoring are different for each, so there are several potential options [21].

The *Chronic Sinusitis Survey* (CSS) (Fig. 30.1) [12] was designed for chronic rhinosinusitis; it contains six items, and was validated for a symptom recall period of 8 weeks. There are two subscales: medication and symptom. The CSS is very sensitive to change over time, although its limited content might exclude some aspects of sinusitis in some patients.

The *Rhinosinusitis Outcome Measure* (RSOM-31) (Fig. 30.2) [26] was initially developed as a 31- question comprehensive assessment of sinusitis-specific symptoms along with some aspects of general health. Since its initial development, it has been simplified and re-validated to be shorter and sinusitis-specific. The current widely used version is the *Sinonasal Outcome Test-20 items* (Fig. 30.3) (SNOT-20), which contains 20 items, with no designated period of symptom recall. There is also a SNOT-22 version, which includes items on loss of smell and nasal obstruction [15]. The SNOT-20 and -22 are scored as a single scale [27]. There is also a 16-item version, the SNOT-16 [3].

The *Rhinosinusitis Disability Index* (RSDI) (Fig. 30.4) [7] is a validated instrument with items phrased in the first person, and it relates symptoms to limitations

#### CHRONIC SINUSITIS SURVEY

Please answer every question by circling the appropriate number. If you are unsure about how to answer a question, please give the best answer you can.

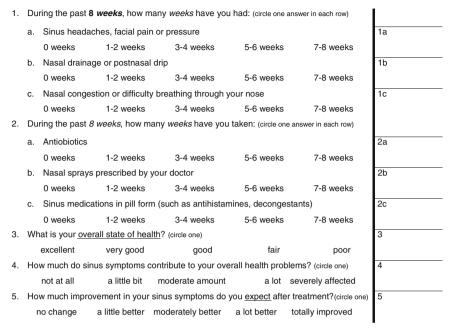


Fig. 30.1 Chronic sinusitis survey (From: Gliklich and Metson [12])

on daily life. It was designed for CRS, contains 30 items, and has no designated period of symptom recall. It is scored into three subscales: emotional, physical and functional. The content of some of the items cover more global QOL issues than other disease-specific instruments.

The *Rhinosinusitis Quality of Life Survey* (RhinoQOL) (Fig. 30.5) [4] is a validated instrument that was designed to be used for both acute and chronic rhinosinusitis. It contains 17 items and uses a recall period of 7 days. It is scored into three subscales: symptom frequency, symptom bother, and symptom effect. The authors report that the instrument can be used in acute or chronic sinusitis.

# The Surgical Approach and Disease Process Impact Frontal Sinus Outcomes

In discussing outcomes after frontal sinus surgery, it is important to understand what underlying disease pathology was present and what type of surgery was performed on the frontal sinus. Primary versus revision, trauma versus non-traumatic, tumor versus inflammatory; all have various surgical interventions and the degree of success may

RHINOSINUSITIS OUTCOME MEASURE (R	SOM	I-31)	)			
To let us know more about your nose and sinus disease, please answer the following questions about the consequences of your nose and sinus disease. There are no "right" or "wrong" answers and only <b>you</b> can provide this information. For each of the statements listed please indicate <b>how much of a problem</b> it is to you. If a statement does not apply to you please circle 0 and go to the next statement. <b>Thank you for your time and participation.</b>	Not present/No problem	Very mild problem	Mild or slight problem	Moderate problem	Severe problem	Problem is as "bad as can be"
1. Stuffy/blocked nose	0	1	2	3	4	5
2. Runny nose	0	1	2	3	4	5
3. Sneezing	0	1	2	3	4	5
4. Decreased sense of taste or smell	0	1	2	3	4	5
5. Postnasal discharge	0	1	2	3	4	5
6. Thick nasal discharge/debris	0	1	2	3	4	5
7. Itchy or watery eyes	0	1	2	3	4	5
8. Sore or swollen eyes	0	1	2	3	4	5
9. Difficulty getting to sleep	0	1	2	3	4	5
10. Wake up during the night	0	1	2	3	4	5
11. Lack of a good night's sleep	0	1	2	3	4	5
12. Wake up tired	0	1	2	3	4	5
13. Fullness in ears	0	1	2	3	4	5
14. Ringing in ears	0	1	2	3	4	5
15. Dizziness	0	1	2	3	4	5
16. Pain in ears	0	1	2	3	4	5
17. Decreased hearing	0	1	2	3	4	5
18. Fatigue / being worn out	0	1	2	3	4	5
19. Reduced productivity	0	1	2	3	4	5
20. Poor concentration	0	1	2	3	4	5
21. Headache	0	1	2	3	4	5
22. Facial pain / pressure	0	1	2	3	4	5
23. Cough	0	1	2	3	4	5
24. Short of breath	0	1	2	3	4	5
25. Inconvenience of having to carry tissues	0	1	2	3	4	5
26. Need to rub nose / eyes	0	1	2	3	4	5
27. Need to blow nose repeatedly	0	1	2	3	4	5
28. Bad breath	0	1	2	3	4	5
29. Frustrated, impatient or irritable	0	1	2	3	4	5
30. Feeling depressed or sad	0	1	2	3	4	5
31. Embarrassed by your symptoms	0	1	2	3	4	5

Fig. 30.2 Rhinosinusitis outcome measure (RSOM-31) (Piccirillo et al. [26])

be highly related to the underlying pathology. In essence, not all frontal sinus surgery is the same and thus the outcomes must be compared to like procedures.

## Endoscopic Frontal Sinus Recess Dissection (Draf 2a)

Considering endoscopic frontal sinus surgery for chronic inflammatory disease, the most commonly performed surgery is a Draf 2a in which the frontal recess is dissected so that the frontal os is patent. This is typically accomplished by

I.D SINO-NASAL OUTCOME TEST DAT	E:			
Below you will find a list of symptoms and social/emotional consequences of your rhinosinusi know more about these problems and would appreciate your answering the following questio ability. There are no right or wrong answers and only you can provide us with this information problems as they have been over the past two weeks. Thank you for your participation. Do n assistance if necessary.	ns to n. Ple	the be ase r	est of ate yo	your our
Considering how severe the problem is when you experience it and how frequently it happens. Please rate each item below on how "bad" it is by circling the number that corresponds with how you feel using this scale: →	No problem	Mild or slight problem	Moderate problem	Severe problem
1. Need to blow nose	0	1	2	3
2. Sneezing	0	1	2	3
3. Runny nose	0	1	2	3
4. Cough	0	1	2	3
5. Post-nasal discharge	0	1	2	3
6. Thick nasal discharge	0	1	2	3
7. Ear fullness	0	1	2	3
8. Dizziness	0	1	2	3
9. Ear pain	0	1	2	3
10. Facial pain/pressure	0	1	2	3
11. Difficult	0	1	2	3
12. Wake up at night	0	1	2	3
13. Lack of a good night's sleep	0	1	2	3
14. Wake up tired	0	1	2	3
15. Fatigue	0	1	2	3
16. Reduced productivity	0	1	2	3
17. Reduced concentration	0	1	2	3
18. Frustrated/restless/irritable	0	1	2	3
19. Sad	0	1	2	3
20. Embarrassed	0	1	2	3

Fig. 30.3 Sinonasal outcome test – 20 (Jay F. Piccirillo, M.D. 1996)

removing the uncinate process, the ethmoid bulla and the agger nasi cell. If there is a complicated ethmoid anatomy in which ethmoid cells extend into the frontal sinus, they may need to be removed as well. Prior studies have reported a 58–100 % success rate using varying criteria such as objective patency of the frontal ostia, CT scan improvement, or improvement in quality of life questionnaires [13, 22, 28]. A recent retrospective study of 109 patients who underwent frontal recess dissection concluded that 193 of 210 (92 %) of frontal sinuses remained patent following surgery with a mean follow-up of 22.9 months. Frontal sinus symptoms resolved in 78 % of the patients. This study found that failure of the frontal outflow tract to remain patent was correlated with persistence of symptoms, infection, polyp recurrence, and the underlying size of the ostium. However, they found no correlation with co-morbid diseases such as asthma and smoking, nor the presence of eosinophilic mucin [24].

#### RHINOSINUSITIS DISABILITY INDEX (RSDI)

The purpose of this scale is to identify difficulties that you may be experiencing because of your nose or sinus problems. Please answer: <b>Never, Almost Never, Sometimes, Almost Always, Always</b> to each item. Answer each item as it pertains to your nose and sinus problem only.	Never	Almost Never	Sometimes	Almost Always	Always
Emotional					
1. Because of my problem I feel stressed in relationships with friends and family.					
2. Because of my problem I feel confused.					
3. Because of my problem I have difficulty paying attention.					
4. Because of my problem I avoid being around people.					
5. Because of my problem I am frequently angry.					
6. Because of my problem I do not like to socialize.					
7. Because of my problem I frequently feel tense.					
8. Because of my problem I frequently feel irritable.					
9. Because of my problem I am depressed.					
10. My problem places stress on my relationships with members of my family or friends.					
Functional					
11. Because of my problem I feel handicapped.					
12. Because of my problem I feel restricted in performance of my routine daily activities.					
13. Because of my problem I restrict my recreational activities.					
14. Because of my problem I feel frustrated.					
15. Because of my problem I feel fatigued.					
16. Because of my problem I avoid travelling.					
17. Because of my problem I miss work or social activities.					
18. My outlook on the world is affected by my problem.					
19. Because of my problem I find it difficult to focus my attention away from my problem and on other things					
Physical					
20. The pain or pressure in my face makes it difficult for me to concentrate.					
21. The pain in my eyes makes it difficult for me to read.					
22. I have difficulty stooping over to lift objects because of face pressure.					
23. Because of my problem I have difficulty with strenuous yard work and housework.					
24. Straining increases or worsens my problem.					
25. I am inconvenienced by my chronic runny nose.					
26. Food does not taste good because of my change in smell.					
27. My frequent sniffing is irritating to my friends and family.					
28. Because of my problem I don't sleep well.					
29. I have difficulty with exertion due to my nasal obstruction.					
30. My sexual activity is affected by my problem.					

Fig. 30.4 Rhinosinusitis Disability Index (RSDI) (Benninger and Senior [7])

## Stereotactic Image Guidance

Stereotactic image guidance is a commonly used modality for primary and revision surgery of the frontal sinus. In a retrospective review of patients who underwent revision ESS of the frontal sinus with the aid of image guidance, 86.6 % of the patients had a significant improvement in both patency of the frontal sinus and subjective improvement [11].

#### RHINOSINUSITIS QOL INSTRUMENT

YOUR RECENT SINUS / NASAL SYMPTOMS	None	A little	Some of the time	Mos	All
Answer questions by checking the box to the right of your answer.	e of t	e of the		t of ti	of the
➔ You are sometimes told to skip over some questions. When this happens, you will see a note that tells you what question to answer next, like this: If None of the time, go to #3.	None of the time	the time	the time	Most of the time	All of the time
<ol> <li>In the last 7 days, how muchof the time did you have sinus headaches, facial pain or facial pressure? If None of the time, go to #2.</li> </ol>					
1a. On a scale of 0 to 10, where 0 is not bothered at all and 10 is bothered a lot, how much were you bothered by the sinus headaches, facial pain or facial pressure?					
0 0 1 0 2 0 3 4 5 6 7 8 9 10					
<ol> <li>In the last 7 days, how much of the time did you have a blocked or stuffy nose? If None of the time, go to #3.</li> </ol>					
2a. On a scale of 0 to 10, where 0 is not bothered at all and 10 is bothered a lot how much were you bothered by having a blocked or stuffy nose?					
0 0 1 0 2 0 3 4 5 6 7 8 9 10					
<ol> <li>In the last 7 days, how much of the time did you have post-nasal drip? If None of the time, go to #4.</li> </ol>					
3a. On a scale of 0 to 10, where 0 is not bothered at all and 10 is bothered a lot how much were you bothered by this post nasal drip?           0         1         2         3         4         5         6         7         8         9         10					
4. In the last 7 days, how much of the time did you have a thick nasal discharge?					
5. In the last 7 days, how much of the time did you have a runny nose?					
For these next questions, please think about all the nasal symptoms you've recently experienced.					
6. In the last 7 days, how much of the time did you have trouble sleeping because of your nasal symptoms?					
7. In the last 7 days, how much of the time did you feel it was harder to concentrate because of your nasal symptoms?					
8. In the last 7 days, how much of the time did you feel it was harder to do the things you normally do because of your nasal symptoms?					
9. In the last 7 days, how much of the time did you feel embarrassed because of your nasal symptoms?					
10. In the last 7 days, how much of the time did you feel frustrated because of your nasal symptoms?					
11. In the last 7 days, how much of the time did you feel irritable because of your nasal symptoms?					
12. In the last 7 days, how much of the time did you feel sad ordepressed because of your nasal symptoms?					
13. In the last 7 days, how much of the time did you think about your nasal symptoms?					

Fig. 30.5 Rhinosinusitis Quality of Life (QOL) instrument (Atlas et al. [4])

## Middle Turbinate Resection

A central tenant offered by Messerklinger concerning ESS is the preservation of the middle turbinate. Of significant concern is that, if the middle turbinate is resected, the stump will lateralize and result in stenosis of the frontal recess. A prospective study was conducted in which 31 patients undergoing ESS had the right turbinate

resected and the left turbinate preserved. The patients were followed up to 2 years. The authors reported that middle turbinate adhesions were present in four patients on the right (the resected side) and three on the left. One patient had bilateral adhesions. Four patients experienced "bilateral sinusitis" in follow up and one patient had "frontal sinusitis"; all improved on antibiotics [31]. Sacrificing the middle turbinate is highly debated, and this appears to be the sole prospective study concerning this topic. Though significance is not sited, the importance of this report is the challenge to the notion that if the middle turbinate is removed, a high rate of frontal sinusitis will ensue. However, caution is advised when analyzing these data due to the small group size, short duration of follow up, and lack of statistical significance.

## Frontal Sinus Rescue Procedure

Frontal sinus stenosis caused by resection of the middle turbinate may necessitate the need for a frontal sinus rescue procedure. The hallmark of a frontal sinus rescue is to reestablish patency of the frontal sinus and more importantly, maintain patency in the post-operative state. Kuhn describes the technique by resecting the bony remnant of the middle turbinate while preserving the medial and lateral mucoperiostium. The medial mucosa is then resected and the lateral mucosa is folded along the skull base in a medial position to aid in maintaining patency of the frontal sinus. In 24 patients who underwent this procedure (32 sides, 9.6 month follow-up) 91 % of the operated sides returned to normal function. Seven sides required an additional procedure; four sides needed two more procedures, and there was one iatrogenic CSF leak [18].

#### Endoscopic Modified Lothrop Procedure

The endoscopic modified Lothrop (EML) procedure is an extended approach to maintain patency of the frontal sinus. The technique commences with the identification of both frontal sinuses. Then a superior septectomy is performed in which the posterior limit is the anterior border of the middle turbinate. This is followed by the removal of the soft tissue and bone between the frontal ostia. This can be safely accomplished by moving from an anterior position in the lateral direction. Care is taken to properly identify the skull base. When the ostia are joined, the frontal sinus floor has been removed, and then the intersinus septum is removed with a drill. This procedure is also termed a Draf 3. A common complication of the procedure is to violate the anterior skull base causing a cerebrospinal fluid leak. These leaks are managed intra-operatively at the time of surgery.

The endoscopic modified Lothrop procedure is used as an alternative to frontal sinus obliteration. Retrospective reviews cite a success rate between 77 and 93 %. Schlosser et al. followed 54 patients with a mean follow-up of 40 months. They

reported that after one procedure, 68 % maintained patency for greater than 1 year. Six patients underwent revision EML that resulted in successful patency. Eighteen percent went on to an osteoplastic flap procedure, including three failed revision surgeries. The complication rate was 11 % [29]. Smith et al. followed 13 patients for a period of 34.5 months, and cite 77 % patency after initial EML; two patients went on to osteoplastic flap [30]. Another study [36] followed 83 patients for an average follow-up of 21.9 months and had a 93 % success rate, and no patients in that cohort went on to osteoplastic flap. Finally, in 2007, a group of 97 patients undergoing EML showed a 98 % improvement in symptom scores, and 22 patients required a revision procedure and three an osteoplastic flap [32]. There was one CSF leak. In a recent review of 339 patients who underwent either primary or revision frontal sinus surgery, 47 patients went on to EML. Comorbidities such as polyps and asthma were associated with failure of standard frontal sinus surgery, as was a Lund-Mackay score >16 and a frontal ostium <4 mm [24].

#### **Osteoplastic Frontal Sinus Obliteration**

The osteoplastic frontal sinus obliteration is reserved for patients with chronic frontal sinusitis that has been unresponsive to other treatment modalities, whether surgical or medical. Performing this procedure involves a coronal incision, opening the anterior table of the frontal sinus and obliterating the sinus by first removing the sinus mucosa and then filling the sinus with fat in an effort to make the sinus nonfunctional. In a retrospective study of 39 patients who underwent an osteoplastic frontal sinus obliteration, the majority had the procedure for chronic frontal sinusitis, followed by mucocele, trauma, and osteoma. The most common presenting symptom was headache. Using the Chronic Sinusitis Survey (CSS), 69.2 % of the patients were deemed "satisfied" with the results of surgery, but only 48.7 % felt that the surgical management was the most effective treatment. Even fewer patients (43.5 %) felt that the frontal sinus obliteration was the most beneficial surgery they had gone though. Pain, congestion, and nasal discharge all improved following surgery; and there was a decrease in clinic visits and the use of antibiotics in the postoperative patients. The most common complications were intraoperative CSF leak (12.8 %) and post-operative abdominal or scalp fluid collection (12.8 %) [1]. However, a smaller series of 17 patients had no intraoperative or perioperative complications reported [2].

#### Various Disease Processes

Successful maintenance of patency of the frontal sinus is highly attributable to the underlying disease process. Patients with chronic rhinosinusitis with nasal polyps have a higher rate of post-surgical failure. In comparing 199 patients who

underwent standard ESS with a Draf 2a versus 139 patients with a Draf 3, polyps were found to occur at rate of 22.7 % in the whole group by 12 months. The frontal recess was the most common site of initial recurrence (55 %). The revision rate was significantly lower in the Draf 3 group, 7 %, versus 37 % in the Draf 2a cohort. The authors state a survival analysis found a significant reduction in the risk of revision in the Draf 3 group [5].

Other indications for more aggressive surgical management of the frontal sinus with drill-out procedures are mucoceles, benign and malignant tumors, and inverted papilloma. Even more, drill-out procedures may be necessary in the repair of CSF rhinorrhea. In analyzing a cohort of 186 patients, 30 required some form of drill-out procedure including a Draf 2B, Draf 3, or transseptal frontal sinusotomy. In these groups, the disease pathology resolved post surgically in 32 %; 56 % had improvement, and 12 % were unchanged [6].

#### Summary of Assessing Outcomes in the Frontal Sinus

There are particular challenges in assessing outcomes after treatment of the frontal sinus. While patient-based, subjective outcomes are important, there are no validated instruments which address only the frontal sinus. In addition, symptoms in this area can have a great deal of overlap with headache and neurologic syndromes, so symptom evaluation alone – without radiologic or endoscopic confirmation – can be a problem. The assessment of frontal sinus ventilation, ostia patency, or mucosal disease are therefore also important. Some common-sense combination of endoscopic, radiologic, and patient-based outcomes should be used to assess outcomes after frontal sinus surgery.

#### References

- Alsarraf R, Kriet JD, Weymuller EA. Quality of life outcomes after osteoplastic frontal sinus obliteration. Otolaryngol Head Neck Surg. 1999;121(4):435–40.
- Al-Qudah M, Graham SM. Modified osteoplastic flap approach for frontal sinus disease. Ann Otol Rhinol Laryngol. 2012;121(3):192–6.
- Anderson ER, Murphy MP, Weymuller Jr EA. Clinimetric evaluation of the Sinonasal Outcome Test-16. Student Research Award 1998. Otolaryngol Head Neck Surg. 1999;121(6):702–7.
- Atlas SJ, Metson RB, Singer DE, et al. Validity of a new health-related quality of life instrument for patients with chronic sinusitis. Laryngoscope. 2005;115:846–54.
- 5. Bassiouni A, Wormald PJ. Role of frontal sinus surgery in nasal polyp recurrence. Laryngoscope. 2013;123(1):36–41.
- Batra PS, Cannady SB, Lanza DC. Surgical outcomes of drillout procedures for complex frontal sinus pathology. Laryngoscope. 2007;117(5):927–31.
- Benninger MS, Senior BA. The development of the rhinosinusitis disability index. Arch Otolaryngol Head Neck Surg. 1997;123:1175–9.

- Bhattacharyya T, Piccirillo FJ, Wippold 2nd FJ. Relationship between patient-based descriptions of sinusitis and paranasal sinus computed tomographic findings. Arch Otolaryngol Head Neck Surg. 1997;123:1189–92.
- 9. Bhattacharyya N. The economic burden and symptom manifestations of chronic rhinosinusitis. Am J Rhinol. 2003;17(1):27–32.
- 10. Bradley DT, Kountakis SE. Correlation between computed tomography scores and symptomatic improvement after endoscopic sinus surgery. Laryngoscope. 2005;115:466–9.
- Chiu AF, Winston CV. Revision endoscopic frontal sinus surgery with surgical navigation. Otolaryngol Head Neck Surg. 2004;130:312–8.
- 12. Gilkrich RE, Metson R. Techniques for outcomes research in chronic sinusitis. Laryngoscope. 1995;105:387–90.
- Har-El G, Lucente FE. Endoscopic intranasal frontal sinusotomy. Laryngoscope. 1995; 105(4 Pt 1):440–3.
- 14. Hwang PH, Irwin SB, Griest SE, et al. Radiologic correlates of symptom-based diagnostic criteria for chronic rhinosinusitis. Otolaryngol Head Neck Surg. 2003;128:489–96.
- 15. Hopkins C, Gillett S, Slack R, Lund JV, Browne JP. Psychometric validity of the 22-item Sinonasal Outcome Test. Clin Otolaryngol. 2009;34(5):447–54.
- 16. Krouse JH. Computed tomography stage, allergy testing, and quality of life in patients with sinusitis. Otolaryngol Head Neck Surg. 2000;123:389–92.
- Krouse J, Lund V, Fokkens W, Meltzer EO. Diagnostic strategies in nasal congestion. Int J Gen Med. 2010;3:59–67.
- Kuhn FA, Javer AR, Nagpal K, Citardi MJ. The frontal sinus rescue procedure: early experience and three-year follow-up. Am J Rhinol. 2000;14(4):211–6.
- 19. Lund VJ, Mackay IS. Staging in rhinosinusitis. Rhinology. 1993;31(4):183-4.
- 20. Lund VJ, Kennedy DW. Staging for rhinosinusitis. Otolaryngol Head Neck Surg. 1997;117(3 Pt 2):S35–40.
- Meltzer EO, Hamilos DL, Hadley JA, Lanza DC, Marple BF, Nicklas RA, Adinoff AD, Bachert C, Borish L, Chinchilli VM, Danzig MR, Ferguson BJ, Fokkens WJ, Jenkins SG, Lund VJ, Mafee MF, Naclerio RM, Pawankar R, Ponikau JU, Schubert MS, Slavin RG, Stewart MG, Togias A, Wald ER, Winther B. Rhinosinusitis: developing guidance for clinical trials. J Allergy Clin Immunol. 2006;118(5 Suppl):S17–6.
- Metson R, Gliklich RE. Clinical outcome of endoscopic surgery for frontal sinusitis. Arch Otolaryngol Head Neck Surg. 1998;124(10):1090–6.
- Naidoo Y, Wen D, Bassiouni, et al. Long-term results after primary frontal sinus surgery. Int Forum Allergy Rhinol. 2012;2(3):185–90.
- Naidoo Y, Bassiouni A, Keen M, Wormald PJ, Risk factors and outcomes for the primary, revision, and modified Lothrop (Draf III) frontal sinus surgery. Int Forum Allergy Rhinol. Int Forum Allergy Rhinol. 2013;3(5):412–7.
- Nair S. Correlation between symptoms and radiological findings in patients of chronic rhinosinusitis: a modified radiological typing system. Rhinology. 2009;47(2):181–6.
- 26. Piccirillo JF, Edwards D, Haiduk A, et al. Psychometric and clinimetric validity of the 31 item Rhinosinusitis Outcome Measure. Am J Rhinol. 1995;9:297–306.
- Piccirillo JF, Merritt Jr MG, Richards ML. Psychometric and clinimetric validity of the 20-item Sino-Nasal Outcome Test (SNOT-20). Otolaryngol Head Neck Surg. 2002;126(1): 41–7.
- Schaefer SD, Close LG. Endoscopic management of frontal sinus disease. Laryngoscope. 1990;100(2 Pt 1):155–60.
- Schlosser RJ, Zachmann G, Harrison S, Gross CW. The endoscopic modified Lothrop: longterm follow-up on 44 patients. Am J Rhinol. 2002;16(2):103–8.
- Schulze SL, Loehrl TA, Smith TL. Outcomes of the modified endoscopic Lothrop procedure. Am J Rhinol. 2002;16(5):269–73.
- Shih C, Chin G, Rice DH. Middle turbinate resection: impact on outcomes in endoscopic sinus surgery. Ear Nose Throat J. 2003;82(10):796–7.

- 32. Shirazi MA, Silver AL, Stankiewicz JA. Surgical outcomes following the endoscopic modified Lothrop procedure. Laryngoscope. 2007;117(5):765–9.
- 33. Stewart MG, Sicard MW, Piccirillo JF, Diaz-Marchan PJ. Severity staging in chronic sinusitis: are CT scan findings related to patient symptoms? Am J Rhinol. 1999;13(3):161–7.
- Stewart MG, Smith TL. Objective versus subjective outcomes assessment in rhinology. Am J Rhinol. 2005;19(5):529–35.
- 35. Stewart MG, Rickert S. Subjective and objective outcomes after revision sinus surgery. In: Kountakis S, Jacobs J, editors. Revision sinus surgery. New York: Springer Publishers; 2008.
- 36. Wormald PJ. Salvage frontal sinus surgery: the endoscopic modified Lothrop procedure. Laryngoscope. 2003;113(2):276–83.

# Chapter 31 Complications of Frontal Sinus Surgery

#### Scott Graham

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No exact figure exists for the likelihood of major complications after endoscopic sinus surgery. To obtain a figure for the chance of a complication involving a single sinus would be even more difficult. Given the complicated anatomy of the frontal sinus and its proximity to vital structures, it is unlikely that complications from frontal sinus surgery would be less frequent than in sinus surgery in general. No study specifically addressing this has been performed. The increasing use of balloon sinuplasty and specifically its application in the frontal sinuses may serve to decrease the overall rate of complications. Balloon sinuplasty, used in isolation in the frontal sinus has an excellent safety profile [1], a safety profile which is likely superior to dissection based techniques. Clearly only a minority of patients and disease processes are suitable for balloon dilation.

• The most commonly quoted figure for complications of surgery involving the sinuses taken as a group is perhaps half of 1 % [2].

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© Springer-Verlag Berlin Heidelberg 2016

S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_31

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This figure is based on survey information with all of the short comings inherent in such a process. Attempts have been made to utilize other parameters in arriving at a figure. Ramakrishnan et al. [3] used insurance claim code analysis to arrive at a figure of 1 % for major complications of sinus surgery in general. Specific complications rates of 0.17 % for CSF leaks and 0.07 % for orbital injury were quoted. Regardless of the exact figure and regardless of the particular sinus or sinuses being operated on a discussion of complications is integral to the process of informed consent. Informed consent requires a discussion and consideration of the risks inherent in sinus surgery. Particularly in the case of the frontal sinus, the risks of no treatment or the risks of a patient not continuing to follow – up for review after an initial decision for conservative treatment also need to be discussed. The consent process also requires a frank discussion of the possibility of "surgical failure". This topic is dealt with in the particular chapters elsewhere in this book dealing with individual surgical techniques.

The utility of image guidance in reducing complications of sinus surgery is a topic that has received renewed interest. Clearly the 'ideal' randomized study prospectively addressing the question of whether image guidance reduces surgical complications is unlikely to ever be performed. Most publications addressing the issue, compare rates of complications in an image guided series with historic controls [4]. Recently two meta – analysis have considered this question with different results. In a 2013 study Sunkaraneni et al. [5], reviewed both their own experience and a meta - analysis of eight other studies concluding that image guidance use imparted no statistically significant benefit in terms of reduction of major complications. The conclusions of this study were in keeping with those of other publications. Also in 2013, in contrast to this report and in contrast to prevailing opinion, Dalgorf et al. [6], published their meta-analysis of 14 comparative cohorts. They concluded that the use of image guidance for sinus surgery is associated with a lower risk of major and total complications. Based on survey results published by Justice and Orlandi [7], frontal sinus surgery is one of the consistent reasons for use of image guidance in the United States. Expert reviews cite the enormous conceptual advantages of image guidance and commend its use in frontal sinus surgery. While there may be argument about whether image guidance provides a statistically significant reduction in complication rates after sinus surgery, the principal statistically proven legacy of image guidance thus far is an increase in the number of sinuses opened.

• Safe and successful frontal sinus surgery requires careful planning and a deliberate effort to positively influence as many surgical variables as possible.

First and foremost in minimizing surgical variables is the CT scan. The CT scan should be appropriately timed after intensive medical treatment and contain, at a minimum, finely cut coronal images. Limited cut CT scans, while useful in resolving diagnostic issues, play no part in surgical planning. Axial cuts provide useful information in assessing frontal sinus wall integrity, and sagittal reconstructions provide invaluable information for endoscopic approaches to the frontal sinus. As a practical matter these images are most often provide together in the multiple planar

images supplied for surgical navigation technology. Image-guided surgery is an unquestioned advance in analyzing the complex anatomy of the frontal recess. While this is no substitute for surgical judgment and experience, it is of particular benefit in revision cases.

- In order to prevent complications our surgical efforts and planning are aimed at:
  - Reducing intraoperative bleeding with consequent increased visualization and by implication, enhanced surgical safety.
  - Reduce mucosal inflammation by the use of pre-operative antibiotics and in selected cases, corticosteroids.

Patients are instructed to avoid the use of any substances which may effect the bleeding time. Intraoperative use is made of topical vaso-constricting solutions as well as hemostatic injections at selected sites. Head of bed elevation and judicious control of blood pressure and heart rate are employed.

• If there is sufficient bleeding intraoperatively to preclude adequate surgical endoscopic visualization, the surgery should be stopped.

Intraoperative blood loss is, of course, recorded in the anesthetic record and, from a medico-legal point of view, an operative complication in the face of significant bleeding becomes difficult to defend. Similarly, total operative time is also recorded and defense of a claim is difficult where the operation may have the appearance of being "rushed" or performed too quickly. A further factor that is scrutinized should a complication occur is the original indication for surgery and whether an adequate trial of appropriate medical therapy had been initially employed.

Complications related to surgery in general, not specific to frontal sinus operations, may also occur. Such events include anesthetic complications, post-operative wound infections and pneumonias and will not be further discussed. In lengthy operations the use of prophylaxis against deep venous thrombosis and pulmonary embolism should be considered [8]. The remainder of this chapter will be devoted to particular complications which may occur with the variety of surgical approaches and operations that exist for the frontal sinus.

## Transnasal Endoscopic Approaches to the Frontal Sinus

CSF leaks can be broadly divided into those leaks recognized at the time of surgery and those leaks diagnosed during the postoperative period. As a general rule, a CSF leak diagnosed intraoperatively should be repaired in the same surgical setting. Local intranasal tissue [9] is generally used to repair skull base defects, although a variety of other tissue including fat from the ear lobule can be considered. Various complications are possible depending on the techniques used to localize the defect and then to reconstruct it. While the use of intrathecal fluorescein is not approved by the FDA, we have used it as a matter of routine in the intraoperative diagnosis and localization of CSF leaks identified secondarily. Specific consent for its use is obtained and a lumbar puncture, performed in our practice by a neurosurgeon, is performed immediately after induction of general anesthesia [10]. 10 cm<sup>3</sup> of CSF is removed, which is mixed with 0.1 cm<sup>3</sup> of 10 % fluorescein, precisely measured in a tuberculin syringe. This is reinjected slowly in a timed 10-min sequence. The variety of fluorescein suitable for intravenous injection is used. Risks of intrathecal fluorescein injection, such as status epilepticus, have generally been associated with bolus injection and dosing errors.

## Anterior Ethmoidal Artery and Orbital Complications

The proximity of the anterior ethmoidal artery to the posterior aspect of the frontal recess places it in special danger during transnasal approaches to the frontal sinus.

• In the event that the anterior ethmoidal artery is damaged and significant bleeding occurs, this is best dealt with by bipolar cautery.

A combined suction bipolar cautery device provides the best instrumentation in this situation. The use of hemostatic packing can also be considered. In general, monopolar cautery should be avoided on the skull base, particularly in close proximity to the orbit. Extended surgical manipulation adjacent to a bleeding anterior ethmoidal artery has the potential for further complications, as this is one of the areas where the skull base is weakest. Unexplained bleeding at the skull base may be suggestive of a concomitant CSF leak.

Damage resulting in transection of the anterior ethmoid artery, if the artery retracts, may produce the rapid onset of an orbital hematoma. The anterior ethmoidal artery also has an intracranial course and, while rare, intracranial bleeding requiring a craniotomy for control has been described. Orbital hematomas may occur more slowly from venous bleeding after breech of the lamina and periorbita [11]. These hematomas are often diagnosed postoperatively, and initial treatment begins with removal of any ipsilateral nasal packing. The more usual situation is a rapid-onset hematoma from an arterial bleed presenting with intraoperative proptosis. Surgery performed under local anesthesia with sedation affords the luxury of being able to assess the patient's vision. Subsequent treatment of the hematoma can be made based on the patient's visual acuity. If the vision is normal and the circulation to the optic nerve, as assessed by funduscopy, is not compromised, carefully selected patients can be closely monitored. Adjunctive treatment such as mannitol or steroids may decrease the intraorbital pressure. It should be emphasized however, that the single most important factor in managing these patients is close observation of their vision.

The principles of managing orbital hemorrhage and complications are:

- If a postoperative orbital hematoma develops, remove any ipsilateral nasal packing.
- Firm pressure to the orbit for 2 min may help control active intraorbital hemorrhage.

- When there is increased orbital pressure, lateral canthotomy and superior and inferior cantholysis may be beneficial.
- · With persistently increased intra-ocular pressure consider orbital decompression.

As a practical matter, most frontal sinus operations are performed under general anesthesia. Vision cannot be assessed and decisions on hematoma management must be made as if the least favorable outcome is likely.

• In cases of orbital hematoma assistance from an ophthalmologist should be immediately sought.

An ophthalmologist will likely examine the fundus, provide an estimate of proptosis, and measure the intraocular pressure. Intraocular pressure can be measured using a tonometer.

- Increased intraocular pressure:
  - As a generalization, an intraocular pressure under 30 mmHg suggests that the eye can be observed
  - An intraocular pressure of 40 mmHg or more may be associated with a poor vision result.

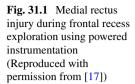
Fundoscopy is used to asses blood flow to the optic nerve, in a 1980 study of central retinal artery occlusion and retinal tolerance time in laboratory monkeys, permanent ischemic damage to vision occurred at approximately 90 min [12].

In reality, immediate ophthalmological assistance may not be available or the ophthalmologist consulted may have limited expertise in orbital surgery. In these common circumstances, the patient relies on the judgment and surgical skills of the otolaryngologist.

- Intraorbital hemorrhage management steps:
  - Ophthalmology consult
  - Remove sinonasal packing if contributing to hematoma
  - Firm four-finger pressure should be applied to the orbit for 2 min
  - Consider diuretics
  - Lateral canthotomy with upper and lower lid cantholysis
  - Consider orbital decompression and exploration

As initial treatment, firm four-finger pressure should be applied to the orbit for 2 min in an effort to control the hemorrhage with digital pressure. The pressure should be stopped if the globe becomes rock hard. This effort to stop the hemorrhage is important; otherwise further surgical steps simply provide a greater volume for the severed and retracted anterior ethmoidal artery to bleed into [13]. A lateral canthotomy with upper and lower lid cantholysis should be performed [14]. This increases orbital volume and lowers the orbital pressure. This lowering of orbital pressure may allow bleeding to restart and the eye requires close observation. If bleeding has stopped, "orbital massage," as described in some publications, to redistribute the intraorbital clot, has a high likelihood of restarting bleeding. After canthotomy and cantholysis, orbital decompression and exploration should be





considered. Orbital decompression with removal of bone and subsequent periorbital incisions can be accomplished via a variety of approaches. These include external ethmoidectomy, endoscopic decompression [15], or via the newer trans-caruncular approach [16]. The choice between these three techniques lies with the particular surgeon's experience and expertise.

Powered instrumentation is associated with special risks within the close confines of the frontal recess [17]. Powered instrumentation is, in fact, "suctionassisted" powered instrumentation, and adjacent tissue is sucked into the rapidly rotating powerful cutting blades. Treatment of the ensuing diplopia from inadvertent medial rectus resection is seldom successful. While restoration of fused vision in primary gaze may represent a surgical triumph in an individual patient, it is seldom of great clinical utility.

• The special impact of powered instrumentation is that an injury that might "only" have resulted in dural exposure or periorbital fat prolapse with conventional forceps dissection can rapidly turn into a catastrophe.

An event that previously would have served simply as a salutary anatomical reminder to the surgeon with little chance permanent significant sequelae is now a major complication. With conventional instrumentation, breech of the lamina and periorbita may produce only fat prolapse and, providing this is recognized and left alone, most of the remaining surgery can still be performed with little by way of lasting sequelae. With powered instrumentation, this tissue, together with whatever lies beneath, most often the medial rectus muscle, is sucked in and severed by the rotating blades in a fraction of a second (Fig. 31.1). Parenchymal brain injuries and intracranial vascular damage may occur in situations which previously would have resulted "only" in CSF leaks. These leaks would have had a high likelihood of

subsequent successful endoscopic repair. Powered instrumentation represents a remarkable advance in surgical technique. Its great power and efficiency of dissection need, however, to be treated with the utmost respect. Its associated suction component, integral to its impressive soft tissue dissection capabilities, has opened up new realms of potentially devastating complications. No information exists as to whether powered instrumentation has increased the incidence of complications of sinus surgery. Unequivocally what it has done is to dramatically escalate the scale of injury.

## Intranasal Modified Lothrop Procedure

Image-guided surgery has done much to increase the comfort of surgeons performing endoscopic-modified Lothrop procedures. Injuries to the orbit and dura may occur in this operation as in any endoscopic procedure. The use of powered dissectors and drills in close proximity to these vital structures calls for particular care and judgment on the part of the surgeon. Long-term patency rates and the potential for restenosis of the common frontal sinus/nasal opening have received a good deal of attention and are most appropriately deal with in the chapter devoted to this operation. These issues need to be carefully reviewed with the patient as part of the informed consent for this procedure.

What has received little attention is the potential for disrupted mucociliary clearance in the scar tissue that exists at the top of the nose and in the septal remnant. Particularly in dry climates, this can produce crusting in the vault of the nose with a sense of nasal fullness. Certain patients find these symptoms nearly as distressing as the symptoms that enticed them to have the intranasal modified Lothrop procedure in the first place.

#### External Fronto-Ethmoidectomy

One of the greatest problems with external fronto-ethmoidectomy occurs as a consequence of resection of the lamina papyracea. With lamina resection, orbital contents can prolapse medially, causing potential obstruction of the frontal recess. The external scar is subject to the vagaries of healing. Interrupting the linear incision with an inverted "v" adjacent to the medial canthus reduces the prominence of the scar by lessening the chance for webbing and by adding a degree of randomization of the surgical wound. Dissection at the junction of the roof and medial wall of the orbit can injure the trochlea of the superior oblique muscle. The trochlea comprises a U-shaped piece of fibrocartilage, closed above by fibrous tissue, which is attached to the fovea or spina trochlearis bone, just behind the orbital rim [18]. Interruption of the trochlea may result in postoperative diplopia.

#### Frontal Sinus Trephine

Misdirected attempts to enter a small frontal sinus may result in an intracranial entry. The procedure is performed through a stab incision, which usually, although not invariable, heals without a noticeable scar. In "above and below" approaches to the frontal sinus, a trephine is combined with an endoscopic approach to the frontal recess. This can be placed in the eyebrow and slid up or in the brow skin crease with good cosmetic effect. A trephine through the anterior wall provides the potential to damage the supra-orbital nerve with associated numbness or paresthesia.

A classically positioned trephine in the floor of the frontal sinus has the potential for trochlea disruption. This potential is minimized by sharply incising and elevating the periosteum, exposing the bone for drill dissection [19].

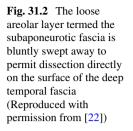
Insertion of a "mini-trephine" with subsequent lavage of the frontal sinus contents has been associated with an intra-orbital collection of lavage fluid [20]. The mini-trephine is often available as part of a pre-made commercially available kit. The presumed pathway was via a likely dehiscence in the frontal sinus floor. The orbital fluid collection was treated in a similar way to an orbital hematoma with an eventual good visual result.

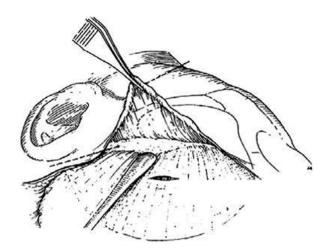
#### **Osteoplastic Frontal Sinus Flap**

Osteoplastic frontal sinus flap surgery can be achieved via a number of approaches. A gull wing or brown incision almost always results in some degree of a noticeable scar. Superior elevation of tissue off of the frontal bone will likely interrupt the supratrochlear and supraorbital nerves with predictable numbness.

Frontal sinus fractures can be sometimes approached through an overlying laceration. Most osteoplastic flap frontal sinus surgeries are, however, approached via a coronal flap. The incision can be sited in a pretrichial location, or most commonly, behind the hairline. Some minor hair loss invariably occurs at the site of the incision. This can be theoretically minimized by beveling of the incision to preserve hair follicles. Of more importance is the potential for visibility of the scar, which may occur with alopecia and advancing male pattern baldness. Numbness occurs over the scalp, posterior to the area of incision.

Elevation of the coronal flap requires a strategy to preserve the frontal branches of the facial nerve [21]. A loose areolar layer known as the subaponeurotic plane lies between the temporoparietal fascia and the deep temporal fascia. This loose tissue can be bluntly swept away to permit dissection directly on the surface of the deep temporal fascia (Fig. 31.2) [22]. This provides protection for the nerve, which travels on the undersurface of the temporoparietal fascia [22].





The potential for inadvertent intracranial entry exists when first entering the frontal sinus and raising the bone flap. This potential is minimized by careful preoperative planning. A surgical template can be fashioned from a nonmagnified plain frontal sinus Caldwell radiograph. Placement of a coin or similar object in the x-rayed field can verify that there is no magnification on the film. Special care must be taken to ensure that the template is able to sit properly at the superior orbital rim. Transillumination may also confirm the boundaries of the frontal sinus. More recently, imaged-guided surgery has provided another means by which to reduce the likelihood of inadvertent intracranial entry, while maximizing frontal sinus exposure.

If frontal sinus obliteration, most often using abdominal fat, is planned, great care must be taken in removing and drilling all mucosa from the sinus. As a practical matter this can be very difficult or indeed impossible with certain anatomic sinus configurations. The danger of postoperative mucocele formation is ever present, and patients must be counseled regarding the need for long-term followup. Great care must also be taken to seal off the frontonasal communication. Contour defects may occur if viability of the bone flap is not completely maintained. Titanium mesh or bone grafts may be used (particularly in revision cases) to restore a more aesthetic contour. Modifications to the classic "osteoplastic frontal sinus flap" procedure where the anterior wall of the sinus is removed and replaced as essentially a free graft, are not associated with increased contour abnormalities [23]. This modification allows a potential pericranial flap to be preserved and raised separately if required. The minor morbidity and potential for complications associated with obtaining an abdominal fat graft for sinus obliteration has led to a search for other more convenient substances. Hydroxyapatite enjoyed some preliminary enthusiasm; however, its use in the frontal sinus has been shown to be associated with the potential for infection and severe problems [24].

## Conclusion

Careful preoperative frontal sinus surgery planning is required to reduce the potential for complications. Improvements in instrumentation have produced unquestioned advances in patient care. The remarkable dissecting capabilities of powered instrumentation, integral to some popular approaches to the frontal sinus, need to be treated with circumspection by experienced and inexperienced surgeons alike.

## References

- Bolger WE, et al. Safety and outcomes of balloon catheter sinusotomy: a multicenter 24 week analysis in 115 patients. Otolaryngology Head Neck Surg. 2007;137:10–20.
- Cumberworth VL, Sudderick RM, Mackay IS. Major complications of functional endoscopic sinus surgery. Clin Otolaryngol. 1994;19:248–53.
- Ramakrishnan VR, Kingdom TT, Nayak JV, et al. Nationwide incidence of major complications in endoscopic sinus surgery. Int Forum Allergy Rhinol. 2012;2:34–9.
- 4. Metson R. Image-guided sinus surgery: lessons learned from the first 1000 cases. Otolaryngol Head Neck Surg. 2003;128:8–13.
- Sunkaraneni VS, Yeh D, Qian H, Javer AR. Computer or not? Use of image guidance during endoscopic sinus surgery for chronic rhinosinusitis at St. Paul's Hospital, Vancouver, and meta-analysis. J Laryngol Otol. 2013;127:368–77.
- Dalgorf DM, Sacks R, Wormald PJ, et al. Image guided surgery influenced perioperspective morbidity from endoscopic sinus Surgery: a systematic renew and meta – analysis. Otolaryngology Head Neck Surg. 2013;149(1):17–29.
- 7. Justice JM, Orlandi RR. An update on attitudes and use of image guided surgery. Int Forum Allergy Rhinol. 2012;2:155–9.
- Moreano EH, Hutchison JL, McCulloch TM, Graham SM, Funk GF, Hoffman HT. Incidence of deep venous thrombosis and pulmonary embolism in otolaryngology head and neck surgery. Otolaryngol Head Neck Surg. 1998;118:777–84.
- Yessenow RS, McCabe BF. The osteo-mucoperiosteal flap in repair of cerebrospinal fluid rhinorrhea: a 20- year experience. Otolaryngol Head Neck Surg. 1989;101:555–8.
- Keerl R, Weber RK, Draf W, Wienke A, Schaefer SD. Use of sodium fluorescein solution for detection of cerebrospinal fluid fistulas: an analysis of 420 administrations and reported complications in Europe and the United States. Laryngoscope. 2004;114:266–72.
- Stankiewicz JA, Chow JM. Two faces of orbital hematoma in intranasal (endoscopic) sinus surgery. Otolaryngol Head Neck Surg. 1999;120:841–7.
- 12. Hayreh SS, Kolder HE, Weingeist TA. Central retinal artery occlusion and retinal tolerance time. Ophthalmology. 1980;87:75–8.
- 13. Graham SM. Complications of sinus surgery. In: Jones N, editor. Practical rhinology. London: Hodder Arnold; 2010.
- 14. Nerad JA. Oculoplastic surgery: the requisites in ophthalmology. St. Louis: Mosby; 2001. p. 38.
- Graham SM, Carter KD. Combined-approach orbital decompression for thyroid-related orbitopathy. Clin Otolaryngol. 1999;24:109–13.
- Graham SM, Thomas RD, Carter KD, Nerad JA. The transcaruncular approach to the medial orbital wall. Laryngoscope. 2002;112:986–9.

- 17. Graham SM, Nerad JA. Orbital complications in endoscopic sinus surgery using powered instrumentation. Laryngoscope. 2003;113:874–8.
- 18. Last RJ. Wolff's anatomy of the eye and orbit. Philadelphia: W.B. Saunders; 1961. p. 236-7.
- Bartley J, Eagleton N, Roser P, et al. Superior oblique palsy after frontal sinus mini-trephine. Am J Otolaryngol. 2012;33:181–3.
- Andrews JN, Lopez MA, Weitzel EK. A case report of intra-operative retro-orbital fluid dissection after frontal mini-trephine placement. Laryngoscope. 2013;123:2969–71.
- Graham SM, Hoffman HT. Chapter XXIII: Extratemporal facial nerve injury: avoidance andpitfalls. In: Shelton C, editor. Update on facial nerve disorders. Rochester: Custom Printing, Inc; 2001. p. 199–208.
- 22. Stuzin JM, Wagstrom L, Kawamoto HK, Wolfe SA. Anatomy of the frontal branch of the facial nerve: the significance of the temporal fat pad. Plast Reconstr Surg. 1989;83:265–71.
- Alqudah M, Graham SM. Modified osteoplastic approach for fontal sinus disease. Ann Otol Rhinol Laryngol. 2012;121(3):192–6.
- Stankiewicz JA, Vaidy AM, Chow JM, Petruzzelli G. Complications of hydroxyapatite use for transnasal closure of cerebrospinal fluid leaks. Am J Rhinol. 2002;16:337–41.

# Chapter 32 Postoperative Care

#### Robert C. Kern and Akaber Halawi

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# Introduction

Post operative care of the frontal sinus is dependent on multiple factors including the procedure performed as well as the indications for that procedure. With advances in surgical navigation and instrumentation, endoscopic approaches have largely displaced open frontal sinus surgery but external procedures such the osteoplastic

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_32

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flap (OPF) are still utilized in occasional cases. Consequently, depending on the approach used, the post operative management and long term monitoring will obviously differ. The indications for frontal sinus surgery may also influence the intensity and duration of post operative care, particularly in the acute setting.

• The presence of severe chronic inflammation or thick bone partitions will likely necessitate more prolonged and aggressive post operative care to facilitate optimal healing of the outflow tract following endonasal surgery.

Elevated inflammatory mediators and exposed bone from extensive drilling result in increased crusting and debris in the post operative setting, and if not addressed, are believed to contribute to early re-stenosis and possibly the need for revision surgery. These factors may also influence outcomes following open frontal sinus surgery, especially when used as an adjunct to an endoscopic technique such as when a trephine is utilized for an "above and below" approach to frontal sinusotomy.

# **External Procedures**

• External procedures on the frontal sinuses with obliteration require considerably less post operative care than the endonasal approach.

In this setting, clinical follow up is focused primarily on cosmetic wound healing and avoidance of hematoma, both of which are dependent on the incision site and management is straight-forward. Persistent post operative pain is more problematic however, and this may be secondary to trauma to the supra-orbital nerve bundle or the use of the diamond burrs. The heat generated from the diamond burr is believed to be helpful in burning any residual mucosa but there is concern that the high thermal energy generated may interfere with post operative bone healing and may be the cause of chronic post operative pain [6]. This can be difficult to distinguish from recurrent frontal sinusitis necessitating radiologic imaging since endoscopic monitoring is of limited value and the patient complaints are often otherwise subtle. Magnetic resonance imaging with fat suppression usually provides adequate details for distinguishing mucoceles, disease recurrence, or intracranial complications.

The principles of postoperative care in external approaches utilized as an adjunct to endoscopic techniques, where the surgical goal is a patent sinus cavity and outflow tract, are similar to purely endoscopic frontal sinusotomy techniques with the additional considerations of proper incisional healing.

# Early Post Operative Care for Draf II and III Procedures

Endonasal procedures on the frontal sinus require extensive post operative management since success is dependent in large measure on patency of the frontal outflow tract, which is an inherently narrow anatomic region, even under the most favorable circumstances. Early post operative care, defined as the first 12 weeks after surgery, is believed to be essential to minimize stenosis. It includes office debridement and medical therapy to suppress inflammation.

# Debridement

There is no consensus on the timing or frequency of post operative debridement following endoscopic sinus surgery in general, or frontal sinus surgery in particular. A recent review of the evidence supported debridement as a mechanism for improved healing and outcomes [17, 18]. This office procedure is performed under topical anesthesia at approximately day 7 post surgery [10].

• While studies have not specifically addressed these issues in regard the frontal sinus, it is our practice to perform debridement at day 7 and then again at day 14 post surgery for both Draf II and III procedures.

Blood clots, mucous plugs, debris, granulation tissue, and un-dissolved packing are removed using frontal sinus instruments and a curved Frasier suction with the aid of a 30, 45 or 70° endoscopes (Fig. 32.1). It is our opinion that this will reduce the inflammatory load on the healing neo-ostium and decrease the risk of scarring. Draf II procedures, with a narrower ostium, likely necessitate more gentle and meticulous debridement. It is also important to evacuate secretions from the frontal sinus cavity, as these can be a nidus for bacterial growth and a resulting inflammatory reaction. The surgeon must ensure proper healing of the middle turbinate in the medial position and lyse any scar bands between the middle turbinate and lateral nasal wall. In the case of a Draf III, the middle turbinate

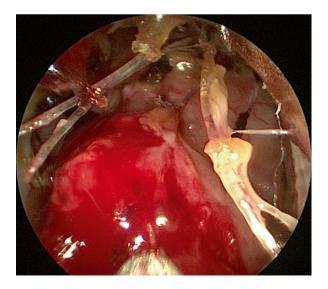


Fig. 32.1 Endoscopic in-office debridement 1 week post EMLP (Image illustrates the steroid eluting stent placed in the surgical bed that is left in place after the first debridement session at 1 week)

has been trimmed so it is not an issue but the extensive drilling and septal resection tend to create granulation tissue that often needs to be addressed. Ultimately, the frequency of post operative debridement is also not standardized, but our primary indication for further procedures is the endoscopic appearance of the outflow tract.

# Nasal Saline Irrigation

Current evidence suggests that nasal saline irrigation (NSI) should be started in the early post operative period for the washout of blood clots, residual secretions and bone fragments [17, 18]. Moreover, keeping the frontal sinus outflow tract moist may promote faster healing, facilitate epithelialization and limit scar formation. The additive effect of NSI with debridement has been demonstrated: a randomized controlled trial comparing NSI with debridement to debridement alone demonstrated the latter to have significant improvement in endoscopic appearance and symptom scores [11]. Interestingly, this effect was only demonstrated in cases of mild CRS, but not moderate or severe CRS. Since Draf II and III procedures are likely to have moderate or severe CRS, it is unclear that there is any added benefit. It is also notable that while NSI may clear debris from the ethmoid, depending on the method of administration, penetration into the sinus cavity itself may be unreliable [20]. The plastic nasal irrigation squeeze bottle is probably to most consistent in this regard.

• Overall, while specific recommendations in regard NSI following frontal sinus surgery do not currently exist [11], it is our practice to utilize saline rinses early, as soon as the patient is able to tolerate the procedure.

## Antibiotics

• Oral antibiotics are considered an option post ESS with relatively little evidence to support improved outcomes or substantial benefit over harm.

Nevertheless, most surgeons utilize antibiotics for 1–2 weeks post surgery to at least theoretically reduce the chance of infection and poor healing. For frontal sinus surgery, it is our practice to place patients on a 2 week course of anti-staphylococcal antibiotics. Infection with this bacterium has been associated with poorer outcomes post Draf III [15]. Alternatively, macrolides are utilized by some surgeons secondary to their added anti-inflammatory effects on sinonasal mucosa, especially in patients with *low* IgE, although improved outcomes with frontal sinus surgery have not been specifically evaluated [21, 22]. Oral doxycycline has also been demonstrated to possess anti- inflammatory properties but this has also not been specifically studied with regard to frontal sinus surgery.

## **Corticosteroids**

Topical steroids in the form of irrigations, sprays and drops as well as oral steroids are used to reduce post-operative inflammation, edema, granulation tissue, fibrin deposition and scar formation. A preponderance of evidence suggests that standard topical intranasal steroids enhance outcomes following ESS, particularly in polyp patients, with no significant side effects even when started in the early post operative period [17, 18]. Non-standard high dose off-label corticosteroid irrigations and sprays have also been utilized; some studies have suggested reduced ostial stenosis and a decreased incidence of revision surgery with corticosteroid irrigations [4]. Budesonide irrigations are particularly popular at present, and short term studies have indicated little systemic absorption [9, 23]. No specific expert recommendations regarding frontal sinus surgery exist but it is our practice to use budesonide irrigations (0.5 mg/2 ml in 240 ml saline BID) after the first debridement for most Draf III procedures. This will typically be continued until endoscopic appearance stabilizes. While evidence supports the use of topical nasal steroids after the surgery, the use of systemic steroids is controversial, primarily due to potential side effects. A level 1 study demonstrated significantly improved post operative endoscopy scores with the use of oral steroids but no difference in patient symptoms [24].

• It is our practice to reserve initiation of postoperative oral corticosteroids for those patients who have significant frontal sinus inflammation by endoscopic evaluation despite the use of budesonide.

Some patients require oral steroids for any number of indications, including asthma control, or reduction of sinonasal inflammation in patients with severe polyposis and/or allergic rhinitis. A detailed discussion of preoperative medical therapy is beyond the scope of the present chapter, but it should be noted that if preoperative oral steroids are utilized, they are tapered gradually in the postoperative period in a manner titrated to the endoscopic resolution of disease. This may take several weeks to even months in patients with severe variants such as aspirin sensitivity triad or allergic fungal CRS. Once the patient is tapered to a low dose (e.g. prednisone 10 mg every other day), he/she is started on budesonide irrigations prior to subsequently stopping oral therapy. Oral steroid use, including dosage schemes and whether they are used at all, must be balanced in the setting of comorbidities such as osteoporosis, cataracts, glaucoma, diabetes, gastritis/stomach ulcer, and reflux.

## Mitomycin C

Mitomycin C has been proposed as an adjunct to help maintain patency of the frontal ostium following Draf II and III procedures. A prospective pilot study reported 86 % patency rate in patients who had three topical mitomycin C applications after revision FESS for a completely stenosed frontal sinus ostium [1]. A prospective, double-blinded, randomized, placebo controlled study applied one intra-operative application of either Mitomycin C (0.5 mg/ml) or placebo for 4 min and found no significant difference in frontal ostium patency [3]. Currently, the utilization of this drug for the post operative management of the frontal sinus is not widespread.

## **Drug Eluting Stents**

The use of rigid frontal recess stents to prevent re-stenosis will be discussed in the section on long term follow up as they are often utilized for an extended time frame. The use of short term, drug-eluting stents has been considered for a number of years, particularly to facilitate healing in the frontal sinus and outflow tract. A pilot study using a doxycycline-eluting frontal sinus stent demonstrated improved healing at 12 weeks post surgery in small prospective trial [8]. Doxycycline has antibacterial and anti-inflammatory properties, both of which may be active in this setting. Clinical device development was not pursued, however. More recently, steroideluting stents which offer the combined ability to help medialize the middle turbinate and release corticosteroids locally into the surgical bed have been introduced (Fig. 32.1) [14]. With regard to frontal sinus surgery, the mechanical stability provided to the middle turbinate is particularly useful in Draf II procedures. Although they have not been specifically studied in terms of frontal sinus patency, they do appear to optimize healing in the early post operative period following sinus surgery. A significant reduction in inflammation, polyp formation, adhesions, frequency of post operative interventions, as well as need for adhesion lysis was demonstrated when compared to placebo stents [5]. In cases of Draf III procedures, these stents can be placed in the cavity at the inflection of the skull base.

• Although drug eluding stents are bio-absorbable, it has been our practice to remove them at the time of the second debridement whether used for a Draf II or Draf III procedure (Fig. 32.1).

Perhaps more significant is the fact that this technology for local drug delivery is theoretically applicable to an array of pharmacologically active agents.

# Long-Term Follow Up for Draf II and III Procedures

## Medical Management

Long term care (>12 weeks post surgery) involves monitoring patient complaints by history as well as nasal endoscopy, which is the key component of the physical exam. Angled nasal endoscopes are often capable of trans-illuminating the frontal sinus demonstrating a patent ostium. The larger cavity in a Draf III makes this

technically easier. The role of post operative CT scanning is typically reserved for evaluation of the symptomatic patient when endoscopic evaluation is unclear or additional surgery is planned for other reasons (Figs. 32.2 and 32.3). Underlying medical co-morbidities such as allergic rhinitis should be treated and standard maintenance therapy for nasal polyposis needs to be continued. Acute infections are treated in a standard fashion. The use of long term intranasal steroids is common after frontal sinus surgery. Long term use of off label corticosteroid irrigations has not been studied, and adverse systemic effects with therapy that is continued indefinitely thus remain a possibility.

• It is has been our practice to utilize budesonide nasal irrigations for up to 3 months at a time, with duration and addition of antibiotics to the irrigations, guided by endoscopic appearance (Figs. 32.4 and 32.5).

The post operative Draf III cavity provides wide access to this form of therapy [15].

• The most problematic long term complication following Draf II and III procedures is stenosis of the frontal outflow tract which may progress to symptomatic obstruction.

In this setting, Draf II procedures may become candidates for Draf III revisions if they fail medical management. Improvements in drill technology and intraoperative imaging over the past decade have greatly facilitated the performance of the Draf III and have lowered the threshold for this procedure. Following Draf III, the neo-ostium will typically narrow to a degree over the first 2 years after surgery [15]. A strict numerical definition of post operative re-stenosis has not been established, but some authors classifying the Draf III drill out site as being either widely patent, stenosed (able to admit a 3-mm curved suction tip), or closed [19] (Fig. 32.6). Others define a stenosed ostium as one that was less than 50–60 % of the original intra-operative opening [2] (Fig. 32.7). From a practical standpoint this is less relevant as the key clinical issue is whether the stenosis, or in some cases complete obstruction, remain symptomatic despite management. Stenosis or even complete obstruction of the outflow tract do not mandate surgery if the patient is asymptomatic.

• Cases of allergic fungal sinusitis and infection with staphylococcus are two conditions that have been associated with symptomatic re-stenosis and a need for revision Draf III [15].

# Stents

Stents have been advocated to prevent stenosis and maintain functional patency of the frontal sinus following open procedures for decades [7]. The application of stents for use following endoscopic frontal sinus surgery is controversial. Currently, there are no absolute indications for post operative stenting of the frontal sinus.

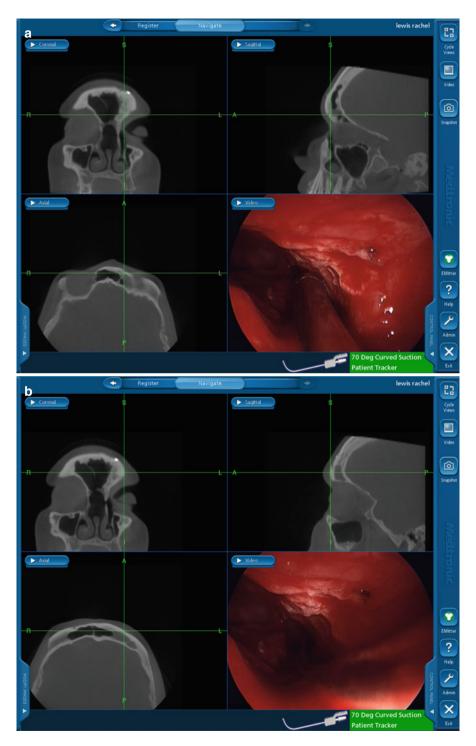


Fig. 32.2 (a, b) Computer tomography scan and endoscopic view using  $70^{\circ}$  scope demonstrating lateral frontal loculation not evident by endoscopic exam despite patent Draft III neo-ostium

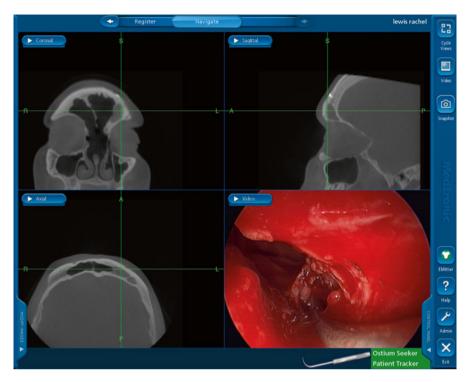
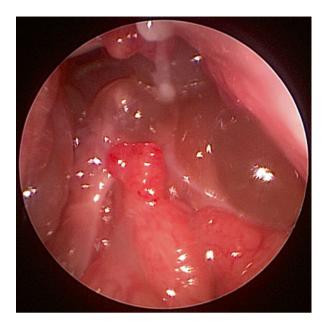


Fig. 32.3 Computer tomography scan and endoscopic view using  $70^{\circ}$  scope demonstrating open sinus cavity after drilling intersinus septum

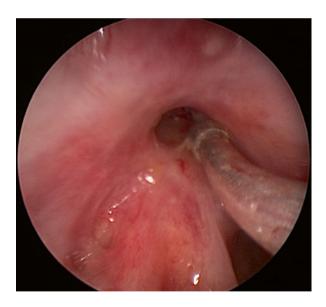


**Fig. 32.4** Endoscopic examination using 45° scope showing discolored drainage in frontal drill out



**Fig. 32.5** Endoscopic examination using 45° scope shows CRSwNP disease recurrence

**Fig. 32.6** Endoscopic examination using 45° scope at 3 months most EMLP shows frontal sinus stenosis



Relative indications for use of stents include:

- Narrow neo-ostium diameter (<5 mm)
- Extensive or circumferential bone exposure or significant mucosal trauma
- Severe polyposis or mucosal hyperplasia
- Destabilized or lateralized middle turbinate

#### 32 Postoperative Care



Fig. 32.7 In-office lysis of midline outflow tract scar band

- Frontoethmoid mucoceles indicative of longstanding chronic sinus disease or prior surgical failures
- · Revision frontal sinus surgery with pre-operative scarring
- Traumatic fracture of the frontal sinus outflow tract [13].

Various non-resorbable materials historically used for frontal sinus stents have included gold, tantalum foil, polyethylene teraphthalate (Dacron®) and polymeric silicone (Silastic) sheeting [7]. Stents also vary in shapes and application techniques. The Freeman frontal sinus stent is a bi-flanged, 20 mm long, hollow silicone tube available in 14 and 16 Fr diameters. The Rains silicone stent on the other hand is made of soft, malleable silicone rubber and has a compressible basket which has a distal end that re-expands to assist maintenance of position in situ. This stent can be used as an irrigation port and is easy to insert under endoscopic guidance. Post-operative care is important in maintaining frontal sinus stent patency, minimize scarring and adhesions, and thus improve

long-term results. Recommendations include daily saline irrigation, long-term topical steroids, and regular outpatient monitoring and debridement.

 There is no consensus of the duration of stent utilization but some surgeons believe that stents should be kept in the frontal recess until adequate reepithelialization and wound healing is achieved.

Experiments on silicone stents in canine models revealed complete reepithelialization of the nasal-frontal communication to be within 8 weeks [16]. Weber et al. kept stents for 6 months post-operatively in Draft type II and found significantly higher patency rate at 12–18 months follow up. Other authors have found the use of stents to be much less helpful [7]. Partial dislodgement of the stent may occur, leading to treatment failure and possible aspiration. Stents may also be obstructed by granulation tissue (across short stents) and crusts (long stents). The presence of stents is also felt by many surgeons to foster biofilm formation and ultimately inhibit patency. In general, it has been our practice to utilize long term frontal sinus stents only rarely, such as cases with unusually narrow frontal sinus outflow tracts or cases of recurrent failures following Draf III procedures.

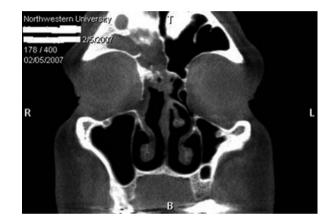
## **Balloon Dilatation**

Stenosis of the post operative frontal outflow tract can be secondary to recurrent polyps, circumferential soft tissue scarring, and/or osteoneogenesis. Symptomatic obstruction for recurrent polyps is often amenable to medical therapy with cortico-steroids and a more open Draf III cavity frequently permits off label steroid irrigations. Osteoneogenesis with symptomatic obstruction often fails to respond to medical therapy requiring surgical revision in the operating room with drilling away of the new bone growth.

• Cases of soft tissue scarring have the potential to be managed with minimally invasive procedures such as balloon dilation.

Theoretically this procedure permits relatively atraumatic dilation of circumferential scarring [12, 25]. Advances in balloon technology have permitted use of these devices in the office setting under local anesthesia. If successful, this obviates the need for formal revision surgery and general anesthesia making the approach much more cost-effective. Under endoscopic visualization, the tip of the guide-wire guides the balloon across the stenotic frontal ostium and the balloon is inflated under a standard protocol. This applies even pressure to dilate the scar tissue. During the process, sinus cavity lavage with saline and/or medications such as antibiotics and corticosteroids can be performed as an adjunct. Close monitoring and occasionally follow up dilations are required as the stenosis may recur. Assuming the device is not damaged, it can be re-utilized in the same patient. Balloon sinuplasty has now become a significant adjunct to post operative management of Draf II and Draf III procedures in our practice, given this potential for office management. Strict indications and long term results are not yet available but the following three cases illustrate some of the principles we currently employ.

**Case 1** This is middle aged male patient who underwent aggressive ESS with complete middle turbinate resection by an outside surgeon. Chronic frontal sinusitis recurred 3 years later (Fig. 32.8) and he was seen in our office. Medical management failed to address his complaints. Endoscopic evaluation revealed a pin point track which was dilated using the balloon. The outflow track remained extremely narrow and clinical response was brief. In retrospect, evaluation of the CT scan reveals significant osteoneogenesis, which could not be addressed using the balloon technique. The intraoperative radiograph illustrates the central narrowing of the balloon at the site of obstructing bone (Fig. 32.9). The patient underwent a Draf III less than 3 months later.

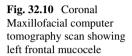


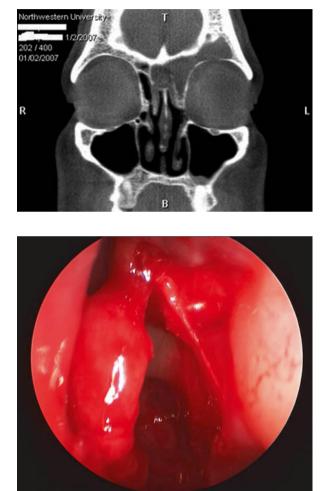


**Fig. 32.8** Coronal maxillofacial computer tomography scan showing right frontal opacification

**Fig. 32.9** Balloon dilatation of frontal sinus tract: fluoroscopic image of the balloon inserted under the wire guidance

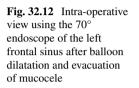
**Case 2** This is middle aged male who underwent ESS 10 years earlier presenting with an isolated left frontal mucocele secondary to a lateralized middle turbinate (Fig. 32.10). The attachments of the middle turbinate were divided and balloon dilation of the outflow tract was performed (Figs. 32.11 and 32.12). The patient has remained asymptomatic for 6 years with a patent frontal sinus on office endoscopy.

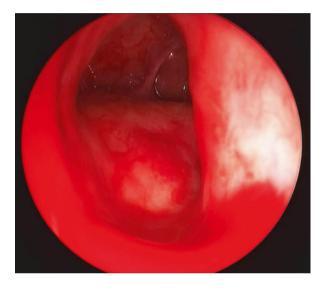




**Fig. 32.11** Intra-operative identification of left frontal mucocele using a 30° endoscope

**Case 3** This is a young male patient who underwent ESS at an outside facility 3 years earlier. He presented to our office with isolated frontal sinusitis that did not respond to aggressive medical therapy (Fig. 32.13). He underwent balloon dilation in the office setting with immediate relief of symptoms. Post procedure CT scan illustrates the enlarged drainage pathway (Fig. 32.14). The endoscopic appearance at 3 months post procedure demonstrates a patent frontal outflow track (Fig. 32.15a, b). The patient has remained asymptomatic for the past 4 years.







**Fig. 32.13** Sagittal maxillofacial computer tomography scan before balloon dilatation



**Fig. 32.14** Computer tomography scan immediately post balloon sinuplasty dilatation

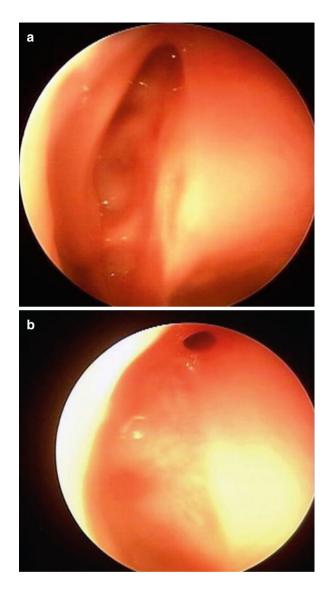
• At present in our practice, patients who experience stenosis and medically recalcitrant frontal sinusitis following Draf II and III procedures are considered potential candidates for office balloon dilation.

Patients must be cooperative and possess a somewhat favorable anatomy. Specifically, the ability to visualize and instrument the frontal outflow tract is essential. Local vasoconstrictive and anesthetic agents are used, similar to the operative room; patients are typically also given an oral anxiolytic 30 min prior to the procedure and post procedure therapy with antibiotics, and possibly a corticosteroid course. Cases with extensive osteoneogenesis are considered poor candidates and are instead offered revision Draf III procedures.

## **Summary**

In general, the management of Draf II and III procedures follows the basic principles used in ESS cases including saline irrigation, debridement and medical management. The use of corticosteroids-topical, oral and off label irrigations-tends to be much higher in the management of post operative frontal sinus surgery however, in an attempt to limit scarring of the narrow outflow tract. The use of steroid-eluting stents has the potential to decrease the need for revision frontal surgery but no data is currently available to support or refute this hypothesis. The application of balloon dilation technology offers the potential to manage frontal stenosis with an office procedure but long term efficacy is also uncertain.

Fig. 32.15 Endoscopic view of the ethmoid cavity (a) and frontal outflow tract (b) at week 12 post balloon dilatation



# Conclusion

Postoperative management and monitoring of the frontal sinus depends on indications for surgery and approach used. Postoperative management for external approach to frontal sinus is essentially external wound care and management of chronic pain secondary drilling sinus mucosa. Monitoring is based on patient symptoms and relies on imaging. Early post postoperative care for endoscopic approach is defined as the first 12 weeks after surgery. Frequent endoscopic monitoring of the neo-ostium is advised.

Treatment is not standardized and includes office debridement, nasal saline irrigation, topical and/or oral steroids and drug eluting stents. Osteoneogenesis, scarring and polyp recurrence are long term complications. Long term monitoring depends on complaints by history as well as nasal endoscopy. Long term management include treatment of co-morbidities and long term intranasal steroids maintenance therapy for nasal polyps as well a further interventions including in office debridement, balloon dilatations and revision surgery.

# References

- 1. Amonoo-Kuofi K, Lund VJ, et al. The role of mitomycin C in surgery of the frontonasal recess: a prospective open pilot study. Am J Rhinol. 2006;20(6):591–4.
- Casiano RR, Livingston JA. Endoscopic Lothrop procedure: the University of Miami experience. Am J Rhinol. 1998;12(5):335–9.
- Chan KO, Gervais M, et al. Effectiveness of intraoperative mitomycin C in maintaining the patency of a frontal sinusotomy: a preliminary report of a double-blind randomized placebocontrolled trial. Am J Rhinol. 2006;20(3):295–9.
- DelGaudio JM, Wise SK. Topical steroid drops for the treatment of sinus ostia stenosis in the postoperative period. Am J Rhinol. 2006;20(6):563–7.
- Han JK, Marple BF, et al. Effect of steroid-releasing sinus implants on postoperative medical and surgical interventions: an efficacy meta-analysis. Int Forum Allergy Rhinol. 2012;2(4): 271–9.
- Hosono N, Miwa T, et al. Potential risk of thermal damage to cervical nerve roots by a highspeed drill. J Bone Joint Surg Br. 2009;91(11):1541–4.
- 7. Hunter B, Silva S, et al. Long-term stenting for chronic frontal sinus disease: case series and literature review. J Laryngol Otol. 2010;124(11):1216–22.
- Huvenne W, Zhang N, et al. Pilot study using doxycycline-releasing stents to ameliorate postoperative healing quality after sinus surgery. Wound Repair Regen. 2008;16(6):757–67.
- 9. Jang DW, Lachanas VA, et al. Budesonide nasal irrigations in the postoperative management of chronic rhinosinusitis. Int Forum Allergy Rhinol. 2013;3(9):708–11.
- Lee JY, Byun JY. Relationship between the frequency of postoperative debridement and patient discomfort, healing period, surgical outcomes, and compliance after endoscopic sinus surgery. Laryngoscope. 2008;118(10):1868–72.
- 11. Liang KL, Su MC, et al. Impact of pulsatile nasal irrigation on the prognosis of functional endoscopic sinus surgery. J Otolaryngol Head Neck Surg. 2008;37(2):148–53.
- 12. Luong A, Batra PS, et al. Balloon catheter dilatation for frontal sinus ostium stenosis in the office setting. Am J Rhinol. 2008;22(6):621–4.
- Man LX, McLean CC, et al. Endoscopic trans(naso)orbital management of supraorbital mucoceles with biliary T-tube stenting. Laryngoscope. 2013;123(2):326–30.
- 14. Murr AH, Smith TL, et al. Safety and efficacy of a novel bioabsorbable, steroid-eluting sinus stent. Int Forum Allergy Rhinol. 2011;1(1):23–32.
- 15. Naidoo YA, Bassiouni et al. Long-term outcomes for the endoscopic modified Lothrop/Draf III procedure: a 10-year review. Laryngoscope. 2014;124(1):43–9.
- Neel HB, Whicker JH, et al. Thin rubber sheeting in frontal sinus surgery: animal and clinical studies. Laryngoscope. 1976;86(4):524–36.
- Rudmik L, Hoy M, et al. Topical therapies in the management of chronic rhinosinusitis: an evidence-based review with recommendations. Int Forum Allergy Rhinol. 2013;3(4):281–98.
- Rudmik L, Soler ZM, et al. Early postoperative care following endoscopic sinus surgery: an evidence-based review with recommendations. Int Forum Allergy Rhinol. 2011;1(6):417–30.

- 32 Postoperative Care
- 19. Schlosser RJ, Zachmann G, et al. The endoscopic modified Lothrop: long-term follow-up on 44 patients. Am J Rhinol. 2002;16(2):103–8.
- Valentine R, Athanasiadis T, et al. A prospective controlled trial of pulsed nasal nebulizer in maximally dissected cadavers. Am J Rhinol. 2008;22(4):390–4.
- 21. Videler WJ, Badia L, et al. Lack of efficacy of long-term, low-dose azithromycin in chronic rhinosinusitis: a randomized controlled trial. Allergy. 2011;66(11):1457–68.
- 22. Wallwork B, Coman W, et al. A double-blind, randomized, placebo-controlled trial of macrolide in the treatment of chronic rhinosinusitis. Laryngoscope. 2006;116(2):189–93.
- Welch KC, Thaler ER, et al. The effects of serum and urinary cortisol levels of topical intranasal irrigations with budesonide added to saline in patients with recurrent polyposis after endoscopic sinus surgery. Am J Rhinol Allergy. 2010;24(1):26–8.
- 24. Wright ED, Agrawal S. Impact of perioperative systemic steroids on surgical outcomes in patients with chronic rhinosinusitis with polyposis: evaluation with the novel Perioperative Sinus Endoscopy (POSE) scoring system. Laryngoscope. 2007;117(11 Pt 2 Suppl 115):1–28.
- 25. Wycherly BJ, Manes RP, et al. Initial clinical experience with balloon dilation in revision frontal sinus surgery. Ann Otol Rhinol Laryngol. 2010;119(7):468–71.

# **Chapter 33 Frontal Sinus Fractures**

Jeremiah A. Alt, Robert T. Adelson, and Timothy L. Smith

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_33

# Abbreviations

- CSF Cerebral spinal fluid
- CT Computed tomography
- FSDP Frontal sinus drainage pathway
- NOE Naso-orbital-ethmoid
- ORIF Open reduction internal fixation

## **Core Messages**

- Frontal sinus fractures can be classified based upon anterior or posterior table involvement, combined fractures, degree of displacement and involvement of the frontal sinus drainage pathway.
- These injuries are usually associated with other significant facial trauma which may require neurosurgical and ophthalmologic consultation.
- High-resolution thin-cut multi-planar CT scanning is essential to accurately characterize these injuries.
- Three main treatment goals include: (1) separation of nasal and intracranial compartments and control of CSF leakage, (2) prevention of late complications, and (3) correction of aesthetic deformity.
- Complications can be life-threatening and involve the orbit and cranium.
- Prevention or reconstruction of aesthetic deformity.

# Introduction

The treatment paradigm for frontal sinus fractures is still under debate. Aesthetic concerns combined with minimization of acute and delayed complications have led to controversies in treatment protocols and decision-making algorithms. Mismanagement of these injuries may lead to complications such as chronic frontal sinusitis, meningitis, brain abscess, mucoceles, mucopyoceles, cerebrospinal fluid leaks, and osteomyelitis which all can present many years after traumatic frontal sinus injury. Unfortunately, most series reported in the literature have relatively small numbers of subjects with limited follow-up periods, further contributing to the continued uncertainty about how these injuries are best managed. The traditional treatment protocols for frontal sinus injury that had been in practice for decades are only now evolving in parallel with the advent of modern endoscopic sinus surgery and advanced imaging techniques. A new treatment paradigm for the management of frontal sinus trauma reflects these advances and is reviewed herein.

## Anatomy

Frontal sinus anatomy is formed by the superior pneumatization of the anterior ethmoid air cells in the fourth fetal month. Significant pneumatization does not begin until age 5 and continues through adolescence. The details of the frontal sinus drainage pathway and mucociliary clearance will not be discussed herein. However, a basic knowledge of the structural boundaries of the frontal sinus and its outflow tract is required for appreciating the advances in management of frontal sinus injury. The frontal bone is composed of horizontal and vertical components, which comprise the orbital roof and forehead respectively. The vertical component is variably pneumatized in the majority of people, dividing the sinus into a thicker anterior table and a thinner posterior table [1]. The posterior table forms the anterior border of the cranial vault and is closely adherent to the underlying dura. The cribriform plate abuts the frontal sinus posteriorly and represents a critical location for postinjury cerebrospinal fluid (CSF) leaks as traumatic energy delivered to the robust frontal bone is distributed through more fragile skull base components. The anterior and posterior walls of the frontal sinus create a funnel directed toward the frontal sinus ostium. The nasofrontal outflow tract or frontal sinus drainage pathway (FSDP) does not form a true duct but rather an hourglass shaped space formed by the boundaries of this drainage pathway. General boundaries include, the agger nasi cell anteriorly, the middle turbinate medially, the skull base posterior superiorly, lamina papyracea laterally and the ethmoid bulla posterior inferiorly.

# Problem

Frontal sinus fractures can be classified into fractures of the anterior table and/or the posterior table with or without displacement or associated FSDP injury. Anterior table fractures alone can cause FSDP obstruction, although there is increased probability of FSDP obstruction when the fracture is associated with naso-orbitalethmoid (NOE) structure or medial supra-orbital rim fractures. The addition of NOE and ethmoid injury makes obstruction of the inferior pathway more likely. Obstruction of the frontal sinus ostia can lead to mucociliary stasis, with subsequent infectious complications and potential for mucocele formation. The consequence of a significantly displaced anterior table fracture usually leads to aesthetic deformity and subsequent reconstruction. Posterior table fractures usually occur in combination with anterior table fractures and are frequently associated with dural, orbital or intracranial injury. The management of CSF leaks and dural tears often dictate acute treatment, although concern for dural exposure predisposing to future infectious complications must also be considered. The same concerns regarding FSDP injury apply to posterior table fractures with the additional increased risk of intracranial spread of infectious complications due to loss of integrity of the anterior cranial vault.

• Unrecognized injury to the frontal sinus ostium and drainage pathway may lead to failure of ventilation with eventual formation of mucoceles, mucopyoceles, meningitis, or intracranial abscess.

# Epidemiology

- Frontal sinus fractures occur in 5–15 % of maxillofacial trauma [2–9].
- In pan facial fracture patients, the frontal sinus is involved in nearly 30 % of the cases [10].

Motor vehicle collisions account for the majority of frontal sinus fractures (42 %), with assaults (14 %), motorcycle collisions (10 %), and ballistic injuries (7 %) being less common [11]. Frontal sinus fractures are often associated with other maxillofacial and intracranial injury as the force of impact required for frontal bone fracture is 800–1,600 lb [12]. Due to the significant transfer of energy frontal sinus fractures are associated with neurologic injury (39–76 %), loss of consciousness (72 %), multiple associated facial and/or skull fractures (87–93 %) or orbital trauma (26–59 %) [13–16].

The predilection for frontal sinus fractures occurring in adults is also due to the timing of pneumatization. The sinuses begin their predominant phase of expansion from age 5 until adolescence, and are usually characterized by two asymmetric sinuses separated by a thin, bony septal plate. They often demonstrate variable pneumatization, with 4–15 % showing developmental failure of one of the frontal sinuses.

## Diagnosis

Although plain films have been used in the past to diagnose frontal sinus fractures, this modality often leads to underdiagnosis of the extent of injury and inadequately assesses the posterior table and outflow tract [5]. The advent of high-resolution thincut multi-planar CT scanning has dramatically improved the assessment of bony facial injury (Table 33.1). Traditional imaging in the axial and coronal planes includes axial and coronal scans, although involvement of the FSDP is not always definitive despite these multiplanar views. The axial plane best visualizes the degree of displacement of the anterior and posterior table while the coronal plane identifies injuries to the anterior ethmoid and medial supra-orbital rim/NOE segments and frontal sinus floor as it relates to the FSDP [17]. Triplanar imaging allows the most helpful radiologic assessment of the FSDP due to the sagittal plane [18–20], which provides useful information for spatial orientation of the FSDP (Fig. 33.1) [21]. Pneumocephalus, air within the intracranial space, is readily identified on computed tomography (CT) scan and can result from injury to the anterior skull base,

Radiographic tri-planar imaging:	
Axial	Degree of displacement of anterior and posterior table
	Pneumocephalus
Coronal	Relationship to anterior and/or posterior ethmoid arteries
	Integrity of the frontal sinus floor
	Damage to medial orbital wall and naso-orbital-ethmoid segments
	Pneumocephalus
Sagittal	Frontal sinus drainage pathway involvement
Three dimensional reconstruction	Visualization of anatomical relationships in extensive pan-facial trauma

 Table 33.1
 Thin cut multi-planar with three dimensional reconstruction imaging has improved diagnostic capability and treatment algorithms in patients with frontal sinus fractures

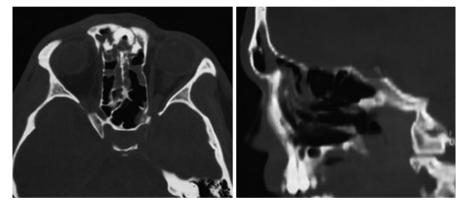


Fig. 33.1 Axial and coronal CT views demonstrating opacification of the frontal sinuses with associated fractures. Although not apparent in the axial view, the sagittal reformatted view demonstrates apparent anatomic patency of the frontal outflow tract despite blood and soft tissue edema

cribriform plate or the orbital roof. Care should be exercised as pneumocephalus is not a sensitive or specific indicator of CSF leaks [4]. In more extensive panfacial fractures, 3-D reconstruction can provide surgeons with a better understanding of anatomic relationships and the optimum sequence for repair [22].

Presence of a CSF leak can be suspected by the presence of a halo sign that develops when the concerning fluid is collected and place on an absorbent tissue because CSF migrates faster than blood. CSF leaks can be confirmed by beta-2 transferrin which is highly specific, although results are typically not available in less than 48 h [4]. Testing for beta trace protein may become more common in the future because this technique has been demonstrated to be more sensitive for the presence of CSF, quicker, less expensive, and requires a smaller volume of fluid for analysis than does testing for beta-2-transferrin [23].

## **Current Management Techniques**

The main goals in the treatment of frontal sinus fractures are generally guided by the following concerns:

- Does the fracture endanger intracranial structures?
- Is there a need to prevent or repair a CSF leak?
- Which fractures, if left untreated will lead to complications, either delayed or immediate?
- Will the fracture lead to aesthetic deformity?
- What appropriate surgical procedures should be undertaken if treatment is deemed necessary?

Although many classification schemes have been proposed, frontal sinus fractures can be simply classified as anterior or posterior table fractures, with or without displacement and/or outflow tract injury (Table 33.2) which dictates treatment decision (Fig. 33.2).

## **Observation with Medical Management**

In appropriate patients medical management patients can avoid surgical procedures with their associated morbidity. Expectant management has been proposed in highly selected cases (Fig. 33.2) where the potential sequalae are amendable to endoscopic techniques with associated satisfactory aesthetic and functional outcomes. Observation is not appropriate for patients with pan-facial fractures or more extensive frontal sinus injury (Tables 33.3 and 33.4).

Good candidates for expectant management include those patients with an isolated outflow tract injury, injuries isolated to the anterior table with or without outflow tract injury, injury limited to aesthetic deformity, and non-displaced uncomplicated posterior table fractures without outflow tract injury (Fig. 33.2). In addition, the safety and success of the expectant strategy relies on the patient's understanding of the risks of future complications and their willingness to undergo periodic follow-up. In these cases, patients are treated with a prolonged course of broad-spectrum antibiotics (4 weeks) and oral steroids, if there are no co-existent medical contraindications. The patients are reassessed with serial CT scans

Table 33.2       Classification of frontal sinus fractures	Anterior table fracture
	± Displacement
	± Outflow tract injury
	Posterior table fracture
	± Displacement
	± Dural injury/CSF leak
	± Outflow tract Injury

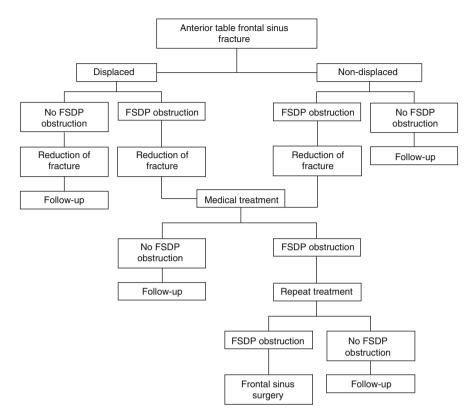


Fig. 33.2 Updated modified treatment algorithm developed from available literature for frontal sinus fractures

Table 33.3       Treatment         options for fractures of the         anterior table	
	Non or minimally displaced
	No treatment necessary
	Displaced
	Immediate: ORIF for cosmesis
	Delayed: Endoscopic brow approach for repair/
	camouflage of the contour depression
	Involvement of the nasofrontal outflow tract
	ORIF anterior table and OPF with obliteration
	Outflow tract reconstruction (not highly recommended)
	Observation and medical management with future endoscopic ventilation if necessary

(1-month, 3-month, 6-month, and yearly thereafter) to check for ventilation and restoration of mucociliary clearance [24].

In patients with non-displaced anterior table fractures there is reduced chance of deformity. Observation is a reasonable consideration with isolated displacement of an anterior table fracture that does not impinge upon the FSDP, as only an aesthetic

i i
Non-displaced without CSF leak
Observation with interval CT scans obtained
Non-displaced with CSF leak
Conservative management of CSF leak with progression to sinus exploration if no resolution in 4–7 days
Displaced (> one table width)
Sinus exploration, repair of dura, obliteration or cranialization depending on involvement of the posterior table
Involvement of the nasofrontal outflow tract
Obliteration or cranialization

Table 33.4 Treatment options for fractures of the posterior table

deformity is expected. Many patients with depressed anterior table fractures will not have perceptible defects 3–6 months post-trauma [25], thereby avoiding morbidity of surgery. Expectant pathway should be considered as most frontal sinus fracture patients will re-establish mucociliary clearance spontaneously as they recover from trauma [24]. In those patients who fail to ventilate the frontal sinus after two courses of antibiotics or suffer an infectious complication would be evaluated for a endoscopic frontal sinus surgery to restore ventilation and communication using the Draf II or Draf III (modified Lothrop procedure) techniques [24, 26]. The Draf II and Draf III procedure are techniques that are widely accepted procedures to ventilate the frontal sinus obliteration [24]. In those patients requiring endoscopic frontal sinus surgery after traumatic injury to the FSDP, image guidance to increase safety of the procedure is suggested. If patients subsequently fail endoscopic frontal sinusotomy, obliteration or cranialization procedures must then be considered.

## **Anterior Table Fractures**

• Depressed anterior table fractures can present with both aesthetic deformity and entrapment of sinus mucosa.

In this situation, the fracture fragments should be reduced and fixed, with careful attention to prevent entrapment of mucosal edges. Exploration can be accomplished via an overlying skin laceration or a coronal flap. Direct glabellar or brow incisions can be considered in male patients with heavy rhytids or loss of scalp hair, though the cosmetic consequences of a direct brow incision may overshadow the aesthetic benefits of anterior table repair.

• Open reduction internal fixation (ORIF) through a coronal flap is the gold standard technique for anterior table fractures with severe comminuted bone fragments, fractured segment displaced greater that one width of the anterior table bone or missing bone. The use of titanium miniplates in frontal sinus fractures is a reliable and durable method of repair with a low complication rate [3]. Small areas of missing bone less than 1 cm may be left untreated, allowing the skin flap to cover the gaps and monitoring for future contour defects which can be treated in a delayed fashion. Alternatively, titanium mesh can be used to span small defects, preserving contour while serving as the method of rigid internal fixation. Larger defects can be reconstructed with split calvarial bone grafts; however, modern internal fixation hardware has obviated the need for bone grafts in the vast majority of cases. If there is extensive contamination of the wound, yet the bone fragments are of moderate size, they may be thoroughly cleansed and soaked in povidone-iodine before replacement [27]. In the setting of extensive fragmentation with gross contamination or infection, it may be best to obliterate or cranialize the sinus and delay reconstruction.

• In some circumstances, noncomminuted isolated anterior table fractures can be addressed endoscopically [28], with excellent outcomes [29].

The fracture segments are exposed using a subperiosteal dissection under endoscopic guidance as for an endoscopic brow lift. Additional small stab incisions can be made in the brow and along forehead mimetic lines to allow for bimanual fracture reduction and miniplate fixation with screws [30]. The endoscopic approach can be converted to the more traditional open approach if fracture reduction or fixation is found to be suboptimal. Persistent aesthetic deformities can be addressed endoscopically for camouflage with alloplastic material with those patients with reliable follow-up. Patients with anterior table fractures in the absence of FSDP injury can often avoid a coronal incision and immediate repair of these fractures, and instead be evaluated 3–6 months with regard to any aesthetic deformity. Cosmetic concerns can be addressed through an endoscopic browplasty approach to the traumatized site with placement of various graft materials to restore normal contour to the anterior table [25].

#### **Posterior Table Fractures**

Many authorities advocate observation alone for uncomplicated, non-displaced posterior table fractures without FSDP involvement, CSF leak or dural exposure [31]. However, others report serious complications resulting from this strategy. One series of five patients reports two infectious complications among non-displaced posterior wall fractures treated non-operatively [32]. The use of regularly scheduled interval CT scans following non-displaced posterior table fracture may decrease the infectious complications reported in the older literature.

- Surgical treatment of posterior table fractures is advised in:
  - Non-displaced posterior table fractures with CSF leakage
  - Involvement of the FSDP



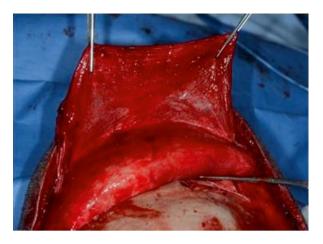
Fig. 33.3 Previously repaired comminuted frontal sinus fracture demonstrating a left lateral frontal sinus mucopyocele with extra sinus extension which required drainage and re-obliteration

 Displaced posterior table fractures (displacement >1 posterior table width) due to increased risk of dural injury.

Consultation with a neurosurgical colleague is required in the aforementioned posterior table fractures. Direct exploration and dural repair or cranialization would both be more commonly recommended than obliteration of the frontal sinus in this scenario. Severe posterior table fractures should prompt neurosurgery evaluation for a frontal sinus cranialization procedure. Posterior table fractures with many unstable fragments are difficult to address properly and pose risks for retained mucosa with delayed mucocele formation as well as intraoperative dural injury during removal.

- · Cranialization is strongly recommended:
  - In fractures of the posterior wall that are severely comminuted
  - CSF leaks that fails to respond to conservative therapy after 5 days or are associated with dural tears or parenchymal injury [14].

Cranialization entails removing the posterior table to create a common intracranial and frontal sinus cavity. By removing the posterior table the brain is allowed to expand forward into the frontal sinus. It is imperative that all mucosa be meticulously removed from the sinus, and the nasofrontal outflow tracts must be obliterated to prevent mucocele formation (Fig. 33.3). A pericranial flap can be used to separate the nasal and frontal cavities in patients with cribriform plate injury and thus augment the skull base and dural repair (Fig. 33.4) [2, 33]. **Fig. 33.4** Photograph of an extended pericranial flap, which can be used for obliteration of the frontal sinus or lining of the anterior cranial fossa in cranialization procedures



# **Nasofrontal Outflow Tract Involvement**

Unrecognized outflow tract injury can lead to sinus drainage failure with subsequent mucocele formation, infectious complications, and symptoms such as headache. Intraoperative assessment of patency by gentle probing of the FSDP or endoscopic examination after administration of fluorescein dye or methylene blue with inspection for intranasal drainage can be unreliable secondary to traumatic mucosal edema and variable responses to topical decongestants. A review of preoperative CT scans with triplanar views has identified several features of frontal sinus fractures that are thought to be associated with compromised frontal sinus function. Those fractures that involve the medial orbital wall and anterior ethmoid system, the frontal sinus floor, and those with bone fragments within the frontal recess and FSDP are at higher risk for compromised frontal sinus function and should be addressed more aggressively [11]. Much controversy has surrounded management of FSDP and treatment consists of endoscopic management of the drainage system, obliteration/ cranialization of the sinus, or observation with medical management.

If the injury is unilateral, some authors have advocated removing the intersinus septum thereby allowing drainage through the contralateral outflow tract [34]. This strategy is not widely supported as it ignores the normal mucociliary clearance pattern and has not been supported by animal models [35]. Another option is prolonged cannulation of the frontal outflow tract [36], but this risks circumferential scarring with outflow tract stenosis, and is considered by many to have an unacceptable failure rate [37]. Alternatively, the outflow tract can be enlarged using modern endoscopic sinus surgery techniques or, less commonly, restored using older procedures that reline the FSDP with mucosa from the middle turbinate such as the (frontal sinus rescue procedure) or from the septum (Sewall-Boyden reconstruction).

• Frontal sinus obliteration should be avoided if possible because of the difficulty in removing all mucosa, which increases risk of failure.

Historically the Reidel procedure eliminated the frontal sinus by removing the anterior sinus wall, plugging the nasofrontal outflow tract, and burring away all mucosa from the posterior wall of the frontal sinus and allowing the skin to collapse against the demucosalized posterior wall. The significant postoperative aesthetic deformity that results from the Reidel procedure lead to the development of the osteoplastic flap technique which exposes the interior of the frontal sinus by creating a flap of the anterior table hinged inferiorly on pericranium. Through this exposure the mucosa of the frontal sinus can be meticulously removed, the nasofrontal outflow tracts plugged, and the sinus obliterated using adipose tissue. The contour of the frontal bone remains normal, but obliteration of the frontal sinus is associated with the development of mucoceles at variably reported rates and times during the post-operative lifetime of the patient [38].

Nasofrontal outflow tract reconstruction attempts have historically been plagued by stenosis and subsequent failure, and these concepts have largely been obviated by modern endoscopic techniques in frontal sinus surgery.

 Endoscopic frontal sinusotomy with Draf II or Draf III procedures have a long history of proven success in treating inflammatory disease of the frontal sinus, and are similarly successful in post-traumatic compromise of the FSDP.

After a trial of medical management in patients meeting criteria for expectant treatment, Draf II/Draf III procedures can appropriately address an obstructed frontal sinus and restore normal function, allowing the patient to be treated in a less invasive fashion than seen in open surgery with coronal approaches and frontal sinus cranialization [24]. The long history of proven success for endoscopic frontal sinusotomy in the management of CRS has, over the last 15 years, has shifted the paradigm for addressing traumatic injuries of the frontal sinus.

#### Complications

The complication rates for the surgical repair of frontal sinus fractures can be divided into late and early complications. Reported complication rates, for the surgical repair of the frontal sinus, range from 1 to 17 % [15, 39, 40].

Early post-operative complications include wound infection, CSF leak, meningitis, forehead pain, damage to the frontal branch of the facial nerve, neuralgia related to surgical trauma of supraorbital and supratrochlear nerves, incisional tenderness, and headaches which can become chronic [41–43]. The complication rate has been reported as 1-3 % for obliterative frontal sinus reconstructions, and as high as 10 % with non-obliterative treatment [13, 44, 45]. CSF leaks postoperatively are present in 3–10 % of patients [39, 41, 46–48]. Early post-operative vigilance is required, as in all endoscopic and cranial base operations, as there have been cases of fatal meningitis in the immediate postoperative period [2].

- Late complications include:
  - Headache
  - Mucocele or mucopyocele

- Scarring/alopecia from coronal incision
- Osteomyelitis
- Hardware infection
- Meningitis.

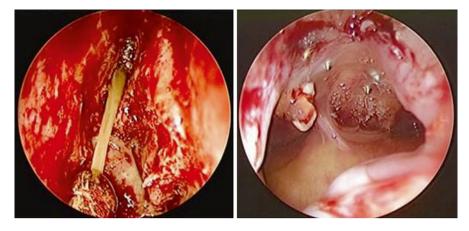
Headaches or chronic pain is one of the most common complication following surgical repair of frontal sinus fractures [40], which presents a diagnostic dilemma due to our limitations in radiographic evaluation of the sinus. This is especially difficult in those patients with frontal obliteration as CT scans lack specificity alone in differentiating a mucocele from a post trauma headache or chronic pain. Mucocele formation has been reported in 6 % of frontal sinus obliteration cases [39]. Mucoceles are encapsulated collections of mucus that cause bony erosion and remodeling as they enlarge (Fig. 33.3). They can erode into the nasal sinuses, orbit, soft tissue or the forehead or the anterior cranial fossa. Mucoceles can occur months to years following initial surgery or time of trauma with no general consensus on the duration, interval, or methodology for monitoring patients after frontal sinus trauma. Current recommendations stem from the fact that a 16 year follow up only captures 50 % of those patients with traumatic frontal sinus mucoceles [38], and that failure to undergo close follow up can result in serious complications [38, 49].

## **Follow-Up Care**

All patients, regardless of type of management, must be followed closely in the first year after injury and annually thereafter. All patients must realize that they have a life-long risk for delayed complications, and must seek immediate medical attention for any complaint of frontal pressure, pain, or headache. They should receive prompt attention with an aggressive workup including appropriate imaging and medical management. These patients require long-term follow-up, as late complications are not uncommon more than a decade after the initial injury.

#### **Case Reports**

**Case 1** A 22-year-old woman presented following a motor vehicle crash in which she sustained an open, displaced anterior table frontal sinus fracture with associated NOE fracture. Fine-cut CT demonstrated these fractures and was highly suspicious for frontal sinus outflow tract fracture. The patient underwent open reduction and internal fixation of the frontal sinus and NOE fractures using a forehead laceration for exposure. There was extensive comminution of the anterior table with small areas of bone loss. The bone fragments were meticulously reduced and fixated with miniplates. Minimal manipulation of the fragments was attempted in an effort to prevent devascularization. She was discharged home with a 4-week course of broad-spectrum antibiotics, nasal spray and close follow-up.



**Fig. 33.5** Intraoperative view of the right frontal sinusotomy (with retained secretions) and the completed modified Lothrop procedure. The tips of the screws used in fixation of the anterior table fracture can be seen penetrating the anterior wall of the frontal sinus, and this is not a finding that should prompt concern or changes in management

At the 4-week follow-up visit, she described pressure over the frontal region. Follow-up CT demonstrated opacification of the frontal sinuses with evidence of frontal outflow obstruction. Endoscopic evaluation revealed no purulence or significant inflammation in the middle meatus. Topical nasal steroid spray, prednisone taper, and empiric antibiotic treatment was initiated for four additional weeks. Follow-up CT demonstrated no improvement.

The patient was prepared for endoscopic frontal sinus surgery. High-resolution thin-cut multi-planar CT scanning was repeated to enable use of computer guidance. A modified endoscopic Lothrop procedure was performed (Fig. 33.5).

Clinical follow-up with endoscopic examination and debridement was performed at day 6, day 13, and then weekly for 6 weeks. Medical therapy was maximized during the initial 6-week postoperative period, which included nasal saline irrigations, topical nasal steroid sprays, perioperative tapering dose of prednisone, and culture-directed antibiotics. Endoscopic examination at 6 months revealed a widely patent nasofrontal communication. At 2 year's follow-up, CT demonstrated excellent ventilation of the sinus with return of mucociliary clearance (Fig. 33.6). At 5 year's follow-up, no clinical evidence of frontal disease is apparent.

**Case 2** A 21-year-old patient presented following a motor vehicle crash in which he sustained an open, displaced anterior table frontal sinus fracture. Fine-cut CT demonstrated these fractures and was highly suspicious for FSDP injury (Fig. 33.7). Displaced fractures of the nasal bones and septum were treated with closed reduction. Open reduction and internal fixation of the frontal sinus fractures was performed. There was extensive comminution of the anterior table. The bone fragments were meticulously reduced and fixated with miniplates. He was

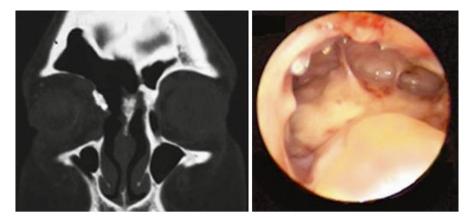


Fig. 33.6 Postoperative views of the frontal sinus, which is widely patent

**Fig. 33.7** Coronal CT demonstrating extensive fractures and soft tissue trauma in the region of the frontal sinus outflow tract



discharged home with a 4-week course of broad-spectrum antibiotics and nasal saline irrigation.

At the 8-week follow-up visit, the patient appeared to be healing without complication and denied frontal pain or pressure. At the 1-year follow-up evaluation, no symptoms related to the frontal sinus were reported, and CT evaluation revealed a well-ventilated frontal sinus and outflow tract. At 4.5 year's follow-up, no clinical evidence of frontal disease is apparent.

# Conclusions

The treatment algorithm for patients with fractures of the frontal sinus continues to evolve as our understanding of frontal sinus function and endoscopic surgical techniques improve. Proper management relies on the type and severity of the injury being treated, the patient population involved, and the experience of the managing trauma team. Frontal sinus trauma patients must receive education about the potential consequences of their injury, the need for continued follow-up, and attention to warning symptoms to prevent life-threatening complications.

## References

- 1. McLaughlin Jr RB. History of surgical approaches to the frontal sinus. Otolaryngol Clin North Am. 2001;34(1):49–58.
- 2. Gerbino G, Roccia F, Benech A, Caldarelli C. Analysis of 158 frontal sinus fractures: current surgical management and complications. J Craniomaxillofac Surg. 2000;28(3):133–9.
- Islamoglu K, Coskunfirat OK, Tetik G, Ozgentas HE. Complications and removal rates of miniplates and screws used for maxillofacial fractures. Ann Plast Surg. 2002;48(3):265–8.
- Manolidis S, Hollier Jr LH. Management of frontal sinus fractures. Plast Reconstr Surg. 2007;120(7 Suppl 2):32S–48.
- May M, Ogura JH, Schramm V. Nasofrontal duct in frontal sinus fractures. Arch Otolaryngol. 1970;92(6):534–8.
- 6. McGraw-Wall B. Frontal sinus fractures. Facial Plast Surg. 1998;14(1):59-66.
- Rodriguez ED, Stanwix MG, Nam AJ, St Hilaire H, Simmons OP, Christy MR, et al. Twentysix-year experience treating frontal sinus fractures: a novel algorithm based on anatomical fracture pattern and failure of conventional techniques. Plast Reconstr Surg. 2008;122(6):1850–66.
- Tiwari P, Higuera S, Thornton J, Hollier LH. The management of frontal sinus fractures. J Oral Maxillofac Surg. 2005;63(9):1354–60.
- Rohrich RJ, Hollier LH. Management of frontal sinus fractures. Changing concepts. Clin Plast Surg. 1992;19(1):219–32.
- 10. Rice DH. Management of frontal sinus fractures. Curr Opin Otolaryngol Head Neck Surg. 2004;12(1):46–8.
- Stanwix MG, Nam AJ, Manson PN, Mirvis S, Rodriguez ED. Critical computed tomographic diagnostic criteria for frontal sinus fractures. J Oral Maxillofac Surg. 2010;68(11):2714–22.
- 12. Nahum AM. The biomechanics of maxillofacial trauma. Clin Plast Surg. 1975;2(1):59-64.
- 13. Levine SB, Rowe LD, Keane WM, Atkins Jr JP. Evaluation and treatment of frontal sinus fractures. Otolaryngol Head Neck Surg. 1986;95(1):19–22.
- 14. Strong EB, Pahlavan N, Saito D. Frontal sinus fractures: a 28-year retrospective review. Otolaryngol Head Neck Surg. 2006;135(5):774–9.
- Chen KT, Chen CT, Mardini S, Tsay PK, Chen YR. Frontal sinus fractures: a treatment algorithm and assessment of outcomes based on 78 clinical cases. Plast Reconstr Surg. 2006; 118(2):457–68.
- Gonty AA, Marciani RD, Adornato DC. Management of frontal sinus fractures: a review of 33 cases. J Oral Maxillofac Surg. 1999;57(4):372–9; 80–1.
- Harris L, Marano GD, McCorkle D. Nasofrontal duct: CT in frontal sinus trauma. Radiology. 1987;165(1):195–8.
- Kim KS, Kim HU, Chung IH, Lee JG, Park IY, Yoon JH. Surgical anatomy of the nasofrontal duct: anatomical and computed tomographic analysis. Laryngoscope. 2001;111(4 Pt 1): 603–8.

- Kanowitz SJ, Shatzkes DR, Pramanik BK, Babb JS, Jacobs JB, Lebowitz RA. Utility of sagittal reformatted computerized tomographic images in the evaluation of the frontal sinus outflow tract. Am J Rhinol. 2005;19(2):159–65.
- Jain SA, Manchio JV, Weinzweig J. Role of the sagittal view of computed tomography in evaluation of the nasofrontal ducts in frontal sinus fractures. J Craniofac Surg. 2010; 21(6):1670–3.
- Hilger AW, Ingels K, Joosten F. Sagittal computerized tomography reconstruction of the lateral nasal wall for functional endoscopic sinus surgery. Clin Otolaryngol Allied Sci. 1999;24(6):527–30.
- Saigal K, Winokur RS, Finden S, Taub D, Pribitkin E. Use of three-dimensional computerized tomography reconstruction in complex facial trauma. Facial Plast Surg. 2005;21(3):214–20.
- Meco C, Oberascher G, Arrer E, Moser G, Albegger K. Beta-trace protein test: new guidelines for the reliable diagnosis of cerebrospinal fluid fistula. Otolaryngol Head Neck Surg. 2003;129(5):508–17.
- Smith TL, Han JK, Loehrl TA, Rhee JS. Endoscopic management of the frontal recess in frontal sinus fractures: a shift in the paradigm? Laryngoscope. 2002;112(5):784–90.
- 25. Strong EB, Kellman RM. Endoscopic repair of anterior table–frontal sinus fractures. Facial Plast Surg Clin North Am. 2006;14(1):25–9.
- 26. Draf W. Endonasal micro-endoscopic frontal sinus surgery: the Fulda concept. Op Technol Otolaryngol Head Neck Surg. 1991;2:234–40.
- 27. Nadell J, Kline DG. Primary reconstruction of depressed frontal skull fractures including those involving the sinus, orbit, and cribriform plate. J Neurosurg. 1974;41(2):200–7.
- Strong EB, Buchalter GM, Moulthrop TH. Endoscopic repair of isolated anterior table frontal sinus fractures. Arch Facial Plast Surg. 2003;5(6):514–21.
- Chen DJ, Chen CT, Chen YR, Feng GM. Endoscopically assisted repair of frontal sinus fracture. J Trauma. 2003;55(2):378–82.
- Onishi K, Osaki M, Maruyama Y. Endoscopic osteosynthesis for frontal bone fracture. Ann Plast Surg. 1998;40(6):650–4.
- Duvall 3rd AJ, Porto DP, Lyons D, Boies Jr LR. Frontal sinus fractures. Analysis of treatment results. Arch Otolaryngol Head Neck Surg. 1987;113(9):933–5.
- 32. Newman MH, Travis LW. Frontal sinus fractures. Laryngoscope. 1973;83(8):1281-92.
- Goodale RL, Montgomery WW. Technical advances in osteoplastic frontal sinusectomy. Arch Otolaryngol. 1964;79:522–9.
- Donald PJ. Frontal sinus and nasofrontoethmoidal complex fractures: a self-instructional package. American association of otolaryngology – head and neck surgery committee on continuing education. 1980.
- Hybels RL, Newman MH. Posterior table fractures of the frontal sinus: I. An experimental study. Laryngoscope. 1977;87(2):171–9.
- 36. Luce EA. Frontal sinus fractures: guidelines to management. Plast Reconstr Surg. 1987; 80(4):500-10.
- Shumrick KA, Smith CP. The use of cancellous bone for frontal sinus obliteration and reconstruction of frontal bony defects. Arch Otolaryngol Head Neck Surg. 1994;120(9):1003–9.
- Koudstaal MJ, van der Wal KG, Bijvoet HW, Vincent AJ, Poublon RM. Post- trauma mucocele formation in the frontal sinus; a rationale of follow-up. Int J Oral Maxillofac Surg. 2004;33(8):751–4.
- 39. Wallis A, Donald PJ. Frontal sinus fractures: a review of 72 cases. Laryngoscope. 1988;98(6 Pt 1):593–8.
- Sivori 2nd LA, de Leeuw R, Morgan I, Cunningham Jr LL. Complications of frontal sinus fractures with emphasis on chronic craniofacial pain and its treatment: a review of 43 cases. J Oral Maxillofac Surg. 2010;68(9):2041–6.
- 41. Correa AJ, Duncavage JA, Fortune DS, Reinisch L. Osteoplastic flap for obliteration of the frontal sinus: five years' experience. Otolaryngol Head Neck Surg. 1999;121(6):731–5.
- 42. Mendians AE, Marks SC. Outcome of frontal sinus obliteration. Laryngoscope. 1999;109(9):1495–8.

- 43. El Khatib K, Danino A, Malka G. The frontal sinus: a culprit or a victim? A review of 40 cases. J Craniomaxillofac Surg. 2004;32(5):314–7.
- 44. Shockley WW, Stucker Jr FJ, Gage-White L, Antony SO. Frontal sinus fractures: some problems and some solutions. Laryngoscope. 1988;98(1):18–22.
- 45. Stanley Jr RB. Management of frontal sinus fractures. Facial Plast Surg. 1988;5(3):231-5.
- Donald PJ. Frontal sinus ablation by cranialization. Report of 21 cases. Arch Otolaryngol. 1982;108(3):142–6.
- 47. Donald PJ, Bernstein L. Compound frontal sinus injuries with intracranial penetration. Laryngoscope. 1978;88(2 Pt 1):225–32.
- Manolidis S. Frontal sinus injuries: associated injuries and surgical management of 93 patients. J Oral Maxillofac Surg. 2004;62(7):882–91.
- 49. Swinson BD, Jerjes W, Thompson G. Current practice in the management of frontal sinus fractures. J Laryngol Otol. 2004;118(12):927–32.

# **Chapter 34 Frontal Sinus Cerebrospinal Fluid Leaks**

Bradford A. Woodworth and Rodney J. Schlosser

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_34

### **Core Messages**

- Identification of a CSF leak etiology; either accidental trauma, surgical trauma, tumors, congenital, or spontaneous; is essential for successful repair.
- Anatomically, frontal sinus CSF leaks are divided into those located adjacent to the frontal recess, within the frontal recess, or within the frontal sinus proper.
- Pre-operative evaluation may include beta-2 transferrin, CT cisternogram, high-resolution CT, MRI, or intrathecal fluorescein and should be individualized for the purposes of diagnosis and localization.
- Frontal sinus CSF leaks involving up to 3 cm of the posterior table can be repaired with proper equipment and expertise.
- Frontal trephine or osteoplastic flap approaches are sometimes required for superior and lateral defects or when sufficient experience in endoscopic techniques are lacking
- Severely comminuted posterior table fractures may require craniotomy and cranialization of the frontal sinus.

## Introduction

Pathology of the frontal sinus represents one of the most challenging and technically demanding areas for the sinus surgeon to reach endoscopically. Over the past two decades, significant advances in endoscopic technique, coupled with the development of instruments designed to specifically address the frontal sinuses, have allowed surgeons to treat an increasing number of complex cases that previously would not have been amenable to endoscopic surgery. Cerebrospinal fluid (CSF) leaks have been repaired with relatively high success rates using accepted endoscopic techniques for nearly 30 years, [1–11] yet the majority of frontal sinus skull base defects are still repaired using traditional open techniques [12]. The use of 70° endoscopes and giraffe instruments allows excellent access to the frontal recess, but post-operative stenosis, anatomic variants, and CSF leaks associated with the posterior table can make repair of these defects very challenging and pushes the limits of endoscopic repairs. Pertinent frontal sinus anatomy, etiologies of CSF leaks, preoperative imaging and considerations, and the technique and type of repair will be discussed.

### **Anatomic Site**

The complex anatomy and variability of the frontal recess is described in great detail elsewhere in this text, but in the most basic sense, the broadest boundaries of the frontal recess are the internal naso-frontal beak anteriorly, the orbit laterally, the attachment of the middle turbinate medially, and the face of the ethmoid bulla (if present) and ethmoid roof posteriorly. This anatomy is highly variable and a number of cells may alter this and encroach upon the frontal outflow tract if present, such as an agger nasi cell antero-laterally or a suprabullar cell posteriorly.

CSF leaks affecting the frontal sinus can be divided anatomically into three general categories [13]:

- · Those adjacent to the frontal recess
- Those with direct involvement of the frontal recess
- Those located within the frontal sinus proper.

While most leaks are limited to one of these distinct sites, some defects encompass multiple anatomic areas.

Skull base defects located in the anterior most portion of the cribriform plate or the ethmoid roof just posterior to the frontal recess do not directly involve the frontal sinus or its outflow tract, but by virtue of their close proximity, the frontal recess must be addressed as described in the Surgical Methods section of this chapter. Endoscopic repairs may cause iatrogenic mucoceles or frontal sinusitis if graft material, packing, or synechiae formation obstructs the frontal sinus outflow tract.

A CSF leak that directly involves the frontal recess is difficult to address surgically, because the superior extent of the defect can be difficult to reach endoscopically and the inferior/posterior extension of the defect may be inadequately visualized from an external approach. Even when long term frontal patency is questionable, expanded techniques (e.g. the Draf III procedure) have allowed access to these regions and also allow for revision "rescue" procedures to be completed if the frontal sinus closes postoperatively [14]. However, individuals with insufficient surgical expertise with such procedures should utilize open approaches such as an osteoplastic flap with thorough removal of all mucosa and obliteration. If the surgeon feels that frontal patency can be maintained, an osteoplastic flap can also be combined with an endoscopic approach to leave the frontal outflow tract patent.

The final anatomic site for frontal sinus CSF leaks is within the frontal sinus proper involving the posterior table above the isthmus of the frontal recess. Significant advancements in equipment and expertise now permit closure of such defects with involvement of up to 3 cm of posterior table in some instances. Defects located superiorly or laterally within the frontal sinus may still require an osteoplastic flap with or without obliteration. Frontal trephination and extended endoscopic techniques are also useful adjuvant techniques. The specific approach depends upon the site and size of the defect, the equipment available, and surgical experience.

### Surgical Goals for Frontal CSF Leaks

#### (Important)

- Goal #1 Successful repair of the skull base defect and cessation of the CSF leak.
- Goal #2 Long term patency of the frontal sinus or a successful obliteration with meticulous removal of all mucosa within the frontal sinus.
- Always be cognizant of both goals when deciding upon a specific surgical approach and repair for each skull base defect.

## Etiology

The underlying cause of a CSF leak will affect the management of the subsequent repair.

CSF leaks are broadly classified into:

- Traumatic (including accidental and iatrogenic trauma)
- Tumor related
- Spontaneous
- Congenital.

These etiologies influence the size and structure of the bony defect, degree and nature of the dural disruption, associated intracranial pressure differential and meningoencephalocele formation. These factors greatly influence medical and surgical treatment and help predict long-term success.

### Trauma

Frontal sinus fractures represent approximately 5–12 % of craniofacial injuries and have a high potential for late mucocele formation, intracranial injury, and aesthetic deformity [15, 16]. Traumatic disruption of the posterior table of the frontal sinus or frontal recess with a dural tear can create an obvious CSF leak or present years later with meningitis, delayed leak, or encephalocele. Projectile injuries from bullets, shot-gun blasts or shrapnel can result in significant comminution of the skull base, and are more likely to involve intracranial injury. CSF leaks usually begin within 48 h and 95 % of them manifest within 3 months of injury [17]. Although over 70 % of traumatic CSF leaks close with observation or conservative treatment, a 29 % incidence of meningitis has been reported in long term follow up when managed non-surgically [18].

Conservative, non-surgical measures are often adequate for injuries limited to the frontal recess and/or posterior table, but severe fractures may require operative intervention due to a high risk of subsequent mucocele formation. Here, the operation addresses both the CSF leak and the potential for future mucocele development, depending upon the anatomic site of the defect. Other considerations include the overall health of the patient, associated intracranial or intraorbital injuries, and other skull base or facial fractures. These additional issues influence surgical treatment and approach.

Functional endoscopic sinus surgery (FESS) and neurologic surgery are the two most common surgeries leading to iatrogenic skull base defects. Significant defects can result from powered instrumentation if they occur during bone resection near the skull base. A CSF leak can occur in the posterior table of the frontal sinus or frontal recess during routine frontal sinusotomy. The posterior table may be less than 1 mm thick and is much thinner than the anterior table. An expansile mucocele or tumor can create dehiscences along the posterior table that are more susceptible to iatrogenic CSF leak during instrumentation. CSF leaks following neurological surgery can occur during frontal craniotomy if the superior or lateral recesses of the frontal sinuses are entered during removal of the bony plate. Individuals with extensive pneumatization are at higher risk. CSF leaks in the lateral recess are difficult to repair endoscopically and may require an osteoplastic flap or trephine approach. Placement of grafts over defects limited to the lateral recess via a frontal trephine may preserve the frontal recess and avoid the need for frontal obliteration.

### **Tumors**

Anterior skull base and sinonasal tumors can create frontal sinus CSF leaks directly through erosion of the posterior table or frontal recess or indirectly secondary to surgical or radiation treatments for the tumor. Persistent tumor following resection and repair will continue to erode the skull base and contribute to frontal sinus CSF leaks. Creating a water tight seal between the sinonasal and intracranial cavities after tumor removal can be difficult. If the tumor is approached intracranially, a pericranial flap is often used to create a barrier. CSF leaks may still occur due to tears in the flap that occur during elevation, devascularization and necrosis, or from inadequate coverage. Posterior table defects and frontal sinus floor defects (after cranialization) may still be present and contribute to CSF leak. Prior chemotherapy or radiation creates significant healing difficulties due to poor vascularity of the wound bed.

## Congenital

Since the frontal sinus is not present at birth, congenital leaks of the frontal sinus proper technically do not exist. However, CSF leaks may develop within or adjacent to the frontal recess, and congenital defects often arise from the foramen cecum [11, 19]. These patients often have a low, funnel-shaped skull base that can make repairs more challenging.

### Spontaneous

Patients with no other recognizable etiology for their CSF leak are deemed spontaneous. Most frequently these leaks occur in obese, middle-age females who demonstrate elevated intracranial pressure (ICP) [20]. The elevated CSF pressures seen in this subset of patients leads to the highest rate (50–100 %) of encephalocele formation, and the highest recurrence rate following surgical repair of the leak (25–87 %), compared to less than 10 % for most other etiologies [21–23]. We recommend adjuvant therapies to treat documented elevation of the ICP as described in the Adjuncts Section of this chapter.

The most common location of spontaneous CSF leaks has varied in the literature with the cribriform plate and the lateral recess of the sphenoid sinus the most frequently quoted areas [8, 24]. Small series and dissimilarities regarding defect site characterization probably account for this variability. However, the first author (BAW) recently published the largest prospectively evaluated series of spontaneous CSF leaks in the literature [25]. This study was the first cohort of spontaneous CSF leak patients to report the frontal sinus posterior table as the most common site of involvement.

# Diagnosis

Establishing the diagnosis and identifying the location of a CSF leak in a patient with intermittent clear nasal drainage and no history of head trauma can be difficult. Pre-operative tests should be based upon the clinical picture and the precise information needed, rather than following a rigid algorithm. In addition, the invasiveness of the test and risks to the patient should be considered. The reported sensitivity and specificity of any test should be interpreted with caution, as these statistics are highly dependent upon the patient population studied, size of the defect, flow rate of the leak, and the individual interpreting the test.

### Techniques for Diagnosing and Localizing CSF Leaks

#### (Attention)

- Beta-2 Transferrin
  - Advantages: Accurate, non-invasive
  - Disadvantages: Non-localizing
- High resolution coronal and axial CT scan
  - Advantages: Excellent bony detail
  - Disadvantages: Inability to distinguish CSF from other soft tissue, bony dehiscences may be present without a leak
- Radioactive cisternograms
  - Advantages: Localizes side of the leak, identifies low volume or intermittent leaks
  - Disadvantages: Localization imprecise, not recommend due to a significant false positive rate and low utility [26].

- CT cisternograms
  - Advantages: Contrast may pool within frontal sinus, good bony detail
  - Disadvantages: Invasive, may not detect intermittent leaks
- MRI/MR cisternography
  - Advantages: Excellent soft tissue (CSF/brain vs secretions) detail, non-invasive
  - Disadvantage: Poor bony detail
- Intrathecal fluorescein

Advantages: Precise localization, blue light filter can improve sensitivity Disadvantages: Invasive, skull base exposure required for precise localization

## **Surgical Technique**

### Endoscopic Approaches

Defects involving up to 3 cm of the posterior table can be repaired endoscopically in select cases with proper equipment and expertise [27]. Lumbar drains are typically utilized to instill fluorescein as previously described [8] in patients with suspected elevated intracranial pressure (spontaneous CSF leaks), questionable defect site (some trauma), or for some large cranial base resections.

• Intrathecal fluorescein protocol: During placement of the lumbar drain, 0.1 cc of preservative-free 10 % fluorescein is placed in 10 cc of the patient's own CSF or normal saline and slowly delivered via the drain over 10 min.

This can aid with intra-operative localization of the defect and confirmation of a water tight seal at the conclusion of the case. To obtain adequate exposure, a total ethmoidectomy, maxillary antrostomy, and frontal sinusotomy, as well as partial middle turbinectomies or Draf III procedure may be indicated. The extent of dissection should be limited to that required for each individual defect. Using 70° nasal endoscopes, any encephalocele present should be ablated with bipolar cautery or radiofrequency coblation [28]. Mucosa is removed from around the defect or from involved fracture segments and the surrounding posterior table. This not only provides an area of adherence for the graft but also contributes to osteoneogenesis and osteitic bone formation. This thickens the bone around the defect and aids bony closure, if a bone graft is used, between the graft and recipient bed. If secondary to trauma, fracture segments can be manually reduced with a suction or probe as necessary to reestablish the contour of the posterior table or removed if multiple fragments are present (Figs. 34.1, 34.2, 34.3, 34.4, 34.5, 34.6, and 34.7).

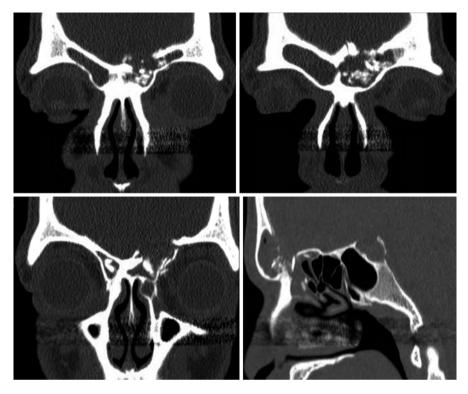


Fig. 34.1 Coronal and sagittal CT scans of a patient with severely comminuted posterior table fracture and CSF leak

The choice of grafts or flaps is often of personal preference, but may include alone or in combination the following:

- Bone
- Cartilage
- Mucosa
- Fascia
- · Alloplastic materials
- Xenografts
- Nasoseptal flap

For underlay grafts, a variety of alloplastic materials can be used, including porcine small intestine submucosal grafts (Biodesign(R), Cook Medical, Bloomington, In), bovine collagen matrix (Duragen, Integra LifeSciences, Plainsboro, NJ) or cadaveric dermal grafts (Alloderm, Lifecell, Bridgewater, NJ). These alloplastic materials vary in handling characteristics and amount of swelling with hydration and selection is typically individual surgeon preference. One universal advantage of alloplastic grafts is the elimination of donor site issues present with temporalis fascia or fascia lata [29]. Bone grafts are useful when the patient has a spontaneous

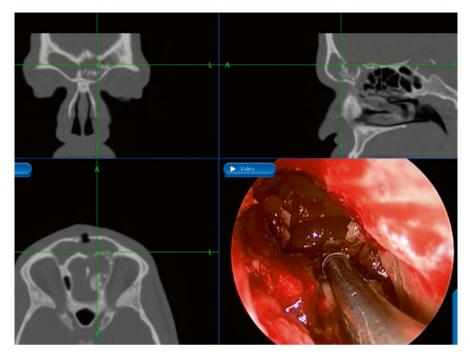
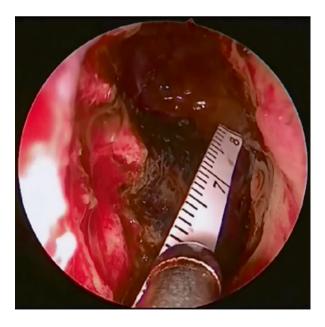


Fig. 34.2 Intraoperative triplanar imaging and endoscopic view following a Draf IIB frontal sinusotomy to expose the fracture segments



**Fig. 34.3** The fracture segments have been removed and the patient is left with a 3 cm defect and exposed dura with multiple tears

**Fig 34.4** Transnasal 70° endoscopic view of the frontal sinus at 1 year demonstrating a patent frontal sinusotomy and well healed skull base defect after repair with a Biodesign® underlay graft and overlay nasoseptal flap



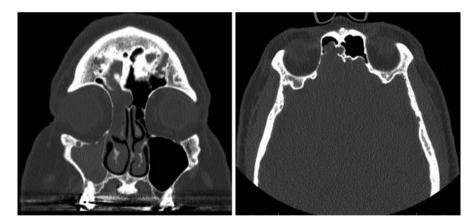
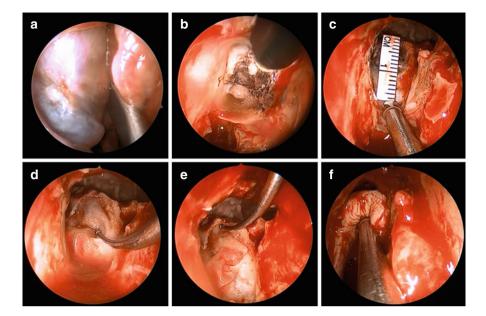


Fig. 34.5 Coronal and axial CT scans of a spontaneous CSF leak and encephalocele originating from the posterior table of the frontal sinus

leak and elevated intracranial pressures. This elevated pressure contributes to disruption of the soft tissue graft or flap and is likely responsible for the higher failure rates in this category. We prefer to use septal bone as this spares an external incision and can easily be harvested from the operative field. Regardless of the choice of bone, the graft is shaped to match the bony defect and placed in an underlay fashion in the epidural space after the underlay soft tissue graft. Care must be taken to avoid enlargement of the existing bony defect or entrapment of mucosa in the epidural space that may lead to an intracranial mucocele. A fascia, mucosal graft or



**Fig. 34.6** Steps in reconstruction. (a) The encephalocele is identified in the middle meatus of the right nasal cavity. (b) Following a right sided endoscopic sinus surgery, the encephalocele is reduced with radiofrequency coblation and bipolar cautery. (c) The defect in the posterior table is measured with a ruler and visualized with  $70^{\circ}$  endoscopy. (d) An underlay Biodesign® dural graft is placed. (e) An underlay septal bone graft is placed. (f) A nasoseptal flap is harvested and manipulated to traverse the posterior table defect



**Fig. 34.7** Six month postoperative endoscopic view illustrates the nasoseptal flap (*arrow*) crossing the base of the sphenoid sinus (*S*) and fixed to the medial orbital wall. (*Left* – 0°). A 70° view of the frontal sinus (*Right*) demonstrates the well healed nasoseptal flap and patent frontal sinus

nasoseptal flap [30] is then placed in an overlay fashion over the skull base defect and supported with gelfoam and intranasal gloved finger cots. To ensure adequate length of the nasoseptal flap, a hemitransfixion incision is performed anteriorly with elevation of the flap to the sphenopalatine foramen/lateral nasal wall as previously described [27]. The nasoseptal flap is vascularized and has been demonstrated to cover up to 3 cm of posterior table in some instances. In any case, multilayer repair with underlay grafts are generally recommended unless defects are small or linear cracks. Fibrin sealant (Eviceal, Ethicon, Somerville, NJ) or dural hydrogel sealant (Duraseal, Confluent Surgical, Waltham, MA) can be useful to "stick" the graft or flap in place during placement of supportive material. Non-absorbable packing is typically removed 7–13 days post-operatively.

Even with meticulous dissection and wide exposure of the frontal recess, the potential for obstruction of the frontal recess by grafts or packing material is high. To avoid this, we often will place a soft Silastic frontal stent until the first postoperative debridement. Careful debridement and cleaning every week for several weeks will lessen the incidence of scarring and make future surveillance easier.

### **Extracranial Repair**

Defects in the posterior table of the frontal sinus may not be amenable to a strict endoscopic approach. Surgeons with limited equipment or expertise should consider traditional open approaches. Leaks that are particularly difficult to repair are those that extend to the isthmus of the frontal sinus outflow tract. It is this site where the skull base transitions from the horizontal (axial) orientation of the ethmoid roof/ cribriform plate to the vertical (coronal) orientation of the posterior table. This area may require a combined approach due to the limits of an external osteoplastic approach from. A frontal trephine can provide access to the superior limits of the defect and endoscopes may be utilized through the trephine as well as from below, but if meticulous removal of mucosa from the entire frontal sinus with subsequent obliteration is required, an osteoplastic flap, rather than a trephine, is recommended. Importantly, posterior table defects can be repaired with an external, extracranial approach using a traditional osteoplastic flap with or without frontal sinus obliteration. A well pneumatized frontal sinus with a defect in the lateral recess can be repaired via an osteoplastic flap or trephine without compromising the frontal outflow tract (Fig. 34.8).

The specific technique for raising osteoplastic flaps is described elsewhere. After elevating the osteoplastic flap with direct access to the frontal sinus, preparation of the recipient bed and grafting is performed in a similar fashion as endoscopic management if the surgeon feels the frontal sinus outflow tract is not compromised, and the frontal drainage pathway will be left open. Fat obliteration should be performed if there is a question about the feasibility of a patent drainage pathway after repair. After all mucosal remnants are stripped and meticulously drilled with a diamond burr, underlay bone and overlay fascia grafts are placed as needed to close the Fig. 34.8 Isolated skull base defect in the lateral aspect of the frontal sinus without involvement of the frontal recess. Such defects could be repaired via trephine while maintaining patency of the frontal recess Property of MUSC Rhinology



defect. Bilateral obliteration for relatively small frontal sinuses or involvement of both posterior tables is recommended. Finally, the mucosa of the frontal recess is stripped and abdominal fat packed in the sinus.

## Intracranial Repair

Large defects in the posterior table, as seen in severe facial trauma or tumors, may benefit more from repair via a craniotomy with cranialization of the frontal sinus and pericranial flap. This approach provides excellent exposure of the defect and allows better access for removal of the mucosa, but does require a craniotomy and retraction on the frontal lobe with possible sequelae such as anosmia, intracranial hemorrhage or edema, epilepsy, and memory and concentration deficits [9].

## **Adjuncts and Postoperative Care**

Lumbar drains are a useful adjunct in the management of spontaneous (or elevated intracranial pressure) frontal sinus CSF leaks as they allow intracranial pressure monitoring after surgery to determine the need for pressure lowering adjuncts.

Additionally, intrathecal fluorescein improves localization when multiple defects are present Patients may have increased pressure postoperatively due to overproduction against a closed defect. We prefer to use a lumbar drain in select patients that will have elevated ICPs postoperatively and generally leave them in place for 2–3 days.

Acetazolamide (Diamox<sup>TM</sup>) is a diuretic that can be a useful adjunct in patients with elevated CSF pressures. It can decrease CSF production up to 48 % [3]. In a prospective study observing acetazolamide's effects on intracranial pressure in patients with CSF leak associated intracranial hypertension, acetazolamide (500 mg orally) significantly reduced ICP from  $32.0\pm7.4$  cm H20 (6 h post-clamping) to  $21.9\pm7.5$  cm H20 in a 4–6 h time frame [31]. However, the optimal timing, dosing, and long-term benefits of this approach have not been proven, but it may reduce the risk of developing subsequent skull base defects in patients with elevated CSF pressures. We periodically monitor electrolytes in any patient placed on long-term diuretic therapy.

### Conclusion

Frontal sinus CSF leaks are a difficult entity to manage. When possible, endoscopic repair will provide the least morbidity, but the location and size of the defect as well as the etiology often dictates customized management. Achieving the best possible results for patients with CSF leaks depends on a thorough understanding of the underlying pathophysiology and fundamental principles of medical and surgical treatment.

## References

- 1. Jones V, et al. Changing paradigms in frontal sinus cerebrospinal fluid leak repair. Int Forum Allergy Rhinol. 2012;2(3):227–32.
- 2. Blount A, et al. Cerebrospinal fluid volume replacement following large endoscopic anterior cranial base resection. Int Forum Allergy Rhinol. 2012;2:217–21.
- 3. Alexander NS, et al. Treatment strategies for lateral sphenoid sinus recess cerebrospinal fluid leaks. Arch Otolaryngol Head Neck Surg. 2012;138(5):471–8.
- 4. Schuster D, et al. Endoscopic resection of intracranial dermoid cysts. J Laryngol Otol. 2011;125(4):423–7.
- 5. Woodworth BA, Palmer JN. Spontaneous cerebrospinal fluid leaks. Curr Opin Otolaryngol Head Neck Surg. 2009;17(1):59–65.
- 6. Purkey MT, et al. Endoscopic repair of supraorbital ethmoid cerebrospinal fluid leaks. ORL J Otorhinolaryngol Relat Spec. 2009;71(2):93–8.
- 7. Banks CA, et al. Endoscopic closure of CSF rhinorrhea: 193 cases over 21 years. Otolaryngol Head Neck Surg. 2009;140(6):826–33.
- Woodworth BA, et al. Spontaneous CSF leaks: a paradigm for definitive repair and management of intracranial hypertension. Otolaryngol Head Neck Surg. 2008;138(6):715–20.
- 9. Woodworth BA, Schlosser RJ. Repair of anterior skull base defects and CSF Leaks. Op Technol Otolaryngol. 2006;18:111–6.

- Woodworth BA, Neal JG, Schlosser RJ. Sphenoid sinus cerebrospinal fluid leaks. Op Technol Otolaryngol. 2006;17:37–42.
- 11. Woodworth BA, et al. Evolutions in the management of congenital intranasal skull base defects. Arch Otolaryngol Head Neck Surg. 2004;130(11):1283–8.
- 12. Choi M, et al. A 10-year review of frontal sinus fractures: clinical outcomes of conservative management of posterior table fractures. Plast Reconstr Surg. 2012;130(2):399–406.
- 13. Woodworth BA, Schlosser RJ, Palmer JN. Endoscopic repair of frontal sinus cerebrospinal fluid leaks. J Laryngol Otol. 2005;119(9):709–13.
- Conger Jr BT, Riley K, Woodworth BA. The Draf III mucosal grafting technique: a prospective study. Otolaryngol Head Neck Surg. 2012;146(4):664–8.
- 15. Gerbino G, et al. Analysis of 158 frontal sinus fractures: current surgical management and complications. J Craniomaxillofac Surg. 2000;28(3):133–9.
- 16. Chaaban MR, et al. Transnasal endoscopic repair of posterior table fractures. Otolaryngol Head Neck Surg. 2012;147(6):1142–7.
- 17. Zlab MK, et al. Cerebrospinal fluid rhinorrhea: a review of the literature. Ear Nose Throat J. 1992;71(7):314–7.
- Bernal-Sprekelsen M, Bleda-Vazquez C, Carrau RL. Ascending meningitis secondary to traumatic cerebrospinal fluid leaks. Am J Rhinol. 2000;14(4):257–9.
- 19. Woodworth B, Schlosser RJ. Endoscopic repair of a congenital intranasal encephalocele in a 23 months old infant. Int J Pediatr Otorhinolaryngol. 2005;69(7):1007–9.
- 20. Schlosser RJ, et al. Spontaneous cerebrospinal fluid leaks: a variant of benign intracranial hypertension. Ann Otol Rhinol Laryngol. 2006;115(7):495–500.
- 21. Gassner HG, et al. CSF rhinorrhea: 95 consecutive surgical cases with long term follow-up at the Mayo Clinic. Am J Rhinol. 1999;13(6):439–47.
- Hubbard JL, et al. Spontaneous cerebrospinal fluid rhinorrhea: evolving concepts in diagnosis and surgical management based on the Mayo Clinic experience from 1970 through 1981. Neurosurgery. 1985;16(3):314–21.
- Schick B, et al. Long-term study of endonasal duraplasty and review of the literature. Ann Otol Rhinol Laryngol. 2001;110(2):142–7.
- Psaltis AJ, et al. A systematic review of the endoscopic repair of cerebrospinal fluid leaks. Otolaryngol Head Neck Surg. 2012;147(2):196–203.
- 25. Chaaban MR, Illing E, Riley KO, Woodworth BA. Spontaneous cerebrospinal fluid leak repair: a five-year prospective evaluation. Laryngoscope. 2014 Jan;124(1):70–5.
- Schlosser RJ, Bolger WE. Nasal cerebrospinal fluid leaks: critical review and surgical considerations. Laryngoscope. 2004;114(2):255–65.
- Virgin F, et al. Frontal sinus skull base defect repair using the pedicled nasoseptal flap. Otolaryngol Head Neck Surg. 2011;145:338–40.
- Smith N, Riley KO, Woodworth BA. Endoscopic Coblator-assisted management of encephaloceles. Laryngoscope. 2010;120(12):2535–9.
- Illing E, et al. Porcine small intestine submucosal graft for endoscopic skull base reconstruction. Int Forum Allergy Rhinol. 2013;3(11):928–32. IN Press.
- 30. Hadad G, et al. A novel reconstructive technique after endoscopic expanded endonasal approaches: vascular pedicle nasoseptal flap. Laryngoscope. 2006;116(10):1882–6.
- Chaaban MR, et al. Acetazolamide for high intracranial pressure CSF leaks. Int Forum Allergy Rhinol. 2013;3(9):718–21.

# **Chapter 35 Inverted Papilloma of the Frontal Sinus**

### Kenneth Rodriguez and Brent A. Senior

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### **Core Messages**

- Inverted papilloma (IP) of the frontal sinus is rare, composing only about 2.5 % of all IP
- CT imaging may identify areas of bone thickening associated with sites of origin
- MRI may help to distinguish tumor from retained secretions
- If open osteoplastic approach is deemed necessary, obliteration of the sinus should be avoided

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_35

Special mention to Dr. Wasman (Dr. Jay K. Wasman, MD, Assistant Professor of Pathology, University Hospitals Case Medical Center) for providing the pathology image and explanation.

## Introduction

Inverted papilloma (IP) represents a tumor that is commonly seen in high volume rhinology practices. Although originating within the frontal sinus is uncommon, lack of identification or misdiagnosis can lead to poor patient outcomes given the risk of malignancy within IP. These tumors most often present with non-specific symptoms such as nasal airway obstruction, facial pressure, or epistaxis and are typically unilateral. A thoughtful history as well as comprehensive exam with nasal endoscopy is crucial to work up these tumors. First line imaging is with non-contrast CT of the sinuses with coronal and sagittal reconstructions. The additional reconstructed views aid in surgical planning as well as evaluation of the cranial base. MRI through the skull base can be a helpful adjunct (with and without contrast) to evaluate for extension into the soft tissues outside of the paranasal sinuses. Treatment is with surgical resection, most commonly via an endoscopic endonasal approach with open assist (trephine, osteoplastic flap) as needed for more complex tumors.

## History

Ward originally reported IP in 1854 and in 1855 Billroth described two cases of papillomatous growths in the nasal cavity characterizing them as "villiform cancers" [1, 2]. Further pathological characterization followed and in 1935 when Kramer and Som published on 86 cases and were the first to distinguish IP from inflammatory polyps [3]. IP has a complex history of nomenclature and has been called more than 50 different names, which has led to confusion about what constitutes IP versus other similar variants. Three morphologically distinct papillomas arise from the mucosa that lines the nasal cavity and paranasal sinuses now called exophytic, inverted, and oncocytic papillomas; collectively known as Schneiderian papillomas [4]. Our focus in this chapter will be the IP subtype.

## Presentation

The incidence of IP has been derived from numerous series and occurs in 0.6–1.5 cases per 100,000 people, usually affecting patients in the fifth and sixth decades of life [5–12]. They represent approximately 0.4–4.7 % of all sino-nasal tumors (second most common benign tumor behind osteoma) and 47–79 % of all Schneiderian papillomas [4]. Symptoms are nonspecific and include unilateral nasal obstruction, epistaxis, nasal drainage or sinusitis [13, 14]. IP has a 2 to 5:1 male to female ratio and has no side predilection [15–17]. Weissler et al. in a review of 223 patients with IP, found 9 % bilateral (most due to direct spread through the septum) and 12 % multi-centric [13].

• Inverted papilloma usually originates from the middle meatus or lateral nasal wall but involves at least one paranasal sinus 82 % of the time and can arise from the septum in 8 % of patients [12, 17, 18].

Diagnosis and prompt treatment of IP is critical given its association with malignancy. A review of 1390 of published IP's demonstrated association with carcinoma in 150 cases or 11 % [4]. Although the most commonly seen cancer is squamous cell carcinoma, verrucous, mucoepidermoid, spindle cell, clear cell and adenocarcinoma have also been reported [19–23].

Grossly IP appears to be exophytic and polypoid and is typically pink to gray in color (Fig. 35.1). Often IP has a discrete pedicled site that is far smaller than the actual tumor mass with the lesion draping over normal mucosa without invasion. This normal mucosa does not need to be resected along with the tumor but finding the actual attachment point should be a priority when resecting these tumors [14]. IP originates from ciliated respiratory mucosa of the sinonasal tract and diagnosis is established via biopsy. Microscopically there is digitiform proliferation of squamous epithelium into the underlying stroma. Its name derives from the inverted (endophytic) growth pattern (Fig. 35.2).

The exact cause of IP remains unclear. There has been significant recent interest in whether there is an association with human papilloma virus (HPV) as seen with recurrent respiratory papillomatosis or squamous cell carcinoma of the oropharynx. The data on this topic is mixed. Some studies suggest that HPV detection is the result of incidental colonization while other studies have implicated HPV as involved in the pathogenesis of the disease and potentially responsible for malignant transformation [24, 25]. The data remains controversial concerning the role of HPV infection in IP formation and possible role in malignant degeneration with no clear consensus presently available.



**Fig. 35.1** Left nasal cavity of a patient who has had two prior attempted resections of a left ethmoid/frontal sinus inverted papilloma that is seen in this image between the inferior and middle turbinates



**Fig. 35.2** Sinonasal (Schneiderian) Papilloma, Inverted Type: This photomicrograph exhibits an endophytic/downward ("inverted") growth pattern. The epithelium is comprised of multiple cell types, including squamous, transitional and columnar cells; admixed goblet cells and intraepithelial mucous cysts are typically present. Scattered chronic inflammatory cells are present within the edematous stroma of the papilloma. (H&E,  $10\times$ )

# **Staging/Treatment**

There is no generally adopted staging system for IP. Many systems have been proposed (Table 35.1) each with advantages and drawbacks. Krouse's system emphasized tumor extension beyond the maxillary sinus as an important prognostic factor and included malignancy as his highest stage [26]. Han's system did not include malignancy, and placed frontal sinus disease or extension beyond the medial maxillary sinus at higher stages [12]. Cannady's system is based on the recurrence risk centered on location [27]. Each system demonstrates high stages for frontal sinus involvement underscoring the increased level of complexity involved with that anatomical location. In the event of pathological discovery of SSC, the TNM staging system as found in the American Joint Committee on Cancer staging manual should be utilized [28]. Malignancies of the frontal sinus are covered in detail elsewhere in this text.

IP management begins with comprehensive history and physical exam with focus on nasal endoscopy. It is critical to assess for pre-operative cranial neuropathies by testing facial sensation, baseline vision, extraocular movements, and sense of smell. Imaging should start with a non-contrast CT of the paranasal sinuses with coronal and sagittal reconstructions.

Imaging for inverted papilloma:

- Computed tomography A unilateral mass is typically seen with focal hyperostosis that can often reflect the point of origin of the tumor [14, 29–31].
- MRI can assist to differentiate tumor from post-obstructive secretions and highlight invasion into the soft tissue of the orbit or brain in regions of bony dehiscence.

Krouse staging system for inverted papilloma (2000)	
T1 confined to the nasal cavity	
T2 ostiomeatal complex region, ethmoid, or medial maxillary involvement (with or withou nasal cavity involvement)	ıt
T3 any wall of maxillary sinus but medial, frontal sinus, or sphenoid with or without T2 cr	d with or without T2 criteria sinus, ethmoid sinus, and group I criteria uses, or medial maxillary wall
T4 any extrasinus involvement or malignancy	
Han staging system for inverted papilloma (2001)	
Group I limited to nasal cavity, lateral nasal wall, medial maxillary sinus, ethmoid sinus, a sphenoid sinus	nd
Group II extension lateral to medial maxillary wall with or without group I criteria	
Group III extension into frontal sinus	
Group IV extension outside sinuses	
Cannady staging system for inverted papilloma (2007)	
Group A inverted papilloma confined to the nasal cavity, ethmoid sinuses, or medial maxillar	ry wal
Group B inverted papilloma with involvement of any maxillary wall (other than the media wall), or frontal sinus, or sphenoid sinus	1

Table 35.1 Common staging systems for sinonasal inverted papilloma

Group C inverted papilloma with extension beyond the paranasal sinuses

MRI is typically not required for routine cases. IP is isodense on T1-weighted images and iso- to hyperdense on T2 weighted images with contrast administration leading to heterogeneous enhancement [32]. It is important to keep in mind that the differential for any unilateral nasal mass should include IP (and other Schneiderian papillomas), antrochoanal polyp, encephalocele, fungus ball, juvenile nasal angiofibroma (males), and malignancy (Squamous cell carcinoma, Sino-nasal undifferentiated carcinoma, lymphoma etc.). Unilateral tumors need to be worked up appropriately and in almost all cases be biopsied or excised to determine the exact pathology.

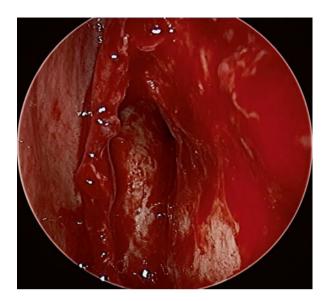
Complete surgical excision has been recognized as the cornerstone to curing IP for several decades. The evolution from external to endo-nasal techniques has evolved as technology and comfort with that approach has become mainstream. Frontal sinus IP represents an intrinsically complicated form of the disease given the need for complete surgical excision in an area that most sinus surgeons are uncomfortable. The need for approaches such as the lateral rhinotomy and midfacial degloving, popular in the 1970s and 1980s for frontal sinus IP, is likely no longer needed for the majority of disease. In addition, combination with an endonasal and coronal approach (osteoplastic flap) spares the patient facial scarring and provides excellent visualization when needed. In 2006 a meta-analysis by Busquets and Hwang helped to more fully validate endoscopic techniques [33]. They divided the literature into two groups: pre (1970-1995) and post (1992–2004) endoscopic periods. Without consideration of tumor size, they found a significantly lower recurrence rate in the endoscopic era compared to the nonendoscopic era (15 % v 20 %, P=0.02). Endoscopically treated patients were found to have a significantly lower recurrence rates (12 % v 20 % (non), P<0.01) and recurrence rate for non-endoscopic treated patients in the endoscopic era was found to be equivalent to that of the non-endoscopic patients in the pre-endoscopic surgery era (20 % v 19 %, P=0.78).

• IP is found to originate from the frontal sinus in only 2.5 % patients [34].

More commonly it is found in the ethmoid region (48 %), maxillary sinus (28 %), and sphenoid sinus (7.5 %) [34]. In the vast majority of patients, en bloc resection is not possible via an endoscopic approach. Endoscopic surgical resection should begin with debulking of the tumor to identify the site of attachment. Mucosal preservation of uninvolved sites should be attempted but the site of origin and the adjacent mucosa and underlying bone should be removed (or thinned) to clear the deep margin [35, 36]. Forethought prior to resection of IP involving regions adjacent to the skull base and lamina is a must. If bone in these regions is removed without complete tumor excision, residual disease can invade the periorbita or dura creating a dangerous clinical picture necessitating removal of these critical tissues at a later time to clear margins exposing the patient to possible medial rectus or superior oblique injury or CSF leak-requiring repair. There is no clear clinical answer in terms of whether to intentionally leave tumor behind over critical bone (assuming that all pathology is benign) versus giving someone potentially debilitating injuries to clear a benign tumor. If these regions of bone can be thinned and not removed that may be a sufficient balance and diligent follow up becomes critical. Frozen section mucosal margins can be helpful to insure clearance of disease at the time of primary resection. Staging of the procedure can also be performed with debulking of the inferior tumor and later open approach if required [37]. There has been no increased risk of malignancy with multiple local recurrences [38].

When considering whether a strictly endoscopic complete resection can be achieved one must address the probable origin of the tumor and if that site can be addressed via an endo-nasal approach, the size of the frontal recess and the extension of the tumor laterally within the frontal sinus. The septum must be taken into account as septoplasty may be needed for access and that time should be accounted for as part of the approach. The middle turbinate can be resected to provide further arc of rotation for instruments within the nasal cavity for further reach laterally within the frontal (Fig. 35.3). As discussed above, focal bony thickening can be an indication of the site of attachment and MRI can help delineate the extent of trapped secretions seen lateral the lesion often seen in frontal recess disease. In a review published in 2009, the authors state that multifocal IP with attachment to the anterior wall, lateral wall, or contralateral frontal sinus is not generally amenable to endoscopic resection [39]. Modified Lothrop procedures can be considered to allow for improved visualization and for medial tumors. Comfort with angled scopes is a must as is familiarity with the array of frontal sinus instrumentation and drill types currently available. If conversion to an open approach is being considered expertise with trephine, osteoplastic flaps, as well as different incision types such as brow, hairline, and coronal are needed and a comprehensive pre-operative discussion of the risks and benefits of each approach should be performed. In addition, adequate time must be allocated for these tumors often much longer than a typical sinus procedure. If tumor is intentionally left behind, any

**Fig. 35.3** Postoperative image of same patient as seen in image 1. He has had complete resection of the inverted papilloma with stripping of all mucosa along lamina (picture *right*), ethmoid skull base, and into the frontal sinus. His middle turbinate was resected (medial) to just below the skull base. Bone was drilled at the level of the frontal recess at the attachment point



aggressive features such as new facial numbness, vision changes, or signs of intra-cranial extension such as mental status changes or new onset headaches should be evaluated to insure malignancy is ruled out and, if present, handled appropriately.

• Frontal sinus obliteration techniques should not be used given the difficulty with differentiating tumor from materials used to fill the sinus.

To determine success with resection of frontal sinus IP, a retrospective chart review of 18 cases with an endonasal endoscopic approach as the primary surgical modality were reviewed [39]. Seventeen cases were benign IP with one case having concurrent squamous cell carcinoma. Six cases (33 %) were primary IP and 12 cases (67 %) were residual or recurrent disease with the IP being unifocal in 10 cases and multifocal in 8 cases. Adjunct open procedures were required in seven cases with five cases necessitating trephine and two requiring osteoplastic flap. Two developed cerebrospinal fluid (CSF) leaks that were managed intraoperatively. Four (22 %) patients developed recurrences and were re-resected. All patients were free of disease at the time of last evaluation with a mean follow-up of 36.6 months (range, 11–114 months).

In 2012, a review of the literature from 1995 to 2010 was performed dealing with frontal sinus IP [40]. Fifty-seven cases were identified in 13 studies, with 49 cases deemed adequate for additional analysis. Twenty-four cases (49 %) were primary, and 25 (51 %) were secondary (residual or recurrent disease). Bilateral frontal sinus involvement was reported in eight cases (16.3 %). The surgical approaches utilized included endoscopic frontal sinusotomy in 21 (42.9 %), endoscopic modified Lothrop in 10 (20.4 %), osteoplastic flap in 13 (26.5 %), and endoscopic trephination and endoscopic frontal sinusotomy in 5 (10.2 %) patients. The overall rate of recurrence was 22.4 %, though when broken down by approach, 23.8 % recurrence

was seen with endoscopic frontal sinusotomy, 30 % with endoscopic modified Lothrop, 15.4 % with osteoplastic flap and 20 % with endoscopic frontal trephination with no statistically significant differences between methods.

Following surgical resection patients should be followed initially at close intervals. Follow up at 1, 2, and 4 weeks post operative intervals allows for debridements and avoidance of scarring at the surgical site that can limit direct surveillance and can confuse imaging later on in regards to differentiating scar from tumor. Since the mucosa is stripped in these patients and the region of the tumor attachment is drilled they will develop significant granulation tissue at the site of resection. Consideration of nonabsorbable packing within the resection site or drug eluding stents can be considered to keep the frontal outflow tract patent. It may be helpful to utilize a similar instrument to the one used in the operating room setting to perform debridements and cannulate the frontal sinus in clinic. Revision procedures may be needed if the frontal sinus outflow scars to re-open the sinus and minimize the need for frequent imaging to follow patients in the long term. Once the surgical site is well healed surveillance is key. We advocate 3-month follow-ups for 2 years then 6 month follow up until 5 years from surgery and then annual surveillance indefinitely.

Radiotherapy is rarely used for histologically benign IPs given that surgical excision is curative. Recent literature supports its use for tumors that are inoperable due to the extent of disease, medical co-morbidities resulting in an unacceptably high risk for peri-operative medical complications (ex: unstable angina) incomplete resection, history of multiple recurrences, or malignant degeneration within the specimen [41].

## Conclusion

IP represents pathology that must be considered in the differential of any nasal or sinus tumor. Comprehensive management requires a stepwise approach. Nasal endoscopy with CT imaging can assist in determining the point of origin. Complete resection, either through an endoscopic, combined, or open approach is the standard of care. Malignant degeneration should be staged through the AJCC guidelines and managed accordingly. Radiation can be considered in select cases for benign IP. Close follow up is mandatory.

### References

- 1. Ward N. A mirror of the practice of medicine and surgery in the hospitals of London: London Hospital. Lancet. 1854;2:480–2.
- 2. Billroth T. Uber dem Bau der Schleimpolyp. Berlin: G. Reimer; 1855.
- 3. Kramer R, Som MI. True papilloma of the nasal cavity. Arch Otolaryngol. 1935;22:22–43.
- Barnes L. Diseases of the nasal cavity, paranasal sinuses and nasopharynx. In: Barnes L, editor. Surgical pathology of the head and neck. New York: Informa Healthcare; 2009. p. 343–422.

- 5. Verner JL, Maguda TA, Yound JM. Epithelial papillomas of the nasal cavity and sinuses. Arch Otolaryngol. 1959;70:574–8.
- Ringertz N. Pathology of malignant tumors arising in nasal and paranasal cavities and maxilla. Acta Otolaryngol. 1938;27:31–42.
- Lampertico P, Russell WO, MacComb WS. Squamous papilloma of the upper respiratory epithelium. Arch Pathol. 1963;75:293–302.
- Buchwald D, Nielsen LH, Nielsen PL, et al. Inverted papilloma: a follow-up study including primarily unacknowledged cases. Am J Otolaryngol. 1989;10:273–81.
- 9. Skolnick EM, Loewy A, Friedman JE. Inverted papilloma of the nasal cavity. Arch Otolaryngol. 1966;84:83–9.
- Buchwald C, Franzmann MB, Tos M. Sinonasal papillomas: a report of 82 cases in Copenhagen County, including a longitudinal epidemiological and clinical study. Laryngoscope. 1995; 105(1):72–9.
- Outzen KE, Grontveld A, Jorgensen K, Clausen PP, Ladefoged C. Inverted papilloma: incidence and late results of surgical treatment. Rhinology. 1996;34(2):114–8.
- Han JK, Smith TL, Loehrl TA, et al. An evolution in the management of sinonasal inverting papilloma. Laryngoscope. 2001;111:1395–400.
- 13. Weissler MC, Montgomery WW, Turner PA, et al. Inverted papilloma. Ann Otol Rhinol Laryngol. 1986;95:215–21.
- Melroy CT, Senior BA. Benign sinonasal neoplasms: a focus on inverting papilloma. Otolaryngol Clin North Am. 2006;39:601–17.
- Krouse JH. Endoscopic treatment of inverted papilloma: safety and efficacy. Am J Otolaryngol. 2001;22:87–99.
- 16. Bielamowicz S, Calcaterra TC, Watson D. Inverting papilloma of the head and neck: the UCLA update. Otolaryngol Head Neck Surg. 1993;109(1):71–6; 109(1):71–6.
- Phillips PP, Gustafson RO, Facer GW. The clinical behavior of inverting papilloma of the nose and paranasal sinuses: report of 112 cases and review of the literature. Laryngoscope. 1990;100(5):463–9.
- Probst L, Stoney P, Jeney E, et al. Nasal polyps, bronchial asthma and aspirin sensitivity. J Otolaryngol. 1992;21:60–5.
- Myers EN, Schramm Jr VL, Barnes Jr EL. Management of inverted papillomas of the nose and paranasal sinuses. Laryngoscope. 1981;91:2071–84.
- Orvidas LJ, Lewis JE, Olsen KD, et al. Intranasal vertucous carcinoma: relationship to inverting papillomas and the human papillomavirus. Laryngoscope. 1999;109:371–5.
- Momose KJ, Weber AI, Goodman M, et al. Radiologic aspects of inverted papilloma. Radiology. 1980;134:73–9.
- 22. Oberman HA. Papillomas of the nose and paranasal sinuses. Am J Clin Pathol. 1964;42:245–58.
- Snyder RN, Perzin KH. Papillomatosis of nasal cavity and paranasal sinuses (inverted papilloma, squamous papillo- ma). A clinicopathologic study. Cancer. 1971;30:668–90.
- 24. Jenko K, Kocjan B, Zidar N, Poljak M, Strojan P, Zargi M, Blatnik O, Gale N. In inverted papillomas HPV more likely represents incidental colonization than an etiological factor. Virchows Arch. 2011;459(5):529–38.
- Hwang CS, Yang HS, Hong MK. Detection of human papillomavirus (HPV) in sinonasal inverted papillomas using polymerase chain reaction (PCR). Am J Rhinol. 1998;12(5):363–6.
- 26. Krouse JH. Development of a staging system for inverted papilloma. Laryngoscope. 2000; 110:965-8.
- 27. Cannady SB, Batra PS, Sautter NB, Roh HJ, Citardi MJ. New staging system for sinonasal inverted papilloma in the endoscopic era. Laryngoscope. 2007;117:1283–7.
- 28. Fleming ID. AJCC cancer staging manual. 7th ed. Published by Springer New York; 2010.
- 29. Som PM, Lawson W, Lidov MW. Simulated aggressive skull base erosion in response to benign sinonasal disease. Radiology. 1991;180:755–9.
- Lee DK, Chung SK, Dhong HJ, Kim HY, Kim HJ, Bok KH. Focal hyperostosis on CT of sinonasal inverted papilloma as a predictor of tumor origin. AJNR Am J Neuroradiol. 2007;28(4):618–21.

- Roobottom CA, Jewell FM, Kabala J. Primary and recurrent inverting papilloma: appearances with magnetic resonance imaging. Clin Radiol. 1995;50:472–5.
- Yousem DM, Fellows DW, Kennedy DW, et al. Inverted papilloma: evaluation with MR imaging. Radiology. 1992;185(2):501–5.
- Busquets JM, Hwang PH. Endoscopic resection of sinonasal inverted papilloma: a metaanalysis. Otolaryngol Head Neck Surg. 2006;134(3):476–82.
- 34. Lawson W, Patel ZM. The evolution of management for inverted papilloma: an analysis of 200 cases. Otolaryngol Head Neck Surg. 2009;140(3):330–5.
- 35. Jameson MJ, Kountakis SE. Endoscopic management of extensive inverted papilloma. Am J Rhinol. 2005;19:446–51.
- Wolfe SG, Schlosser RJ, Bolger WE, et al. Endoscopic and endoscope-assisted resections of inverted sinonasal papillomas. Otolaryngol Head Neck Surg. 2004;131:174–9.
- Dubin MG, Sonnenburg RE, Melroy CT, Ebert CS, Coffey CS, Senior BA. Staged endoscopic and combined open/endoscopic approach in the management of inverted papilloma of the frontal sinus. Am J Rhinol. 2005;19(5):442–5.
- Lawson W, Le Benger J, Som P, Bernard PJ, Biller HF. Inverted papilloma: an analysis of 87 cases. Laryngoscope. 1989;99:1117–24.
- Yoon BN, Batra PS, Citardi MJ, Roh HJ. Frontal sinus inverted papilloma: surgical strategy based on the site of attachment. Am J Rhinol Allergy. 2009;23:337–41.
- 40. Walgama E, Ahn C, Batra PS. Surgical management of frontal sinus inverted papilloma: a systematic review. Laryngoscope. 2012;122:1205–9.
- Gomez JA, Mendenhall WM, Tannehill SP, Stringer SP, Cassisi NJ. Radiation therapy in inverted papillomas of the nasal cavity and paranasal sinuses. Am J Otolaryngol. 2000;21: 174–8.

# **Chapter 36 Fibro-osseous Lesions of the Frontal Sinus**

Kenneth Rodriguez, Mohamed Tomoum, and Brent A. Senior

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_36

#### **Core Messages**

- Osteomas involving the frontal sinus are common though controversy exists as to the appropriate timing for removal
- Fibrous dysplasia (FD) involving the frontal sinus has diffuse borders on CT imaging and frequently causes cosmetic and functional disability requiring surgical management
- Ossifying fibroma may be confused with FD by imaging though having more defined borders but should be differentiated in light of its more aggressive growth

## Introduction

Fibro-osseous lesions of the frontal sinus represent an uncommon spectrum of paranasal sinus pathology. This chapter will review the clinical presentation, radiographical findings, and treatment of the most common fibro osseous lesions of the frontal sinus: osteoma, fibrous dysplasia, and ossifying fibroma. Although not covered in detail in this chapter, eosinophilic granuloma, Paget's disease, and malignant lesions (see Chap. 37) should be considered in any differential of frontal sinus pathology.

## Osteoma

Veiga is credited as being the first to describe a frontal sinus osteoma in 1506 [1]. In 1733, Vallisnieri detailed their bony origin as opposed to representing petrified brain [2]. Paranasal sinus osteomas are seen in 3 % of computed tomography (CT) examinations obtained for sino-nasal symptoms [3]. The frontoethmoid region is the most common location for osteomas representing more than 90 % of those found in the paranasal sinuses [4]. These bony tumors typically present in the third to fourth decade of life and have a male to female ratio of 1.5-2:1 [5, 6].

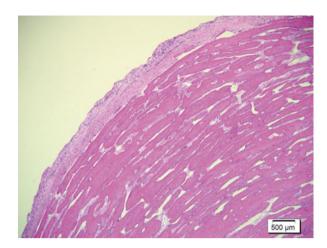
• Osteomas tend to grow slowly as demonstrated in a study of 89 patients were the mean growth rate was 0.79 mm/year in the cephalocaudal direction and 0.99 mm/ year in the mediolateral direction [7].

The mean follow up of that group was 54 months and 46 of 89 patients showed an increase in the size of their osteoma demonstrating persist growth is not necessarily to be expected [7].

# Etiology/Histology

Since the diagnosis of paranasal sinus osteoma is established incidentally in most cases, it is typically not possible to know the age of a patient's lesion. There are three accepted theories of the etiology of frontal sinus osteoma: developmental,

Fig. 36.1 Osteoma: this lesion is composed of interconnecting trabeculae of lamellar bone with vascular channels similar to haversian systems and minimal fibrous stroma. Both osteoblasts and osteocytes are small, inconspicuous, and difficult to appreciate on this low power image. (H&E, 40×)



traumatic, or infectious [8]. The developmental theory states that these tumors tend to arise in areas of fusion between tissues of varying embryologic origin such as the membranous frontal bone and cartilaginous ethmoid bone but many paranasal osteomas arise at sites distant from these junctions [9]. The traumatic theory and the infectious theory both rely on an inflammatory process as the inciting force for bony tumor formation. It is difficult to determine if the osteoma or the infection is the primary process as tumor and infection frequently coexist at the time of diagnosis. However, the type of bone in an osteoma differs significantly from the bony hyperplasia expected to characterize reactive osteitis [8].

• When dealing with multiple paranasal osteomas in the clinical context of intestinal polyps, fibromas, lipomas, neurofibromas, epidermoid cysts, abnormal teeth, and pigmented skin lesions, Gardner's syndrome must be considered [10].

It is autosomal dominant, and typically presents in the second decade of life with rectal bleeding, diarrhea, or abdominal pain [11]. With a propensity to malignant degeneration of the intestinal polyps, early diagnosis is critical.

Histologically, there are three types of osteomas; eburnated, mature, or mixed (Fig. 36.1). The eburnated type, also known as the ivory or compact type, is very dense and lacks Haversian canals [12, 13]. The mature type, or osteoma spongiosum, is composed of softer bone more similar to cancellous bone [14]. The mixed type of osteoma contains elements of both the eburnated and mature forms.

### **Clinical Manifestations**

While as many as 60 % of patients with frontal osteomas present with a chief complaint of headache, many patients diagnosed with an osteoma of the paranasal sinuses are asymptomatic [15–17]. As a result of this slow, asymptomatic growth, frontal osteomas can present with complications secondary to slow expansion. Anterior table erosion can lead to cosmetic deformity or palpebral abscess, orbital compression can



**Fig. 36.2** Coronal noncontrast CT image of a left midline frontal region osteoma with resultant mucosal thickening in the frontal sinus

lead to diplopia from mass effect and blockage of sinus ostia can lead to mucocele formation and chronic frontal sinusitis [7]. Posterior table erosion with intracranial extension can lead to CSF leakage, pneumocephalus, meningitis or brain abscess [18].

High-resolution computed tomography (CT) without contrast with coronal and sagittal reconstructions is the study of choice to assess the extent of the tumor. Frontal osteomas are usually easily identified, appearing as round or oval-shaped, dense, homogeneous, well-circumscribed masses on CT (Fig. 36.2), however, small lesions occurring in the frontal recess may be mistaken for thickening of the bony frontal sinus beak [7].

### Management

• The primary dilemma in patients with an osteoma is determining if or when surgical removal is indicated with a variety of opinions represented in the literature.

There is general agreement that rapidly growing or symptomatic tumors should be removed [19]. Since most osteomas are asymptomatic, many investigators advocate periodic imaging to follow growth and allow intervention before the development of complications [20]. It can be argued that expected tumor growth combined with radiation risk of annual imaging and the relatively minimal morbidity from endoscopic resection, particularly of smaller lesions, warrants earlier surgical intervention. Clearly this concept is predicated on having no complications associated with the resection so careful consideration combined with detailed discussion with the patient about potential risks and about prognosis of having an osteoma should be undertaken prior to any attempted resection. A reasonable observation plan for small, asymptomatic lesions would entail follow up non-contrast sinus CT at 6 months after initial discovery and an annual follow-up CT thereafter. If there is no growth of the osteoma the imaging can be spaced to longer intervals. Patients should be educated about symptoms and complications of osteoma growth and should be instructed to seek evaluation with any new symptoms [8].  Larger tumors at discovery that occupy more than 50 % of the frontal sinus or posterior table based tumors should be considered for early removal to prevent future complications [12, 20–22].

Surgical approaches to the frontal sinus for removal of osteomas can be categorized as endoscopic, external, or combined. In deciding which approach is ideal for a given patient, consideration should be made for total resection with minimal associated morbidity while minimizing frontal sinus mucosal trauma during resection in order to maintain healthy function of the frontal sinus post-operatively.

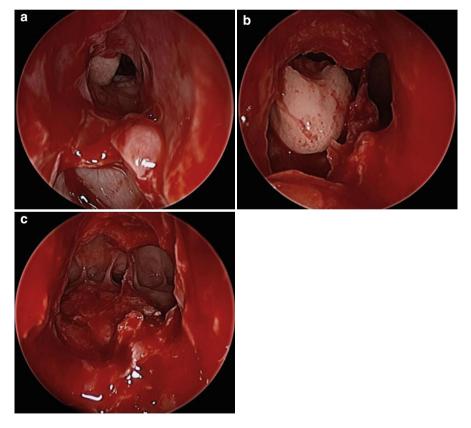
The first widely applied method used to treat symptomatic frontal or frontoethmoid osteomas was the external frontoethmoidectomy (Lynch procedure). While effective for removal of fairly large lesions, this technique has the potential for greater risk of intracranial penetration and subsequent CSF leak (compared to endoscopic approaches), as well as scarring with a relatively high rate of frontal recess stenosis [21]. An alternative to the Lynch procedure, the osteoplastic approach, has also been widely used for frontal sinus osteomas [23]. This approach provides excellent visualization and wide access to the frontal sinus but it does carry the risks of poor cosmesis with scarring and possible post-operative frontal bossing, as well as postoperative frontal pain, paresthesias or anesthesia from supraorbital nerve damage. With open approaches the surgeon must also decide whether the sinus should be obliterated following resection. In a patient with multiple previous surgeries for chronic sinusitis, or a large osteoma that resulted in significant stripping of mucosa after its removal, obliteration should be considered [4]. In most cases, obliteration is not necessary, and is best avoided in order to provide restoration of function to the sinus while preserving the ability to monitor for tumor recurrence either radiographically or by endoscopy [24]. If the frontal sinus is obliterated, then the added morbidity of an addition tissue harvest, such as abdominal incision for fat is introduced, as well as the risk of late mucocele formation, which can be as high as 9 % after 2 years [21].

Craniofacial resection has also been advocated for extremely large lesions with significant extension into the surrounding structures. A report of eight patients by Blitzer with massive residual and/or recurrent lesions revealed no recurrences at 4-year follow-up using that technique [25].

The first report of an endoscopic approach to a frontal sinus osteoma was by Busch in 1992 [26]. Subsequently, Kennedy et al. reported on the endoscopic management of bony tumors with intracranial or intraorbital extension, while Senior and Lanza reported on the use of endoscopic techniques in isolation and in combination with open approaches to remove tumors with frontal sinus involvement [27, 28]. As technology has advanced (image guidance, angled drill burrs, etc) surgeons have begun to remove even more extensive osteomas using transnasal endoscopic approaches in isolation, or possibly with the addition of small trephines making the limit of what can be removed transnasal somewhat unclear.

The endoscopic approach begins with thorough dissection of normal tissue around the tumor to widen the surgical field. Once adequate exposure of the tumor has been achieved, the tumor can be addressed. If there is a small pedicle to normal bone, that junction can be fractured with en bloc removal of the tumor. Care must be taken if the tumor is pedicled on the posterior table or anterior ethmoid cranial base as resultant CSF leakage can occur. Identification of the anterior ethmoid artery on pre-operative imaging and with image guidance can prevent inadvertent damage while drilling. With large tumors, the tumor must be debulked to be removed transnasally. Once the tumor is sufficiently debulked, it may be separated from adjacent structures using angled instruments and removed. Again, care must be taken to avoid CSF leak. Typically, neither frontal sinus stents nor packing is utilized (Fig. 36.3). It cannot be overstated that although simple in description large tumors can take hours of continuous drilling to remove in their entirely and a decision must be made whether the added morbidity of an open approach warrants the time savings and added visualization that may be provided. Constant awareness of the position of the skull base and orbit is required given that during the drilling process anatomy can become distorted as tumor is removed.

The choice of a transnasal endoscopic, external, or combined approach is primarily determined by the size and location of the tumor as well as the surgeon's comfort level with various endoscopic and open techniques. Table 36.1 is a grading system that was designed by Chiu et al. to reflect the variables used to describe the location



**Fig. 36.3 (a)** Intraoperative image of right frontal sinus osteoma following standard frontal sinusotomy. **(b)** The osteoma has been mobilized from the skull base. It was too large to be removed through the prior frontal sinusotomy so bone was drilled laterally and anteriorly. **(c)** Final defect showing gross total tumor resection. No CSF leak was noted

Grade 1:
Base of attachment is posterior-inferior along the frontal recess
Tumor is medial to a virtual sagittal plane through the lamina papyracea
Anterior–posterior diameter of the lesion is $<75$ % of the anterior–posterior dimension of the frontal recess
Grade II:
Base of attachment is posterior-inferior along the frontal recess
Tumor is medial to a virtual sagittal plane through the lamina papyracea
Anterior–posterior diameter of the lesion is >75 % of the anterior–posterior dimension of the frontal recess
Grade III
Base of attachment is anterior or superiorly located within the frontal sinus
And/or
Tumor extends lateral to a virtual sagittal plane through the lamina papyracea
Grade IV
Tumor fills the entire frontal sinus

Table 36.1 Frontal sinus osteoma grading system

of the osteoma within the frontal sinus that should be considered when deciding which surgical approach to use for the removal of tumor [4]. The variables include base of attachment, location of the lesion in relation to a virtual sagittal plane through the lamina papyracea, and the anterior-posterior diameter of the osteoma in relation to the anterior-posterior dimension of the frontal recess. Grade I frontal osteomas are considered particularly accessible via an endonasal approach. These lesions are pedicled on the cribriform plate or located at the posterior aspect of the frontal recess can often be removed endoscopically after removal of an underlying agger nasi cell and superior ethmoid cells along the skull base. Grade II lesions may be similarly pedicled, but occupy a larger area in the anterior posterior diameter of the frontal recess, and hence, may benefit from an extended frontal sinus procedure such as a Draf IIb or III in order to gain more working room within the frontal recess; these lesions may also benefit from a frontal sinus trephine. Working "above and below," a large lesion may be manipulated from above to fit through a narrow frontal recess. Lesions that are Grade III or IV are located in places that are more difficult to access endoscopically and are more likely to benefit from a combined approach.

In 2007, Bignami et al. on the basis of 26 osteomas stated that an endoscopic approach was not feasible in cases with intracranial extension, large orbital involvement, anteroposterior diameter of the frontal sinus smaller than 10 mm, lateral extension behind a virtual plane through the lamina papyracea, or erosion of the posterior or anterior wall of the frontal sinus [29]. We feel that these may be relative indications to consider an open approach or combined approach but not absolute, with individual situations varying. We have found the utility of a Draf IIb or III to gain wide endoscopic access to even very large lesions, negating the need for an open approach in many situations. However, in our hands, lesions of the frontal sinus that are significantly lateral to the lamina papyracea will likely require a combined approach with trephination or osteoplastic procedure (with or without endoscopic assist). Given the benign nature of these tumors, even with subtotal resection as long as drainage pathways are established, complications related to these tumors can be avoided.

Regardless of surgical approach, following discharge, the patient should return for frequent debridements until all mucosal surfaces have healed. If stenosis evolves, then office-based techniques such as balloon dilation of the frontal sinus may be performed. Once the area has stabilized, follow up intervals may be lengthened. If the surgical site is visible no further imaging is needed, however, if the surgical site cannot be assessed endoscopically, repeat CT imaging may be needed.

All of the known complications of frontal sinus surgery are possible during resection of frontal osteomas and, indeed, are heightened compared to frontal sinus surgery for inflammatory disease. Injury to the periorbita or other intraorbital structures with resultant diplopia or blindness as well as injury to the cribriform or posterior table with resultant CSF leak [8]. CSF leak caused during endoscopic resection high on the posterior table can be especially difficult to treat given its location and increased risk of stenosis of the frontal sinus outflow following repair. Endoscopic and open approaches may both lead to disruption of the sinus walls and violation of the mucosa with a subsequent reactive bony hyperplasia. This subsequent bony regrowth should be distinguished from tumor recurrence and managed appropriately to prevent stenosis [8]. In addition, post-operative bleeding, infection, and possible need for revision surgery should all be discussed with the patient as known risks of resection. Open approaches can be complicated by all of those noted above as well as wound infection, scarring, or facial sensation changes.

# **Fibrous Dysplasia**

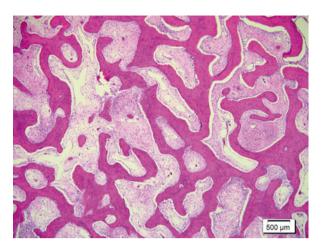
Fibrous dysplasia (FD) was first described in 1891 by von Recklinghausen, and was later given its current nomenclature by Lichtenstein in 1938 [30, 31].

• It is a typically benign, idiopathic, slowly progressive lesion where normal bone is replaced by fibrous tissue and immature woven bone [32].

In contrast to osteoma, whose borders are well defined, FD is less clearly localized both histologically and radiographically. Often presenting as painless swelling with facial deformity, 75 % of patients with FD are diagnosed before the age of 30 [10]. It is often most active during puberty, but can continue to progress into adulthood [33]. The prevalence of this disorder during puberty, and reported exacerbations during pregnancy have suggested a hormonal link [34].

The most common form of FD is a monostotic (single bone) lesion, representing 70–75 % of FD; craniofacial involvement is seen in 10–30 % of monostotic cases [35–37]. These lesions arise most commonly in the ribs followed by the femur, tibia, maxilla, mandible, calvarium, and humerus [35, 38]. In terms of cranio-facial involvement, it is most commonly found in the maxilla followed by frontal bone, mandible, parietal bone, and occipital bone [39]. Thirty percent of all FD is polyostotic (multiple bones) with 50–100 % having craniofacial involvement [35–46]. Deserving

**Fig. 36.4** Fibrous dysplasia: this section shows replacement of normal bone with fibrous stroma containing irregularly shaped bony trabeculae that consist of woven bone without osteoblastic rimming. (H&E, 40×)



special mention, McCune Albright syndrome is characterized by polyostotic lesions with the addition of skin pigmentation abnormities (Café-au-lait spots) and endocrine abnormities, representing about 4 % of FD [36]. There is an approximately 0.5 % risk of malignant transformation in monostotic FD and up to 4 % in McCune-Albright syndrome most commonly to osteosarcoma, fibrosarcoma, or chondrosarcoma [40]. Microscopically normal bone is replaced with fibrous stroma containing irregularly shaped bony trabeculae that consist of woven bone without osteoblastic rimming (Fig. 36.4).

Presentation of FD is dependent on the location of the lesion. Most commonly it presents with painless, slowly progressive swelling remaining asymptomatic until a critical structure is compressed. Orbital involvement can present with proptosis, often followed by diplopia and possible vision loss if there is optic nerve compression. While isolated frontal sinus involvement is extremely rare, these lesions can often involve the frontal sinus from adjacent extension presenting with facial asymmetry or an obstruction of the frontal sinus outflow tract [41]. Symptoms may mimic findings seen with chronic rhinosinusitis including facial pain and pressure, nasal drainage, and if the lesion is large enough, nasal airway obstruction or epiphora.

FD lesion size can vary from clinically insignificant, very small lesions to massive cranial bone involvement. On CT imaging, FD lesions are characterized by hazy borders and a fairly homogeneous "ground glass" appearance representing the disorganized spicules of bone (Fig. 36.5) [42]. With MRI, the T1 signal is intermediate and the T2 signal is hypointense [42].

FD can stabilize over time and given its low malignant potential, asymptomatic or non-disfiguring lesions can be observed. If treatment is indicated, surgery is employed. The exact approach depends on the size, extent, and location of the lesion. The goal is to alleviate compression and resultant symptoms (cranial neuropathies/diplopia/etc.) and/or restore facial symmetry whether through total resection if possible or by debulking of gross tumor. An endoscopic endonasal approach

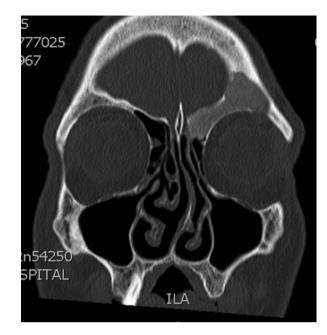


Fig. 36.5 Fibrous dysplasia of the left frontal sinus. Note the hazy borders and homogeneous "ground glass" appearance. This was completely resected using an osteoplastic flap via coronal approach

may be optimal for some lesions of the paranasal sinuses or cranial base, including some frontal sinus lesions, however, other open access options may be required. Subtotal resections may require multiple procedures over a patient's lifetime, though the disease frequently "burns out" in adulthood.

# **Ossifying Fibroma**

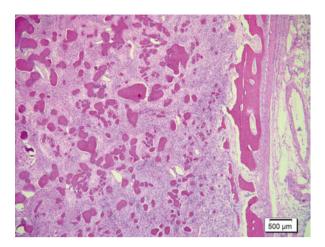
• Ossifying fibroma (OF) is most commonly encountered in the mandible (75 %) but should be included in the differential of frontal sinus fibro osseous lesions [32].

These lesions typically present in the second to fourth decades of life and have a male to female ratio of 1:5 [32]. Malignant transformation is extremely rare. They present as painless swelling in the face and can result in the same mechanical obstructive symptoms (epiphora, proptosis, blockage of frontal drainage) as the other lesions presented in this chapter due to mass effect.

• Ossifying fibroma in the paranasal sinuses and surrounding bone are believed to behave more aggressively than their mandibular counterparts with potential for significant growth and local tissue destruction [43, 44].

These lesions can mimic FD both radiographically and histopathologically [45]. The primary distinction between OF and FD in regards to histology is the presence of lamellar bone and peripheral osteoblasts in OF and the absence of both in FD

Fig. 36.6 Ossifying fibroma: this photomicrograph shows variably cellular stroma containing deposits of woven bone with lamellar bone present at the periphery on the right side of the image. While not readily identifiable in this low magnification image, one also sees cementumlike basophilic deposits and prominent osteoblastic rimming within the lesion. (H&E, 40×)



(Fig. 36.6) [46]. In regards to CT, OF presents is an expansile lesion with sharp demarcation from the adjacent bone, whereas FD tends to have more diffuse margins.

Given its locally destructive characteristics treatment is complete resection, which is curative. The exact surgical approach depends on the location and extent of the tumor but whereas with FD and osteoma observation is an option, with OF complete resection is recommended to prevent future expansion and resultant complications.

# Conclusion

Although uncommon, fibro-osseous lesions of the frontal sinus represent a spectrum of important diseases seen routinely within high volume rhinology practices. Management of these lesions in the paranasal sinuses can be challenging, especially when located in the frontal sinus. The surgical approach as well as the risks of resection versus observation should be thoughtfully balanced and post-operative care and monitoring of the surgically managed frontal sinus must be accomplished in order to prevent long term complications. As with many complex sino-nasal disorders, a team approach is key and enlisting the help of a neurosurgeon or ophthalmologist to assist in resection may be warranted.

# References

- 1. Teed RW. Primary osteomas of the frontal sinus. Arch Otolaryngol. 1941;33:255-81.
- 2. Broniatowski M. Osteomas of the frontal sinus. Ear Nose Throat J. 1984;63:267-71.
- 3. Earwalker J. Paranasal sinus osteomas: a review of 46 cases. Skelet Radiol. 1993;22:417-23.

- Chiu AG, Schipor I, Cohen NA, Kennedy DW, Palmer JN. Surgical decisions in the management of frontal sinus osteomas. Am J Rhinol. 2005;19(2):191–7.
- 5. Atallah N, Jay MM. Osteomas of the paranasal sinuses. J Laryngol Otol. 1981;95:291-304.
- 6. Larrea-Oyarbide N, Valmaseda-Castellon E, Berini-Aytes L, Gay-Escoda C. Osteomas of the craniofacial region: review of 106 cases. J Oral Pathol Med. 2008;37:38–42.
- 7. Buyuklu F, Akdogan MV, Ozer C, Cakmak O. Growth characteristics and clinical manifestations of the paranasal sinus osteomas. Otolaryngol Head Neck Surg. 2011;145:319–23.
- Eller R, Sillers M. Common fibro-osseous lesions of the paranasal sinuses. Otolaryngol Clin N Am. 2006;39:585–600.
- 9. Seiden AM, Hefny YI. Endoscopic trephination for the removal of frontal sinus osteoma. Acta Otorhinolaryngol Ital. 1995;112:607–11.
- 10. London S, Schlosser RJ, Gross CW. Endoscopic management of benign sinonasal tumors: a decade of experience. Am J Rhinol. 2002;16:221227.
- 11. Sayan NB, Ucok C, Karasu HA, Gunhan O. Peripheral osteoma of the oral and maxillofacial region: a study of 35 new cases. J Oral Maxillofac Surg. 2002;60(11):1299–301.
- 12. Summers LE, Mascott CR, Tompkins JR, Richardson DE. Frontal sinus osteoma associated with cerebral abscess formation: a case report. Surg Neurol. 2001;55:235–9.
- 13. Osma U, Yaldiz M, Tekin M, Topcu I. Giant ethmoid osteoma with orbital extension presenting with epiphora. Rhinology. 2003;41:122–4.
- Naraghi M, Kashfi A. Endonasal endoscopic resection of ethmoido-orbital osteoma compressing the optic nerve. Am J Otolaryngol. 2003;24:408–12.
- 15. Mahabir RC, Szymczak A, Sutherland GR. Intracerebral pneumatocele presenting after air travel. J Neurosurg. 2004;101:340–2.
- 16. Tsai CJ, Ho CY, Lin CZ. A huge osteoma of paranasal sinuses with intraorbital extension presenting as diplopia. J Chin Med Assoc. 2003;66:433–5.
- Tsai TL, Ho CY, Guo YC, Chen W, Lin CZ. Fibrous dysplasia of the ethmoid sinus. J Chin Med Assoc. 2003;66:131–3.
- Gil-Carcedo LM, Gil-Carcedo ES, Vallejo LA, de Campos JM, Herrero D. Frontal osteomas: standardizing therapeutic indications. J Laryngol Otol. 2011;125:1020–7.
- Moretti A, Croce A, Leone O, D'Agostino L. Osteoma of maxillary sinus: case report. Acta Otorhinolaryngol Ital. 2004;24:219–22.
- Johnson D, Tan L. Intraparenchymal tension pneumatocele complicating frontal sinus osteoma: case report. Neurosurgery. 2002;50:878–9.
- Georgalas C, Goudakos J, Fokkens WJ. Osteoma of the skull base and sinuses. Otolaryngol Clin North Am. 2011;44:875–90.
- Rappaport JM, Attia EL. Pneumocephalus in frontal sinus osteoma: a case report. J Otolaryngol. 1994;23(6):430–6.
- Goodale RL, Montgomery WW. Experiences with the osteoplastic anterior wall approach to the frontal sinus: case histories and recommendations. AMA Arch Otolaryngol. 1958;68: 271–83.
- Melroy CT, Dubin MG, Senior BA. Management of benign frontal sinus tumors with osteoplastic flap without obliteration. Oper Tech Otolaryngol–Head Neck Surg. 2004;15:16–22.
- Blitzer A, Post KD, Conley J. Craniofacial resection of ossifying fibromas and osteomas of the sinuses. Arch Otolaryngol Head Neck Surg. 1989;115:1112–5.
- Busch RF. Frontal sinus osteoma: complete removal via endoscopic sinus surgery and frontal sinus trephination. Am J Rhinol. 1992;4:139–43.
- Kennedy DW, Senior B, Lanza DC. Endoscopic approach to tumors of the anterior skull base and orbit. Op Tech Otolaryngol Head Neck Surg. 1996;7:257–63.
- Senior BA, Lanza DC. Benign lesions of the frontal sinus. Otolaryngol Clin North Am. 2001;34:253–67.
- Bignami M, Dallan I, Terranova P, Battaglia P, Miceli S, Castelnuovo P. Frontal sinus osteomas: the window of endonasal endoscopic approach. Rhinology. 2007;45(4):315–20.
- Von Recklinghausen F. Die Fibrose oder deformierende Ostitis, die Osteomalacie und die oesteoplastische carcinose in ihren gegenseitigen Beziehungen. Festschrift Rudolf Virchow zum 13. Oktober, Berlin, 1891.

- 31. Lichtenstein L. Polyostotic fibrous dysplasia. Arch Surg. 1938;36:874-98.
- Commins DJ, Tolley NS, Milford CA. Fibrous dysplasia and ossifying fibroma of the paranasal sinuses. J Laryngol Otol. 1998;112(10):964–8.
- Davies ML, MacPherson P. Fibrous dysplasia of the skull: disease activity in relation to age. Br J Radiol. 1991;164:576–9.
- Stevens-Simon C, Stewart J, Nakashima II, White M. Exacerbation of fibrous dysplasia associated with adolescent pregnancy. Adolesc Med. 1991;12:403–5.
- 35. Bibby K, McFadzean R. Fibrous dysplasia of the orbit. Br J Ophthalmol. 1994;78:266-70.
- Mohammadi-Araghi H, Haery C. Fibro-osseous lesions of craniofacial bones. Radiol Clin North Am. 1993;31:121–34.
- Stompro BE, Wolf P, Haghighi P. Fibrous dysplasia of bone. Am Fam Physician. 1989; 39:179–84.
- Schlumberger HG. Fibrous dysplasia of single bones (monostotic fibrous dysplasia). Mil Surg. 1947;99:504–27.
- Van Rompaey D, Schmelzer B, Verstraete W, Cammaert T. Fibrous dysplasia in the frontoethmoidal complex: diagnosis and surgical aspects. Acta Otorhinolaryngol Belg. 1994;48(1): 37–40.
- Ruggieri P, Sim FH, Bond JR, Unni KK. Malignancies in fibrous dysplasia. Cancer. 1994; 73:1411–24.
- Charlett SD, Mackay SG, Sacks R. Endoscopic treatment of fibrous dysplasia confined to the frontal sinus. Otolaryngol Head Neck Surg. 2007;136(4 Suppl):S59–61.
- 42. MacDonald-Jankowski DS. Fibro-osseous lesions of the face and jaws. Clin Radiol. 2004;59(1):11–25.
- Cox VS, Rimell FL, Marenttete LJ, Ness JA. Ethmoidal cemento-ossifying fibroma: the transglabellar/subcranial approach. Otolaryngol Head Neck Surg. 1996;114:335–8.
- 44. Vaidya AM, Chow JM, Goldberg K, Stankiewicz JA. Juvenile aggressive ossifying fibroma presenting as an ethmoid sinus mucocele. Otolaryngol Head Neck Surg. 1998;119:665–8.
- 45. Ferris NJ, Tien RD. Ethmoid mucocele in an infant with benign fibro-osseous lesion. Am J Neuroradiol. 1995;16:473–5.
- Post G, Kountakis SE. Endoscopic resection of large sinonasal ossifying fibroma. Am J Otolaryngol. 2005;26(1):54–6.

# **Chapter 37 Frontal Sinus Malignancies**

Joanne Rimmer and Valerie J. Lund

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_37

#### **Core Messages**

- Frontal sinus malignancies are very rare; extension from adjacent structures is more common than primary frontal sinus tumours
- Squamous cell carcinoma is the most common frontal sinus malignancy
- Tumours are often very advanced at presentation due to vague early symptoms
- High index of suspicion for patients with persistent or worsening sinus problems despite medical therapy
- Treatment is usually with surgery; postoperative radiotherapy may improve local control ± survival
- Prognosis is generally poor
- Unilateral nasal symptoms and/or proptosis warrant urgent investigation
- All frontal sinus malignancies should be reviewed by an appropriate multidisciplinary team with expert histopathology and radiology

## Introduction

Sinonasal tumours are rare, accounting for less than 1 % of all malignancies [60]. Approximately 3 % of all upper respiratory tract malignancies arise in the nose and paranasal sinuses, but only 0.3-5 % of these originate within the frontal sinus [16, 17, 34]. However, with large tumours it may be impossible to determine the site of origin due to spread into adjacent structures. The global incidence of sinonasal malignancy is less than 1 per 100,000 population per year, but there is some geographical variation [34]. European countries report an incidence of 0.6-1.2:100,000 population per year, while in Japan and some areas of Africa it is as high as 2.6 per 100,000 per year [22, 42]. Whilst they can occur at any age, they are most frequent in the sixth and seventh decades [17, 34]. These tumours are generally more common in men [11, 21].

The nose and paranasal sinuses are known to display the most diverse range of histological diagnoses of anywhere in the body [40, 41]. The World Health Organisation (WHO) classification of nasal cavity and paranasal sinus tumours is shown in Box 37.1 [4]. Whilst different series report a variable prevalence, it is accepted that epithelial malignancies are the most common (Box 37.2). Although frontal sinus malignancies are one of the least common sinus tumours, the pathology seen there is similar to that of the other sinuses. Extension from the adjacent ethmoid sinuses and nasal cavity is more common than tumours arising primarily within the frontal sinus itself [46].

#### WHO Classification of Nasal Cavity and Paranasal Sinus Tumours [4]

- 1. Malignant epithelial tumours
- 2. Neuroendocrine tumours
- 3. Malignant soft tissue tumours
- 4. Borderline and low malignant potential tumours of soft tissue
- 5. Hematolymphoid tumours
- 6. Neuroectodermal tumours
- 7. Germ cell tumours
- 8. Secondary tumours

Most Common Sinonasal Malignant Tumours Squamous cell carcinoma Adenocarcinoma Olfactory neuroblastoma Adenoid cystic carcinoma Other minor salivary gland tumours Sinonasal undifferentiated carcinoma Mucosal melanoma Sarcoma Lymphoma Plasmacytoma Metastases

The rarity and histological diversity of sinonasal tumours means that most reported series are very heterogeneous, in both the type and site of tumours. This leads to difficulties in determining prognosis and standardising management. Whilst the most recent American Joint Committee on Cancer (AJCC) staging document was updated to include staging for ethmoid as well as maxillary sinus malignancies, there is no such system for the frontal sinus, which adds to these difficulties [13].

A variety of aetiological factors have been implicated in the development of sinonasal malignancy including smoked foods, heavy metal particles and fumes, isopropyl alcohol, thorium dioxide, chromates and nickel compounds [42, 46, 49]. Occupations such as the leather and textile industries are reported to increase the incidence, and the well-established link between hardwood dust and ethmoid ade-nocarcinoma was first reported in furniture workers in 1967 [1]. There is conflicting evidence for the aetiological impact of smoking [22, 34]. Human papilloma virus (HPV) has been detected in 4 % of paranasal sinus squamous cell carcinomas (SCC), although there is currently insufficient evidence for causation [46].

## Presentation

• Tumours affecting the frontal sinus are often at an advanced stage at presentation because there are little or no symptoms initially

Symptoms are usually related to the mass effect of the tumour, and therefore may not become apparent until the air-filled sinus cavity is completely filled with tumour. As the tumour enlarges and symptoms do develop, they are often vague and/or mimic benign inflammatory conditions such as chronic rhinosinusitis. Symptoms have often been present for several months before the diagnosis is made, with an average of 8.5 months [15].

- Depending on the site and extension of the tumour, symptoms include [16, 21, 35, 43]:
  - Pain or headache (34 %)
  - Nasal obstruction (35–55 %)
  - Swelling (29–59 %)
  - Epistaxis (23-49 %)
  - Numbness (14 %)
  - Epiphora (11 %)
  - Diplopia (11 %)
  - Proptosis and visual impairment (56 %)

Primary frontal sinus tumours most commonly present with forehead swelling, pain or numbness, or diplopia, but tumours extending into the frontal sinus from the nose or ethmoid sinuses are more likely to present with nasal obstruction or epistaxis. Occasionally, frontal sinus tumours will present acutely and be misdiagnosed as acute sinusitis or a mucopyocele, or occasionally the two may occur together [8, 51, 53].

• One should retain a high index of suspicion for patients with persistent or worsening sinus symptoms despite medical treatment, and have a low threshold for imaging in such cases [60].

Cervical lymph node metastases occur in up to 21 % of patients during the course of their malignant sinonasal disease, but are present at diagnosis in 2–10 % [11, 21, 25, 43, 60]. The lymphatic drainage of the frontal sinus mucosa is to retropharyngeal nodes, and from the overlying skin to the parotid or submandibular nodes.

Distant metastases are present in up to 4 % at presentation, and develop in 4-32 % overall depending on the histology; this rate has increased as survival has improved [6, 24, 25, 43].

## Diagnosis

Imaging should be performed prior to biopsy if possible.

• A combination of computed tomography (CT) and magnetic resonance imaging (MRI) is accepted as the gold standard for evaluation of both the extent and spread of paranasal sinus malignancy [32].

Both axial and coronal CT images should be obtained with contrast, and still provide optimum information regarding bony erosion. MRI with gadolinium delineates soft tissue lesions and differentiates tumour from secretions and inflammation [32]. Tumours are usually of intermediate signal intensity compared to secretions and cerebrospinal fluid (CSF) on T2-weighted images, and usually enhance with gadolinium on T1-weighted images [46]. MRI is also helpful in assessing whether there is any dural enhancement even if frank intracranial involvement is not seen.

Either CT or MRI can be used to assess the neck for cervical lymphadenopathy, although ultrasound can also be used for this. It is also becoming more common to perform positron-emission tomography (PET)-CT prior to treatment, to check for distant metastases.

Histological diagnosis is often difficult because of the wide differential diagnoses, usually requires immunohistochemical staining and should be undertaken by a pathologist with expertise in this area. Biopsy is best performed in the operating theatre, ideally under general anaesthesia, as many sinonasal tumours are very vascular and can be associated with surrounding edema and inflammation.

## Pathology

Frontal sinus malignancies can be divided into four groups according to their site of origin: primary tumours of frontal sinus mucosa; tumours that extend into the frontal sinus from adjacent regions; tumour deposits within the frontal sinus; and tumours arising in the bony walls of the frontal sinus.

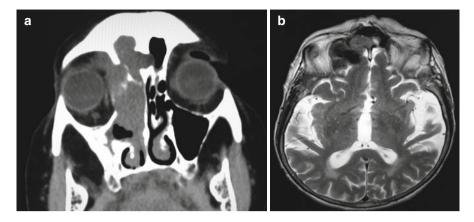
### **Primary Frontal Sinus Tumours**

Epithelial malignancies are the most common primary sinonasal tumours, and most series report SCC as the most frequent tumour within this group [16, 17, 42].

• Whilst SCC accounts for up to 60 % of paranasal sinus malignancies, it is probably nearer to 90 % of primary frontal sinus tumours [35, 42].

Actuarial disease-free survival for SCC is generally poor, reported at just 53 % at 5 and 35 % at both 10 and 15 years in one large series [24]. It is worth noting that these patients were undergoing craniofacial resection and therefore most were likely to have involvement of the frontal sinuses. Other authors report an overall survival of 60–64 % at 5 years but were generally dealing with less extensive disease [11, 22].

SCC arises within inverted papilloma (IP) in less than 5 % of cases (Fig. 37.1), but this malignant transformation is much more likely in IP of the frontal sinus compared to other sites [29, 40]. IP involves the frontal sinuses in 11-16 % of cases, with higher "recurrence" rates probably due to inadequate removal [19]. If IP appears to arise bilaterally, one should be highly suspicious of a SCC on one (or both) sides. In a personal series of 123 IP, two patients had IP on one side and SCC on the other, in one case involving the frontal sinus.



**Fig. 37.1** (a) (coronal CT) and (b) (axial T2-weighted MRI): *Right* sinonasal SCC arising within a pre-existing IP with extension into the frontal sinus

Verrucous carcinoma, thought to be a distinct variant of SCC, is a rarely metastasizing, locally invasive neoplasm, which has been reported to arise within the frontal sinus [47].

 Adenocarcinoma accounts for 10–20 % of all sinonasal malignancies, but is very rarely reported to arise in the frontal sinus [39].

It is most common in the nose and ethmoid sinus, where it is associated with hardwood dust [45]. Some series do report a higher prevalence of adenocarcinoma than SCC, and this perhaps reflects occupational exposure [5, 24]. Adenocarcinoma can be divided into salivary (5–10 %) and non-salivary types, which are then further subdivided in the WHO classification system [4, 31]. Adenocarcinoma perhaps has a better prognosis than SCC, with one large series reporting a 5-year disease specific survival of 78 % [11]. Another large series of sinonasal adenocarcinoma resected endoscopically reported an 83 % overall survival and 72 % disease-specific survival at 5 years [37]. However, other series have not found such a difference, with an overall 5-year overall survival of 58 % for adenocarcinoma, worse with increasing age and grade of tumour [45]. These tumours will sometimes spread anteriorly into the glabella and thence into the frontal bone as well as spreading directly from the middle meatus and ethmoids (Fig. 37.2).

Sinonasal melanoma accounts for less than 1 % of all malignant melanoma and approximately 4 % of all sinonasal malignancies [38]. It is much more common in the nasal cavity than the sinuses, but has been reported arising within the frontal sinus [26]. Sinonasal melanoma has a universally poor prognosis, with survival rates of less than 25 % at 5 years [38]. This is predominantly due to local recurrence but cervical metastases also occur in up to 33 % of patients [26]. Interestingly, the largest reported series of 115 cases of sinonasal melanoma treated at a single institution found that those treated with endoscopic resection had a better prognosis irrespective of initial extent [38]. Radiotherapy did not show a significant survival benefit, although there was a trend towards it.

## **Tumours Extending into the Frontal Sinus**

The floor of the frontal sinus, adjacent to the orbit and the common wall with the ethmoid sinus, is the thinnest and most easily eroded by tumour.

• Most malignant tumours involving the frontal sinus do so by extension from the adjacent ethmoid sinus or nasal cavity (Fig. 37.3).

SCC and adenocarcinoma are often seen in this way, sinonasal melanoma less so as it tends to arise within the nasal cavity in nearly 80 % of cases [38].

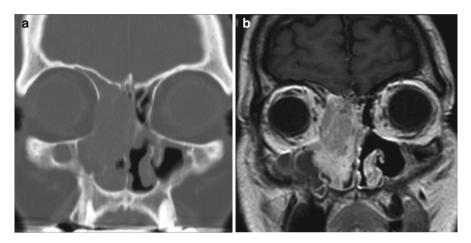
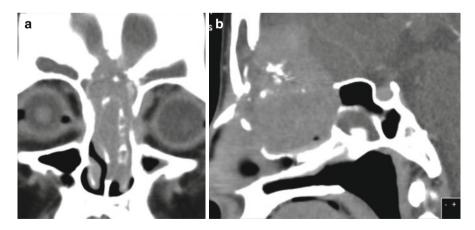


Fig. 37.2 (a) (coronal CT) and (b) (coronal T1-weighted MRI with gadolinium): *Right* sinonasal adenocarcinoma with extension into frontal sinus



**Fig. 37.3** (a) (coronal CT) and (b) (sagittal CT): Sinonasal undifferentiated carcinoma involving the nasal cavities, ethmoid and frontal sinuses bilaterally with erosion of the intersinus septum and posterior wall of the frontal sinus

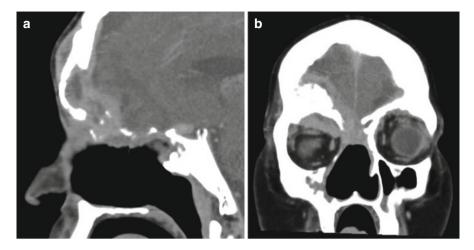


Fig. 37.4 (a) (sagittal CT) and (b) (coronal CT): Recurrent olfactory neuroblastoma involving frontal sinus and dura 10 years after craniofacial resection

Sinonasal adenoid cystic carcinoma (ACC) accounts for 10–25 % of all ACC in the head and neck, although it has not been reported arising within the frontal sinus [43, 52]. The overall survival of sinonasal ACC may be as high as 86 % at 5 years, although disease-free survival is generally lower, approximately 50 % at 5 years [35]. Distant metastases and perineural spread can occur much later; disease-free survival falls to 31 % after 10 years and will continue to drop thereafter [24, 52]. Life-time follow-up is therefore required for these tumours.

Olfactory neuroblastoma is a rare malignant tumour of neuroectodermal origin that accounts for approximately 5 % of nasal malignant tumours [36]. It commonly arises within the olfactory niche, and from there it may extend into the frontal sinus (Fig. 37.4). Survival rates are generally better than for SCC and adenocarcinoma, with 5-year disease-free survival rates of up to 77 % [36]. However, recurrence may occur after many years and again lifelong follow-up is advised.

Sinonasal undifferentiated carcinoma is rare, accounting for approximately 1-5 % of sinonasal malignancies [20, 43]. It has not been reported as arising primarily within the frontal sinus, but again may extend into it from the adjacent ethmoids.

Other minor salivary gland tumours, such as mucoepidermoid carcinoma, account for 4 % of sinonasal tumours [17]. Such tumours may be low or high grade, with better long-term outcomes for low-grade lesions which probably do not require postoperative radiotherapy [22].

### **Tumour Deposits Within the Frontal Sinus**

Lymphomas of the paranasal sinuses may be under-reported as they have a different International Classification of Disease (ICD) coding [42]. Sinonasal lymphoma accounts for only 0.17 % of all lymphoma, and is mainly seen in the maxillary and

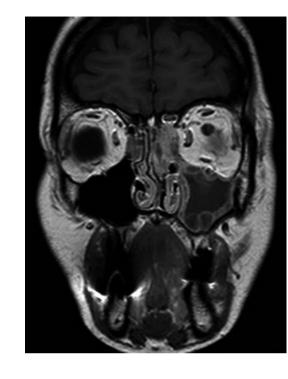


Fig. 37.5 Coronal T1-weighted MRI with gadolinium showing *left* sinonasal lymphoma with extension into frontal sinus

ethmoid sinuses [23]. Non-Hodgkin's lymphoma accounts for 9–29 % of sinus malignancies (Fig. 37.5) [12, 59].

• Primary frontal sinus lymphoma has been reported, and together with plasmacytoma accounts for up to 5 % of frontal sinus malignancies [14, 28, 44].

Overall 5-year survival for all nasal cavity and paranasal sinus lymphoma is 52 %, but this is better in younger patients, those without "B" symptoms such as fever, night sweats and weight loss, and those at an early stage [61].

Deposits of leukemia, known as "chloroma", can also occur within the frontal sinus. We have seen two cases present in this way with unsuspected haematological disease and previous histology from the frontal sinus reported as chronic rhinosinusitis. This emphasises again the need for a low threshold of suspicion and expert histopathology.

Plasmacytoma is a rare plasma cell neoplasm, 80 % of which occur in the upper respiratory tract [62]. Metastatic spread to soft tissue and/or bone occurs in approximately 50 % of cases, and 10-year survival is 50 %.

Infraclavicular malignancies, most commonly renal cell carcinoma, are known to metastasize to the head and neck [41]. Breast, lung, gastrointestinal tract and prostate metastases are also seen [55]. Renal cell metastases to the nose and sinuses are very vascular and present as epistaxis in 70 % of cases. In a series of over 1,700 sinonasal neoplasms, we have seen ten cases of metastases to the sinuses, one of which affected the frontal sinus and arose from the prostate. Metastatic deposits within the sinuses may be treated with debulking surgery and/or radiotherapy as appropriate, for palliation of symptoms.

### **Tumours Arising Within the Bone of the Frontal Sinus**

Head and neck sarcomas account for 4-10 % of all sarcomas, but 5-20 % of sinonasal tumours [17, 22, 46]. Osteosarcoma is rare, just 0.5–1 % of sinonasal tumours, but osteogenic sarcoma arising in the frontal sinus has been described [18]. Rhabdomyosarcoma has also been described, but again only accounts for 1 % of all sinonasal tumours [12, 22]. Leiomyosarcoma and fibrosarcoma have also been rarely reported within the sinuses [22, 57].

Up to 20 % of chondrosarcomas arise within the head and neck, but rarely from the frontal sinus [54]. They more commonly arise within the maxillary sinus, septum or skull base as their origin is from cartilage, nests of which rarely if ever occur in the frontal bone [22]. They accounted for 9 % of the sinonasal tumours in a large series of 209 cases [35]. Chondrosarcomas encompass a spectrum, from well-differentiated tumours with an almost benign course through to aggressive high-grade malignancy, with a 5-year survival of 33–79 % depending on the grade [24, 54]. Local disease is the most common cause of death, in 88 % of cases. A very rare variant, mesenchymal chondrosarcoma can affect the frontal region as part of its rapid and inexorable spread. It generally affects younger people.

Meningiomas very rarely arise in the nose and sinuses, but primary extracranial meningiomas have been reported there [50]. This is thought to be due to arachnoid cells carried with nerves or vessels during embryological development. Meningiomas more commonly arise intracranially, but may extend into the frontal sinus [48].

## Treatment

• Multimodality treatment with surgery and radiotherapy leads to significantly better survival than radiotherapy alone for most sinus malignancies [2, 25].

The timing of adjuvant radiotherapy varies, but most centres tend to give it postoperatively [3, 11]. A systematic review of the literature found no improvement in outcomes with the use of hyperfractionated or neutron beam radiotherapy [11]. For adenocarcinoma, radiotherapy may improve local control but there is no convincing evidence that it affects overall survival [39, 45]. Proton beam radiotherapy has shown favourable outcomes for sarcomas involving the skull base compared with standard radiotherapy in some small series, and fast neutron radiotherapy appears helpful for salivary gland malignancies within the head and neck [30]. The role of chemotherapy remains uncertain, although it shows promise as an adjunctive treatment [11, 33]. Lymphomas are the exception to the above, and are treated with appropriate chemoradiotherapy by haemato-oncologists.

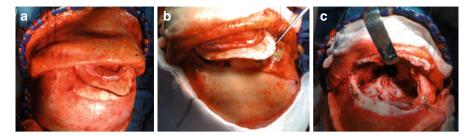
There are few regional lymph node relapses, particularly without failure at the primary site, and overall it is felt that elective treatment of a clinically and radiologically negative neck is not warranted [21].

## **Surgical Approaches**

Endoscopic approaches to the frontal sinus are now widely used for inflammatory disease and benign tumours. However, for malignant tumours endoscopic surgery is often not possible if oncologic principles are to be followed and if the surgeon is not trained in endoscopic skull base resection techniques. As a rule of thumb, if a tumour crosses the mid-pupillary line to involve the lateral recess of the frontal sinus then an endoscopic approach is contraindicated [40, 58]. The same is true for tumours involving the posterior wall, which is also very difficult to reach with endoscopic instruments. If a tumour is very medial, or just extends into the frontal sinus from the ethmoids, then it may be possible to remove it endoscopically, with or without the need for an endoscopic modified Lothrop procedure for access. However, in the majority of cases, frontal sinus malignancy requires an external approach.

Small tumours or those confined to the frontal sinus with no bony erosion may be removed via a Lynch-Howarth incision; a combined external-endoscopic approach may also be helpful in such cases [7]. An incision is made just below the medial eyebrow and extended inferomedially onto the lateral wall of the nose as required. A "v" can be inserted here to prevent epicanthal notching. The periosteum is elevated over the floor of the frontal sinus, which can then be entered using a cutting burr. The supraorbital neurovascular bundle is at risk with this approach and should be preserved if possible. The soft tissue in the region of the trochlea should be approximated with a non-absorbable monofilament suture at the end of the procedure to try and minimise diplopia. This incision can be extended across to the other side as a spectacle incision if required.

Larger tumours, those crossing the intersinus septum or involving the bony walls of the frontal sinus require wider access, which is best obtained with a formal craniofacial resection (CFR) [9]. Either an extended lateral rhinotomy or coronal scalp incision can be used, extending from the postauricular region on one side across the vertex of the scalp to the contralateral postauricular region, behind the hairline. The coronal incision may be slightly wavy to reduce scar visibility, and scalp clips can be placed along the wound edges for haemostasis. The flap is then elevated in the loose areolar tissue plane beneath the galea aponeurotica as far forward as the supraorbital rims. In this plane the superficial temporal artery and temporal branches of the facial nerve will be avoided. If a pericranial flap is required, for example to repair a defect in the posterior wall of the frontal sinus or adjacent dura, then particular care should be taken to preserve the supraorbital and supratrochlear vessels as they provide the blood supply to this flap. The frontal sinus can now be entered unilaterally or bilaterally via an anterior osteotomy, assuming the bone is not involved by tumour. To do this, the pericranium should be incised 2 cm around the line of the planned bony cuts, except inferiorly where it should be left intact to preserve blood supply to the bone. The pericranium is then elevated and the bony cuts made with a cutting burr (Fig. 37.6a), taking care to bevel the edges inwards to allow the bone flap to sit properly once plated or wired at the end of the procedure (Fig. 37.6b). If the anterior wall of the frontal sinus is involved by tumour then it



**Fig. 37.6** (a) Anterior osteotomies in frontal bone after elevation of overlying pericranium. (b) Frontal bone being replaced prior to plating; note the bevelled edges. (c) Wide exposure of frontal sinus bilaterally

must be resected, and reconstruction will be required using split calvarial bone grafts, titanium mesh or plates, or local or free tissue transfer [35, 46]. If the posterior wall of the frontal sinus is involved then a craniotomy may be required for complete extirpation of tumour, again with appropriate reconstruction. Formal CFR was popularized in the 1980s, due to the wide access (Fig. 37.6c), low morbidity and excellent cosmesis it affords, and became the gold standard for resection of malignant sinonasal tumours [5, 9].

Management of the orbit depends on the degree of orbital involvement and the preoperative function of the eye. Most studies have shown that orbital involvement is associated with worse prognosis [35]. However, there has been a growing trend towards preservation of the eye if only the periorbita are involved. The orbital periosteum can be removed without exenteration in such cases, thus preserving a functional eye, though inevitably patients with periosteal involvement have a somewhat poorer prognosis than those without [10, 24]. A split-thickness skin graft or fascia can be used for reconstruction of the medial orbital wall. If there is gross involvement of orbital contents, or if the eye is non-functioning, then orbital clearance should be undertaken.

## Complications

The complications of surgery depend on the approach used, although all include infection and bleeding. CFR complications specifically include CSF leak with the attendant risk of meningitis, as well as postoperative seizures [5, 24]. Diplopia is a potential complication for any surgery around the bony orbit, but it is unusual and rarely requires subsequent surgery [24].

Radiotherapy complications include: osteoradionecrosis, particularly of the frontal shield osteotomy if created; hypopituitarism; radiation-induced cataracts; blindness; and radiation fibrosis of the frontal lobes which may cause seizures [24, 27]. These risks are reduced by giving postoperative radiotherapy rather than radiotherapy alone, as a lower dose may be given, but should not be underestimated.

### Outcomes

It is difficult to report outcomes for such rare and histologically diverse tumours, but overall survival is poor as a result of the advanced stage at presentation in most cases. The prognosis for frontal sinus malignancies is actually better than that for other sinuses of the same stage, but most frontal sinus tumours present at a more advanced stage, often with intracranial extension [46]. The complex anatomy of the frontal sinus and its adjacent structures also makes adequate surgical resection difficult in many cases.

Death is normally due to a failure of local control [56]. Overall survival at 5 years is variably reported at 35–61 %, falling to 48 % after 10 years [3, 5, 25]. A large series of 308 sinonasal tumours treated with CFR with or without radiotherapy reported a disease-free 5-year survival of 59 % for malignant disease overall, dropping to 33 % at 15 years, but this varied considerably for the different tumour types [24]. Two factors were found to significantly affect outcome and survival, namely orbital involvement, and extension to dura and/or brain. Advanced tumour stage and the presence of regional and/or distant metastases have also been shown to be independent prognostic indicators [43].

Disease-specific survival varies between series, from 45-69 % at 5 years [11, 25]. This falls to 35-59 % at 10 years [3, 43]. There is some variation between histological subtypes, as discussed above.

Post-treatment surveillance with contrast-enhanced MRI should include imaging of the neck for tumours such as olfactory neuroblastoma with a propensity for lymphatic spread. Local or regional recurrence may be amenable to salvage treatment, and should be identified as early as possible. Although there is no widely accepted protocol, it is generally advised to scan patients every 4 months for the first 2 years, every 6 months for a further 3 years, then every 9–12 months thereafter, depending on the type of malignancy [40].

## Conclusion

Frontal sinus malignancy is rare, and tends to present at an advanced stage. Depending on the histological type, surgical resection plus postoperative radiotherapy offers the best chance of survival. Treatment failure is usually due to local recurrence but regional and distant metastases may also occur.

## References

- 1. Acheson ED, Hadfield EH, Macbeth RG. Carcinoma of the nasal cavity and accessory sinuses in woodworkers. Lancet. 1967;1:311–2.
- Alvarez I, Suarez C, Rodrigo JP, Nunez F, Caminero MJ. Prognostic factors in paranasal sinus cancer. Am J Otolaryngol. 1995;16:109–14.

- Arnold A, Ziglinas P, Ochs K, Alter N, Geretschlager A, Ladrach K, et al. Therapy options and long-term results of sinonasal malignancies. Oral Oncol. 2012;48:1031–7.
- Barnes L, Eveson JW, Reichart P, Sidransky D. Pathology and genetics of head and neck tumours. Lyon: IARC Press; 2005.
- Bridger GP, Kwok B, Baldwin M, Williams JR, Smee RI. Craniofacial resection for paranasal sinus cancers. Head Neck. 2000;22:772–80.
- Cantu G, Bimbi G, Miceli R, Mariani L, Colombo S, Riccio S, et al. Lymph node metastases in malignant tumors of the paranasal sinuses. Arch Otolaryngol Head Neck Surg. 2008;134: 170–7.
- Catalano PJ, Hecht CS, Biller HF, Lawson W, Post KD, Sachdev V, et al. Craniofacial resection. An analysis of 73 cases. Arch Otolaryngol Head Neck Surg. 1994;120:1203–8.
- Chaturvedi VN, Chauhan AN, Gode D, Moghe KV. Primary carcinoma of the frontal sinus. Ear Nose Throat J. 1978;57:47–52.
- 9. Cheesman AD, Lund VJ, Howard DJ. Craniofacial resection for tumors of the nasal cavity and paranasal sinuses. Head Neck Surg. 1986;8:429–35.
- Curran AJ, Gullane PJ, Waldron J, Irish J, Brown D, O'Sullivan B, et al. Surgical salvage after failed radiation for paranasal sinus malignancy. Laryngoscope. 1998;108:1618–22.
- 11. Dulguerov P, Jacobsen MS, Allal AS, Lehmann W, Calcaterra T. Nasal and paranasal sinus carcinoma: are we making progress? Cancer. 2001;92:3012–29.
- Duncavage JA, Campbell BH, Hanson GA, Kun LE, Hansen RM, Toohill RJ, et al. Diagnosis of malignant lymphomas of the nasal cavity, paranasal sinuses and nasopharynx. Laryngoscope. 1983;93:1276–80.
- 13. Edge S, Byrd D, Compton C, Fritz A. AJC cancer staging manual. New York: Springer; 2011.
- 14. El-Hakim H, Ahsan F, Wills LC. Primary non-Hodgkin's lymphoma of the frontal sinus: how we diagnosed it. Ear Nose Throat J. 2000;79:738–43.
- Fasunla AJ, Lasisi AO. Sinonasal malignancies: a 10-year review in a tertiary health institution. J Natl Med Assoc. 2007;99:1407–10.
- Gerlinger I, Göbel G, Tóth E, Szanyi I, Weninger C. Primary carcinoma of the frontal sinus: a case report and a review of the literature. Eur Arch Otorhinolaryngol. 2008;254: 593–7.
- 17. Gil Z, Carlson DL, Gupta A, Lee N, Hoppe B, Shah JP, et al. Patterns and incidence of neural invasion in patients with cancers of the paranasal sinuses. Arch Otolaryngol Head Neck Surg. 2009;135:173–9.
- Gupta D, Vishwakarma SK. Osteogenic sarcoma of the frontal sinus. Ann Otol Rhinol Laryngol. 1990;99:489–90.
- Han JK, Smith TL, Loehrl T, Toohill RJ, Smith MM. An evolution in the management of sinonasal inverting papilloma. Laryngoscope. 2001;111:1395–400.
- 20. Haraguchi H, Ebihara S, Saikawa M, Mashima K, Haneda T, Hirano K. Malignant tumors of the nasal cavity: review of a 60-case series. Jpn J Clin Oncol. 1995;25:188–94.
- Harbo G, Grau C, Bundgaard T, Overgaard M, Elbrond O, Sogaard H, et al. Cancer of the nasal cavity and paranasal sinuses. Acta Oncol. 1997;36:45–50.
- Harvey RJ, Winder M, Parmar P, Lund V. Endoscopic skull base surgery for sinonasal malignancy. Otolaryngol Clin N Am. 2011;44:1081–140.
- Hatta C, Ogasawara H, Okita J, Kubota A, Ishida M, Sakagami M. Non-Hodgkin's malignant lymphoma of the sinonasal tract – treatment outcome for 53 patients according to REAL classification. Auris Nasus Larynx. 2001;28:55–60.
- Howard DJ, Lund VJ, Wei WI. Craniofacial resection for sinonasal neoplasia a twenty-five year experience. Head Neck. 2006;28:867–73.
- Jakobsen MH, Larsen SK, Kirkegaard J, Hansen HS. Cancer of the nasal cavity and paranasal sinuses. Acta Oncol. 1997;36:27–31.
- Jayaraj SM, Hern JD, Mochloulis G, Porter GC. Malignant melanoma arising in the frontal sinuses. J Laryngol Otol. 1997;111:376–8.
- 27. Katz TS, Mendenhall WM, Morris CG, Amdur RJ, Hinerman RW, Villaret DB. Malignant tumors of the nasal cavity and paranasal sinuses. Head Neck. 2002;24:821–9.

- Kim K, Kim MJ, Ahn S, Bae SY, Kim WS, Yoon J-H. Frontal sinus lymphoma presenting as progressive multiple cranial nerve palsy. Yonsei Med J. 2011;52:1044–7.
- 29. Kim K, Kim D, Koo Y, Kim C-H, Choi EC, Lee J-G, et al. Sinonasal carcinoma associated with inverted papilloma: a report of 16 cases. J Craniomaxillofac Surg. 2012;40:e125–9.
- Laramore GE. Role of particle radiotherapy in the management of head and neck cancer. Curr Opin Oncol. 2009;21:224–31.
- Leivo I. Update on sinonasal adenocarcinoma: classification and advances in immunophenotype and molecular genetic make-up. Head Neck Pathol. 2007;1:38–43.
- 32. Lloyd G, Lund VJ, Howard D, Savy L. Optimum imaging for sinonasal malignancy. J Laryngol Otol. 2000;114:557–62.
- LoRusso P, Tapazoglu E, Kish JA, Ensley JF, Cummings G, Kelly J, et al. Chemotherapy for paranasal sinus carcinoma. Cancer. 1988;62:1–5.
- Lund VJ. Malignancy of the nose and sinuses. Epidemiological and aetiological considerations. Rhinology. 1991;29:57–68.
- 35. Lund VJ, Howard DJ, Wei WI, Cheesman AD. Craniofacial resection for tumours of the nasal cavity and paranasal sinuses a 17-year experience. Head Neck. 1998;20:97–105.
- Lund VJ, Howard D, Wei W, Spittle M. Olfactory neuroblastoma: past, present, and future? Laryngoscope. 2003;113:502–7.
- Lund V, Howard D, Wei WI. Endoscopic resection of malignant tumors of the nose and sinuses. Am J Rhinol. 2007;21:89–94.
- Lund VJ, Chisholm EJ, Howard DJ, Wei WI. Sinonasal malignant melanoma: an analysis of 115 cases assessing outcomes of surgery, postoperative radiotherapy and endoscopic resection. Rhinology. 2012;50:203–10.
- 39. Lund VJ, Chisholm EJ, Takes RP, Suarez C, Mendenhall WM, Rinaldo A, et al. Evidence for treatment strategies in sinonasal adenocarcinoma. Head Neck. 2012;34:1168–78.
- 40. Lund VJ, Stammberger H, Nicolai P, Castelnuovo P, Beale T, Beham A, et al. European position paper on endoscopic management of tumours of the nose, paranasal sinuses and skull base. Rhinol Suppl. 2012;22:1–144.
- 41. Lund VJ, Howard D, Wei W. Tumours of the nose, sinuses and nasopharynx. Stuttgart: Thieme; 2014.
- Muir CS, Nectoux J. Descriptive epidemiology of malignant neoplasms of nose, nasal cavities, middle ear and accessory sinuses. Clin Otolaryngol. 1980;5:195–211.
- 43. Myers LL, Nussenbaum B, Bradford CR, Teknos TN, Esclamado RM, Wolf GT. Paranasal sinus malignancies: an 18-year single institution experience. Laryngoscope. 2002;112: 1964–9.
- 44. Nemet AY, Deckel Y, Kourt G. Orbital invasion of frontal sinus lymphoma. Orbit. 2006;25: 149–51.
- 45. Orvidas LJ, Lewis JE, Weaver AL, Bagniewski SM, Olsen KD. Adenocarcinoma of the nose and paranasal sinuses: a retrospective study of diagnosis, histologic characteristics, and outcomes in 24 patients. Head Neck. 2005;27:370–5.
- Osguthorpe JD, Richardson M. Frontal sinus malignancies. Otolaryngol Clin N Am. 2001;34:269–81.
- 47. Paleri V, Orvidas LJ, Wight RG, Bradley PJ. Verrucous carcinoma of the paranasal sinuses: case report and clinical update. Head Neck. 2004;26:184–9.
- Papavasiliou A, Sawyer R, Lund VJ. Effects of meningiomas on the facial skeleton. Arch Otolaryngol. 1982;108:255–7.
- 49. Pedersen E, Hogetveit AC, Andersen A. Cancer of respiratory organs among workers at a nickel refinery in Norway. Int J Cancer. 1973;12:32–41.
- Perzin KH, Pushparaj N. Nonepithelial tumors of the nasal cavity, paranasal sinuses, and nasopharynx. A clinicopathologic study. XIII: Meningiomas. Cancer. 1984;54:1860–9.
- Reddy KT, Gilhooly M, Wallace M, Ali MH. Frontal sinus carcinoma presenting as acute sinusitis. J Laryngol Otol. 1991;105:121–2.
- Rhee C-S, Won T-B, Lee CH, Min Y-G, Sung M-W, Kim K-H, et al. Adenoid cystic carcinoma of the sinonasal tract: treatment results. Laryngoscope. 2006;116:982–6.

- 53. Robinson JM. Frontal sinus cancer manifested as a frontal mucocele. Arch Otolaryngol. 1975;101:718–21.
- 54. Ruark DS, Schlehaider UK, Shah JP. Chondrosarcomas of the head and neck. World J Surg. 1992;16:1010–6.
- 55. Simo R, Sykes AJ, Hargreaves SP, Axon PR, Birzgalis AR, Slevin NJ, et al. Metastatic renal cell carcinoma to the nose and paranasal sinuses. Head Neck. 2000;22:722–7.
- Sisson GA, Toriumi DM, Atiyah RA. Paranasal sinus malignancy: a comprehensive update. Laryngoscope. 1989;99:143–50.
- 57. Spiro JD, Soo KC, Spiro RH. Nonsquamous cell malignant neoplasms of the nasal cavities and paranasal sinuses. Head Neck. 1995;17:114–8.
- Stammberger H, Anderhuber W, Walch C, Papaefthymiou G. Possibilities and limitations of endoscopic management of nasal and paranasal sinus malignancies. Acta Otorhinolaryngol Belg. 1999;53:199–205.
- 59. Svane-Knudsen V, Jorgensen KE, Hansen O, Lindgren A, Marker P. Cancer of the nasal cavity and paranasal sinuses: a series of 115 patients. Rhinology. 1998;36:12–4.
- Tufano RP, Mokadam NA, Montone KT, Weinstein GS, Chalian AA, Wolf PF, et al. Malignant tumors of the nose and paranasal sinuses: hospital of the University of Pennsylvania experience 1990–1997. Am J Rhinol. 1999;13:117–23.
- Vidal RW, Devaney K, Ferlito A, Rinaldo A, Carbone A. Sinonasal malignant lymphomas: a distinct clincopathological category. Ann Otol Rhinol Laryngol. 1999;108:411–9.
- 62. Waldron J, Mitchell DB. Unusual presentations of extramedullary plasmacytoma in the head and neck. J Laryngol Otol. 1988;102:102–4.

# Chapter 38 Extended Endonasal Approaches to the Anterior Skull Base with Emphasis on the Frontal Sinus

Eric Mason, Hachem Jammal, and Clementino A. Solares

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# Introduction

Endoscopic surgery has undergone tremendous advancement in the past years. From the management of sinus and nasal pathologies, endoscopic surgery has developed to manage a variety of diseases. Current anatomical knowledge and navigation systems have enabled surgeons to tackle an array of lesions in the paranasal sinuses and

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© Springer-Verlag Berlin Heidelberg 2016 S.E. Kountakis et al. (eds.), *The Frontal Sinus*, DOI 10.1007/978-3-662-48523-1\_38

**Electronic supplementary material** The online version of this chapter (doi:10.1007/978-3-662-48523-1\_38) contains supplementary material, which is available to authorized users.

even lesions extending beyond the boundaries of the sinuses themselves. Management of benign diseases via endoscopic routes is nowadays a commonality, whereas the role of endoscopic techniques in sinonasal malignancies is still being defined.

Currently, it is possible to endoscopically manage different lesions situated not only in the ventral skull base, but also extending laterally (infratemporal fossa and petrous apex) and even, in select cases, within the orbit. The utilization of the endoscope endonasally could prove to be the preferred method of accessing these anatomical areas. Transnasal endoscopic approaches possess an invaluable advantage over the microscope because they allow a wide angle of vision. Therefore, new approaches using the endoscope allow access to anatomic regions that were typically inaccessible or that required extensive and highly morbid approaches. Angled endoscopes provide an even greater visual advantage when working around obstructions. With the use of a coupled camera, surgeons are afforded a unique, close-up view that minimizes tissue damage and allows precise navigation. Technology related to endoscopic surgery continues to advance, expanding the limits of endonasal surgery [1].

The safety and feasibility of expanded endonasal approaches (EEA) is currently well established and documented in several publications [2–4]. Expanded endonasal approaches allow for addressing lesions of the anterior, medial and posterior cranial fossa [4–5]. The accumulating experience that has been gained in different specialized centers worldwide make EEA to the skull base recognized as important and critical tools for skull base and sinus surgeons. Traditional skull base surgery involves invasive, open surgical procedures that are often difficult to reconstruct and come with a high degree of morbidity and iatrogenic damage [6]. Addressing these issues has remained a source of increasing interest in endoscopic endonasal surgery.

Important advantages of EEA compared to classical surgical techniques and approaches are better access to deeply seated lesions, a more direct exposure of the midline, reduced trauma to brain parenchyma, less manipulation of the neurovascular structures, rapid decompression of the optical structures, and more efficient devascularization of neoplasms from their surroundings [5, 7, 8]. Additional advantages of the endoscopic approaches are the often shorter duration of the operations, a decreased hospital stay, improved quality of life for the patients, as well as the lack of external cuts significantly reducing morbidity and cosmetic damage when compared to open approaches [9].

The experience of the surgical team and the technical prerequisites is of the utmost importance to ensure optimal surgical results [10]. The surgical teams performing EEA should be capable of handling vascular complications and performing appropriate reconstruction of skull base defects [11–13]. At the present time, a clear disadvantage of endoscopic endonasal surgery is the reduced mobility of surgical tools. In addition, intraoperative bleeding presents a greater challenge to control, and endoscopic visualization is more easily compromised [14]. Lastly, reconstruction of skull base defects continues to be a challenge in endonasal surgery. These obstacles may be better overcome in the future as experience and technology becomes more sophisticated.

A number of studies have documented the efficacy of endoscopic methods proving comparable results to open surgical approaches for tumors of the paranasal sinuses, the sella, and the skull base, and have formed the basis for the acceptance of EEA as the preferred approach [2, 8, 15, 16]. However, EEA are still in the process of definition and standardization. The extensive number of reports in the literature coming from different surgical groups from around the world is a good indicator of the enthusiasm about using EEA for different skull base pathologies. This topic has not been exhausted fully and there will be new indications to come and improvements along the way.

## **Anatomical Considerations**

Important and complex bony, neuronal, and neurovascular structures are abundant within the nasal cavity and skull base, and this makes strong anatomical knowledge a critical requirement to the success of any endoscopic endonasal surgery.

The anterior cranial fossa is formed mostly by the frontal bone, which forms the ethmoid roof, with contributions from the cribriform plate of the ethmoid bone and the sphenoid bone. Following sinonasal exenteration and septectomy, an orbit-to-orbit exposure of the anterior cranial base is achieved (Figs. 38.1, 38.2, 38.3, 38.4, 38.5, 38.6, 38.7, 38.8, and 38.9). The anterior limit is formed by the frontal sinus and the posterior limit by the planum sphenoidale. The anterior ethmoid artery is a common landmark for describing the position of the ethmoid roof, frontal recess, and crista galli, as well as the connection between the posterior wall of the frontal recess and the ethmoid roof. The posterior ethmoidal artery is located just a few



Fig. 38.1 Endoscopic view during a cadaver dissection following complete sphenoethmoidectomy and posterior septectomy

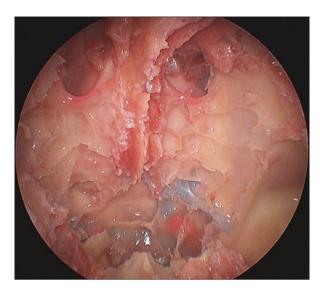


Fig. 38.2 Endoscopic view of the anterior cranial base following complete sphenoethmoidectomy and septectomy. Anteriorly the frontal sinuses are visualized and posteriorly the sphenoid is seen

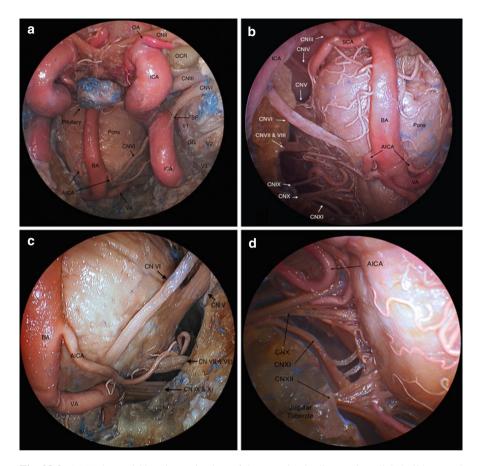
millimeters anterior to the planum sphenoidale. Following the resection of the cranial base through the ethmoid roof, the gyri recti, interhemispheric fissure, dura of the olfactory nerves and the basal surface of the frontal lobes are visualized.

As evident by the brief review of the anatomy, depending on the endonasal corridor utilized, a unique anatomical viewpoint will need to be topographically understood and navigated. This is not an exhaustive review of all the anatomy that can be encountered, thus, further reading is recommended.

### Technique

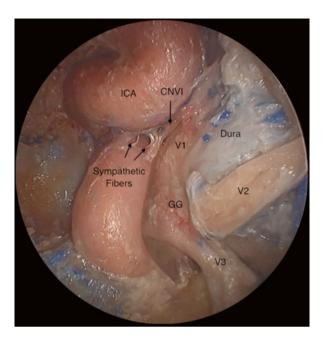
Patients are positioned supine on the operating table in Mayfield head holders, with the head in a neutral position or extended and slightly rotated to the right side. The patient position remains the same for all EEA. For more rostral or caudal lesions, 45° or 70° endoscopes may be used. Bimanual dissection is essential for microneurosurgery and is an absolute prerequisite for endoneurosurgery. To allow for bimanual dissection, a binasal approach, two-surgeon technique is a must.

Skull base lesions located in the most anterior portion of the cribriform plate or the ethmoid roof just posterior to the frontal recess do not directly involve the frontal sinus or its outflow tract, however, by virtue of their close proximity, the frontal recess must be addressed by an Endoscopic Modified Lothrop Procedure (EMLP) or Draf III and will be the main focus of this chapter. The EMLP or Draf III entails resection of the superior nasal septum, frontal sinus floor and frontal sinus septum.



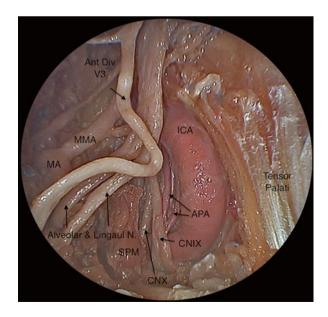
**Fig. 38.3** (a) Endonasal 0° endoscopic view of the completely dissected medial skull base and Meckel's cave. *AICA* anterior inferior carotid artery, *BA* basilar artery, *CN* cranial nerve, *GG* Gasserian ganglion, *ICA* internal carotid artery, *OA* ophthalmic artery, *OCR* optic carotid recess, *SF* sympathetic fibers, *V1* ophthalmic nerve, *V2* maxillary nerve, *V3* mandibular nerve (b) Endonasal 0° endoscopic view of the laterally dissected clivus showing cranial nerves III to XI with relevant vasculature. *AICA* anterior inferior carotid artery, *CC* endonasal 0° endoscopic view of the laterally dissected clivus showing the lower cranial nerve. *AICA* anterior inferior carotid artery, *BA* basilar artery, *CN* cranial nerve, *SA* superior cerebellar artery, *VA* vertebral artery. (c) Endonasal 0° endoscopic view of the laterally dissected clivus showing the lower cranial nerve. *AICA* anterior inferior carotid artery, *BA* basilar artery, *CN* cranial nerve, *VA* vertebral artery. (d) Endonasal 45° endoscopic view of the laterally dissected clivus showing cranial nerve XII, inserting into the hypoglossal canal. *AICA* anterior inferior carotid artery, *CN* cranial nerve

Endoneurosurgical tumor resection is done using standard microneurosurgical techniques. The two suction technique has been recognized as the safest means for debulking soft lesions [17]. For harder consistency tumors, an ultrasonic aspirator or a suction-cutting device can be used [5]. Internal debulking is continued until the capsule is reached, and then the extracapsular dissection is performed. Critical neurovascular structures are then identified and protected. The capsule is coagulated using the appropriately shaped endoscopic bipolar cautery.

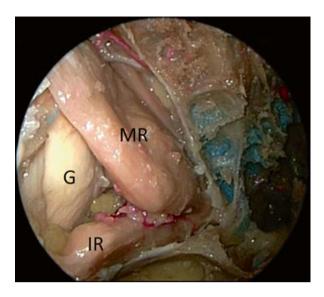


**Fig. 38.4** Endoscopic view of Meckel's cave with the lateral and posterior walls of the sphenoid sinus removed to reveal the internal carotid artery with gasserian ganglion and respective branches of the trigeminal nerve. *CNVI* abduscens nerve, *Dura* Dura mater, *GG* gasserian ganglion, *ICA* internal carotid artery. V1 – ophthalmic branch of the trigeminal nerve, V2 – the maxillary branch of the trigeminal nerve, V3 – mandibular branch of the trigeminal nerve

**Fig. 38.5** Endoscopic dissection of the parapharyngeal space with removal of the tensor palatini and the mandibular branch of the trigeminal nerve. *APA* ascending pharyngeal artery, *CNIX* glossopharyngeal nerve, *CNX* vagus nerve, *ICA* internal carotid, *IJV* internal jugular vein, *MA* maxillary artery, *MMA* middle meningeal artery



**Fig. 38.6** Endoscopic view of the medial orbit. The periorbita has been removed to reveal the underlying medial rectus (*MR*) and inferior rectus (*IR*) muscles. With dissection of periorbital fat the globe (*G*) may also be visualized



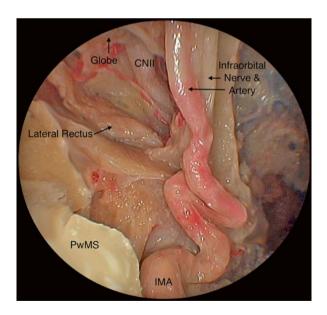


Fig. 38.7 Right transantral endoscopic view of the inferior orbital floor (removed) showing the dissection of the posterior wall of the maxillary sinus (PwMS) revealing the internal maxillary artery (IMA). The infraorbital nerve and artery were used as landmarks for the medial border of the dissection

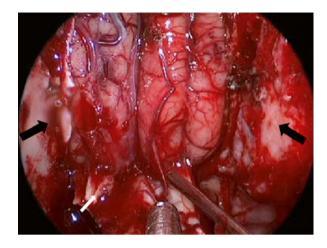
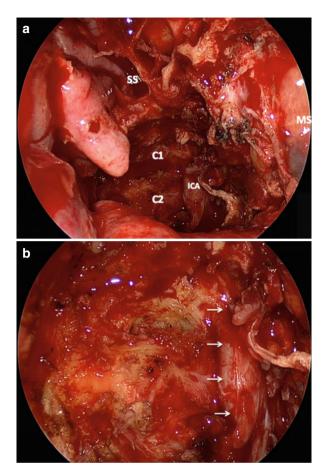


Fig. 38.8 Endoscopic view following a resection of the anterior cranial base. The *black arrows* identify the orbits and the *white arrow* points to the right olfactory bulb (transected)

# Approaches

## Transcribriform Approach

The transcribriform approach is indicated for sinonasal malignancies that extend through the anterior skull base, and olfactory groove meningiomas. The approach should be completed by Draf III nasalization of the frontal sinuses. A large cavity extending from one lamina papyracea to the other should be created and visualized. Therefore, a total anterior and posterior ethmoidectomy is performed with exposure and ligation of the anterior and posterior ethmoid arteries. The planum sphenoidale should also be defined with identification of the optic nerves, ICA, and sellar floor. The transcribriform approach is accomplished by resecting the attachment of the anterior portion of the nasal septum to the skull base. The olfactory sulcus is then further exposed by removing mucosa and remnants of air cells. These sulci extend on both sides of the crista galli, from the cribriform plate rostrally to the anterior margin of the planum caudally. It is understood, that for primarily intradural tumors the sinonasal exposure leads to the tumor while in the case of sinonasal malignancies the sinonasal malignancies this dissection is intimately involved with the tumor resection. The cribriform plate can then be removed bilaterally, leaving the crista galli in the midline. The exposed dura mater is further coagulated. This leaves the anterior falcine artery as the primary residual supply to anterior skull base tumors. Following coagulation, the dura mater is opened on both sides of the falx individually. The midline is kept intact intentionally to avoid bleeding from the anterior falcine artery. After debulking is done on each side, the feeding vessels arising from the anterior falcine artery are coagulated along with the falx [5]. The falx is transected at this point to provide a single intradural working cavity (Fig. 38.8). Identification of the optic nerves and the anterior communicating artery may greatly facilitate the extracapsular dissection of the tumor from surrounding vascular structures. In selected cases, a unilateral approach can be performed with the aim of preserving olfaction [18].



**Fig. 38.9** (a) Endoscopic view following tumor removal. The specimen included the Eustachian tube and prevertebral muscles. The vertebral bodies of C1 and C2, as well as the parapharyngeal internal carotid artery (ICA), which is coursing medially before it enters the carotid canal at the skull base are exposed. (*SS* sphenoid sinus, *MS* maxillary sinus). (b) Post-resection cavity of endoscopic transnasal and transoral pharyngectomy for recurrent squamous cell carcinoma of the naso-pharynx and oropharynx. Endoscopic transoral view shows the left parapharyngeal internal carotid artery (*arrows*) coursing superiorly towards the skull base

## Isolated Transfrontal Approach for Skull Base Lesions

Trauma, projectile injuries from bullets, anterior skull base and sinonasal tumors can create frontal sinus CSF leaks. Endoscopic closure of anterior skull base CSF leaks is now recognized as the treatment of choice for most CSF leaks. In those cases of frontal sinus CSF leaks after firearm injuries, the role of endoscopy is less clear. Many patients require transcranial procedures to address these intracranial injuries. Repairs can be achieved by mucosal grafting or a pedicled flap based on branches of the facial and anterior ethmoidal arteries [19]. Also, collagen matrix materials, fascia or fat can be used as inlay grafts to help seal theses defects.

Mucoceles are the most common benign lesions of the paranasal sinuses, from which the frontal sinus is the most common location. Although they usually compress the orbit, they can also have intracranial extension through the posterior table of the frontal sinus that can lead to CSF leaks and meningitis. Some patients with frontal mucoceles can be asymptomatic, despite of this, slow concentric expansion of the paranasal sinus mucoceles can result from accumulation of secretions, eventually remodeling or eroding bony structures. Endoscopic frontal sinusotomy is the preferred technique for the management of frontal sinusitis associated with posterior table erosion, since it is a functional approach and avoids morbidity of more destructive alternatives [20].

Isolated frontal sinus tumors are rare. Most commonly, inverted papillomas and osteomas. Asymptomatic osteomas should be observed. The exposure of lesions in the frontal sinus and near its outflow tract is achieved with graded dissection of the frontal sinus and the ethmoid labyrinth as necessary. Adequate exposure is key to a successful tumor resection outcome.

### Discussion

The EEA allow access to skull base lesions extending from the crista galli to the foramen magnum. The nature of the disease entity and its location dictate the most appropriate approach. There are few absolute contraindications to the EEA for pathological conditions of the ventral skull base. These may include larger lesions and those expending too far laterally [8]. The success of EEA depends heavily on the expertise of the surgical team and on the correct indications for each and every approach. The close proximity of the orbit and the skull base make the EMLP a technically demanding procedure. Complications of the EMLP or Draf III include: dural injury and CSF leak, orbital injury, and injury of the nasal bones and skin, thus, knowledge of frontal sinus and ventral skull base anatomy is a must. At this point, optimum training programs are being perfected for surgeons to enhance overall success [21].

It goes without saying that getting familiar with endoscopic endonasal surgery necessitates getting familiar with skull base anatomy viewed from below. This is essential for identification of structures, especially neurovascular structures, so that accurate dissection can be performed and unnecessary complications avoided. The feasibility of different endoscopic approaches is mainly determined by the topographical relation of critical anatomical structures along the skull base and their locations with respect to the trajectory of the approach. The associated risks of these new methods are therefore less dependent on the size of the targeted lesion but more on the neurovascular structures along the path of the approach [22].

The EEA are proving to be oncologically sound procedures with the ability to ensure good resection margins as with traditional open approaches. Dural involvement and brain invasion are still challenges for both EEA and open approaches to skull base lesions. Still, it seems that the most important tumor characteristic in selecting an open approach versus EEA is the exact character and relationship of the lesion to critical neurovascular structures [5]. The endoscope grants a much wider viewpoint than a traditional microscope, and obstructions might be better and more safely circumvented with EEA [1]. However with a smaller corridor, en bloc tumor removal may be difficult or impossible depending on the lesion. Above all, the patient should be healthy and have no contraindication for a procedure under anesthesia.

The patient also should have the appropriate indication. In the literature, extensive reports have recognized EEA as being the optimum surgical option for a variety of non-neoplastic and neoplastic conditions of the ventral skull base [23, 24]. The most common non-neoplastic diagnosis that we treat is a CSF leak (traumatic, iatrogenic, and spontaneous). The most common benign neoplasms of the anterior cranial base treated are meningiomas. In terms of malignant lesions, esthesioneuro-blastomas and sinonasal cancers with cranial base involvement are the most common neoplasms treated with the EEA [20].

Simply put, EEA is ideal for lesions where the critical neurovascular structures are around and above the capsule of the tumor. If a major vessel is to be encountered, then open approaches are more favored. The key predetermined factors that define the goals of surgery are the patient's age, premorbid conditions, symptoms, and natural history of the lesions. What might outweigh other factors in determining the applicability of EEA for skull base access is the experience of the surgical team. Therefore, the most desirable situation would be in a setting where a dedicated skull base team is available and collaborating extensively, including surgeons from different specialties, (neurosurgery, otolaryngology, and endovascular surgery). These teams should be equipped with the best instrumentation, and with strong auxiliary staffing and post-operative care facilities.

## Conclusion

The use of the endonasal endoscope for treating skull base and intracranial lesions is a relatively new, yet exciting development. The exact parameters are still unclear, and more research into this area will help solidify what is possible with this type of surgery. Advancement in technology is an integral part of medicine. As tools, imaging, safety, and surgical expertise continually evolve, new possibilities can be opened. It is safe to say that that endoscopic endonasal surgery is becoming an accepted and permanent component of skull base surgery.

## References

- 1. Lee S, Senior B. Endoscopic skull base surgery. Clin Exp Otolaryngol. 2008;1(2):53-62.
- Laufer I, Anand VK, Schwartz TH. Endoscopic, endonasal extended transsphenoidal, transplanum transtuberculum approach for resection of suprasellar lesions. J Neurosurg. 2007; 106:400–6.

- 3. Carrabba G, Dehdashti AR, Gentili F. Surgery for clival lesions: open resection versus the expanded endoscopic endonasal approach. Neurosurg Focus. 2008;25:E7.
- de Divitiis E, Cavallo LM, Cappabianca P, Esposito F. Extended endoscopic endonasal transsphenoidal approach for the removal of suprasellar tumors: part 2. Neurosurgery. 2007; 60:46–58.
- 5. Kassam AB, Snyderman CH, Mintz AH, Gardner P, Carrau RL. Expanded endonasal approach: the rostrocaudal axis. Part I. Crista galli to the sella turcica. Neurosurg Focus. 2005;19:E3.
- DiLuna M, Bulsara K. Surgery for petroclival meningiomas: a comprehensive review of outcomes in the skull base surgery era. Skull Base. 2010;20(5):337–42.
- Kassam AB, Gardner P, Snyderman CH, Mintz AH, Carrau RL. Expanded endonasal approach: fully endoscopic, completely transnasal approach to the middle third of the clivus, petrous bone, middle cranial fossa, and infratemporal fossa. Neurosurg Focus. 2005;19:E6.
- Dehdashti AR, Ganna A, Witterick I, Gentili F. Expanded endoscopic endonasal approach for anterior cranial base and suprasellar lesions: indications and limitations. Neurosurgery. 2009; 64:677–87.
- Zada G, Kelly DF, Cohan P, Wang C, Swerdloff R. Endonasal transsphenoidal approach for pituitary adenomas and other sellar lesions: an assessment of efficacy, safety, and patient impressions. J Neurosurg. 2003;98:350–8.
- Kassam AB, Snyderman CH, Carrau RL, Gardner P, Mintz AH. Endoneurosurgical hemostasis techniques: lessons learned from 400 cases. Neurosurg Focus. 2005;19:E7.
- 11. Kassam AB, Carrau RL, Snyderman CH, Gardner P, Mintz AH. Evolution of reconstructive techniques following endoscopic expanded endonasal approaches. Neurosurg Focus. 2005; 19:E8.
- Kassam AB, Thomas A, Carrau RL, Snyderman CH, Vescan A, Prevedello D, Mintz A, Gardner P. Endoscopic reconstruction of the cranial base using a pedicled nasoseptal flap. Neurosurgery. 2008;63:ONS44–52.
- Zanation AM, Snyderman CH, Carrau RL, Kassam AB, Gardner PA, Prevedello DM. Minimally invasive endoscopic pericranial flap: a new method for endonasal skull base reconstruction. Laryngoscope. 2009;119:13–8.
- 14. Thongrong C, Kasemsiri P, Carrau R, Bergese S. Control of bleeding in endoscopic skull base surgery: current concepts to improve hemostasis. ISRN Surg. 2013;2013:1–11.
- 15. Fraser JF, Nyquist GG, Moore N, Anand VK, Schwartz TH. Endoscopic endonasal transclival resection of chordomas: operative technique, clinical outcome, and review of the literature. J Neurosurg. 2010;112:1061–9.
- 16. O'Malley Jr BW, Grady MS, Gabel BC, Cohen MA, Heuer GG, Pisapia J, Bohman LE, Leibowitz JM. Comparison of endoscopicand microscopic removal of pituitary adenomas: single-surgeon experience and the learning curve. Neurosurg Focus. 2008;25:E10.
- Kassam A, Thomas A, Snyderman C, Carrau R, Gardner P, Mintz A, Kanaan H, Horowitz M, Pollack I. Fully endoscopic expanded endonasal approach treating skull base lesions in pediatric patients. J Neurosurg Pediatr. 2007;106(2):75–86.
- Ong YK, Solares CA, Carrau RL, Prevedello DM, Kassam AB. Preservation of olfactory function following endoscopic resection of select malignancies of the nasal vault. Surg Tech Dev. 2012;2:e5. doi:10.4081/std.2012.e5.
- Gustavo H, Rivera-Serrano CM, Bassagaisteguy LH, Carrau RL, Fernandez-Miranda J, Prevedello DM, Kassam AB. Anterior pedicle lateral nasal wall flap: a novel technique for the reconstruction of anterior skull base defects. Laryngoscope. 2011;121:1606–10.
- 20. Solares CA, Citardi MJ, Budev M, Batra PS. Management of frontal sinus mucoceles with posterior table erosion in the pretransplant cystic fibrosis population. Am J Otolaryngol. 2007;28(2):110–4.
- Snyderman C, Kassam A, Carrau R, Mintz A, Gardner P, Prevedello D. Acquisition of surgical skills for endonasal skull base surgery: a training program. Laryngoscope. 2007;117(4): 699–705.

- Wagenmann M, Schipper J. The transnasal approach to the skull base. From sinus surgery to skullbase surgery. Laryngorhinootologie. 2011;90Suppl1:S1–15.doi:10.1055/s-0030-1270443. Epub 2011 Apr 26. Review. German.
- Bhatki A, Carrau R, Snyderman C, Prevedello D, Gardner P, Kassam A. Endonasal surgery of the ventral skull base—endoscopic transcranial surgery. Oral Maxillofac Surg Clin N Am. 2010;22(1):157–68.
- 24. Snyderman C, Pant H, Carrau R, Prevedello D, Gardner P, Kassam A. What are the limits of endoscopic sinus surgery? The expanded endonasal approach to the skull base. Keio J Med. 2009;58(3):152–60.

# Chapter 39 Open Approaches to the Frontal Sinus

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### **Core Messages**

- Open approaches to the frontal sinus are an important component of the sinus surgeon's armamentarium
- Open and endoscopic approaches to the frontal sinus can complement one another
- Sinus surgeons should choose an approach to the frontal sinus which can safely achieve the surgical objectives with the least morbidity

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## Introduction

Historically, frontal sinus disease has been treated surgically through external approaches. Since the introduction of endoscopic sinus surgery in the 1980s, its growth and evolution has increased our ability to treat and manage a wide variety of paranasal sinus disease endoscopically [1-3]. The limits of the endoscopic approach continue to be expanded and challenged with advances in technique and instrumentation. There is ongoing evolution of the role for open surgical approaches in the endoscopic era.

Despite the recent advances in endoscopic sinus surgery, frontal sinus disease remains challenging to treat given the complex anatomy of the frontal recess and its anatomic relationship to vital structures, such as the orbit and anterior skull base. There remain certain situations in which open frontal sinus surgery provides unparalleled views and access to the sinus which is not possible solely via the endoscope. Combined open and endoscopic frontal sinus procedures are also becoming more prevalent.

Current indications for open frontal sinus surgery include:

- · Chronic frontal sinusitis which has failed endoscopic procedures
- · Acute frontal sinusitis with impending complications
- Benign and malignant frontal sinus lesions that are inaccessible endoscopically (generally when attached to the lateral or anterior frontal sinus wall)
- Lateral frontal sinus mucoceles
- · Osteoneogenesis of the frontal recess
- Osteomyelitis of the frontal bone
- · Frontal bone fractures with comminuted bone
- Cerebrospinal fluid (CSF) leak
- Frontal recess injury.

It is critical that the sinus surgeon understand the underlying pathophysiology, know the relevant anatomy, set realistic surgical objectives, and then accomplish those objectives in as safe a manner as possible while causing the least morbidity. Whether this is to be accomplished endoscopically or via an open approach depends on the surgeon's experience and skill, the availability of necessary technology, and the specific disease entity being treated.

### **Historical Perspectives**

The first open frontal sinus approaches date back to 1870, when simple incision and drainage of frontal pyoceles was described by Wells [4, 5]. In 1884, Ogston proposed frontal sinus anterior wall perforation via a forehead incision [6].

The removal of the anterior wall of the frontal sinus was first described by Kuhnt in 1895 [7]. Then, Riedel proposed complete obliteration of the frontal sinus by removing the floor and anterior wall, creating a significant cosmetic deformity. In 1904, Killian modified this approach by leaving a small bridge of bone across the supraorbital rim to lessen the deformity [4].

The history of osteoplastic flap dates back to 1904 when Hoffman described the frontal sinus obliteration procedure [4]. In 1956, Goodale and Montgomery popularized the osteoplastic flap technique with fat obliteration, and it soon became the gold-standard surgical treatment for frontal sinus disease [8]. The osteoplastic operation took its name from the fact that the anterior wall of the frontal sinus was opened as an inferior-based flap, fractured of off the orbital roof with intact periosteum, resulting in a vascularized bone flap. The opened cavity was then obliterated with fat, which became vascularized and prevented mucosal ingrowth.

In 1908, Knapp advocated for an extensive ethmoidectomy through the medial orbital wall and enlarging the frontonasal recess [9]. In 1914, Lothrop modified the external approach by leaving the anterior wall intact, but removing the interfrontal sinus septum and upper nasal septum, creating a large inferior drainage fistula into the nose.

In 1921, Lynch combined removal of the floor of the frontal sinus through an external approach via the medial orbital wall with dilation and stenting of the nasofrontal drainage system. This became the preferred procedure for some time, but was complicated by restenosis of the nasofrontal passage and mucocele/pyocele formation [10]. Further studies show that this procedure has a 30 % failure rate [11].

Endoscopic sinus surgery was first described in American literature in 1985 by Kennedy [12, 13]. Since that time, there have been significant advances in techniques and equipment which have increased our ability to treat paranasal sinus disease endoscopically. Frontal sinus disease remains challenging to treat, and there are certain situations in which open approaches remain necessary, either performed alone or in combination with endoscopic procedures.

### Modern Day Open Frontal Sinus Surgery

### Frontal Sinus Trephination

### **Surgical Indications**

Indications for trephination include:

- · Acute frontal sinusitis with or without orbital and intracranial complications
- Frontal sinus exploration and/or biopsy
- As an adjunct to an endoscopic approach for chronic frontal sinusitis or frontal sinus mucoceles

There are three principal situations in which the frontal sinus trephination is useful. First, a small or mini trephination may be used to simply irrigate the sinus contents in complicated cases or to ensure clearance of pus, mucus or fungal material when the surgeon does not wish to place an instrument through the frontal ostium and risk damage to the mucosa. Secondly, the frontal sinus trephination is a very useful technique for a combined open and endoscopic procedure when the drainage pathway of the frontal sinus is not easily seen endoscopically. A cannula can be placed through the trephine and the sinus is flushed with fluorescein-stained saline until the outflow tract is visualized endoscopically to allow for safe frontal sinusotomy. Alternatively, a Fogarty catheter can be inserted through the trephination, into the frontal recess, and visualized intra-nasally for localization of the frontal recess. Thirdly, the trephination can be enlarged to fit endoscopes and endoscopic instruments for visualization and to perform biopsies or resections of lesions within the frontal sinus.

In 2009, Seiberling et al. published a series of 188 frontal sinus trephinations that were performed during combined open and endoscopic procedures [14]. The author states that trephines were placed when there was difficulty finding the frontal recess, severe edema/polyps, obstructing frontal cells (type 3 or 4 frontoethmoidal cells and intersinus septum cells), and to aid in the dissection and postoperative irrigation during modified Lothrop procedure.

The advantages of the frontal sinus trephine are:

- · It provides fast and easy access to the frontal sinus
- It allows placement of a drain for continued irrigation of the sinus in the postoperative period

The main disadvantages are:

- · Associated scarring
- Risk of sinocutaneous fistula formation
- Risk of injury to the supraorbital nerve bundle and the trochlea which can lead to diplopia [15].

The complication rate of frontal sinus trephination is reported at 6 % with infection at the trephine site being the most common complication [14]. The success rate after frontal sinus trephination is reported at 86-92 % [14, 16].

### **Description of Procedure**

Surgical steps:

- · Incision planned at inferior aspect of the medial eyebrow
- · Incision site is injected with local anesthetic
- Incision carried down through all layers up to the periosteum
- Tissue incision retracted cephalad and medial to approximately 1 cm from midline
- Incision carried through periosteum
- · Periosteum elevated with a Cottle elevator
- Cutting bur is then used to create bony trephine
- · Trephine is widened with a cutting bur or rongeur to the appropriate size



Fig. 39.1 Left frontal sinus trephination

After thorough review of the CT scan, the planned incision site is injected with local anesthetic and adrenaline. The incision may be placed through the inferior aspect of the medial eyebrow to hide the scar or alternatively in a vertical frown line. The planned incision site and is carried down through all layers including the periosteum. The periosteum is elevated with a Cottle elevator. The landmark for placement of the trephine is determined by drawing an imaginary horizontal line connecting the middle of the medial end of each eyebrow, and the trephine is placed approximately 1 cm from the midline along this imaginary line [17, 18]. If the trephine is placed too laterally, it may endanger the supratrochlear neurovascular bundle or be outside of the sinus. Studies have shown that the sinus can be successfully trephined at 5 mm, 10 mm, or 15 mm from midline in the majority of patients provided that the intersinus septum is in the midline and the frontal sinus is well developed laterally [18]. It is best to enter the sinus through the lamellar bone of the floor of the frontal sinus rather than the cancellous bone of the anterior wall, which has a marrow space. Contamination of the marrow space may theoretically lead to osteomyelitis. Image guidance may also be used to identify the appropriate trephination site, especially for small frontal sinuses.

A cutting bur is then used to create a 5 mm bony trephine while taking care to not pass the drill into the sinus to avoid injuring the posterior table. The trephine is widened with a cutting bur or rongeur to the appropriate size (Fig. 39.1). The sinus may then be irrigated with saline, topical decongestant, or fluorescein-stained saline. Cultures are obtained as indicated.

Once the trephination is performed, it may be widened enough to place an endoscope and examine the sinus mucosa or to perform biopsy or resection of focal frontal sinus pathology. Trephines that are enlarged greater than 1 cm are generally reconstructed with titanium mesh. Although the standard trephine is placed in the inferomedial aspect of the frontal sinus, Zacharak et al. showed that with image guidance, the trephination site can actually be placed anywhere on the frontal sinus tailored to the location of the target lesion [19]. This modified technique is ideal for patients with focal frontal sinus pathology such as fibrous dysplasia, type 3 or 4 frontal cells, and benign frontal sinus tumors.

Postoperatively, the patient is treated with antibiotics as indicated. In cases of acute infection, two cannula are placed; a long red rubber Robinson catheter for irrigation, and a truncated catheter for egress of irrigation. The catheter is irrigated four times daily with saline and topical nasal decongestant spray. The recommended irrigation mixture is a liter of saline with 0.5 cc of 0.05 % xylometazoline or oxymetazoline. When patency of the nasofrontal drainage system is confirmed by free flow of irrigation solution into the nose, the catheters are removed.

### The Lynch Procedure

### **Surgical Indications**

The original Lynch procedure (also known as the Lynch-Howarth procedure) describes removal of the bone surrounding the frontal recess including the entire lamina papyracea and all frontal sinus mucosa. Due to frequent post-operative frontal recess stenosis from medial collapse of the orbital contents, Lynch placed a 1 cm rubber tube stent in the frontal recess for 5 days [20]. The Lynch procedure has largely been replaced by endoscopic techniques because stenosis of the frontal recess developed in approximately a third of patients who underwent the procedure [21, 22]. Surgical indications for the Lynch procedure include: [20]

• Failed prior external frontal sinus surgery

- Extensive bony destruction of the frontal sinus (especially over the dura or around the orbit)
- Frontal sinus fractures

In 1976, the Neel-Lake modification was described, which begins with an intranasal ethmoidectomy, preserves mucosa, preserves the frontal process of the maxilla, and uses a rolled silastic sheet to stent the naso-frontal duct [23]. This modification has been shown to have a 20 % long-term failure rate [20].

#### **Description of Procedure**

Surgical steps

- Curvilinear incision is made from the infero-medial aspect of the eyebrow to the upper third of the nose, between nasal dorsum and medial canthus
- Incision carried down through periosteum
- · Medial canthal tendon released, retracted laterally
- · Anterior ethmoidal artery identified and divided
- Anterior ethmoid cavity is entered via the lacrimal fossa
- · Remove anterior ethmoid cells and anterior lamina papyracea
- Resect the frontal sinus floor

A curvilinear incision is made from the infero-medial aspect of the eyebrow to the upper third of the nose approximately half way from the midline nasal dorsum to the medial canthus. The incision is carried down through skin, subcutaneous tissue, and periosteum. The medial canthal tendon is released, and the orbital contents are retracted laterally with a Sewell retractor. The anterior ethmoid artery is identified, and a medium sized hemoclip is placed on the lateral (orbital) side while the medial (nasal) side is then cauterized with a bipolar cautery and transected.

The anterior ethmoid cavity is entered via the lacrimal fossa, and the bone of the anterior lacrimal crest is removed with a Kerrison rongeur. The anterior ethmoid cells are removed along with the anterior portion of the lamina papyracea. Next, the floor of the frontal sinus is resected with the Kerrison rongeur, and the diseased mucosa is removed from the frontal sinus. Historically, a large stent was placed into the frontal recess, but it is now recognized that large stents cause pressure necrosis. Local rotational mucosal flaps or composite grafts can be placed around a loose stent. This avoids pressure necrosis and provides lateral support with reduced chance of stenosis. A commonly used stent is a rolled piece of silastic sheeting which is secured to the nasal septum, though commercially produced stents are available. They are typically removed 3–6 weeks post-operatively, but some stents can be left in place for months or years.

### The Osteoplastic Flap

### **Surgical Indications**

The frontal osteoplastic flap with or without obliteration remains an option for surgical treatment of frontal sinus pathologies that cannot be treated through a purely endoscopic approach.

Indications for osteoplastic flap:

- Previously failed endoscopic approaches to the frontal recess for chronic sinusitis [4]
- Malignant or benign lesions of the frontal sinus that cannot be reached endoscopically
- Severe frontal bone fractures requiring an open approach for reduction, fixation and dural repair
- Situations where injury to the frontal sinus drainage pathway is not amenable to an endoscopic approach

Although the indications for endoscopic sinus surgery are rapidly growing, there remain specific situations in which the osteoplastic flap is beneficial for inflammatory frontal sinus disease. In 2009, a series of 683 patients with chronic frontal sinusitis showed that 4.7 % of patients required external procedures. The most common indication was for osteoneogenesis of the frontal recess, usually secondary to failed endoscopic procedures [24]. Correa et al. stated that patients with a narrow

anterior-posterior diameter of the frontal recess, a highly compartmentalized frontal sinus, an extensive polypoid degeneration of the frontal sinus mucosa or those with highly thickened secretions are also more likely to require an osteoplastic flap procedure [25].

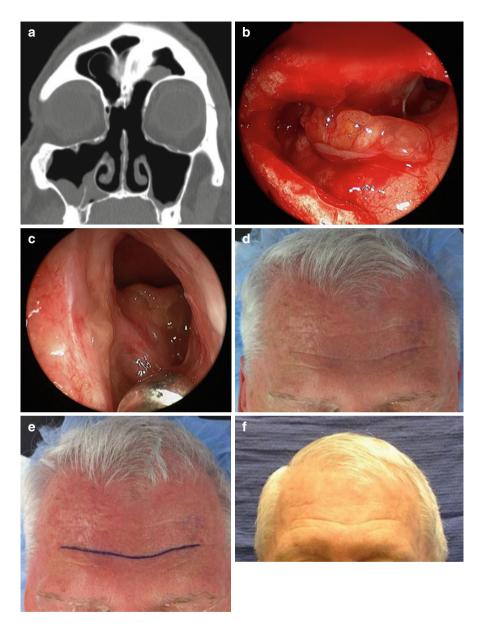
Despite success rates of 85-95 %, long term studies have shown that frontal osteoplastic flap with obliteration can have significant morbidity [26, 27]. In a study by Hardy and Montgomery of 250 osteoplastic flaps with frontal sinus obliteration, the long-term failure rate was 18 % and the revision rate was 5 % [28].

Complication rates of osteoplastic flap surgery [27–31]:

- 35 % forehead numbness
- 10 % post-operative mucocele formation
- 6–10 % moderate to severe headaches
- 6-8 % cosmetic defect including frontal bossing or depression
- 2.8 % cerebrospinal fluid intraoperative leaks
- 1.7 % subdural or epidural hematoma.
- Other complications include meningitis, brain abscess, fat graft donor site morbidity, and osteomyelitis of the bone flap

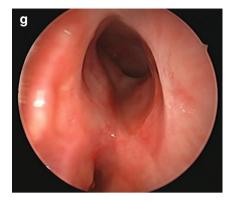
The frontal osteoplastic flap is also beneficial for frontal sinus lesions. Numerous studies have attempted to predict which frontal sinus masses are most likely to fail endoscopic resection and require an osteoplastic flap. Studies have demonstrated that frontal sinus inverted papillomas that are multifocal, associated with malignancy, or attached to the anterior or lateral frontal sinus walls are best managed with the osteoplastic flap (Fig. 39.2a–g) [32, 33]. Chiu et al. suggested that frontal sinus osteomas with anterior or superior attachments (grade III), tumors which extended lateral to a sagittal plane through the lamina papyracea (grade III), and tumors that filled the entire frontal sinus (grade IV) are best managed with the aid of an osteoplastic flap [34]. Mucoceles situated in the lateral aspect of the frontal sinus are also more likely to require an osteoplastic flap procedure for drainage [35].

Although the original description of this procedure describes obliteration of the frontal sinus, it is becoming more common to perform this procedure without obliteration. Obliteration can be avoided if care is taken to preserve the mucosal lining of the sinus and if the frontal recess can be opened via combined external and endoscopic approach (Fig. 39.3a–e). Many authors prefer this technique because it restores physiologic function of the sinus and avoids late complications related to fat reabsorption and mucocele formation. Smith et al. demonstrated that a select group of patients with frontal sinus and outflow tract fractures can be managed with open repair of anterior table fractures without osteoplastic obliteration [36]. The majority of patients in the study developed spontaneous frontal sinus ventilation, and those who had persistent obstruction were successfully managed with endoscopic frontal sinus surgery [36]. Also, obliteration is undesirable in cases of frontal sinus tumors given the difficulty of monitoring for recurrence. If obliteration is avoided, the frontal sinus can be monitored more accurately with cross-sectional imaging and directly via the endoscope.



**Fig. 39.2** (a) Non-contrast coronal CT of a patient with recurrent left frontal sinus inverted papilloma after attempted endoscopic resection. The base of the papilloma is noted to be lateral to a sagittal plane through the lamina papyracea. (b) Intra-operative photo after osteoplastic flap is performed shows papilloma within the left frontal sinus. (c) Endoscopic photo shows the left frontal sinus inverted papilloma through the prior frontal sinusotomy. (d) Pre-operative photo shows existing forehead crease. (e) Intra-operative photo shows planned mid-forehead incision in existing deep forehead crease. (f) This clinic photo taken 2 years after surgery shows a well healed mid-forehead incision. (g) Clinic endoscopic photo taken 2 years after surgery shows a patent frontal sinusotomy with no evidence of recurrent disease

#### Fig. 39.2 (continued)



### **Description of Procedure**

The keys to success of the osteoplastic flap with frontal sinus obliteration are:

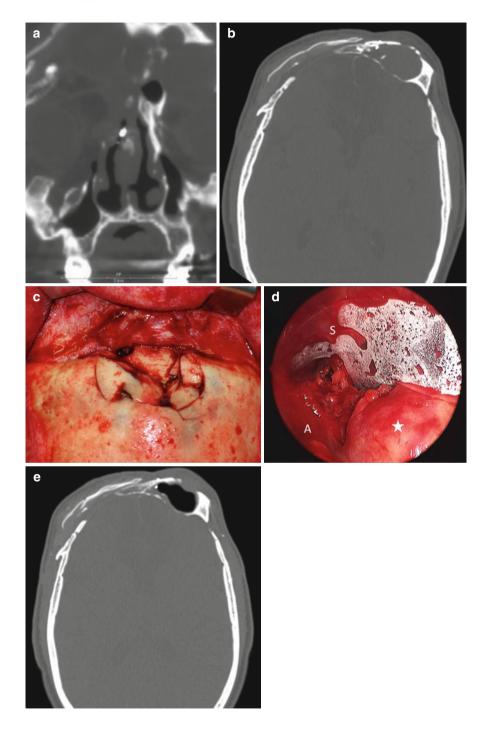
- Proper delineation of the frontal sinus boundaries
- Complete removal of all frontal sinus mucosa
- Burring the inner table of bone throughout the sinus cavity
- · Permanently occluding of the frontal recess
- Avoiding damage to the supraorbital nerve

Surgical steps:

- · Brow, midforehead, or coronal incision
- Skin flap is elevated in a plane superficial to the periosteum
- The sinus is outlined
- · Periosteum incised and elevated
- Anterior frontal wall incised with sagittal saw
- Bone flap fractured inferiorly at the level of the superior orbital rims

The sinus is approached through a brow, midforehead, or coronal incision. The skin flap is elevated in a plane superficial to the periosteum, down to the supraorbital rim. The supratrochlear and supraorbital nerves are spared and may be released from foramina as needed. The extent of the sinus must be mapped

**Fig. 39.3** (a) Non-contrast coronal CT of a patient with remote trauma to the frontal bone and orbit shows stenosis and osteoneogenesis of the frontal recess. (b) Non-contrast axial CT shows displacement of the anterior table of the frontal sinus. (c) Intra-operative photo after coronal flap elevation demonstrates multiple fractures of the anterior table of the frontal sinus. A trephination was created in an existing area of dehiscence in order to assist with proper placement of the frontal sinusotomy, which was performed both via a combined open and endoscopic approach. (d) Intra-operative view of the frontal sinus from above using an endoscope *S* silastic sheet in the shape of a "T" to be removed 6 weeks post-operatively from below, *A* anterior, \* an area of dehiscence in left orbital roof. (e) Non-contrast axial post-operative CT shows aeration of the frontal sinus



using a reliable method to prevent intracranial entry while outlining the frontal bone flap. The perimeter can be determined by using transillumination, placing a wire or bayonet forceps through a trephination site to palpate and map the extent of the sinus, or using a template made from a 6 ft PA Caldwell's view radiograph. For the 6 ft PA "penny" Caldwell view, a penny is taped to the patient's forehead when the film is taken to provide a standard measurement to judge magnification. A properly done film will have no magnification or reduction of the image and will reflect the true size of the frontal sinus shadow. CT image guidance technology is now commonly used, and recent studies have shown that this method is superior to other techniques for determining sinus extent [37].

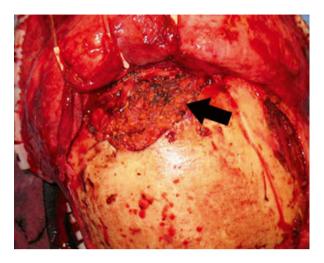
Using any of these methods, the sinus is outlined and an oscillating or sagittal saw is used to cut the frontal bone 2–4 mm inside the limits of the frontal sinus. The saw blade should be beveled inward toward the center of the sinus to provide a shelf for the bone flap to rest on and prevent collapse of the anterior table into the sinus postoperatively. Prior to removing the entire anterior wall, it is suggested that miniplates be fixed to the anterior wall in proper position for ease in replacing the bone flap at the completion of the procedure.

At the supraorbital rims, the very thick bone must be completely transected. A 4 mm osteotome is used to fracture the supraorbital rims and glabella while taking care to preserve the supratrochlear neurovascular pedicles. The osteotome is then and used to divide the interfrontal sinus septum. In the conventional description, the osteoplastic flap with vascularized periosteum adherent to its anterior wall is then fractured inferiorly. Other authors suggest dissecting the periosteum off of the frontal bone down to the supraorbital margins and removing the entire anterior table as a free bone graft without attached periosteum. With this technique, the periosteum can be utilized as part of a pericranial flap for re-lining the floor of the anterior cranial fossa, if necessary.

It is vital to remove all of the mucosa of the frontal sinus before it is obliterated to avoid formation of a mucocele. The inner cortical bone, including the posterior wall of the anterior frontal plate, should be drilled utilizing magnification with a diamond bur to ensure that all mucosa has been removed. Care must be taken not to enter the orbit through the thin floor of the frontal sinus.

Once all of the mucosa has been removed and the bone drilled, the frontal recess is occluded with muscle or fascia, and the sinus is obliterated. A wide variety of implant material for obliteration has been described, but an autologous fat graft is most commonly used (Fig. 39.4) [35]. This is harvested from a periumbilical incision or the left lower quadrant of the abdomen so as not to be confused in the future with an appendectomy incision.

The bone flap is then replaced and secured with mini-plates at the previously planned locations, and the pericranium and coronal flap are closed with absorbable sutures and staples over closed suction drains. Careful suspension of the periosteum and elevated soft tissue is important to prevent a cosmetic deformity. The anterior bone flap is susceptible to infection, so prophylactic antibiotic coverage should be prescribed. A pressure dressing is placed for 3 days to prevent hematoma.



**Fig. 39.4** Frontal sinus osteoplastic flap via coronal incision with fat placed in the sinus for obliteration

# **Reidel's Procedure**

Riedel's procedure may be indicted in the following situations:

- · Osteomyelitis of the anterior wall of the frontal sinus
- Tumor involvement of the anterior table of the frontal sinus
- Failure of frontal sinus obliteration

Riedel's procedure can help eradicate frontal sinus disease and symptoms when endoscopic techniques and osteoplastic flap with obliteration have failed. Riedel's procedure has less morbidity than frontal sinus cranialization since it maintains a barrier in the form of the posterior sinus wall isolating the resection cavity from the intracranial contents. Because the frontal sinus mucosa is completely removed, the chance of recurrent complications is very low, and if recurrence occurs, it can easily be recognized. Postoperative disfigurement is the main morbidity associated with Riedel's procedure [38]. Reconstruction of the anterior wall can be performed at a later date if desired.

Riedel's procedure is performed via a technique similar to the osteoplastic flap except that the anterior wall and floor of the frontal sinus are removed along with all of the mucosal lining of the frontal sinus. The edges are beveled to allow the frontal skin to fall into the cavity against the posterior frontal sinus wall.

# Frontal Sinus Cranialization

### **Surgical Indications**

Frontal sinus cranialization is employed most commonly after trauma. During this procedure, the disrupted posterior wall of the frontal sinus is removed, mucosa is drilled away, and the brain and dura are permitted to rest against the repaired

anterior frontal sinus wall and floor. Judgment is necessary to determine when repair versus cranialization of the posterior table and dura is necessary.

The indications for cranialization include:

- Displaced posterior table fracture with moderate to severe comminution, involving more than 25 % of the sinus [39]
- Posterior table fracture with CSF leak
- Anterior skull base tumors

#### **Description of Procedure**

The surgical approach to frontal sinus cranialization is identical to the approach used in frontal sinus obliteration. With use of a standard coronal incision, a skin flap is elevated to the level of the supraorbital rims. The skin flap can be elevated to preserve a pericranial or galeal-pericranial flap depending on the desired thickness. The flap must be preserved during this approach as it will be needed for closure of dural tears. The borders of the flap are incised sharply using the superior temporal lines as lateral limits of dissection. The posterior edge of the flap is incised near the vertex to provide adequate length. The length can be extended by beginning the harvest posterior to the coronal incision. The flap is then carefully elevated from posterior to anterior. In trauma, exposure of the sinus rarely requires osteotomies due to the severity of the anterior table injury. If an osteotomy is needed, this is done in a similar fashion as the obliteration procedure, though a direct view into the sinus is often available. Any free bone segments from the posterior table are removed and any foreign material or necrotic tissue is removed from the sinus. With the use of a rongeur, the displaced posterior frontal sinus wall is taken down, flush with the floor of the anterior cranial fossa. Bony septations within the sinus are also removed. Large pieces of posterior table bone should be preserved for possible reconstruction of the anterior table. The dura is inspected. Simple lacerations of the dura can be repaired with 5-0 nylon suture. More complex injuries may require neurosurgical debridement and dural closure.

All mucosa is removed from the bony segments of the anterior and posterior tables using a cutting bur. If the wound is grossly contaminated, the bone fragments are soaked in Betadine (povidine-iodine) until needed for reconstruction. All remaining mucosa is meticulously removed from within the frontal sinus. The mucosa in each frontal sinus infundibulum is elevated and inverted, then the bone is drilled out, and each frontal recess is occluded with a temporalis muscle or temporoparietal fascia plug. A small bone fragment may be placed on top. The pericranial flap is then draped over the denuded frontal sinus anterior wall and floor as well as anterior cranial fossa floor to cover any bony defects. When replacing the anterior table bone, the anterior/inferior region of the craniotomy must be widened to create a space which will allow the pericranial flap to pass intracranially without occluding the blood supply. The anterior table is reconstructed with mini-plates or titanium mesh. Outer table calvarial bone grafts are used as necessary to supplement native

bone. The intracranial contents are then allowed to expand forward and fill the newly created cavity. The skin wound is closed in multiple layers over a closed-suction drain.

# Conclusions

Open approaches to the frontal sinus are an important part of the sinus surgeon's armamentarium. Despite the recent advances in endoscopic sinus surgery, frontal sinus disease remains challenging to treat, and there are certain situations in which open frontal sinus surgery provides exposure and access to the sinus which is not possible via the endoscope alone.

# References

- Ung F, Sindwani R, Metson R. Endoscopic frontal sinus obliteration: a new technique for the treatment of chronic frontal sinusitis. Otolaryngol Head Neck Surg. 2005;133(4):551–5.
- 2. Rice DH. Chronic frontal sinus disease. Otolaryngol Clin N Am. 1993;26(4):619-22.
- 3. Schaefer SD, Close LG. Endoscopic management of frontal sinus disease. Laryngoscope. 1990;100(2 Pt 1):155–60.
- Close LG, Stewart MG. Looking around the corner: a review of the past 100 years of frontal sinusitis treatment. Laryngoscope. 2009;119(12):2293–8.
- 5. Wells R. Abscess of the frontal sinus. Lancet. 1870;1:694-5.
- 6. Ogston A. Trephining the frontal sinus for catarrhal diseases. Med Chron. 1884;3:235–8.
- McLaughlin Jr RB. History of surgical approaches to the frontal sinus. Otolaryngol Clin N Am. 2001;34(1):49–58.
- Goodale RL, Montgomery WW. Experiences with the osteoplastic anterior wall approach to the frontal sinus; case histories and recommendations. AMA Arch Otolaryngol. 1958;68(3): 271–83.
- 9. Knapp A. The surgical treatment of orbital complications in disease of the nasal accessory sinuses. JAMA. 1908;51:299.
- 10. Lynch R. The techniques of radical frontal sinus operation which has given me the best results. Laryngoscope. 1921;31:1–5.
- 11. Anderson C. External operation on the frontal sinus. Arch Otolaryngol Head Neck Surg. 1932;15:739–45.
- 12. Kennedy DW. Functional endoscopic sinus surgery. Technique. Arch Otolaryngol. 1985; 111(10):643–9.
- Kennedy DW, Zinreich SJ, Rosenbaum AE, Johns ME. Functional endoscopic sinus surgery. Theory and diagnostic evaluation. Arch Otolaryngol. 1985;111(9):576–82.
- 14. Seiberling K, Jardeleza C, Wormald PJ. Minitrephination of the frontal sinus: indications and uses in today's era of sinus surgery. Am J Rhinol Allergy. 2009;23(2):229–31.
- Lewis D. Surgical management; Sinusitis. Boca Raton, Florida: Taylor and Francis Group; 2006. p. 257–64.
- Batra PS, Citardi MJ, Lanza DC. Combined endoscopic trephination and endoscopic frontal sinusotomy for management of complex frontal sinus pathology. Am J Rhinol. 2005;19(5): 435–41.

- 17. Chiu AG, Vaughan WC. Management of the lateral frontal sinus lesion and the supraorbital cell mucocele. Am J Rhinol. 2004;18(2):83–6.
- Lee JM, Palmer JN. Indications for the osteoplastic flap in the endoscopic era. Curr Opin Otolaryngol Head Neck Surg. 2011;19(1):11–5.
- Zacharek MA, Fong KJ, Hwang PH. Image-guided frontal trephination: a minimally invasive approach for hard-to-reach frontal sinus disease. Otolaryngol Head Neck Surg. 2006;135(4): 518–22.
- Neel 3rd HB, McDonald TJ, Facer GW. Modified Lynch procedure for chronic frontal sinus diseases: rationale, technique, and long-term results. Laryngoscope. 1987;97(11):1274–9.
- 21. Williams HL, Holman CB. The causes and avoidance of failure in surgery for chronic suppuration of the frontoethmo-sphenoid complex of sinuses: with a previously unreported anomaly which produces chronicity and recurrence, and the description of a surgical technique usually producing a cure of the disease. Laryngoscope. 1962;72:1179–227.
- 22. Goodale RL. Some causes for failure in frontal sinus surgery. Ann Otol. 1942;51:648.
- 23. Neel HB, Whicker JH, Lake CF. Thin rubber sheeting in frontal sinus surgery: animal and clinical studies. Laryngoscope. 1976;86(4):524–36.
- 24. Hahn S, Palmer JN, Purkey MT, Kennedy DW, Chiu AG. Indications for external frontal sinus procedures for inflammatory sinus disease. Am J Rhinol Allergy. 2009;23(3):342–7.
- Correa AJ, Duncavage JA, Fortune DS, Reinisch L. Osteoplastic flap for obliteration of the frontal sinus: five years' experience. Otolaryngol Head Neck Surg. 1999;121(6):731–5.
- Goodale RL, Montgomery WW. Anterior osteoplastic frontal sinus operation. Five years' experience. Ann Otol Rhinol Laryngol. 1961;70:860–80.
- 27. Langton-Hewer CD, Wormald PJ. Endoscopic sinus surgery rescue of failed osteoplastic flap with fat obliteration. Curr Opin Otolaryngol Head Neck Surg. 2005;13(1):45–9.
- Hardy JM, Montgomery WW. Osteoplastic frontal sinusotomy: an analysis of 250 operations. Ann Otol Rhinol Laryngol. 1976;85(4 Pt 1):523–32.
- 29. Weber R, Draf W, Kratzsch B, Hosemann W, Schaefer SD. Modern concepts of frontal sinus surgery. Laryngoscope. 2001;111(1):137–46.
- 30. Catalano PJ, Lawson W, Som P, Biller HF. Radiographic evaluation and diagnosis of the failed frontal osteoplastic flap with fat obliteration. Otolaryngol Head Neck Surg. 1991;104(2): 225–34.
- Alsarraf R, Kriet J, Weymuller Jr EA. Quality-of-life outcomes after osteoplastic frontal sinus obliteration. Otolaryngol Head Neck Surg. 1999;121(4):435–40.
- 32. Yoon BN, Batra PS, Citardi MJ, Roh HJ. Frontal sinus inverted papilloma: surgical strategy based on the site of attachment. Am J Rhinol Allergy. 2009;23(3):337–41.
- 33. Walgama E, Ahn C, Batra PS. Surgical management of frontal sinus inverted papilloma: a systematic review. Laryngoscope. 2012;122(6):1205–9.
- Chiu AG, Schipor I, Cohen NA, Kennedy DW, Palmer JN. Surgical decisions in the management of frontal sinus osteomas. Am J Rhinol. 2005;19(2):191–7.
- Kristin J, Betz CS, Stelter K, Berghaus A, Leunig A. Frontal sinus obliteration a successful treatment option in patients with endoscopically inaccessible frontal mucoceles. Rhinology. 2008;46(1):70–4.
- Smith TL, Han JK, Loehrl TA, Rhee JS. Endoscopic management of the frontal recess in frontal sinus fractures: a shift in the paradigm? Laryngoscope. 2002;112(5):784–90.
- Melroy CT, Dubin MG, Hardy SM, Senior BA. Analysis of methods to assess frontal sinus extent in osteoplastic flap surgery: transillumination versus 6-ft Caldwell versus image guidance. Am J Rhinol. 2006;20(1):77–83.
- Raghavan U, Jones NS. The place of Riedel's procedure in contemporary sinus surgery. J Laryngol Otol. 2004;118(9):700–5.
- 39. Donald PJ, Gluckman J, Rice DH, editors. The sinuses. New York: Raven Press; 1995.

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