Complications in Cranio-Maxillofacial and Oral Surgery

Robert Gassner
Editor

Springer

Complications in Cranio-Maxillofacial and Oral Surgery

Robert Gassner
Editor

Complications in Cranio-Maxillofacial and Oral Surgery

Springer

Editor
Robert Gassner
Department of CMF and Oral Surgery
Medical University of Innsbruck
Innsbruck
Austria

ISBN 978-3-030-40149-8 ISBN 978-3-030-40150-4 (eBook)
https://doi.org/10.1007/978-3-030-40150-4

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

To all my inspiring local and international friends, colleagues, companions, and great individuals on this small planet on my academic, surgical, and personal paths who enrich my life, especially my family

Robert Gassner

Foreword

It is a pleasure and a honor for me to have been invited to write the introduction for the first edition of the book *Complications in Cranio-Maxillofacial and Oral Surgery* edited by Prof. Robert Gassner from Innsbruck, Austria.

In this international endeavor, Prof. Gassner's deep knowledge and great experience in the field of oral and cranio-maxillofacial surgery are reflected in a work that will fill an important space in the literature of the specialty. This book cannot be missed in the library of all those professionals who are dedicated to the study and treatment of the different areas of cranio-maxillofacial and also oral surgery.

The topic "complications" is of great importance due to its frequency and the consequences that it implies, both in terms of the impact on the patients and also the legal aspects. We appreciate the contribution of this work to the best knowledge of this aspect of oral and cranio-maxillofacial surgery, rarely systematized in such a structured way. It is really a pleasure to highlight the great international group of coauthors of recognized prestige who have contributed to make this book a reality. Throughout the book, the authors have achieved to make a very complete review of the various aspects of the topic "complications," which will contribute to an update of the reader's knowledge regarding the understanding and management of the possible complications in such important fields like the treatment of congenital or acquired deformities, facial trauma, TMJ pathology or tumors, and reconstructive surgery, among other areas. A special mention is deserved for the inclusion of very specific chapters such as complications of skull base surgery or radiation therapy; the latter written by the editor of the book itself.

Summarizing, based on the fact that a better knowledge of the complications will help to prevent and diagnose them in an early and adequate way, I am sure that this book will contribute to the best treatment of our patients. May I congratulate Robert Gassner for the initiative as well as all the prestigious authors who have participated in the edition of this new contribution to the scientific literature.

Julio Acero
Past President of International Association of Oral and Maxillofacial Surgeons, President of the European Association of CranioMaxillofacial Surgeons, Department of Oral and Maxillofacial Surgery
Ramón y Cajal and Puerta de Hierro University Hospitals, University of Alcala,
Madrid, Spain

Prologue: A Short Note on Wisdom Teeth and Related Complications

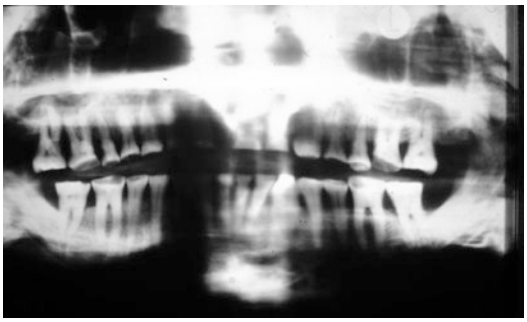


Fig. 1 Zonarc panoramic radiograph of the Iceman—September 1991. (Gassner, K: The Dentition of the Iceman. Diploma Thesis, 2018)

The Iceman (en.wikipedia.org-wiki-oetzi), a 5300-year-old mummy, was found in the Tyrolean Alps in September 1991 and revealed to have a dentition of 28 teeth without wisdom teeth.

This image was taken in September 1991 when the Iceman underwent a Zonarc Panoramic Radiograph at the University Hospital of Innsbruck, Tyrol, Austria, which allowed the examination of patients in a supine position.

Regarding the field of dentistry and medicine, especially oral surgery but also craniomaxillofacial surgery, one of the stunning findings of the radiograph revealed that the iceman did not have wisdom teeth.

Until today, wisdom tooth removal may pose tremendous life-lasting consequences due to their position in the jaw. Therefore, while at the dental office adolescents should be evaluated whether wisdom teeth develop correctly and whether there is enough space for them before they harm neighboring teeth, interfere with their roots, or the infra-alveolar nerve.

There is always a plethora of general risks due to wisdom tooth removal such as pain, swelling, bleeding, inhibition of swallowing, limited mouth opening, mild fever, changes of blood pressure or heart rate, and side effects affecting nervous system (anxiety, seizures, breathing). Wisdom teeth themselves may develop pain, tooth decay, cysts, damage of neighboring roots, pressure on dental arches, and promote inflammatory conditions with bone infections surrounding them. Finally retained wisdom tooth may inhibit the insertion of crowns or bridges on second molars, the implementation of partial or full dentures.

Special risks of wisdom tooth removal are damage of the neighboring tooth/crown, displacement of the tooth or parts of it in the soft tissue, remaining root tips, broken drill heads, oroantral communication or tuber fracture in the upper jaw, nerve damage of the intra-alveolar nerve, lingual nerve, or mandible fracture even weeks after successful wisdom tooth removal. In very rare cases, bone infections may not only develop into osteomyelitis with the leading sign of lip numbness and limited mouth opening but also into life-threatening sepsis and even death.

In conclusion, it can be stated that the presence of all these wisdom tooth-related risks put humans nowadays in a precarious situation. They are at a higher risk than the Iceman with his 28 teeth when discussing wisdom tooth-related survival.

Robert Gassner

Acknowledgments

I am thankful to Dr. Sverre Klemp from Springer who approached me in Hong Kong at the ICOMS 2017 to edit a book on *Complications in Cranio-Maxillofacial and Oral Surgery*. I hesitated but he insisted based on Springer's analysis that I know clinically active experts around the globe who will accept the invitation to prepare book chapters.

I am also thankful to Ahmad Ejaz from Springer for his advice who was extremely helpful and effective in collecting all chapters from around the globe. I am thankful to Daniela Heller from Springer. She was always there for all questions I had regarding the book project.

I am deeply thankful to all my coauthors for their great efforts and commitment to contribute to this book especially **Srinivas Gosla Reddy** from Hyderabad, India, on Complications in Cleft Lip Palate Surgery, **B J Costello** from Pittsburgh, Pennsylvania, on Complications in Craniofacial Surgery, **Suzanne McCormick** from Encinitas, California, on Complications in Distraction Surgery, **Roger Moreira** from Goiânia, Brazil, on Complications in Orthognathic Surgery, **Radhika Chigurupati** from Boston, Connecticut, on Complications of Odontogenic and Non-odontogenic Infections, **Bilal Al-Nawas** from Mainz, Germany, on Complications in Oral Implant Placement, **Tetsu Takahashi** from Tohoku, Japan, on Complications in TMJ Surgery, **Sven OTTO** from Munich, Germany, on Complications treating MRONJ, **Andreas Kolk** from Innsbruck, Austria, on Complications due to Removal of Plates and Screws, **Wilhelm Eisner** from Innsbruck, Austria, on Complications and Orofacial Pain, **Gregorio Sanchez Anicheto** from Madrid, Spain, on Complications in Skull Base Surgery, **Bernhard Frerich** from Lübeck, Germany, on Complications in Maxillofacial Cancer Resection, **Volker Schartinger** from Innsbruck, Austria, on Complications in Neck Dissection, **Christos Perisanidis** from Athens, Greece, on Complications in Free Flap Reconstruction, and their coworkers, respectively. They are all highly educated and accomplished surgeons and also scientists. It is a real privilege and pleasure to share this book authorship with all of them.

Contents

Part I

Malformations

1 Complications in Cleft Lip and Palate Surgeries

Srinivas Gosla Reddy and Ashish Fanan

Contents

S. G. Reddy (✉) · A. Fanan
GSR Institute of Craniomaxillofacial and Facial Plastic Surgery, Hyderabad, Telangana, India
e-mail: goslareddy@craniofacialinstitute.org

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_1

1.1 Introduction

Cleft lip and palate (CLP) is a three-dimensional anomaly involving hard and soft tissues of the face. The comprehensive treatment of CLP deformities requires a thorough understanding of the deformed anatomy and a balance between intervention and growth.

In the past, poor outcomes were the result of nonexistent protocols, fragmented care, and lack of periodic assessment. Many of the developed countries now have a well-structured and organized cleft team centers. However, the situation in most of the developing countries is quite different, where they lack properly trained medical personnel which hampers delivery of high-quality care to the orofacial cleft patients. Funding in these resource-poor nations is managed through outreach programs of various philanthropic organizations around the world.

Complications can occur in the management of cleft patients due to lack of understanding of surgical principles and improper techniques. This chapter provides a detailed classification of complications in cleft lip and palate surgeries and insights into their comprehensive multidisciplinary management.

1.2 Overview of Complications Associated with Cleft Lip and Palate Patients

Classification of complications
General anesthesia
I. Airway complications
1. Difficult intubation
2. Tube disconnection and tube compression
3. Desaturation
4. Disconnection or accidental extubation
5. Laryngospasm and bronchospasm
II. Other complications
1. Arrhythmia
2. Hyper-/hypothermia

Classification of complications
Surgery
I. Cleft lip
A. Intraoperative complications
1. Incorrect markings
2. Blood loss
B. Immediate postoperative complications
1. Infection and tissue necrosis
2. Wound dehiscence
3. Hematoma formation
C. Late postoperative complications
1. White roll mismatch
2. Vermillion notching (whistle lip deformity)
3. Hypertrophic scar/contracture
4. Shortening of lip length
5. Nostril asymmetry
6. Differences in alar base
7. Infra-sill depression
8. Prolabium necrosis

Classification of complications
Surgery
II. Cleft palate
A. Preoperative complications
1. Otitis media
2. Aspiration pneumonia
3. Nutritional deficiency
B. Intraoperative complications
1. Blood loss
2. Damage to the pedicle
C. Immediate postoperative complications
1. Bleeding
2. Airway obstruction
3. Wound dehiscence/infection
4. Hanging palate
5. Erosion of corner of mouth

D. Late Postoperative complications
1. Fistula formation
2. Velopharyngeal incompetence
3. Maxillary hypoplasia
4. Recurrent ear infections

Classification of complications
Surgery
III. Alveolar bone graft
Recipient site
A. Intraoperative complications
1. Perforation of nasal layer
2. Damage to the tooth/tooth bud

B. Postoperative complications
1. Graft exposure

Donor site (anterior iliac crest)
A. Intraoperative complications
1. Bleeding
2. Fracture of iliac cortex
3. Peritoneal perforation
4. Injury to inguinal ligament
5. Injury to ilioinguinal nerve

B. Postoperative complications
1. Deep hematoma
2. Seroma
3. Infection and wound dehiscence
4. Meralgia paresthetica
5. Hernia
6. Gait changes
7. Foot drop
8. Cosmetic defect:
   (a) Hypertrophic scar
   (b) Pelvic brim defect

Classification of complications
Surgery
IV. Orthognathic surgery
A. Intraoperative complications
1. Hemorrhage
2. Bad split/fracture
3. Nerve injury
4. Damage to the tooth buds

B. Postoperative complications
1. Vascular compromise; complete or partial necrosis
2. Palatal perforation
3. Malunion/nonunion
4. Malocclusion
5. Relapse
6. Temporomandibular joint effects
7. Sensory impairment
8. Cranial nerve palsies/cavernous sinus thrombosis
9. Blindness; partial or complete
10. Maxillary aneurysm
11. Avascular necrosis
12. Velopharyngeal insufficiency (VPI)

Classification of complications
Surgery
V. Distraction osteogenesis
A. Intraoperative complications
1. Hemorrhage
2. Bad split/fracture
3. Nerve injury
4. Damage to the tooth buds

B. Postoperative complications
Intradistraction complications
1. Pin infections, pin and device loosening
2. Device failure
3. Inappropriate distraction vector
4. Premature consolidation
5. Coronoid process interference
6. Fibrous pseudoarthrosis
7. Trismus

Postdistraction complications
1. Delayed consolidation
2. Premature consolidation
3. Malocclusion
4. Growth disturbances

Classification of complications
Surgery
VI. Rhinoplasty
A. Intraoperative complications
1. Bleeding
2. Cartilage fracture
3. Buttonholing of skin
4. Perichondrium tear

B. Immediate postoperative complications
1. Transient numbness and pain
2. Hematoma
3. Infection and wound dehiscence
4. CSF rhinorrhea
5. Olfactory disturbances

C. Late postoperative complications
1. Columellar skin necrosis
2. Graft/implant migration
3. Nasal stenosis/blockage
4. Septal perforation
5. Nasal valve collapse
6. Poor hypertrophic scar

Classification of complications
Surgery
VII. Complications associated with syndromic patients
A. Pierre Robin syndrome
B. Velocardiofacial syndrome

1.3 Complications Associated with Anesthesia

1.3.1 Airway Complications

Most anesthetic complications in cleft surgeries are related to *airway management* namely difficult intubation, tube disconnection, tube compression, desaturation, disconnection, or accidental extubation. Other complications are laryngospasm, bronchospasm, and airway edema.

Children with hypoplastic mandible or wide cleft (common in Pierre Robin syndrome) are at increased risk of tongue prolapse into the pharynx and pose a problem during induction of anesthesia. Postoperative respiratory obstruction may result following the closure of wide cleft palate or syndromic cleft associated with hypoplasia of mandible, hematoma, or due to accidental leftover packs. There is also change in oral/nasal airway dynamics especially in syndromic children that may present with the problem of respiratory obstruction in the postoperative period. Use of nasopharyngeal airway or/and tongue suture to allow forward traction can help in such a situation.

The optimum anesthetic management will depend on the age of the patient, the availability of intraoperative monitoring equipment, anesthetic drugs and expertise, and the level of postoperative care that is available.

1.3.1.1 Difficult Intubation

If the airway becomes obstructed after induction of the patient, the maneuvers to be followed are turning the patient lateral or semi-prone and the use of a nasal or oral pharyngeal airway. *No neuromuscular blocking agents should be given until one is sure that the lungs can be ventilated with a mask*. The laryngeal mask airway may help and enable the patient to achieve adequate depth of anesthesia for direct laryngoscopy and intubation to be tolerated. In the event of being unable to secure the airway, consideration should be given to allowing the patient to wake up and deferring surgery to when they are older and have more structural and neuromuscular maturity [1, 2].

1.3.1.2 Tube Disconnection and Tube Compression

The ET tube should be properly secured with the help of adhesive tapes. Scrubbing solutions should not interfere with the adhesive tapes. Muhammed et al. devised an L-shaped metal rod used to prevent endotracheal compression during palatopharyngeal or intraoral surgery (Fig. 1.1a) [3]. Also tongue blade with longer groove for the endotracheal tube can be used to prevent tube dislodgement (Fig. 1.1a, b).

1.3.1.3 Desaturation

Desaturation can occur due to preoperative infection and inflammation of the respiratory tract,

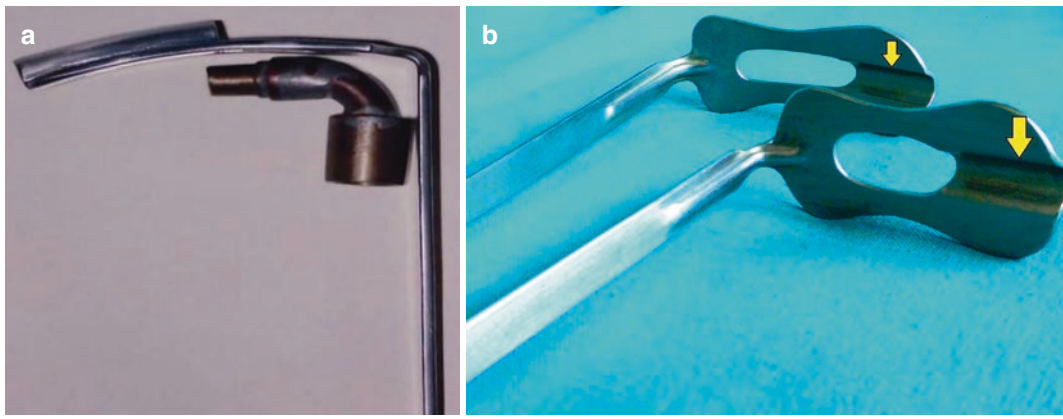


Fig. 1.1 (**a**) Metal rod with tongue blade. (**b**) Tongue blade with longer groove

tube compression, laryngospasm, or bronchospasm. Hence it is essential to defer the surgery till the patient's pulmonary health and functional reserves return to the normal.

1.3.1.4 Accidental Extubation

Accidental extubation when positioning the head for surgery is minimized if the tube is placed 1.5 cm above the carina. Inadvertent extubation is mostly associated manipulation during placement of gag for cleft palatal surgery. Immediate reintubation should be performed with constant monitoring of the oxygen saturation. Hence, it is mandatory to inform the anesthesia team during manipulation of the gag during surgery.

1.3.1.5 Laryngospasm and Bronchospasm

Laryngospasm is common in children with history of asthma or upper airway infections. Precipitating factors of laryngospasm are airway manipulation, blood/secretions in the pharynx, regurgitation/vomiting, surgical stimulus, moving patient, irritant volatile agent, or failure of anesthetic delivery system.

Bronchospasm usually manifests during anesthesia as an expiratory wheeze, prolonged expiration, and/or increased inflation pressures during intermittent positive pressure ventilation. The chest may be silent on auscultation and the diagnosis may rest on correct assessment of increased inflation pressures.

Laryngospasm is relieved by giving muscle relaxant, by chin lift jaw thrust maneuver, and by oxygenation (100%) whereas bronchospasm is relieved through intravenous hydrocortisone and deriphyllin.

1.3.2 Other Complications

1.3.2.1 Arrhythmia

Preoperative, perioperative, and postoperative arrhythmias in cleft lip and palate patients are associated mainly with hemodynamic change or any previously diagnosed underlying cardiac disease.

1.3.2.2 Hypo-/Hyperthermia

Children tend to lose more heat through conduction and radiation than adults, due to less insulating subcutaneous fat and a higher surface area to volume ratio. Core temperature monitoring probes, body warmers, and fluid warmers should be available in the operatory. It is recommended that recommend active warming be used during the first 30 min if the surgery is expected to last for <2 h, and no such measures are required if the expected duration is >2 h [4]. Operatory temperature should be maintained between 21 and 24° C with a humidity of 40–50%.

Anesthetic management of cleft repair surgeries requires a skilled professional, meticulous monitoring, and postoperative care in an intensive care unit set up to minimize the complications.

1.4 Complications Associated with Surgery

1.4.1 Complications Associated with Cleft Lip Surgery

1.4.1.1 Intraoperative Complications

Incorrect Markings

Incorrect markings on the lip are due to lack of proper understanding of the anatomical landmarks and cleft anatomy. This error can also occur due to improper magnification and illumination.

Blood Loss

Mean estimated blood loss during unilateral cleft lip surgery was 26–47 mL. Mean estimated blood loss in unilateral cleft lip surgery was not significantly different from that of bilateral cleft lip surgery (P = 0.46) [5]. However, a significant positive correlation between duration of surgery and blood loss is established.

How to Avoid This Complication

Compression of superior labial artery during dissection with index finger and thumb can significantly prevent blood loss during surgery.

How to Treat This Complication
Cleft lip surgery is a low volume blood loss surgery. This surgery very rarely requires blood transfusion.

1.4.1.2 Immediate Postoperative Complications

Infection, Wound Dehiscence, and Tissue Necrosis

This can occur due to tension in the repair or improper suturing of the orbicularis oris muscle, trauma, etc. Maintaining a clean wound is essential. It is widely accepted that the cleft lip wound should be cleaned after each feeding, with gentle action and no repetitive rubbing. A sterile cotton swab, normal saline solution, and topical antibacterial ointments are recommended for wound dressing.

Omitting this essential step paves the way for infections which leads to wound dehiscence and finally even to tissue necrosis (Fig. 1.2).

How to Avoid This Complication
Tension in the repair can be avoided by extensive subperiosteal elevation of the facial mask especially in wide clefts. Also suturing of the orbicularis oris muscle should be performed meticulously.

How to Treat This Complication
Infection must be controlled and if necessary secondary surgery is required after 3–6 months.

1.4.1.3 Late Postoperative Complications

White Roll Mismatch

Malalignment of white roll of even 1 mm is visible from a conversational distance. It is one of the most common and easily discernable sequela of cleft lip surgery. In bilateral cleft lip repairs, preservation of prolabial and lateral element white rolls can result in the triple line effect of the prolabial white roll, scar, and lateral white roll underneath.

How to Avoid This Complication
A good tip is to mark the white roll points with ink before local anesthesia injection to ensure accurate alignment which is maintained by placing 6-0 prolene sutures above and below the white roll. This deformity can be avoided by adequate rotation of the noncleft segment.

How to Treat This Complication
White roll mismatch is corrected by a diamond-shaped excision of the white roll scar extending above and below the roll (Fig. 1.3).

Vermillion Notching (Whistle Lip Deformity)

The vermilion of the lip is a composite structure consisting of the orbicularis oris muscle, fat, vermilion, and specialized epithelium. Vermillion notching is a discontinuity in the free border of the vermillion. This may be central ("whistle deformity") or lateral along the line of the scar. It may be caused by:

- Inadequate rotation
- Inversion of the sutured edges
- Orbicularis oris marginalis muscle deficiency
- Straight line scar contracture
- Failure to fill central tubercle with lateral vermillion tissues
- Diastasis of the orbicularis muscle at the base of the nose resulting in an upward pull on the central tissue or a combination of these (Fig. 1.4a, b)

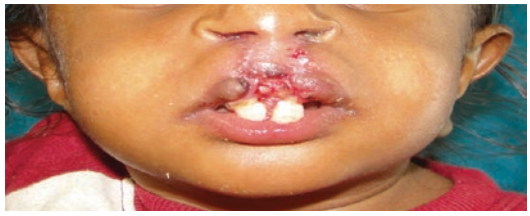


Fig. 1.2 Infection wound dehiscence and tissue necrosis

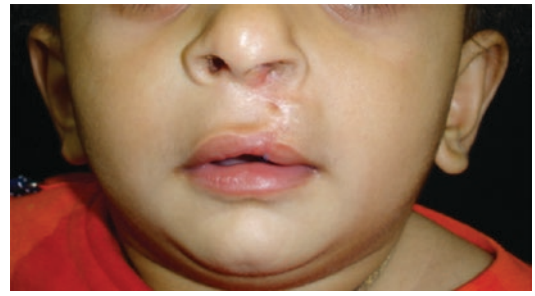


Fig. 1.3 White roll mismatch

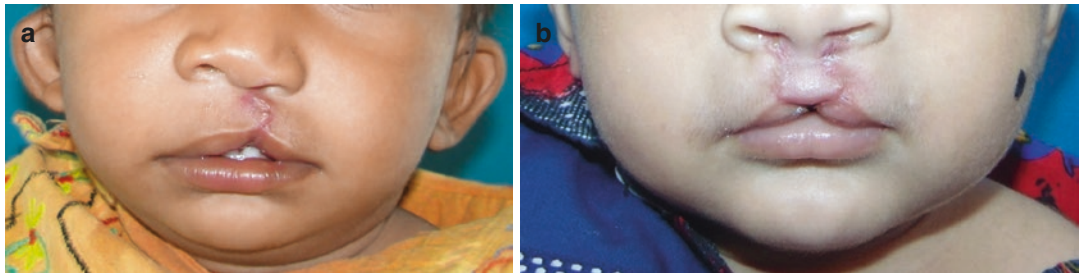


Fig. 1.4 (**a**) Vermillion notching. (**b**) Vermillion notching whistle lip deformity

How to Avoid This Complication

This deformity may be prevented by properly choosing the Cupid's bow point on the lateral lip segment, i.e., Nordhoff's point. An incision placed too laterally can cause short lip in horizontal/transverse dimension. An incision placed too medially compromises the vertical height of the lip and vermillion. Also excessive dissection of pars marginalis muscle should be avoided to avoid this complication.

How to Treat This Complication

There are various techniques to correct the vermillion deficiency, the choice of which should be based upon the underlying cause. If the deformity is due to a scar contracture, a z plasty can be performed to release the scar. If the deformity is due to lack of orbicularis muscle bulk in the vermillion, it can be addressed by a wedge-/diamond-shaped excision and reorientation and resuturing of the orbicularis muscle. This can prevent scar contracture and notching during function. The vermillion bulk can also be augmented by free fat graft, fillers, or any autologous material. A severe vermillion deficiency in the central tubercle region with no locally available tissues can be corrected by Abbe's flap.

Hypertrophic Scar Contracture

Hypertrophic scars can result due to

- Local wound tension
- Infection
- Improper tissue handling
- Genetic predisposition (Fig. 1.5)

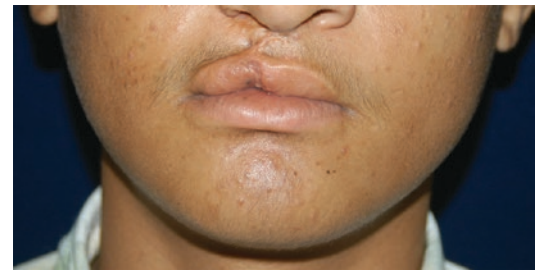


Fig. 1.5 Hypertrophic scar contracture

How to Avoid This Complication

Hypertrophic scars can be prevented by relieving any unnecessary tension, meticulous dissection of involved structures, and proper muscle approximation. This allows skin suture placement without any tension. A good wound care is equally important to prevent infections. These scar management methods should be employed for 6–12 months until the scar matures completely. Choice of suture materials and atraumatic suturing techniques also play an important role in the fate of the scar.

How to Treat This Complication

Scar management methods are also employed to achieve an acceptable scar which includes taping, scar massage, and sun protection. Unsightly scars can be managed by scar excision procedure down to the orbicularis muscle layer and sufficiently mobilizing the skin flaps for tension-free closure. The scar tissue including the skin and muscle if scarred is excised and resuturing is performed. If a hypertropic raised scar starts developing post surgery, it can be managed by

intralesional steroid injections. Parents of the patients should be counseled for the amount of the time it will take for the scar to fade and not vanish completely.

Shortening of Lip Length

Short lip length can result from

- Failing to balance the cupids bow
- Poor scarring
- Straight line scar
- Under rotation of the flap

How to Avoid This Complication

The cupids bow should be horizontal before the start of suturing. A straight line scar should be avoided. Adequate rotation of the flaps should be achieved before suturing.

How to Treat This Complication

Lip deficiencies of up to 1 mm can be corrected by an elliptical excision of previous scar by *Rose Thompson effect.* Any major discrepancy in vertical height requires complete revision of the lip which includes release of all the abnormal attachments, meticulous muscle suturing, and accurate leveling of cleft side of cupids bow by giving an adequate back cut (Fig. 1.6).

Nostril Asymmetry and Differences in Alar Base

Nasal asymmetry in cleft patients is caused by three important anatomical changes:

- Nasal septum deviation
- Significant distortion of the alar cartilages caused by separation of the bone structures and soft tissues
- Unleveling of the maxillary and alveolar bone resulting from the spreading of the palatal shelves

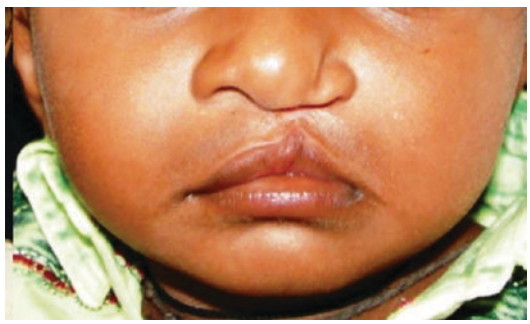


Fig. 1.6 Shortening of lip

Unleveling of the bone of the alveolar arches, viz. greater segment and smaller segment and maxilla, exerts a vector of caudal force over a nasal wing, as well as overall nose structures; this is one of the important anatomical limitation to obtain final nasal symmetry after lip repair.

How to Avoid This Complication

Morphofunctional reposition of the septal cartilage from its physiological position to its anatomical position is also an important factor to obtain nasal symmetry. Mulliken and Martínez-Pérez observed 23% postoperative nasal asymmetry in unilateral cleft patients [6]. Cutting and Dayan described the inclusion of percutaneous sutures that exert a vector of force contrary to the primary caudal force originating from the alveolar gap resulting from the spreading of the palatal shelves [7]. Postoperative nasal splinting in the primary management of the unilateral cleft nasal deformity serves to preserve and maintain the corrected position of the nostril after primary lip and nasal correction, resulting in a significantly improved aesthetic result. Therefore, it is recommended that all patients undergoing primary correction of complete unilateral cleft deformity use the nasal retainer postoperatively for a period of at least 6 months [8] (Fig. 1.7).

How to Treat This Complication

Preschool (5–6 years of age) rhinoplasty is generally performed if there is a significant deformity mostly involving the lower lateral cartilages or is delayed until the nasal growth is complete (16 years of age).

Nasal Stenosis

Nasal stenosis can occur due to excessive scarring of the incision taken around the ala similar to conventional Millard's incision. Salyer recommended leaving the nostril larger because tightening a laterally displaced alar base is easier than secondary correction of tight external nares (Fig. 1.8).

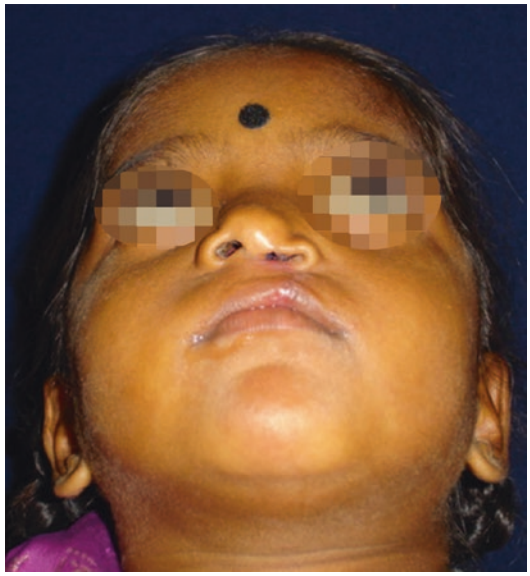


Fig. 1.7 Nostril asymmetry

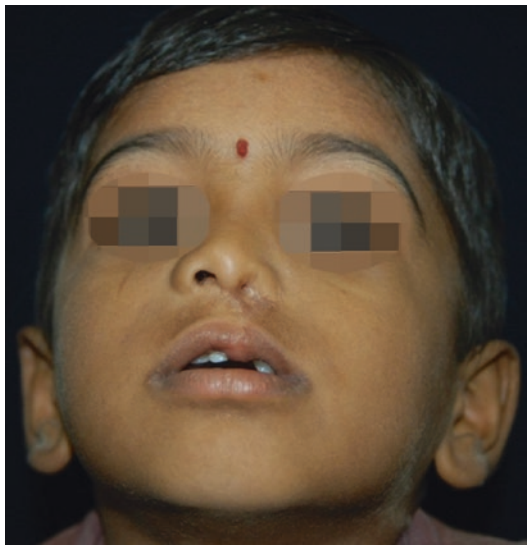


Fig. 1.8 Nasal stenosis

How to Avoid This Complication
Nasal stent post surgery can be used to prevent nasal stenosis.

How to Treat This Complication
Surgical repair can be carried out, viz. nasolabial flaps, boomerang flap, vestibular rotation flap.

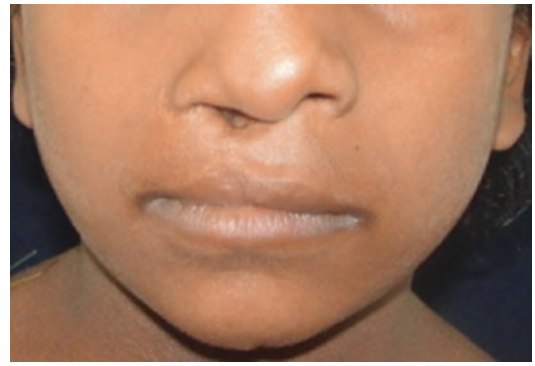


Fig. 1.9 Infra-sill depression

Infra-sill Depression

An infra-sill depression indicates lack of muscle bulk at the base of the nose. Another cause of this depression is the thin dermis in the tip of the advancement flap (Fig. 1.9).

How to Avoid This Complication
A good amount of orbicularis oris muscle bulk at the base of the nose is recommended during the suturing of the muscular part.

How to Treat This Complication
A dermal draft or dermal fillers can be used to elevate the infra-sill depression.

Prolabial Necrosis (Bilateral Cleft Lip Cases)

Devitalization and necrosis of prolabium is a feared complication in bilateral cleft lip surgeries. It was initially thought that a bilateral cleft lip repair required staged procedure since the prolabium could not bear the surgical insult of a single-stage technique. However, the true incidence of prolabial necrosis is not known and the risk of prolabium necrosis still exists. Any aggressive open technique in the nose during primary reconstruction also carries a higher risk of skin necrosis to the columella and prolabium [9] (Fig. 1.10a, b).

How to Avoid This Complication
It is important to increase the prolabial flap thickness when it is raised from the philtral

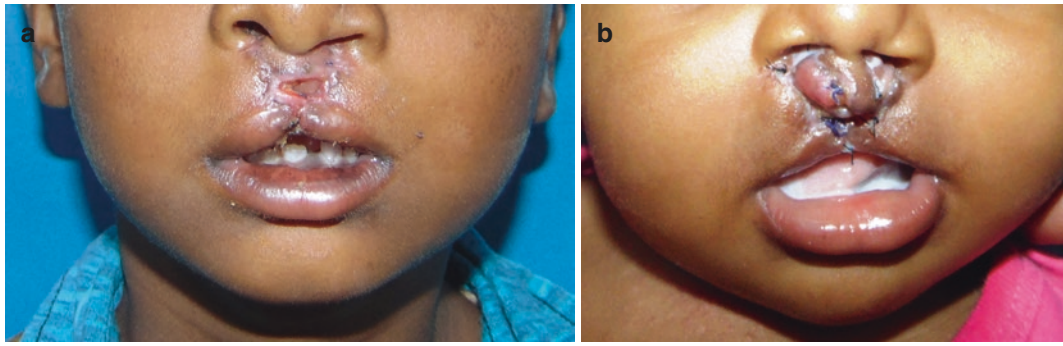


Fig. 1.10 (**a**) Prolabial necrosis. (**b**) Prolabial necrosis

notch and ensure a tension-free closure. Capillary refill of the flap and its color should be checked periodically intraoperatively and postoperatively. Bipolar cautery should be used judiciously on the prolabial flap. After surgical intervention, the prolabium should be pink and perfused to ensure survival. If it appears blanched, the sutures should be removed to help prevent necrosis.

How to Treat This Complication

Prolabial flap necrosis will heal as a scar with poor cosmetic outcomes and may lead to short and tight upper lip. Local tissue arrangement including complete muscle repositioning methods should be employed. Abbes flap should be employed when there is a loss of tissue from multiple operations or when the prolabium has been discarded. Abbes flap reconstructs the lip in all the three layers.

1.4.2 Complications Associated with Cleft Palate Surgery

1.4.2.1 Preoperative Complications

Otitis Media

The hearing loss due to otitis media in a cleft patient is a well-known complication, but generally gets ignored. The abnormal reflux of fluids and food around eustachian tube causes chronic inflammation and obstruction of the tube leading to otitis media.

How to Treat This Complication

These patients should undergo regular otological and audiological follow-ups with appropriate interventions. These interventions can be in the form of grommets and myringotomy tubes to drain the fluid in middle ear cavity. These can be used in patients with age group of 6 months to 12 years [10]. Eustachian tube dysfunction resolves in at least 50% patients after palate repair and resolves in most patients by the age of 5 [10].

Aspiration Pneumonia

Aspiration pneumonia was defined as the coexistence of pneumonia at chest radiography with a history of frequent choking during feeding. The incidence of aspiration pneumonia in infants with cleft palate is more than infants born without cleft palate. The faulty feeding position (on the child's back) is the main reason for aspiration of the contents.

How to Avoid This Complication

The parents and the caretakers should be educated and demonstrated about feeding the baby in a proper way (Fig. 1.11).

Nutritional Deficiency

Studies have shown that children with clefts have lower height and weight when compared to a control group, especially during the first year of life [11, 12]. Children with either cleft lip or palate have a short, fast, uncoordinated, and ineffective intraoral suction, due to abnormal muscular

Fig. 1.11 Feeding position for a cleft baby

attachments which may cause asphyxia because of the entrance of milk into the nasal cavity, and also excessive air ingestion [13, 14]. The factors that optimize weight gain include proper feeding advice and multidisciplinary integrated care. The main priority during the first months of life, including those with cleft palate, should be appropriate nutrition along with multivitamin, calcium, and other dietary supplements.

How to Avoid This Complication

The parents and the caretakers should be educated about the feeding technique, viz.:

- Positioning the baby
- Burping the baby after the feeding
- Feeding the baby in short interval of times

1.4.2.2 Intraoperative Complications

Blood Loss

An average blood loss of 46 mL with palatoplasty (3–12% of expected blood volume) was reported by Kulkarni et al. [2]. The alteration of the heart rate is a direct consequence and the lowering of the body temperature is an indirect consequence of blood loss. The reduction of the intraoperative blood loss is one approach to decreasing the probability and the severity of intra- and postoperative complications. A blood loss of about 50 mL during infant surgery with total patient blood volume of 400–700 mL can disturb the circulation, requiring a transfusion of banked blood or plasma substitutes. A precise assessment of the blood loss is therefore vital in order to find the balance between over-transfusing and unnecessary transfusion. Ringers lactate should be infused (considering 4-2-1 rule), 8–12 mL/kg/h throughout the procedure and 4–6 mL/kg/h for 4–6 h postoperative period. Intraoperative continuous monitoring should include heart rate, blood pressure, ECG, SpO_2, end tidal carbon dioxide ($EtCO_2$), temperature, and blood loss.

How to Avoid This Complication

The shortening of the duration of a cleft surgery is an important step to reduce the total loss of blood.

How to Treat This Complication

Blood loss up to 20% of total blood volume can be corrected with crystalloids (in 1:3 ratio) and if >20% with blood transfusion (in 1:1 ratio). Surgical site should be infiltrated with local anesthetic (1–1.5%) containing adrenaline (1:100,000).

Damage to the Pedicle

Damage to the pedicle can occur due to aberrant anatomy or improper surgical technique. Hence, a careful approach when dissecting in and around the pedicle is required. There is a network of anastomoses between the vessels that supply the hard palate and soft palate. Avoid using sharp instruments for elevation of the mucoperiosteal flaps.

How to Avoid This Complication

A wet gauze along with a freer elevator can be used to elevate the mucoperiosteal flap to prevent damage to the pedicle.

How to Treat This Complication

The most important are the anastomoses between the ascending palatine and lesser palatine arteries which acquire importance when the greater palatine artery is sectioned accidentally during palatoplasty. When a pedicle is damaged, minimal dissection in the soft palate region should be the rule.

1.4.2.3 Immediate Postoperative Complications

Bleeding

Slight oozing from the raw surfaces of the flaps is expected; however, it is very important that the patient does not leave the operating room until the bleeding is controlled. A fine tip bipolar cautery is used to cauterize the edges of the flaps. The Dingman retractor should be left in place until bleeding is controlled. Gelfoam or Surgicel or other hemostatic agents can also be able to control active bleeding. Patient's parents should be counseled beforehand regarding possibility of postoperative bleeding and the need to take the patient back to the operatory to reexplore the surgical site and cauterize the active bleeders.

How to Treat This Complication

Active bleeding can occur mostly due to accessory greater palatine or lesser palatine vessels. The patient should be taken to the operatory to cauterize the active bleeders.

Airway Obstruction

Patients with Pierre Robin sequence or other additional congenital anomalies have an increased risk of airway problems following palatoplasty. There is also change in oral and nasal airway dynamics especially in children with Pierre Robin Syndrome which may present itself in postoperative period and if severe enough can lead to respiratory obstruction. These patients must be identified prior to surgery so that they can be monitored and managed appropriately, minimizing the likelihood of major complications or death.

Postoperative respiratory obstruction may occur due to:

- Closure of wide cleft palates
- Hypoplastic mandibles in syndromic patients (PRS)
- Hematoma
- Accidental left-over packs
- Aspiration of secretions or blood, collected in the nasopharynx

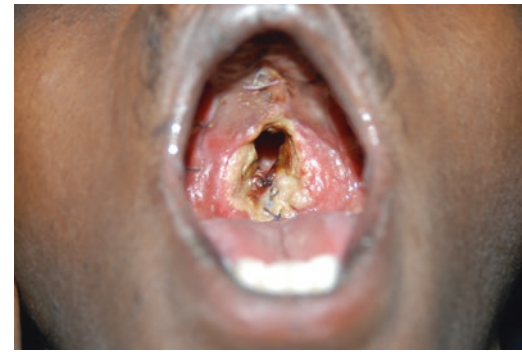


Fig. 1.12 Wound dehiscence

How to Treat This Complication

Nasopharyngeal airways and tongue stitch can be used on emergency basis. Hence continuous monitoring of the respiratory distress (use of accessory respiratory muscles) and oxygen saturation (SpO_2) is essential.

Wound Dehiscence

Wound dehiscence is a surgical complication that results from poor wound healing. The nasal and oral layers of the surgical wound separate or the whole wound splits open. This dehiscence usually occurs between 7 and 10 days post surgery (Fig. 1.12).

Wound dehiscence varies depending on the technique of surgery and the type of cleft. The generalized causes are:

- Infection at the wound
- Tight sutures
- Poor knotting or tissue handling
- Inadvertent trauma to the wound after surgery
- Weak tissue or muscle at the wound area
- Incorrect suture technique used to close operative area
- Vitamin C deficiency

How to Avoid This Complication

Maintaining a good oral hygiene with strict diet instructions to the parents/caretakers of the patient.

How to Treat This Complication

Fistula formation can be a potential complication of wound dehiscence. No immediate management is required post-wound dehiscence.

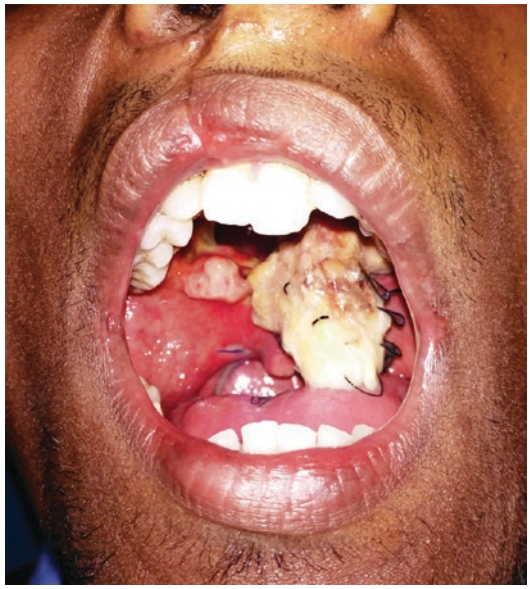


Fig. 1.13 Hanging palate

The management of fistula formation is described further in details.

Hanging Palate

The anterior wound dehiscence results in the detachment of the mucoperiosteal flap from the alveolar margin (Fig. 1.13). This complication is very troublesome for the parents as well as the surgeon. It mostly occurs on the 4th or 5th postoperative day, the cause being hematoma formation between the mucoperiosteal flap and nasal layer.

How to Avoid This Complication

Good adaptation sutures preventing the dead space, including nasal layer and oral layer, is very important to prevent this complication.

How to Treat This Complication

An innovative management protocol using a methyl methacrylate obturator fixed to the alveolar arch has been described by K. Agrawal [15]. Use of adhesion systems, i.e., fibrin glue has also been advocated.

Erosion of Corner of Mouth

This can occur due to long operating hours for the palate repair (Fig. 1.14).

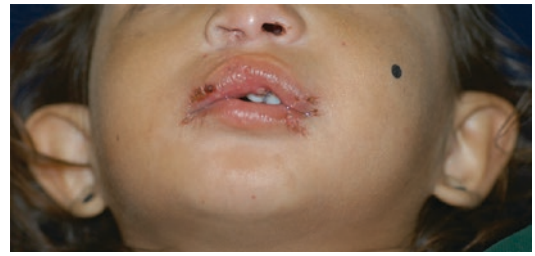


Fig. 1.14 Erosion of corner of mouth

How to Avoid This Complication

Release the gag if long operating hours are expected during the surgery.

How to Treat This Complication

Application of emolients and moisturizing ointments.

1.4.2.4 Late Postoperative Complications

Fistula Formation

An oronasal fistula is an abnormal communication between the oral cavity and the nose that occurs after surgical repair of a cleft palate. There is no consensus for the rate of fistula formation following surgery despite extensive research in cleft palate surgeries. A meta-analysis by Bykowski MR et al. reports fistula formation rates was 4.9% following cleft palate surgeries. However, the rate of ONF varies from 4 to 35% or even more in case of primary palatoplasty surgeries. Oronasal fistulae have been associated with severity and type of the cleft (Fig. 1.15a–c).

Palatal fistula can occur due to:

- Poor wound healing due to nutritional deficiency and anemia
- Tension or improper suturing technique
- Wound dehiscence
- Partial mucoperiosteal flap necrosis
- Infection
- Injury to the pedicle
- Hematoma formation

Most of the fistulas are observed in the early stages, i.e., within 2 weeks after palatal surgery.

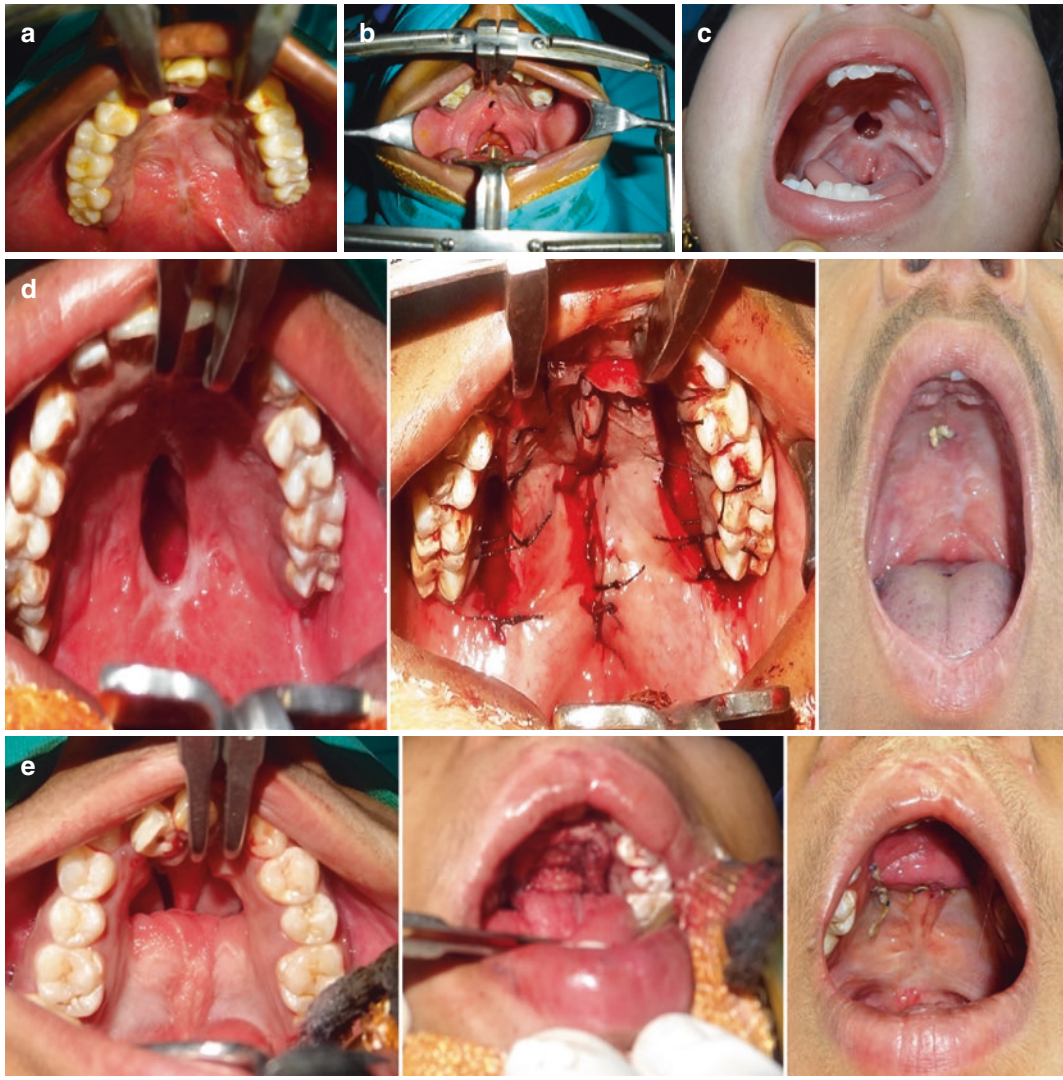


Fig. 1.15 (**a**) Anterior palatal fistula. (**b**) Midpalatal fistula. (**c**) Junctional palatal fistula. (**d**) Junctional palatal fistula closure with local flaps. (**e**) Junctional palatal fistula closure with tongue flap

This condition has functional consequences because of fluid and air leakage. Air leakage can cause problems such as speech impairments due to nasal escape and nasal regurgitation. Fistulas causing disturbance in speech should undergo early repair, whereas the closure of fistulas not associated with speech problems should be delayed, if possible, until completion of orthodontic maxillary arch expansion. Fistula closures can also be combined with secondary alveolar bone grafting surgery [16].

How to Avoid This Complication

- Reduction in tension during suturing: A good nasal layer repair is critical in reducing the incidence of postoperative fistula. No technique is good if it does not allow closure of palate without tension. Any tension during palate repair will cause tear in mucoperiosteum when one tries to tighten the knots. The tension can be reduced by releasing the pedicle from the foramen, releasing lateral incision like in Von Langenbeck palate repair or separating the pedicle from the flap

especially in very wide clefts, e.g., syndromic patients.
- Oral hygiene maintenance: Infection is unlikely in younger babies, unless they are compromised either immunologically or nutritionally. In older children, infection is seen especially with poor dental and oral hygiene.
- Postoperative antibiotic pack: The rate of fistula formation after primary palatoplasty is significantly reduced if a pack soaked with antibiotic cream is placed on the palate postoperatively.

How to Treat This Complication

It is preferred to wait until 4 years of age when objective examination with the help of nasopharyngoscope or videofluoroscope is possible for the management of fistula closure with velopharyngeal incompetence. A turn-over flap and transposition flap from buccal, vestibular, or facial regions can be used. It is preferable to use local palatal mucosa or mucoperiosteal tissue to replace the defect (Fig. 1.15d). The aim of surgical management of the fistula closures should be *two layered tension free closure.* For large fistulas local tissue, a tongue flap may be employed. The base of the flap should be at least half the width of the tongue or two thirds the width of the fistula to ensure a rich blood supply [17]. This flap is beneficial for recurrent and recalcitrant fistulas with extensively scarred tissues (Fig. 1.15e).

With extremely collapsed alveolar arches with large fistulas, orthodontic expansion should be performed followed by free tissue transfer. A radial forearm flap is mostly preferable since it is thin, freely mobile, mostly hairless with a long vascular pedicle. For multiple hard palate fistulas, complete redo-palatoplasty should be employed. Fistulas in soft palate and junction of hard and soft palate are mostly associated with VP dysfunction, a Furlow's double-opposing palatoplasty should be performed. It has the advantage of closing the fistula as well as lengthening the soft palate which helps to improve speech [16].

Velopharyngeal Incompetence

Velopharyngeal insufficiency (VPI) is described as the inability to close the velopharyngeal sphincter because of an anatomical or structural deficit during the production of sounds during speech. The inadequacy of speech and voice derived from velopharyngeal insufficiency is a major stigma for cleft palate patients.

Complete velopharyngeal closure effectively seals off the nasal cavity from the oral cavity, which is necessary for the production of all vowels and oral consonants. Patients with VPI present with nasal air escape and hypernasality during speech.

The evaluation of a patient with repaired cleft palate includes meticulous speech assessment by a trained speech therapist with familiarity of patients with cleft palate. Speech therapists assess the patient's intelligibility in speech, nasal emissions, and vowel and consonant production errors. Speech articulation errors are primarily addressed with the use of intensive and regular speech therapy. Hypernasality due to VPI requires surgical management.

How to Treat This Complication

The main aim of the VPI surgeries should be
- Separation of the oral and nasal components
- Adequate velar length
- Functional transverse orientation of the levator muscle sling

The velopharyngeal closure pattern and size of the velopharyngeal gap are the two important factors that guide the surgical treatment plan which includes:

- Pharyngeal flap
- Furlow's double-opposing Z-plasty palatoplasty
- Modified Furlow's Z-plasty (Fig. 1.16a)
- Buccal myomucosal flap (Fig. 1.16b)
- Combination of the above procedures

The most common age group for performing VPI surgeries is 4–6 years. Early interventions are associated with better speech outcomes. Fukushiro

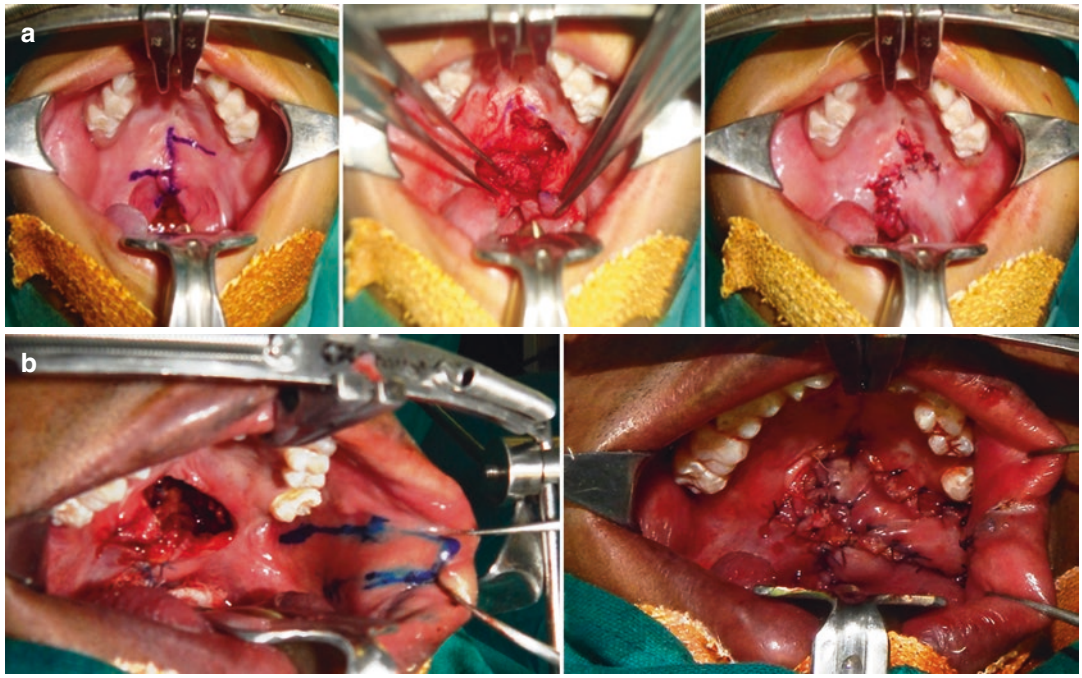


Fig. 1.16 (**a**) Modified Furlow's Z plasty. (**b**) Buccal myomucosal flap

and Trindade suggested that patients 6–12 years of age had better outcomes than their older counterparts [18]. Older children are unable to retrain their developed speech pattern after surgery. Surgeons and speech-language pathologists should counsel the patients and their parents on the increased risks associated with increasing age.

Maxillary Hypoplasia

Cleft lip and palate patients normally undergo surgical soft tissue repair of the cleft lip and palate during childhood. The resulting secondary deformities of the jaw and malocclusion are a consequence of early soft tissue repair of the cleft palate. It has been reported that 25–60% of cleft lip and palate patients need to undergo maxillary advancement to correct the resulting midface hypoplasia [19, 20]. Maxillary advancement in cleft lip and palate patients can be achieved using conventional Le Fort I osteotomy and rigid fixation or through distraction osteogenesis (DO). Each technique has its indications and advantages (Fig. 1.17).

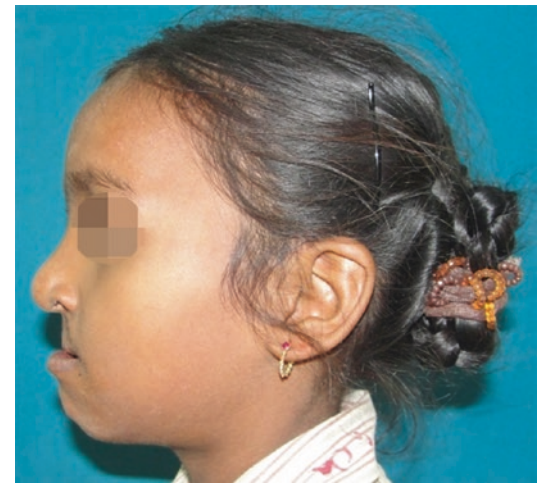


Fig. 1.17 Maxillary hypoplasia

As a result of severe maxillary hypoplasia, the mandible often undergoes autorotation in anterior and superior direction with subsequent over closure of the vertical dimension and a loss of facial height, pseudoprognathism, and upward inclination of the occlusal plane.

How to Treat This Complication
It can be managed by orthognathic surgery and intraoral or extraoral distraction.

Maxillary advancement using conventional Le Fort I osteotomy is an accepted treatment modality for the treatment of cleft maxilla. It is performed as a single-stage surgical procedure but has a high relapse tendency. A significantly high relapse rates are noted in cleft patients than non-cleft patients.

Intraoral distraction osteogenesis is a successful alternative technique in maxillary advancement in patients with cleft lip and palate who need an advancement of less than 10 mm with stable posterior occlusion. It produces improvements in the skeletal and soft profile. Also intraoral distraction devices do not have any psychological impact and have longer consolidation phases.

Extraoral distraction devices, i.e., rigid distraction devices, allow large advancement of the underlying skeletal foundation with bony regeneration and soft tissue distraction histiogenesis. This gives better stability for large advancements with severe palatal scarring. Newly formed bone noted in the pterygoid region after maxillary distraction reduces the risk of relapse. A consolidation period of 3 months is a must in order to ensure and preserve the bony formation in the pterygoid region.

Recurrent Ear Infections
The middle ear function may not improve with palatoplasty. Cleft palate teams need to follow up all such children beginning at birth and going into adulthood, decades after a "successful" palate repair.

1.4.3 Complications Associated with Alveolar Bone Grafting Surgery

1.4.3.1 At the Recipient Site

Intraoperative Complications

Perforation of Nasal Layer
Nasal layer perforations can occur during dissecting the nasal layer from the palatal mucosa.

How to Treat This Complication
Perforations if small should be sutured with a resorbable suture. Large perforations require placement of collagen sheet depending on the perforation site.

Damage to the Tooth/Tooth bud
Damage to the tooth/tooth buds can occur during the dissection procedure and closure. Hence a careful approach is required to prevent damage to the tooth buds.

Postoperative Complications

Graft Exposure/Failure
It can occur due to:
- Improper oral hygiene leading to wound dehiscence
- Large intersegmental gap which causes increased tension on the mucosal closure over the bone graft, risking postoperative wound dehiscence (Fig. 1.18)

How to Avoid This Complication
A releasing mucosal incision or scoring of the periosteum should be done to avoid tension on the mucosal closure. A periodontal pack can be placed after the completion of suturing of the wound.

1.4.3.2 At the Donor Site (Anterior Iliac Crest)

Intraoperative Complications

Bleeding
Cancellous bone bleeding can be controlled by hemostatic agents like Gelfoam and Surgicel.

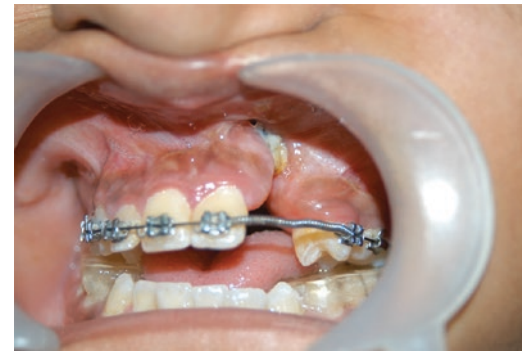


Fig. 1.18 Graft exposure

Fracture of Iliac Cortex

Fracture of iliac crest, a very unusual complication, has been reported in cases where there was injudicious use of osteotome and mallet leading to comminuted fracture and necessitating reduction and fixation.

Postoperative fracture is conservatively treated, with a period of bed rest followed by progressive ambulation. It is thought to mainly occur due to sudden contraction of the outer muscles in a weakened structure of the iliac crest.

Peritoneal Perforation

The use of a trephine to procure corticocancellous bone cores from the anterior iliac crest was found to carry a high risk of peritoneal perforation [21].

Injury to Inguinal Ligament

Inaccurate markings due to poor understanding of the anatomy and inadvertent use of curettes and bone gouges for removal of cancellous bone can cause inguinal ligament injury.

Injury to Ilioinguinal Nerve

The risk of nerve injury is significantly higher in cases in which graft deeper than 30 mm is harvested.

Postoperative Complications

Deep Hematoma

Hematoma formation results from inadequate intraoperative hemostasis, improper subperiosteal dissection, or cancellous bone bleeding. Significant deep hematomas have been reported when the layer-wise closure of the wound is improper. It can also occur due to strenuous activities post surgery. Water tight hermetic closure of Scarpa's fascia is required to prevent this complication.

Seroma

It is a collection of serous fluid that may develop after surgery in a dead space. The fluid contains blood plasma and inflammatory exudate which is derived from ruptured small blood vessels. Dead space elimination should be carried out with the help of external pressure dressing.

Infection and Wound Dehiscence

It can occur due to improper wound hygiene. Patient immunity and nutritional deficiency also play an important role in wound healing.

Meralgia Paresthetica

Meralgia paresthetica is described as a syndrome of dysesthesia or anesthesia in the distribution of the lateral femoral cutaneous nerve. Injury to the lateral femoral cutaneous nerve (Bernhardt's syndrome) or meralgia paresthetica occurs after harvest of the bone from the anterior iliac crest. The lateral femoral cutaneous nerve is a terminal sensory nerve that originates from L2 to L3 and innervates the skin of the thigh laterally.

How to Treat This Complication

Symptoms improve with occasional nerve blocks, analgesics, and desensitization. Some patients may require operative decompression or nerve sectioning.

Hernia

Herniation of the abdominal contents can occur through the donor site defect. Patients may present with lower abdominal pain and a sensation of fullness in the donor site. Computed tomography usually clearly shows the hernia.

How to Treat This Complication

Local tissue closure, Bosworth's method of crest resection and closure, rotation of regional fascial flaps, and the use of synthetic mesh grafts have all been suggested to treat these defects.

Gait Changes

For the morbidity of the donor site, the muscular attachments to the ilium play a significant role, when postoperative pain and gait disturbance are concerned. The reflection and retraction of the tensor fascia lata muscle seem to be the primary reasons for the increased morbidity observed with the anterior approach.

Foot Drop

It can occur due to peroneal nerve injury.

Cosmetic Defect

- **Hypertrophic scar**

  Tension-free skin closure along with meticulous multiple layer closure so that the skin edges sit in approximation or near approximation before subcuticular closure of the skin is important.
- **Pelvic rim defect/Contour defect**:

  It was assessed by palpation and graded as small/medium/large. Proper approximation of the cartilages is important to prevent this complication.

1.4.4 Complications Associated with Orthognathic Surgery

1.4.4.1 Intraoperative Complications

Hemorrhage

Severe bleeding can occur if the inferior alveolar, superior alveolar, maxillary, retromandibular, facial, and sublingual vessels become damaged. Bleeding can be stopped by applying pressure, using bone wax or resorbable hemostatic materials, or by performing thrombin or epinephrine impregnated gauze packing or electrocautery. Nasal epistaxis can occur after Le Fort I osteotomy and must be controlled by performing one of the following procedures:

- Anterior and/or posterior nasal packing, packing of the maxillary antrum
- Reoperation with clipping or electrocoagulation of bleeding vessels
- Application of hemostatic agents in the pterygomaxillary region
- External carotid artery ligation
- Selective embolization of the maxillary artery

However, these hemostatic therapies can cause the aseptic necrosis. The severity of intraoperative bleeding and the possibility of developing complications vary from patient to patient. For this reason, it is important to measure the relative blood loss during intraoperative bleeding by using a patient specific measure. Extensive surgery and reduced body mass index are associated with increased intraoperative relative blood loss.

How to Avoid This Complication

Hypotensive anesthesia using sevoflurane and propofol can effectively reduce bleeding and secure excellent vision during surgery. Injection tranexamic acid given intravenously or applied topically is found to significantly reduce intraoperative blood loss.

How to Treat This Complication

Bleeding is occasionally heavier, and surgeons should be prepared for heavier bleeding by reserving blood at a blood bank or by preparing an autotransfusion.

Bad Split/Fracture

Unfavorable fracture lines can cause fragmentation of the bones in the maxilla and may produce difficulty in fixation and may also permeate through to the skull base producing deleterious effects like blindness. The chances for a bad split is more common in a Le Fort I osteotomy when the conventional lines are modified to include the infra-orbital rim or zygoma where the osteotomy may have to be blind behind the zygoma.

How to Avoid This Complication

It is recommended to preplate the maxilla across the previous alveolar cleft to maintain it as a single unit prior to osteotomy in cleft cases. An arch bar or a heavy wire can be incorporated into the surgical splint and fixed to the maxilla.

Nerve Injury

Sensory nerve deficit following orthognathic surgery may be transient or permanent. Permanent sensory deficit is certainly an undesirable outcome, which has to be discussed with the patient and explained in detail. An informed consent explaining the expected complications and their consequences would allow a better understanding of the problems by the patient and prevent a postsurgical embarrassment.

How to Avoid This Complication
The infraorbital nerve is mostly at risk of injury from retraction of the soft tissue during maxillary osteotomy, although it can be injured directly during higher osteotomies or from injudicious placement of fixation. Careful retraction of the mucoperiosteal flap with the help of retractors over the infraorbital nerves is important. A wet gauze should be kept over the infraorbital nerves before retraction.

How to Treat This Complication
Steroids, anti-inflammatory drugs, and Vitamin B12 supplements have been found to be useful.

Damage to the Tooth
Transient or persistent dental hypersensitivity is a common problem associated with post-osteotomy discomfort. Patients may also exhibit obliteration of the pulp canal, which is more prevalent in the cleft maxilla. Spontaneous pulpal necrosis or internal resorption of the tooth is not common. The transient ischemia following an osteotomy for the first 2 days followed by increased pulpal blood flow may be the cause of the hyperemia and sensitivity.

How to Avoid This Complication
At least 5 mm of the distance should be maintained between the tooth roots and the osteotomy cuts to prevent devitalization of the teeth.

How to Treat This Complication
Postoperative endodontic treatment is advised if the teeth are devitalized.

1.4.4.2 Postoperative Complications

Vascular Compromise: Complete or Partial Necrosis
Complete or partial necrosis of the osteotomized segment due to vascular compromise has been reported and has been attributed more predominantly to cases requiring multisegment osteotomies in conjunction with superior repositioning and or transverse discrepancy corrections. Partial necrosis of the anterior maxilla after an anterior maxillary osteotomy has also been reported. The occurrence of palatal perforations also compromises the vascularity of the anterior maxilla. Other factors contributing to the vascular compromise are type of incision, ligature of the descending palatine arteries, amount of bone-teeth repositioning, lesion of the vascular pedicle, and hypotensive anesthesia.

How to Avoid This Complication
Modified incisions maintaining the additional pedicle in the anterior region, and preservation of the descending palatine artery.

How to Treat This Complication
Treatment involves conservative debridement, hyperbaric oxygen, reconstruction with free grafts, or microvascular flaps depending upon the size of the defect. This is followed by placement of dental implants and prosthetic rehabilitation.

Palatal Perforation
Palatal osteotomy is usually performed with a curved osteotome or a saw placed through the horizontal osteotomy site, with the surgeon's finger on the palatal tissues to ensure complete osseous sectioning gauging and minimizing palatal perforation.

Malunion/Nonunion
Nonunion or delayed union has an incidence of 2.6% [22]. The main causes of non- or delayed union are instability in dental occlusion, postsurgical infections, or osteosynthesis failures. Investigations with a 3D-computed tomography reconstruction are always superior to conventional radiography in making a diagnosis of nonunion.

How to Avoid This Complication
Accurate preoperative diagnosis and proper anatomical reduction of the osteotomized segments are essential to preventing postsurgical malunion and malocclusion.

How to Treat This Complication
Nonunion requires secondary surgery with curettage, bone grafting and adequate rigid fixation is required as a corrective measure. Malunion

requires reosteotomy and realignment followed by rigid fixation.

Malocclusion

It can occur due to:

- Relapse at the Le Fort I osteotomy site
- Fibrous union at the Le Fort I osteotomy site
- Relapse at the segmental osteotomy sites
- Continued mandibular growth
- Unstable orthodontic tooth positioning

Relapse

No orthognathic procedure is relapse proof, and when the surgical procedure is not planned, modified, or over-corrected with this in mind, it may lead to an undesirable result in due course. Relapse is related to the magnitude of surgical advancement, nonrigid type of fixation, palatal scarring, tightness of upper lip, and interference with nasal septum and presence or absence of pharyngeal flaps [23]. The concept of stability can be understood better in terms of percentage changes anticipated, which will enable us to plan in countering relapse.

- **Highly stable:** Lesser than 10% chance of significant postoperative change
- **Stable:** Less than 20% chance of significant change and almost no chance of major change postoperatively
- **Stable with modification:** E.g., rigid fixation after osteotomy
- **Problematic:** Considerable chances for major postoperative changes [22]

Relapse after Le Fort I osteotomy is mainly related to:

- Inadequate mobilization of the down-fractured maxilla before the placement of fixation.
- Inadequate bone contact across the osteotomy site after the fixation is applied.
- Inadequate (nonrigid) plate and screw fixation across the osteotomy site or
- Excessive occlusal forces or movement transmitted across the osteotomy site during initial healing.
- Special attention should be paid to the patient with bilateral cleft, multiple missing teeth, and shallow postoperative overbite.

A clear understanding of the stability and planning accordingly will reduce the incidence of postsurgical problems. Two movements fall in the problematic category, viz. maxillary downgraft and maxillary expansion. Knowledge, counseling, and awareness of these help us to achieve better results and counsel the patients on what to anticipate.

How to Avoid This Complication

Relapse can be prevented by

- Preoperative alignment of the arch and leveling of the teeth
- Sufficient mobilization of the osteotomized segments intraoperatively
- Use of interpositional bone grafts when the amount of the advancement is more
- Rigid skeletal fixation
- IMF for a variable period of time

How to Treat This Complication

It may require a secondary surgical procedure in the form of external/internal maxillary distraction or anterior maxillary distraction.

Temporomandibular Joint Effects

TMJ problems arise due to changes in occlusion, mandible position, and when there are pre-existing TMJ problems. Treatment of pre-existing TMJ disorder prior to orthognathic surgery helps in achieving stable results.

Sensory Impairment

Sensory innervation of the area affected by the Le Fort I osteotomy is mainly through the maxillary division of the trigeminal nerve. During the Le Fort I osteotomy, the superior alveolar nerves on each side are transected as part of the osteotomy and the terminal labial branches of the infraorbital nerve at the time of Le Fort I osteotomy that results in temporary paresthesia.

Cranial Nerve Palsies/Cavernous Sinus Thrombosis

Cranial nerve damage is one of the serious complications that can occur after Le Fort I osteotomy. Cranial nerve damage after the Le Fort I osteotomy is related to direct or indirect injury. CN VI crosses the cavernous sinus close to the wall of the sphenoid sinus, enters the superior orbital fissure, and innervates the lateral rectus muscle and is thus the most likely nerve to be injured if a comminuted fracture of the sphenoid bone occurs. In the cavernous sinus, CN V_1 and V_2 are located lateral proximity to the CN VI. Indirect injuries to the cranial nerves, such as contusion or force applied during the osteotomy, might result in the ischemia of a nutrient artery for the cranial nerves. Cranial nerve palsies can occur due to unexpected sphenoid bone fracture and subsequent trauma in the cavernous sinus during pterygomaxillary disjunction [24].

How to Treat This Complication

The treatment comprises of empirical antibiotic therapy, corticosteroids, and continuous monitoring.

Blindness

Partial or complete/orbital compartment syndrome or blindness after Le Fort I surgery has been reported due to:

- Arterial aneurysm
- Induced hypotension
- Hypoperfusion of the optic nerve
- Propogation of the fracture lines to the skull base during pterygomaxillary disjunction
- Unknown (Fig. 1.19a–c)

Le Fort I osteotomy should be performed with extreme care, and the informed consent should include visual loss as a complication of the procedure. It is also suggested that all patients undergoing Le Fort osteotomies undergo a preoperative ophthalmic assessment of vision.

Maxillary Aneurysm

Pseudoaneurysm (PA), also known as false aneurysm, is an incomplete injury of the blood vessel, with the rupture of one or more layers of the vessel wall rather than the expansion of all layers of the vessel, as occurs in a true aneurysm. Once the hematoma is formed, the onset of the PA occurs when the blood flow between the arterial laceration and hematoma is maintained.

The development of an aneurysm is determined by the degree of vascular injury, blood flow, and elasticity of the neighboring tissues. CT and MRI are useful aids in the diagnosis of maxillary aneurysms. The formation of an aneurysm beginning with the initial injury until the appearance of a pulsating mass takes between 1 and 8 weeks.

Various treatment modalities are:

- Nonsurgical – Transcatheter interventions, viz. embolization, stents, stent grafts
- Surgical – Excision of the aneurysmal sac following external carotid ligation

Avascular Necrosis

Complete or partial necrosis of the osteotomized segment due to vascular compromise has been reported more in the maxilla and has been attributed more predominantly to cases requiring multisegment osteotomies in conjunction with superior repositioning and or transverse discrepancy corrections. The occurrence of palatal perforations also compromises the vascularity of the anterior maxilla.

How to Avoid This Complication

- Divide into as few segments as possible and avoid small segments anteriorly.
- Maintain the integrity of the palatal mucosa.
- Perform sagittal segmentation in paramedian sites as the mucosa is thicker and the bone thinner than the midline.

How to Treat This Complication

- Local flaps for small defects
- Pedicled regional flaps
- Microvascular free tissue transfers, viz. radial forearm flap, free fibula flap.
- Prosthetic maxillary obturators

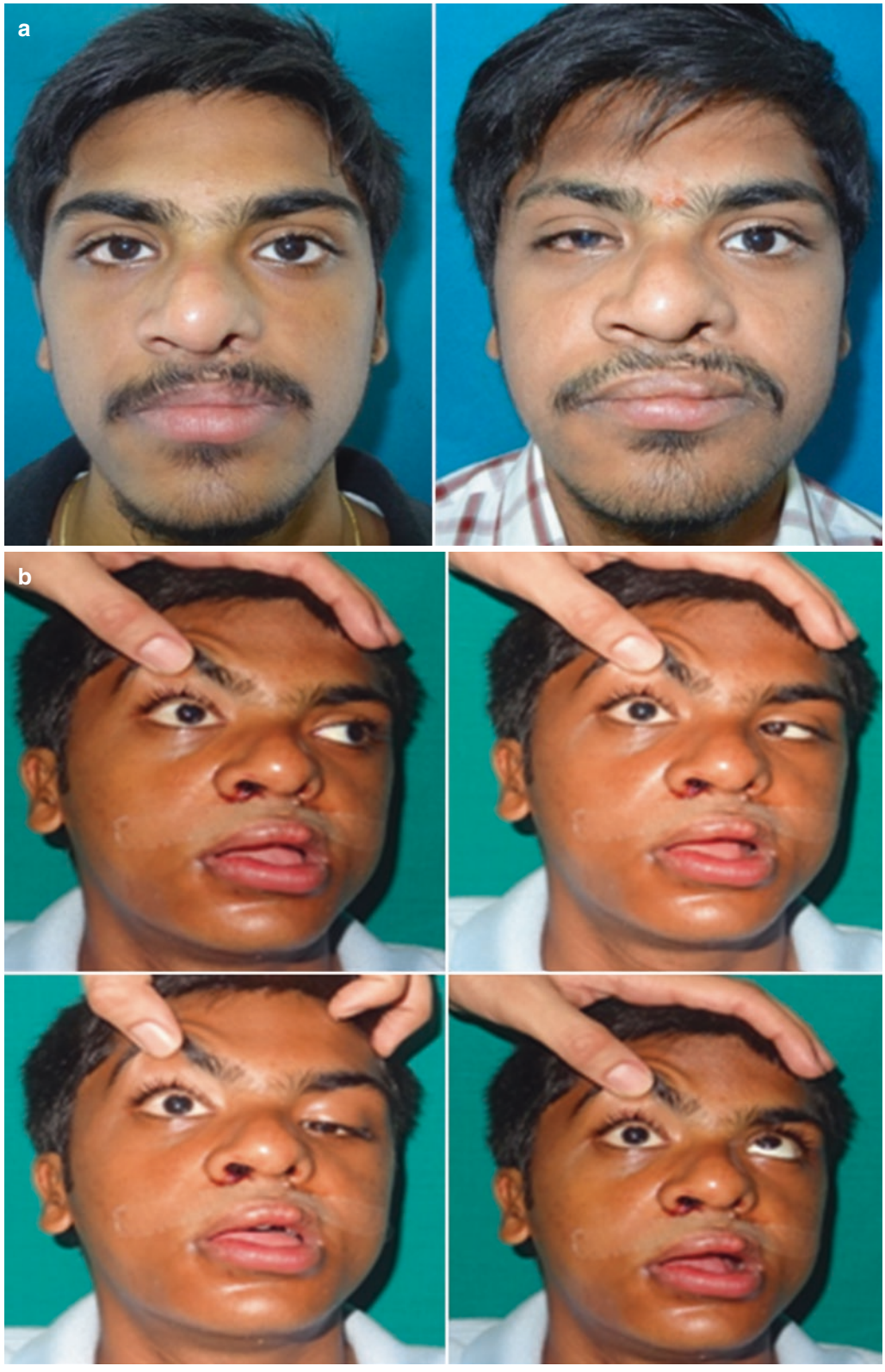


Fig. 1.19 (**a**) Orbital compartment syndrome drooping of eyelids. (**b**) Lack of eye movements. (**c**) Blindness

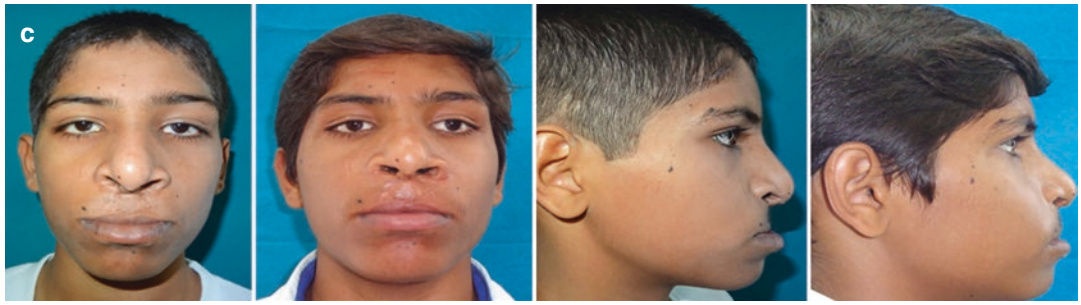


Fig. 1.19 (continued)

Velopharyngeal Insufficiency (VPI)

Many patients with cleft lip and palate who undergo orthognathic surgery have previously had speech surgery to correct velopharyngeal insufficiency (VPI), and, if not, they are certain to have spent time with a speech pathologist for normal speech. The mechanism that contributes to VPI includes the failure of the velum to obstruct nasal air flow by elevating against the posterior pharynx. It is understood that, if a surgeon advances the maxilla, this mechanism may be disrupted regardless of whether the patient has had a pharyngeal flap or not; however, this is not the case. Cephalometric assessment of the velum and pharynx may aid in predicting which patients may need a pharyngeal flap after surgery. Another treatment option in maxillary advancement is distraction osteogenesis (DO). It was once thought that DO may provide patients with time to adapt to the skeletal changes, and therefore show a decreased chance of velopharyngeal deterioration. However, it has been concluded that DO has no advantage compared with traditional procedures for the purpose of preventing VPI. VPI and the effects of maxillary advancements should be discussed with the patient and family before surgery.

Studies have indicated that there is an alteration in the anatomy and functioning of the velopharyngeal apparatus after a total maxillary osteotomy and this may not be very different between cleft and noncleft patients. However, the magnitude of the change may be different in the two groups, which may need consideration during advancements of the maxilla in cleft patients. The presence of a short soft palate and a deep pharynx contribute to the development of a velopharyngeal insufficiency following Le Fort 1 osteotomy. Patient and their parents should be counseled about the requirement of any further measures, viz. surgical or nonsurgical for correction of VPI.

1.4.5 Complications Associated with Distraction Osteogenesis

1.4.5.1 Intraoperative Complications

The following have been discussed in section orthognathic surgery

1. Hemorrhage
2. Bad split/fracture
3. Nerve injury
4. Damage to the tooth buds

1.4.5.2 Postoperative Complications

Intradistraction

Pin infections, Pin and Device Loosening

Pin loosening occurs when one or more halo-fixation pins could be freely twisted without resistance or tip visibility at the edge of the skin [25]. Pin track infection decreases the stability of the pin–bone interface. Conversely, instability of the fixator pin–bone construct can lead to half-pin loosening and infection. It is a common misconception that pin loosening only results from pin track infection whereas in actual fact pin loosening is often the initiating event resulting in pin track sepsis. Hence it is essential to monitor

the looseness of the screws during every distraction schedule.

How to Avoid This Complication

A topical antibiotic dressing and proper hygiene is essential to prevent this complication.

Device Failure

Device failure can occur due to mechanical errors in distraction screws or accidental trauma to the halo frame. A familiarity with the components of the device and clear understanding of their function are essential. Any component of the distraction device can experience hardware failure during the placement, activation, and consolidation phase of distraction.

Inappropriate Distraction Vector/Frame Migration

Instability of the frame during the distraction period in craniofacial patients might impair DO and thus functional and aesthetic outcome (Fig. 1.20).

How to Avoid This Complication

Accurate fixation of the distractor is very important step for the vector control.

Premature Consolidation

It has been reported in the range of 1.9–7.6% [26]. This can occur if the latency period is too long or the distraction rate is too sluggish.

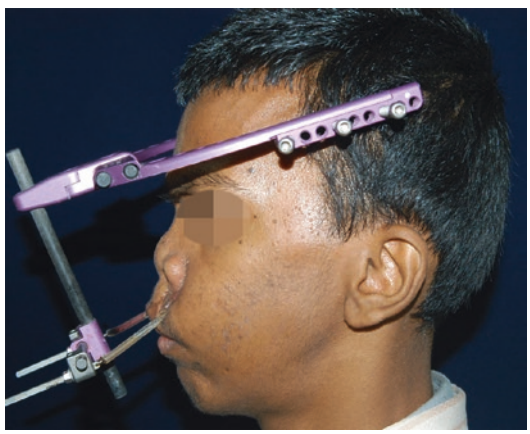


Fig. 1.20 Inappropriate distraction vector and frames

Coronoid Process Interference

Coronoid process can interfere with the skull base and prohibit elongation of the proximal segment. A preoperative planning with the help of CT scans and analysis of the movements that recognizes interferences and collisions are useful.

How to Avoid This Complication

A coronoidectomy can be planned prior to the distraction device placement to avoid anatomic interferences.

Fibrous Pseudoarthrosis

This complication can occur if the rate of distraction is too fast. The rate of distraction depends on the age of the patient, area of distraction (cortical or alveolar), and the type of distraction.

Trismus

Trismus occurs due to prolonged intermaxillary fixation during consolidation period. It can be relieved by regular jaw physiotherapy.

Post Distraction

1. **Delayed Consolidation**
   It can occur due to too rapid rates of distraction.
2. **Premature Consolidation**
   It can occur due to too sluggish rates of distraction and long latency period.
3. **Malocclusion**
   Minor malocclusions can be corrected by postoperative orthodontics.
4. **Growth Disturbances**
   It should be noted that forward growth of the maxilla ceases after the distraction in most cases.
   Two factors that might prevent forward growth of the maxilla following the distraction are as follows:
   - The pterygomaxillary junction is an important growth site and is in the line of the osteotomy cut and the region of new bone formation during distraction. Forward growth of the maxilla may be affected adversely due to disturbance of the growth site or obliteration of this sutural junction.

- Pressure from the soft tissue and musculature, which may have a longer-lasting effect on the maxillary growth.

1.4.6 Complications Associated with Rhinoplasty

1.4.6.1 Intraop Complications

Bleeding

Adequate hemostatic measures are required to control intraoperative bleeding. Bleeding reduces the visibility, increased operating difficulty, and operating time. Any genetic or acquired coagulopathy should be investigated prior to the surgery.

How to Avoid This Complication

Reverse Trendelenburg position (15° angle) reduces intraoperative bleeding in rhinoplasty patients while facilitating the procedure compared to the supine position**.** A single bolus dose of intravenous tranexamic acid (10 mg/kg) upon induction of anesthesia is found to achieve satisfactory hemostasis in patients of open rhinoplasty.

Cartilage Fracture (Dorsal Caudal L Structure)

Loss of dorsal support due to fracture of the L frame. This can occur when the cartilage is inherently weak. The extended septal graft which is harvested from the septum can fracture due to improper handling of the cartilage or inherent weakness.

How to Avoid This Complication

An adequate dorsal-caudal L structure of at least 1 cm is necessary for structural support to prevent this type of complication.

Buttonholing of Skin

This complication can occur during undermining of skin, particularly over the dome region and is best avoided by prudent technique. Buttonholing may lead to scarring.

How to Treat This Complication

Nontension suturing should be done if buttonholing occurs.

Perichondrium Tear

This can be prevented by meticulous dissection technique. It is mostly associated with noses which have undergone previous surgery. Unilateral tears heal uneventfully, but bilateral tears may result in septal perforation. Tears in septum and lateral wall may lead to synechiae formation (adhesions), which may require subsequent release.

1.4.6.2 Immediate Postoperative Complications

Transient Numbness and Pain

Numbness in the tip of the nose, upper front teeth, or roof of the mouth following surgery is to be expected because rhinoplasty surgery typically causes a temporary disruption of some of the nerves in the area. Sensation will generally return over a period of several weeks, and rarely, months.

Hematoma

This complication occurs due to bleeding in the septal compartment. This complication can go unnoticed in the immediate postop period. Thus, examination of the nostrils must be systematic at the time of removal of the splint and nasal packing. In case of hematoma, it should be evacuated and compressive dressing should be given. In the absence of treatment, infection caused by this hematoma results in perichondritis followed by perforation of the nasal septum. A tight transseptal suture is required to close the dead space and further prevent this complication which can lead to septal perforation.

Infection and Wound Dehiscence

Postoperative infections after rhinoplasty are rare due to good vascular supply to the nose. This complication can be prevented by possible blood evacuation and removal of bony spicules and debris before packing. Large hematomas and prolonged packing are also a concern for infections.

CSF Rhinorrhea

It is a dreaded complication of rhinoplasty. Trauma to the septum during dorsal hump removal or lateral osteotomy to narrow the nose may inadvertently result in cribriform plate fracture at its junction with perpendicular plate. Also imprudent use of bony septum as a fulcrum for the osteotome may cause fracture of the cribriform plate and collapse of the nasal dorsum. Treatment is required for prevention of potentially fatal infections, meningitis, cerebral abscess, septic cavernous sinus.

How to Treat This Complication

Most CSF leaks heal spontaneously. Persisting leaks need localization and repair by extracranial and/or intracranial techniques.

Olfactory Disturbances

The olfactory function shows a temporary decline following open rhinoplasty but tends to revert to its preoperative levels 6 months post surgery. Documentation of the preoperative olfactory function before surgery is important.

1.4.6.3 Late Postoperative Complications

Columellar Skin Necrosis

This complication is more common with external approach than closed approach and mainly occurs due to compromised vascular supply to the columellar skin. It appears after damage in the lateral nasal arteries due to an incorrect plane of dissection or following excessive nose tip fat tissue reduction, in an attempt to reduce its size (Fig. 1.21).

How to Avoid This Complication

- Dissect the nasal tissues attached to the cartilaginous framework without getting into the superficial plane.
- Avoid defatting techniques of the nose tip or reduce it to a minimum.
- Avoid firm and tight dressings, especially in revision cases.
- Limit alar wedge resections under the alar crease.

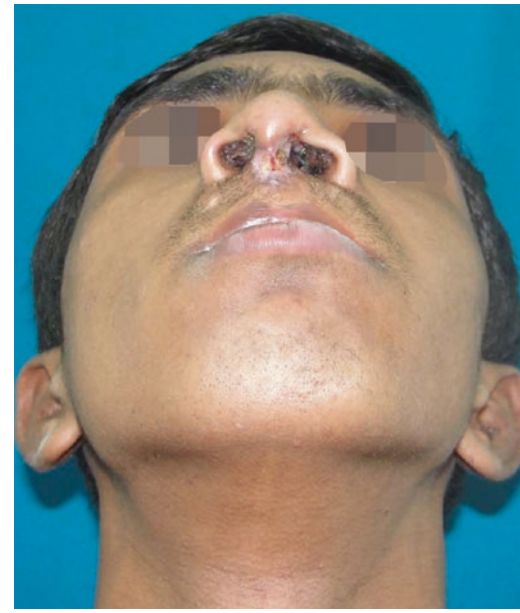


Fig. 1.21 Columellar skin necrosis

How to Treat This Complication

Treatment of skin necrosis is complex and can range from conservative approaches such as second-intention wound healing to complex reconstruction procedures with local, regional, or free flaps. Whatever the approach, skin tropism and elasticity are a primary goal before intending more complex repair. This can be achieved through platelet-rich plasma and micro-fat filling sessions.

Graft/Implant Migration

Graft migration mainly occurs with tip grafts and dorsal augmentation grafts.

How to Avoid This Complication

The longest lasting suture material should be utilized for graft fixation, unless other factors are of overriding importance.

How to Treat This Complication

This may require a repeat procedure if graft fixes in abnormal location.

Nasal Stenosis/Blockage

Nasal vestibule contracture, resulting from either the primary repair or revisions, is a difficult problem encountered after rhinoplasty. Scaring can

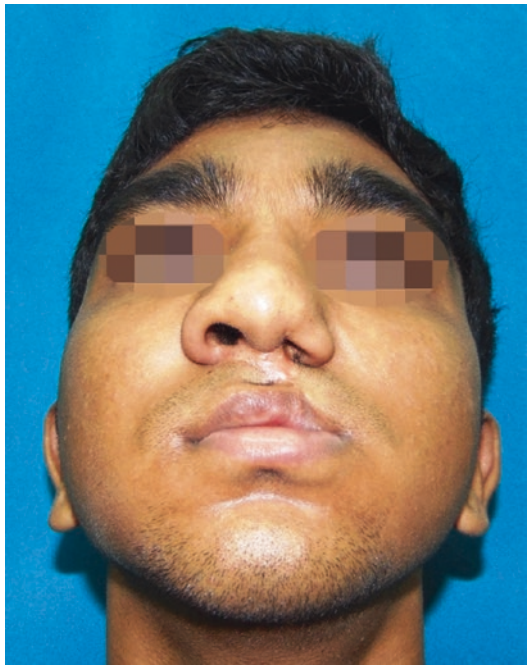


Fig. 1.22 Nasal stenosis

lead to nostril stenosis and collapse of the external nasal valve. *Micronostril is a very difficult problem to correct, and the creation of this deformity during primary repair should be recognized and avoided at all costs* (Fig. 1.22).

How to Treat This Complication

Local tissue flaps, viz. perialar nasolabial flap, can help reconstruct this and disrupt the constrictive forces in the external nasal valve. Redundant skin can also be used to line a stenotic nasal vestibule. If there is significant stenosis of the nostril and loss of tissue along the nostril sill, the nasal alae should be repositioned.

Septal Perforation

The etiology of septal perforation is diverse and may be iatrogenic. With subperichondrial septum dissection, caution should be taken not to trespass the mucoperichondrial flaps. If mucosal flaps are damaged bilaterally, an iatrogenic septal perforation is bound to be produced. It can also occur due to formation of hematoma between the septal cartilage and perichondrium.

How to Treat This Complication

Meticulous dissection of the septal perichondrium, taking care not to breach the continuity of the mucoperichondrial flap, should be performed. Septal perforation which forms due to hematoma formation can be prevented by tight transseptal sutures and an antibiotic nasal pack for 5 days post surgery.

How to Treat This Complication

Septal perforations can be treated by advanced flap, a rotation flap, or a combination of both flaps, depending on the size and location of the perforation.

Nasal Valve Collapses

Valve problems are very often caused by the separation of the upper lateral cartilages from the septum. Deep osteotomies can narrow the airways at the piriform aperture. Excessive alar cartilage resections can also cause alar collapse. Excessive removal of mucosa in the valve area or an improperly placed incision can produce stenosis and nasal obstruction.

How to Treat This Complication

Internal nasal valve collapse can be treated with spreader grafts, alar batten grafts, or lateral crural strut grafts. These involve removal of cartilage from either the nasal septum or ear and adding strength to the nose to prevent it from collapsing inwards.

Poor Hypertrophic Scar

A retracted columella scar after open rhinoplasty can occur because of the following:

- Inadequate cartilage support
- Linear skin incision (Fig. 1.23)

1.4.7 Complications Associated with Syndromic Patients

1.4.7.1 Pierre Robin Syndrome

It includes a triad of retrognathism, glossoptosis, and a median palatal cleft.

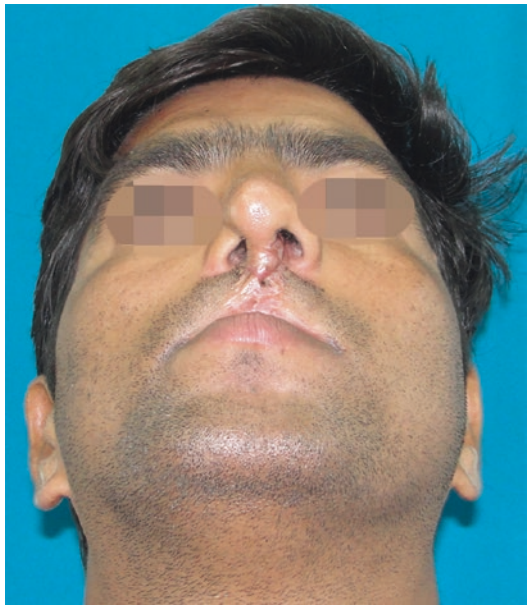


Fig. 1.23 Poor hypertrophic scar

The complications associated with it are:

- Airway obstruction with recurrent episodes of cyanosis
- Feeding difficulties
- Malnutrition
- Pulmonary hypertension
- Congestive cardiac failure
- Cerebral hypoxia
- Positioning the child upright will resolve the airway obstruction in ~70% of cases. In the correct position, most children will also be able to feed normally.

  The two most common surgical procedures for treatment include tongue–lip adhesion and distraction osteogenesis of the mandible.

1.4.7.2 Velocardiofacial Syndrome

It is one of the most common multiple anomaly syndromes in humans. It is associated with congenital heart disease (70%), immune disorders, feeding problems, cleft palate, and developmental disorders.

Complications associated with it are:

- Multiple abnormalities of the heart including ventricular septal defect (VSD), pulmonary atresia, tetralogy of Fallot, truncus arteriosus, interrupted or right-sided aortic arch and transposition of the great arteries
- Learning disabilities in one or more areas
- Hearing loss due to recurrent otitis media
- Speech problems
- Behavior problems, including anxiety, ADHD, and depression

Depending on the presence and severity of various features, a child with VCFS might need one or more of the following surgeries:

- Repair of heart defects
- Repair of cleft palate
- Psychological evaluation and therapy

1.5 Conclusion

Complications are inherent in surgical practice. Key to the management of surgical complications is an understanding of the cause, arising from issues related to surgeon's knowledge of medical literature, surgical technique, and surgical judgment. In cleft lip and palate surgeries, one must carefully choose a technique depending upon the patient needs, master it and follow up the cases for a reasonably long period of time, and be ready to learn from mistakes.

References

1. Gunawardana R. Difficult laryngoscopy in cleft lip and palate surgery. Br J Anaesth. 1996;76(6):757–9.
2. Kulkarni K, Patil M, Shirke A, Jadhav S. Perioperative respiratory complications in cleft lip and palate repairs: an audit of 1000 cases under ‘Smile Train Project’. Indian J Anaesth. 2013;57(6):562.
3. Ahmad M, Malik SA. A novel tool to prevent endotracheal tube compression in cleft palate surgery. Iran J Med Sci. 2011;36(3):231–2.
4. Rajan S, Halemani R. Are active warming measures required during paediatric cleft surgeries? Indian J Anaesth. 2013;57(4):377.
5. Adeyemo WL, et al. Frequency of homologous blood transfusion in patients undergoing cleft lip and palate surgery. Indian J Plast Surg. 2010;43(1):54–9.
6. Mulliken J, Martínez-Pérez D. The principle of rotation advancement for repair of unilateral complete

cleft lip and nasal deformity: technical variations and analysis of results. Plast Reconstr Surg. 1999;104(5):1247–460.
7. Cutting C, Dayan J. Lip height and lip width after extended mohler unilateral cleft lip repair. Plast Reconstr Surg. 2003;111(1):17–23.
8. Yeow V, Chen P, Chen Y, Noordhoff S. The use of nasal splints in the primary management of unilateral cleft nasal deformity. Plast Reconstr Surg. 1999;103(5):1347–54.
9. Mcgregor J, Kelley P, Gruss J, Khosla R. Contemporary concepts for the bilateral cleft lip and nasal repair. Semin Plast Surg. 2013;26(04):156–63.
10. Sharma R, Nanda V. Problems of middle ear and hearing in cleft children. Indian J Plast Surg. 2009;42(3):144.
11. Mizuno K, Ueda A, Kani K, Kawamura H. Feeding behaviour of infants with cleft lip and palate. Acta Paediatr. 2007;91(11):1227–32.
12. Endriga MC, Speltz ML, Maris CL, Jones K. Feeding and attachment in infants with and without orofacial clefts. Infant Behav Dev. 1998;21(4):699–712.
13. Ize-Iyamu I, Saheeb B. Feeding intervention in cleft lip and palate babies: a practical approach to feeding efficiency and weight gain. Int J Oral Maxillofac Surg. 2011;40(9):916–9.
14. Marques IL, Nackashi J, et al. Longitudinal study of growth of children with unilateral cleft lip and palate: 2 to 10 years of age. Cleft Palate Craniofac J. 2015;52(2):192–7.
15. Agrawal K, Panda KN. An innovative management of detached palatal mucoperiosteal flap from the hard palate (hanging palate). Plast Reconstr Surg. 2005;115(3):875–9.
16. Diah E, Lo LJ, Yun C, Wang R, Wahyuni LK, Chen YR. Cleft oronasal fistula: a review of treatment results and a surgical management algorithm proposal. ChangGung Med J. 2007;30(6):529–37.
17. Ogle OE. The management of oronasal fistulas in the cleft palate patient. Oral Maxillofac Surg Clin North Am. 2002;14(4):553–62.
18. Fukushiro AP, Trindade IE. Nasometric and aerodynamic outcome analysis of pharyngeal flap surgery for the management of velopharyngeal insufficiency. J Craniofac Surg. 2011;22(5):1647–51.
19. Rachmiel A. Treatment of maxillary cleft palate: distraction osteogenesis versus orthognathic surgery—part 1. Maxillary distraction. J Oral Maxillofac Surg. 2007;65(4):753–7.
20. Panula K, Lovius BB, Pospisil OA. The need for orthognathic surgery in patients born with complete cleft palate or complete unilateral cleft lip and palate. Oral Surg Oral Diagn. 1993;4:23–8.
21. Caminiti MF, Sándor GK, Carmichael RP. Quantification of bone harvested from the iliac crest using a power-driven trephine. J Oral Maxillofac Surg. 1999;57(7):801–5.
22. Bonanthaya K, Anantanarayanan P. Unfavourable outcomes in orthognathic surgery. Indian J Plast Surg. 2013;46(2):183.
23. Painatt J, Veeraraghavan R. Profile changes and stability following distraction osteogenesis with rigid external distraction in adult cleft lip and palate deformities. Contemp Clin Dent. 2017;8(2):236.
24. Kim J, Chin B, Park H, Lee S. Cranial nerve injury after Le Fort I osteotomy. Int J Oral Maxillofac Surg. 2011;40(3):327–9.
25. Nout E, Wolvius E. Complications in maxillary distraction using the RED II device: a retrospective analysis of 21 patients. Int J Oral Maxillofac Surg. 2006;35(10):897–902.
26. Swennen G, Schliephake H. Craniofacial distraction osteogenesis: a review of the literature. Part 1. Clinical studies. Int J Oral Maxillofac Surg. 2001;30(2):89–103.

2 Complications in Craniofacial Surgery

Samuel Liu and Bernard J. Costello

Contents

S. Liu
Fellow in Pediatric Craniomaxillofacial Surgery, Department of Oral and Maxillofacial Surgery, University of Pittsburgh, Pittsburgh, PA, USA
e-mail: lius9@upmc.edu

B. J. Costello (✉)
Dean and Thomas W. Braun Endowed Professor, Department of Oral and Maxillofacial Surgery, Children's Hospital of the University of Pittsburgh Medical Center, University of Pittsburgh School of Dental Medicine, Pittsburgh, PA, USA
e-mail: bjc1@pitt.edu

2.1 Introduction

The most appropriate definition of craniofacial surgery describes those procedures that traverse the craniofacial barrier, rather than procedures in the general craniofacial region, such as orthognathic procedures or rhinoplasty. Transcranial craniofacial operations are needed in pediatric patients due to functional and aesthetic issues

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_2

associated with congenital and acquired deformities of the craniomaxillofacial complex. Many of these procedures have significant risk associated with them. Surgical intervention for craniosynostosis is one such indication which requires a reconfiguring of dysmorphic craniofacial tissues into a more normal confirmation that is more aesthetic and provides improved volume for functional growth of the brain. Complications are mostly rare and include significant blood loss, infection, blindness, embolism, disability, or even death. Figures show that mortality in craniofacial surgery can range from 0.1% to up to 2.7% [1].

There is evidence that suggests that children born with single- or multiple-suture synostosis may suffer from increased intracranial pressure and papilledema that may eventually lead to developmental abnormalities or blindness if not treated [2]. However, for single-suture synostosis, there is minimal compelling research that is able to clearly and definitively link its presence on a consistent basis with deleterious effects. In individuals with craniosynostosis, the cranium is able to expand via the unaffected sutures in a fashion described by Virchow's law, where compensatory growth is created perpendicular to the fused suture, thereby creating an abnormal head shape [3, 4]. Craniofacial surgeons must then decide whether the cosmetic benefit and presumed functional benefit of expansion of the volume early in the growth phase of this highly invasive correction procedure is worth the concomitant surgical risk—which can be substantial. Add a few associated comorbidities that tend to be present in patients with syndromes, and the perioperative management can become highly complicated and complex. In this chapter, we review the risks and complications associated with transcranial procedures in children, mostly used to treat craniosynostosis, craniofacial dysostoses, craniofacial trauma, and various other craniofacial syndromes.

Another key element in avoiding complications is being aware and proactive in the team's thought process. The entire clinical care team should have an anticipatory attitude that problems are always possible, and a culture that allows anyone in the room to speak up should they feel that an issue may be present. A "level playing field" is a key aspect of team care, and an operating room team will perform better if issues are raised verbally with a culture of respect and openness. This is also true for the perioperative team.

2.1.1 Timing of Pediatric Craniofacial Procedures

The timing of craniofacial procedures depends on many factors including the growth potential of the structures involved, associated visceral tissue considerations, and the possibility of late growth restriction [5, 6]. Cranio-orbital growth is mostly complete by 1–2 years. Midfacial and mandibular growth plateau much later, and these procedures are usually delayed until the teen years for this reason. Procedures are typically timed for the plateau phase of growth of the area of concern (Fig. 2.1).

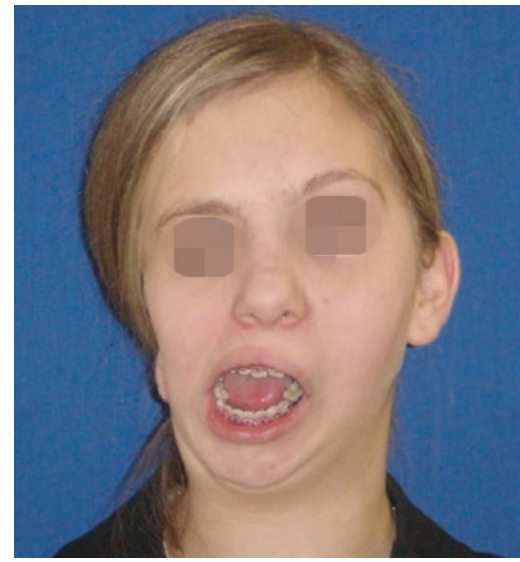


Fig. 2.1 A 16-year-old female with oculoauriculovertebral dysplasia (OAVS) with effects across the entire craniomaxillofacial skeleton, including cranial vault asymmetry, orbital dystopia, and maxillomandibular asymmetry that is highly complex. Timing of each intervention should consider the growth curves of the cranio-orbital and maxillofacial complexes separately, as they plateau at different times. This can dictate a more ideal timing of intervention based on the maximum amount of growth being finished prior to intervention

We constantly balance the needs for intervention against the potential biological consequences of intervening in growing tissues, which often will yield growth restriction later—further necessitating revision surgery which tends to be more complicated than the initial reconstruction. Early surgery is often associated with growth restriction, and this decision should be made carefully. If compelling reasons exist to operate early in a growth phase, then the potential consequences should be recognized. For cranial procedures such as synostosis release and reshaping for both syndromic and nonsyndromic patients, repair and cranioplasty by 6–12 months of age is preferred unless increased intracranial pressure measurements dictate an earlier intervention. Allowing cranial growth until this point is viewed as favorable because reconstructed cranial vault and upper orbital shape are better maintained over long term due to more of the growth being completed, and hemodynamic stability is often easier to maintain because of larger blood volumes, among other reasons that limit anesthetic and resuscitation complexities.

Another area often requiring reconstruction in childhood is the lower orbital and malar region, such as in complex facial clefting or Treacher-Collins syndrome. Reconstruction of the malar region and lower orbit is usually reserved for after 5 years of age [7]. The vast majority of growth associated with the orbitozygomatic complex is complete by 5–7 years, which would mean a reconstruction at this point is often definitive for adult-sized structures [8, 9]. Repairs in this region that are performed earlier often require revision later. As with most craniofacial procedures, there is concern that continued growth would alter initial results due to growth restriction in the area or that surrounding tissue growth would limit the results. In addition, if split-thickness cranial vault bone is the desired bone graft, the bi-cortical nature of cranial bone in patients older than 5 years allows for easier splitting and separation. Splitting of the cranial bone can occur at a younger age but is often technically more challenging and limits available bone.

Maxillomandibular surgery is usually reserved for individuals who have reached skeletal maturity of the jaws, which occurs in individuals following typical growth curves, between 14 and 16 years of age in girls and 16 and 18 years in boys. Disproportionate growers may differ from this average pattern, so there is individual adjustment that should be accounted for when planning. Class III patients are repaired later while patients with hemifacial microsomia could be considered for early surgery by some based on a poorly formed and poorly functioning condyle segment. Considerations for surgery before the time of early skeletal maturity (13–15 years) are made for patients with Kaban IIB or III hemifacial deformities that may benefit from early intervention to optimize the mandibular position [10]. However, most all of these patients can wait until they are older, from a functional standpoint. These treatments are typically performed conjunction with orthodontic treatment and may involve early maneuvers such as costochondral grafting or mandibular distraction. This is significant because some patients will require glendoid fossa reconstruction which may include craniofacial grafting (see Fig. 2.1).

Another indication for early surgery is potential significant psychosocial benefit for patients who are being teased because of their deformity. Families of the patient must be counseled that these interventions would be considered first-stage surgical procedures, and that the patient would most likely require definitive surgery at a later time, after skeletal maturity is achieved. Another reason to intervene early is obstructive sleep apnea, and revision surgery is sometimes required in this population as well—again, typically in a more complicated environment.

The timing of pediatric craniomaxillofacial procedures affects the risk of complications in a variety of ways. From a hemodynamic standpoint, small patients are not able to compensate physiologically as readily as patients with large blood volumes and substantial reserves. Other issues may be associated with long-term issues such as growth restriction or infection risk, which may not manifest into obvious issues for over a decade. The complexity increases in a number of areas with infants and children. The following sections describe some of these instances.

2.1.2 Differences in Pediatric Versus Adult Patients

As is commonly stated in pediatric specialty care, pediatric patients are not small adults. This is even more true conceptually in craniofacial surgery, and particularly for surgical intervention of the neonate and infant. Hemodynamic instability is a major consideration as the craniofacial complex is highly vascular. Another is the potential for negative effects on future growth that can yield not just aesthetic asymmetry issues, but also functional problems with malocclusion, trismus, or other problems. Surgeons must carefully time their treatment with periods where structures are either mostly done growing or functional concerns encourage earlier intervention because they are compelling. The choices that a surgeon makes with respect to the relative aggressiveness of intervention and the materials one uses for reconstruction can have significant effects over the long term (Fig. 2.2).

In regards to trauma, infants and small children have a relatively large head compared to the rest of the body. Due to the prominence of the frontal bar and occiput from the rapidly growing brain in the infant, these structures are at higher risk of injury for motor vehicle accidents, falls, or animal bites (Fig. 2.3a–j). In fact, this was recognized as early as 1974 where Harris and colleagues reported that up to 78% of all dog bite injuries occur in the head and neck in children, whereas adults were only affected 10% of the time in this region [11]. Furthermore, the lack of well-developed neck musculature as well as a shorter neck makes the pediatric airway more prone to obstruction following injury. Their risk for particular types of craniofacial injury are different in larger children, and the procedures that we choose to address these issues can also have long-lasting effects on growth if aggressive maneuvers are utilized. Cranium and transcranial injuries are somewhat common in this group and infection in this area is a significant risk. Blood loss at the scene or in the perioperative phase can be significant.

From a physiologic perspective, the pediatric cardiovascular system is immature in that cardiac output, which is normally dependent upon stroke volume and heart rate, is reliant primarily on the heart rate. The contractile properties of the pediatric heart are underdeveloped due to lower concentration of contractile fibers in the cardiac muscle meaning the Frank-Starling law does not

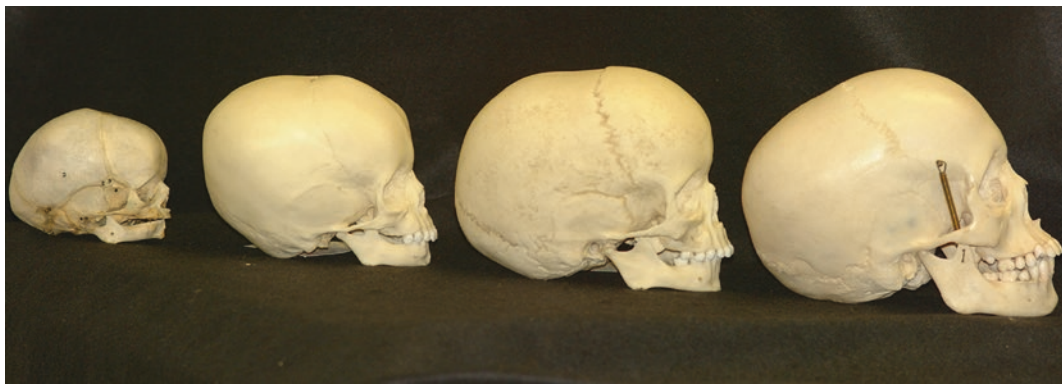


Fig. 2.2 Skulls at different stages of growth which include infancy through the mature adult craniomaxillofacial skeletal. The cranium and upper orbital region is complete with the vast majority of growth velocity by 1–2 years of age, while the maxillomandibular complex finishes its growth much later beyond 14–18 years of age with some variability-based gender and individual variation. The optimal time for intervention for a definitive treatment of a dysmorphic bone area is once the majority of growth is complete. The later this is completed, the more definitive the repair

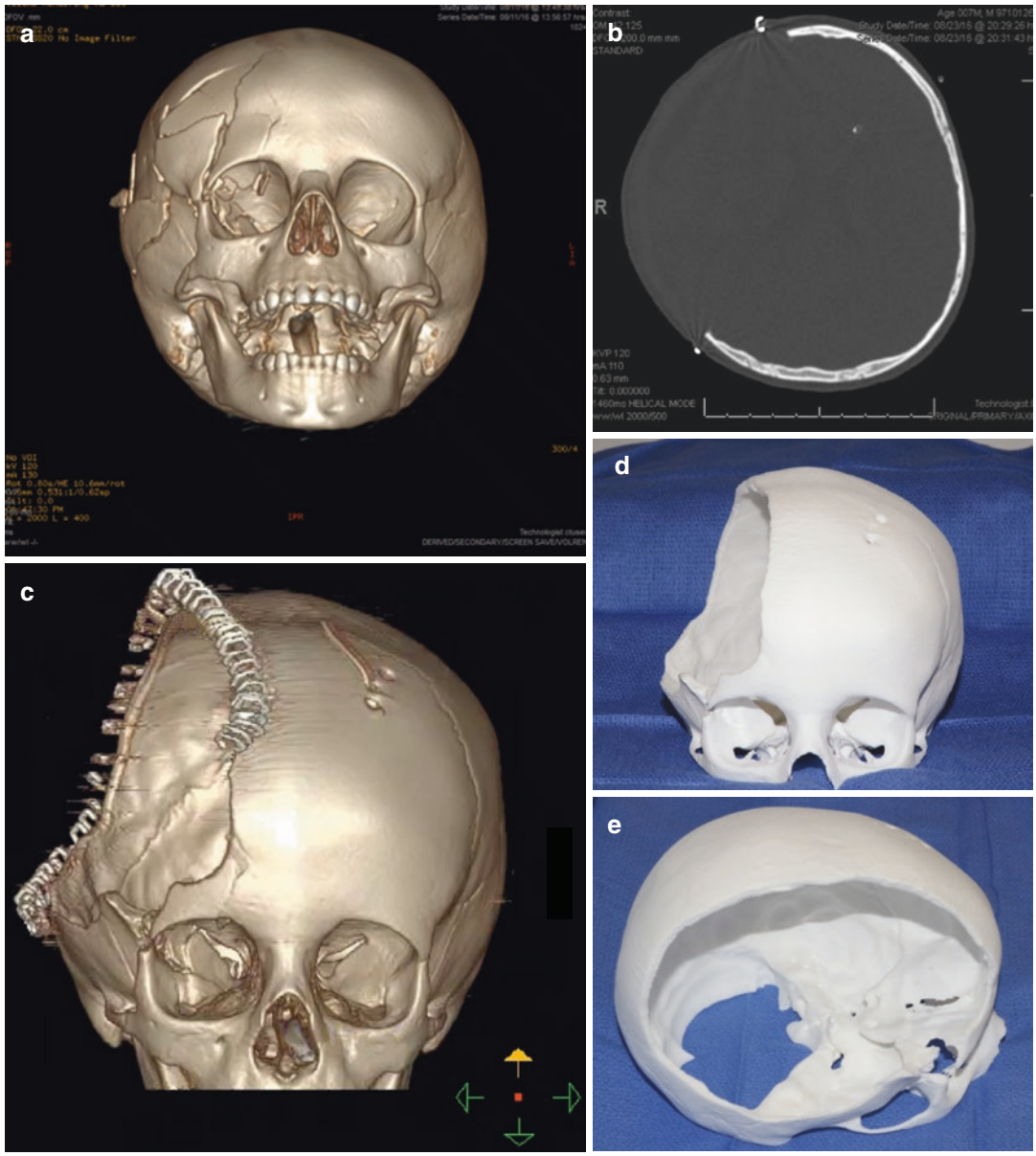


Fig. 2.3 (**a**–**j**) A young child who fell from a roller-coaster ride after attempting to disembark part-way through the ride with a resultant fall of over 20 ft. The contaminated and comminuted cranial vault was discarded during the neurosurgical debridement at the initial presentation. The remaining defect encompassed approximately 40% of the vault in a patient without a significant diploe for splitting. A poly-ether-ether-ketone custom implant was fabricated from a 3-D printed model generated from his computed tomography scan. The implant was held in place with titanium alloy plates and screws, provided perfect symmetry based upon its design, and was easy to place. While the aesthetic outcome is excellent compared with other autogenous techniques that tend to be more unpredictable, the lifetime risk for infection is significant

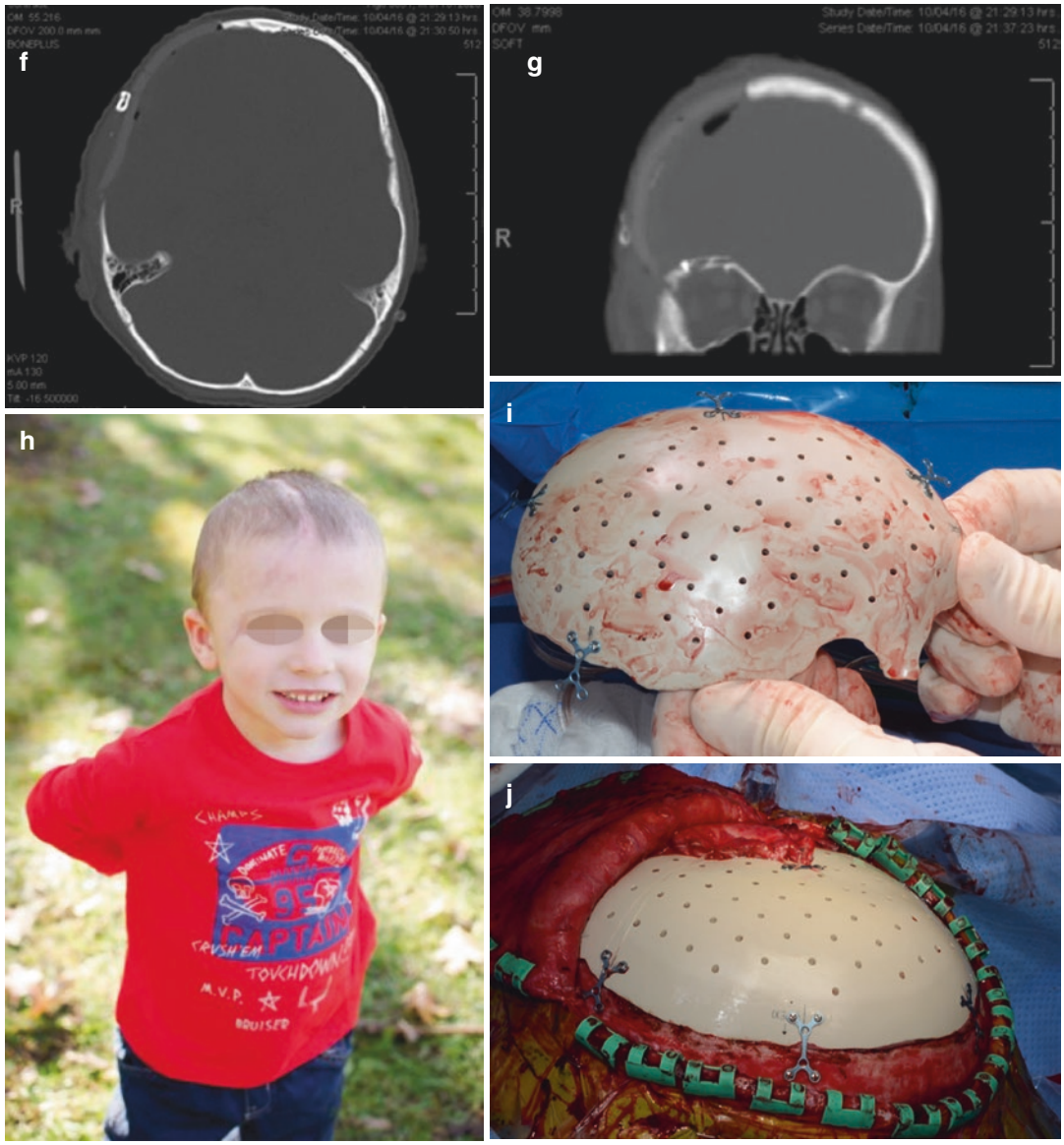


Fig. 2.3 (continued)

apply in the same manner to pediatric physiology [12]. As heart rate is the major determinant for cardiac output and perfusion, any bradycardias must be evaluated seriously, as a persistent bradycardia is usually a late sign for systemic compromise and potential impending cardiovascular collapse. Furthermore, blood loss must be treated much more aggressively in infants. The oft-cited figure of the 1% mortality rate of transcranial procedures is mostly due to exsanguinating hemorrhage, and in some instances, an inability to keep pace with loses when providing volume substitutions [13]. Anticipatory guidance and open communication between all key members

are the hallmarks of good team work during craniofacial procedures. This starts during the preparatory phase with blood being available when anticipated and recognizing the risk level in a particular patient.

2.2 Intraoperative Complications

2.2.1 Ocular Complications

Blindness is a rare complication of craniofacial surgery but has been reported with osteotomies that involve the orbit, distraction osteogenesis of the midface, and rarely in orthognathic procedures [14]. Munro and Sabatier reported four cases of blindness in their cohort of 1098 patients who underwent craniofacial surgery [15]. Intraoperative misadventure is a possible etiology, as is anatomic variation with unusual fracture patterns of the cranial base affecting either the nerves directly or blood vessels in the area—particularly in syndromic or growing patients. An increased incidence of blindness is seen in patients with growing skeletons and syndromic patients with hypoplasia of the cranial base, orbits, and/or midface. On rare occasions, bleeding in the posterior orbit at or near the palatine bone can cause a compressive neuropathy of the optic nerve. Prevention of ocular complications using corneal shields and navigation-assisted surgery can be used as appropriate. Intervention in an expeditious manner is important when these complications can be recognized early. Steroids and possible decompressive approaches to the optic nerve may be helpful depending upon the etiology. In addition, endoscopic navigation-assisted optic nerve decompression can be utilized to relieve pressure, but must be done quickly to avoid permanent effects. The anatomic nature of the orbit and its confined space makes this area susceptible to pressure from swelling, bleeding, or displacement of structures, and the presentation of symptoms often occurs late.

Another consequence of manipulating tissues in the ocular region is the incidence of bradycardia, or even asystole, associated with globe and orbital manipulation. Known as the *oculocardiac reflex*, this phenomenon is mediated by connections from the ophthalmic branch of the trigeminal nerve (V1) to the vagus nerve, causing aberrant signals to be fired by the vagus nerve in response to stimulation received by the trigeminal nerve [16]. When oculocardiac reflex is recognized, it is important for the anesthesia team to request pause from the surgeon to prevent cardiac collapse, though thankfully these complications are rare. Medications that function to limit parasympathetic conduction (e.g., glycopyrrolate or atropine) as well as epinephrine in severe cases can be used to counteract the physiologic response.

2.2.2 Dural Tear

Dural tears may occur during osteotomy creation or elevation of bony flaps. The design of many drill systems limits this occurrence, but when the dura is bound to the structures of the inner vault, such as at the suture sites, or the recess under the sphenoid wing, this can occur more frequently. Isolated tears are more likely to occur during elevation of dysmorphic or synostotic bone and is usually amenable to simple repair with direct suturing. Larger tears may be treated with patches using pericranial flap, collagen membrane, or other materials [17]. Experienced teams of neurosurgeons and craniofacial surgeons can sometimes avoid these issues by knowing the typical locations when this is prone to occur, but other instances are unavoidable based on anatomy or variations seen during revision procedures. When performing the tenon extension cuts of the frontal bar, it is often necessary to cut inferior to the sphenoid wing in an area that is somewhat blind if not dissected completely. This can cause injury to the dural and potential brain tissue. Not addressing tears properly may make the patient prone to cerebrospinal leaks, particularly in the anterior cranial vault area after trauma or extensive osteotomies that tend to leave pathways to the facial sinus areas and/or dead space in this recess. Those situations could lead to retrograde bacterial migration and infection.

2.2.3 Blood Loss, Fluid Shifts, and Electrolyte Imbalances

When compared with other orthopedic procedures in different anatomic locations, craniofacial surgeons are afforded significant latitude and can expect fairly predictable healing because of the rich blood supply of the craniomaxillofacial region. However, this rich blood supply also can be problematic. Significant bleeding can be seen in craniofacial procedures and must be managed efficiently—sometimes quickly, particularly in children with small blood volumes [18, 19]. While there are many factors involved, the technique utilized can have a significant impact on the volume of blood loss. It is recommended that if significant bleeding is anticipated, that appropriately typed and cross-matched blood be available for use by the anesthesia team to avoid delay in providing colloid replacement when needed. The authors often have a cell-salvage system in use when significant blood loss is expected in transcranial procedures, but with good operative technique most patients will not produce enough collected blood to give it back. In those instances where more substantial bleeding is encountered, the blood collecting system can be quite helpful. This is especially useful in patients who, for religious reasons or otherwise, do not accept blood transfusions. Breach of large vessels such as the sagittal or sigmoid sinuses can produce impressive bleeding that necessitates an immediate response.

A bi-coronal flap is often associated with significant bleeding because of the rich blood supply within the scalp and the vascular supply to the periosteum through the bone. Many surgeons will utilize a needle-tip bovie on a low setting to both cut and dissect the initial incision to minimize blood loss. When turning a scalp flap, dissection superficial to the periosteal plane (as opposed to in the sub-periosteal plane) significantly decreases the loss of red cell mass. Additionally, it also allows for a pericranial flap to be raised at the last portion of the flap elevation simplifying meticulous hemostasis, and provides a versatile flap for use later in the procedure. Appropriate local anesthetic with vasoconstrictor, bone wax, gel foam with thrombin, and other local measures can be utilized to minimize bleeding areas of bone while elevating the flap, improving hemostasis and minimizing volume loss. The amount of bleeding seen is often more severe with increased intracranial pressure, and anticipating that this may be the case in certain patients will help with planning and intraoperative fluid management.

Maximizing these efforts can be vitally important when turning the flap initially as well as during closure. While the majority of craniofacial procedures can be done without uncontrolled bleeding, some patients will have more blood loss than others for a variety of reasons. For many patients, this may be the first procedure they encounter, and blood dyscrasias may be discovered in the most inopportune of times. The senior author has diagnosed several patients with platelet disorders after a craniomaxillofacial procedure happened to be their first significant surgery. Even with the best of hemostasis measures and blood conservation measures such as the use of a cellular blood reprocessing machine, over 80% of infant patients who undergo open cranial vault surgery will require transfusion in the perioperative period.

Additional bleeding can be seen with the craniotomy from the diploic space, dura, and sagittal or sigmoid sinuses. Bleeding along the osteotomy sites can be controlled with bone wax. Sagittal sinus or sigmoid sinus bleeding is a more significant blood loss that requires immediate hemostatic control. Direct suturing of the vessel is often performed. Significant blood loss can be encountered and requires an aggressive approach to resuscitation. Additionally, late effects including intraluminal clotting of the sinus can propagate thrombosis or altered flow of the sinus which can lead to neurologic complications, including death [20].

Significant bleeding is also seen during the closure phase of the operation whether clip appliances are used or not. As the flap is turned back for closure, additional bleeding is seen and should be addressed with local measures and quick closure techniques. It is often the case that trainees are left to close the wound at this portion

of the case. It is important to note the blood loss potential to the surgeons closing the wound and to the anesthetic team as there is a common sense of calm that the major operative events are complete once closure begins—this is not the case in craniofacial surgery. Experienced teams understand this risk. Planning for this blood loss avoids complications and prepares the patient for the most effective and efficient recovery postoperatively. The team should not find out that the hemoglobin levels have dropped once arrival occurs at the intensive care unit and blood is not immediately available.

2.2.4 Air Embolism

In children, air embolism can be seen with flap elevation or craniotomies. These events are likely due to the rich supply of emissary vessels and venous lakes in the pediatric cranial vault and surrounding area [21]. In 35% of patients, a patent foramen ovale is present, and these patients are at risk for aberrant travel of this air to the left heart chambers and passage to the cerebral circulation or to the coronary arteries causing arrest. Usually, the left atrial pressure is enough to keep the foramen ovale closed, but a venous air embolism may enter the pulmonary circulation and create increased right atrial pressure, causing a right to left shunting of blood.

The outcomes are variable, but some patients who experience a large embolism that causes an effect on the cardiac or central nervous systems do not survive. While less significant venous air embolism occurs with some higher frequency, the event usually does not produce a life-threatening episode. This event contributes significantly to the 1% mortality rate seen in craniofacial procedures.

In the event that an air embolism is detected, rapid closure of the air entry point is essential. The Bernoulli effect can contribute to this risk when a rotary bur is utilized to cut the bone during the osteotomies, creating a whirling effect around potential entry points with resultant entrance of air. Bone wax application to cut bony edges and physical compression of openings with coagulation adjuncts (e.g., gelatin sponges or oxidized cellulose sheets) will assist in decreasing further air entry.

2.2.5 Mishandling of Flaps

All bony flap manipulation should be completed over a sterile back table, and the transfer of such flaps should be deliberate. Many experienced craniofacial surgeons have moments involving the dreaded "thud" with the bone flap being dropped on the floor by any number of team members. In addition to the careful transfer of such flaps using clamps or other methods, they should be stored in a moist location that is announced to the entire team and signed out should the scrub tech transfer duties to another without error. The tissue should not be allowed to dry completely as surface cells would become desiccated and potentially become nonviable. Furthermore, the use of a *clean* gauze is important as the scrub technician or other team member would not be tempted to discard the flap in a misguided attempt at cleanliness. When flaps are still attached, it is crucial to keep the areas moist using frequent irrigation with physiologic saline. Antibiotics are not preferred as the higher concentrations can be harmful to viable bone cells. As the flaps are inset, it is often beneficial for the scrub technician and circulator to do a quick count of gauze and sponges to ensure that they do not become implanted as well.

2.2.6 Loss of Airway or Accidental Extubation

The risk of inadvertent extubation intraoperatively has been documented well and is much higher in craniomaxillofacial procedures—particularly if there is an intraoral component or if the patient is prone [22]. In pediatric patients, simple flexion at the neck is sometimes enough to displace the endotracheal tube superiorly to the vocal cords—particularly with some types of endotracheal tubes in patients with longer neck structures. This can be problematic with

down-fracture of midface structures, maxillomandibular fixation, bleeding, or other obstructions of the airway making re-intubation difficult. The risk of intraoperative extubation is increased when the patient is positioned prone for posterior cranial vault reconstructions. Because of these considerations, it is imperative that the endotracheal tube is secured well with taping, wires, and/or sutures, and that the length of the tube is measured either at the lips or nares and checked regularly either by the anesthesiologist or surgeon. Anticipatory plans should be made in cases that are high risk and scenarios should be discussed regarding what would be done if the endotracheal tube became dislodged at various points in the procedure.

Another common complication is obstruction of the secured airway by kinking or pressure on the tube during the procedure. The conscientious surgeon must always pay attention not only to the surgical field, but also to the multitudes of monitors and sounds fielded by the anesthesia team. When airway pressures increase suddenly or volumes change, the endotracheal tube should be examined and checked for poor positioning. If the patient is placed on a volume-controlled ventilation setting and the anesthesiologist does not recognize the increased airway pressures caused by a kink in the endotracheal tube, the patient may endure barotrauma, or issues with flash pulmonary edema can occur. This can limit effective respiration and be difficult to manage.

2.3 Early Postoperative Complications

2.3.1 Infection, Wound Breakdown, and Meningitis

Infection of the craniofacial bones or in the soft tissue flaps is rare, with one study showing postoperative infection in only 0.4% of their 248 consecutive patients for single suture craniosynostosis repair [23]. A subperiosteal abscess is occasionally seen. More significant infections of the bone flaps are thankfully rare, but do occur. Loss of bone segments occurs most often due to lack of blood supply in compromised areas such as those patients with significant acute soft tissue injury, radiation to the site, or scarred soft tissue from multiple procedures. Even the most biocompatible of implants can become infected, and tend to do so in those instances that involve compromised tissue (Fig. 2.4a, b). They also carry a lifetime infection risk, so the decision to

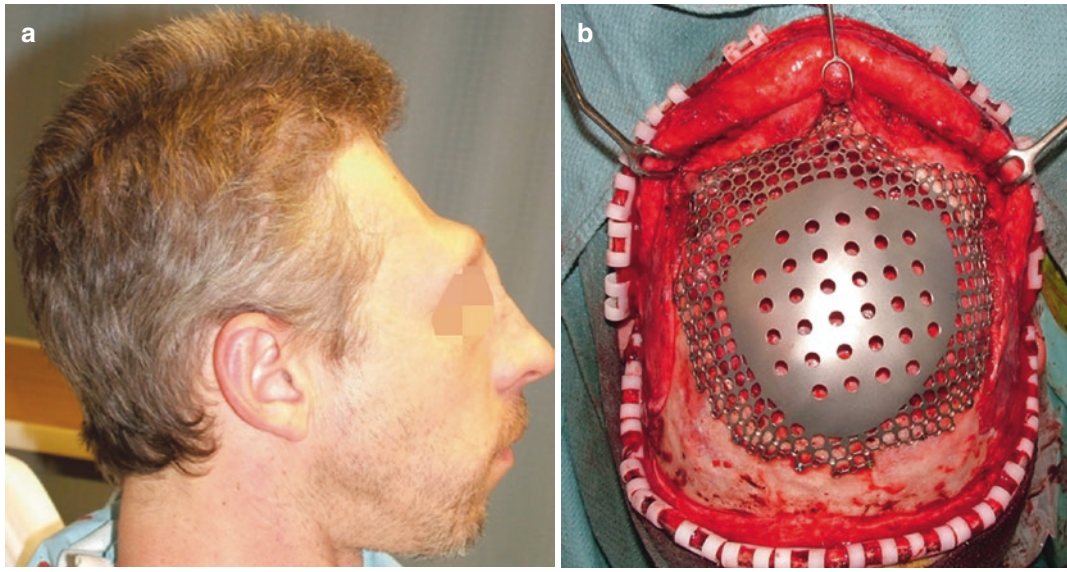


Fig. 2.4 (**a**, **b**) An adult male who sustained trauma to the anterior cranial vault with a postoperative infection and loss of his entire reconstruction, performed by other surgeons. The remaining defect was reconstructed with a custom CAD-CAM generated custom titanium frontal bandeau and entire frontal complex

use artificial materials in children should be considered carefully when revision may be a certainty over a lifetime of an individual patient (Figs. 2.5 and 2.6). Regenerative solutions are not quite ready at this point, and the limited results of bone healing modulators, such as bone morphogenic protein, has been less than impressive. Patients who have infections with collections should be cultured, particularly those with long histories of revision surgery or extensive hospital visits. The infection rate tends to be higher in those procedures that involve the sinuses and dead space, such as the monobloc or bi-partition osteotomies [24]. Intracranial infections are rare in congenital craniofacial procedures, but are seen more commonly after removal of neoplasms—particularly after adjuvant therapies. They are addressed with antibiotics and occasionally drainage procedures.

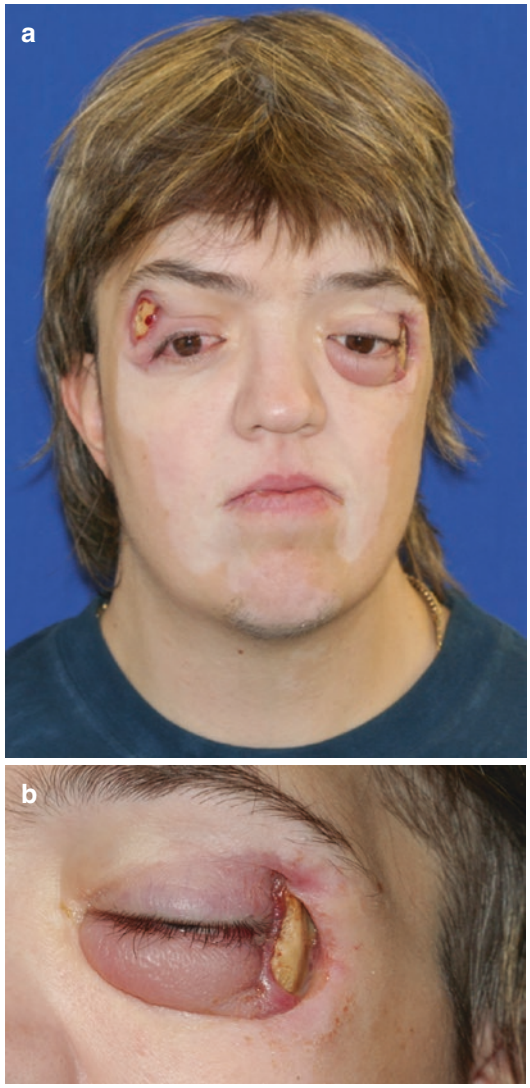


Fig. 2.5 (**a**, **b**) An adult female with Crouzon syndrome that had placement of polymethylmethacrylate paste that was custom fabricated in situ by another surgeon, but later became infected with exposure in multiple locations, including the periorbital tissues. While the reconstruction lasted for years, the resultant infection and chronic wounds were devastating to the local tissues. Complete removal is difficult when this material fragments and is locked into existing wire or plate fixation

Occasionally, surgical incision sites can open at the scalp due to seroma, abscess formation, or inflammatory reactions. Most wounds of this type are addressed easily with local wound care measures. Occasionally, debridement and reclosure is needed, although most are managed with local measures. In these instances, every effort should be made to address these concerns with a tension-free closure. Alopecia may form in these areas if a hypertrophic scar band results due to the secondary intention healing. Excision and with local flap rotation can aid in limiting the area of alopecia once the site has healed for at least 6 months. At times, tissue expansion for recruitment of local tissue may be helpful to close larger defects or limit areas with alopecia.

2.3.2 Cerebrospinal Fluid Leak

Cerebrospinal fluid leaks are occasionally encountered with severe cranial base fractures or extensive surgery at the cranial base. In a study by Esparza et al., the surgeons encountered cerebrospinal fluid leakage postoperatively in 2.7% of their cases [25]. Leaks are often limited by careful blockage of the cranial base with various materials including pericranial flaps, fibrin glue, dermal grafts, bone, fat, or others. However, leaks can still occur rarely despite the best of efforts intraoperatively. Endoscopic techniques with or without navigation can be utilized to localize defects and utilize internal septal flaps for closure [26]. Patients can be given a lumbar drain, and precautions can be taken such as elevating the head to limit intracranial pressure. These maneuvers will often allow small leaks to heal. Larger

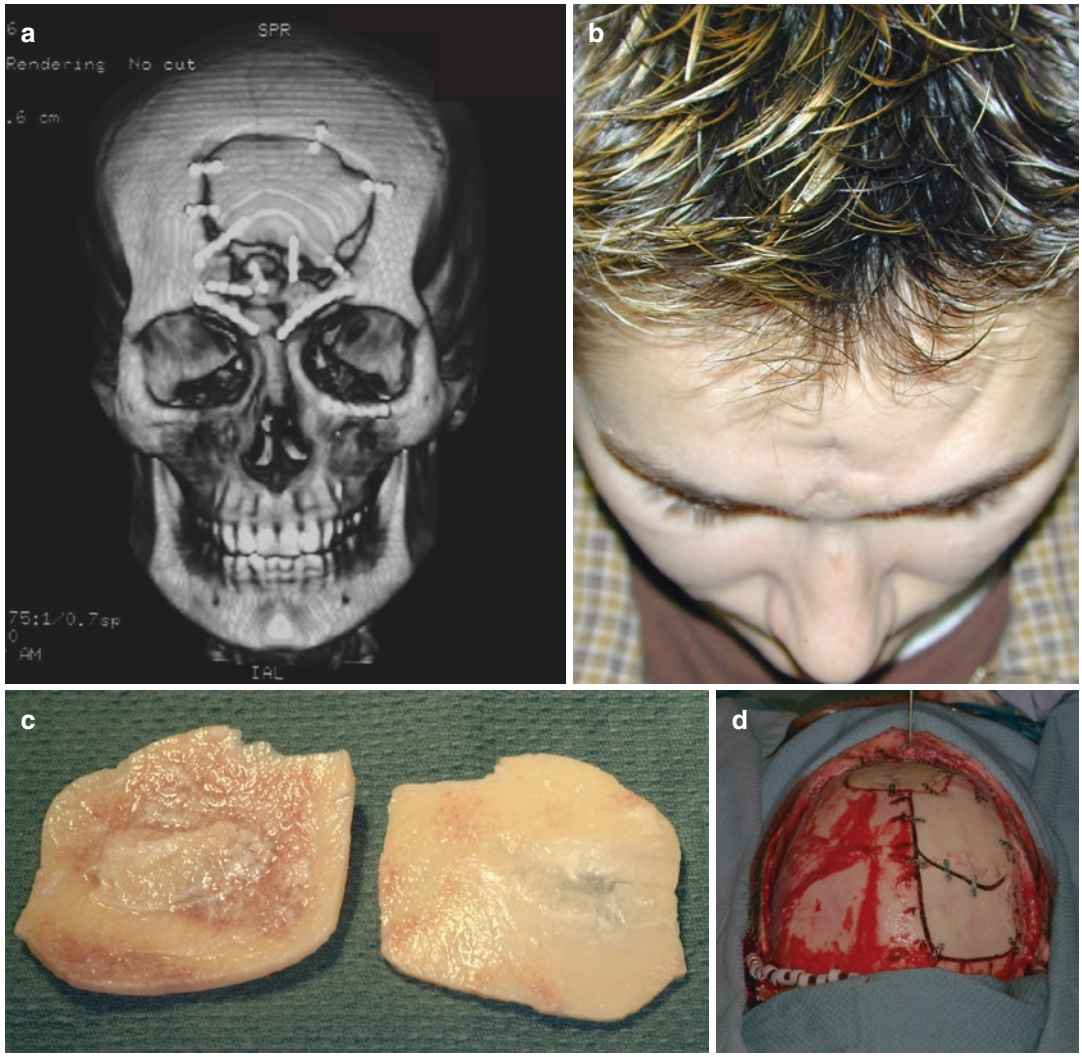


Fig. 2.6 (**a**–**d**) An adult female who had a primary reconstructive procedure after a crush injury to the forehead from a large concrete block that fell from an overlying bridge onto her motor vehicle. The traumatic blow crushed the overlying soft tissue making it difficult from smaller segments to heal. A nonunion resulted and was replaced with autogenous split thickness cranium with limited fixation; the authors preferred reconstruction when conditions permit

leaks can be addressed operatively with open procedures to re-address the cranial base [26].

2.3.3 Increased Intracranial Pressure

One of the indications for surgical repair of craniosynostosis cases is the risk or presence of raised intracranial pressures. As was mentioned previously, there is not good evidence suggesting a predictable risk of increased intracranial pressure that results in functional deficits in a predictable manner for patients with single-suture craniosynostosis. On the other hand, increased intracranial pressure is thought to occur in up to 42% of untreated children with multiple suture synostosis [27]. Cranial vault reshaping and decompression is usually effective at allowing room for brain expansion without large

fluctuations in intracranial pressure, which directly impact the cerebral perfusion pressure. Intraoperatively, some surgeons elect to place small bur holes into bony flaps to allow for escape of accumulated blood. However, some authors have documented a small group of patients that present with raised intracranial pressure even after vault reshaping and decompression [28]. In addition, many children will require multiple revisions of craniosynostosis repairs that will refine shape and accommodate for a growing brain. It is well described that the brain triples in size within the first year of life and is four times the size at birth by age 2 in the normally developing child. Interesting research has also shown that the brain volumes in children with autism spectrum disorders are up to 6% greater than in unaffected individuals [29]. Thus, surgeons should anticipate revision surgeries in craniosynostosis release to prevent increased intracranial pressures during development. Close monitoring is important to decrease the events of undetected increases in intracranial pressure and irreversible neurologic damage.

2.3.4 Electrolyte Imbalances

Electrolyte abnormalities can occur with the extensive fluid shifting that may occur with more complicated craniofacial reconstructions. As such, hyponatremia, hypokalemia, and acid-base imbalances are relatively common, with sodium imbalances being seen the most often. Indeed, in their prospective study, Levine and colleagues showed that all of their patients who underwent a craniofacial procedure had a brief period of hyponatremia postoperatively [30]. Etiologies of these sodium abnormalities can include syndrome of inappropriate antidiuretic hormone (SIADH), salt wasting, or aggressive crystalloid replacement. The pathophysiology of each of these entities is closely tied to kidney function (or dysfunction) and its processing of water. In the syndrome of inappropriate antidiuretic hormone secretion, a central nervous system disorder allows an inappropriate and persistent release of antidiuretic hormone. The subsequent water retention via the proximal tubules of the kidney creates dilutional hyponatremia. Cerebral salt wasting is a debated phenomenon where impaired sympathetic neural input could reduce sodium absorption in the proximal tubule of the kidney.

Electrolyte parameters should be monitored carefully and addressed in an expeditious manner to avoid large shifts in electrolytes. If untreated, sodium abnormalities may create mental status changes that could mimic those seen in increased intracranial pressures. In addition, reversal could cause central nervous system disorders if it is undertaken too rapidly. An active management strategy is recommended in patients with electrolyte abnormalities that do not respond to traditional therapies and consultation with an endocrinologist may be helpful. Specialized replacement regimens are sometimes needed to address these abnormalities and avoid other complications such as cardiac instability, arrhythmias, or central effects.

2.4 Late Postoperative Complications

2.4.1 Nonunion of Flaps/ Reconstruction

Once initial healing has occurred, most complications that occur late are a consequence of nonviable tissue and poorly healed bone. For patients with substantial reconstructions of the craniorbital region, nonhealing bone segments are a relatively rare occurrence. Smaller fragments may have difficulty healing, especially in compromised soft tissue beds. Defects smaller than 15 millimeters may not require reconstruction and post little risk to the patient. However, even small defects in sensitive areas may present aesthetic concerns such as the superior orbital rim or frontal bar region. When areas of bone resorb, hardware may become more palpable or mobile, causing inflammation and possible necessitation of removal of hardware. Defects that are larger than 15 millimeters may require reconstruction with autogenous grafts, titanium mesh, or alloplastic materials (Figs. 2.5 and 2.6). Current alloplasts are less

desirable in patients who have already had a failed primary reconstruction, or those with other compromises to healing (Fig. 2.5).

2.4.2 Alloplastic Materials

There is no doubt that technologies such as intraoperative navigation and three-dimensional printing have greatly influenced the current state of operative technique and the precision with which we can deliver symmetric, custom implants. The ability to create custom cranial vault implants that perfectly mirror the unaffected side of a patient or even use normative models is an advancement that allows even novice surgeons to create aesthetic repairs of large defects. However, even with significant improvements in alloplast development, no material is perfectly biocompatable or has the healing potential of autogenous bone (Figs. 2.4, 2.5 and 2.6). Traditionally used titanium mesh is easily customizable in the operating room setting and is useful for small to medium size defects but carries a lifetime infection risk. Custom computer-aided design/computed-aided manufactured titanium and polyether-ether-ketone (PEEK) implants are now commonly used for large size defects but carries the same drawbacks of lifetime infection risk [31]. Indeed, patients who receive these implants have vast aesthetic improvements with relative ease, but questions regarding infection arise each time they have a headache or other symptoms due to possible infection.

Ongoing research into regenerative techniques may provide additional options, but are currently not approved for most craniofacial indications at this time [32]. The future may hold promising new materials that utilize the patient's own stem cells and a custom scaffold to create a fully osteogenic solution [33]. This would be similar in theory to some intraoral tissue engineering that dentists currently use for dental implant site development. Until then, split thickness cranium or titanium mesh still provide the best and most-predictable solutions for small to medium size defects while custom-printed PEEK implants are the standard for larger reconstructions.

2.4.3 Relapse Requiring Revision

Relapse of cranio-orbital advancements occurs occasionally, and does so most often in instances of large advancements and/or patients with syndromes and poor bone quality. Good fixation is important for retention of advancements in the immediate postoperative time period. Late relapse occurs in patients who have earlier procedures during early phases of growth, and really a manifestation of growth restriction rather than true "relapse." This is particularly evident in patients with unicoronal synostosis as the asymmetry becomes increasingly more evident with time as the child grows. Single-suture craniosynostosis reconstructions all have a revision rate of appropriately 5%, but certain dysmorphologies, such as more severe unicoronal synostosis, may predispose to higher rates of revision [34]. Syndromic craniosynostosis reconstructions have a higher revision rate, but often require additional procedures for other reasons than relapse (e.g., increased intracranial pressure). Nonetheless, understanding the limits of the soft tissue envelope and the quality of bone are important when deciding the amount of advancement of the frontal bandau, or other components. While surgeons often speak of "overcorrecting" the deformity during an early treatment, there is no predictable way to determine if this is helpful and how much "overcorrection" might be helpful in an individual patient—this is true for every technique available.

2.5 Conclusion

In general, craniofacial surgery is predictable, safe, and successful. A clear understanding of the nuances, optimal outcomes, and complications is important to treat patients with these disorders at the highest level. Management of complications and optimizing outcomes requires an awareness of the possibility of these issues as well as the technical ability and experience to address them effectively.

References

1. Sharma RK. Unfavourable results in craniofacial surgery. Indian J Plast Surg. 2013;46(2):204–14.
2. Hertle RW, Quinn GE, Minguini N, Katowitz JA. Visual loss in patients with craniofacial synostosis. J Pediatr Ophthalmol Strabismus. 1991;28(6):344–9.
3. Delashaw JB, Persing JA, Broaddus WC, Jane JA. Cranial vault growth in craniosynostosis. J Neurosurg. 1989;70(2):159–65.
4. Virchow R. Über den cretinismus, nametlich in Franken, under über pathologische. Schadelformen Verk Phys Med Gesellsch Wurzburg. 1851;2:230–71.
5. Ruiz RL, Ritter AM, Turvey TA, Costello BJ, Ricalde P. Nonsyndromic craniosynostosis: diagnosis and contemporary surgical management. Oral Maxillofac Surg Clin North Am. 2004;16(4):447–63.
6. Turvey TA, Ruiz RL. Craniosynostosis and craniofacial dysostosis. In: Fonseca RJ, Baker SB, Wolford LM, editors. Oral and maxillofacial surgery. Philadelphia: WB Saunders; 2000. p. 195–220.
7. Posnick JC, Ruiz RL, Tiwana PS. Craniofacial dysostosis syndromes: stages of reconstruction. Oral Maxillofac Surg Clin North Am. 2004;16(4):475–91.
8. Posnick JC, Ruiz RL. The craniofacial dysostosis syndromes: current surgical thinking and future directions. Cleft Palate Craniofac J. 2000;37(5):433.
9. Waitzman AA, Posnick JC, Armstrong D, Pron GE. Craniofacial skeletal measurements based on computed tomography. Part II. Normal values and growth trends. Cleft Palate Craniofac J. 1992;29:118–28.
10. Kaban LB, Padwa BL, Mulliken JB. Surgical correction of mandibular hypoplasia in hemifacial microsomia: the case for treatment in early childhood. J Oral Maxillofac Surg. 1998;56:628–38.
11. Harris D, Imperato PJ, Oken B. Dog bites—an unrecognized epidemic. Bull N Y Acad Med. 1974;50(9):981–1000.
12. Katz AM. Ernest Henry Starling, his predecessors, and the "law of the heart". Circulation. 2002;106(23):2986–92.
13. Poole MD. Complications in craniofacial surgery. Br J Plast Surg. 1988;41(6):608–13.
14. Lo LJ, Hung KF, Chen YR. Blindness as a complication of Le fort I osteotomy for maxillary distraction. Plast Reconstr Surg. 2002;109(2):688–98; discussion 699–700.
15. Munro IR, Sabatier RE. An analysis of 12 years of craniomaxillofacial surgery in Toronto. Plast Reconstr Surg. 1985;76:29.
16. Lang S, Lanigan DT, Van der wal M. Trigeminocardiac reflexes: maxillary and mandibular variants of the oculocardiac reflex. Can J Anaesth. 1991;38(6):757–60.
17. Greensmith AL, Meara JG, Holmes AD, Lo P. Complications related to cranial vault surgery. Oral Maxillofac Surg Clin North Am. 2004;16(4):465–73.
18. Jones BM, Jani P, Bingham RM, et al. Complications in paediatric craniofacial surgery: an initial four year experience. Br J Plast Surg. 1992;45:225–31.
19. Steig PE, Mulliken JB. Neurosurgical complications in craniofacial surgery. Neursurg Clin N Am. 1991;2:703–8.
20. Resnick DK, Pollack IF, Albright AL. Surgical management of the cloverleaf skull deformity. Pediatr Neurosurg. 1995;22(1):29–37; discussion 238.
21. Phillips RJ, Mulliken JB. Venous air embolism during a craniofacial procedure. Plast Reconstr Surg. 1988;82(1):155–9.
22. Shaw I, Kumar C, Dodds C. Oxford textbook of anaesthesia for oral and maxillofacial surgery. Oxford: Oxford University Press; 2010.
23. Fearon JA, Ruotolo RA, Kolar JC. Single sutural craniosynostoses: surgical outcomes and long-term growth. Plast Reconstr Surg. 2009;123(2):635–42.
24. Whitaker LA, Munro IR, Salyer KE, Jackson IT, Ortiz-Monasterio F, Marchac D. Combined report of problems and complications in 793 craniofacial operations. Plast Reconstr Surg. 1979;64:198–203.
25. Esparza J, Hinojosa J. Complications in the surgical treatment of craniosynostosis and craniofacial syndromes: apropos of 306 transcranial procedures. Childs Nerv Syst. 2008;24(12):1421–30.
26. Rivera-Serrano CM, Oliver CL, Sok J, Prevedello DM, Gardner P, Snyderman CH, Kassam AB, Carrau RL. Pedicled facial buccinator (FAB) flap: a new flap for reconstruction of skull base defects. Laryngoscope. 2010;120(10):1922–30.
27. Gault DT, Renier D, Marchac D, Jones BM. Intracranial pressure and intracranial volume in children with craniosynostosis. Plast Reconstr Surg. 1992;90(3):377–81.
28. Siddiqi SN, Posnick JC, Buncic R, et al. The detection and management of intracranial hypertension after initial suture release and decompression for craniofacial dysostosis syndromes. Neurosurgery. 1995;36(4):703–8.
29. Piven J, Arndt S, Bailey J, Havercamp S, Andreasen NC, Palmer P. An MRI study of brain size in autism. Am J Psychiatry. 1995;152(8):1145–9.
30. Levine JP, Stelnicki E, Weiner HL, Bradley JP, McCarthy JG. Hyponatremia in the postoperative craniofacial pediatric patient population: a connection to cerebral salt wasting syndrome and management of the disorder. Plast Reconstr Surg. 2001;108(6):1501–8.
31. Lethaus B, Safi Y, Ter laak-poort M, et al. Cranioplasty with customized titanium and PEEK implants in a mechanical stress model. J Neurotrauma. 2012;29(6):1077–83.
32. Costello BJ, Shah G, Kumta P, Sfeir CS. Regenerative medicine for craniomaxillofacial surgery. Oral Maxillofac Surg Clin North Am. 2010;22(1):33–42.
33. Thesleff T, Lehtimäki K, Niskakangas T, et al. Cranioplasty with adipose-derived stem cells and biomaterial: a novel method for cranial reconstruction. Neurosurgery. 2011;68(6):1535–40.
34. Selber JC, Brooks C, Kurichi JE, Temmen T, Sonnad SS, Whitaker LA. Long-term results following fronto-orbital reconstruction in nonsyndromic unicoronal synostosis. Plast Reconstr Surg. 2008;121(5):251e–60e.

3 Complications Associated with Distraction Osteogenesis

Suzanne U. Stucki-McCormick and Louis F. Clarizio

Contents

Since the application of distraction osteogenesis to the bones of the craniofacial skeleton in the early 1990s, the DO technique has gained success and acclaim [1, 2]. The primary advantage of the distraction osteogenesis (DO) technique is that the slow application of force over time allows for histiogenesis and the generation of all tissues: skin, muscle, nerves, blood vessels, and bone (Fig. 3.1a–c) [3]. The changes in the facial skeleton are impressive, with secondary correction of the affected skeleton not in the original site of distraction including improvement of the airway [4, 5].

S. U. Stucki-McCormick (✉)
Department of Oral and Maxillofacial Surgery, School of Dentistry, University of California Los Angeles, Los Angeles, CA, USA

L. F. Clarizio
Private Practice, Portsmouth, NH, USA

Conventional orthoghnathic surgery allows for the immaediater movement of a bone to its new positon, held in place and allowed to heal. In contrast, the distraction osteogenesis technique requires the application of forec over time with the bones gradually moved to the final position. Conseqently, it is vital that the surgcial team ensures close pateint follow-up during the entire DO process and consolidation phases [6]. As with other techniques of the bony skeleton, complications encountered during and after DO surgery are similar to that of conventional orthognathic and dentoalveolar surgery, and discussed elsewhere [7]. However, the complications unique to distraction osteogenesis can be divided into three categories: poor planning, poor execution, and lack of attention to detail with a lack of close follow-up.

The "consolidation phase" when the DO device is in neutral fixation and the segment has been advanced to its optimal position is the most important for this close follow-up. It is during the

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_3

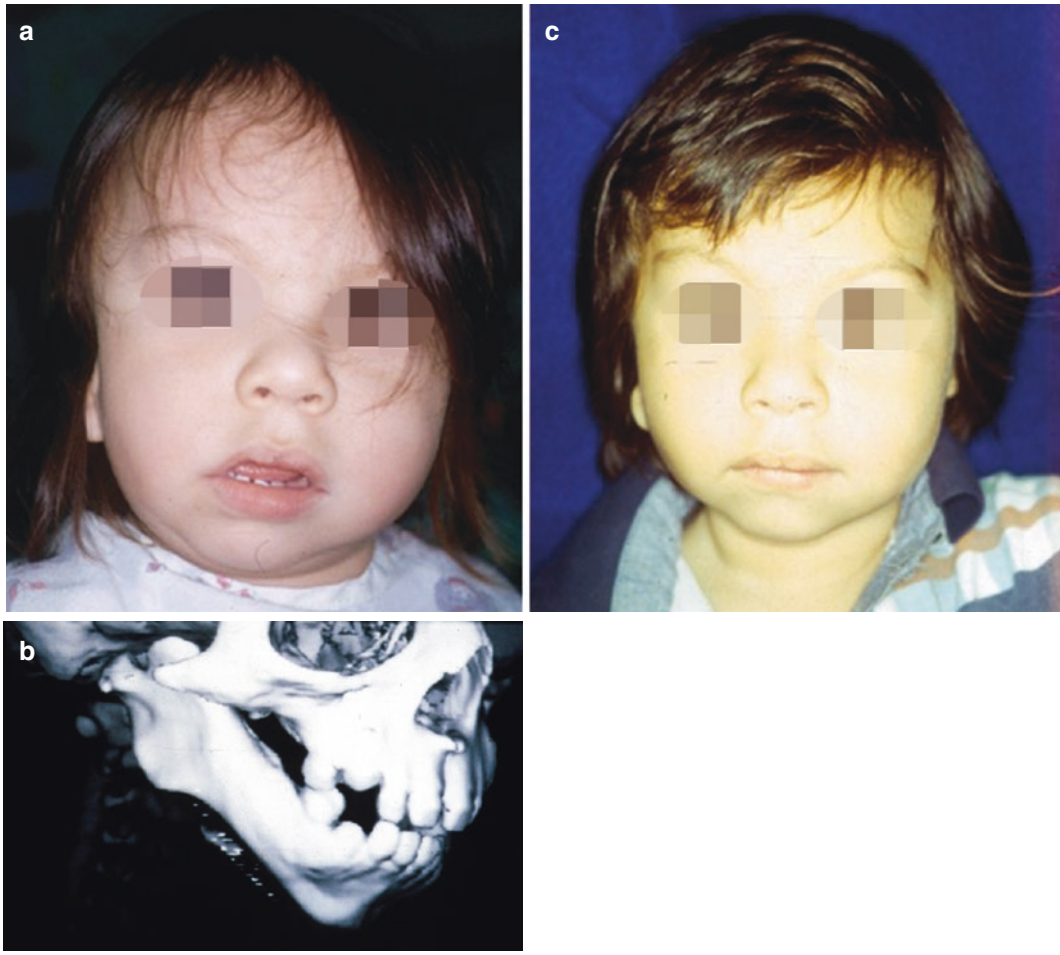


Fig. 3.1 (**a**) A young child with unilateral craniofacial microsomia prior to mandibular distraction osteogenesis (DO). Chin point deviation, occlusal cant, hypoplastic right zygona, and right microtia are noted. (**b**) 3-D CT scan of the patient with Type IIb craniofacial macrosomia. (**c**) After mandibular DO, facial form is reestablished. The power of the distraction technique is noted by the soft tissue response

consolidation phase when the new regenerate bone is at its softest, with minimal ossification. Similarly, at the end of DO, that the DO device is fully "open," when structural stability of the device at its weakest and minor local muscle forces can rotate and torque even the best designed DO devices. Consequently, the regenerate is susceptible to adjacent muscle pull resulting in complications such as open bite, misshaped regenerate, tipping of the regenerate, and other force-related phenomenon including those as a consequence of a patient parafunctional habits.

Distraction osteogenesis allows for the expansion of the osseous skeleton in vectors outside those of traditional orthognathic surgery including mandibular widening (Fig. 3.2a–d). Again all tissues are created allowing for orthodontic movement of teeth into the newly distracted bone. However, as the distraction plane is counter to that of the physiologic skeleton-muscular envelope of the face, the rate of relapse was initially high. This relapse was primarily due to local muscle pull. The advent of newer hybrid distraction devices have overcome this challenge [8].

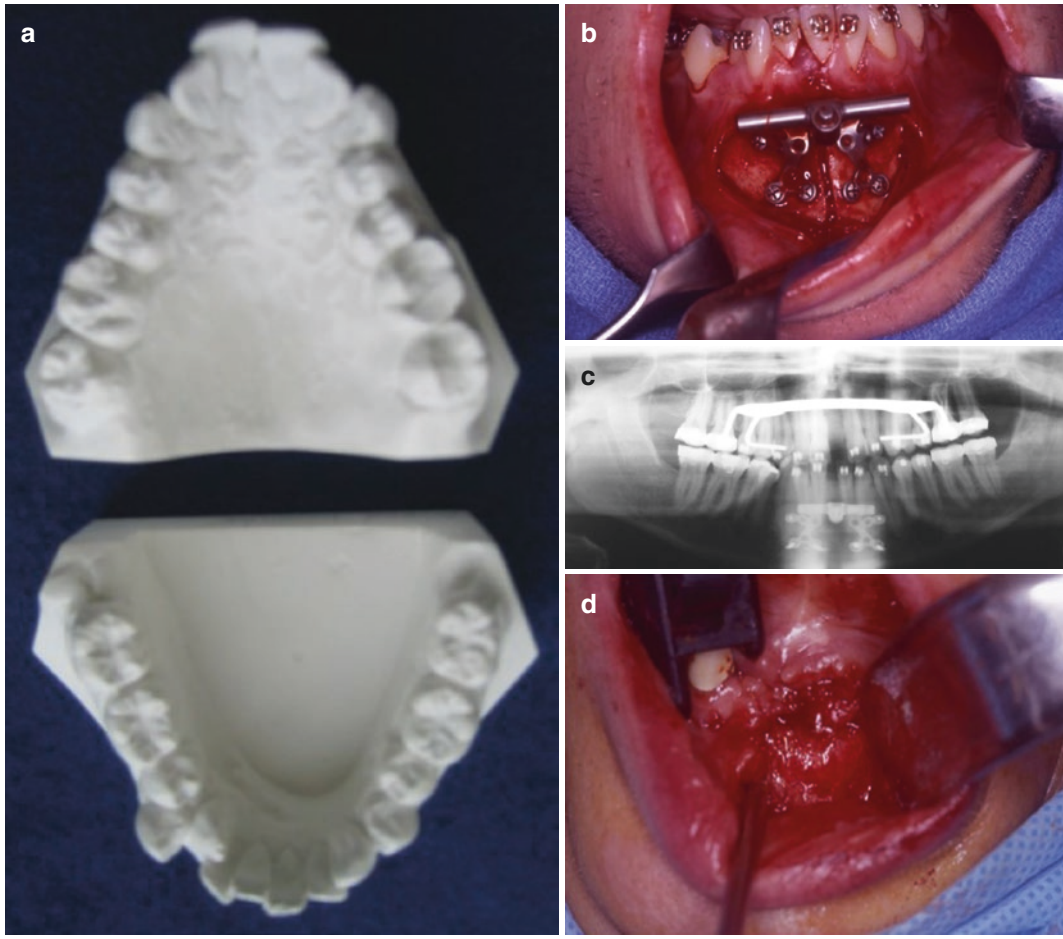


Fig. 3.2 (**a**) A patient with severe constriction of the maxilla and mandible underwent maxillary and mandibular widening using DO. (**b**) The osseous-borne DO device in place for mandibular widening. (**c**) The panoramic radiograph showing maxillary and mandibular widening during DO. (**d**) The osseous regenerate created from the DO process is noted at the time of DO device removal

With the advent of Virtual Surgical Planning (VSP), many of the complications associated with the planning phase of the DO technique have been obviated (Fig. 3.3). VSP allows the clinician to reproduce the osseous anatomic site in 3-D, both on the computer and in a stereolithic model (SLA) (Fig. 3.4). This allows for visualization of critical anatomic structure including neurovascular structures, and unerupted teeth. The computer models can now predict the bony movements planned and the vector of DO device as it is positioned on the bone (Fig. 3.5). The volumetric airway change can also be predicted using VSP simulation [9]. Thus the clinician can customize: 1. choice of size/length of the DO device, 2. the positioning of the DO device, and 3. the placement of DO device retention screws, all as to avoid vital structures and trajectory concerns. Using VSP, the osteotomy can also be planned in 3-D. Here, the bone cut can be modified, angled, or stepped to enhance osseous gain during DO as well as to avoid vital structures [10]. The resultant planned osteotomy is converted to a custom surgical guide (Fig. 3.6a, b). The VSP of the osteotomy and planned movement can identify sites of potential bony interfer-

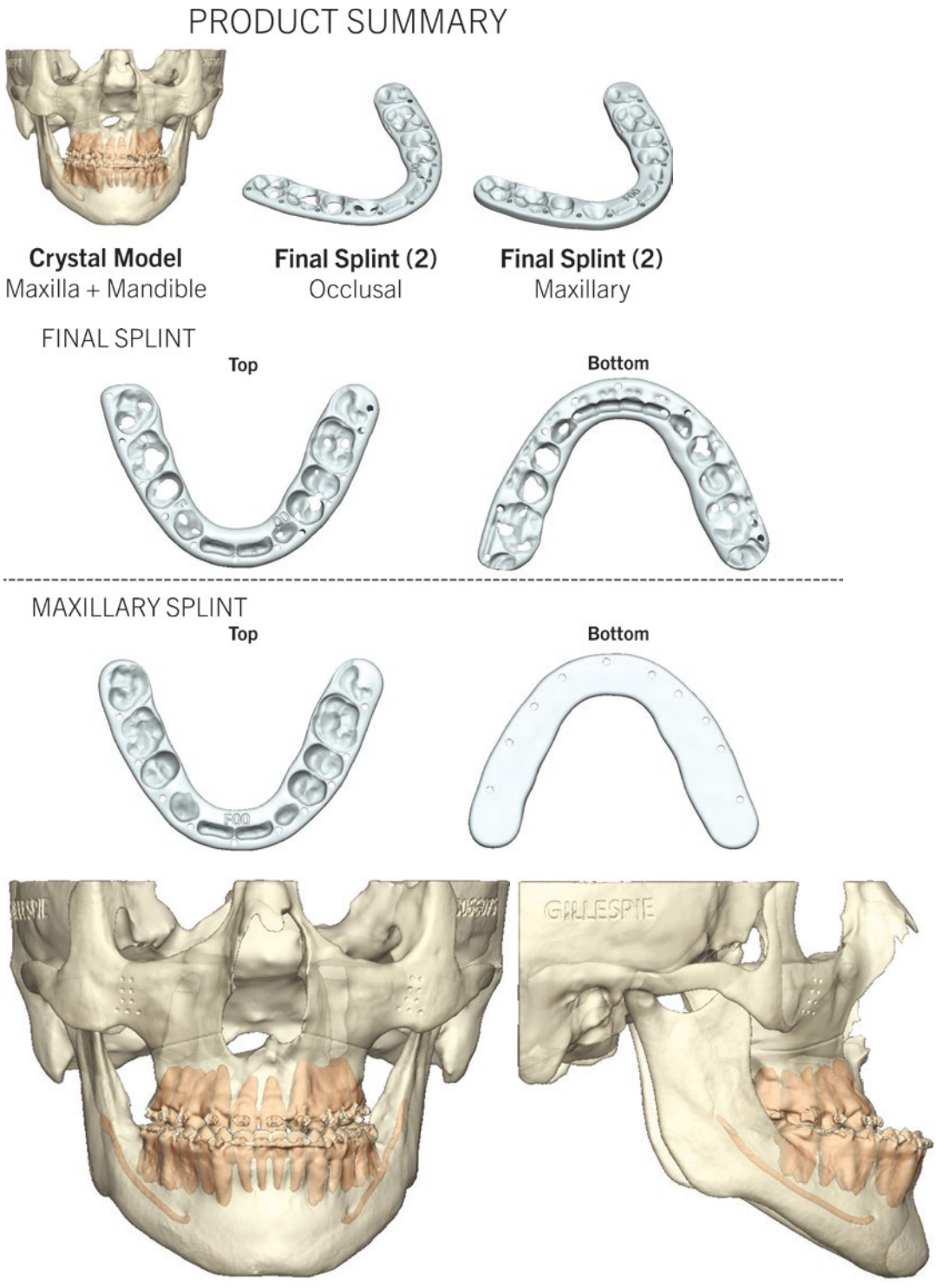


Fig. 3.3 Virtual surgical planning (VSP) allows the surgeon to visualize the maxillary osseous structure, planned osteotomy, and DO device/screw placement here in 3-D. (With permission from Dr. Richard Burton)

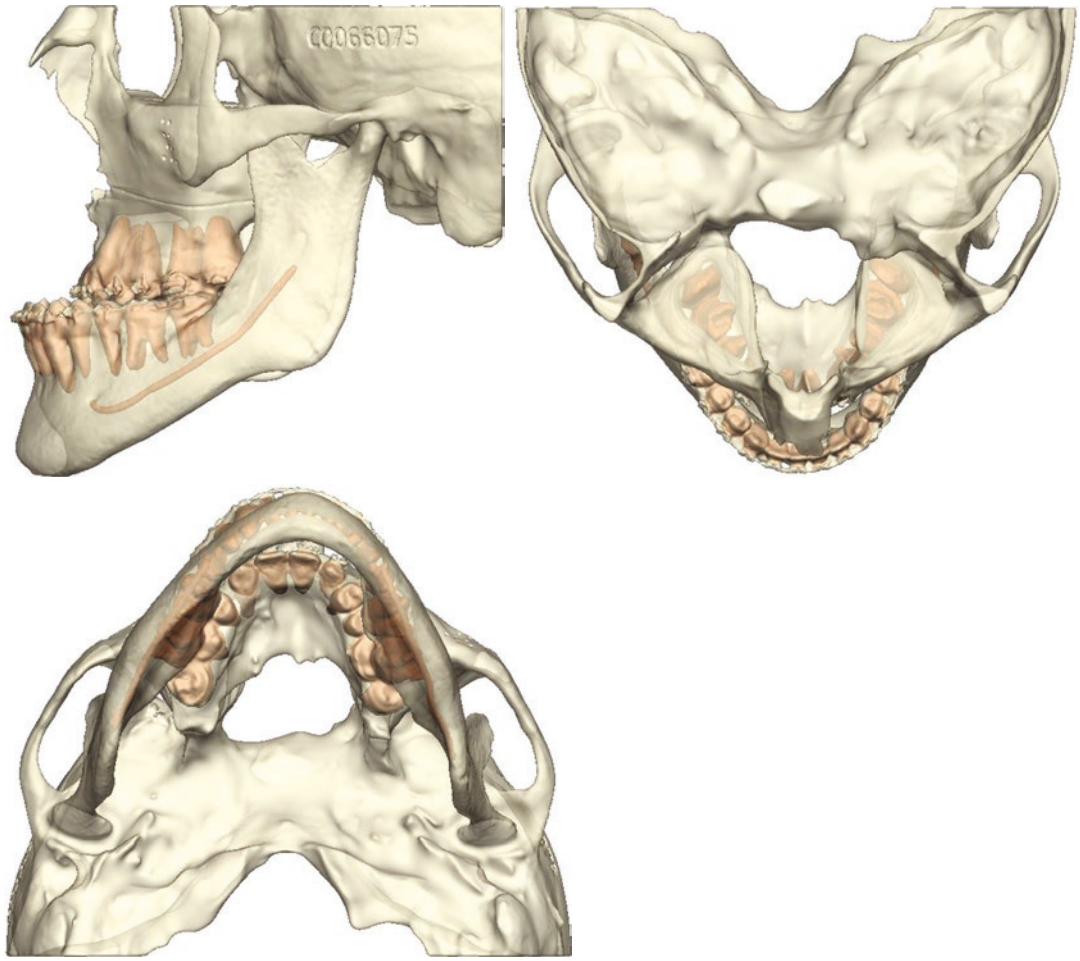


Fig. 3.3 (continued)

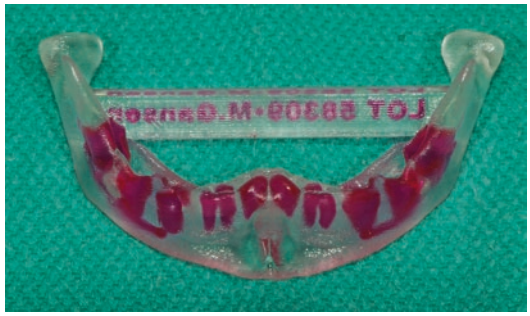


Fig. 3.4 A stereolithic model can be generated during the VSP planning process. The model allows for visualization of vital intraosseous structures including the neurovascular bundle and unerupted teeth in this infant with micrognathia. (With permission from Dr. Richard Burton)

ences or protuberances that may need to be removed prior to closure of the site which are useful and verified during surgery (Fig. 3.7a, b). VSP planning can also identify areas of potential technical/device limitations and failures. In general, submerged devices exhibit less technical failures [11].

Care must be taken during the VSP phase as to verify the location of the planned osteotomies versus the local muscles. A bone cut anterior to the masseteric muscle sling can result in proximal segment rotation due to local muscle pull, much like an unfavorable fracture of the mandible. For large mandibular advancements, the infrahyoid musculature is most pronounced to

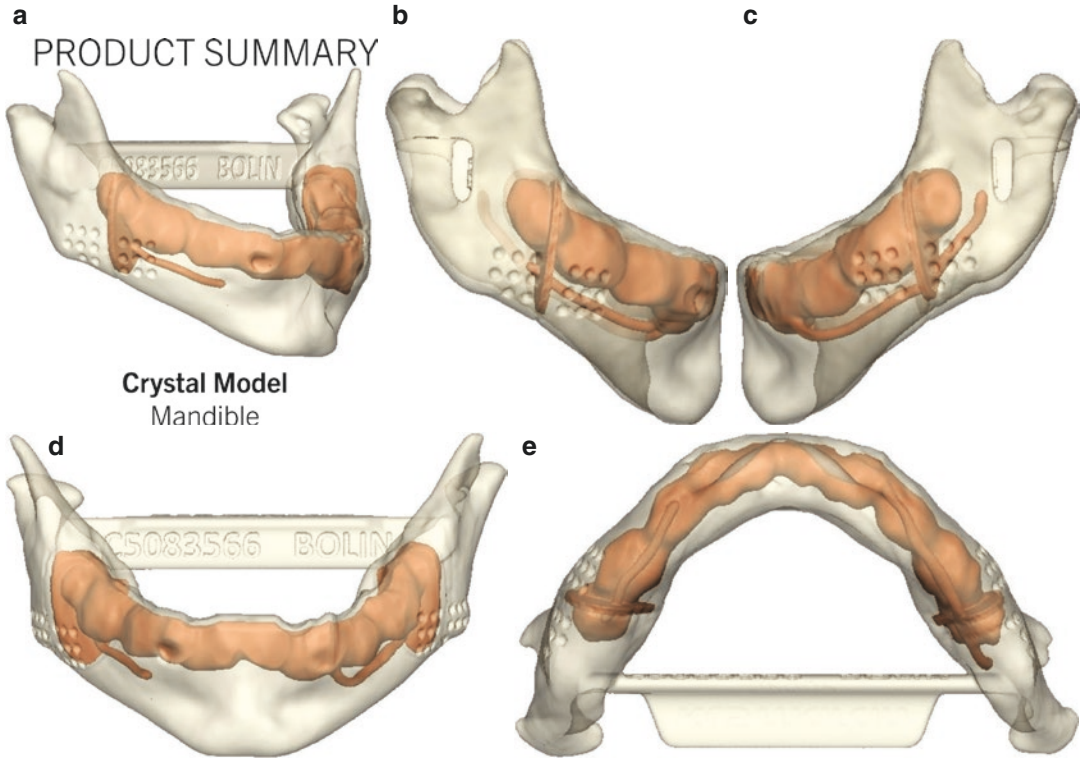


Fig. 3.5 (**a**) Virtual surgical planning (VSP) allows for accurate identification of anatomic landmarks including the IAN and tooth buds for infant distraction osteogenesis. (**b**, **c**) VSP allows the clinician to identify and plan the site of the DO bone cut as well as the device placement/trajectory and retention screw sites for the infant airway DO device in both lateral views right (**b**) and left (**c**). (**d**, **e**) The device placement and trajectory can be verified in the frontal (**d**) and submandibular (**e**) views. Additionally, the device footplate and retention screw hole sites can be verified in these views as to avoid vital structures

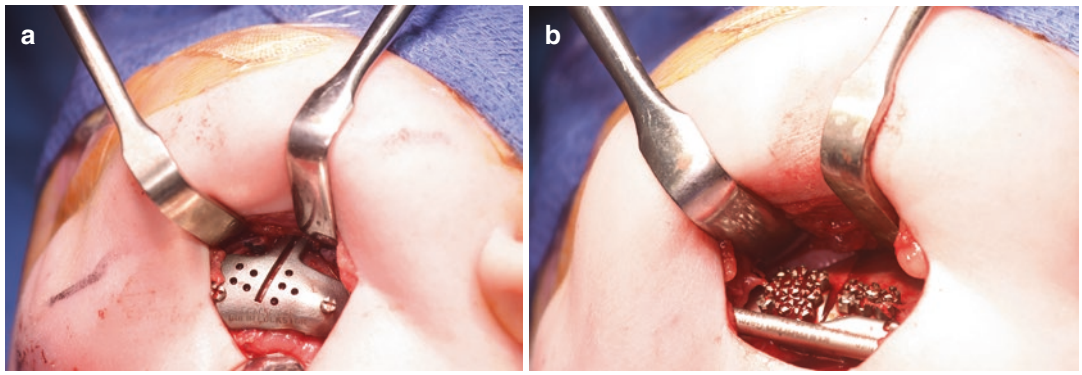


Fig. 3.6 (**a**, **b**) The planned surgical guide is in place. Here the osteotomy is angled, as to avoid vital intraosseous structures. The DO device is placed in the planned orientation. (With permission from Dr. Richard Burton)

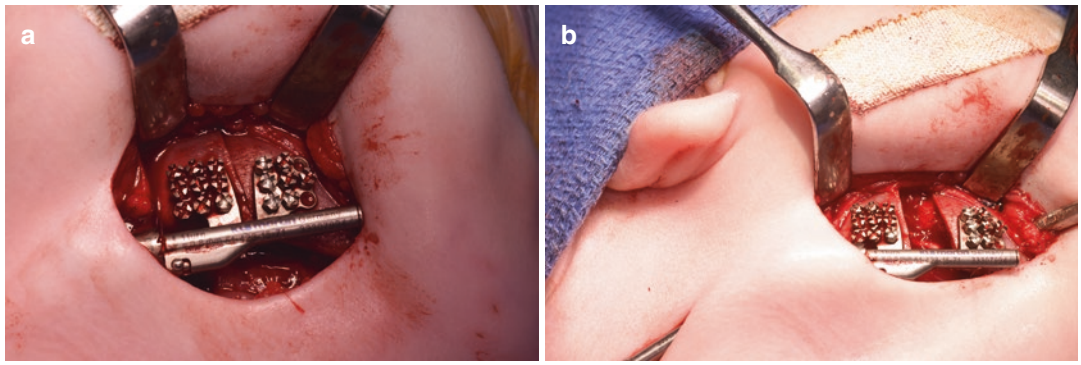


Fig. 3.7 (**a**, **b**) On the contralateral side, the device is placed in the planned position. The device is activated to ensure free movement of the DO site. The IAN can be visualized. (With permission from Dr. Richard Burton)

affect a clockwise rotation of the distal segment, especially during the end of DO, during the consolidation phase. Similarly, vertical alveolar DO can be affected by the pull of the mylohyoid if its insertion is high on the lingual aspect of the mandible. Thus unexpected challenges can be encountered during surgery necessitating a "Plan B." The following are four complications representing categories of challenges that occur during distraction osteogenesis. Many of these complications are "old school," and occurred prior to the advent of virtual surgical planning, VSP. However even with VSP, these occurrences represent the most common complications associated with DO of the craniofacial skeleton. Thus identifying these challenges/complications and how they were addressed gives insight and highlights the need for attention to detail during the entire DO process, from planning to final DO device removal.

3.1 Case 1: Small Bone Segment DO

A 35-year-old male presented to the office with complaints of periodontal involvement around a dental implant to area #8. Several years earlier, he was playing water polo and was struck in the face, with damage to tooth #8 (Fig. 3.8a). At the time #8 was removed and an immediate implant placed. The implant was placed, immediately into the remaining bone, 2 mm below the crest of the bone, as was the standard of care then, back at the time when the implant was placed into an immediate extraction site (Fig. 3.8c, d). This led to the implant being placed significantly below the level of the alveolus to the adjacent teeth. A longer crown and long custom abutment were fabricated which over time led to localized periodontal involvement, as the site was difficult to clean (Fig. 3.8e).

Physical examination revealed that the overlying gingival tissue had acceptable contour and concern was raised that removal of the implant and subsequent localized bone grafting might result in a lesser gingival contour (Fig. 3.8f–h). Consequently, it was decided to perform small segment distraction osteogenesis, DO whereby the implant would be part of the small DO/transport disc, as to vertically reposition the osseointegrated implant [12–15]. First, the overly elongated crown was removed and a temporary crown fabricated, as to allow adjustment/reduction of the incisal edge of the crown during the DO process, as the implant was distracted vertically downwards, from its original submerged positon, towards the crest of the alveolar ridge (Fig. 3.8i).

The site was approached through a vestibular incision. The small alveolar DO device (Track 1.0, KLS Martin LLP) was modified and used for the DO (Fig. 3.8j). The osteotomy was planned to be a two vertical bone cuts and one horizontal cut leaving approximately 1 mm of bone around the

implant laterally and 2 mm of bone vertically to the implant, as to avoid the teeth on both sides and the floor of the nose.

Taking advantage of the curvature of the alveolus and the concavity of the bone in the cuspid region, the vertical activation portions was located in the cuspid fossa with the activation site exposed in the vestibule between the cuspid and lateral incisor (Fig. 3.8k, l). This site was chosen as to help hide the DO device when smiling versus placement of the DO device more proximally, adjacent to the central incisors. As the bone segment was

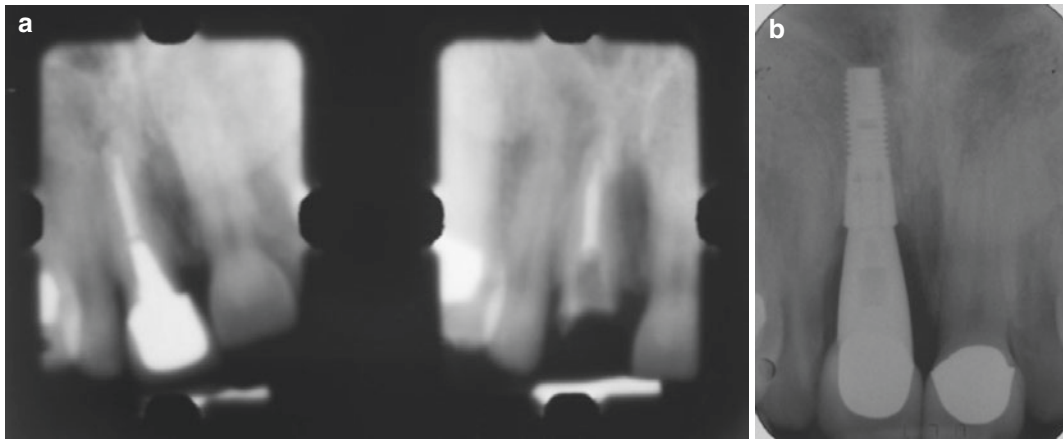


Fig. 3.8 (**a**) Adult patient with endodontically treated central incisor, now with water polo sports injury resulting in root fracture. At the time of tooth removal, there was existing vertical alveolar bone loss. (With permission from Dr. Richard Burton). (**b**–**d**) The fractured tooth was removed and an immediate implant placed. The immediate implant was placed 2 mm below the remaining crest of the ridge, according to the protocol at the time. This resulted in the need for a custom abutment with a long abutment neck as noted on the periapical (**b**), panoramic (**c**), and lateral cephalometric (**d**) views. (**e**) The implant integrated and the bone remodeled as noted on this periapical radiograph 5 years after implant placement. (**f**, **g**) The gingival esthetics and health were compromised due to the long custom abutment noted on the facial (**f**) and palatal (**g**). (**h**) A low smile line is noted that helped to camouflage the gingival compromise. (**i**) The crown was removed and a new resin crown was fabricated to allow for small bone segment distraction osteogenesis (DO). (**j**) A 1.0 Track alveolar DO device (KLS Martin LLP) was modified. Note the bending of the lateral arms in a "butterfly" pattern. As to allow adaptation of the DO device to the curvature of the maxilla. (**k**) The planned osteotomy was created as to avoid the teeth on either side and the floor of the nose: two vertical bone cuts with 1 mm cuff of bone lateral to the implant and one horizontal cut leaving approximately 2 mm of bone apical to the implant. (**l**) The aid in esthetics and comfort, track device was positioned so that the activation site and vertical arm was positioned in the canine fossa (O). (**m**) As the site was small, only the lower arm with a single screw was used in the transport disc containing the implant. (**n**) During active DO, the resin crown was reduced vertically, as the distraction proceeded. (**o**) At the end of DO, the small segment was held in place to allow for ossification. The implant remained integrated during the DO process. (**p**) After DO, the implant positon was improved yet short of the ideal, as the small segment rotated, due to the long cantilever arm for the Track device positioned in the canine fossa. (**q**) A 5-year periapical radiograph revealed the distracted implant to be well healed with good bone stability of the distraction sites and crestal bone levels

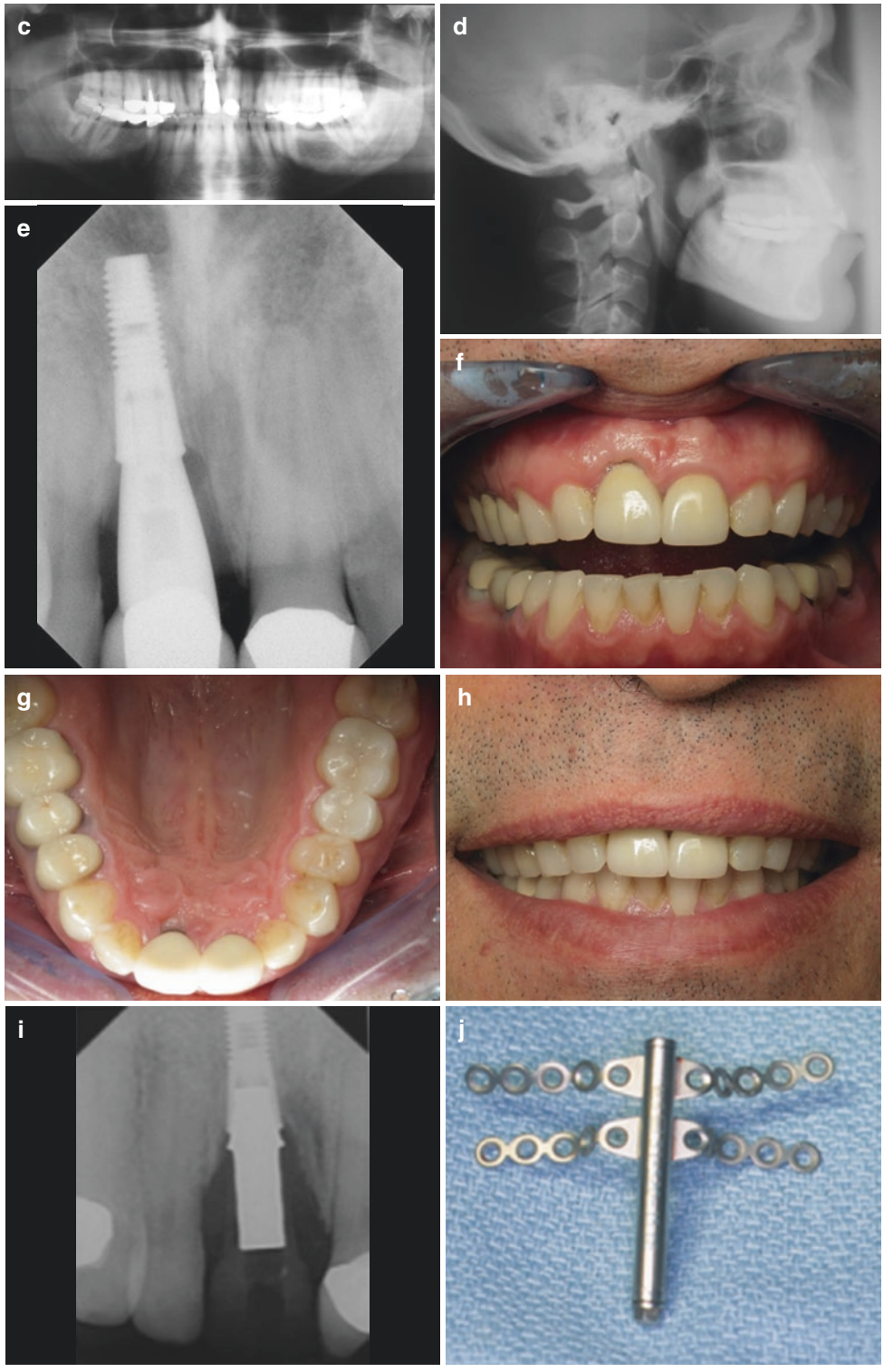


Fig. 3.8 (continued)

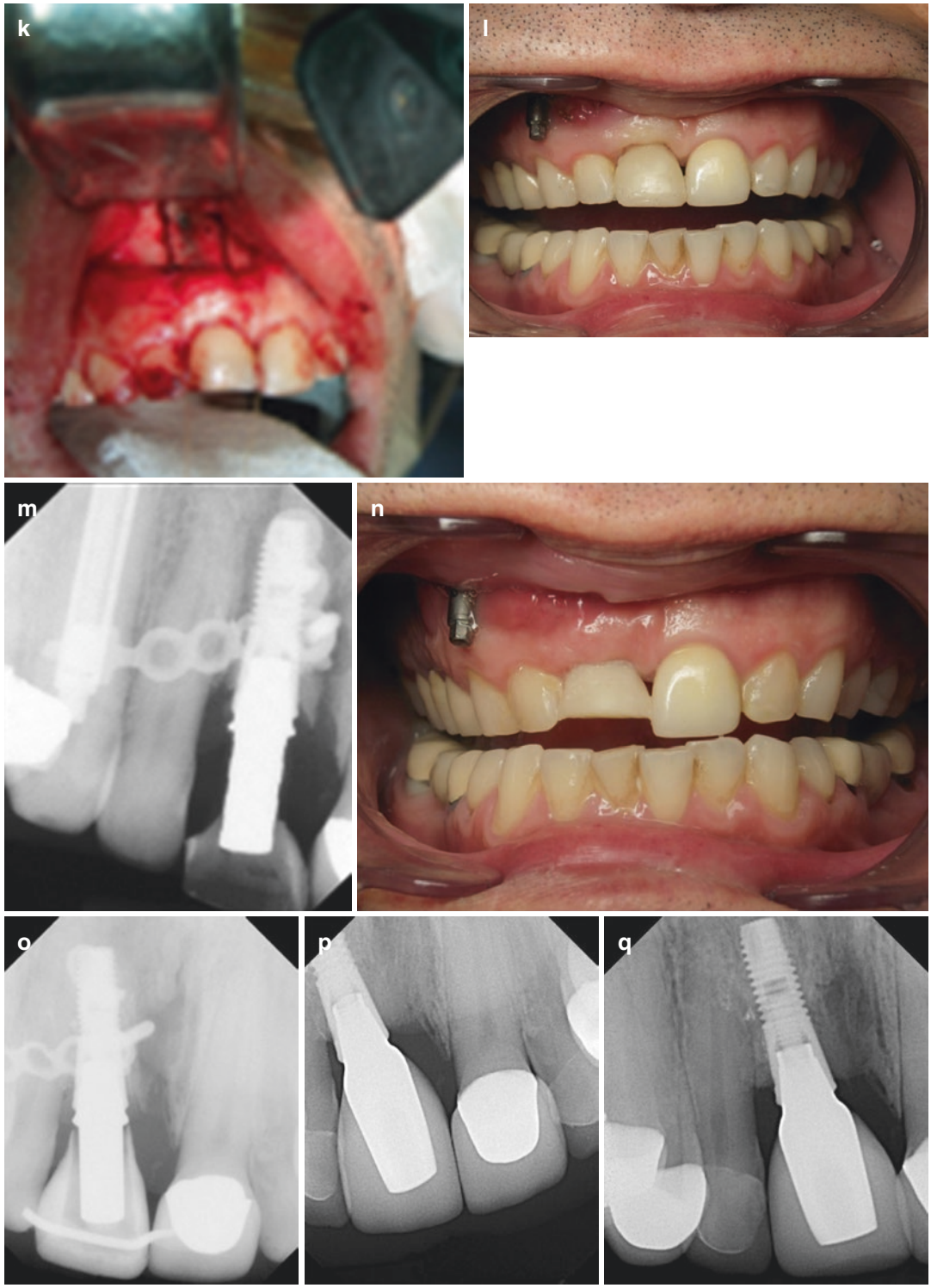


Fig. 3.8 (continued)

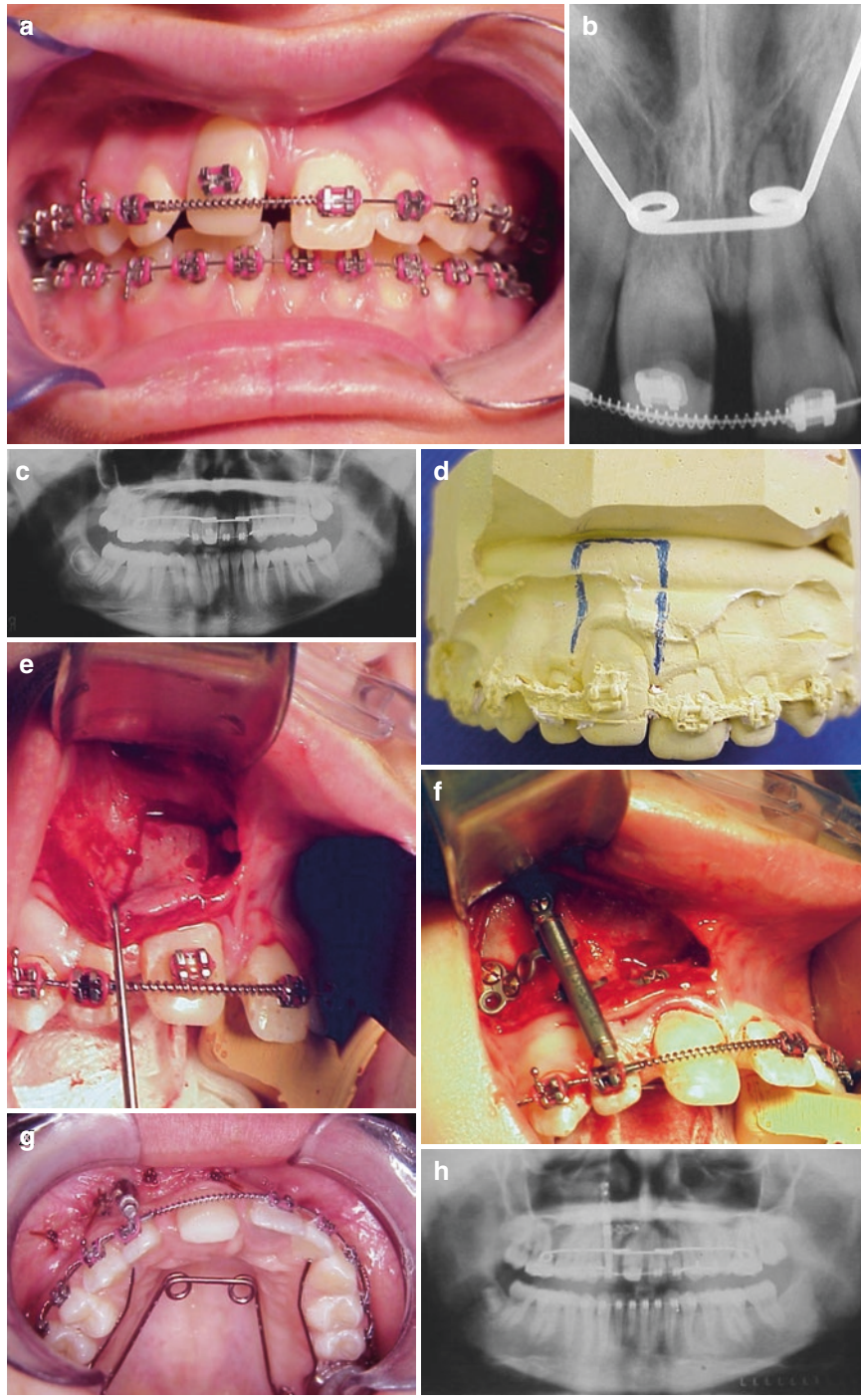


Fig. 3.9 A second case of dentoalveolar ankylosis, was a patient whose the central incisor underwent trauma as a child. To camouflage, tooth colored material was placed at the incisive edge to make the two central incisors symmetrical (**a**). The tooth is noted to have internal/external resorption on the periapical radiograph (**b**). The associated alveolar bone is located vertically high, as compared to the remaining alveolus as seen on the periapical and panoramic radiograph (**b**, **c**). (**d**, **e**) The distraction segment was planned on the model and created in the maxilla leaving a cuff of bone as to protect the adjacent teeth and floor of nose. (**f**) The 1.0 Track device (KLS Martin LLP) was adapted to fit the curvature of the maxilla. Due to the small segment size, only one of the lateral arms was utilized for the transport DO. (**g**, **h**) Orthodontic traction was utilized to guide the tooth and associated bone and soft tissue, down into proper position

small, only one of the horizontal arms could be used on both sides, with only one screw placed into the bony segment apical to the implant, and two screws placed into the horizontal bone above the apices of the premolar teeth (Fig. 3.8m).

The site was closed and active DO was started 5 days after surgery. The distraction technique went well with the implant and surrounding bone being transported vertically towards the crest of the ridge with the adjacent soft tissue. The incisal edge of the crown was periodically reduced, to allow for vertical distraction, thus bringing the implant, along with the surrounding bone and soft tissue, down towards the crest of the ridge from its original submerged location (Fig. 3.8n). Although greatly improved, the final planned result was shy of the optimal vertical position in which the implant would be coincident with the remaining alveolar height (Fig. 3.8o, p). A 5-year follow-up shows maintenance of the implant health and osseous integrity in the new distracted site (Fig. 3.8q). Close evaluation of the radiographs revealed the following complications occurred, which limited the complete vertical distraction to the preplanned site:

1. The long lever arm of the DO device allowed for bending of the horizontal arms of the distraction device and rotation of the transport segment. The vertical portion of the DO device was placed near the cuspid fossa to avoid the nasal floor and take advantage of the piriform rim for esthetics and patient comfort. This placement did create a long lever arm of the horizontal portion of the DO device. Therefore as DO progressed, the horizontal DO arm bent during the later stages of active DO. Additionally as there was only one screw in the transport segment for fixation, the transport segment was able to rotate as the DO device was advanced. Note the angle change of the implant. Originally the implant was parallel with the roots of the adjacent teeth. At the end of DO, the implant was slightly angled from vertical (Fig. 3.8n–q).
2. VSP could have helped with the planning portion of this surgery, especially to create bone cuts as to allow more rigid fixation of the DO device. The newer Micro TRACK is ideal for this clinical situation. Additionally it must be remembered that during active DO, there is NOT a 1:1 correlation between the activation of the device and the amount of movement of the transport/DO site. Here this phenomenon was heightened as the lever arm from the vertical, activation site of the DO device was very long to the site of force application into the transport segment. With a longer lever arm, the amount of DO advancement per turn of device activation was significantly reduced.
3. Use of orthodontic traction would have helped guide the DO transport bone segment containing the implant into the final site (Fig. 3.9a–h).
   (a) A 42-year-old female presented for implant consultation. She had prior trauma to tooth # 8 as a child, resulting in ankyloses of the tooth in a more vertical position (Fig. 3.9a–c). This was camouflaged by placing acrylic on the incisal edge. With time, the tooth experienced internal resorption requiring removal. However, to achieve optimal bone and soft tissue contour, it was planned to distract the tooth and alveolus prior to extraction of the tooth (Fig. 3.9d).
   (b) A similar vertical and horizontal bone cuts were created, and using distraction osteogenesis, the tooth and alveolus were distracted vertically along with the soft tissue (Fig. 3.9e–g). Here, orthodontic guidance was used to assist in the path of draw of the transport segment (Fig. 3.9h). Additionally the new TRACK alveolar device (KLS Martin) with the vertical foot plate was utilized, which prevented lateral rotation of the DO device. The scalloped gingival contour was maintained and respotitoned vertically as a result of the distraction technique. Once osseous healing occurred, the tooth was removed and optimal implant reconstruction completed.

3.2 Case 2. Preprosthetic Augmentation

A 42-year-old male presented for implant reconstruction of three missing maxillary teeth: first bicuspid, canine, and lateral incisor. The patient had a history of wearing a removable partial denture such that there was adequate bone width, yet inadequate bone height and a "U"-shaped vertical alveolar deformity (Fig. 3.10a). The defect was appreciated as the patient could extend his tongue through the defect while in maximal occlusion (Fig. 3.10b).

Dentoalveolar distraction was planned and performed [6, 7]. Using a vestibular incision, the bone cuts were made, the DO device placed, path of draw verified, the site closed, and DO commenced after a 5-day latency (Fig. 3.10c, d). DO proceeded without incident continuing until the site was distracted fully, "over-distracted" with the regenerate extending beyond the crest of the alveolus (Fig. 3.10e). It has been suggested that the DO site should be planned for a 20% over-distraction to allow for maturation of the site prior to implant placement [16–19].The site was held in neutral fixation for osseous consolidation/healing. During the consolidation period, a bony protuberance was noted on the palatal (Fig. 3.10f). Additionally, exposure of the distraction device and screws were noted (Fig. 3.10g). The site was managed without incident and sufficient bone was generated through the DO process as to place three osseointegrated implants. This case highlights two complications that can occur during alveolar DO: 1. displacement of the small DO transport segment and 2. exposure of the distraction device and screws.

1. During the final stage of DO, the soft tissue pull upon the site of distraction can be considerable, especially in the alveolus where there is a significant difference in the tightness of the soft tissue: loose tissue buccal, and dense tissue on the palatal. Here, at the end of DO, when the DO device is fully expanded, the differential alveolar tissue pull, the effect of local muscles including the orbicularis oris and the patient's parafunctional habit of placing their tongue through the surgical site, allowed the DO device to "fall" toward the palate thus dislodging the transport disc/alveolar DO segment. This was managed by manually pushing the alveolar segment buccal as to align the site (Fig. 3.10h, i).
   (a) This complication was common with early cases of dentoalveolar DO, and led to the advancement of DO technology. The DO device was modified and a small footplate added to the base of the vertical portion of the TRACK distraction devices, as to prevent the tipping of the device and bone segment. Utilizing this footplate is essential to ensure clinical success with DO for preprosthetic augmentation [13–15].
   (b) Additionally, orthodontic and/or prosthetic appliances can be constructed to prevent this tipping and guide the transport disc to ideal position.
2. VSP would also help in this case, as the computer 3-D image would show that the "U"-shaped bony deformity was actually not uniform: the bone height was taller next to the central incisor as compared with the bone height adjacent to the bicuspid. Recognizing this would allow the surgeon to trim the bone slightly on the one edge of the distraction segment. The shape of the new alveolar bone can be visualized when the path of draw of the distraction device is verified, prior to closure of the site.
3. The exposure of the DO device plate and screw can occur. It is best to treat the site locally with chlorohexidine both as rinses and topically. If tissue tension is noted, the DO protocol can be modified to allow for smaller daily incremental advancements of the DO device. For example, ½ turn 4 times a day versus 1 turn twice a day. Slow application of the distraction force allows the soft tissue envelope to stretch and passively advance the osseous segment.

3.3 Case 3: Mandibular Distraction, Vector Control

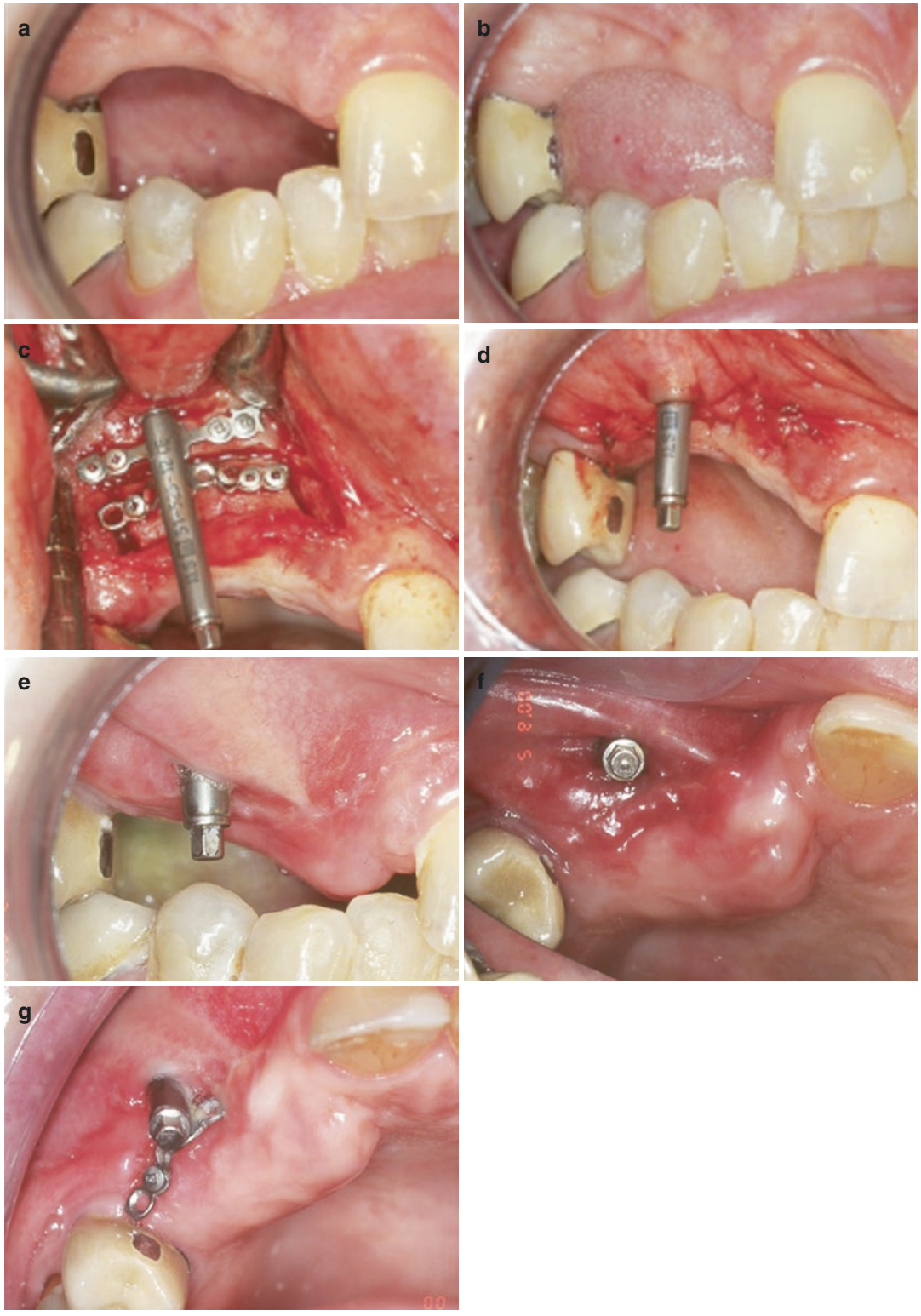
A 20-year-old female with mandibular hypoplasia underwent mandibular distraction. The mandibular DO proceeded uneventfully yet an open bite was created during the distraction process. Careful review of the radiographs revealed poor planning of the vector of DO as well as the effect of muscle pull on the distraction site. This case was managed with the removal of the DO device prior to the completion of the consolidation phase, and elastic traction; "bone floating" was performed to close the open bite [20–23].

1. Review of the radiographs revealed that the DO device has been placed with the DO device oriented more parallel to the inferior boarder rather than more parallel with the occlusal plane. As distraction advanced, the mandible moved in a forward and downward direction (Fig. 3.11a). Additionally, the osteotomy was placed anterior to the masseteric muscle sling such that the proximal segment was influenced by vertical muscle pull, and the distal segment affected by the supra-hyoid muscles in an inferior direction both contributing to an open bite (Fig. 3.11b). As this case was early in the evolution of DO devices, the number of screw holes available in the footplates of the DO device were few in number. This led to the development of DO devices designed with larger array of footplate screw fixation sites.
2. VSP would aid in the prevention of this complication. However, attention to detail of the location of the osteotomy versus the location of potential muscle pull vectors must be maintained. In this case, the open bite only became apparent after active DO was completed and the site held in neutral fixation as to allow ossification of the regenerate. As the open bite was noticed early, the regenerate could be manipulated and correct the complication. Here the DO device was removed prior to complete ossification, and using elastic traction, the open bite was closed and elastic force applied until consolidation was complete (Fig. 3.11c).

3.4 Case 4: Maxillary DO and Arc of Rotation Around First Molar

Maxillary DO has changed the treatment options especially for severe maxillary cleft lip and palate hypoplasia and other craniofacial deformities [24–28]. Even from the early experience with maxillary DO using a Petit Delaire mask in the nonsyndromic patient, it was noted that when slow force is applied to the freed maxilla, an anterior open bite usually occurs (Fig. 3.12a, b). This is because there is an arc of rotation of the maxilla centered above the root of the maxillary first molar, rotating the maxilla in a counterclockwise vector to produce an open bite [29]. A 13-year-old female presents with her mother for maxillary DO. She is status post repair of a bilateral cleft lip and pate and is in need of 15+

Fig. 3.10 (**a**, **b**) A patient with a "U"-shaped alveolar defect was evaluated for distraction osteogenesis. The defect was large enough to allow the patient to protrude his tongue while in occlusion. (**c**, **d**) The bone cuts were created using a vestibular incision and the distraction device adapted and placed. Note the distraction segment is trapezoidal in shape with the alveolar height is taller adjacent to the central incisor (**c**). The site was closed and distraction proceeded without incident (**d**). (**e**) The site was over-distracted such that the segment was distracted vertically above the level of the CEJ of the adjacent teeth. On the mesial, a triangular bony protuberance was noted. This protuberance occurred as the defect was "U" shaped. Consideration for trimming of such bony irregularities/sites at the time of surgery should be included in the treatment plan. (**f**) The bony protuberance is also noted on the palatal, highlighting the need to plan the distraction segment in 3-D. (**g**) During the portion of active distraction and during consolidation, a small portion of the DO device arm became exposed. Exposure is controlled with local measures. Note the gingiva is pink and not inflamed/infected despite the exposure


a
b
c
d
e
f
g

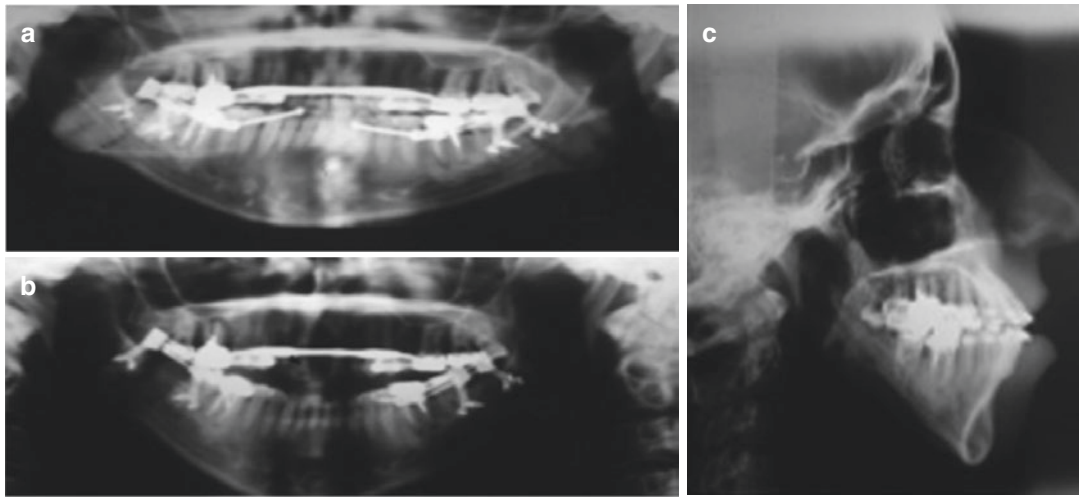


Fig. 3.11 (**a**, **b**) A 20-year-old patient underwent mandibular distraction. The vector of the DO device was not ideal, with the vector parallel to the steep mandibular plane angle (**a**). Consequently an open bite occurred during active DO (**b**). (**c**) The open bite was addressed by removing the DO device prior to complete consolidation, and using elastic/orthodontic traction, the open bite was closed

mm advancement of the maxilla (Fig. 3.12c). She underwent Le Fort I maxillary advancement using the RED device (KLS Martin) [30–32] (Fig. 3.12d). The DO technique proceeded uneventful, and she did well so that after maxillary DO, her facial form married that of her mother (Fig. 3.12e, f). This case highlights a commonly overlooked complication of maxillary DO: the potential for the creation of an open bite.

1. The arc of rotation of the maxilla around the skull base is centered just above the maxillary first molar. This phenomenon is commonly observed with maxillary advancement including the use of a frame such as a Petit DeLaire mask. All patients undergoing maxillary DO using either intraoral or external devices should be carefully monitored and followed for this occurrence. Using an external halo frame, RED device, forces to the maxilla can be adjusted to allow for the DO advancement of the maxilla uneventfully adjusting the arms for elastic traction inferiorly as DO progresses. When using an intraoral device, care must be taken during the planning stage to orient the device in a vector to counteract this usual arc of rotation. For intraoral devices, elastic traction can be used but is not as effective as direct device reorientation, as can be accomplished with the RED, halo device. For either intraoral or extra-oral devices, at the time of maxillary DO device removal, additional intraoral elastic traction can be used via orthodontic appliances to address any residual concerns.

Distraction osteogenesis is a powerful tool to correct bone and soft tissue deformities associated with the craniofacial skeleton. As such, the technique is intuitive as DO correlates with conventional orthognathic surgery. With the advent of virtual surgical planning, VSP, and newer DO devices, many of the complications encountered by early DO techniques have been obviated. Yet close attention to detail must be maintained throughout the entire DO process. The rate for DO of the craniofacial skeleton has been established at 1.0 mm per day, yet a rate of 2 mm per day is suggested for children less than 12 months of age [11]. Yet should activation of the device become difficult, especially near the end of the planned distraction, then premature ossification should be considered.

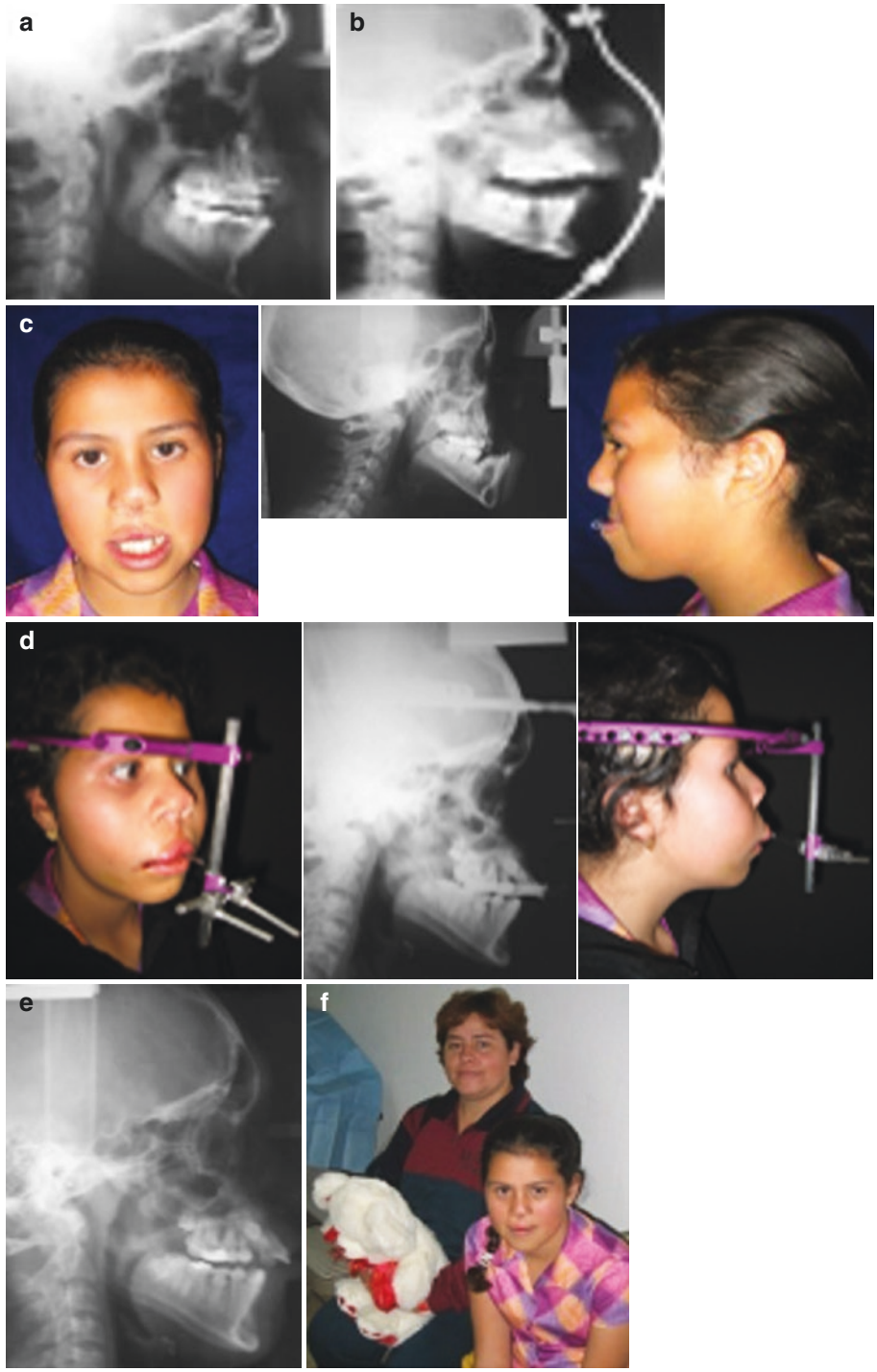


Fig. 3.12 (**a**, **b**) Open bite occurs with DO, either via skeletal halo frame (**a**) or tooth bone (**b**) Petit Delaire devices as the maxilla rotates around a point centered above the maxillary first molar. (**c**) A 13-year-old female with repaired cleft lip-cleft palate presents with severe maxillary hypoplasia. (**d**) She underwent maxillary Le Fort DO for maxillary advancement. The open bite was corrected during active DO, by adjusting the vertical and horizontal aspects of the traction arms. (**d**, **e**) The maxilla was overcorrected during DO to achieve a positive result. (**f**) Facial harmony is resorted after maxillary distraction; now the child's face mirrors her mother's

This attention to detail is paramount after active DO, during consolidation when both the surgical team and patient/parent of a patient are more "relaxed" often assuming that the only challenge is the final osseous healing. It is during this time of neutral DO fixation that the device is fully extended, and the regenerate is malleable that forces can affect the shape of the mandibular regenerate. Some have reported early open, surgical callus manipulation as to obtain the desired functional and esthetic results [21, 23]. Consequently all extrinsic forces, especially the local muscles attached to the distal bony DO site, can work and pull to affect the final shape and positon of the distracted bone. Close observation during this and all time periods associated with the DO process can avoid these muscular forces as well as to intervene and correct for them as necessary. This may necessitate early device removal and placing elastic traction to allow the bone to be guided to its final, correct position. These incidents are usually minor in nature and easily addressed [33]. Both the patient and or the parents of the patient are a useful member of the team as to identify and assist in the shaping of the final regenerate form. Active involvement and observation by the patient and family is encouraged. Long-term follow-up is recommended as active physical therapy may be required to overcome learned muscle motion such as deviation with opening, which has been associated with "late relapse" of mandibular distraction [20]. Simple techniques such as chewing gum placed on the contralateral side of the deviation with opening will assist in avoiding this occurrence. TMJ ankyloses has been reported, however rarely, after mandibular DO [34]. This too can be avoided with active opening exercises during and long term after DO. It cannot be assumed that a congenital deformity be overcome with DO during infancy/early childhood, without observation and gentle orthodontic/orthopedic therapy during growth.

Interestingly, unlike mandibular DO where vectors are influential, relapse is the primary concern for both maxillary DO, at all levels Le Fort I, II, and III as well as vertical alveolar DO. It has thus been suggested that for dentoalveolar DO, the site should be over-distracted by 20% as to account for the potential vertical relapse [12]. Yet, once an implant is placed in the new, DO-generated bone, the bone continues to mature and acts as the native bone with similar implant success rates. Similarly, maxillary DO is stable once the tenancy for the rotation around the maxillary first molar during active DO has been accounted for. Yet age-related relapse has been reported for treatment of cleft maxillary hypoplasia, with the least amount of relapse occurred when the surgery was performed when the child was 11–15 years old 6% versus 16–25% for all other age groups [35]. This may be due to the nature of a multiple operated site associated with cleft maxillary hypoplasia. Periodic follow-up is recommended for all patients after DO until the surgeon is satisfied the incidence of long-term occurrences is rare.

Dentoalveolar DO has two unique complications reported: tipping of the distraction segment 16% and fracture of the basal bone 2% occurrence [18]. Fracture of the basal bone can be obviated by avoiding sharp internal line angles to the osseous cut [20]. Our tendency is to create a "box like cut" to the DO segment (Fig. 3.13). For the maxilla this is less critical. Yet there is unique muscle pull on the mandible exerting compressive/tension forces on the superior boarder and expansive forces on the

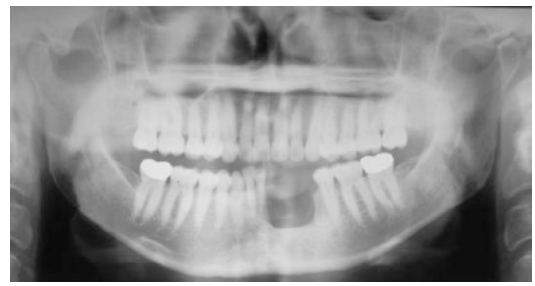


Fig. 3.13 For alveolar DO, the osteotomy created should have rounded internal lines as to avoid stress concentration at the corners of the osteotomy. Here the DO is fully extended, with the transport disc at the level of the alveolus and the osseous regenerate site radiographically darker, until it ossifies

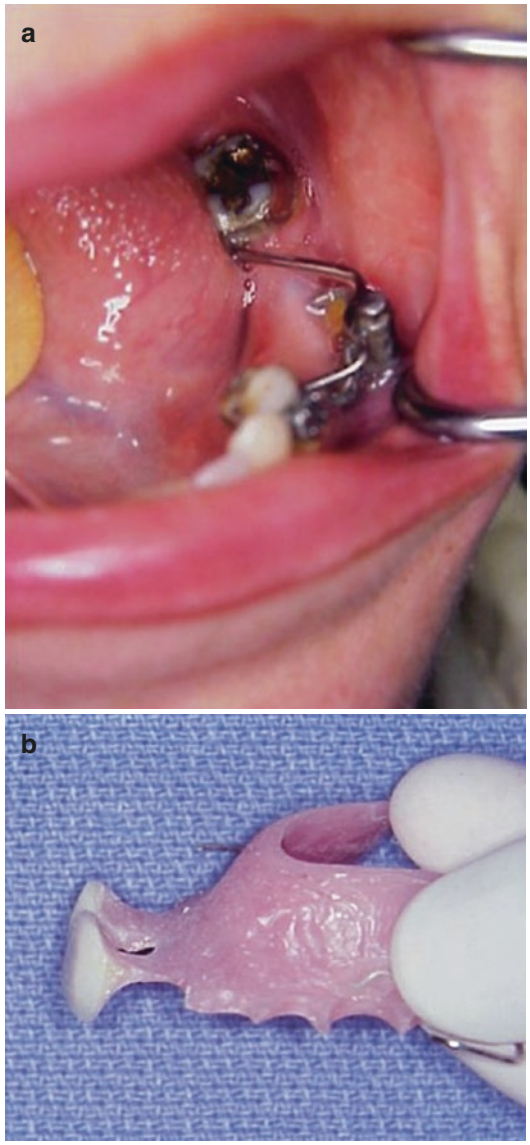


Fig. 3.14 (**a**, **b**) Appliances are created to stabilize the vertical DO activation arm here with an orthodontic retained design (**a**), or using a prosthetic device with an access hole (**b**)

inferior boarder of the mandible as one opens and closed their mouth. These forces are transmitted throughout the bone such that a box-shaped osteotomy for DO can lead to fracture of the basal bone right at the internal angle/junction of the horizontal and vertical components of the osteotomy. Rounded, "U"-shaped internal line angles are suggested for alveolar DO. The most common minor complication associated with alveolar DO is displacement of the transport segment. This can be overcome with the use of orthodontic or prosthetic guidance appliances (Fig. 3.14a, b).

Distraction osteogenesis is a powerful tool as it allows for the reconstruction of all tissues in and adjacent to the surgical site (Fig. 3.12a–f). However used in the growing child, it must be recognized that a second orthognathic procedure may be revised later, at the end of normal physiologic growth [36, 37]. It has been shown that DO itself does not hinder normal growth of the site such that early correct of a dentofacial deformity can improve socialization and self-perception as the child progresses in school.

References

1. McCarthy JG. Our surgical past: an aid to understanding the present and a guide to the future. J Craniofac Surg. 2017;28(5):1121–2. https://doi.org/10.1097/SCS.0000000000003806.
2. Vyas RM, Alperovich M, Grayson BH, McCarthy JG, Rodriquez ED. From multidisciplinary to interdisciplinary to transdisciplinary care: an evolution in craniofacial surgery. Plast Reconstr Surg. 2015;135(4):796e–7e. https://doi.org/10.1097/PRS.0000000000001137.
3. Rossini G, Vinci B, Rizzo R, Pinho TM, Deregibus A. Mandibular distraction osteogenesis: a systematic review of stability and the effects on hard and soft tissues. Int J Oral Maxillofac Surg. 2016;45(11):1438–44. Epub 2016 Sep 5. https://doi.org/10.1016/j.ijom.2016.08.012.
4. Archual A, Black JS. Avoidance of tracheostomy using mandibular distraction in an infant with severe condylar dysplasia and airway obstruction. J Craniofac Surg. 2017;28(8):2053–5. https://doi.org/10.1097/SCS.0000000000003978.
5. da Costa A, Manica D, Schweiger C, Kuhl G, et al. The effect of mandibular distraction osteogenesis on airway obstruction and polysomnographic parameters in children with Robin sequence. J Craniomaxillofac Surg. 2018;46(8):1343–7. Epub 2018 May 18. https://doi.org/10.1016/j.jcms.2018.05.030.
6. Wang X, Feng S, Tang X, Shi L, et al. Incidents of mandibular distraction osteogenesis for hemifacial microsomia. Plast Reconstr Surg. 2018;142(4):1002–8. https://doi.org/10.1097/PRS.0000000000004812.
7. Moran I, Virdee S, Sharp I, Sulh J. Postoperative complications following LeFort 1 maxillary advancement surgery in cleft palate patients: a 5-year retrospective study. Cleft Palate Craniofac J. 2018;55(2):231–7.

https://doi.org/10.1177/1055665617736778. Epub 2017 Dec 14.
8. Rougier G, Diner PA, Rachwalski M, Galliani E, Tomat C, et al. Mandibular symphyseal distraction osteogenesis: 20 years of experience treating transverse deficiencies with an internal hybrid device. J Craniomaxillofac Surg. 2019;47(4):586–91. https://doi.org/10.1016/j.jcms.2019.01.001. Epub 2019 Jan 11.
9. Humphries LS, Yates EK, Mhlaba JM, Collins JM, Baroody FM, Reid RR. Airway volume simulation in virtual mandibular distraction: a cohort study. Plast Reconstr Surg. 2018;141(4):1003–9. https://doi.org/10.1097/PRS.0000000000004249.
10. Şensoy AT, Kaymaz I, Ertaş Ü, Kiki A. Determining the patient-specific optimum osteotomy line for severe mandibular retrognathia patients. J Craniofac Surg. 2018;29(5):e449–54. https://doi.org/10.1097/SCS.0000000000004470.
11. Breik O, Tivey D, Umapathysivam K, Anderson P. Does the rate of distraction or type of distractor affect the outcome of mandibular distraction in children with micrognathia? J Oral Maxillofac Surg. 2016;74(7):1441–53. https://doi.org/10.1016/j.joms.2016.01.049. Epub 2016 Feb 2.
12. Elnayef B, Monje A, Gargallo-Albiol J, Galindo-Moreno P, et al. Vertical ridge augmentation in the atrophic mandible: a systematic review and meta-analysis. Int J Oral Maxillofac Implants. 2017;32(2):291–312. https://doi.org/10.11607/jomi.4861.
13. Kofod T, Würtz V, Melsen B. Treatment of an ankylosed central incisor by single tooth dento-osseous osteotomy and a simple distraction device. Am J Orthod Dentofacial Orthop. 2005;127(1):72–80.
14. Kisnisci RS, Iseri H. Dentoalveolar transport osteodistraction and canine distalization. J Oral Maxillofac Surg. 2011;69(3):763–70. https://doi.org/10.1016/j.joms.2010.11.035.
15. Senışık NE, Koçer G, Kaya BÜ. Ankylosed maxillary incisor with severe root resorption treated with a single-tooth dento-osseous osteotomy, vertical alveolar distraction osteogenesis, and mini-implant anchorage. Am J Orthod Dentofacial Orthop. 2014;146(3):371–84. https://doi.org/10.1016/j.ajodo.2013.10.02.
16. Jensen OT, Block M. Alveolar modification by distraction osteogenesis. Atlas Oral Maxillofac Surg Clin North Am. 2008;16(2):185–214. https://doi.org/10.1016/j.cxom.2008.04.001.
17. Reininger D, Rodriguez-Grandjean A, López-Quiles J. Analysis of resorption and need for overcorrection in alveolar distraction osteogenesis. Int J Oral Maxillofac Implants. 2016;31(4):865–9. https://doi.org/10.11607/jomi.4424.
18. Zhao K, Wang F, Huang W, Wu Y. Clinical outcomes of vertical distraction osteogenesis for dental implantation: a systematic review and meta-analysis. Int J Oral Maxillofac Implants. 2018;33(3):549–64. https://doi.org/10.11607/jomi.6140.
19. Block MS. Dental implants: the last 100 years. J Oral Maxillofac Surg. 2018;76(1):11–26. https://doi.org/10.1016/j.joms.2017.08.045. Epub 2017 Oct 13.
20. Stucki-McCormick SU, Fox RM, Mizrahi R, Erikson M. Distraction osteogenesis for congenital mandibular deformities. Atlas Oral Maxillofac Surg Clin North Am. 1999;7(1):85–109.
21. Hoffmeister B. The effect of sagittal orientation of the distractor on the biomechanics of mandibular lengthening. J Oral Maxillofac Surg. 1999;57(10):1221–2.
22. Samchukov ML, Cope JB, Cherkashin AM. The effect of sagittal orientation of the distractor on the biomechanics of mandibular lengthening. J Oral Maxillofac Surg. 1999;57(10):1214–22; discussion 1221-2.
23. Pereira AR, Neves P, Rosa J, Bartlett S. Curvilinear segmental mandibular reconstruction utilizing distraction osteogenesis and early open callus manipulation. Plast Reconstr Surg Glob Open. 2017;5(1):e1229. eCollection 2017 Jan. https://doi.org/10.1097/GOX.0000000000001229.
24. Guerrero CA. Cleft lip and palate surgery: 30 years follow-up. Ann Maxillofac Surg. 2012;2(2):153–7. https://doi.org/10.4103/2231-0746.101342.
25. Molina F. From midface distraction to the "true monoblock". Clin Plast Surg. 2004;31(3):463–79, vii.
26. Arnaud E, Marchac D, Renier D. Reduction of morbidity of the frontofacial monobloc advancement in children by the use of internal distraction. Plast Reconstr Surg. 2007;120(4):1009–26.
27. Mathijssen I, Arnaud E, Marchac D, Mireau E, Morisseau-Durand MP, Guérin P, Renier D. Respiratory outcome of midface advancement with distraction: a comparison between Le Fort III and frontofacial monobloc. J Craniofac Surg. 2006;17(4):642–4.
28. Figueroa AA, Polley JW, Figueroa AD. Biomechanical considerations for distraction of the monobloc, Le Fort III, and Le Fort I segments. Plast Reconstr Surg. 2010;126(3):1005–13. https://doi.org/10.1097/PRS.0b013e3181e3b70f.
29. Staggers JA. From MS Thesis, Orthodontics. Personal communication, private practice Winchester, VA. 2006.
30. McCarthy JG, Stelnicki EJ, Mehrara BJ, Longaker MT. Distraction osteogenesis of the craniofacial skeleton. Plast Reconstr Surg. 2001;107(7):1812–27.
31. Troulis MJ, Padwa B, Kaban LB. Distraction osteogenesis: past, present, and future. Facial Plast Surg. 1998;14(3):205–15.
32. Nout E, Wolvius EB, van Adrichem LN, Ongkosuwito EM, van der Wal KG. Complications in maxillary distraction using the RED II device: a retrospective analysis of 21 patients. Int J Oral Maxillofac Surg 2006;35(10):897-902. Epub 2006 Sep 27.
33. Verlinden CR, van de Vijfeijken SE, Tuinzing DB, Becking AG, Swennen GR. Complications of mandibular distraction osteogenesis for acquired deformities: a systematic review of the literature. Int J Oral Maxillofac Surg. 2015;44(8):956–64. https://

doi.org/10.1016/j.ijom.2014.12.008. Epub 2015 Apr 1.
34. Galié M, Candotto V, Elia G, Clauser LC. Temporomandibular joint ankylosis after early mandibular distraction osteogenesis: a new syndrome? J Craniofac Surg. 2017;28(5):1185–90. https://doi.org/10.1097/SCS.0000000000003612.
35. Richardson S, Krishna S, Khandeparker RV. A comprehensive management protocol to treat cleft maxillary hypoplasia. J Craniomaxillofac Surg. 2018;46(2):356–61 . Epub 2017 Dec 13. https://doi.org/10.1016/j.jcms.2017.12.005.
36. Ruiz RL, Turvey TA, Costello BJ. Mandibular distraction osteogenesis in children. Oral Maxillofac Surg Clin North Am. 2005;17(4):475–84.
37. Zhang RS, Lin LO, Hoppe IC, Swanson JW, Taylor JA, Bartlett SP. Early mandibular distraction in craniofacial microsomia and need for orthognathic correction at skeletal maturity: a comparative long-term follow-up study. Plast Reconstr Surg. 2018;142(5):1285–93. https://doi.org/10.1097/PRS.0000000000004842.

4 Complications in Orthognathic Surgery

Roger William Fernandes Moreira,
Sergio Monteiro Lima Jr,
and Fernanda Brasil Daura Jorge Boos Lima

Contents

R. W. F. Moreira (✉)
OMFS Red Cross Hospital, São Paulo, SP, Brazil

S. M. Lima Jr
OMFS MaterDei HealthCare Network,
Belo Horizonte, Minas Gerais, Brazil

OMFS, Federal University of Minas Gerais,
Belo Horizonte, Minas Gerais, Brazil

F. B. D. J. B. Lima
OMFS, Federal University of Minas Gerais,
Belo Horizonte, Minas Gerais, Brazil

4.1 Introduction

Orthognathic surgery is a set of facial-bone surgical procedures with esthetical and functional aims. Like any other surgical procedure, orthognathic surgery has complications that must be approached as soon as possible to avoid definitive sequels to the face and mouth. A complication can be defined as an unwanted or unpredictable event that increases the patient's morbidity after

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_4

surgery. Complications may be a consequence of problems in tissue repair, may be a consequence of the disease, homeostasis, or even surgeon's errors. Orthognathic surgery complications may also come from errors during planning. A complication may happen during surgery, which can be called as a surgical accident, or during the postoperative period.

A search in the literature shows that classification and methods vary a lot between studies, making it difficult to organize and separate strong scientific evidence of complications in orthognathic surgery. Also, most of the manuscripts published are case reports or case series. It is important to make the reader understand that a complication is different from a consequence of the surgical technique. One of the most common subjects discussed as a complication is the neurosensory deficit of the lower lips after bilateral sagittal split osteotomies (BSSOs). One must keep in mind that paresthesia after a BSSO, is a consequence of the sprocedure and not necessarily a complication. A complication may occur when the nerve is inadvertently sectioned.

Therefore, in order to bring the best possible evidence and an approach directed to complication treatment, this chapter focused in possible intraoperative and postoperative complications and how to treat them, surgically or not, based on available published information and clinical experience.

In general, one can affirm that complication rates during or after orthognathic surgery are low, varying from 3 to 12% [1, 2]. Neurosensory disturbances are significantly associated with mandibular surgery, and intraoperative complications have a lower incidence when compared to postoperative complications [3]. Major complications are rare, and for such, the literature agrees that orthognathic surgery is considered a safe procedure for patients.

Factors that affect complication rates include surgeon's experience, length of surgery, and type of surgery. Surgeon's experience is directed correlated with duration of surgery [4, 5]. The longest the surgery, the more prone to contamination and infection. Also, experienced surgeon treats soft tissues better, decreasing the risk of laceration and/or hypoxemic lesions of the incision.

Protocols for patient security are the best way to prevent complications such as foreign bodies, ulcers, and procedures on the wrong body side. International guidelines are available to prevent such problems, like corneal ulcers and operating on the wrong temporomandibular joint [6, 7]. Complications other than those directly associated with the orthognathic surgery, like deep vein thrombosis, recurrent laryngeal nerve palsy, and arytenoid cartilage dislocation, will not be discussed here.

Before discussing specific complications, surgeons should remember that ethics is absolutely necessary in order to disclose any complication to a patient. An informed consent that contains all complications and alternative procedures must be obtained before surgery, so that patients can make informed decisions. After surgery, surgeons should clearly disclose any complication, if it happens. Keeping a good medical–patient relationship will make treatment of a complication, including reoperation, easier. It will also improve patient cooperation during the postoperative period. Training, study, and delivery of a clear message to the patient can avoid legal problems, especially in profit-based health treatment delivery, including recklessness and malpractice. Most of the complications of orthognathic surgery are described below.

4.2 Bad Split of the Mandible

The bilateral sagittal split osteotomy (BSSO) was first described by Trauner and Obwegeser [8–10] during the 1950s, and after many modifications [11–14], it is one of the most performed procedures in orthognathic surgery, because it is versatile, allowing large movements in virtually any direction, and it can be completely performed through an intraoral incision and allows easy application of internal fixation, making immediate postoperative stability easier.

The modifications of the BSSO made it longer, increasing bone contact between fragments. Therefore, the longer the osteotomy, the longer

the cut at the inferior border of the mandible (Fig. 4.1). A bad split of the BSSO occurs when the inferior border of the mandible is incompletely transected [15, 16], and an undesirable fracture of the proximal segment, that contains the condyle, happens. This bad split usually presents as a fracture of the distal segment of the buccal plate [17].

Understanding the evolution of the BSSO helps the surgeon to treat this intraoperative accident easier. Obwegeser initial report of BSSO [9, 10] described a short osteotomy at the lateral aspect of the mandible, with the cortical bone cut being performed horizontally at the middle of the ramus. A modern design of the BSSO osteotomy is usually made with a reciprocating saw, and the bone cut laterally is extended near the first molar (Fig. 4.2). If the surgeon has the modern design and the initial Obwegeser design in mind when a bad split occurs, the better way to finish the split is to continue the surgery as a classic Obwegeser osteotomy. The fractured buccal plate is then removed, and fixation is applied over it (Fig. 4.3). After plating the fractured buccal plate, it is replaced and stabilized within the ramus. After this reconstruction, the complete BSSO can be then stabilized with plates and screws as an uncomplicated BSSO (Fig. 4.4). In this way, there will be no prejudice in the amount of movement planned for the osteotomy. A similar technique was described in 2004 [18].

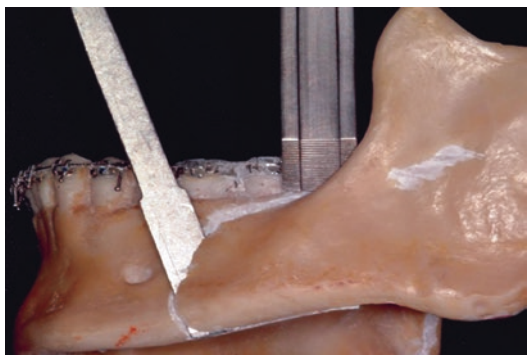


Fig. 4.1 Cut of the inferior border of the mandible to prevent a bad split during BSSO

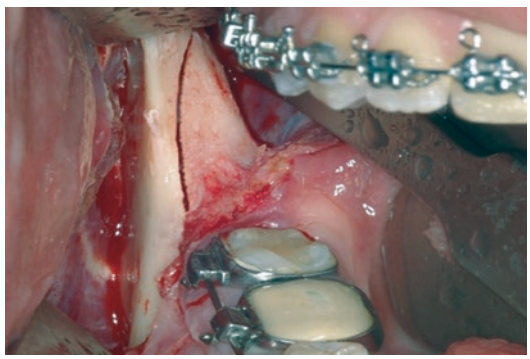


Fig. 4.2 Ideal design of the BSSO. The osteotomy is performed with a reciprocating saw

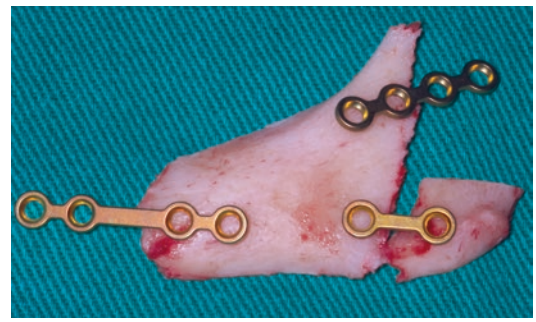


Fig. 4.3 Bad split of a left sagittal split osteotomy. The image shows the bone fragment with plates on ideal position, before fixation. The fixation applied used the fragment as a bridge. After securing the segment with two small plates on the right side of the picture, another plate was applied on the left side of the picture to stabilize the osteotomy

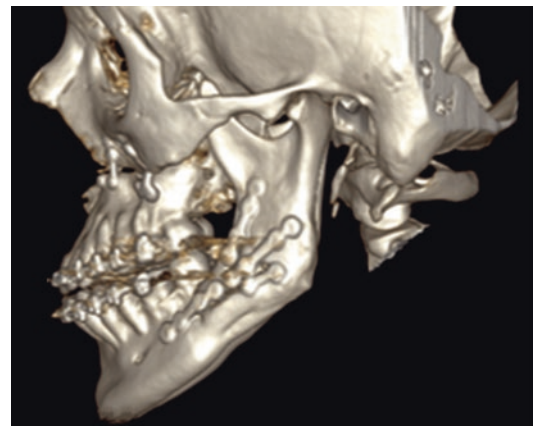


Fig. 4.4 Postoperative CT of the bad split shown in Fig. 4.3. The distal fractured segment was fixed using two extra miniplates and screws

If a bad split occurs near the condyle, a decision must be made whether to stabilize it with plates and screws or not. If technically difficult, an option will be a postoperative intermaxillary fixation for 30–45 days to allow bone repair.

4.3 Bad Split of the Chin

A bad split of the horizontal chin osteotomy is a rare event. The literature is poor in describing it. Figure 4.5 shows a bad split that occurred because of two errors during surgery: first, the osteotomy line was made above the mental nerve. Second, the inferior border of the mandible was not completely cut with the reciprocating saw. The sum of these two factors led to the bad split seen. After the bad split, the inferior border was aligned by means of drills to allow good bone contact, and the chin was advanced as planned. It healed uneventfully, without esthetic complaints. The paresthesia from the right mental nerve resolved after 4 months.

A bad split of the inferior border during a chin osteotomy may cause problems in adapting the distal (chin) segment, which will lead to gaps and vertical asymmetries. Therefore, in order to avoid such complication, the surgeon must completely cut the inferior border until the distal segment is released. A bone cut like this will ensure good and symmetrical bone contact.

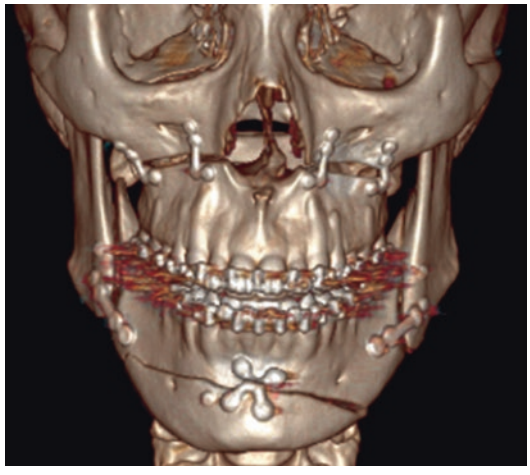


Fig. 4.5 Bad split of chin osteotomy

4.4 Condylar Malposition

A condylar malposition in orthognathic surgery will lead to a relapse in occlusion. If the condyle is not properly positioned at the mandibular fossa during intermaxillary fixation and mandibular plating, as soon as the patient awakes, the temporomandibular joint (TMJ) will move to a more functional and stable position, leading to malocclusion. This relapse in occlusion that follows a change in condylar position is also known as condylar sag [19].

Reyneke et al. [19] classifies sag as central or peripheral (types I and II). Central condylar sag occurs when the condyle makes no contact with the fossa. Peripheral condylar sag type I occurs when the condyle makes a peripheral contact with the fossa, while peripheral condylar sag type II occurs when tensions of the fixation move the condyle away from the fossa after releasing the intermaxillary fixation (Fig. 4.6).

Condylar malposition classified as a central condylar sag will present as an immediate postoperative malocclusion because the condyle will assume a more stable position, seated in the mandibular fossa, as soon as the patient awakens from general anesthesia. A condylar malposition where a contact still exists between condyle and fossa will appear as a late complication, because of condylar remodeling during follow-up. A condylar malposition needs

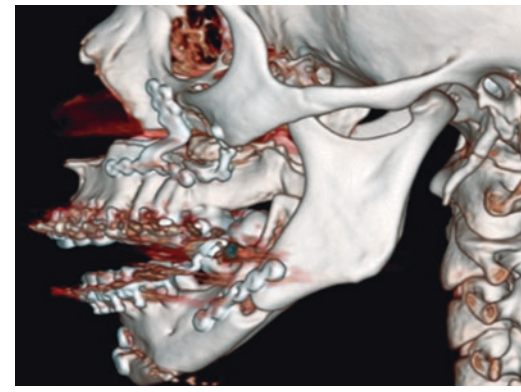


Fig. 4.6 Condylar malposition after orthognathic surgery (peripheral condylar sag type I). Note the open bite and the condyle in an advance position. This complication was treated by reoperation of the mandible

surgery as soon as possible to reposition the condyle and replace plates and screws. Navigation and transoperative images may be the best way to prevent such kind of complication, since the surgeon is able to diagnose and treat it before surgery ends [20].

4.5 Condylar Resorption

Condylar resorption is one of the most common causes of long-term relapse in orthognathic surgery, although its incidence is small [21]. Condylar remodeling should be differentiated from condylar resorption. The first one is expected to have no loss to the clinical result, especially occlusion and mandibular position, while condylar resorption will have adverse effects on the final clinical result of orthognathic surgery. Condylar resorption after orthognathic surgery is an uncommon but recognized clinical problem affecting the TMJ. It is defined as a progressive alteration of shape and volume of the mandibular condyles following orthognathic surgery [22]. Condyle resorption causes a decrease in ramus height, a progressive mandibular retrusion followed by a class II malocclusion, and an anterior open bite.

Literature shows that patients submitted to maxillary impaction and mandibular advancement are positively correlated with postoperative condylar resorption (Fig. 4.7) [22, 23]. Mandibular advancements greater than 10 mm increase in 20 times the risk for such complication [24]. Other factors associated with condylar resorption are age, gender [23, 25], class II long-face dentofacial deformity patients [23, 25, 26], condylar shape and neck inclination [23, 27], estrogen deficiency [28], TMJ dysfunction, and internal derangements [29].

The treatment of condylar resorption varies in the literature. The resorbed TMJ can be treated by surgery or by clinical therapy. Surgery is indicated when radiological and clinical stabilization of the TMJ is present. The patients should undergo another mandibular advancement by means of a BSSO, often followed by TMJ surgery. Orthognathic surgery alone may be indicated to treat the relapse when the surgeon does not recognize risk factors for condylar resorption [30].

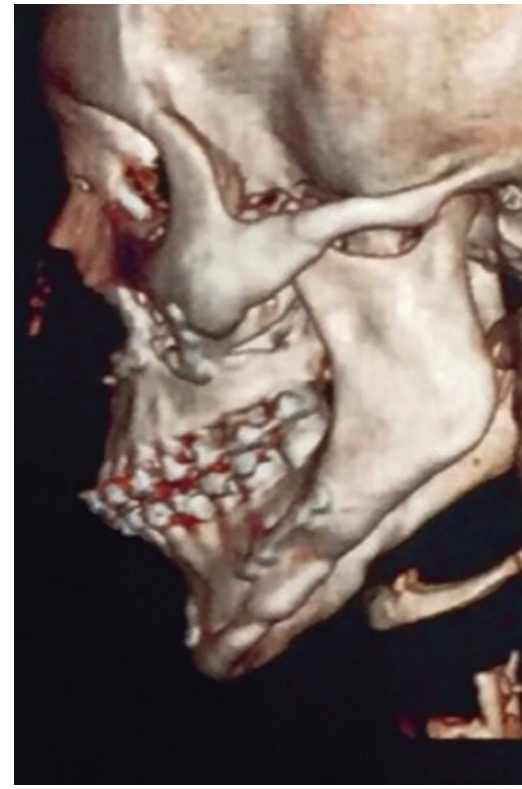


Fig. 4.7 Condyle resorption and relapse following orthognathic surgery—maxillary impaction, mandibular, and chin advancement associated with counterclockwise rotation of the maxillomandibular complex. Note a flat condyle morphology and loss of articular space is visible in the 3D computed tomography

The TMJ disc plays a crucial role in joint function, that reducing loads on the underlying bone and contributing to bone remodeling [31]. If a magnetic resonance image proves the presence of a dislocated health disc, it is logical to perform a surgery to reposition the disc, whether by open or arthroscopic techniques (Fig. 4.8) [29, 32, 33]. TMJ total joint prosthesis are also indicated, especially in severe cases of resorption, where the condyle has lost most of its volume, and the disc has perforations or deformations [33]. A recent systematic review has shown the stability of approaching the TMJ in conjunction with orthognathic surgery to avoid relapse [33].

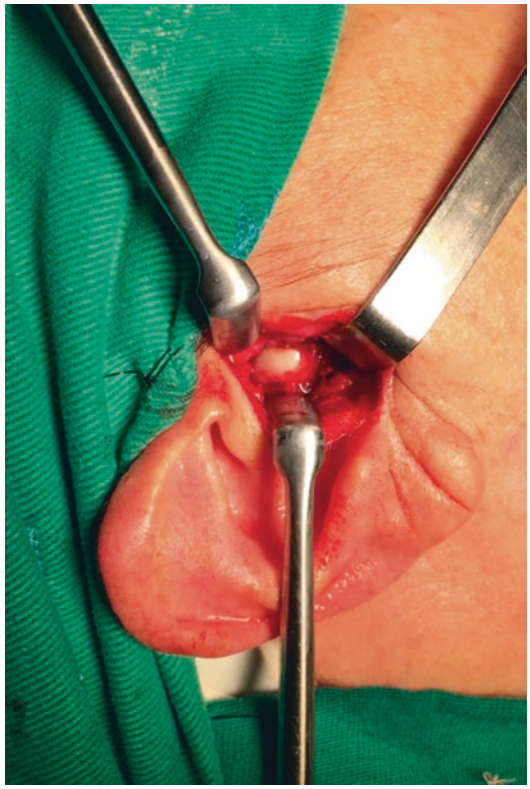


Fig. 4.8 The patient from Fig. 4.7 underwent another mandibular advancement and open surgery disc reposition. The patient is stable after 2 years of second surgery

The clinical treatment of condylar resorption is pharmacologically based, targeting specific pathways to disturb or interrupt bone resorption [34]. The article shows promising results, but randomized controlled clinical trials are still necessary to understand the role of each drug to prevent further condylar resorption.

4.6 Failure of Fixation

Fixation of the BSSO can be performed in many ways, including one or two miniplates with monocortical screws, bicortical screws in line or in an inverted L arrangement, and the hybrid technique—which uses one plate with monocortical screws and at least one extra bicortical screw. The fixation of the Le Fort I maxillary osteotomy is usually performed with four L plates at canine and zygomatic buttresses, bilaterally. Understanding how a plating system works under mechanical forces is essential to understand and choose between techniques of fixation. However, the failure of fixation is not only because of mechanical overload but also a sum of patient's factors, like muscular forces and amount of advancement.

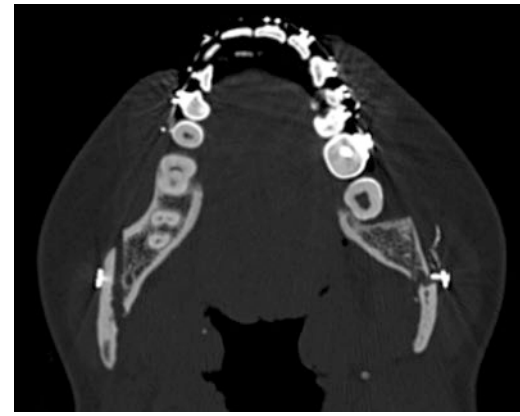


Fig. 4.9 Loosening of screws of the right sagittal split osteotomy two weeks after orthognathic surgery. The patient presented with malocclusion

Three main reasons are the cause of failure of fixation in orthognathic surgery. The first is the use of an inadequate fixation, which is not able to hold forces on maxilla and mandible. The second cause is a change in stress distribution after releasing the intermaxillary fixation, leading to forces that may cause failure. The third cause is an injured soft tissue over the plates and screws. An injury could be ischemia, infection, or even lack of tissues to adequately cover the fixation.

In a study published by Hammer and colleagues, screw loosening was always associated with a chronic infection, causing fistula. These authors, however, state that infection may not be the cause of loosening [35]. This information can be extrapolated to an orthognathic surgery patient. After a screw is loosened, the soft tissue around it starts an inflammatory reaction that leads to pain and local edema (Fig. 4.9).

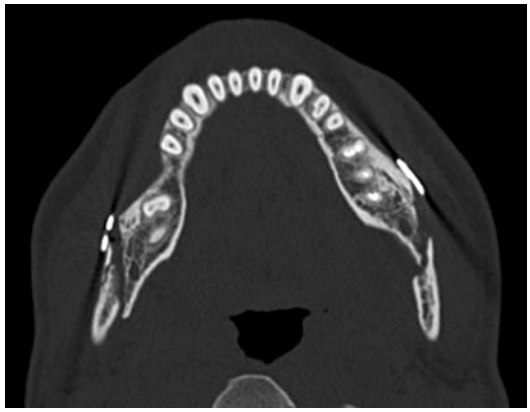


Fig. 4.10 Failure of mandibular osteosynthesis. The probable cause of failure in this patient was an incidence of unwanted forces over the system that overcome the system resistance. Note the midline shift to the left and an asymmetry at the osteotomy gaps

A clinical comparison between three methods of fixation for BSSO showed no difference in relapse after 6 months. The comparison was made between one plate and monocortical screws, three bicortical screws, and the hybrid technique [36]. Similar clinical results are found elsewhere in the literature. Biomechanical tests showed that inverted L arrangement and hybrid method of fixation delivered the better results in load peak and peak displacement and better stress distribution in finite element analysis [36, 37]. A systematic review showed that two miniplates placed in parallel and a grid plate showed most stability [38]. When the forces over the system are above the peak resistance, and the osteotomy is not stable yet, a subtle malocclusion will happen, usually followed by loss of the facial profile obtained. In Fig. 4.10, the patient presents with an acute malocclusion and deviation of the midline, probably because of stress overload under the fixation system.

Plate fracture after orthognathic surgery is rare and may be caused by changing forces, also with a rotational component. These forces may be low but still can cause fatigue fracture if occurring with high frequency. Figure 4.11 shows a female patient 1-month postoperative computed tomography (CT) which BSSO was

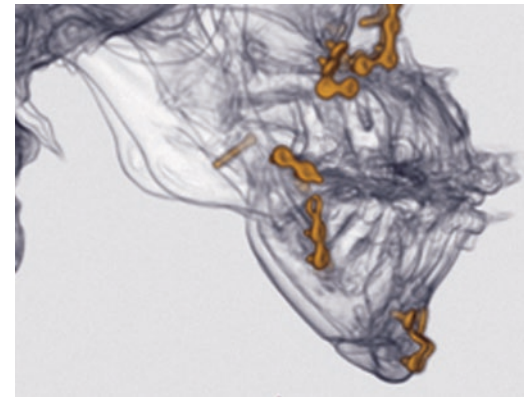


Fig. 4.11 Plate fracture four weeks after BSSO. The proximal segments rotated causing losing of angle contour and a high volume of soft tissue at the cheeks

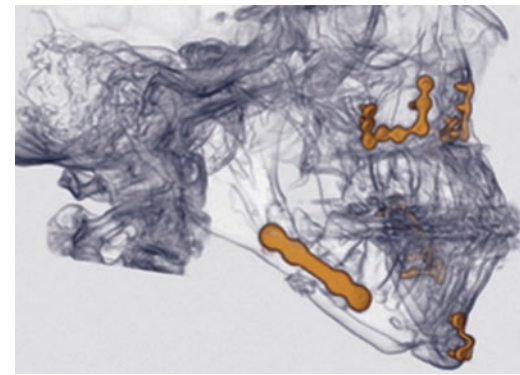


Fig. 4.12 The patient was treated by changing the fractured miniplates for larger 2.0 mm locking plates

fixed using the hybrid technique. Despite adequate fixation, the patient presented fracture of the plates and rotation of the condylar segment, causing loss of the mandibular angle. The patient underwent surgery again to change the fractured plates and received thicker locking 2.0 mm plates. The healing was then uneventfully (Fig. 4.12). Usually, a lot of loose connective tissue is found around lose plates and screws. This diseased tissue should be removed, and bone margins clearly exposed to apply a new fixation method.

Failure of the fixation may happen at the maxilla also. The failure may present as loose screws, or in rare patients, as pseudarthrosis of the

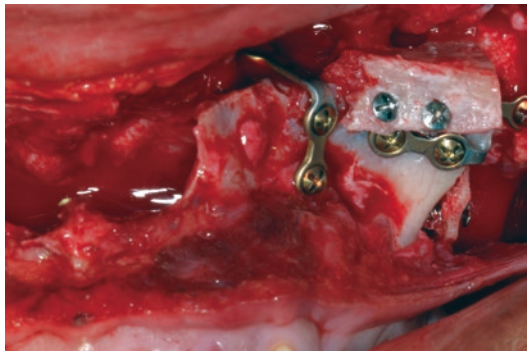


Fig. 4.13 Nonunion of the Le Fort I osteotomy. The maxilla was approached again, and gaps were filled with cranial graft

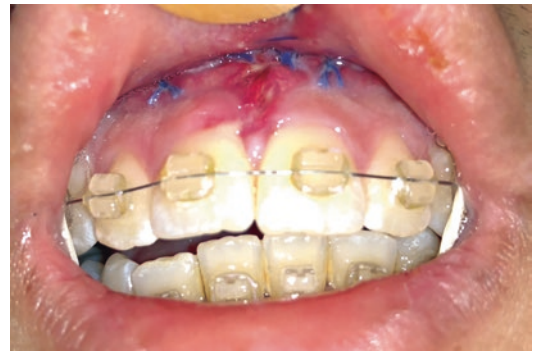


Fig. 4.14 Avascular necrosis of a small portion of the anterior maxilla after midline segmentation

maxilla. At this point, it is important to explain the concept of pseudarthrosis. Pseudarthrosis can be classified as nonunion, or the absence of bone formation between gaps. Figure 4.13 shows a patient that went on to a second maxillary surgery because of a complete instability of the Le Fort I osteotomy 6 months after surgery. The patient had a pseudarthrosis of the entire maxilla, which was treated by removal of the connective tissue and filling of the gaps with iliac bone graft. A meta-analysis showed a good evidence that bone grafting increases the stability of Le Fort I [39]. The maxilla was initially fixated with standard four L plates at canine and zygomatic buttresses. This fixation is a classic method of fixation which has proven to be clinically efficient and mechanically stable [40–42].

4.7 Avascular Necrosis

The basis for Le Fort I maxillary osteotomy was given by a series of studies published by Bell and colleagues [43–47]. Since its development, one great concern of total maxillary osteotomy was the avascular necrosis. It has been shown that irrigation for the maxilla comes from the buccal and from pharyngeal arteries that irrigate it, not from palatine arteries. Therefore, the integrity of palatine arteries is not essential for Le Fort osteotomy. However, compromised blood supply may cause partial or total necrosis of the maxilla, becoming a devastating complication.

The literature of craniomaxillofacial surgery is poor in describing the mechanism of avascular necrosis. Few cases were reported, and possible mechanism included stretching the soft tissue pedicle following Le Fort I osteotomy [48–50]. Blood flow at osteotomized segments can decrease up to 89% after osteotomy, but it tends to be transitory [51]. In the light of more recent knowledge, a flaw exists, because avascular necrosis will be time-dependent due to a long-term ischemia or an ischemia-reperfusion injury [52, 53]. Figure 4.14 shows a case of small avascular necrosis of the anterior maxilla after a Le Fort I osteotomy.

Based on our current knowledge, it is impossible to determine the exact pathophysiology of the disease. The long-term ischemia may have venous thromboembolism (VTE) as a cause. VTE is a common cause of morbidity on which risk varies from 0.4% to 80% in surgical subpopulations, depending on the site of deep veins involved, nature of the underlying operation, and patient comorbidities [54]. In a survey of thromboembolism events, the incidence of VTE in 2589 surgeries at month, esophagus and pharynx was 0.85% [55]. This risk is compatible with the low risk rate commonly seen in our surgical practice. Even more, the VTE is a plausible cause of ischemia of small vessels of the maxilla.

The second cause of avascular necrosis may be an ischemia-reperfusion injury, since transitory ischemia decreases up to 89% of blood flow immediately after surgery [51, 53]. The reperfusion of oxygenated blood to the oxygen-deprived

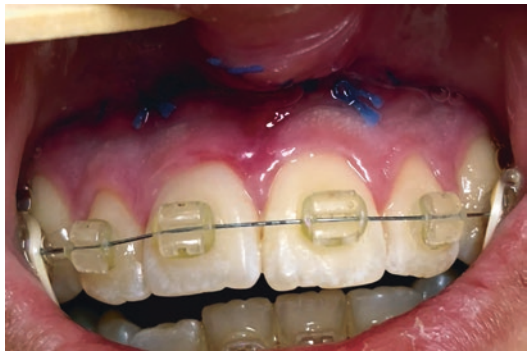


Fig. 4.15 Healing of the avascular necrosis after two weeks of local care (oral hygiene and topical antibiotics) and 03 days of anticoagulation with LMWH. A second option would be ten sessions of hyperbaric oxygen

areas may enhance the tissue damage. The process of ischemia-reperfusion is multifactorial, and there are several mechanisms involved, including the generation of free radicals [53]. The established pro-inflammatory environment caused by ischemia intensifies as free radicals present in the wound causes further tissue destruction [56]. The free radicals consequently initiate an inflammatory response that may affect the extremities of the maxilla.

Contemporary treatment of the avascular necrosis includes the use of warm saline solution during surgery to stimulate tissue reperfusion of the maxilla and early postoperative use of low molecular weight heparin (LMWH) to anticoagulation. Another possibility is the use of hyperbaric oxygen therapy (HBOT). HBOT has many effects, including an increase in VEGF-mRNA levels in endothelial cells and macrophages, and increased VEGF is noted in wound fluid of patients receiving this treatment, [57] which induces endothelial progenitor cells (EPCs) to migrate out of bone marrow, circulate, and settle in the peripheral wound, forming vascular buds. Consequently, HBOT increases wound vascularity [58]. Figure 4.15 shows the clinical result of local care and LMWH for 3 days.

A systematic review showed an incidence of 2,14% of avascular necrosis after a segmental Le Fort I osteotomy, a low rate in comparison with other complications cited in this study [50].

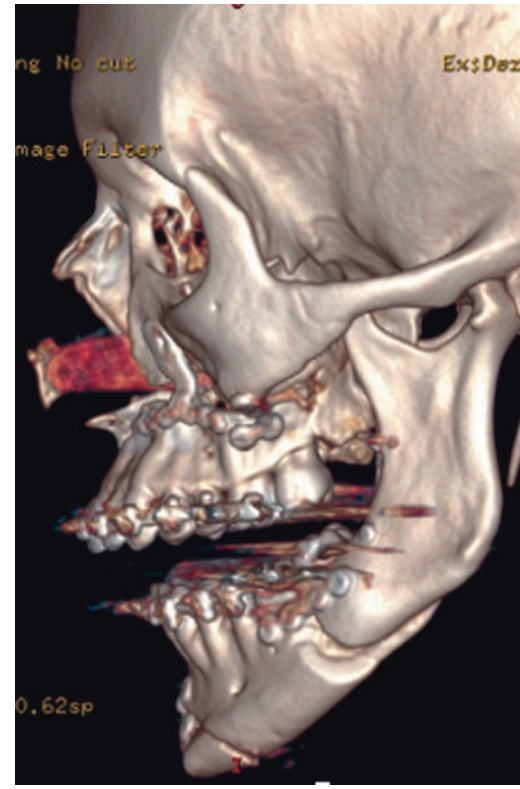


Fig. 4.16 Immediate postoperative CT of a patient submitted to Le Fort I osteotomy, BSSO, and chin advancement

Segmental Le Fort I segmental osteotomy has an increased risk of avascular necrosis, because of the increase in the number of bone cuts and a higher risk of losing blood flow in smaller segments.

The BSSO is also subject to avascular necrosis. Figure 4.16 shows an immediate postoperative CT of an orthognathic surgery to advance the maxilla, mandible, and chon. Two months later, the patient presented an image similar to a bad split of the buccal plate. Since there was no communication within the oral cavity and wounds closed, the best hypothesis was an avascular necrosis of this segment (Fig. 4.17). The patient underwent surgery to remove the necrotized segment (Fig. 4.18). An avascular necrosis of the distal segment of a BSSO is caused by a complete lack of blood flow, because the soft tissue is completely elevated from the bone (Figs. 4.19 and 4.20).

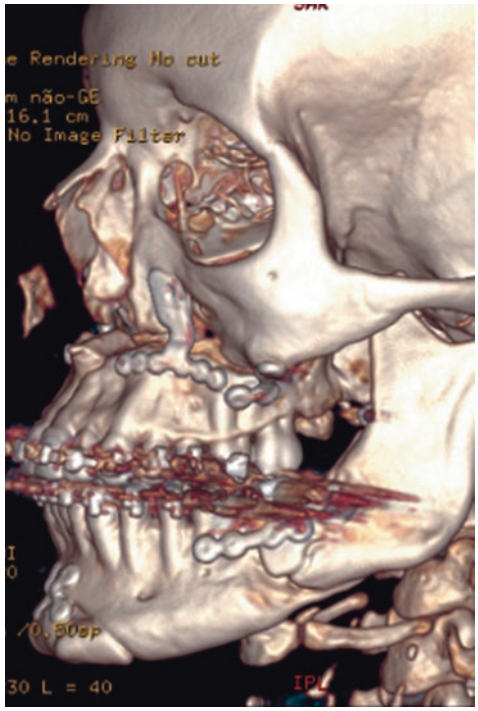


Fig. 4.17 CT 2 month post-op, showing a fracture of the buccal plate. This segment presented an avascular necrosis

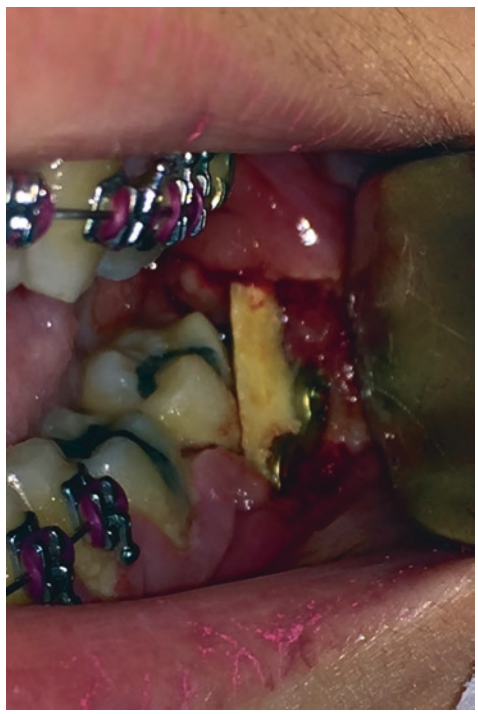


Fig. 4.18 Clinical image of the patient shown in Fig. 4.17, when he underwent surgery to remove the necrotized segment

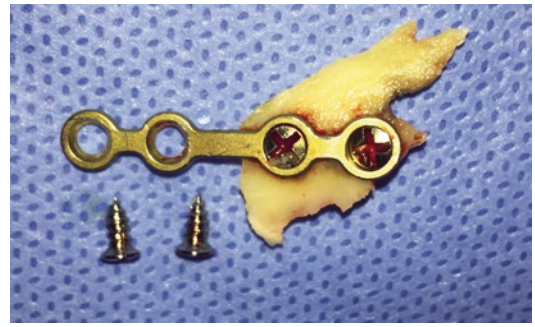


Fig. 4.19 Necrotized buccal plate from the same patient after removal

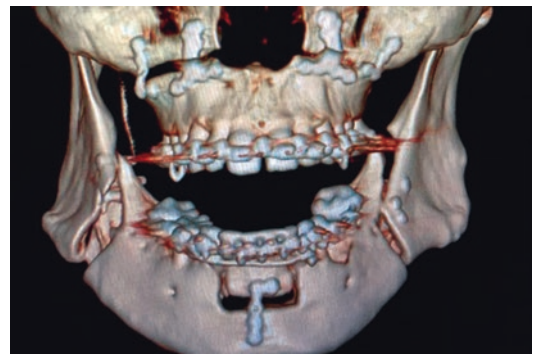


Fig. 4.20 3D reconstruction of a computed tomography showing an anterior dentoalveolar segmental osteotomy

4.8 Hematoma

Hematoma is the collection of blood in an anatomic space. During orthognathic surgery, hematoma formation is a rare complication. A hematoma can be formed from any bleeding artery on which hemorrhage is not controlled. Hematomas from the retromandibular vein, inferior alveolar nerve, facial artery, maxillary artery, and masseteric artery have been reported [59].

An important area that presents hematoma is the sublingual and submental spaces. The literature is full of case reports about life-threatening hematomas of this area [60–65]. This complication usually occurs after oral surgery procedures, like implant placement or any kind of surgery that risks rupturing the lingual aspect of the anterior mandible or entering the muscles in this area. Sublingual hematoma can cause airway obstruction and intubation, or tracheotomy

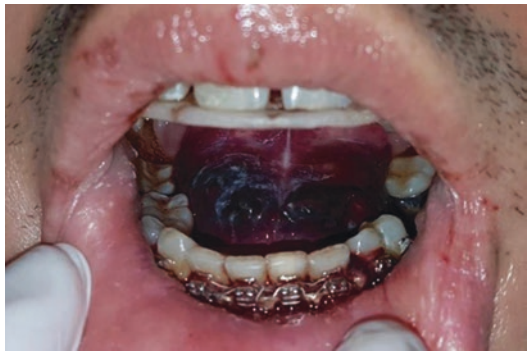


Fig. 4.21 Immediate postoperative sublingual hematoma after a dentoalveolar segmental osteotomy to correct dental leveling and BSSO to advance the mandible

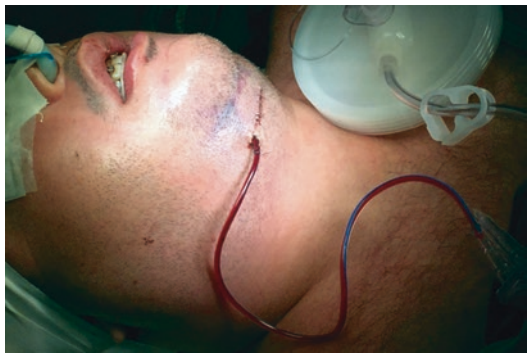


Fig. 4.22 Drainage of the hematoma by means of a submandibular incision

may be necessary to preserve the airway. General anesthesia has two indications in this complication: analgesia during surgery and airway protection.

The blood supply to the sublingual artery comes from the facial artery, and the arterial supply to an incisor tooth is often from the submental artery [66]. Figure 4.21 shows a hematoma at the floor of the mouth, occupying the sublingual space. The patient had a massively enlarged and ecchymotic tongue that was displaced upward with a blood-filled floor of the mouth. The patient needs hospitalization for drainage under general anesthesia (Fig. 4.22). The ecchymosis eventually followed the gravity force and disappeared around the chest of the patient three weeks later (Fig. 4.23).

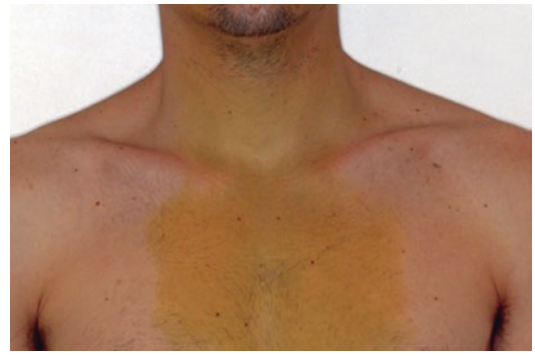


Fig. 4.23 Huge ecchymosis of the neck and breast after orthognathic surgery

4.9 Nasal Septum Deviation

The nasal septum is an important part of the nose. It divides the nasal airway symmetrically and defines the position and height of the tip of the nose [67]. The presence of nasal deformities associated with dentofacial deformities is acknowledged in the literature, and maxillary Le Fort I osteotomies will always present some degree of changes on the nose [68, 69].

After releasing the septum from the maxilla, the septum will assume a new position that will vary according to the previous deviation, nasotraqueal tube position and type of maxillary movement. A nasal deformity can occur when an intrinsic deformity aggravates after a Le Fort I procedure or when a well-balanced nose is distorted [69]. Figure 4.24 shows these side effects as an immediate septum deviation after orthognathic surgery.

Maxillary impactions have a higher risk of septum deviation if a septoplasty is not performed on the same surgery. Maxillary impaction will result in raising the nasal tip, septum deviation, and widening of the alar base, associated with a poor nasal function.

Performing orthognathic surgery with rhinoplasty has many advantages, including simultaneous manipulation of the soft tissue envelope around mouth and nose on the same procedure, decreasing the number of surgeries, better transoperative hemostasis, and a direct approach to inner structures of the nose, like the turbinates [69].

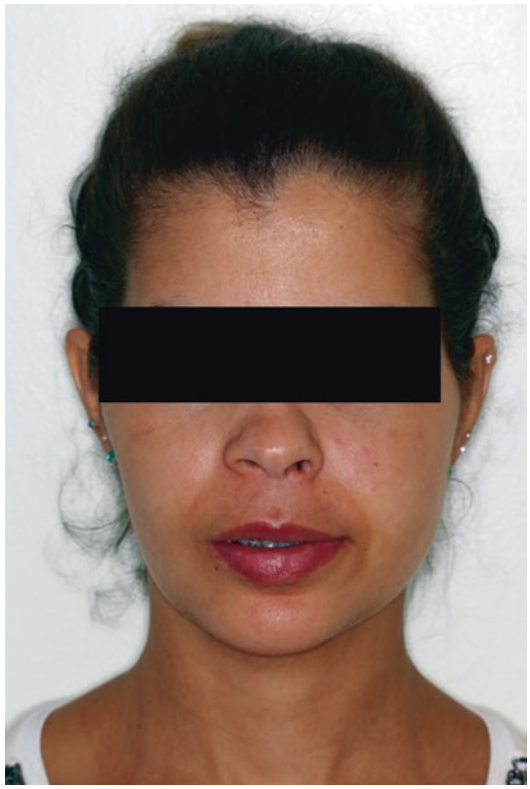


Fig. 4.24 Immediate postoperative result of maxillary and mandible advancement, showing a nasal septum deviation. Note the shift of the nasal tip to the left side

Performing orthognathic surgery with rhinoplasty will also improve patients satisfaction [68].

Septum deviation is an indication for surgery. Figure 4.25 shows a transoperative view of the deviated nasal septum that was corrected and remained stable after 1 year (Fig. 4.26).

4.10 Nerve Paresthesia

As cited in the introduction of this chapter, nerve paresthesia is not necessarily a complication, but a consequence of stretching the nerve after a BSSO. However, the presence of paresthesia is considered alone a complication in most of the studies published. In general, performing BSSO will damage the inferior alveolar nerve (IAN), and when BSSO and genioplasty are performed together, more damage is expected to the mental nerve than BSSO performed alone [70].

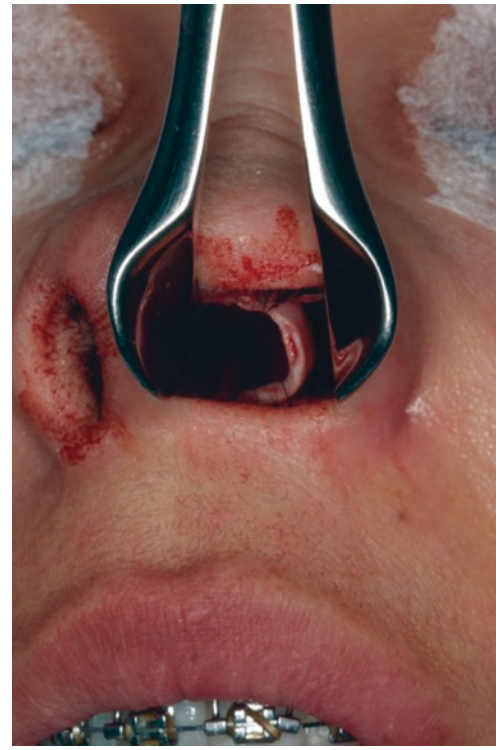


Fig. 4.25 Correction of nasal septum deviation

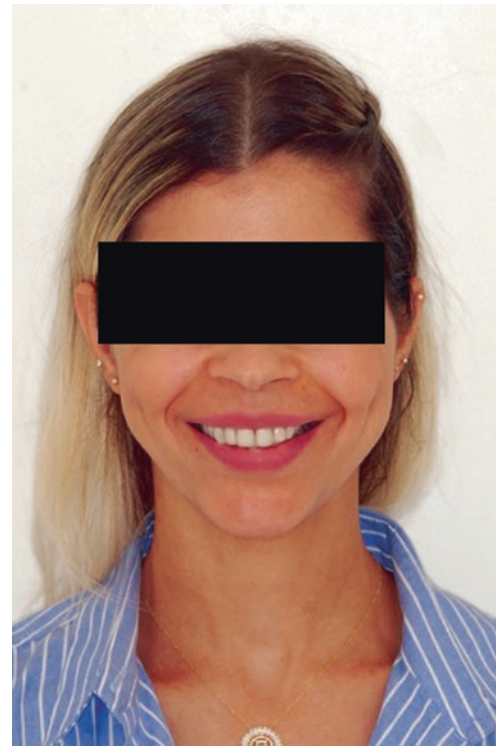


Fig. 4.26 One-year postoperative result after alignment of the nasal septum

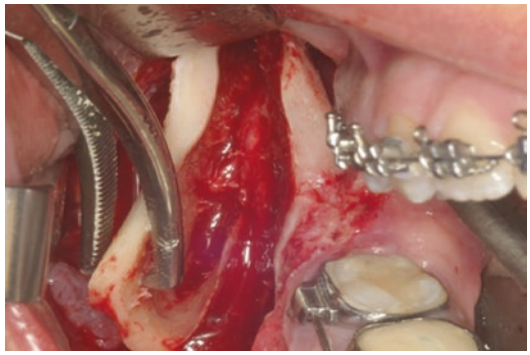


Fig. 4.27 BSSO with exposure of the inferior alveolar nerve. The exposure of the neurovascular bundle may cause intraoperative bleeding. Paresthesia is a result of surgical manipulation, not necessarily a complication. Complication will occur as definitive paresthesia when the nerve is sectioned

Damage of the inferior alveolar nerve is caused by intraoperative trauma during BSSO and genioplasty, causing alterations in lower lip sensibility. This altered sensation has a significant effect on the majority of patients' everyday lives [71]. Its resolution occurs up to 2 years after orthognathic surgery [72].

Exposing the IAN during splitting of the mandible (Fig. 4.27) and surgical manipulation results in edema and the release of inflammatory mediators that temporarily irritate the nerves [73]. Clinically, most of the patients will develop transient paresthesia, hypoesthesia, or dysesthesia. Few treatment methods have been proposed to treat IAN injuries, including corticosteroids, low-level laser therapy, ozone, photobiomodulation, and vitamin B complex [73–76]. Keep in mind that the less is the manipulation during surgery, the preserved is the perineurium and axons, and the faster is the recovery. A complete transection of the nerve can be treated by nerve grafts, which are difficult to do and have variable success rates.

Nonsurgical therapeutics methods can be used alone or in combination. Corticoids will prevent inflammation around the nerve, low-level laser therapy may modulate action potential amplitude [73, 77], and ozone is an extremely strong oxidant that oxidizes all surfaces to the highest oxidation stage, increases and stimulates oxygen metabolism in the circulation, disrupts tumor metabolism, and kills pathogens [75, 78]. Photobiomodulation stimulates the proliferation and differentiation of fibroblasts, osteoblasts, and chondroblasts [75]. Vitamin B complex may stimulate nerve regeneration in the acute phase of healing [76]. Although of low morbidity to the patients, time is necessary to accomplish nerve healing, and these therapies will work as complements, not as a definitive treatment.

4.11 Nerve Paralysis

As the cranial nerves run through and in close proximity to the facial skeleton, they are at risk of injury during several stages of oral and maxillofacial operations [79]. The facial nerve, including the main trunk or smaller nerves, lays over the periosteum near the border of the mandible, especially in the mouth-open position while performing BSSO. The incidence of injuries on the facial nerve is less than 1% in BSSO [80]. The main causes of facial palsy are compression or traction of the nerve after incision and periosteal rupture, getting into the mimic muscles during tissue retraction [81].

Recovery depends on the extent of the injury to the facial nerve. When nerve structures are preserved, the patient will develop neuropraxy that clinically appears as a temporary facial palsy (Fig. 4.28). Regeneration will be complete and facial palsy resolves completely (Fig. 4.29). In more severe injuries of the facial nerve, where axons were disrupted, the recovery is slower and usually incomplete [81, 82]. When complete nerve section occurs, recovery of motion of the facial mimics muscles is not expected, and the only attempt is surgical repair, which presents variable results.

Pharmacological treatment of motor nerves is similar to those of sensitive nerves, which is present in the anterior section of this chapter. It works as a complement, not as an effective treatment. A long follow-up is necessary for all patients with facial palsy.

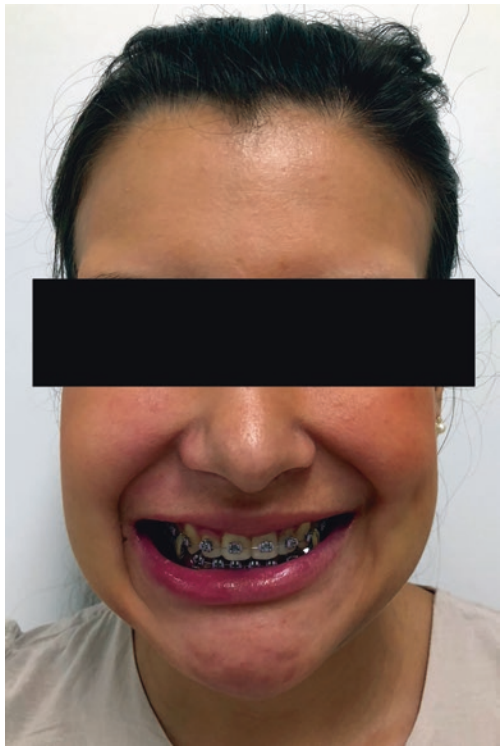


Fig. 4.28 Temporary facial nerve injury after BSSO

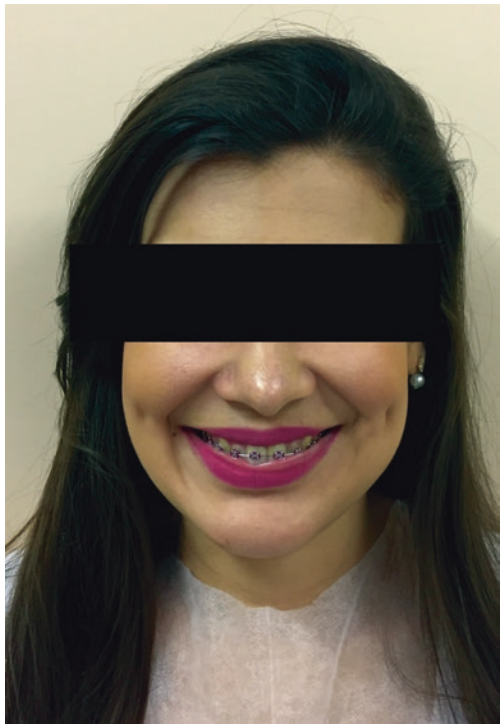


Fig. 4.29 Same patient as in Fig. 4.28, showing total resolution of the facial nerve weakness after 4 months

4.12 Rare Complications

Rare complications in orthognathic surgery include vascular and ophthalmic complications. The literature has less information about these rare complications. Vascular complications include severe bleeding from the maxillary artery or its branches and can be of difficult management, because of the intensity of bleeding or the possibility of recurrence during early recovery [83]. Aneurisms may also be present, and the treatment for both hemorrhage and aneurisms from large vessels is embolization [84].

Another rare situation is an ophthalmic complication after orthognathic surgery [85]. These complications may be caused by direct injury of the optical nerve during pterygoid plate fracture or the formation of a retrobulbar hematoma that compresses the orbital contents. Both complications occur after orthognathic maxillary surgery, after a bad split involving the orbital component of the palatine bone. Tearing of the optical nerve will need an urgent surgery to decompress the nerve, and the hematoma will need drainage and corticoids to control postoperative edema.

4.13 Complications in Le Fort III Osteotomies

Le Fort III osteotomies are indicated to correct the middle third of the face in syndromic and non-syndromic patients [86, 87]. Currently, to treat severe discrepancies and achieve an adequate intermaxillary relationship and a stable occlusion, LF III osteotomy in combination with LF I osteotomy is indicated for the treatment of syndromic and non-syndromic patients presenting with severe class III malocclusion and 10 mm or more of reverse overjet [86–88].

The complications of classic Le Fort III osteotomies include strabismus, anosmia, zygoma fracture, graft exposure, respiratory infections, liquorrhea, and amaurosis. The subcranial Le Fort III osteotomy is performed away from the central nervous system, decreasing the possibility of major complications seen on the classic one. The design of this subcranial osteotomy allows correction of the midface, nose, and orbits

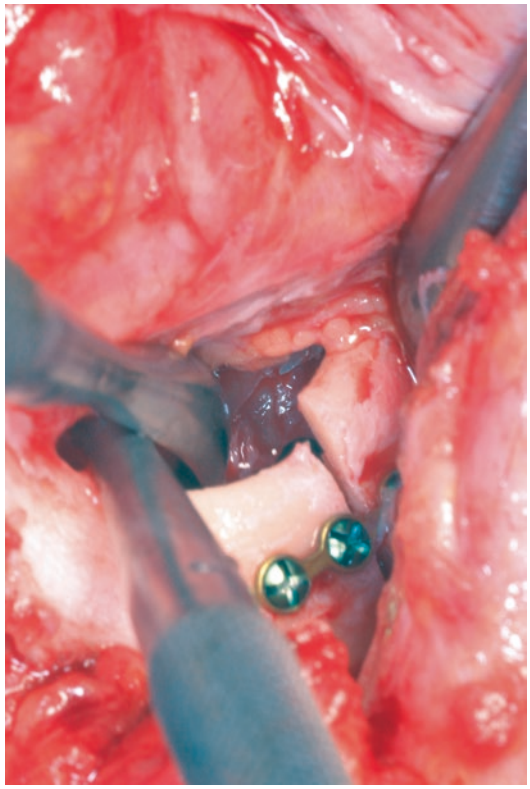


Fig. 4.30 Intraoperative right orbital floor fracture during a subcranial Le Fort III osteotomy

in adult patients by means of two surgical approaches: a coronal incision and an intraoral incision [87, 89].

The authors would like to share their experience with the subcranial Le Fort III osteotomies, discussing four complications that might happen in this surgery: orbital floor fracture, asymmetrical advancement of the middle third of the face, vertical asymmetry of the middle face, and the scar from the coronal flap.

The orbital osteotomy during subcranial Le Fort III osteotomy is performed through the orbital floor, up to 5 mm behind the orbital border. This bone cut is preferably done with a piezo instrument to avoid injuries to the contents of the orbit. Nonetheless, as the orbital floor is very thin, it will fracture during mobilization of the middle third of the face (Fig. 4.30). Treatment should be done after middle third reposition and fixation to understand the final size of the defect. Usually, the orbital floor osteotomy and the insertion of the titanium mesh are made through the coronal incision, avoiding a new incision at the lower eyelid (Fig. 4.31).

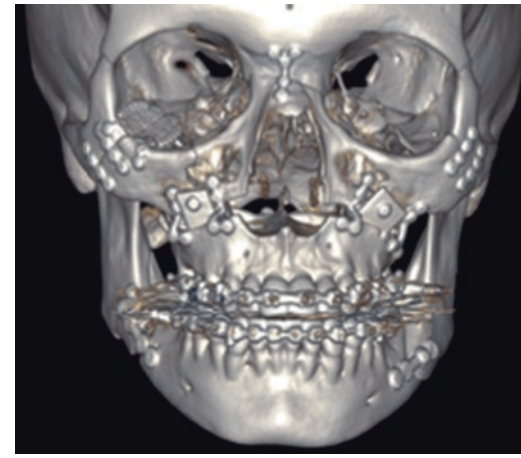


Fig. 4.31 The fracture was treated with a titanium mesh to avoid orbital herniation to the maxillary sinus

The second complication is the difficult to symmetric advance the middle third of the face. Downfracturing and mobilizing it is difficult because all maxillary-pterygoid junctions must be separated. If the surgeon fails to correctly separate this junction after completing all osteotomies, he will fail to make the planned advancement. An asymmetrical advancement will have a direct impact on the anterior–posterior projections of the zygoma bodies, causing an unaesthetic result. The surgeon must check the amount of advancement at the zygomatic osteotomy before fixation is done (Fig. 4.32).

The third complication may occur when the osteotomies are not parallel on both sides. Performing a horizontal osteotomy at the body of the zygoma and nasal bones during subcranial Le Fort III osteotomy, the surgeon will achieve a large surface on which the midface will slide forward and in an upper direction under the patient's skull. This design will improve the stability by increasing the contact and avoiding gaps between segments, thus avoiding the need for grafts. In addition, regarding the design of the zygoma body osteotomies, they should be parallel to each other. By failing to make them parallel after advancement, a differential contact may be created between sides,

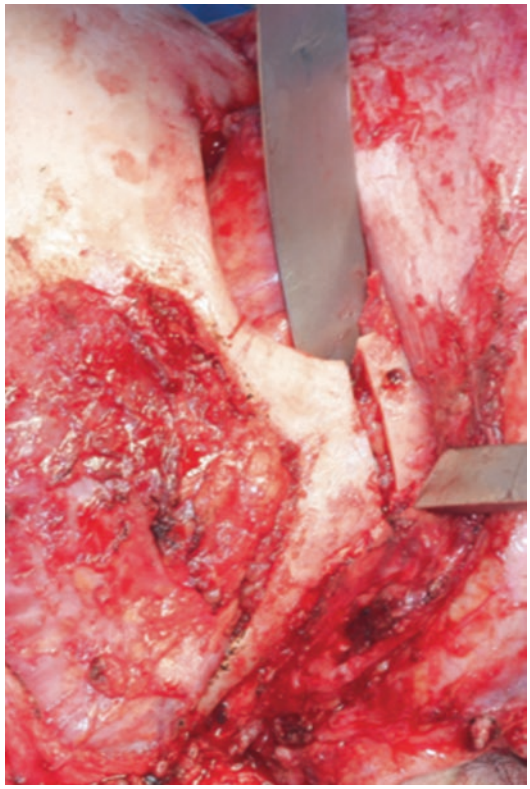


Fig. 4.32 Subcranial Le Fort III osteotomy after down-fracture. Note the advancement of the zygoma body

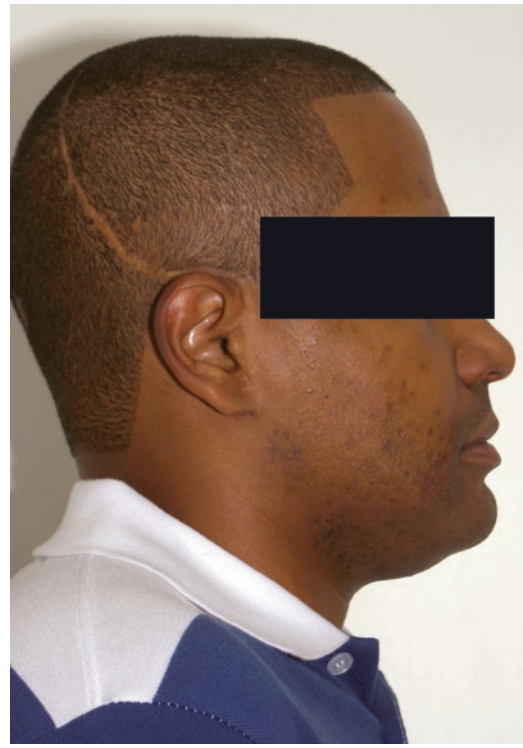


Fig. 4.33 Scar crossing the hair in a patient submitted to Le Fort III osteotomy by means of a coronal incision. In a patient with long hair, the same scar would be invisible

creating gaps and different heights of the zygoma, thus causing a vertical asymmetry and the necessity of grafting one side of the osteotomy to fill the created gap [87].

The fourth complication is the scar of the coronal incision. Figure 4.33 shows a patient with a visible scar 4 years after a subcranial Le Fort III osteotomy to advance the face. The best way to avoid this scar is to perform a zigzag incision or make an incision at the division of the hair with the skin [90]. This is an esthetical complaint with no adequate treatment until now.

4.14 Conclusions

Orthognathic surgery is safe, and most of the complications are not life-threatening. Prevention and knowledge about the complication are the best way to avoid definitive consequences for the patient.

References

1. Patel PK, Morris DE, Gassman A. Complications of orthognathic surgery. J Craniofac Surg. 2007;18(4):975–85. https://doi.org/10.1097/scs.0b013e318068442c.
2. Kim S-G, Park S-S. Incidence of complications and problems related to orthognathic surgery. J Oral Maxillofac Surg. 2007;65(12):2438–44. https://doi.org/10.1016/j.joms.2007.05.030.
3. Olate S, Sigua E, Asprino L, de Moraes M. Complications in orthognathic surgery. J Craniofac Surg. 2018;29(2):e158–61. https://doi.org/10.1097/SCS.0000000000004238.
4. Zenk K, Finze S, Kluess D, Bader R, Malzahn J, Mittelmeier W. Einfluss der Erfahrung des Operateurs in der Hüftendoprothetik. Orthopade. 2014;43(6):522–8. https://doi.org/10.1007/s00132-014-2292-4.
5. Cahill PJ, Pahys JM, Asghar J, Yaszay B, Marks MC, Bastrom TP, et al. The effect of surgeon experience on outcomes of surgery for adolescent idiopathic scoliosis. J Bone Jt Surg. 2014;96(16):1333–9. https://doi.org/10.2106/JBJS.M.01265.
6. Haynes AB, Weiser TG, Berry WR, Lipsitz SR, Breizat AHS, Dellinger EP, et al. A surgical safety

checklist to reduce morbidity and mortality in a global population. N Engl J Med. 2009;360(5):491–9. https://doi.org/10.1056/NEJMsa0810119.
7. Conley DM, Singer SJ, Edmondson L, Berry WR, Gawande AA. Effective surgical safety checklist implementation. J Am Coll Surg. 2011;212(5):873–9. https://doi.org/10.1016/j.jamcollsurg.2011.01.052.
8. Trauner ROH. Zur Operationstechnik bei der Progenia und anderen Unterkieferanomalien. Dtsch Zahn Mund Kieferhlkd. 1955;23:11–25.
9. Trauner R, Obwegeser H. The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. Oral Surg Oral Med Oral Pathol. 1957;10(7):677–89. https://doi.org/10.1016/S0030-4220(57)80063-2.
10. Trauner R, Obwegeser H. The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. Part II. Operating methods for microgenia and distoclusion. Oral Surg Oral Med Oral Pathol. 1957;10(8):787–92. https://doi.org/10.1016/0030-4220(57)90105-6.
11. Dal Pont G. Retro-molar osteotomy for correction of prognathism. Minerva Chir. 1959;14:1138–41.
12. Hunsuck EE. A modified intraoral sagittal splitting technic for correction of mandibular prognathism. J Oral Surg. 1968;26(4):250–3. https://doi.org/10.1002/acn3.201.
13. Epker BN. Modifications in the sagittal osteotomy of the mandible. J Oral Surg. 1977;35:157–9.
14. Wolford LM, Davis WM. The mandibular inferior border split: a modification in the sagittal split osteotomy. J Oral Maxillofac Surg. 1990;48(1):92–4. https://doi.org/10.1016/0278-2391(90)90190-D.
15. Gil JN, Marin C, Claus JDP, Lima SM. Modified osteotome for inferior border sagittal split osteotomy. J Oral Maxillofac Surg. 2007;65(9):1840–2. https://doi.org/10.1016/j.joms.2005.12.045.
16. Böckmann R, Schön P, Neuking K, Meyns J, Kessler P, Eggeler G. In vitro comparison of the sagittal split osteotomy with and without inferior border osteotomy. J Oral Maxillofac Surg. 2015;73(2):316–23. https://doi.org/10.1016/j.joms.2014.08.005.
17. Steenen SA, Becking AG. Bad splits in bilateral sagittal split osteotomy: systematic review of fracture patterns. Int J Oral Maxillofac Surg. 2016;45(7):887–97. https://doi.org/10.1016/j.ijom.2016.02.001.
18. O'Ryan F, Poor DB. Completing sagittal split osteotomy of the mandible after fracture of the buccal plate. J Oral Maxillofac Surg. 2004;62(9):1175–6. https://doi.org/10.1016/j.joms.2003.12.032.
19. Reyneke J, Ferretti C. Intraoperative diagnosis of condylar sag after bilateral sagittal split ramus osteotomy. Br J Oral Maxillofac Surg. 2002;40(4):285–92. https://doi.org/10.1016/S0266-4356(02)00147-X.
20. Nova I, Kallus S, Berger M, Ristow O, Eisenmann U, Freudlsperger C, et al. Computer assisted positioning of the proximal segment after sagittal split osteotomy of the mandible: preclinical investigation of a novel electromagnetic navigation system. J Cranio Maxillofac Surg. 2017;45(5):748–54. https://doi.org/10.1016/j.jcms.2017.01.036.
21. Nunes de Lima V, Faverani LP, Santiago JF, Palmieri C, Magro FO, Pellizzer EP. Evaluation of condylar resorption rates after orthognathic surgery in class II and III dentofacial deformities: a systematic review. J Cranio Maxillofac Surg. 2018;46(4):668–73. https://doi.org/10.1016/j.jcms.2018.02.002.
22. Catherine Z, Breton P, Bouletreau P. Condylar resorption after orthognathic surgery: a systematic review. Rev Stomatol Chir Maxillofac Chir Orale. 2016;117(1):3–10. https://doi.org/10.1016/j.revsto.2015.11.002.
23. de Moraes PH, Rizzati-Barbosa CM, Olate S, Moreira RWF, de Moraes M. Condylar resorption after orthognathic surgery: a systematic review. Int J Morphol. 2012;30(3):1023–8. https://doi.org/10.4067/S0717-95022012000300042.
24. Scheerlinck JPO, Stoelinga PJW, Blijdorp PA, Brouns JJA, Nijs MLL. Sagittal split advancement osteotomies stabilized with miniplates. A 2–5-year follow-up. Int J Oral Maxillofac Surg. 1994;23(3):127–31. https://doi.org/10.1016/S0901-5027(05)80285-1.
25. Arnett GW, Milam SBGL. Progressive mandibular retrusion—idiopathic condylar resorption. Part I. Am J Orthod Dentofacial Orthop. 1996;110:8–15.
26. Bouwman JPB, Kerstens HCJ, Tuinzing DB. Condylar resorption in orthognathic surgery. Oral Surg Oral Med Oral Pathol. 1994;78(2):138–41. https://doi.org/10.1016/0030-4220(94)90135-X.
27. Hwang SJ, Haers PE, Sailer HF. The role of a posteriorly inclined condylar neck in condylar resorption after orthognathic surgery. J Cranio Maxillofac Surg. 2000;28(2):85–90. https://doi.org/10.1054/jcms.2000.0129.
28. Campbell JH. Effects of sex hormones on protein and collagen content of the temporomandibular joint disc of the rat. J Oral Maxillofac Surg. 1996;54(6):727–8. https://doi.org/10.1016/S0278-2391(96)90691-6.
29. Wolford LM, Reiche-Fischel O, Mehra P. Changes in temporomandibular joint dysfunction after orthognathic surgery. J Oral Maxillofac Surg. 2003;61(6):655–60. https://doi.org/10.1053/joms.2003.50131.
30. Chigurupati R, Mehra P. Surgical management of idiopathic condylar resorption. Oral Maxillofac Surg Clin North Am. 2018;30(3):355–67. https://doi.org/10.1016/j.coms.2018.05.004.
31. Singh M, Detamore MS. Biomechanical properties of the mandibular condylar cartilage and their relevance to the TMJ disc. J Biomech. 2009;42(4):405–17. https://doi.org/10.1016/j.jbiomech.2008.12.012.
32. Goizueta Adame CC, Muñoz-Guerra MF. The posterior double pass suture in repositioning of the temporomandibular disc during arthroscopic surgery: a report of 16 cases. J Cranio Maxillofac Surg. 2012;40(1):86–91. https://doi.org/10.1016/j.jcms.2011.01.022.
33. Al-Moraissi EA, Wolford LM. Does temporomandibular joint pathology with or without surgical management affect the stability of counterclockwise rotation

of the maxillomandibular complex in orthognathic surgery? A systematic review and meta-analysis. J Oral Maxillofac Surg. 2017;75(4):805–21. https://doi.org/10.1016/j.joms.2016.10.034.
34. Gunson MJ, Arnett GW, Milam SB. Pathophysiology and pharmacologic control of osseous mandibular condylar resorption. J Oral Maxillofac Surg. 2012;70(8):1918–34. https://doi.org/10.1016/j.joms.2011.07.018.
35. Hammer B, Schier P, Prein J. Osteosynthesis of condylar neck fractures: a review of 30 patients. Br J Oral Maxillofac Surg. 1997;35(4):288–91. https://doi.org/10.1016/S0266-4356(97)90050-4.
36. Sato FRL, Asprino L, Fernandes Moreira RW, de Moraes M. Comparison of postoperative stability of three rigid internal fixation techniques after sagittal split ramus osteotomy for mandibular advancement. J Cranio Maxillofac Surg. 2014;42(5):e224–9. https://doi.org/10.1016/j.jcms.2013.08.012.
37. Sato FRL, Asprino L, Consani S, de Moraes M. Comparative biomechanical and photoelastic evaluation of different fixation techniques of sagittal split ramus osteotomy in mandibular advancement. J Oral Maxillofac Surg. 2010;68(1):160–6. https://doi.org/10.1016/j.joms.2009.09.004.
38. Kuik K, De Ruiter MHT, De Lange J, Hoekema A. Fixation methods in sagittal split ramus osteotomy: a systematic review on in vitro biomechanical assessments. Int J Oral Maxillofac Surg. 2019;48(1):56–70. https://doi.org/10.1016/j.ijom.2018.06.013.
39. Alyahya A, Swennen GRJ. Bone grafting in orthognathic surgery: a systematic review. Int J Oral Maxillofac Surg. 2019;48(3):322–31. https://doi.org/10.1016/j.ijom.2018.08.014.
40. Erkmen E, Ataç MS, Yücel E, Kurt A. Comparison of biomechanical behaviour of maxilla following Le Fort I osteotomy with 2- versus 4-plate fixation using 3D-FEA. Int J Oral Maxillofac Surg. 2009;38(2):173–9. https://doi.org/10.1016/j.ijom.2008.10.006.
41. Ataç MS, Erkmen E, Yücel E, Kurt A. Comparison of biomechanical behaviour of maxilla following Le Fort I osteotomy with 2- versus 4-plate fixation using 3D-FEA. Part 1: advancement surgery. Int J Oral Maxillofac Surg. 2008;37(12):1117–24. https://doi.org/10.1016/j.ijom.2008.10.004.
42. Ataç MS, Erkmen E, Yücel E, Kurt A. Comparison of biomechanical behaviour of maxilla following Le Fort I osteotomy with 2- versus 4-plate fixation using 3D-FEA: Part 2: impaction surgery. Int J Oral Maxillofac Surg. 2009;38(1):58–63. https://doi.org/10.1016/j.ijom.2008.10.005.
43. Bell WH. Revascularization and bone healing after anterior maxillary osteotomy: a study using adult rhesus monkeys. J Oral Surg. 1969;27(4):249–55.
44. Bell W, Levy B. Revascularization and bone healing after posterior maxillary osteotomy. J Oral Surg (Chic). 1971;29(5):313–20.
45. Bell WLB. Revascularization and bone healing after maxillary corticotomies. J Oral Surg. 1972;30(9):640–8.
46. Bell WH, Fonseca RJ, Kenneky JW, Levy BM. Bone healing and revascularization after total maxillary osteotomy. J Oral Surg. 1975;33:253–60.
47. Quejada JG, Kawamura H, Finn RA, Bell WH. Wound healing associated with segmental total maxillary osteotomy. J Oral Maxillofac Surg. 1986;44(5):366–77. https://doi.org/10.1016/S0278-2391(86)80032-5.
48. Lanigan DT, Hey JH, West RA. Aseptic necrosis following maxillary osteotomies: report of 36 cases. J Oral Maxillofac Surg. 1990;48(2):142–56. https://doi.org/10.1016/S0278-2391(10)80202-2.
49. Lanigan DT, West RA. Aseptic necrosis of the mandible: report of two cases. J Oral Maxillofac Surg. 1990;48(3):296–300. https://doi.org/10.1016/0278-2391(90)90397-K.
50. Haas Junior OL, Guijarro-Martínez R, de Sousa Gil AP, da Silva Meirelles L, de Oliveira RB, Hernández-Alfaro F. Stability and surgical complications in segmental Le Fort I osteotomy: a systematic review. Int J Oral Maxillofac Surg. 2017;46(9):1071–87. https://doi.org/10.1016/j.ijom.2017.05.011.
51. Nelson RL, Path MG, Ogle RG, Waite DEMM. Quantitation of blood flow after a Le Fort I osteotomy. J Oral Surg. 1977;35(1):10–6.
52. Gunduz A, Turkmen S, Turedi S, Mentese A, Yulug E, Ulusoy H, et al. Time-dependent variations in ischemia-modified albumin levels in mesenteric ischemia. Acad Emerg Med. 2009;16(6):539–43. https://doi.org/10.1111/j.1553-2712.2009.00414.x.
53. Halladin NL. Oxidative and inflammatory biomarkers of ischemia and reperfusion injuries. Dan Med J. 2015;62:B5054.
54. Geerts WH, Pineo GF, Heit JA, Bergqvist D, Lassen MR, Colwell CW, et al. Prevention of venous thromboembolism. Chest. 2004;126(3):338S–400S. https://doi.org/10.1378/chest.126.3_suppl.338S.
55. Rogers SO, Kilaru RK, Hosokawa P, Henderson WG, Zinner MJ, Khuri SF. Multivariable predictors of postoperative venous thromboembolic events after general and vascular surgery: results from the patient safety in surgery study. J Am Coll Surg. 2007;204(6):1211–21. https://doi.org/10.1016/j.jamcollsurg.2007.02.072.
56. Eisenbud DE. Oxygen in wound healing. Clin Plast Surg. 2012;39(3):293–310. https://doi.org/10.1016/j.cps.2012.05.001.
57. Schreml S, Szeimies RM, Prantl L, Karrer S, Landthaler M, Babilas P. Oxygen in acute and chronic wound healing. Br J Dermatol. 2010;163(2):257–68. https://doi.org/10.1111/j.1365-2133.2010.09804.x.
58. Goldstein LJ, Gallagher KA, Bauer SM, Bauer RJ, Baireddy V, Liu Z-J, et al. Endothelial progenitor cell release into circulation is triggered by hyperoxia-induced increases in bone marrow nitric oxide. Stem Cells. 2006;24(10):2309–18. https://doi.org/10.1634/stemcells.2006-0010.
59. Lanigan DT, Hey J, West RA. Hemorrhage following mandibular osteotomies: a report of 21 cases. J Oral Maxillofac Surg. 1991;49(7):713–24. https://doi.org/10.1016/S0278-2391(10)80235-6.

60. Frohna WJ, Lowery RC, Pita F. Lingual and sublingual hematoma causing upper airway obstruction. J Emerg Med. 2012;43(6):1075–6. https://doi.org/10.1016/j.jemermed.2011.05.075.
61. Pathak R, Supplee S, Aryal MR, Karmacharya P. Warfarin induced sublingual hematoma: a Ludwig angina mimic. Am J Otolaryngol. 2015;36(1):84–6. https://doi.org/10.1016/j.amjoto.2014.08.008.
62. de Moraes HHA, de Santana Santos T, Camargo IB, de Holanda Vasconcellos RJ. Sublingual hematoma after usual warfarin dose. J Craniofac Surg. 2013;24(5):1858–9. https://doi.org/10.1097/SCS.0b013e3182997ca0.
63. Puri A, Nusrath MA, Harinathan D, Lyall J. Massive sublingual hematoma secondary to anticoagulant therapy complicated by a traumatic denture: a case report. J Med Case Reports. 2012;6(1):105. https://doi.org/10.1186/1752-1947-6-105.
64. Kalpidis CDR, Setayesh RM. Hemorrhaging associated with endosseous implant placement in the anterior mandible: a review of the literature. J Periodontol. 2004;75(5):631–45. https://doi.org/10.1902/jop.2004.75.5.631.
65. Zhao Y-F, Jia J, Jia Y. Complications associated with surgical management of ranulas. J Oral Maxillofac Surg. 2005;63(1):51–4. https://doi.org/10.1016/j.joms.2004.02.018.
66. Nakajima K, Tagaya A, Otonari-Yamamoto M, Seki K, Araki K, Sano T, et al. Composition of the blood supply in the sublingual and submandibular spaces and its relationship to the lateral lingual foramen of the mandible. Oral Surg Oral Med Oral Pathol Oral Radiol. 2014;117(1):e32–8. https://doi.org/10.1016/j.oooo.2012.03.032.
67. Moroi A, Yoshizawa K, Tsutsui T, Saida Y, Hotta A, Fukaya K, et al. Assessment of nasal septum after Le Fort I osteotomy with computer tomography. J Cranio Maxillofac Surg. 2016;44(9):1187–93. https://doi.org/10.1016/j.jcms.2016.05.024.
68. Seah TE, Bellis H, Ilankovan V. Orthognathic patients with nasal deformities: case for simultaneous orthognathic surgery and rhinoplasty. Br J Oral Maxillofac Surg. 2012;50(1):55–9. https://doi.org/10.1016/j.bjoms.2010.12.009.
69. Sun AH, Steinbacher DM. Orthognathic surgery and rhinoplasty. Plast Reconstr Surg. 2018;141(2):322–9. https://doi.org/10.1097/PRS.0000000000004020.
70. Gianni A. Neurosensory alterations of the inferior alveolar and mental nerve after genioplasty alone or associated with sagittal osteotomy of the mandibular ramus. J Cranio Maxillofac Surg. 2002;30(5):295–303. https://doi.org/10.1016/S1010-5182(02)90311-2.
71. Lee EGL, Ryan FS, Shute J, Cunningham SJ. The impact of altered sensation affecting the lower lip after orthognathic treatment. J Oral Maxillofac Surg. 2011;69(11):e431–45. https://doi.org/10.1016/j.joms.2011.07.013.
72. Borstlap W, Stoelinga PJ, Hoppenreijs TJ, van't Hof M. Stabilisation of sagittal split advancement osteotomies with miniplates: a prospective, multicentre study with two-year follow-up. Int J Oral Maxillofac Surg. 2004;33(5):433–41. https://doi.org/10.1016/j.ijom.2004.02.003.
73. de Lima VN, Lemos CAA, Faverani LP, Santiago Júnior JF, Pellizzer EP. Effectiveness of corticoid administration in orthognathic surgery for edema and neurosensorial disturbance: a systematic literature review. J Oral Maxillofac Surg. 2017;75(7):1528.e1–8. https://doi.org/10.1016/j.joms.2017.03.039.
74. Miloro M, Criddle T-R. Does low-level laser therapy affect recovery of lingual and inferior alveolar nerve injuries? J Oral Maxillofac Surg. 2018;76(12):2669–75. https://doi.org/10.1016/j.joms.2018.06.001.
75. Yucesoy T, Kutuk N, Canpolat DG, Alkan A. Comparison of ozone and photo-biomodulation therapies on mental nerve injury in rats. J Oral Maxillofac Surg. 2017;75(11):2323–32. https://doi.org/10.1016/j.joms.2017.04.016.
76. Altun I, Kurutaş E. Vitamin B complex and vitamin B 12 levels after peripheral nerve injury. Neural Regen Res. 2016;11(5):842. https://doi.org/10.4103/1673-5374.177150.
77. Li H, Xie W, Strong JA, Zhang J-M. Systemic antiinflammatory corticosteroid reduces mechanical pain behavior, sympathetic sprouting, and elevation of proinflammatory cytokines in a rat model of neuropathic pain. Anesthesiology. 2007;107(3):469–77. https://doi.org/10.1097/01.anes.0000278907.37774.8d.
78. Azarpazhooh A, Limeback H. The application of ozone in dentistry: a systematic review of literature. J Dent. 2008;36(2):104–16. https://doi.org/10.1016/j.jdent.2007.11.008.
79. Bowe DC, Gruber EA, McLeod NMH. Nerve injury associated with orthognathic surgery. Part 1: UK practice and motor nerve injuries. Br J Oral Maxillofac Surg. 2016;54(4):362–5. https://doi.org/10.1016/j.bjoms.2016.01.026.
80. de Vries K, Devriese PP, Hovinga J, van den Akker HP. Facial palsy after sagittal split osteotomies. J Cranio Maxillofac Surg. 1993;21(2):50–3. https://doi.org/10.1016/S1010-5182(05)80147-7.
81. Choi B-K, Goh RCW, Chen PKT, Chuang DCC, Lo L-J, Chen Y-R. Facial nerve palsy after sagittal split ramus osteotomy of the mandible: mechanism and outcomes. J Oral Maxillofac Surg. 2010;68(7):1615–21. https://doi.org/10.1016/j.joms.2010.01.010.
82. Jones JK, Vansickels J. Facial-nerve injuries associated with orthognathic surgery—a review of incidence and management. J Oral Maxillofac Surg. 1991;49(7):740–4.
83. Lanigan DT, Hey JHWR. Major vascular complications of orthognathic surgery: hemorrhage associated with Le Fort I osteotomies. J Oral Maxillofac Surg. 1991;45:561–73.
84. Lanigan DT, Hey JH, West RA. Major vascular complications of orthognathic surgery: False aneurysms and arteriovenous fistulas following orthognathic

surgery. J Oral Maxillofac Surg. 1991;49(6):571–7. https://doi.org/10.1016/0278-2391(91)90337-L.
85. Lanigan DT, Romanchuk K, Olson CK. Ophthalmic complications associated with orthognathic surgery. J Oral Maxillofac Surg. 1993;51(5):480–94. https://doi.org/10.1016/S0278-2391(10)80502-6.
86. Tiwana PS, Turvey TA. Subcranial procedures in craniofacial surgery: the Le Fort III osteotomy. Oral Maxillofac Surg Clin North Am. 2004;16(4):493–501. https://doi.org/10.1016/j.coms.2004.08.001.
87. Boos Lima FBDJ, Hochuli Vieira E, Juergens P, Lima Junior SM. Is subcranial Le Fort III plus Le Fort I osteotomy stable? J Cranio Maxillofac Surg. 2017;45(12):1989–95. https://doi.org/10.1016/j.jcms.2017.09.004.
88. Nout E, Cesteleyn LLM, van der Wal KGH, van Adrichem LNA, Mathijssen IMJ, Wolvius EB. Advancement of the midface, from conventional Le Fort III osteotomy to Le Fort III distraction: review of the literature. Int J Oral Maxillofac Surg. 2008;37(9):781–9. https://doi.org/10.1016/j.ijom.2008.04.006.
89. Cheung LK, Ow A, Chua HDP. Simultaneous modified oblique Le Fort III and segmentalized Le Fort I osteotomies. J Oral Maxillofac Surg. 2010;68(4):915–23. https://doi.org/10.1016/j.joms.2009.06.009.
90. Munro IR, Chir KMB, Fearon JA. The coronal incision revisited. Plast Reconstr Surg. 1994;93(1):185–7. https://doi.org/10.1097/00006534-199401000-00031.

Part II

Infection

Complications of Odontogenic and Non-odontogenic Infections

5

Radhika Chigurupati and Michael Shemkus

Contents

5.1 Introduction

5.1.1 Odontogenic and Non-odontogenic Infections in Adults

Odontogenic infections: Infections of the head and neck region may originate from odontogenic or non-odontogenic sources. Infections arising from the teeth and supporting tissues of the teeth are known as *odontogenic infections*. Reports in the

R. Chigurupati (✉)
Department of Oral and Maxillofacial Surgery, Boston University, Boston, MA, USA
e-mail: rchiguru@bu.edu

M. Shemkus
Oral and Maxillofacial Surgeon, Quincy, MA, USA

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_5

literature reveal that 50–70% of head and neck infections and the related complications arise from an odontogenic source [1, 2]. Huang et al. reported that 50% of the 185 cases of deep neck infections were odontogenic in origin [3]. Bridgeman et al. [4] found that 53% of their 107 major maxillofacial infection cases were due to an odontogenic source. Byers et al. did a cross-sectional review of their cases in 2010 and reported that 86% of their cervicofacial infections in Scotland were of dental origin [5], and Bross-Soriano et al. [6] reported that 89% of their 121 patients with Ludwig's angina had an odontogenic source of infection. These infections are frequently due to poor oral hygiene, acute or chronic periodontitis, severe dental caries, pericoronitis, and sometimes secondary to dental surgical procedures [7, 8]. The posterior mandibular teeth, particularly third molars followed by mandibular second and first molars, are the most frequently involved teeth in deep neck space infections in adults [9–11]. The maxillary posterior and anterior teeth can be a source of orbital or intracranial infections.

Non-odontogenic infections, on the other hand, arise usually from tonsils, the upper respiratory tract, paranasal sinuses, middle ear, retropharyngeal lymph nodes, salivary glands, cervical lymph nodes, and skin. Less common sources of infection are penetrating traumatic injuries and foreign bodies, infected developmental cysts such as a branchial cleft cyst or thyroglossal duct cyst, and malignant lymph nodes [12, 13]. Occasionally, the exact source of head and neck infection may be unclear [14]. Non-odontogenic infections such as tonsillitis, pharyngitis, otitis interna/media, parotitis, and suppurative cervical adenitis are more common in children and adolescents in comparison to adults where an odontogenic source is usually the cause of infection [15].

5.1.2 Pediatric Odontogenic and Non-odontogenic Infections

Epidemiological studies from the United States estimated the national incidence of deep neck space infections (DNSIs) to be 4.6 per 100,000 children, and most cases occur in children under 6 years of age. The majority of DNSIs in children occur due to tonsillitis, pharyngitis, and suppurative cervical adenitis in comparison to adults where an odontogenic source is usually the cause of infection. When odontogenic infections occur in children, they are usually upper face, maxillary, or buccal infections [16]. Unlike adults, children respond well to intravenous (IV) antibiotic therapy and may not need surgical drainage; therefore, a trial of IV antibiotic therapy and fluids is recommended before surgical intervention [17]. Indications for surgical intervention of DNSIs include signs of airway compromise, presence of complications, no clinical improvement after 48 h of IV antibiotics, abscess >2.2 cm on CT imaging, age <4 years, and ICU admission [18]. Children with odontogenic infections also have fewer complications compared to adults and require a shorter length of stay when admitted to the hospital [16]. Cheng et al. reviewed 178 cases of deep neck space infections in children and reported a complication rate of 2.2%. Retropharyngeal abscesses caused by infected cervical lymph nodes that receive drainage from the nose, sinuses, and pharynx occur in early childhood. These abscesses can cause life-threatening complications in younger children. The typical symptoms are fever: irritability, neck pain, neck swelling, unilateral bulging of the posterior pharynx, dysphagia, excessive drooling, and airway distress or stridor. These infections become less frequent in later years as the lymph nodes become smaller with age [19].

5.1.3 Complications of Odontogenic and Non-odontogenic Infections

The majority of patients with moderate to severe odontogenic or non-odontogenic infections can be treated by admission to the hospital, removal of the source of infection, surgical drainage of the abscess, intravenous antibiotic therapy, and supportive care with fluids and nutrition. Sometimes, these infections progress to develop life-threatening complications due to *airway compromise, or due to extension of the infection into the mediastinum, lungs, orbit and/or brain, and blood resulting in severe sepsis or septic shock, and occasionally death* [7, 20, 21]. Airway distress and obstruction is the most commonly reported complication of head and neck infections [22].

Other frequently reported complications include *descending necrotizing mediastinitis (DNM)* [23], *cervical necrotizing fasciitis (CNF)* [24], *Lemierre's syndrome or internal jugular vein thrombosis (IJVT)* [25], *thoracic empyema* [26], *brain abscess* [27, 28], *cavernous sinus thrombosis (CST)* [29], *orbital apex syndrome,* subdural empyema [30], *osteomyelitis, severe sepsis*, *septic shock* [31, 32], and occasionally *death* [33, 34]. Such serious life-threatening complications have been reported to occur in about 10–40% of all patients treated for head and neck infections [35, 36]. These complications are often difficult to manage, and they can cause severe morbidity and mortality; in particular, airway obstruction, descending necrotizing mediastinitis (DNM), and septic shock frequently result in fatal outcomes [37–40]. Therefore, it is important that the treating specialist is able to recognize the early signs and symptoms of severe infections, and manage the complications to avoid a fatal outcome.

5.1.4 Morbidity and Mortality of Odontogenic and Non-odontogenic Infections

Before the availability of antibiotics, odontogenic infections were associated with significant mortality in the range of 40–50% [41–44]. In recent years, the mortality has decreased considerably and reports range from <1% to 10% [45, 46]. When fatalities do occur, they are usually in patients who have severe infections with concomitant underlying systemic illness, particularly immunocompromising disorders [34].

There is considerable variability in the reported mortality and morbidity of odontogenic and non-odontogenic infections, depending on the type of complication, comorbid illness, anatomical spaces involved, source of infection, and the virulence of the pathogen. Additionally, the lack of a standard method of grading the severity of odontogenic infections, differences in the study design, and the geographic variations contribute to some of the observed differences in morbidity and mortality [36]. This makes it difficult to interpret the results and to estimate the overall morbidity and mortality of odontogenic infections. Despite these challenges in interpreting results, there are similarities in the signs and symptoms, the principles of management, and treatment outcomes of complications of infections.

In 1998, *Chen, Mu Kan et al.* reviewed data gathered from 214 Taiwanese patients admitted with deep neck infections over a 10-year period (1988–1996) to identify predisposing risk factors of developing serious complications. They reported life-threatening complications in 18/214 (morbidity 8.5%) and death in 2/214 (mortality 0.9%) of their patients who were treated for odontogenic and non-odontogenic deep neck space infections. Complications included septic shock, descending mediastinitis, upper airway obstruction, pleural empyema, and disseminated intravascular coagulation (DIC) among others. A logistic regression analysis revealed that patients with an underlying comorbid systemic illness, delay time from onset of symptoms to treatment, and those with bilateral neck swelling had a positive correlation to major complications [47]. In a similar nationwide survey in Taiwan, Wong [48] reported <1% mortality or 1 death for every 150 patients admitted with maxillofacial infections. In this study, 2790 patients with maxillofacial infections were admitted to 33 medical centers over a 3-year period [48]. In another retrospective study of 185 patients treated for deep neck space infections between 1997 and 2002, serious complications were reported in 16.4% and death in 2.4% of their patients. In this study, older patients and those with underlying systemic illness had the most severe deep neck space infections with complications [3].

In recent years, Seppanen et al. [49] analyzed the records of 35 patients in Finland, hospitalized with severe odontogenic infections treated over a 2-year period (2000–2003) and reported 28% morbidity (10/35 patients) and 8.5% mortality (3/35 patients). In this study, death occurred in a higher proportion of admitted patients, likely due to study design, which included only records of severe odontogenic infections. They also reported more severe complications such as septicemia, endocarditis, brain abscess, and pulmonary complications in patients with comorbid illness [49]. According to a cross-sectional study conducted by the UK National Health Service (NHS) in 2006, about 3% of patients hospitalized for management

of odontogenic infections recovered with complications and 1% required a surgical airway [50]. Larawin et al. [36] reviewed the records of 103 patients with deep neck space infections (DNSIs) treated at Port Moresby General Hospital in Papua New Guinea between 1993 and 2005. Almost 50% of the DNSI in their series had an odontogenic source of infection. They reported death in 9/103 patients (8.9% mortality) and multiple complications including airway distress, descending mediastinitis, internal jugular vein thrombosis, orbital cellulitis, intracranial extension of infection, disseminated intravascular coagulation (DIC), and septic shock in 36/103 (37% morbidity). Similar to other studies, they found that patients with underlying systemic comorbid conditions such as cardiac valve disorders, liver cirrhosis, diabetes, lung sarcoidosis, and schizophrenia had increased the length of hospital stay and fatal outcomes [36].

More recently, Boscolo-Rizzo et al. reported life-threatening complications in 67/365 patients (18.4%) and death in 1/365 (0.3%) patients with deep neck infections who underwent surgical drainage and/or intravenous antimicrobial therapy. Diabetes mellitus and involvement of multiple deep neck spaces were the strongest independent predictors of developing complications [38]. In another retrospective analysis of 814 patients admitted with odontogenic infections over an 8-year period between 2004 and 2011, the reported mortality was 0.12% (1/814 patients) and serious complications occurred in 2% (14/814 patients) of the patients. All 14 patients who required intensive care had some comorbid illness such as diabetes, HIV infection, obesity, alcohol or tobacco use, and hypertension [43]. Adamson et al. [51] categorized their patients with deep neck space infections based on the type of complication. They reported a higher mortality in patients with necrotizing fasciitis (1/5) and Ludwig's angina (1/7) in their series of 55 patients treated at the Lagos University Teaching Hospital over a 1-year period (2014–2015). A linear regression analysis of their data revealed that the involvement of multiple spaces and anemia (low hemoglobin) were predictors of poor outcomes. Fatal outcomes occurred in 2/55 patients (3.5% mortality) [51].

Table 5.1 Risk factors for complications in patients with head and neck infections

Patient-related
– Systemic comorbid illness or compromised host immunity: diabetes mellitus, alcoholism, hepatitis C/HIV infection, substance use, psychiatric illness, chronic kidney disease, anemia, systemic lupus erythematosus, and cardiac or pulmonary disorders
– Local tissue factors: injury, radiation (XRT), chemotherapy, dehydration, and malnourishment
– Demographic factors: advanced age, geographic location, and socioeconomic status
– Socioeconomic: delay in seeking care, limited access to healthcare, and poor health literacy
Disease- or pathogen-related
– Type of pathogen (bacterial, viral, or fungal)
– Virulence of the pathogen
– Resistance of the pathogen to antibiotic therapy
– Anatomic spaces involved: parapharyngeal, retropharyngeal, prevertebral, pre-tracheal, and infratemporal
– Proximity of infection to vital structures—airway, great vessels, mediastinum, orbit, and skull base
Treatment-related
– Delay in diagnosis
– Inadequate imaging
– Delay in initiation of treatment
– Failure to identify and/or remove the source of infection
– Failure to drain fluid/pus collection adequately
– Failure of response to surgical treatment or resistance to antibiotic therapy

Risk factors of complications: A review of the literature on odontogenic and non-odontogenic infections revealed the following typical characteristics of patients who are at risk of developing complications: older males (age >55 years), with underlying systemic illness and dental source of infection and poor access to a tertiary care facility. The predisposing risk factors can be divided broadly into three categories: *host* factors, *pathogen or disease* factors, and *treatment factors* (Table 5.1). Understanding these risk factors will allow the surgeon/physician to predict and anticipate the potential complications, the degree of difficulty in airway management, the length of hospital stay, and the need for interdisciplinary or intensive care. More often than not, complications occur due to delay in recognition of the early signs of severe infection, delay in diagnosis or misdiagnosis or delay in treatment, and/or inadequate treatment.

Individuals with underlying systemic comorbidities such as diabetes and other immunosuppressive disorders such as HIV infection, lupus erythematosus, alcoholic liver cirrhosis, chronic renal failure, anemia, and substance misuse (alcohol, tobacco, and intravenous drugs) disorders are more likely to develop complications [47, 52]. Extremes of age (<5 years, >55 years), elevated BMI, and nutritional deficiencies are other factors that can increase the risk of complications. In a study comparing patients with and without HIV infection, it was noted that those with HIV infection had slightly increased length of stay, more serious complications of airway obstruction, sepsis, mediastinitis, and slightly increased mortality rate compared to patients without HIV infection. The authors of the study noted that HIV patients older than 55 years of age and CD4 count less than 350/mm^3 were more likely to have complications [53]. Similarly, patients with diabetes and involvement of multiple bilateral fascial neck spaces are at higher risk of developing Ludwig's angina compared to those without diabetes [54, 55]. Hidaka et al. also found that patients with diabetes were almost twice as likely to suffer from deep neck infections extending into multiple spaces compared to those without diabetes. Also, there is a difference in the cultured microorganisms with higher chance of isolating *Klebsiella pneumoniae* in patients with diabetes than those without (RR, 3.28; 95% CI, 2.52–4.26) [56, 57].

Several studies have identified the involvement of deep neck spaces, namely, the retropharyngeal and parapharyngeal spaces, or multiple fascial space involvement (Ludwig's angina) as the strong predictors of developing complications [58]. Jundt and Gutta [42] reported that patients with severe odontogenic infections who had systemic comorbidities and those with the involvement of a greater number of deep neck fascial spaces stayed longer in the hospital (average 8.25 days, range 5–26 days) and required intensive care compared to those without these risk factors [42].

Frequently, individuals in the lower socioeconomic strata with limited access to healthcare, poor health literacy, and alcoholism or psychiatric illness may delay seeking care and present to the Emergency Department (ED) with advanced signs of infection such as sepsis, dehydration, and airway distress. They often present after the infection has already violated anatomic barriers, and after it has spread to potential danger spaces allowing extension of infection into the mediastinum or brain or the bloodstream. Elderly and malnourished patients living in poor hygienic conditions or nursing homes are also at higher risk of developing complications. The treating surgeon or physician should identify predictors of potential complications based on the rapidity of progression of symptoms and clinical and radiographic signs (Table 5.2). Prompt interven-

Table 5.2 Clinical and radiographic signs in patients with head and neck infections at risk for complications

a. Clinical symptoms and signs	
Deep neck space infections	Upper or midface infections
• Dyspnea/difficulty breathing	• Fever and malaise
• Dysphagia and/or odynophagia	• Severe periorbital edema
• Drooling of saliva	• Proptosis of eyes
• Dysphonia	• Injection of conjunctiva
• Neck pain and limitation of movement	• Limited extraocular movements
• Severe trismus (MIO <15 mm)	
• Swelling or elevation of floor of mouth and tongue	
• Skin discoloration and crepitus	

b. Radiographic signs
– Thickening and diffuse enhancement of the cervical fascia
– Gas/air in the subcutaneous tissues in the absence of trauma/surgery
– Fluid collection or edema in retropharyngeal, parapharyngeal, and infratemporal spaces
– Involvement of bilateral neck spaces: submandibular, sublingual, and submental
– Involvement of infrahyoid spaces: pre-tracheal, retropharyngeal, prevertebral, and danger space
– Involvement of infratemporal, temporal, and periorbital spaces
– Involvement of paranasal sinuses, particularly sphenoid and frontal
– Severe constriction or deviation of oropharyngeal and hypopharyngeal airway

tion with adequate surgical drainage, careful management of airway, and removal of the source can improve the treatment outcomes of complications [59].

Perhaps, the most difficult risk factor to control is the pathogen itself or disease-related factors. The offending pathogens in the majority of odontogenic and non-odontogenic infections are mixed aerobic and anaerobic bacteria, but occasionally mycobacterial or fungal pathogens or a virus may be the causative organisms. Some bacteria such as the *Streptococcus milleri* bacteria within the *Streptococcus anginosus* group are more virulent than others and have the potential to cause rapid tissue destruction by necrosis or release of exotoxins allowing the fulminant spread of infection across tissue planes. Hasegawa et al. [60] reported an increased risk of complications in patients with non-odontogenic tonsillar and salivary gland infections caused by the *Streptococcus milleri* group of bacteria [60]. Kuriyama et al. noted that 24% of the isolated organisms failed to respond to two commonly used antibiotics (ceftriaxone and amoxicillin–clavulanate) [61]. The ability of the organisms to produce the enzyme beta-lactamase is one of the important mechanisms of developing antibiotic resistance. The beta-lactamase-producing bacteria can protect themselves and other penicillin-susceptible organisms from beta-lactam antibiotics, thereby decreasing their response to treatment. Bacteria that produce the *enzyme β-lactamase* or the enzyme *penicillinase* can *hydrolyze* the β-lactam ring of the antibiotic, rendering the antibiotic ineffective. Therefore, they have to be combined with a β-lactamase inhibitor. The lack of response to antibiotics increases the risk of spread of infection and associated complications, despite treatment [10, 11, 46].

The proximity of the anatomic spaces to vital structures such as airway, thoracic cavity, great vessels in the neck, paranasal sinuses, and cranial base increases the risk of complications by facilitating the spread of infection into lungs, mediastinum, orbit, or brain. Infections that involve the infrahyoid fascial spaces such as the pre-tracheal space, retropharyngeal space, danger space, and prevertebral space or infection that involve bilateral neck spaces increases the risk of complications such as airway distress, mediastinitis, Internal jugular vein thrombosis, and/or sepsis [62]. Infections of the midface region and periorbital region and paranasal sinuses increase the risk of complications such as cavernous sinus thrombosis or brain abscess.

Applied anatomy and imaging: One of the most critical steps in the management of head and neck infections and the associated complications is careful consideration of the anatomic boundaries of the spaces involved. Understanding the anatomy helps the surgeon to trace the path of spread of the infection to the critical regions from the source and to plan the surgical intervention. Identifying the involved spaces and their proximity to vital structures and organs such as the airway, major vessels, lungs, brain, and eyes can help the treating surgeon or physician to predict the risk of potential complications (Table 5.3).

Applied anatomy: The fascia of the neck is divided into two main layers: superficial and deep. The *superficial cervical fascia* is a part of

Table 5.3 Risk severity of deep neck space infections based on anatomy

Low to moderate risk
Suprahyoid spaces spanning across the neck bilaterally:
Submandibular
Sublingual
Submental
Moderate to high risk
Suprahyoid spaces with indirect access to thorax and brain:
Pterygomandibular space
• Infratemporal
• Temporal
• Sub-masseteric
Parotid
Parapharyngeal space
High risk
Infrahyoid spaces with direct access to mediastinum:
Anterior visceral space
High risk
Spaces spanning the entire length of the neck and thorax:
Prevertebral space
Danger space
Carotid space
Retropharyngeal space

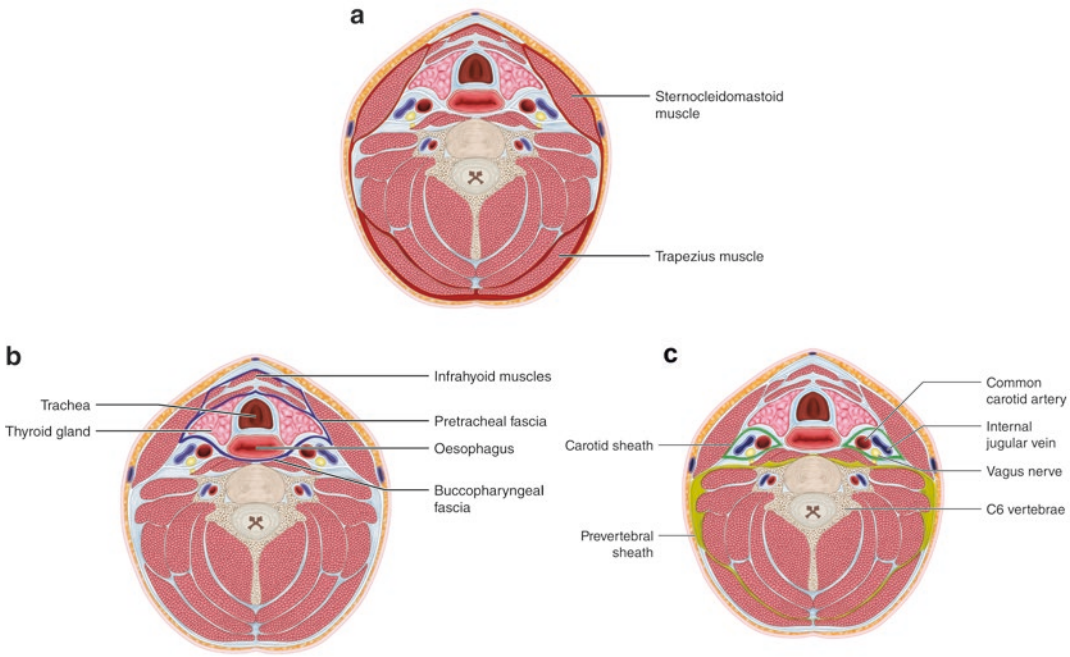


Fig. 5.1 (**a**, **b**, **c**) Spaces between the three layers of the deep cervical fascia

the subcutaneous fatty tissue that extends all the way from head to the chest shoulder and axilla covering the anterior, posterior, and lateral aspects of the neck. This superficial layer splits to enclose the platysma muscle in the anterolateral neck.

The deep layer is divided into three fascial layers: superficial (investing), middle (visceral), and deep (pre and paravertebral) (Fig. 5.1a–c).

The *investing layer of the deep cervical fascia* extends from nuchal line of the skull to clavicles and axillary region. It surrounds the neck and encompasses two muscles, the sternocleidomastoid (SCM) and trapezius muscles, and two glands, the parotid and submandibular salivary glands. In the anterolateral aspect of the neck, this investing layer has a suprahyoid and infrahyoid component.

The *visceral or middle layer of the deep cervical fascia* has a muscular and visceral division. The muscular division invests the strap muscles of the neck (sternohyoid, sternothyroid, thyrohyoid, and omohyoid). It attaches superiorly to the hyoid bone and the thyroid cartilage and inferiorly to the sternum and clavicle. The visceral division encloses the important viscera in the neck: pharynx, larynx, esophagus, trachea thyroid, and parathyroid glands.

The *deep layer of the deep cervical fascia* arises from the cervical spine and nuchal ligament forming a complete fascial sheath around the neck. The deep layer splits into the prevertebral and the alar divisions. The *prevertebral layer* extends from the base of skull to the coccyx and lines the vertebral bodies and transverse processes and forms the posterior wall of the danger space. The *alar layer*, which extends all the way from the base of the skull and extends to the second thoracic vertebra (T2), forms the anterior wall of the danger space and separates this space the retropharyngeal space.

The great vessels of the neck (carotid artery and internal jugular vein) lie just outside the deep cervical fascial sheath, and the phrenic nerve lies within this sheath. The carotid sheath is formed by all three layers of the deep cervical fascia and extends from the base of the skull through the posterior portion of the pterygomandibular space to the chest below the clavicle. It contains the internal jugular vein, carotid artery, and vagus nerve [63, 64].

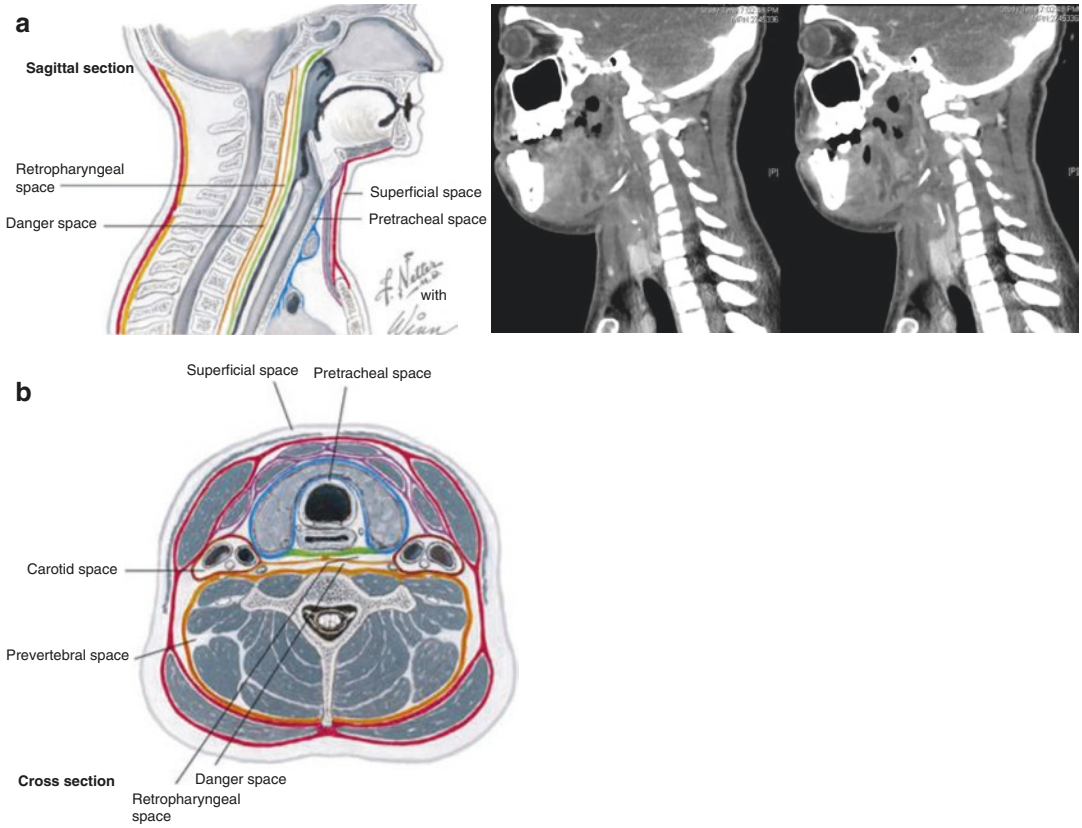


Fig. 5.2 (**a**) Pathways to mediastinum: sagittal view of the deep neck spaces. (**b**) Pathways to the mediastinum: axial view of the deep neck spaces between the layers of cervical fascia. (Netter medical illustration used with permission of Elsevier. All rights reserved)

There are three potential pathways for spread of a neck infection into the mediastinum (Fig. 5.2a, b): (1) the pre-tracheal route to the anterior mediastinum, (2) the lateral pharyngeal route to the middle mediastinum, and (3) the retropharyngeal-retro-visceral route to the posterior mediastinum. The absence of barriers between the cervical fasciae and the anatomic continuity between the neck and mediastinum facilitates the extension of the infection from deep neck spaces via the anterior visceral space or retropharyngeal space into the thoracic cavity.

5.2 Infrahyoid Spaces

The *anterior visceral space* or pre-tracheal space extends from the hyoid to the sternal notch and allows direct extension of infections from the neck to the anterior mediastinum.

The *retropharyngeal space* extends from the skull base to the upper mediastinum and contains only loose fatty tissue in its infrahyoid portion. Retropharyngeal space infections are considered high risk because of the proximity of this space to the airway and to the mediastinum. The tonsils or adenoids are the primary sources of retropharyngeal infection, but odontogenic infections from the parapharyngeal space and other suprahyoid spaces can spread to the retropharyngeal and danger space. Infections can spread to the mediastinum and cause acute mediastinitis, which may further spread and cause thoracic empyema and pericarditis. Spread of infection into the systemic circulation can result in sepsis or intracranial infection.

The *danger space* lies posterior to the retropharyngeal space and extends from the skull base down into the posterior mediastinum to the level of the diaphragm. The prevertebral fascia forms

the posterior wall of the danger space, and alar layer forms the anterior wall of the danger space and separates this space the retropharyngeal space. Infections in the retropharyngeal and parapharyngeal spaces can spread to the danger space. Danger space infections may initially present in the same clinical manner as retropharyngeal infections. Involvement of this space can result in the extension of infection into the posterior mediastinum and serious complications such as mediastinitis, empyema, and sepsis.

5.3 Suprahyoid Spaces

Parapharyngeal space is shaped like an inverted pyramid with its base at the base of the skull and tip at the hyoid bone, and lacks superior and inferior boundaries. This space is divided into the pre-styloid and post-styloid compartments. The *post-styloid* compartment has vital neurovascular structures of the carotid sheath (carotid artery, internal jugular vein, cervical sympathetic chain, and cranial nerves IX, X, XI, and XII). The *pre-styloid* compartment of this space contains fat. The parapharyngeal space is central to spread of infection to and from surrounding spaces. It is surrounded by and communicates with the other spaces, namely—retropharyngeal, submandibular, parotid, and pterygomandibular (masticator) spaces, allowing infections to spread from or extend into deeper spaces of the neck. Early diagnosis of parapharyngeal infections is important because of the potential to develop airway edema and obstruction and more severe complications such as Lemierre's syndrome (internal jugular vein thrombosis), septicemia, carotid artery aneurysm or rupture, ipsilateral Horner's syndrome, and cranial nerve IX–XII palsies.

Pterygomandibular/masticator space (Fig. 5.3a)*:* Infection arising from the lower posterior molars, particularly, the third molars, spreads into the deeper spaces via the masticator space. The masticator space encompasses the posterior body of the mandible, ramus, and condyle, and the muscles of mastication. This space is frequently involved in odontogenic infections arising from mandibular third molars. From the masticator space, the infection can spread medially into the parapharyngeal space, which is adjacent and medial to the medial pterygoid muscle. Infections from the masticator space can spread posterolaterally into the parotid space [65]. The submandibular and pterygomandibular spaces communicate with the parapharyngeal space and deeper visceral spaces of the neck which in turn communicate with the mediastinum allowing the spread of infection from the superior portion of the neck into the deeper neck spaces causing mediastinitis. The involvement of bilateral neck spaces allows the infection to spread across the neck and floor of the mouth causing the clinical presentation of Ludwig's angina.

5.4 Applied Anatomy of Upper Facial and Orbital Infections and Pathways (Fig. 5.3b, c)

The infection from the maxillary teeth can spread into the canine space, buccal space, and infratemporal and temporal spaces. Infections of the temporal and infratemporal spaces can be complicated due to the proximity to the cranial base. These infections can be complicated by spread into the periorbital tissues causing periorbital cellulitis and further spread of infection into the orbit. Infection from the posterior teeth can spread via the pterygoid venous plexus into the cavernous sinus.

The cavernous sinuses are located in the middle cranial fossa on either side of the sella turcica, and they are connected by intercavernous sinuses with septa within the sinuses. The anatomy of the cavernous sinus lends itself to an increased likelihood of entrapment of emboli or thrombus formation and hematogenous spread of infection into the brain as well as to other organs. The cavernous sinus is most often affected by thrombosis of all dural venous sinuses. Cranial nerve VI runs along with ICA within the sinus whereas CN III, IV, and V2 are in the lateral wall of the sinuses. The spread of infection from the teeth usually occurs via the pterygoid venous plexus in the infratemporal fossa, which communicates via the

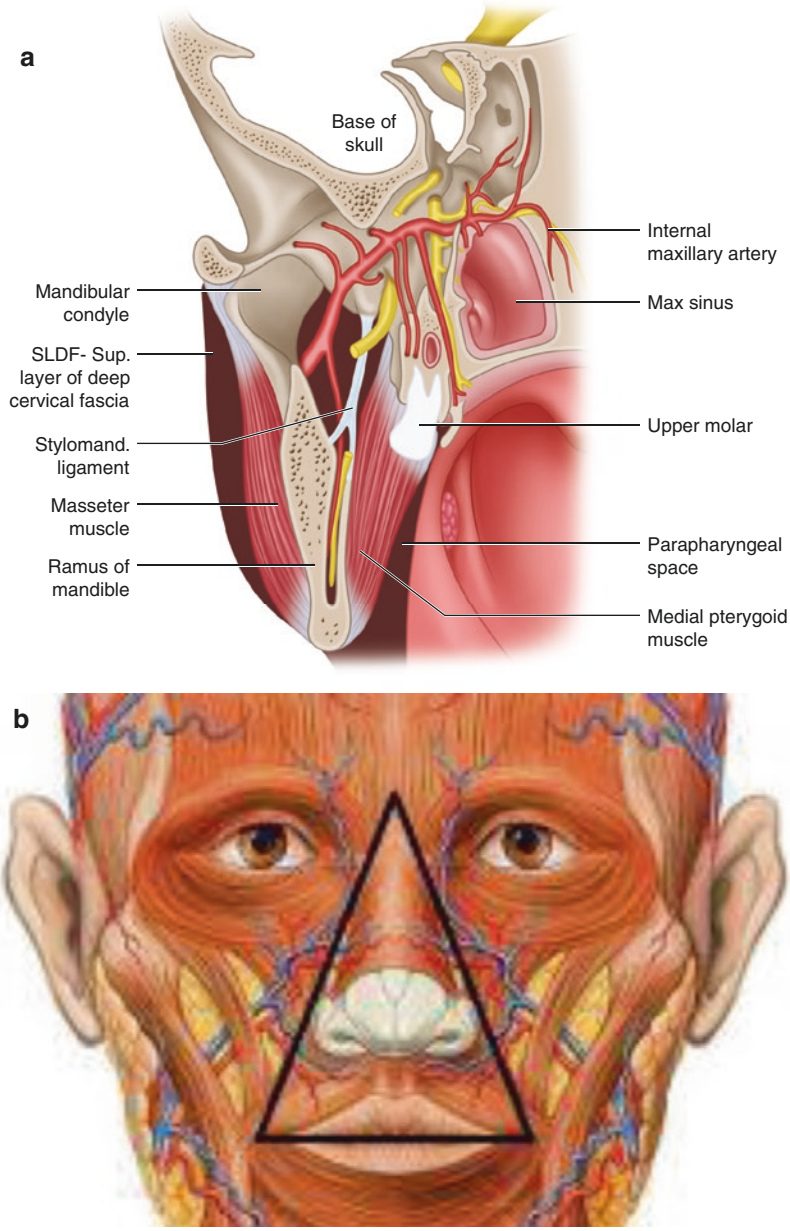


Fig. 5.3 (**a**) Masticator space in frontal view. Confluence of sub-masseteric, pterygomandibular, temporal, and infratemporal spaces. (**b**) Danger triangle and the pathway of infection to the orbit and cavernous sinus. (Netter medical illustration used with permission of Elsevier. All rights reserved)

inferior orbital fissure to the cavernous sinus. Paranasal sinus infection can spread via the pterygoid venous plexus or by direct extension from the sphenoid sinus, which lies adjacent to the cavernous sinus.

Alternatively, the infection may spread from the midfacial region via the facial venous system, which drains into the angular vein at the inner canthus of the orbit and is continuous with the nasofrontal vein and superior ophthal-

mic vein, which communicates with the anterior aspect of the cavernous sinus. The angular and ophthalmic veins do not have valves and permit retrograde flow and spread of infection from the upper-middle face into the brain via the cavernous sinus, and cause septic emboli [66]. The primary source of sepsis may also be a distant focus with septicemia preceding thrombosis of the cavernous sinus. Bacterial vegetations can seed heart valves, which, in turn, can cause septic emboli to disseminate into the lungs causing multilobar consolidation and pneumonia.

Diagnostic imaging: Contrast-enhanced CT scan is the gold standard and the main diagnostic imaging modality for infections of the deep neck spaces and midface and upper face. Reviewing the diagnostic imaging to identify the abscess or cellulitis and its relationship to vital anatomic structures and airway helps the surgeon to plan management of the airway and surgical drainage while preventing further spread of the infection. The decision to consider medical treatment in the absence of a fluid collection or image-guided aspiration versus incision and drainage in the presence of fluid collection is only possible with good imaging.

Fluid and fat stranding and obliteration of the fat and loss of the definition in the subcutaneous tissues along the fascial planes may be representative of cellulitis. Single or multiple low-attenuation areas may be seen in the path of spread of infection. Enlargement of the involved muscles, increased density, and enhancement of muscles may be seen in myositis. Heterogeneous changes in the muscles with areas of lower attenuation are suggestive of abscess formation, and rim-enhanced hypodense areas indicate abscess formation.

Magnetic Resonance Imaging (MRI) is valuable especially in patients with intracranial involvement or complications as in brain abscess or CST. MR is also a valuable tool to assess marrow edema and changes in the surrounding soft tissues in the evaluation of suspected osteomyelitis [67].

5.5 Complications of Odontogenic and Non-Odontogenic Infections

5.5.1 Airway Obstruction or Distress

Airway distress is one of the most frequent and well-documented complications in the management of head and neck infections [68, 69]. The reported incidence of airway complications is high and varies in different studies ranging from 5% to 40% [22, 70, 71]. The high incidence of airway-related complications is due to the alteration of the anatomy of the oropharynx and laryngopharynx caused by the edema and/or abscess formation. There can be deviation or constriction of the upper airway, swelling of the soft palate, inflammation of the surrounding pharyngeal soft tissues, and distortion of the epiglottis and tongue base. Furthermore, access to the upper airway and endotracheal intubation can be extremely difficult because of severe trismus with <15 mm of mouth opening as in patients with masticator space (pterygomandibular, temporal, and sub-masseteric spaces) infection or due to elevation of floor of the mouth and tongue as in patients with bilateral submandibular, sublingual, and submental space involvement (*Case 1*, Fig. 5.4a–d). *Infections of the temporal space are characterized by severe trismus (MIO <10 mm), severe pain over the temporal, and mandibular condyle areas making access to the airway difficult.* The presence of oral and nasal secretions, bleeding of friable or inflamed tissue, and pain make intubation challenging in these patients. Manipulation of the airway during intubation also increases the risk of rupture of the abscess and aspiration of the infected material and oral secretions. Involvement of the parapharyngeal, retropharyngeal, and anterior visceral space increases the risk of direct extension of the infection into the upper airway, lower airway, and mediastinum. This further increases the risk of pulmonary complications and sepsis resulting in high morbidity and mortality [45].

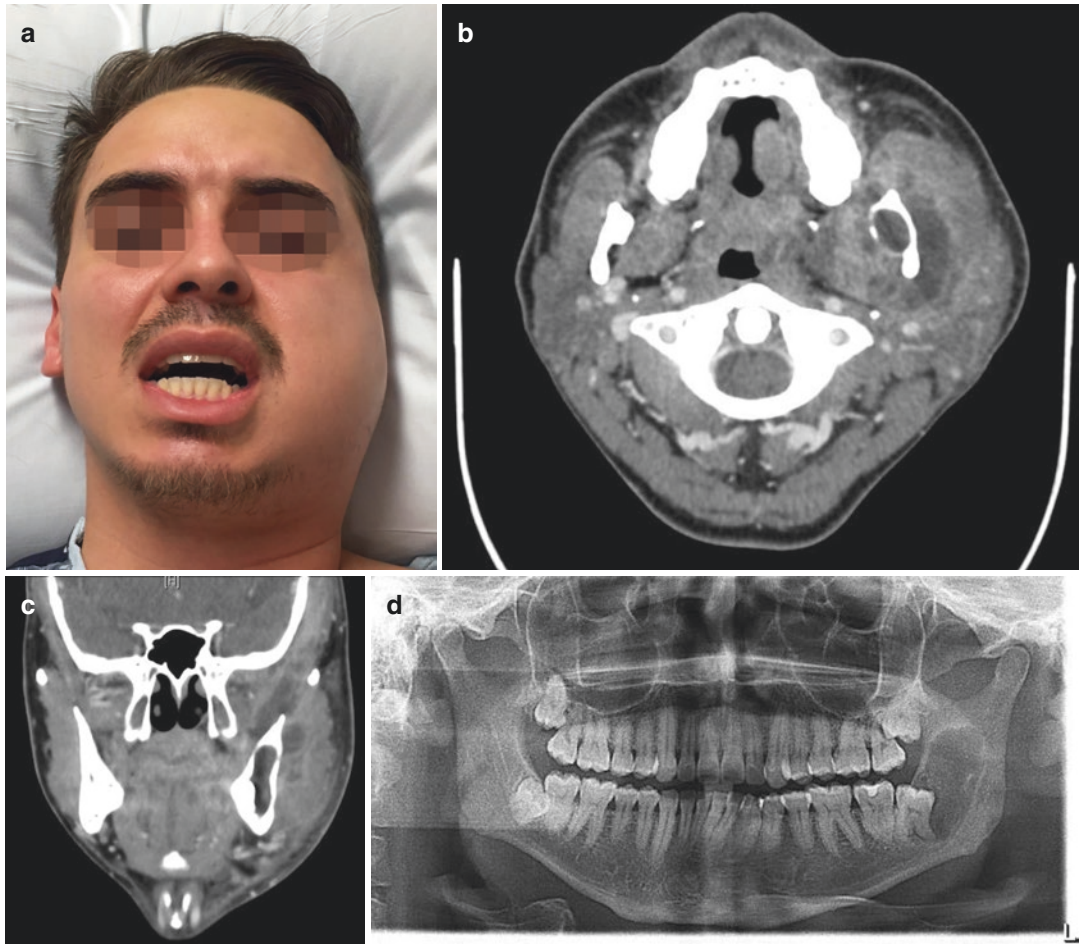


Fig. 5.4 (**a**, **b**, **c**, **d**) Airway access made difficult due to severe trismus. *Case 1*: 31-year-old Portuguese-speaking male presented to the Boston Medical Center ED with a 1 week history of left-sided facial pain, swelling, and severe trismus with opening to 1 cm. (**a**) 31-year-old male with severe trismus and maximal incisal opening <15 mm secondary to masticator space requiring endoscopic airway intubation. (**b**) Axial view of CT Scan, and (**c**) coronal view of CT scan showing infection involving the infratemporal, sub-masseteric, and pterygomandibular spaces arising from left mandibular third molar and associated odontogenic keratocyst. (**d**) Panoramic radiograph of infected left mandibular third molar and associated odontogenic keratocyst

The duration and progression of symptoms, the number and location of deep neck spaces involved, and the overall severity of infection influence the degree of difficulty of airway intubation. The signs of potential airway complications include severe trismus, dysphagia or odynophagia, change in voice or hoarseness, stridor, suprasternal retraction, tracheal deviation, and elevated tongue and floor of mouth with the involvement of bilateral neck spaces as in Ludwig's angina).

Complications of airway management in patients with odontogenic and non-odontogenic infections can be prevented the majority of the time by avoiding delay in securing the airway, by meticulous planning, and good communication between the surgical and anesthesia team members. The anesthesiologist should establish the

plan for airway management after a thorough assessment of the upper airway by measuring mouth opening, neck swelling and girth, tongue position, oral secretions, patient's ability to breathe in supine position, and CT or endoscopic examination of the oropharynx when feasible and available.

Airway management techniques in patients with head and neck infections include direct or indirect laryngoscopy/video laryngoscopy, awake fiberoptic endoscopic nasal or oral intubation, blind nasal intubation, and elective awake tracheotomy under local anesthesia [72–74]. Each technique of airway management has its advantages and disadvantages. Fiberoptic endoscopic intubation allows indirect visualization of the vocal cords and more accurate placement of the endotracheal tube. However, it is technique sensitive and based on the experience of the anesthesiologist. It can be difficult in an airway with copious secretions, blood, or purulence and abscess in the proximity of the airway. Also, an awake fiberoptic intubation can be unpleasant for patients, and manipulation of airway may have adverse effects such as hypertension, tachycardia, epistaxis, and oversedation with related hypoxemia. In a retrospective study by Tapiovaara et al. [75], the authors reviewed the records of 202 patients treated for deep neck space infections to assess mode of airway management: intubation versus tracheostomy [75]. Overall, 17% (35/202) of their patients had primary tracheotomy, and the majority of these were performed awake with local anesthesia to secure the airway safely. Tracheotomy after prolonged intubation for >7 days was necessary in 12% (25/202). The majority of their patients (165/202—82%) were managed by intubation and immediate extubation (102/202—50%) after surgical drainage [75]. Similarly, reports by other authors reveal that the need for tracheotomy was relatively high in patients with Ludwig's angina [76]. Others have shown that an awake tracheotomy can be avoided most of the time by using advanced airway management techniques such as upright positioning and endoscopic awake intubation, or GlideScope intubation in patients with airway compromise and deep neck space infections [77]. Cho et al. [22] noted that 42/71(56.8%) patients with deep neck space infections were intubated with direct laryngoscope using a Macintosh blade, and a video laryngoscope (Pentax AWS and McGrath) was used in 11/71 patients (14.9%). Fiberoptic bronchoscope was used in 13 patients (17.6%), and 4 patients (5.4%) already had a tracheostomy prior to arrival [22]. Video laryngoscopy can provide superior images of the larynx compared with a direct laryngoscopy, and when available should be used as an alternative in difficult airway situations. The use of a GlideScope requires an adequate opening of the mouth. Schumann et al. found that the longer the duration of infection symptoms, the smaller the interincisal distance. They were able to use the glide scope successfully in their patients to manage the difficult airway with improvement in interincisal distance from 20 to 26 mm after induction [74].

The need for tracheostomy and timing of tracheostomy should be critically assessed in each individual case. A tracheotomy is generally recommended in case of difficulty in intubating safely or in case of prolonged intubation greater than 7–10 days, which can increase the risk of laryngeal injury particularly posterior commissure stenosis. Based on the current literature, primary tracheotomy is required less often than was previously documented. The advantages of tracheotomy in comparison to prolonged intubation are decreased risk of laryngeal injury, avoiding the risk of accidental extubation, improved patient comfort by decreasing need for sedation, and the need for mechanical ventilation, shorter ICU stay, and lower cost [78].

Management of acute airway obstruction: In the event of an acute airway emergency, treatment should be aimed at relieving the hypoxia immediately. Epinephrine aerosols, a dose of steroids with topical vasoconstrictors to decrease the edema, can be useful in acute air-

way obstruction. Nasopharyngeal or oropharyngeal airways can be useful adjuncts, if the patient can tolerate. A large-bore 16-gauge angiocath for transtracheal positive pressure ventilation, intubation over a bougie, or a cook catheter are other useful adjunctive devices that can aid in the management of acute upper airway obstruction. If intubation fails and the patient cannot be ventilated adequately, then a cricothyroidotomy or tracheotomy must be performed. Less experienced trainees can perform a cricothyroidotomy more easily in an emergency, with less instruments and lesser risk of bleeding and pneumothorax. However, opponents may argue that a tracheotomy can be performed safely as well. The main disadvantage of a cricothyroidotomy is the development of voice changes due to stretching of cricothyroid muscles. The recommendation is to convert a cricothyroidotomy to a tracheotomy, after securing the airway emergently, if the need for long-term surgical airway is anticipated.

Prevention of airway complications: It is very important for the surgeon to perform a good clinical exam of the head and neck region before incision and surgical drainage. Emergency room physicians should have an airway management protocol in place when assessing patients with deep neck space infection. For patients who present to the ED with deep neck space infection or Ludwig's angina and airway distress, secure the airway prior to further investigations such as CT imaging (*Case 2,* Fig. 5.5a, b). Patients should be appropriately consented for tracheotomy in the event of loss of airway or inability to ventilate and secure the airway. The surgical and anesthesia team members should review the imaging (CT scan) and clinical examination findings to assess deviation or constriction of the airway, and they should communicate with each other the plan to secure the airway safely. They should be especially cautious, when there are signs of *trismus with MIO <15 mm*, *swelling or elevation of the floor of mouth and tongue*, *involvement of bilateral neck spaces*, *deviation of soft palate and parapharyngeal or retropharyngeal edema or abscess formation,* and *when deviation or constriction of the upper airway is*

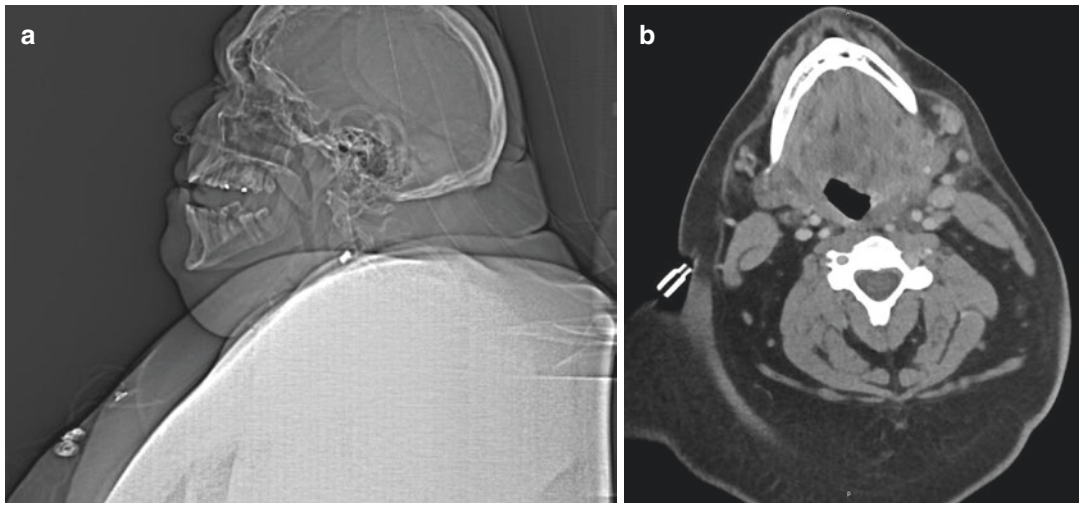


Fig. 5.5 (**a**, **b**) Ludwig's angina in a patient with morbid obesity. *Case 2*: (**a**) Patient with morbid obesity and Ludwig's angina showing the bilateral neck swelling, tongue elevated in the floor of the mouth making airway access difficult. (**b**) Axial view of CT scan of the patient showing airway deviation to the right, hypodense area in the floor of mouth and sublingual space indicating edema or abscess formation

noted on imaging (*Case 3,* Fig. 5.6a–d). An endoscopic examination of the upper airway can be performed with local anesthesia and vasoconstrictors before administering general anesthesia. Awake fiberoptic endoscopic intubation via the nasal cavity or oral cavity or video laryngoscope should be used (GlideScope Ranger (Verathon, Inc, Bothell, WA); McGrath MAC (Aircraft Medical Ltd, Edinburgh, UK)) when the upper airway is edematous or there is a distortion of the surrounding tissues. Careful management of the airway and removal of the source of infection are critical factors in achieving the most desirable treatment outcomes in the management of patients with severe odontogenic infections [59].

5.5.2 Descending Necrotizing Mediastinitis (DNM)

Mediastinitis is a rare life-threatening complication of severe odontogenic and non-odontogenic deep neck space infections typically involving retropharyngeal and anterior visceral spaces that extend into the thoracic cavity. It is second to airway obstruction among the list of reported complications of head and neck infections [79]. Descending necrotizing mediastinitis (DNM) as the name suggests is a rapid downward dissection of deep neck space infection into mediastinum with systemic spread of infection leading to sepsis, septic shock, and end-organ failure, resulting in fatal outcomes. Pearse first described this condition in 1938 with death in almost 50% of their 110 patients [80]. The reported mortality of this condition ranges between 11 and 40% [81, 82]. Reports of mortality in the last decade are slightly lower and range from 5 to 20%. Severe sepsis, septic shock, and complications such as pleural effusion, pneumonia, pericardial effusion, respiratory failure, and pneumothorax were the risk factors that correlated with a poor prognosis and fatal outcome [23].

There are three potential pathways for the spread of a neck infection into the mediastinum: (1) the pre-tracheal route to the anterior mediastinum, (2) the lateral pharyngeal (viscero-vascular) route to the middle mediastinum, and (3) most frequently via the retropharyngeal route to the posterior mediastinum [37]. The absence of barriers between the cervical fasciae and the anatomic continuity between the neck and mediastinum facilitates the extension of the infection from deep neck spaces via the anterior visceral space or retropharyngeal space into the thoracic cavity. In addition, to the absence of barriers, there is gravity and negative intrathoracic pressure, which contributes to the spread of these infections from the neck to chest [45]. The extension of infection from the neck via the retropharyngeal and danger space into the posterior mediastinum is seen in >70% cases in comparison to spread via the pre-tracheal space or anterior visceral space occurs less often in about 8% of neck infections [83].

In a recent systematic review on DNM, the authors reported that a large proportion of these infections arise from an odontogenic source (36–47%) and the pharyngeal area (33–45%). The non-odontogenic causes include peri-tonsillar abscess, cervical lymphadenitis, or post-traumatic neck abscess, or acute suppurative sialadenitis [83]. Diagnosis of DNM is established by clinical exam and imaging typically, CT scan extending from head to chest. Dyspnea, hypoxia, jugular distention, thoracic/chest pain, crepitus/crackling on palpation over neck and chest, and respiratory failure are the classic clinical findings of mediastinitis. The clinical signs and symptoms may also be subtle or vague such as high fever, neck pain, restriction of neck movements, dysphagia, and dysphonia [45].

The radiologic findings on CT scan include swelling and thickening of the cervical fascia, edema of the muscles, and tracking of infection down through the neck with signs of fluid collec-

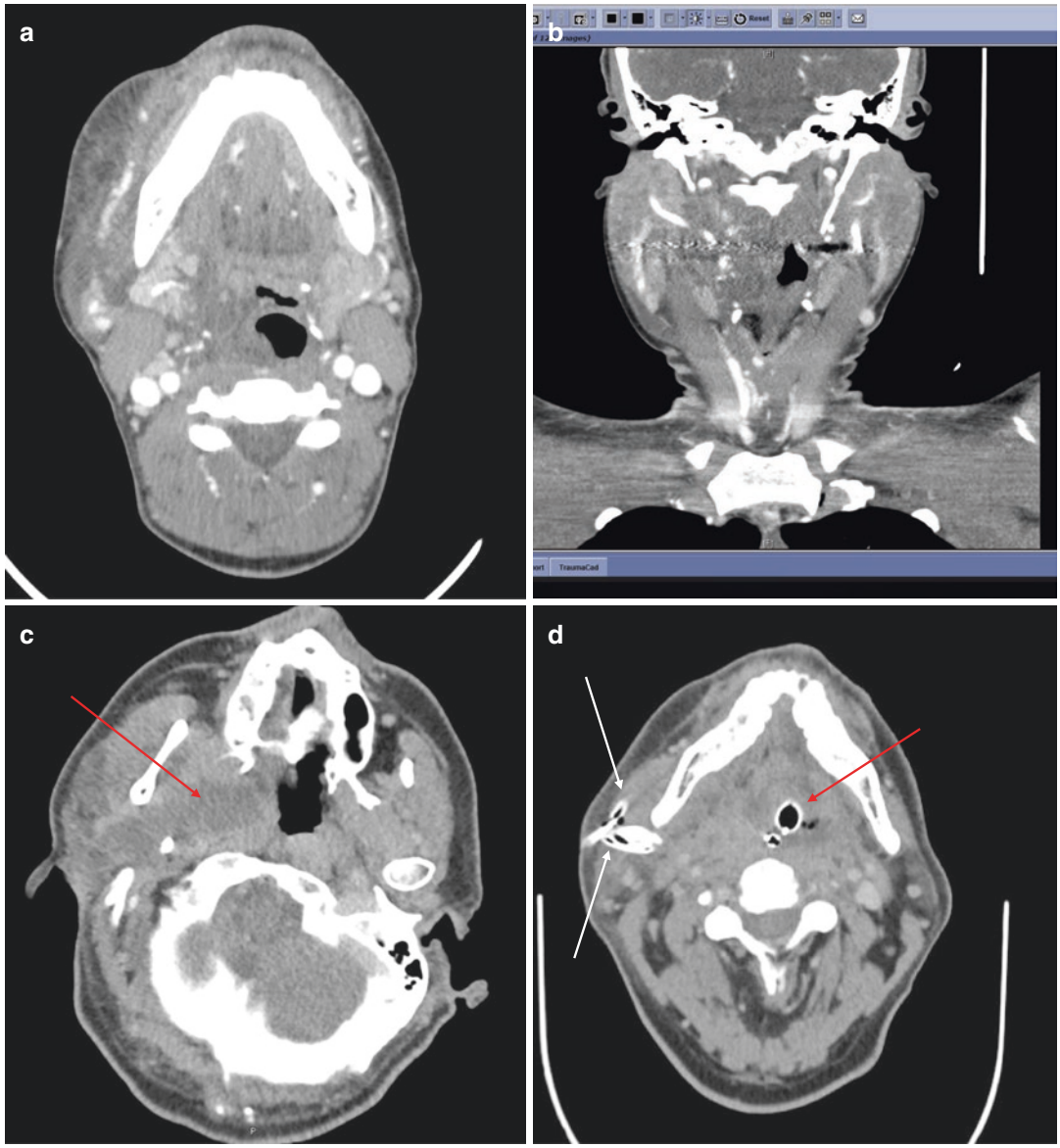


Fig. 5.6 (**a**, **b**, **c**, **d**) 56 Y M with airway deviation and constriction due to parapharyngeal and retropharyngeal infection making intubation difficult. *Case 3*: 56 Y M presented to his PCP with right facial pain and swelling of 1 week duration. He was referred to the ED with chills, and a fever of 101.3. Clinical exam was notable for marked trismus to 1 cm, asymmetric swelling of right face and jaw with tenderness, cervical lymphadenopathy and a non palpable inferior border of the mandible. (**a**) Axial view of CT scan and (**b**) coronal view of CT scan showing upper airway deviation and constriction causing airway distress due to lateral pharyngeal and retropharyngeal space infection. CT scan showing significant mass effect and partial effacement of the pharyngeal airway and cellulitis extending caudally into the mid and lower neck. (**c**) Image of airway before drainage of infection, and (**d**) edema of airway around endotracheal tube 10-days after drainage. Patient required a tracheotomy after 10 days of intubation (white arrows pointing to penrose drains and red arrow pointing to endotracheal tube)

tion or gas within the mediastinum (*Case 4,* Fig. 5.7a–j). Estrera et al. reported on the four classic diagnostic criteria for descending necrotizing mediastinitis: (1) a clinical manifestation of severe oropharyngeal infection, (2) the radiologic features of mediastinitis on CT, (3) docu-

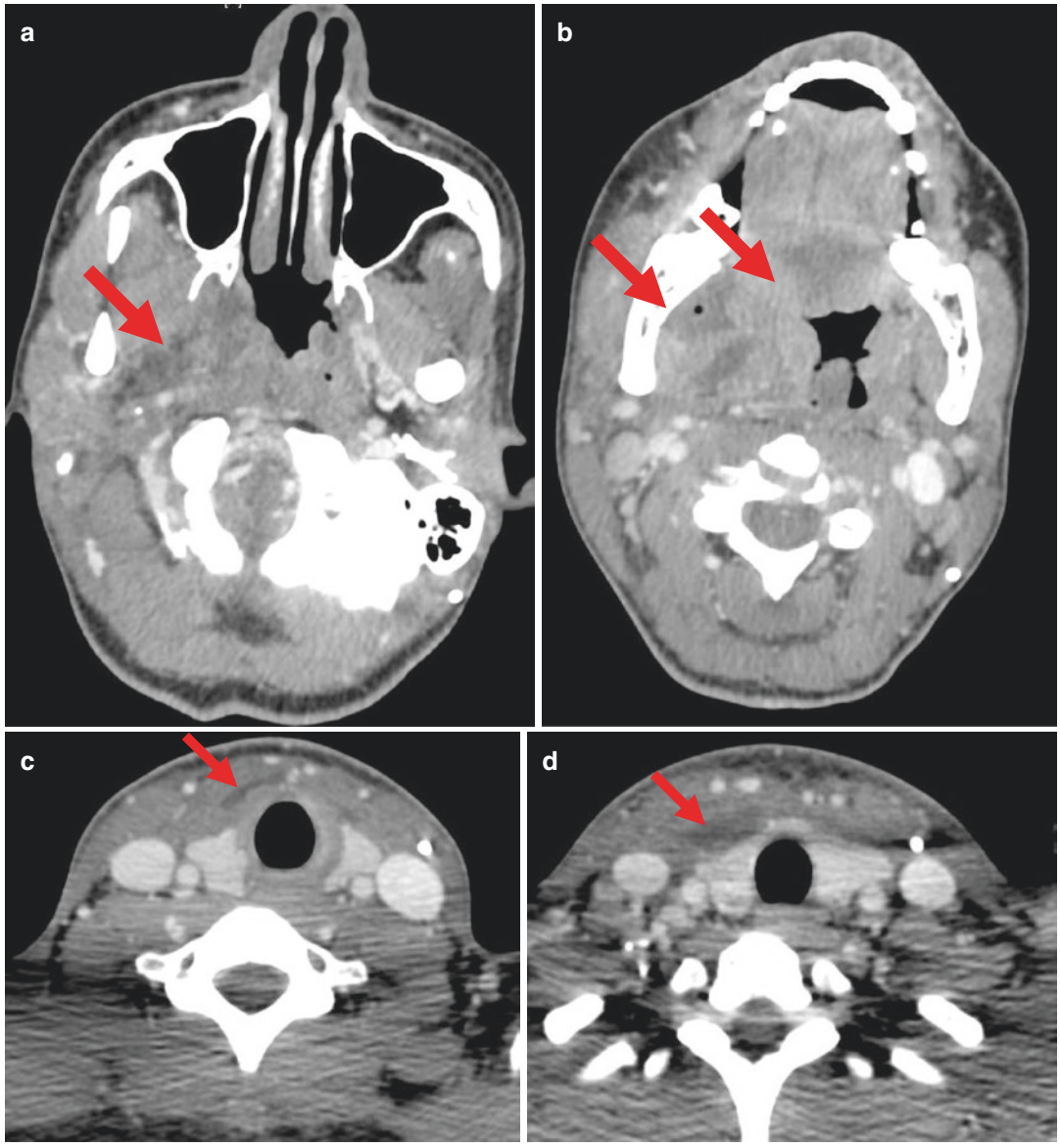


Fig. 5.7 (**a**–**d**) 31-year F with h/o anemia, seizure disorder, depression and anxiety presenting with odontogenic infection associated with lower molar that progressed to descending necrotizing mediastinitis. *Case 4*: 31-year-old female presents to the ED with 4 day history of lower right molar pain and increasing swelling on right side of face, and low grade temperature of 100.2° F was discharged with amoxicillin. The patient returned 2 days later to the emergency department with odynophagia, increased facial swelling, fevers and chills, dysphagia with difficulty handling secretions. (**a**–**d**) In these axial views the fluid collection in the lateral pharyngeal, submandibular, sublingual appears to be tracking from the pterygoid plates along the right mandible with extension into the pre-tracheal space to the right lobe of the thyroid gland and continuing into the anterior mediastinum. In these coronal views, the fluid collection in the lateral pharyngeal, submandibular, sublingual appears to be tracking from the **e**, **f** pterygoid plates along the right mandible with extension into **g**, **h** the pre-tracheal space to the right lobe of the thyroid gland and continuing into the anterior mediastinum. (**i**) POD1 showed successful drainage of pretracheal abscess with drains in the pre-tracheal space. (**j**) Three days after repeat I&D of all neck spaces, repeat CT scan shows deeper retrosternal collections requiring cardiothoracic intervention for mediastinotomy via transcervical approach (red arrow pointing to retrosternal collection)

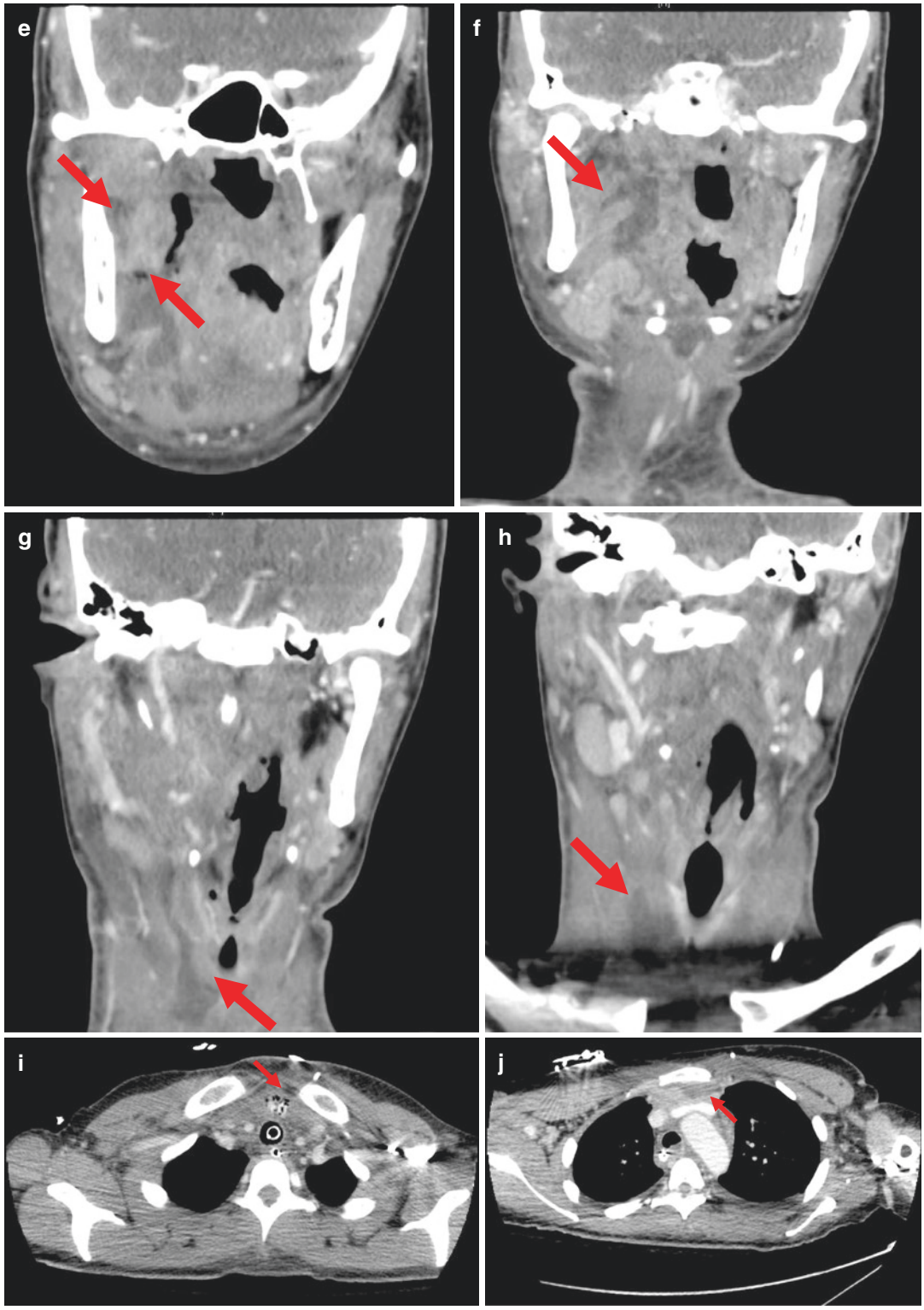


Fig. 5.7 (continued)

mentation of a necrotizing mediastinal infection at surgery or on postmortem examination, and (4) an established relationship between oropharyngeal infection and development of descending necrotizing mediastinitis [84]. Endo et al. classified the extent of DNM based on CT and intraoperative findings into Type I, where the infection is above the tracheal bifurcation or carina; Type IIA, when infection extends to the lower part of the anterior mediastinum; and Type IIB, when infection extends to the lower anterior and posterior mediastinum [85]. The microbiology of mediastinitis due to odontogenic infections typically shows mixed aerobic and anaerobic organisms. The most commonly reported organisms are beta-hemolytic streptococci, staphylococci *Bacteroides* species, *Pseudomonas aeruginosa*, and *Escherichia coli* [40].

The treatment of DNM consists of adequate drainage from the neck and mediastinum, removal of the source, and intravenous broad-spectrum antibiotics with close monitoring and intensive care. Management is challenging due to the difficulty in establishing good drainage of the mediastinum, leading to sepsis and organ failure resulting morbidity and mortality with reports ranging from 25 to 40% [81, 86]. Patients often require multiple operations with drainage of spaces and washout of wounds with postoperative cervicothoracic CT scans to monitor progress. The average length of hospital stay of these patients is about 35 days and ranges between 12 and 100 days.

Access for drainage can be obtained via transcervical, standard posterolateral thoracotomy, median sternotomy, and transthoracic via subxiphoid or clamshell approach. The thoracoscopic approach can also be used in select patients [87]. Surgical drainage through a cervical approach is possible if the infection is restricted to spaces above T4 or tracheal bifurcation, and a combined cervical and thoracic approach is usually necessary in all other cases. Corsten et al. reported a higher mortality of 47% in patients who had only transcervical approach for drainage of mediastinum in comparison to those who had drainage via transthoracic and transcervical mediastinal drainage (19%) [88]. It is important to monitor progress closely with cervicothoracic CT scans every 48–72 h after the initial drainage especially if there are signs of clinical deterioration [87]. Aggressive cervical drainage, early mediastinal exploration—debridement and drainage through a subxiphoid incision or thoracotomy is advocated to salvage the patient with descending necrotizing mediastinitis [89]. Inadequate drainage can result in sepsis and organ failure and fatal outcomes [90]. Early diagnosis and use of aggressive mediastinal drainage are fundamental to reducing the incidence of complications and the risk of developing septic shock [23].

5.5.3 Cervical Necrotizing Fasciitis

Necrotizing fasciitis (NF) is a fulminant infection with sudden onset, rapid progression, and high morbidity and mortality if treatment is inadequate or delayed. In the head and neck region, this life-threatening infection can arise from an odontogenic or non-odontogenic source such as penetrating trauma, tonsillar infection, burns, intravenous drug use, sinusitis, osteoradionecrosis, or minor surgical procedures [91–93]. The infection spreads quickly across tissues planes by liquefaction necrosis of subcutaneous fat and connective tissues. Tissue necrosis and release of bacterial toxins can mount a severe systemic inflammatory response leading to systemic toxicity and septic shock, vascular thrombosis, end-organ failure, and ultimately death. NF has been well documented throughout the history of medicine, particularly by surgeons who operated in the battlefield. Some of the former synonyms include *necrotizing cellulitis*, *gangrenous erysipelas*, *Fournier gangrene, and Meleney gangrene*. Meleney in 1924 was among the early reporters of this extensive infection with tissue destruction in a series of 20 patients caused by beta-hemolytic streptococci [94].

NF is rare and has a reported incidence of 0.2–1.7 per 100,000 person-years. Necrotizing fasciitis is even more rare in the head and neck region when compared to the other regions such as the extremities, abdominal wall, or perineum.

This is due to the rich blood supply and vascularity of the tissues. Cervical necrotizing fasciitis (CNF) constitutes about 2% (range 1–10%) of all cases of necrotizing fasciitis [95]. In general, older individuals with immunosuppressive disorders, diabetes, malnutrition, substance misuse, peripheral vascular disease, and renal failure are at greater risk of CNF, but it can occur in healthy young patients as well [94]. Pediatric necrotizing fasciitis due to odontogenic infection is very rare but can occur and has been reported [96].

In the head and neck region, the infection typically follows the superficial musculo aponeurotic system (SMAS) or the investing layer of the deep cervical fascia with rapid progressive liquefaction necrosis of the subcutaneous fat and connective tissues while sparing the skin, allowing the spread of infection into the mediastinum. Early signs and symptoms include fever, tachycardia, and dehydration with severe pain and firm, brawny swelling, and erythema of overlying skin [97]. Early symptoms of this condition are often similar to other deep neck space infections and therefore can be misleading. Malik et al. [98] in their case series describe some of the early warning symptoms and signs that should alert a clinician or provoke a high degree of suspicion of CNF [98] (Table 5.4).

Contrast-enhanced computed tomography (CT) of the neck is the main diagnostic investigation, and the chest should be included in the scan to determine if there is a mediastinal extension. Diffuse thickening of the subcutaneous fat cervical fascia and cervical muscles with gas and fluid collections are typical. Diffuse thickening with enhancement of the superficial and deep cervical fascia and fluid collections in the neck spaces is present in 100% of the cases. The presence of gas is a common finding in the imaging studies, but the absence of gas in the tissues does not preclude the diagnosis of CNF as seen in our illustrated case (*Case 5,* Fig. 5.8a–d) [79, 93].

Table 5.4 Characteristics of patients with cervical necrotizing fasciitis

Facial and/or neck swelling with unusual induration
Pain out of proportion to clinical signs
Pharyngeal swelling and pain
Subcutaneous emphysema with crepitus
Skin with erythema or purplish discoloration or mottled appearance
Immunocompromised Host—advanced age, diabetes, peripheral vascular disease, liver cirrhosis, alcoholism, and HIV infection
Signs of systemic toxicity—fever, tachycardia, rising WBC, and CRP
CT scan with diffuse enhancement and thickening of cervical fascia and fluid collections in neck compartments (100%)
CT scan with air/gas in tissues in the absence of trauma or surgery (60–65%)

(Adapted from Malik et al. [98])

Early diagnosis and aggressive debridement of the tissues to prevent further spread of infection is key to decreasing morbidity and mortality. Sarna et al. noted that early aggressive surgical therapy, tissue debridement, appropriate intravenous antibiotic administration, and intensive care improved outcomes with a reported mortality rate ranging between 7 and 20%. Early identification of the extent and severity of disease looking for signs of mediastinal or cranial extension is important. CT neck should include chest to assess the extension of infection into the thorax. Petitpas et al. [99] reported that among their cohort of 130 patients with CNF, 28% (37/130) had mediastinitis. In their retrospective study, the risk factors for the likelihood of mediastinal extension of the cervical infection were: (1) presence of a pharyngeal source of infection, (2) presence of gas at the source site of infection, and (3) use of oral corticosteroids prior to admission. The majority of their patients had either a pharyngeal source (42%) or dental source (37%) of infection [99].

Sarna et al. [79] reviewed the literature on patients with CNF and descending mediastinitis. Among the 100 cases of CNF identified, 76% were due to odontogenic infection. They reported a mortality rate of 41% in patients with CNF with DNM, compared to 20% in patients with CNF alone. The progression to sepsis was the worst prognostic indicator for both CNF and CNF with DNM with a mortality rate of 64% [79]. Similarly, Umeda et al. [100] reported a mortality rate of 19.2% in 125 patients with necrotizing fasciitis of odontogenic origin. Increased mortality was noted in patients who had (1) underlying immu-

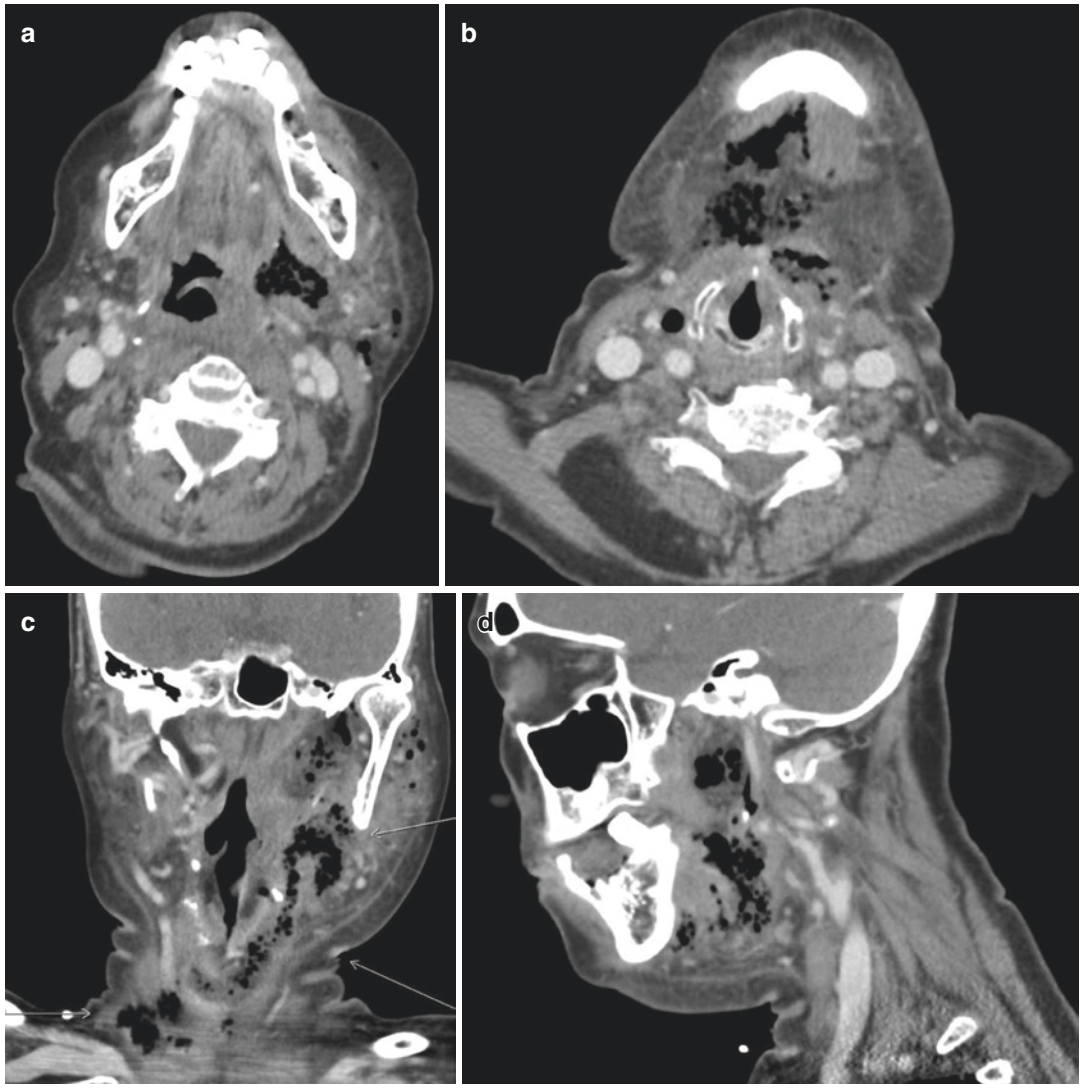


Fig. 5.8 *Case 5*: (**a**–**d**) Cervical necrotizing fasciitis in a 79-year-old female presented to ED with concerning neck infection after extraction of teeth #19,20 by an outside local oral surgeon. Her postoperative course was complicated by sudden onset neck swelling and sever dysphagia 3 days after extractions recalcaitrant to clindamycin. She has a h/o colon cancer s/p colectomy, complicated by metastasis to the lungs undergoing palliative chemotherapy. (**a**) Diffuse fluid collection and gas due to fulminant spread of infection from lower left posterior teeth (#19, 20) extraction sites to the right submental, submandibular, and left pretracheal can be seen on axial views of CT scan. (**b**) Left lateral pharyngeal space gas bubbles on axial cut. (**c**) Extensive subcutaneous emphysema indicating Left sub-masseteric, infratemporal, submandibular, lateral pharyngeal, and pre-tracheal abscesses extending to the contralateral side. (**d**) Sagittal view with diffuse subcutaneous emphysema a sign of ascending necrotizing fasciitis

nosuppressive disorder, (2) delay in surgical intervention, and (3) mediastinal extension of the infection [100]. Prognosis is poor with very high mortality rates approaching 100% in patients with CNF when surgical treatment is delayed or not rendered.

Morbidity is severe, even when these patients survive due to the facial disfigurement and loss of function, scar formation secondary to loss of tissue. Unlike cervical necrotizing fasciitis, craniofacial or facial NF is much less likely to progress to DNM, but can leave a significant cosmetic

deformity. Treatment consists of prompt surgical drainage of all the spaces involved and debridement of the necrotic tissues with adequate incisions to access the infection. Simultaneous medical therapy that includes the administration of intravenous fluids and intravenous antibiotics to avoid progression to septic shock is the hallmarks of treatment for NF. Antimicrobial agents for mixed aerobic and anaerobic bacteria that generally provides coverage for *S. aureus* as well as anaerobic bacteria include cefoxitin, clindamycin, imipenem, and the combinations of a b-lactamase inhibitor (i.e., clavulanic acid) and a penicillin (i.e., ticarcillin) and the combination of metronidazole plus a b-lactamase-resistant penicillin [101]. Some have advocated the use of HBO treatment. The rationale for hyperbaric oxygen (HBO) therapy in CNF is that it will improve tissue oxygenation, change the microbial environment, and inhibit the survival of anaerobic organisms.

Frequently, the initiating organisms are group A beta-hemolytic streptococci, *Streptococcus pyogenes,* and *Staphylococcus aureus*. The causative organisms may be aerobic, anaerobic, or mixed. Almost 60% of the main pathogens are anaerobic: *Peptostreptococcus*, *Bacteroides*, and *Fusobacterium* species [102]. In patients with CNF due to odontogenic source, it is suspected that the toxin-producing Gram-positive cocci and Gram-negative anaerobes are may be more virulent and have the potential for tissue destruction compared with infections originating from pharyngeal injury caused by ingestion of a sharp object or an iatrogenic injury during endoscopy or catheterization [79].

Lemierre's syndrome also known as internal jugular vein thrombosis (IJVT) or post-anginal septicemia is a rare vascular complication of deep neck space infections. It occurs most often as a complication of oropharyngeal infections such as acute tonsillitis or pharyngitis rather than odontogenic infection [103, 104]. It was first described by Dr. André Lemierre in 1936 with high mortality of 90% in the era before antibiotic therapy whereas the estimated fatality rate at present is around 4–12% [105]. The majority of patients affected by this condition are generally healthy young adults without a history of immunosuppression [106].

Suppurative thrombophlebitis of the internal jugular vein is most commonly associated with *Fusobacterium* species, which can spread from acute pharyngeal, periorbital, mastoidal, or occasionally from periodontal/dental tissues [107, 108]. *Fusobacterium necrophorum*, a Gram-negative, anaerobic normal flora of the oropharynx and female genital tract, has been identified as the most common causative agent. The bacterial cultures usually yield *Fusobacterium* species—*F. necrophorum*, or *F. nucleatum*, or Enterococcus species, streptococci group B, C, *Streptococcus oralis*, Staphylococci, *Eikenella corrodens*, *Peptostreptococcus* species, and *Bacteroides* species. In a small number of cases, the organism may not be identified [109].

Clinical symptoms include chills, sore throat, spiking fevers, and painful neck swelling at the angle of the lower jaw or along the sternocleidomastoid muscle (SCM). IJVT produces bacteremia, circulating septic thrombi with distant infection, or pulmonary embolism [104]. Progression of the disease is due to dissemination of the septic emboli to the lungs and other end organs leading to a severe septicemia, septic shock, and ultimately death if untreated. Post-contrast neck and chest CT scan is the main diagnostic imaging test. It allows the detection of pulmonary septic emboli in addition to examination of the internal jugular vein. The imaging typically demonstrates a filling defect of the IJV, thickening and enhancement of the vessel wall, and swelling of the surrounding soft tissues. Chest X-rays and chest CT scan will show pulmonary opacities indicating an abscess or parenchymal consolidation. Doppler ultrasound can also be used for the diagnosis of venous thrombi [104, 110].

Management includes medical treatment with antibiotic therapy with good anaerobic coverage, anticoagulation therapy, and supportive care in case of sepsis. Ampicillin with sulbactam or clindamycin or a third-generation cephalosporin in combination with metronidazole is typically used. The exact duration and type of anticoagulation therapy—heparin, low-molecular weight heparin, or vitamin K antagonists is not well established, but a mean duration of 3–4 weeks has been reported [104]. Surgical procedures may be required in some patients. These can

include drainage of the abscesses, tissue debridement, and removal of the source of infection. Internal jugular vein ligation is suggested only in case of persistent septic emboli despite antibiotic treatment [111]. Systemic septic embolism is the most severe complication, which can lead to severe sepsis, septic shock, and death.

Cavernous sinus thrombosis (CST): Septic cavernous sinus thrombosis is a rare and serious complication of non-odontogenic and odontogenic infections with severe morbid long-term consequences and high risk of mortality. Paranasal sinusitis, especially sphenoid and ethmoid sinusitis, has been reported as common source of infection in septic CST [112]. Skin infections of the midface, upper lip, nose and forehead (also known as the danger area of the face), mastoid and ear infections (otitis media), infection from the oropharynx, and teeth can spread to the cavernous sinus. Approximately, 10% of reported cases of cavernous sinus thrombosis (CST) may be due to an odontogenic source of infection [29]. The primary odontogenic foci of infection are usually maxillary posterior or maxillary anterior teeth, but mandibular teeth have also been implicated.

The anatomy of the cavernous sinus lends itself to an increased likelihood of entrapment of emboli or thrombus formation and hematogenous spread of infection into the brain as well as to other organs. Dental infection of posterior maxillary teeth can spread via the pterygoid venous plexus and the inferior orbital fissure into the orbit and the inferior ophthalmic vein, or the veins of foramen ovale and lacerum, which communicate with the cavernous sinus. Paranasal sinus infection can spread via the pterygoid venous plexus or by direct extension from the sphenoid sinus, which lies adjacent to the cavernous sinus. Alternatively, the infection may spread from the midfacial region via the facial venous system, which drains into the angular vein at the inner canthus of the orbit and is continuous with the nasofrontal vein and superior ophthalmic vein, which communicates with the anterior aspect of the cavernous sinus [113]. The angular and ophthalmic veins do not have valves and permit retrograde flow and spread of infection from the upper-middle face into the brain via the cavernous sinus, and cause septic emboli [66]. The primary source of sepsis may also be a distant focus with septicemia preceding thrombosis of the cavernous sinus.

The presenting clinical symptoms of CST can include fever, malaise, headache, facial and eye pain, periorbital swelling, and painful ophthalmoplegia. The most frequent symptoms and signs are fever, chemosis, ptosis, proptosis, external ophthalmoplegia/limitation of extraocular muscle movements—CN VI palsy (abducens) and periorbital swelling [113, 114]. The average latency period between the onset of the primary lesion and the onset of clinical signs is 5–6 days, and it can range anywhere between 1 and 27 days. A fundoscopic examination should be performed early, when CST is suspected, to confirm the diagnosis. *Blurred optic disc margin due to venous congestion* is *pathognomonic for CST*. The differential diagnosis of CST includes orbital cellulitis, orbital neoplasm, ethmoiditis, carotid-cavernous fistula, or exophthalmic goiter [113].

The cultured organisms vary based on the primary source of infection. The most commonly cultured organisms in CST are *Staphylococcus aureus* (60–70%) followed by *Streptococcus species* 20%. In cases of sinusitis, the isolated organisms are *Streptococcus* species usually *S. pneumoniae*, anaerobic *Streptococcus*, *Haemophilus* spp., and Gram-negative bacilli, i.e., *Prevotella*, *Bacteroides* spp., and *Fusobacterium* spp. Occasionally fungal organisms such as *Aspergillus* may cause CST [113].

Both high-resolution CT with contrast and MRI are useful imaging studies for diagnosis. The abnormal radiographic signs of CST include expansion of the cavernous sinus, convexity of the lateral wall instead of the normal concavity seen on coronal views, abnormal irregular filling defects of the cavernous sinus, dilatation of the superior ophthalmic vein, and thrombi in the sinus tributaries to the cavernous sinus. MRI is most helpful when reassessing patients with a non-diagnostic CT scan or for follow-up of residual neurological deficits. MR venography will show the absence of flow within the cavernous sinuses [115]. Management of patients with CST includes prompt empirical antibiotic therapy covering aerobic Gram-positive, Gram-negative, and anaerobic

organisms. Until the specific organisms are identified. Usually, a third-generation cephalosporin (ceftriaxone) and vancomycin or nafcillin and metronidazole have been suggested for initial broad-spectrum antibacterial coverage. Once the culture and sensitivity results are available, narrow-spectrum antibiotics can be administered. Simultaneous identification of the source of infection and surgical treatment to remove or treat the source of infection is essential. Surgical treatment in the form of functional endoscopic sinus surgery to drain sinuses or craniotomy may be necessary to drain subdural empyema (*Case 6*, Fig. 5.9a–g) or for clot retrieval and sometimes for orbital decom-

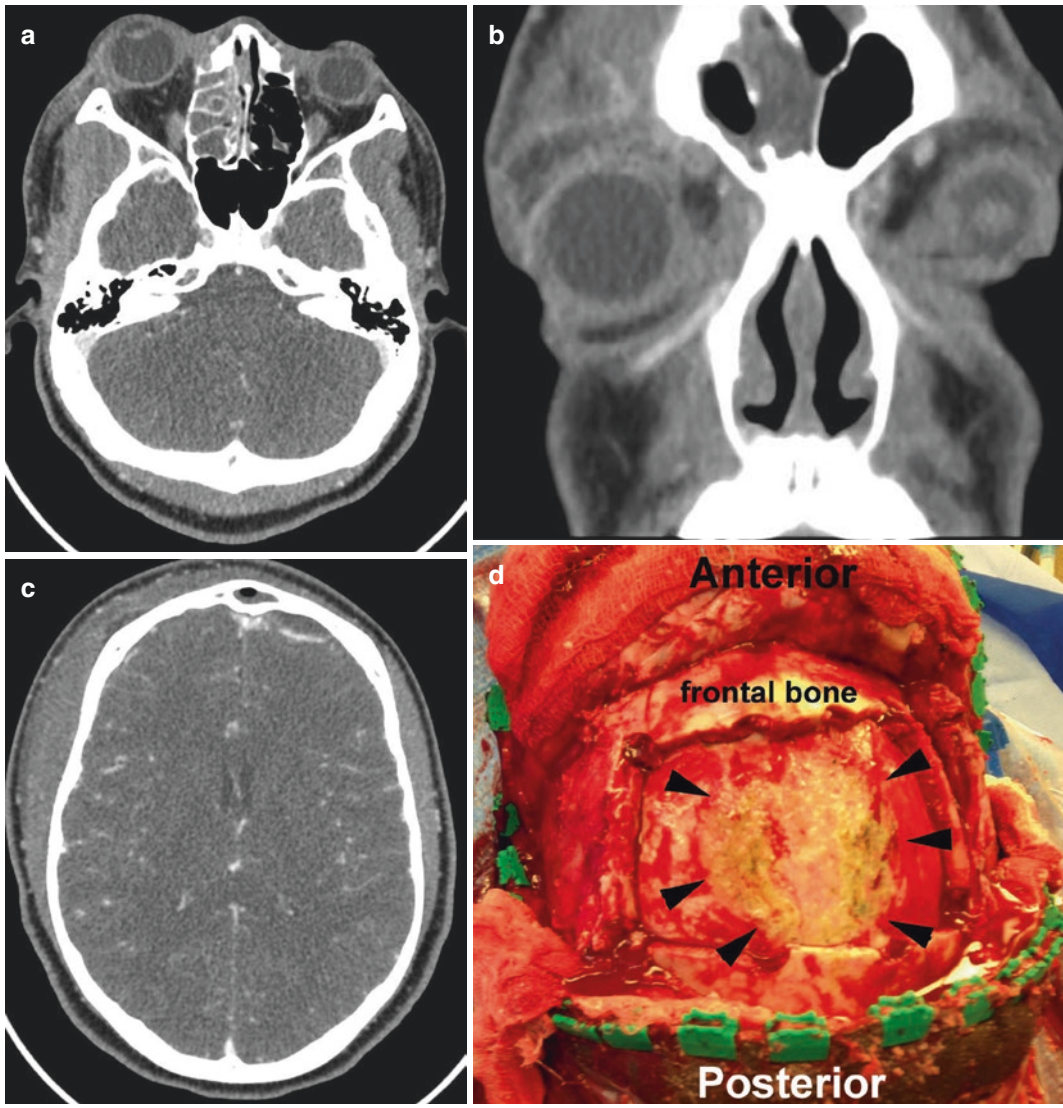


Fig. 5.9 *Case 6*: (**a**–**g**) 15-year-old M with severe ethmoid sinusitis complicated orbital and epidural abscess requiring FESS, craniotomy. 1 year after initial treatment patient developed bilateral pansinusitis and sepsis. He underwent repeat functional endoscopic sinus surgery. Antibiotic therapy included Unasyn, Vancomycin, Flagyl. (**a**) Axial view of CT scan showing right ethmoid sinusitis and proptosis of right eye. (**b**) Coronal view of CT scan showing right frontal sinusitis and right orbital abscess. (**c**) Left side frontal epidural space empyema due to extension of infection from bilateral frontal and ethmoid sinuses. (**d**) Craniotomy and drainage of epidural abscess. (**e**) Large intracranial mesh implant after craniotomy. (**f**, **g**) Axial and coronal views of CT scan showing bilateral pansinusitis and sepsis 1 year after initial treatment and was taken for repeat functional endoscopic sinus surgery

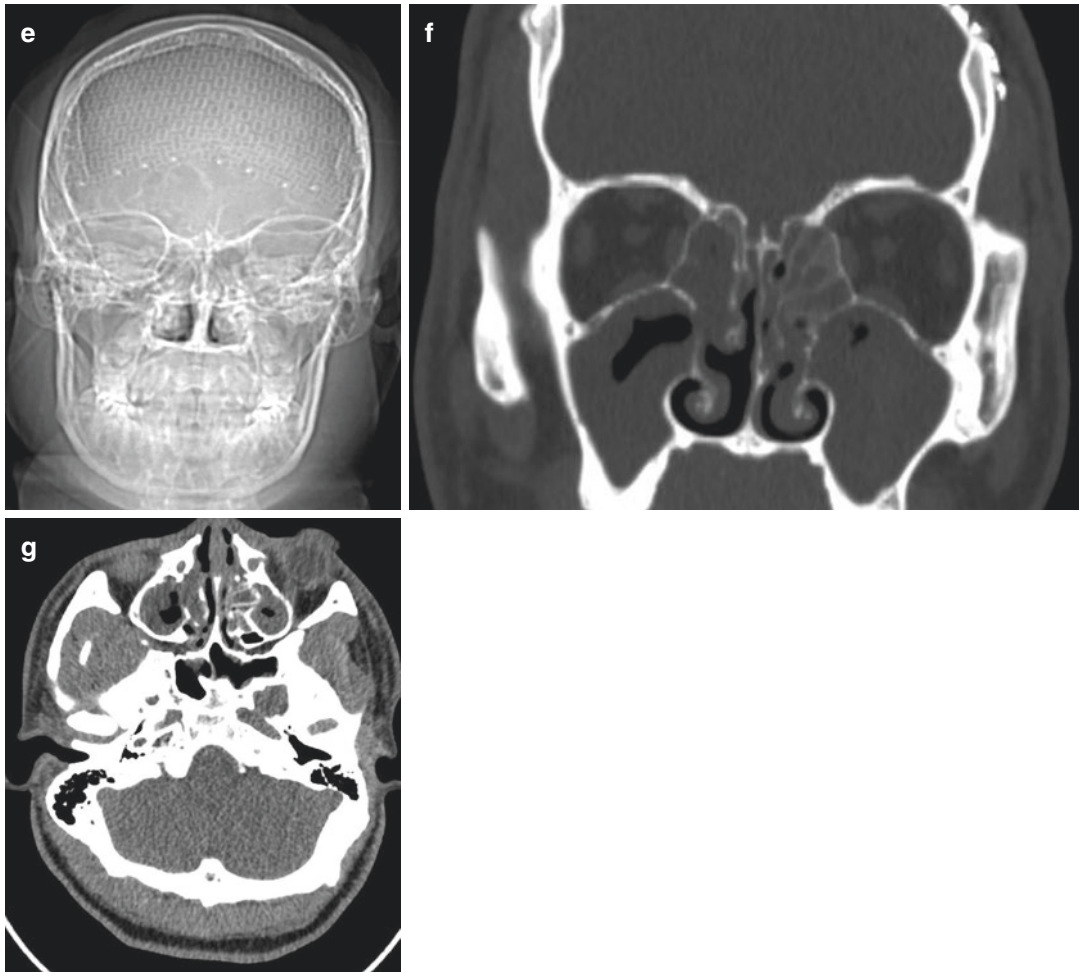


Fig. 5.9 (continued)

pression [29]. The data on anticoagulation therapy show improved morbidity and mortality. Anticoagulant therapy if initiated early, i.e., within 7 days of diagnosis, may reduce morbidity. The duration of anticoagulation is variable and can range between 2 and 6 weeks and up to 3 months in a few cases [29]. More patients with anticoagulation made full recovery (54% vs. 32%), and fewer (12% vs. 28%) patients died in comparison to those who were not anticoagulated [112]. The role of steroids is still unclear. The benefits of decreasing edema of the involved cranial nerves or periorbital swelling must be weighed against the risks of immunosuppressive effects and the probability of hyperglycemia and elevated serum lactate levels for each patient [115].

Up until 1960, the reported mortality (80%) and morbidity (75%) were very high. Then Southwick et al. reviewed the literature between 1940 and 1984, and reported mortality around 30%. Although there has been significant improvement in the mortality and morbidity of CST over the years, the overall prognosis of CST is not good. Among cases treated between 1980 and 2015, the reported mortality and morbidity were lower at 11% and 15%, respectively [116]. Early diagnosis with better imaging techniques and appropriate management with administration of broad-spectrum antibiotics has reduced the mortality and morbidity of patients diagnosed with CST in the last three decades [117]. At present, mortality ranges between 11% and 13%, and death can

occur within 4–7 days, if diagnosis or treatment is delayed [117]. The long-term morbidity from CST still remains high. More than half of the survivors have residual neurological deficits in the form of cranial nerve deficits. Dysfunction of CN III and VI resulting in limitation of extraocular movements is the most common long-term sequela. Other sequelae include unilateral blindness, focal seizures, hemiparesis, pituitary insufficiency, and syndrome of inappropriate antidiuretic hormone secretion (SIADH). Metastatic infection from septic emboli can cause pulmonary complications such as pulmonary embolism, pneumonia, and thoracic empyema [29, 112].

Sepsis and septic shock: Sepsis and septic shock have been reported in severe odontogenic as well as non-odontogenic infections as a complication of bacterial endocarditis, descending necrotizing mediastinitis (DNM), Ludwig's angina, cervical necrotizing fasciitis, internal jugular vein thrombosis (IJVT), or cavernous sinus thrombosis (CST) [31, 32, 118, 119].

Sepsis is defined as the presence of (probable or documented) infection together with systemic manifestations of infection such as tachycardia, pyrexia, neutrophilia, tachypnea, and hypotension. Sepsis is a systemic, deleterious host response to infection that can lead to severe sepsis. *Severe sepsis* is defined as acute organ dysfunction or tissue hypoperfusion in the presence of infection. According to the third international consensus meeting the term "*septic shock"* is defined as a subset of sepsis in which profound circulatory, cellular, and metabolic abnormalities are associated with a greater risk of mortality than with sepsis alone.

Organ dysfunction can be documented by an increase in the sequential [sepsis-related] organ failure assessment (SOFA). The clinical criteria that together constitute the new clinical score termed quick SOFA (qSOFA) are: *respiratory rate of 22/min or greater, altered mentation, or systolic blood pressure of 100 mmHg or less*. A score of 2 points or more is associated with an in-hospital mortality greater than 10%. Adult patients with suspected infection are likely to have poor outcomes typical of sepsis, if they have at least two of the above (qSOFA) criteria.

Patients with septic shock can be clinically identified by a vasopressor requirement to maintain a mean arterial pressure of 65 mmHg or greater and serum lactate level greater than 2 mmol/L (>18 mg/dL) in the absence of hypovolemia. This combination is associated with hospital mortality rates greater than 40% [120].

The principles of treatment of sepsis are: (1) initial resuscitation with fluids to maintain urine output greater than or equal to 0.5 mL/kg/h, and MAP greater than or equal to 65 mmHg, and normalize blood lactate concentration if elevated; (2) early diagnosis with blood cultures for aerobic and anaerobic bacteria without delaying antimicrobial therapy for more than 45 min; (3) administration of antimicrobial therapy; (4) source control or eradication of the infectious focus; (5) adequate goal-directed nutrition; and (6) infection prevention to reduce the incidence of ventilator assisted pneumonia. Treatment should focus on monitoring and preserving end-organ function, i.e., renal function, respiratory function, nutritional status, and mentation.

Pulmonary complications such as *pulmonary embolism (PE), pleural empyema, pneumonia and ARDS* [121] have been documented as complications of odontogenic infections. Usually, these complications are sequelae of sepsis, prolonged intubation, mediastinitis, or internal jugular venous thrombosis. In severe sepsis, the body fails to contain the infection which can result in release of toxins and cause diffuse damage to the alveolar capillary walls, increased permeability, and subsequent interstitial and intra-alveolar edema which can impede gaseous exchange and eventually cause respiratory failure.

Osteomyelitis (*Case 7*, Fig. 5.10a–e). This is an inflammatory disorder of the cortical bone, bone marrow, and surrounding soft tissues resulting in destruction of bone and caused by infecting bacterial and/or fungal microorganisms. Osteomyelitis of the jaws can occur as a complication of chronic odontogenic infection *by direct extension of infection* into the bone from carious or periodontally involved teeth, after a traumatic injury or surgical procedures such as tooth extraction or endosseous implant placement [122, 123]. Osteomyelitis can also be caused *by hematoge-*

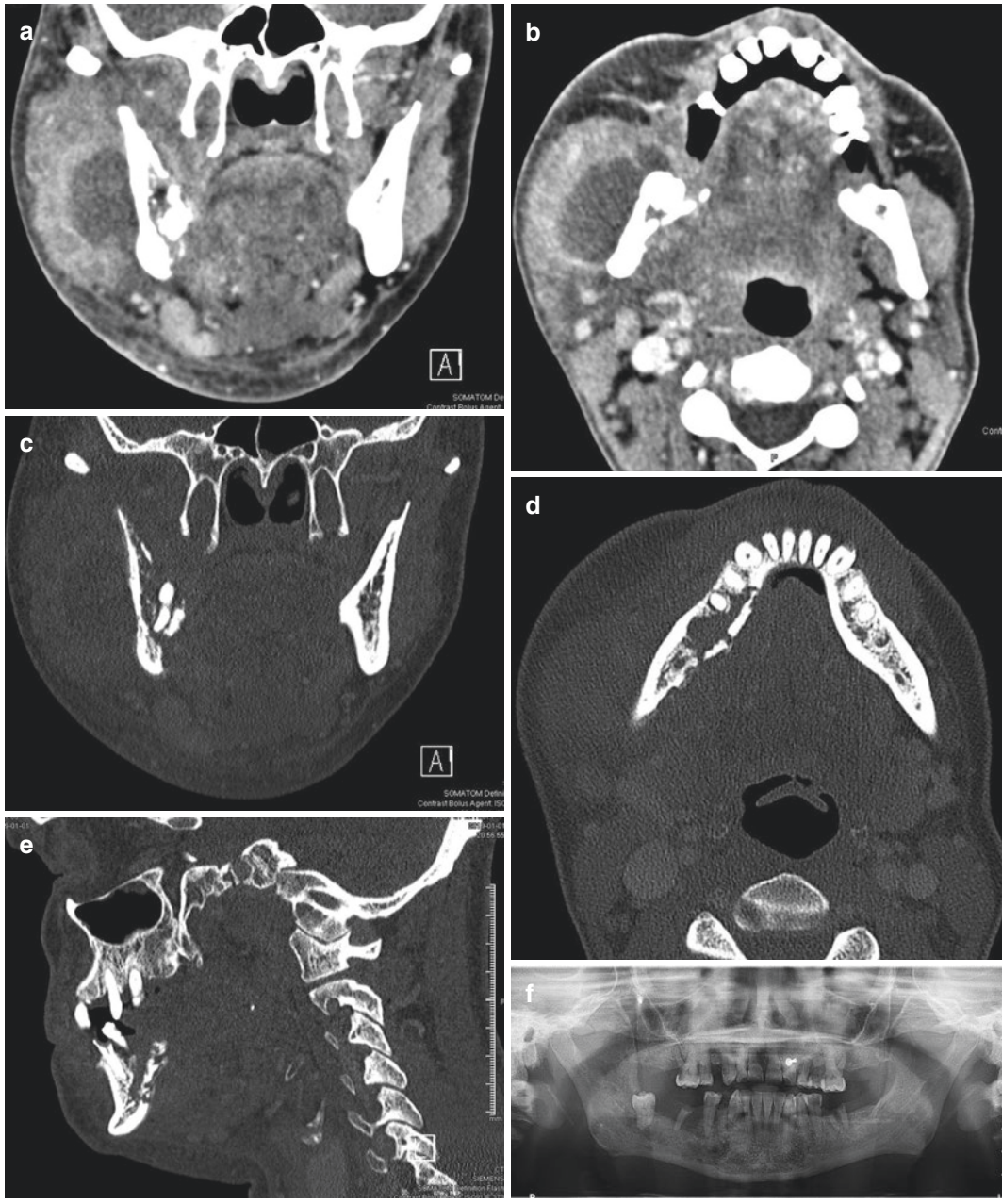


Fig. 5.10 *Case 7*: (**a**–**f**) 37 year old female with a history significant for IVDU and Hepatitis C presented with right-sided facial swelling and dental pain of 2 weeks duration. Patient did not seek any medical/dental help and only used antibiotics that was originally prescribed for her right arm cellulitis. (**a**, **b**) Right mandibular body osteomyelitis with sub-masseteric space abscess. (**c**) Right mandibular body acute osteomyelitis. (**d**) Right body acute osteomyelitis. (**e**) Right body osteomyelitis in sagittal cut. (**f**) Panoramic radiograph of right body osteomyelitis

nous spread of distant infection into the jaw bone, e.g., acute suppurative osteomyelitis in children. Osteomyelitis can occur *due to vascular insufficiency or marrow fibrosis* as in patients who have diabetes, treatment with certain antiresorptive and antiangiogenic medications, or radiation therapy [124].

Etiologic risk factors: Patients who develop osteomyelitis as a complication of odontogenic or non-odontogenic infections often have some local or systemic risk factors. Among the local risk factors that enable the bacterial extension from the oral cavity into the alveolar bone are: *inadequate mucosal barrier, poor vascularity of the overlying periosteal and mucosal tissues, venous stasis, neurosensory deficit, and radiation-induced fibrosis with the presence of exposed bone or implant surface.* Occasionally, this complication may arise due to an iatrogenic cause after a prolonged oral surgical procedure such as traumatic tooth extraction, or placement of endosseous implants. These risk factors facilitate seeding and colonization of bacteria in the bone. S*ystemic comorbidities such as diabetes, sickle cell disease, chronic kidney disease, autoimmune disorders, malnourishment tobacco smoking, alcohol and IV drug use, and immunosuppression after organ transplant put patients* at greater risk of developing this complication. Patients with bone disorders such as osteopetrosis and Paget's disease of bone are also at higher risk for developing osteomyelitis from dental infections [122, 125]. In the above patients, administration of appropriate perioperative antibiotics, use of proper atraumatic surgical technique with primary wound closure, and close follow-up are recommended to prevent or avoid this complication.

Osteomyelitis is more common in the mandible than in the maxilla. The posterior mandible is the most common location, but it can occur in any part of the mandible or maxilla and other facial bones (temporal/mastoid) [126]. Clinical presentation of osteomyelitis can vary based on the duration of infection and extent of disease. Patients may present with pain, swelling of jaw, erythema of skin, paresthesia, and in later stages with fracture and orocutaneous fistula. Osteomyelitis in adults has been classified based on the progression of disease into acute or chronic, and based on the extent of disease into four stages [127]:

- Stage 1—Medullary osteomyelitis medullary bone involved without cortical involvement.
- Stage 2—Superficial osteomyelitis (less than 2 cm cortical bony defect without medullary involvement).
- Stage 3—Localized osteomyelitis (less than 2 cm cortical bony defect without the involvement of both bony cortices).
- Stage 4—Diffuse osteomyelitis (greater than 2 cm cortical defect with both cortices involved and pathologic fracture, nonunion).

Diagnosis of osteomyelitis is usually established by correlating clinical findings with radiographic imaging studies and microbiological test results. The main imaging tools for diagnosis of osteomyelitis are MRI and CT scan. Often is it necessary to obtain both MRI and CT scan to assess the soft tissue and marrow changes as well as the extent of the cortical bone destruction and to plan surgical treatment. The classic radiographic signs include the presence of irregular bone destruction with the moth-eaten appearance and bony sequestra. MRI with fat-suppressed T2-weighted images is useful to detect early changes of bone marrow edema and periosteal reaction and muscle inflammation [128]. Contrast-enhanced T1-weighted images are helpful in determining the heterogeneity of bone marrow. The benefits of early detection and the ability to monitor progression or response to treatment with no radiation make MRI a very valuable imaging tool. MRI has a high sensitivity of 85% and moderate specificity of 60%, respectively, for the diagnosis of osteomyelitis. Surveillance may be better with a nuclear medicine imaging study such as TC ^{m99} bone scan rather than an MRI, as postsurgical changes can make interpretation difficult up to 6–8 months after surgery, due to the inability to differentiate between postsurgical edema and infection [129]. Plain films such as a panoramic radiograph can play a role in evaluating late changes of osteomyelitis such as sequestrum formation, fractures, and help in identifying the

odontogenic source of infection (*Case 8*, Fig. 5.11a–d).

These changes in bone do not become apparent on a plain film until there is at least 30% reduction in bone mineral density. Therefore, plain films have a limited role in the early diagnosis. Additionally, it is hard to evaluate the periosteal tissue changes and marrow changes. Reported sensitivity and specificity of plain films are 14% and 70%, respectively [130].

Pathogens: Unlike osteomyelitis of the long bones where *Staphylococcus aureus* is the main

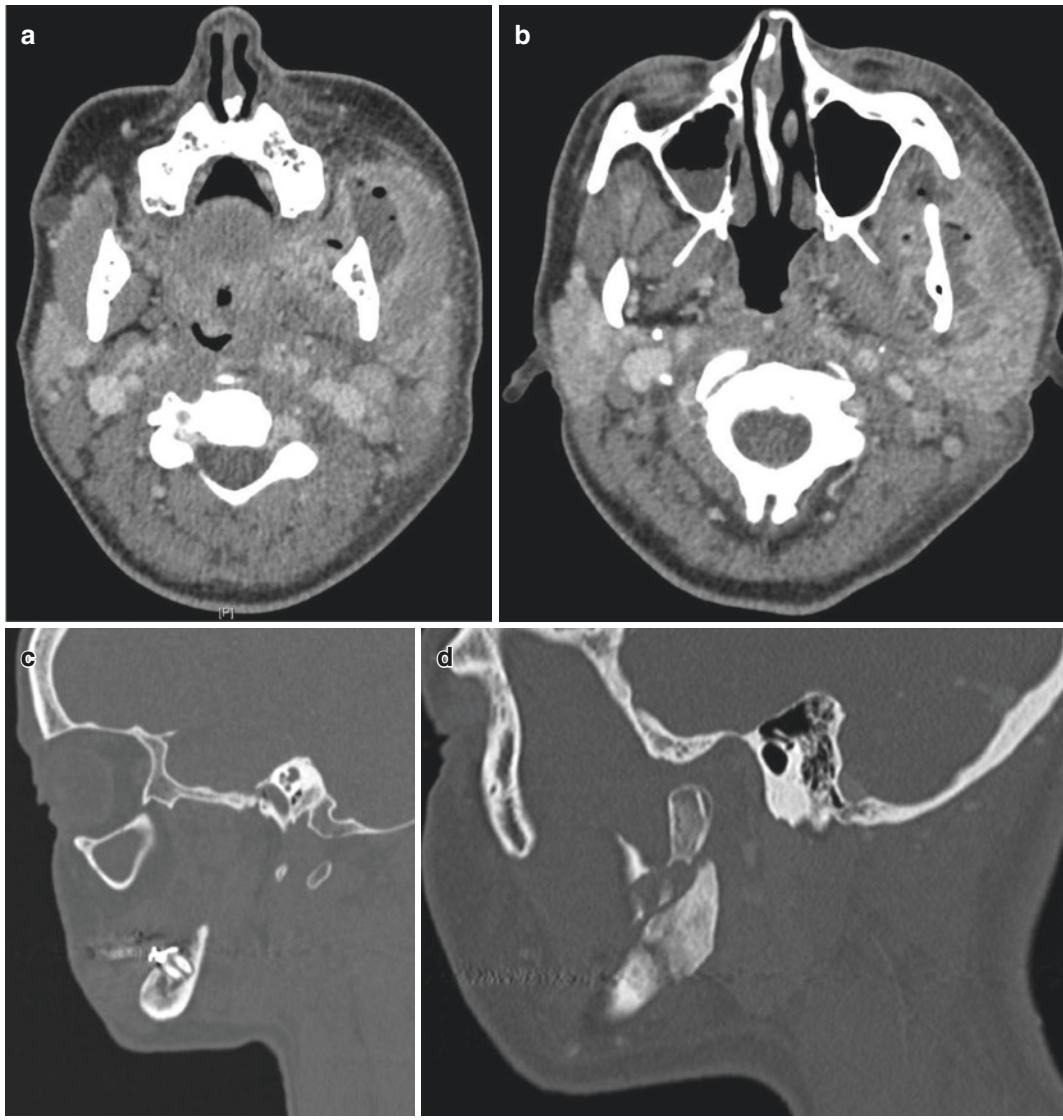


Fig. 5.11 *Case 8*: (**a**–**d**) 28 y.o. woman with a medical history significant for IV drug abuse and hepatitis C initially presented with left masticator space infection requiring intraoral and transcervical drainage and extraction of #19 in the OR. She returned to the ED 2 months later, now with chronic osteomyelitis and pathologic fracture of the left mandible requiring washout, debridement, and closed reduction. Cultures were positive for *Parvimonas micra*, Actinomyces *Serratia marcescens*. She was lost to follow up with only partial treatment course of IV unasyn as incomplete treatment course of 3 days with meropenem. (**a**) Axial cut left submasseteric space abscess secondary to osteomyelitis. (**b**) Same patient more superior axial cut left submasseteric space abscess secondary to osteomylitis. (**c**) Infected tooth #19 that resulted in osteomyelitis and ultimately a pathological fracture. (**d**) 5 months after sub-masseteric abscess and incomplete follow up to treatment patient presented with osteomyelitis

offending pathogen, 90–95% of osteomyelitis of the jaws is due to polymicrobial infection involving one to four organisms. Better culturing techniques in recent years have helped us to isolate the organisms; however, the treatment can be challenging due to the growing number of antibiotic-resistant bacteria. In order to obtain the best culture results, the surgeon should send bone and soft tissue samples rather than fluid/purulent discharge for aerobic, anaerobic, mycobacterial, and fungal cultures. Tissue samples yield higher bacterial counts than swabs of the discharge. The specimens should be delivered within 10–15 min of obtaining them in ideal temperatures (37°) to improve the chances of culturing anaerobic bacteria. Specimens for direct microscopic examination will reveal the growth of organisms on the surface of the bone, and histopathology can help to confirm the diagnosis.

In a recent study by Haeffs et al. [123], the bacteria cultivated from patient samples with suppurative osteomyelitis revealed that α-hemolytic *Streptococcus* of the *Streptococcus milleri group* was the most common bacterial isolate. Other common microorganisms included coagulase-negative *Staphylococcus* species, *Propionibacterium acnes*, and *Actinomyces* species. In their study, the average number of species identified for each patient was 1.81±0.917 [123]. Reports from other studies also showed similar organisms in chronic suppurative osteomyelitis as well as other organisms—*Fusobacterium*, *Parvimonas*, *Staphylococcus*, and *Eikenella corrodens*. These results suggest that mixed aerobic and anaerobic organisms cause chronic suppurative osteomyelitis of the jaws, reinforcing the concept that oral microorganisms and oral environment/periodontal health are risk factors for osteomyelitis of the jaws [131].

Management of osteomyelitis: Surgical debridement of the bone along with the removal of the source of infection, with simultaneous intravenous antibiotic therapy, is the main treatment modalities. Monitoring treatment outcome with close clinical follow-up, imaging, and systemic inflammatory markers such as WBC, CRP, and ESR is essential to ensure adequate resolution of the infection. Surgical treatment consists of thorough debridement of medullary and cortical bone by sequestrectomy, decortication and removal of nonviable bone, and excision of orocutaneous fistula [132]. Adequate bone and soft tissue samples must be obtained for culture and direct microscopic and pathologic examination. When indicated, stabilization of the bony segments with maxilla-mandibular fixation or rigid fixation may be necessary to prevent reentry of microorganisms from the oral cavity into bone. The surgeon should decide on the timing of fixation by weighing the risks of introducing a foreign body that can form a biofilm, versus the need for rigid fixation to establish stability [133, 134]. Treatment of the bone and surrounding soft tissue with direct placement of antibiotic-impregnated polymethylmethacrylate beads (Gentamicin and clindamycin beads) has also been described [130]. Hyperbaric oxygen can be considered as an adjunctive treatment in select cases and in refractory osteomyelitis given the benefits of free oxygen radicals and neo-angiogenesis to improve the oxygen tension in poorly vascularized tissue beds [135]. The average duration of intravenous antibiotics for mandibular osteomyelitis is a period of 6 weeks (range 2–8 weeks). Intravenous antimicrobial therapy may be followed by oral antimicrobial therapy for longer periods (3–4 months) in some patients. In general, Penicillin-based antibiotics such as imipenem, ertapenem, ampicillin with sulbactam or clavulanic acid are commonly used. Second- or third-generation cephalosporins and vancomycin may also be used for the management of chronic suppurative osteomyelitis. A fluoroquinolone such as moxifloxacin has broad-spectrum activity against Gram-positive and Gram-negative organisms and anaerobes. It has good bioavailability even with oral administration [130].

Microbiology and antibiotic therapy for head and neck infections: Most of the head and neck infections are polymicrobial, i.e., mixed aerobic and anaerobic bacteria. The viridans group of streptococci (VGS) are a group of Gram-positive cocci and normal oral commensals, which are responsible for most of the severe head and neck infections. This comprises *Strep. anginosus* (*Strep. milleri*), *Strep. mitis*, *Strep. mutans*, *Strep. sanguinis*, and *Strep. salivarius*. The organisms cultured are native to the ana-

tomic regions where they inhabit, namely, the oropharynx, nasopharynx, upper respiratory tract, ears eyes, and skin. There are differences in the microbiology of infections of the midface/upper face compared to the deep neck space infections, and there are differences between odontogenic and non-odontogenic infections. The organisms also vary based on the host immunity and the geographic region of the population.

The upper respiratory tract and the oral cavity together have several normal commensals including coagulase-negative *Staphylococci*, nonhemolytic and viridans streptococci, *Corynebacterium* spp., micrococci, saprophytic *Neisseria* spp., *Haemophilus* spp., and a wide range of anaerobes including *Propionibacterium*, *Lactobacillus*, *Peptostreptococcus*, and *Veillonella*, which can cause infections in an immunosuppressed host. Other pathogenic organisms commonly found at these sites and responsible for the majority of head and neck infections include *Staphylococcus aureus*, *Streptococcus pneumoniae*, *beta-hemolytic streptococci*, *Neisseria meningitidis*, *Haemophilus influenzae*, *H. parainfluenza*, *Moraxella catarrhalis*, and *Eikenella*, *Fusobacterium*, *Bacteroides*, *Prevotella*, *Porphyromonas*, and *Actinomyces* spp. [109] (Table 5.5).

In general, empiric antibiotic therapy covering mixed aerobic and anaerobic bacteria is effective in the majority of patients with odontogenic and non-odontogenic infections. Once the specific

Table 5.5 Microbiology of head and neck infections and associated complications

Infections	Common bacterial strains (aerobic)	Common bacterial strains (anaerobic)
Odontogenic DSNI [139, 140]	*Streptococcus viridans* *Staphylococci* spp. Enterobacteria	*Prevotella melaninogenica* *Bacteroides* spp. *Peptostreptococcus* spp. *Eikenella corrodens* *Clostridium* spp. *Parvimonas micra*
Non-odontogenic DNSI Acute tonsillopharyngitis/quinsy	*Streptococcus pyogenes* (group A *Streptococcus* [GAS]) *Staphylococcus aureus* (including methicillin-resistant *S. aureus* [MRSA])	*Fusobacteria* spp. *Prevotella* spp. *Veillonella* species
Acute sinusitis	*Streptococcus pneumoniae* *M. catarrhalis*	*H. influenzae* *Prevotella* *Porphyromonas* *Fusobacterium* *Peptostreptococcus*
Lemierre's syndrome (IJVT)	Streptococci group B, C, *Streptococcus oralis*, *Staphylococci* spp.	*Fusobacterium necrophorum* *Fusobacterium nucleatum* *Prevotella* *Peptostreptococcus*
Cervical necrotizing fasciitis (CNF)	Alfa-hemolytic, *Streptococcus anginosus group* (*Streptococcus milleri*) Beta-hemolytic *Streptococcus* (Group A) *Staphylococcus aureus*	*Prevotella* *Prevotella* spp. *Bacteroides fragilis* *Peptostreptococcus*
Cavernous sinus thrombosis (CST)	*Staphylococcus aureus* *Streptococcus* spp. *Pseudomonas aeruginosa*	*Peptostreptococcus* *Fusobacterium* spp.
Brain abscess [141, 142]	*Staphylococcus aureus* *Streptococci microaerophilic* *Pseudomonas aeruginosa* *E. coli*	*Prevotella* spp. *Bacteroides* spp. *Peptostreptococcus* *Fusobacterium* spp.
Osteomyelitis	*Staphylococcus aureus* Viridans streptococci Milleri group streptococci	Actinomyces spp. *Parvimonas micra* *Propionibacterium* spp. *Prevotella* spp. *Fusobacterium* spp.

organisms have been identified, it is important to direct and target the organisms with the appropriate narrow-spectrum antibiotic of choice. Some anaerobic organisms are difficult to culture; therefore, care should be taken to include anaerobic coverage even though these organisms are not evident in the results. Patients with history of diabetes, chronic renal insufficiency, skin ulcers or wounds, and intravenous drug use are at a higher risk for developing methicillin-resistant *Staphylococcus aureus* (MRSA) infection; therefore, the antibiotic coverage should include adequate coverage against MRSA. Other factors such as the source of infection, host immunity, response to treatment, previous exposure and resistance, geographic region, and allergies should be considered before prescribing antibiotics. When obtaining specimens for cultures, it is important to get tissue (soft or hard tissue) whenever possible. For necrotizing fasciitis, the edge of the necrotic tissues or bone for the osteomyelitis is better specimen for culture than a swab.

An important question is the length of treatment especially in patients with severe infections who have complications. In patients with complications of infections, particularly those involving vital organs, vascular structures or bone, or in patients with sepsis, the antibiotic therapy may be prolonged, ranging from 3 to 8 weeks with an average of 4 weeks. In patients with osteomyelitis, oral antibiotics for up to 3–4 months often follow the IV antibiotic therapy. Adequate nutrition and supportive therapy as well as proper simultaneous management of systemic comorbid disorders are essential for the successful treatment of infection. Surrogate markers of infection such as WBC, CRP, physiologic signs of sepsis such as fever, and clinical signs of erythema, pain, swelling, and drainage should be considered when deciding on the length of treatment.

The usual empiric antibiotics include ampicillin/sulbactam or penicillin and metronidazole, or ceftriaxone, or clindamycin [136]. When the bioavailability of the drug is good or almost equivalent to the intravenous route, the antibiotics can be administered orally, as in the case of levofloxacin, ciprofloxacin, and metronidazole. Farmahan et al. studied odontogenic infections and response antibiotic treatment. The most common bacteria isolated were streptococci spp. Seventy percent of the bacteria were sensitive to amoxicillin and 84% to amoxicillin and metronidazole, while 14% (mainly *Staphylococcus aureus* from infections of the skin) were resistant to penicillin. They compared their results with previous literature, which showed no major changes in the microbiology and antibiotic sensitivity of odontogenic infections in the head and neck in the past three decades [137]. Poeschl et al. [138] reported on bacterial sensitivity to empiric antibiotics used in odontogenic infections. In their study of 206 patients who underwent treatment for deep neck and facial infections, they reported high resistance of the tested strains for clindamycin (11%–18% of tested strains) and erythromycin (14% of the tested strains). The rates of resistance to penicillin (6–7%) and aminopenicillins were lower (0–3%) [138]. Flynn et al. [10] reported a higher failure to treatment response (21%) with penicillin in severe odontogenic infections. Resistance to penicillin is more common among anaerobes—*Prevotella*, *Bacteroides*, and *Fusobacterium*.

5.6 Prevention of Complications in the Management of Head and Neck Infections (Table 5.6)

The best way to reduce morbidity and mortality of severe odontogenic and non-odontogenic infections is to prevent the complications. This is somewhat difficult to control as the patients, the providers, and the healthcare delivery systems can influence the factors that contribute to complications. Barber et al. [143] attempted to determine factors that predict more severe deep neck space infections (DNSI). They found that patients with a low level of education, patients living greater than 1 h from a tertiary care center, and those with frequent *s*treptococcal infections of tonsils have an increased risk of developing a severe DNSI. Patients with diabetes, current smokers, and those that presented with airway

Table 5.6 Tips to prevent complications in patients with head and neck infections

Admission	Triage to appropriate specialists Recognize high-risk symptoms and signs of airway distress Document comorbid illness Document SIRS criteria (BP, HR, WBC with diff., temperature) Initiate empiric antibiotic therapy Initiate supportive therapy with fluids
Diagnosis	CT neck with contrast with extension to chest or brain, if indicated Identify all involved spaces Identify the source of infection Assess the risk of airway deviation and/ or constriction Assess the risk of mediastinal or intracranial extension of infection Document severity of infection based on the *number of spaces and proximity to vital structures (airway, great vessels, mediastinum and eyes, and brain)* Use a severity score (low, intermediate, and high)
Treatment	Anesthesiologist and surgeon communicate the plan to safely secure airway Empiric antibiotic therapy Surgical drainage of all involved spaces when fluid collection is present Remove or manage the source of infection Identify the need for intensive care monitoring after treatment Manage comorbid illness Evaluate cultures and response to antibiotic and surgical therapy Monitor clinical progress for improvement or deterioration

compromise were more likely to have a prolonged hospital stay [143].

Prevention requires a change in the healthcare delivery to improve access to dental and medical care and health literacy, as well as improvement in clinician's knowledge and ability to recognize patients at risk of developing life-threatening complications. *Emergency room physicians, primary care doctors, and triage nurses should be educated to recognize signs of deep neck space infection and an impending airway distress* in patients. House officers in oral and maxillofacial surgery and otolaryngology should be trained to recognize *high-risk patients* based on *medical history, clinical symptoms and signs, and findings on imaging that are predictors of life-threatening complications, e.g., signs of sepsis and airway distress* [9].

Patients with early signs of bilateral neck space involvement and signs of Ludwig's angina should have airway secured promptly. In some instances, evaluating and securing the airway prior to imaging and further diagnosis may be necessary to avoid airway obstruction. Documentation of physiologic parameters such as temperature, BP, HR, and inflammatory markers such as CRP, WBC with neutrophil count to monitor for signs of sepsis should be standard. If sepsis is suspected, it is important to obtain blood for cultures and inflammatory markers before the administration of antibiotics. Patients with signs of skin discoloration, severe pain and air/gas in the tissues with crepitus, rapid progression of symptoms, and multiple space involvement are at high risk for developing further complications [100]. In patients with severe deep neck space infections, one should also closely monitor changes in airway patency and swelling of oropharyngeal tissues, and neck pain [5, 10, 11]. In the case of upper face infections, clinicians should evaluate for vague symptoms that may indicate orbital or intracranial extension, for example, headache, nausea, vomiting, alteration in mental status, fever, malaise and eye pain, conjunctival redness, disturbances visual acuity, extraocular movements, etc. Adequate imaging with post-contrast-enhanced CT scan is important for early accurate diagnosis and surgical treatment. Empiric antibiotic therapy in conjunction with adequate surgical incision and drainage, if indicated, and removal of the source of infection should be planned upon admission.

Gallo et al. [144] in their recent prospective study proposed an algorithm to avoid life-threatening complications in patients with deep neck space infections [144]. They used a scoring system based on the risk of developing major complications. They classified patients into three risk groups: low risk (Score 0–5), intermediate risk (Score 6–10), and high risk (Score 11–15). The risk score was assigned based on the severity of airway obstruction, difficulty in swallowing, degree of mouth opening (trismus), number and type of neck spaces involved, and history of anti-

biotic use. Their algorithm allowed them to identify patients who could be treated as outpatient (low risk), and patients who required hospitalization (intermediate risk and high risk). Similar to previous studies, they noted that the presence of comorbid illness and involvement of multiple lateral deep neck spaces were the strongest predictors of complications [144].

Flynn et al. conducted a prospective study on severe odontogenic infections treated in four urban hospitals in New York City over a 3-year period between 1996 and 1999. They proposed a severity score for infections, which is helpful to analyze data and to standardize the documentation of clinical and radiographic assessment. They classified odontogenic infections as low (1), moderate (2), and severe (3) based on clinical and radiographic involvement of the spaces, and the risk to airway and other vital structures. Their infection severity score can be used along with other objective serum markers such as C-reactive protein (CRP), ESR, WBC (absolute neutrophil count), and temperature as predictors of complications and prognosis. The number of anatomic deep neck spaces involved and their relationship to vital structures are useful predictors of complications [10, 11].

Prevention of airway complications requires meticulous planning, communication, and prompt action by the surgical and anesthesia team members. In general, it is better to reserve the options for tracheostomy only for those select cases when the alternative plans for securing the airway fail. It is sometimes difficult to differentiate between cellulitis and abscess on the diagnostic imaging. When in doubt, consider admitting the patient for IV antibiotic therapy and monitoring, rather than discharge with a prescription of oral antibiotics. The need for surgical drainage can be predicted based on the progression of clinical symptoms, signs of sepsis, and serum markers for prognosis of infection [145]. Further imaging may be necessary if the clinical condition of the patient does not improve with the removal of source and antibiotic therapy.

Repeat imaging to assess for new or residual collections, and repeat surgical intervention for debridement of necrotic tissue and wash out fascial spaces. In the event of sepsis or other complications, interdisciplinary care and intensive care monitoring are often necessary.

With the advent of electronic records, we can use the subjective and objective data to improve our prediction of complications and identify high-risk, moderate-risk, and low-risk patients using artificial intelligence. By documenting a risk profile for every patient admitted with head and neck infection, we can develop intelligent algorithms to treat high-risk patients (airway obstruction, mediastinal or intracranial extension of infection).

5.7 Conclusion

The morbidity and mortality of complications of odontogenic and non-odontogenic infections are relatively high despite advances in diagnostic imaging, airway management, antibiotic therapy, surgical treatment, and intensive coordinated care. The current literature and our experience indicate that complications occur most often in patients with systemic comorbidities such as immunosuppressive disorders, poor nutritional status, current smokers, alcoholism, and psychiatric disorders. Other predictors of complications are extremes of age, low socioeconomic status, and limited access to healthcare. Delay in diagnosis and treatment, improper airway management, failure to identify or remove the source of infection, and failure of response to surgical and antibiotic therapy also contribute to complications.

Although little has changed in the microbiology of odontogenic and non-odontogenic infections in the head and neck regions, the growing number of antibiotic-resistant bacteria will continue to be a challenge.

When managing patients with severe head and neck infections, we can ***prevent complications*** by:

1. Identifying high-risk patients: demographic variables—age, socioeconomic status, systemic comorbid illness, and anatomical spaces involved clinical symptoms and signs (trismus, dysphagia) that are predictors of life-threatening complications.

2. Documenting and monitoring important physiologic parameters (mental status, systolic BP, HR, RR, WBC, temperature) of sepsis for early signs of septic shock.
3. Early imaging using post-contrast-enhanced computed tomography, extending to chest and brain when mediastinal or intracranial extension, respectively, is suspected.
4. Educating emergency room house staff, colleagues, and triage nurses to recognize an impending airway distress in patients with deep neck infections.
5. Prevention of airway complications requires meticulous planning, communication, and prompt action by the surgical and anesthesia team members.
6. In general, it is better to reserve the options for tracheostomy only for those select cases when the alternative plans for securing the airway fail.
7. A careful review of the number and location of the anatomical spaces involved, as well as the boundaries of the fascial spaces involved.
8. Identifying and removing the source of infection rendering timely and adequate surgical treatment, keeping in mind that almost 60% of the infections are odontogenic in origin.
9. Being mindful of antibiotic-resistant bacteria and choosing the appropriate broad-spectrum antibiotics for mixed aerobic and anaerobic bacteria.
10. Providing adequate supportive therapy and close monitoring with interdisciplinary and intensive care when necessary.

References

1. Daramola OO, Flanagan CE, Maisel RH, Odland RM. Diagnosis and treatment of deep neck space abscesses. Otolaryngol Head Neck Surg. 2009;141(1):123–30.
2. Mathew GC, Ranganathan LK, Gandhi S, Jacob ME, Singh I, Solanki M, Bither S. Odontogenic maxillofacial space infections at a tertiary care center in North India: a five-year retrospective study. Int J Infect Dis. 2012;16(4):e296–302.
3. Huang TT, Liu TC, Chen PR, Tseng FY, Yeh TH, Chen YS. Deep neck infection: analysis of 185 cases. Head Neck. 2004;26(10):854–60.
4. Bridgeman A, Wiesenfeld D, Hellyar A, Sheldon W. Major maxillofacial infections. An evaluation of 107 cases. Aust Dent J. 1995;40(5):281–8. PMID:8629955.
5. Byers J, Lowe T, Goodall CA. Acute cervico-facial infection in Scotland 2010: patterns of presentation, patient demographics and recording of systemic involvement. Br J Oral Maxillofac Surg. 2012;50(7):626–30.
6. Bross-Soriano D, Arrieta-Gómez JR, Prado-Calleros H, Schimelmitz-Idi J, Jorba-Basave S. Management of ludwig's angina with small neck incisions: 18 years experience. Otolaryngol Head Neck Surg. 2004;130(6):712–7.
7. Ogle OE. Odontogenic infections. Dent Clin N Am. 2017;61(2):235–52.
8. Kewani et al. J Maxillofac Oral Surg. 2019;18(3):345–353. https://doi.org/10.1007/s12663-018-1152-x.
9. Alotaibi N, Cloutier L, Khaldoun E, Bois E, Chirat M, Salvan D. Criteria for admission of odontogenic infections at high risk of deep neck space infection. Eur Ann Otorhinolaryngol Head Neck Dis. 2015;132(5):261–4.
10. Flynn TR, Shanti RM, Hayes C. Severe odontogenic infections, part 2: prospective outcomes study. J Oral Maxillofac Surg. 2006a;64(7):1104–13.
11. Flynn TR, Shanti RM, Levi MH, Adamo AK, Kraut RA, Trieger N. Severe odontogenic infections, part 1: prospective report. J Oral Maxillofac Surg. 2006b;64(7):1093–103.
12. Gujrathi AB, Ambulgekar V, Kathait P. Deep neck space infection—a retrospective study of 270 cases at tertiary care center. World J Otorhinolaryngol Head Neck Surg. 2016;2(4):208–13.
13. Kataria G, Saxena A, Bhagat S, Singh B, Kaur M, Kaur G. Deep neck space infections: a study of 76 cases. Iran J Otorhinolaryngol. 2015;27(81):293–9.
14. Ridder GJ, Technau-Ihling K, Sander A, Boedeker CC. Spectrum and management of deep neck space infections: an 8-year experience of 234 cases. Otolaryngol Head Neck Surg. 2005;133(5):709–14.
15. Dodson TB, Barton JA, Kaban LB. Predictors of outcome in children hospitalized with maxillofacial infections: a linear logistic model. J Oral Maxillofac Surg. 1991;49(8):838–42.
16. Wang J, Ahani A, Pogrel MA. A five-year retrospective study of odontogenic maxillofacial infections in a large urban public hospital. Int J Oral Maxillofac Surg. 2005;34(6):646–9.
17. Biederman GR, Dodson TB. Epidemiologic review of facial infections in hospitalized pediatric patients. J Oral Maxillofac Surg. 1994;52(10):1042–5.
18. Lawrence R, Bateman N. Controversies in the management of deep neck space infection in children: an evidence-based review. Clin Otolaryngol. 2017;42(1):156–63.

19. Cheng J, Elden L. Children with deep space neck infections: our experience with 178 children. Otolaryngol Head Neck Surg. 2013;148(6):1037–42.
20. Baqain ZH, Newman L, Hyde N. How serious are oral infections? J Laryngol Otol. 2004;118(7):561–5.
21. Rubin MM, Cozzi GM. Fatal necrotizing mediastinitis as a complication of an odontogenic infection. J Oral Maxillofac Surg. 1987;45(6):529–33.
22. Cho SY, Woo JH, Kim YJ, Chun EH, Han JI, Kim DY, Baik HJ, Chung RK. Airway management in patients with deep neck infections: a retrospective analysis. Medicine (Baltimore). 2016;95(27):e4125.
23. Qu L, Liang X, Jiang B, Qian W, Zhang W, Cai X. Risk factors affecting the prognosis of descending necrotizing mediastinitis from odontogenic infection. J Oral Maxillofac Surg. 2018;76(6):1207–15.
24. Ord R, Coletti D. Cervico-facial necrotizing fasciitis. Oral Dis. 2009;15(2):133–41.
25. Osborn TM, Assael LA, Bell RB. Deep space neck infection: principles of surgical management. Oral Maxillofac Surg Clin North Am. 2008;20(3):353–65.
26. Lalwani AK, Kaplan MJ. Mediastinal and thoracic complications of necrotizing fasciitis of the head and neck. Head Neck. 1991;13(6):531–9.
27. Boyd D, Paterson P, Dunphy L, Carton A, Hammersley N. A case report of Lemierre's syndrome associated with dental sepsis. Scott Med J. 2013;58(1):e24–7.
28. Laupland KB. Vascular and parameningeal infections of the head and neck. Infect Dis Clin North Am. 2007;21(2):577–90, viii.
29. Desa V, Green R. Cavernous sinus thrombosis: current therapy. J Oral Maxillofac Surg. 2012;70(9):2085–91.
30. Martines F, Salvago P, Ferrara S, Mucia M, Gambino A, Sireci F. Parietal subdural empyema as complication of acute odontogenic sinusitis: a case report. J Med Case Reports. 2014;8:282.
31. Carter L, Lowis E. Death from overwhelming odontogenic sepsis: a case report. Br Dent J. 2007;203:241.
32. Pota V, Passavanti MB, Sansone P, Pace MC, Peluso F, Fiorelli A, Aurilio C. Septic shock from descending necrotizing mediastinitis - combined treatment with IgM-enriched immunoglobulin preparation and direct polymyxin B hemoperfusion: a case report. J Med Case Reports. 2018;12(1):55.
33. Dugan MJ, Lazow SK, Berger JR. Thoracic empyema resulting from direct extension of Ludwig's angina: a case report. J Oral Maxillofac Surg. 1998;56(8):968–71.
34. Green AW, Flower EA, New NE. Mortality associated with odontogenic infection! Br Dent J. 2001;190(10):529–30.
35. Bakir S, Tanriverdi MH, Gun R, Yorgancilar AE, Yildirim M, Tekbas G, Palanci Y, Meric K, Topcu I. Deep neck space infections: a retrospective review of 173 cases. Am J Otolaryngol. 2012;33(1):56–63.
36. Larawin V, Naipao J, Dubey SP. Head and neck space infections. Otolaryngol Head Neck Surg. 2006;135(6):889–93.
37. Bonapart IE, Stevens HP, Kerver AJ, Rietveld AP. Rare complications of an odontogenic abscess: mediastinitis, thoracic empyema and cardiac tamponade. J Oral Maxillofac Surg. 1995;53(5):610–3.
38. Boscolo-Rizzo P, Stellin M, Muzzi E, Mantovani M, Fuson R, Lupato V, Trabalzini F, Da Mosto MC. Deep neck infections: a study of 365 cases highlighting recommendations for management and treatment. Eur Arch Otorhinolaryngol. 2012;269(4):1241–9.
39. Bulut M, Balci V, Akkose S, Armagan E. Fatal descending necrotising mediastinitis. Emerg Med J. 2004;21(1):122–3.
40. Zeitoun IM, Dhanarajani PJ. Cervical cellulitis and mediastinitis caused by odontogenic infections: report of two cases and review of literature. J Oral Maxillofac Surg. 1995;53(2):203–8.
41. Bali RK, Sharma P, Gaba S, Kaur A, Ghanghas P. A review of complications of odontogenic infections. Natl J Maxillofac Surg. 2015;6(2):136–43.
42. Jundt JS, Gutta R. Characteristics and cost impact of severe odontogenic infections. Oral Surg Oral Med Oral Pathol Oral Radiol. 2012;114(5):558–66.
43. Opitz D, Camerer C, Camerer DM, Raguse JD, Menneking H, Hoffmeister B, Adolphs N. Incidence and management of severe odontogenic infections—a retrospective analysis from 2004 to 2011. J Craniomaxillofac Surg. 2015;43(2):285–9.
44. Uluibau IC, Jaunay T, Goss AN. Severe odontogenic infections. Aust Dent J. 2005;50(4 Suppl 2):S74–81.
45. Celakovsky P, Kalfert D, Tucek L, Mejzlik J, Kotulek M, Vrbacky A, Matousek P, Stanikova L, Hoskova T, Pasz A. Deep neck infections: risk factors for mediastinal extension. Eur Arch Otorhinolaryngol. 2014;271(6):1679–83.
46. Ferneini EM, Goldberg MH. Management of oral and maxillofacial infections. J Oral Maxillofac Surg. 2018;76(3):469–73.
47. Chen MK, Wen YS, Chang CC, Huang MT, Hsiao HC. Predisposing factors of life-threatening deep neck infection: logistic regression analysis of 214 cases. J Otolaryngol. 1998;27(3):141–4.
48. Wong TY. A nationwide survey of deaths from oral and maxillofacial infections: the Taiwanese experience. J Oral Maxillofac Surg. 1999;57(11):1297–9; discussion 1300.
49. Seppanen L, Lauhio A, Lindqvist C, Suuronen R, Rautemaa R. Analysis of systemic and local odontogenic infection complications requiring hospital care. J Infect. 2008;57(2):116–22.
50. Carter LM, Layton S. Cervicofacial infection of dental origin presenting to maxillofacial surgery units in the United Kingdom: a national audit. Br Dent J. 2009;206:73.
51. Adamson OO, Gbotolorun OM, Odeniyi O, Oduyebo OO, Adeyemo WL. Assessment of predictors of treatment outcome among patients with

bacterial odontogenic infection. Saudi Dent J. 2018;30(4):337–41.
52. Kityamuwesi R, Muwaz L, Kasangaki A, Kajumbula H, Rwenyonyi CM. Characteristics of pyogenic odontogenic infection in patients attending Mulago Hospital, Uganda: a cross-sectional study. BMC Microbiol. 2015;15:46.
53. Sittitrai P, Srivanitchapoom C, Reunmakkaew D. Deep neck infection in patients with and without human immunodeficiency virus: a comparison of clinical features, complications, and outcomes. Br J Oral Maxillofac Surg. 2018;56(10):962–7.
54. Chen MK, Wen YS, Chang CC, Lee HS, Huang MT, Hsiao HC. Deep neck infections in diabetic patients. Am J Otolaryngol. 2000;21(3):169–73.
55. Zheng L, Yang C, Zhang W, Cai X, Kim E, Jiang B, Wang B, Pu Y, Wang J, Zhang Z, Zhou L, Zhou J, Guan X. Is there association between severe multispace infections of the oral maxillofacial region and diabetes mellitus? J Oral Maxillofac Surg. 2012;70(7):1565–72.
56. Hidaka H, Yamaguchi T, Hasegawa J, Yano H, Kakuta R, Ozawa D, Nomura K, Katori Y. Clinical and bacteriological influence of diabetes mellitus on deep neck infection: systematic review and meta-analysis. Head Neck. 2015;37(10):1536–46.
57. Kamat RD, Dhupar V, Akkara F, Shetye O. A comparative analysis of odontogenic maxillofacial infections in diabetic and nondiabetic patients: an institutional study. J Korean Assoc Oral Maxillofac Surg. 2015;41(4):176–80.
58. Sakarya EU, Kulduk E, Gundogan O, Soy FK, Dundar R, Kilavuz AE, Ozbay C, Eren E, Imre A. Clinical features of deep neck infection: analysis of 77 patients. Kulak Burun Bogaz Ihtis Derg. 2015;25(2):102–8.
59. Caccamese JF Jr, Coletti DP. Deep neck infections: clinical considerations in aggressive disease. Oral Maxillofac Surg Clin North Am. 2008;20(3):367–80.
60. Hasegawa J, Hidaka H, Tateda M, Kudo T, Sagai S, Miyazaki M, Katagiri K, Nakanome A, Ishida E, Ozawa D, Kobayashi T. An analysis of clinical risk factors of deep neck infection. Auris Nasus Larynx. 2011;38(1):101–7.
61. Kuriyama T, Nakagawa K, Karasawa T, Saiki Y, Yamamoto E, Nakamura S. Past administration of beta-lactam antibiotics and increase in the emergence of beta-lactamase-producing bacteria in patients with orofacial odontogenic infections. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2000;89(2):186–92.
62. Yuan H, Gao R. Infrahyoid involvement may be a high-risk factor in the management of non-odontogenic deep neck infection: retrospective study. Am J Otolaryngol. 2018;39(4):373–7.
63. Lindner HH. The anatomy of the fasciae of the face and neck with particular reference to the spread and treatment of intraoral infections (Ludwig's) that have progressed into adjacent fascial spaces. Ann Surg. 1986;204(6):705–14.
64. Marchand P. The anatomy and applied anatomy of the mediastinal fascia. Thorax. 1951;6(4):359–68.
65. Yonetsu K, Izumi M, Nakamura T. Deep facial infections of odontogenic origin: CT assessment of pathways of space involvement. Am J Neuroradiol. 1998;19(1):123–8.
66. Pavlovich P, Looi A, Rootman J. Septic thrombosis of the cavernous sinus: two different mechanisms. Orbit. 2006;25(1):39–43.
67. Hedge A, Mohan S, Lim WE. Infections of the deep neck spaces. Singapore Med J. 2012;53(5):305–11; quiz 312.
68. Karkos PD, Leong SC, Beer H, Apostolidou MT, Panarese A. Challenging airways in deep neck space infections. Am J Otolaryngol. 2007;28(6):415–8.
69. Li RM, Kiemeney M. Infections of the neck. Emerg Med Clin North Am. 2019;37(1):95–107.
70. Candamourty R, Venkatachalam S, Babu MR, Kumar GS. Ludwig's Angina—an emergency: a case report with literature review. J Nat Sci Biol Med. 2012;3(2):206–8.
71. Neff SP, Merry AF, Anderson B. Airway management in Ludwig's angina. Anaesth Intensive Care. 1999;27(6):659–61.
72. Ovassapian A, Tuncbilek M, Weitzel EK, Joshi CW. Airway management in adult patients with deep neck infections: a case series and review of the literature. Anesth Analg. 2005;100(2):585–9.
73. Rosenstock CV, Thogersen B, Afshari A, Christensen AL, Eriksen C, Gatke MR. Awake fiberoptic or awake video laryngoscopic tracheal intubation in patients with anticipated difficult airway management: a randomized clinical trial. Anesthesiology. 2012;116(6):1210–6.
74. Schumann M, Biesler I, Borgers A, Pfortner R, Mohr C, Groeben H. Tracheal intubation in patients with odentogenous abscesses and reduced mouth opening. Br J Anaesth. 2014;112(2):348–54.
75. Tapiovaara L, Back L, Aro K. Comparison of intubation and tracheotomy in patients with deep neck infection. Eur Arch Otorhinolaryngol. 2017;274(10):3767–72.
76. Botha A, Jacobs F, Postma C. Retrospective analysis of etiology and comorbid diseases associated with Ludwig's angina. Ann Maxillofac Surg. 2015;5(2):168–73.
77. Wolfe MM, Davis JW, Parks SN. Is surgical airway necessary for airway management in deep neck infections and Ludwig angina? J Crit Care. 2011;26(1):11–4.
78. Potter JK, Herford AS, Ellis E 3rd. Tracheotomy versus endotracheal intubation for airway management in deep neck space infections. J Oral Maxillofac Surg. 2002;60(4):349–54; discussion 354-345.
79. Sarna T, Sengupta T, Miloro M, Kolokythas A. Cervical necrotizing fasciitis with descending

mediastinitis: literature review and case report. J Oral Maxillofac Surg. 2012;70(6):1342–50.
80. Pearse HE. Mediastinitis following cervical suppuration. Ann Surg. 1938;108(4):588–611.
81. Mora R, Jankowska B, Catrambone U, Passali GC, Mora F, Leoncini G, Passali FM, Barbieri M. Descending necrotizing mediastinitis: ten years' experience. Ear Nose Throat J. 2004;83(11):774, 776-780.
82. Roccia F, Pecorari GC, Oliaro A, Passet E, Rossi P, Nadalin J, Garzino-Demo P, Berrone S. Ten years of descending necrotizing mediastinitis: management of 23 cases. J Oral Maxillofac Surg. 2007;65(9):1716–24.
83. Prado-Calleros HM, Jimenez-Fuentes E, Jimenez-Escobar I. Descending necrotizing mediastinitis: Systematic review on its treatment in the last 6 years, 75 years after its description. Head Neck. 2016;38(Suppl 1):E2275–83.
84. Estrera AS, Landay MJ, Grisham JM, Sinn DP, Platt MR. Descending necrotizing mediastinitis. Surg Gynecol Obstet. 1983;157(6):545–52.
85. Endo S, Murayama F, Hasegawa T, Yamamoto S, Yamaguchi T, Sohara Y, Fuse K, Miyata M, Nishino H. Guideline of surgical management based on diffusion of descending necrotizing mediastinitis. Jpn J Thorac Cardiovasc Surg. 1999;47(1):14–9.
86. Ridder GJ, Maier W, Kinzer S, Teszler CB, Boedeker CC, Pfeiffer J. Descending necrotizing mediastinitis: contemporary trends in etiology, diagnosis, management, and outcome. Ann Surg. 2010;251(3):528–34.
87. Mihos P, Potaris K, Gakidis I, Papadakis D, Rallis G. Management of descending necrotizing mediastinitis. J Oral Maxillofac Surg. 2004;62(8):966–72.
88. Corsten MJ, Shamji FM, Odell PF, Frederico JA, Laframboise GG, Reid KR, Vallieres E, Matzinger F. Optimal treatment of descending necrotising mediastinitis. Thorax. 1997;52(8):702–8.
89. Wheatley MJ, Stirling MC, Kirsh MM, Gago O, Orringer MB. Descending necrotizing mediastinitis: transcervical drainage is not enough. Ann Thorac Surg. 1990;49(5):780–4.
90. Singhal P, Kejriwal N, Lin Z, Tsutsui R, Ullal R. Optimal surgical management of descending necrotising mediastinitis: our experience and review of literature. Heart Lung Circ. 2008;17(2):124–8.
91. Cavalcante MB, Lima ALO, Moreira RT, de Oliveira ESED, Branco BLC. Cervical-thoracic necrotizing fasciitis of odontogenic origin in a diabetic patient: a case report. Gen Dent. 2017;65(4):25–9.
92. Elander J, Nekludov M, Larsson A, Nordlander B, Eksborg S, Hydman J. Cervical necrotizing fasciitis: descriptive, retrospective analysis of 59 cases treated at a single center. Eur Arch Otorhinolaryngol. 2016;273(12):4461–7.
93. Lin WL, Yeh TC, Lu CH, Huang HK, Chiu WY. A catastrophic cervical necrotizing fasciitis after tooth extraction. Intern Emerg Med. 2016;11(8):1135–6.
94. Weiss A, Nelson P, Movahed R, Clarkson E, Dym H. Necrotizing fasciitis: review of the literature and case report. J Oral Maxillofac Surg. 2011;69(11):2786–94.
95. Tung-Yiu W, Jehn-Shyun H, Ching-Hung C, Hung-An C. Cervical necrotizing fasciitis of odontogenic origin: a report of 11 cases. J Oral Maxillofac Surg. 2000;58(12):1347–52; discussion 1353.
96. Ricalde P, Engroff SL, Jansisyanont P, Ord RA. Paediatric necrotizing fasciitis complicating third molar extraction: report of a case. Int J Oral Maxillofac Surg. 2004;33(4):411–4.
97. Cariati P, Monsalve-Iglesias F, Cabello-Serrano A, Valencia-Laseca A, Garcia-Medina B. Cervical necrotizing fasciitis and acute mediastinitis of odontogenic origin: a case series. J Clin Exp Dent. 2017;9(1):e150–2.
98. Malik V, Gadepalli C, Agrawal S, Inkster C, Lobo C. An algorithm for early diagnosis of cervicofacial necrotising fasciitis. Eur Arch Otorhinolaryngol. 2010;267(8):1169–77.
99. Petitpas F, Blancal JP, Mateo J, Farhat I, Naija W, Porcher R, Beigelman C, Boudiaf M, Payen D, Herman P, Mebazaa A. Factors associated with the mediastinal spread of cervical necrotizing fasciitis. Ann Thorac Surg. 2012;93(1):234–8.
100. Umeda M, Minamikawa T, Komatsubara H, Shibuya Y, Yokoo S, Komori T. Necrotizing fasciitis caused by dental infection: a retrospective analysis of 9 cases and a review of the literature. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2003;95(3):283–90.
101. Lee TC, Carrick MM, Scott BG, Hodges JC, Pham HQ. Incidence and clinical characteristics of methicillin-resistant Staphylococcus aureus necrotizing fasciitis in a large urban hospital. Am J Surg. 2007;194(6):809–12; discussion 812-803.
102. Brook I. Microbiology and management of peritonsillar, retropharyngeal, and parapharyngeal abscesses. J Oral Maxillofac Surg. 2004;62(12):1545–50.
103. Rae J, Misselbrook K. Lemierre's syndrome—a rare cause of disseminated sepsis requiring multi-organ support. J Intensive Care Soc. 2017;18(4):329–33.
104. Righini CA, Karkas A, Tourniaire R, N'Gouan JM, Schmerber S, Reyt E, Atallah I. Lemierre syndrome: study of 11 cases and literature review. Head Neck. 2014;36(7):1044–51.
105. Noy D, Rachmiel A, Levy-Faber D, Emodi O. Lemierre's syndrome from odontogenic infection: review of the literature and case description. Ann Maxillofac Surg. 2015;5(2):219–25.
106. Plymyer MR, Zoccola DG, Tallarita G. Pathologic quiz case: an 18-year-old man presenting with sepsis following a recent pharyngeal infection. Lemierre syndrome. Arch Pathol Lab Med. 2004;128(7):813–4.
107. Busko JM, Triner W. Lemierre syndrome in a child with recent pharyngitis. CJEM. 2004;6(4):285–7.

108. Malis DD, Busaidy KF, Marchena JM. Lemierre syndrome and descending necrotizing mediastinitis following dental extraction. J Oral Maxillofac Surg. 2008;66(8):1720–5.
109. Roscoe DL, Hoang L. Microbiologic investigations for head and neck infections. Infect Dis Clin North Am. 2007;21(2):283–304, v.
110. Lai YJ, Lirng JF, Chang FC, Luo CB, Teng MM, Chang CY. Computed tomographic findings in Lemierre syndrome. J Chin Med Assoc. 2004;67(8):419–21.
111. Karkos PD, Asrani S, Karkos CD, Leong SC, Theochari EG, Alexopoulou TD, Assimakopoulos AD. Lemierre's syndrome: a systematic review. Laryngoscope. 2009;119(8):1552–9.
112. Weerasinghe D, Lueck CJ. Septic cavernous sinus thrombosis: case report and review of the literature. Neuroophthalmology. 2016;40(6):263–76.
113. Ebright JR, Pace MT, Niazi AF. Septic thrombosis of the cavernous sinuses. Arch Intern Med. 2001;161(22):2671–6.
114. Southwick FS, Richardson EP Jr, Swartz MN. Septic thrombosis of the dural venous sinuses. Medicine (Baltimore). 1986;65(2):82–106.
115. Prabhu S, Jain SK, Dal Singh V. Cavernous sinus thrombophlebitis (sans thrombosis) secondary to odontogenic fascial space infection: an uncommon complication with unusual presentation. J Maxillofac Oral Surg. 2015;14(Suppl 1):168–72.
116. Weerasinghe D, Lueck CJ. Septic cavernous sinus thrombosis: case report and review of the literature. Neuroophthalmology. 2016;40(6):263–76. eCollection 2016 Dec.
117. van der Poel NA, Mourits MP, de Win MML, Coutinho JM, Dikkers FG. Prognosis of septic cavernous sinus thrombosis remarkably improved: a case series of 12 patients and literature review. Eur Arch Otorhinolaryngol. 2018;275(9):2387–95.
118. Lewandowski B, Pakla P, Wolek W, Jednakiewicz M, Nicpon J. A fatal case of descending necrotizing mediastinitis as a complication of odontogenic infection. A case report. Kardiochir Torakochirurgia Pol. 2014;11(3):324–8.
119. Marulasiddappa V, Tejesh CA. Lemierre's syndrome presenting with septic shock. Indian J Crit Care Med. 2013;17(6):382–4.
120. Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, Bellomo R, Bernard GR, Chiche JD, Coopersmith CM, Hotchkiss RS, Levy MM, Marshall JC, Martin GS, Opal SM, Rubenfeld GD, van der Poll T, Vincent JL, Angus DC. The Third International Consensus definitions for sepsis and septic shock (Sepsis-3). JAMA. 2016;315(8):801–10.
121. Rajab B, Laskin DM, Abubaker AO. Odontogenic infection leading to adult respiratory distress syndrome. J Oral Maxillofac Surg. 2013;71(2):302–4.
122. Adekeye EO, Cornah J. Osteomyelitis of the jaws: a review of 141 cases. Br J Oral Maxillofac Surg. 1985;23(1):24–35.
123. Haeffs TH, Scott CA, Campbell TH, Chen Y, August M. Acute and chronic suppurative osteomyelitis of the jaws: a 10-year review and assessment of treatment outcome. J Oral Maxillofac Surg. 2018;76(12):2551–8.
124. Lew DP, Waldvogel FA. Osteomyelitis. N Engl J Med. 1997;336(14):999–1007.
125. Andre CV, Khonsari RH, Ernenwein D, Goudot P, Ruhin B. Osteomyelitis of the jaws: a retrospective series of 40 patients. J Stomatol Oral Maxillofac Surg. 2017;118(5):261–4.
126. Shen JY, Futran ND, Sardesai MG. Craniofacial Actinomyces osteomyelitis evolving from sinusitis. Radiol Case Rep. 2018;13(1):104–7.
127. Cierny G III, Mader JT, Penninck JJ. A clinical staging system for adult osteomyelitis. Clin Orthop Relat Res. 2003;(414):7–24.
128. Lee YJ, Sadigh S, Mankad K, Kapse N, Rajeswaran G. The imaging of osteomyelitis. Quant Imaging Med Surg. 2016;6(2):184–98.
129. Koorbusch GF, Deatherage JR, Cure JK. How can we diagnose and treat osteomyelitis of the jaws as early as possible? Oral Maxillofac Surg Clin North Am. 2011;23(4):557–67, vii.
130. Coviello V, Stevens MR. Contemporary concepts in the treatment of chronic osteomyelitis. Oral Maxillofac Surg Clin North Am. 2007;19(4):523–34, vi.
131. Gaetti-Jardim Junior E, Fardin AC, Gaetti-Jardim EC, de Castro AL, Schweitzer CM, Avila-Campos MJ. Microbiota associated with chronic osteomyelitis of the jaws. Braz J Microbiol. 2010;41(4):1056–64.
132. Hudson JW, Daly AP, Foster M. Treatment of osteomyelitis: a case for disruption of the affected adjacent periosteum. J Oral Maxillofac Surg. 2017;75(10):2127–34.
133. Baur DA, Altay MA, Flores-Hidalgo A, Ort Y, Quereshy FA. Chronic osteomyelitis of the mandible: diagnosis and management—an institution's experience over 7 years. J Oral Maxillofac Surg. 2015;73(4):655–65.
134. Hudson JW. Osteomyelitis of the jaws: a 50-year perspective. J Oral Maxillofac Surg. 1993;51(12):1294–301.
135. Ferrera PC, Busino LJ, Snyder HS. Uncommon complications of odontogenic infections. Am J Emerg Med. 1996;14(3):317–22.
136. Flynn TR. What are the antibiotics of choice for odontogenic infections, and how long should the treatment course last? Oral Maxillofac Surg Clin North Am. 2011;23(4):519–36, v-vi.
137. Farmahan S, Tuopar D, Ameerally PJ, Kotecha R, Sisodia B. Microbiological examination and antibiotic sensitivity of infections in the head and neck. Has anything changed? Br J Oral Maxillofac Surg. 2014;52(7):632–5.
138. Poeschl PW, Spusta L, Russmueller G, Seemann R, Hirschl A, Poeschl E, Klug C, Ewers R. Antibiotic susceptibility and resistance of the odontogenic microbiological spectrum and its clinical impact on severe deep space head and neck infections. Oral

Surg Oral Med Oral Pathol Oral Radiol Endod. 2010;110(2):151–6.
139. Rega AJ, Aziz SR, Ziccardi VB. Microbiology and antibiotic sensitivities of head and neck space infections of odontogenic origin. J Oral Maxillofac Surg. 2006;64(9):1377–80.
140. Zirk M, Buller J, Goeddertz P, Rothamel D, Dreiseidler T, Zoller JE, Kreppel M. Empiric systemic antibiotics for hospitalized patients with severe odontogenic infections. J Craniomaxillofac Surg. 2016;44(8):1081–8.
141. Brook I. Microbiology and treatment of brain abscess. J Clin Neurosci. 2017;38:8–12.
142. Das ST, Tiwari DP, Sharma V, Nath G. Microbiological profile of brain abscess. World J Med Res. 2013;6.
143. Barber BR, Dziegielewski PT, Biron VL, Ma A, Seikaly H. Factors associated with severe deep neck space infections: targeting multiple fronts. J Otolaryngol Head Neck Surg. 2014;43(1):35.
144. Gallo O, Mannelli G, Lazio MS, Santoro R. How to avoid life-threatening complications following head and neck space infections: an algorithm-based approach to apply during times of emergency. When and why to hospitalise a neck infection patient. J Laryngol Otol. 2018;132(1):53–9.
145. Ban MJ, Jung JY, Kim JW, Park KN, Lee SW, Koh YW, Park JH. A clinical prediction score to determine surgical drainage of deep neck infection: a retrospective case-control study. Int J Surg. 2018;52:131–5.

6 Complications in Oral Implant Placement

Peer W. Kämmerer and Bilal Al-Nawas

Contents

6.1 Introduction

The demographic change is one of the central challenges of the dental profession, which manifests itself in daily practice. As the patients are becoming more aged, there is also an increase in

P. W. Kämmerer (✉) · B. Al-Nawas
Department of Oral, Maxillofacial and Plastic Surgery, University Medical Centre Mainz, Mainz, Germany
e-mail: Al-Nawas@uni-mainz.de

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_6

risk factors. In “major surgery”, age itself is an independent risk factor for perioperative mortality [1]. In implant dentistry, age alone could not be associated with a decreased implant prognosis [2]. Even so, not only implant prognosis requires our attention as potential negative consequences for the general condition of the patient have to be considered as well [3]. For example, bleeding triggered by implant-associated measures can become vitally threatening. The patient on antiresorptive medication may consider the potential triggering of bone necrosis to be more relevant than implant prognosis or even having fixed dentures; the benefit of a dental implant therapy is always to be evaluated with medical risk in the sense of a cost–benefit analysis. Accordingly, this chapter of the book will focus on potential individual, surgical and post-surgical complications of dental implant placement.

6.2 Implant Prognosis

Potential problems in the context of osseointegration, soft tissue healing and of the implant-supported rehabilitation should be discussed first. Of importance are all diseases and drugs that will affect the hard- and soft tissue healing as well as the immunological reaction.

Interestingly, modern implant systems seem to heal so predictably that high success rates are possible even in high-risk patients [4–6]. Frequently, however, these successes, for example in patients that experienced a radiotherapy (Fig. 6.1), are based on a particularly non-invasive surgical approach [6]. Long-term prognosis may be impaired due to several local and systemic factors. In addition, the subjective importance of oral hygiene may be reduced in the case of stressful, severe general illness. Modern implant dentistry should necessarily include these aspects in the planning. Therefore, an adequate risk analysis is essential and must be included in the dentists’ and patients’ education. Under these conditions, implant-supported rehabilitation can be a significant quality of life benefit. On the other hand, if the indication is incorrect, complications of the implant surgery can become vitally threatening. In brief:

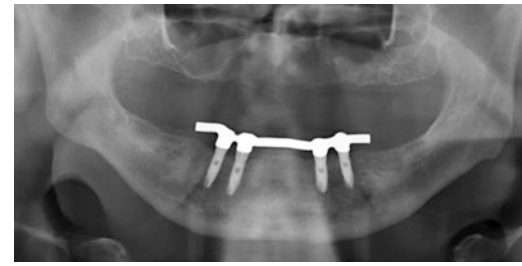


Fig. 6.1 Panoramic X-ray: dental implants in an irradiated patient 10 years after placement without any sign of pathology

- General diseases and medication should be evaluated for the effects on hard and soft tissue healing as well as immune defense.
- Patients with severe general conditions may have oral hygiene deficits more frequently.
- The potential gain in quality of life must be balanced against the risk of complications.
- The patient should be informed about this risk assessment; this results in participatory decision-making.

6.3 Complication Prediction

6.3.1 Pre-surgical Selection of Patients and Evaluation of Risk Factors

6.3.1.1 Diabetes Mellitus

In patients with diabetes mellitus, tooth loss is more common and the risk of periodontitis has reported to be significantly increased [7]. The acute blood glucose impairment during medical measures such as surgical implant placement is an emergency that can be easily solved by adding glucose to the patient, though the influence of bone metabolism on the elevated blood sugar levels is more difficult. Here, it is important to distinguish disorders of ossointegration and problems in the long-term course. The Hba1c value refers to glycated hemoglobin and offers an overall picture of the average plasma glucose levels over a period from weeks to months. In brief, it has established

itself as an objective measure of how well the blood sugar is adjusted; a value of below 6% is considered to be normal, of 6–6.4% to be pre-diabetic and of >6.5% to indicate manifest diabetes. For dental implant therapy, a value >10% is regarded as "badly" adjusted and a slower osseointegration can be expected. However, after 1 year of loading or more, no altered implants' survival could be seen [8, 9]. In a recent systematic review, hints towards a greater risk of peri-implantitis in patients with diabetes mellitus/hyperglycaemia was found [10].

It is striking that there are virtually no solid data on the question of augmentation procedures and diabetes. Clinically, no abnormalities appear here for circumscribed augmentations (sinus lift, Guided Bone Regeneration (GBR)), whereas extensive augmentations in patients with diabetes are poorly scientifically proven. On the other hand, the favorable effect of perioperative antibiotic prophylaxis is relatively clear in patients with diabetes.

On this basis, the German S3 guideline concludes as follows [11]:

- Dental implant rehabilitation in patients with diabetes mellitus is a safe and predictable procedure.
- Before the start of treatment, the practitioner should inform himself about the cessation of diabetes mellitus. Patients with poorly controlled diabetes mellitus appear to have delayed osseointegration after implantation.
- Due to evidence of delayed osseointegration, the indication for immediate and early loading as well as transgingival healing should be critically assessed.
- Patients with diabetes mellitus are at a higher risk for peri-implantitis, so risk-adjusted follow-up care should be provided after implant placement.
- There are indications that adjuvant therapy with prophylactic administration of an antibiotic and use of chlorhexidine-containing mouthwashes improves the success of treatment.

6.3.1.2 Smoking

Tobacco products are known to contain more than 4000 potentially toxic substances that were attributed to be responsible for a broad variety of oral and extra-oral diseases. In terms of oral health, it is known that tobacco smokers have an increased prevalence of periodontitis and a higher number of missing teeth when compared to non-smokers [12]. For the interface between dental implants and the local bone, a series of deleterious effects on osteogenesis and angiogenesis were reported [13] that will lead to increased postoperative infection, marginal bone loss and dental implant failure [14, 15]. Even so, there is a lack of evidence in the literature on the potential dose-related effect of smoking with regard to dental implant healing [16]. In brief, smoking is no contraindication for dental implant therapy. Even so, the surgeon as well as the patient should be aware on the potential higher rate of complications in the short and the long term.

6.3.1.3 Osteoporosis and Antiresorptive Drugs

In general, bone density is determined radiographically (Bone Mineral Density—BMD). As it decreases in age and is lower in women than in men, the diagnosis of a deficit relative to normal distribution is age and sex dependent. Depending on the severity, osteopenia or osteoporosis is defined. This disproportion between bone formation and degradation typically affects postmenopausal women. From the view of implant dentistry, especially in restorations in the lateral maxilla, a very soft bone—often compared to styrofoam by the feeling of drilling—may require under-dimensioned processing and possibly prolonged healing times. Considering these peculiarities, an implant treatment is feasible even in osteoporosis. A current cohort study on implant treatment in older women shows no impact of osteoporosis on implant prognosis [17]. Similarly, there is no difference in the long-term bone level [18]. Systematic reviews on this topic also confirm the safety of implant dentistry in patients with osteoporosis [19, 20].

Since 1986, bisphosphonates have been used to treat a variety of resorbing bone diseases reducing osteoporosis-related bone fractures, though bisphosphonate-related osteonecrosis of the jaws (Figs. 6.2 and 6.3) constitutes a severe side effect, also seen in cases of other

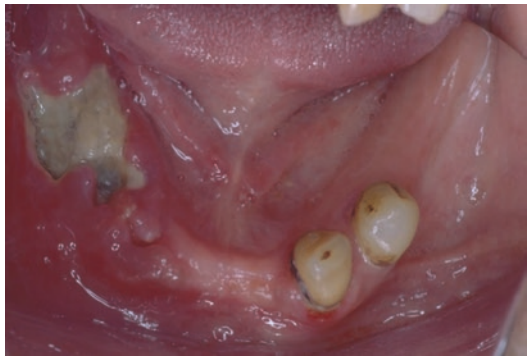


Fig. 6.2 Bisphosphonate-related osteonecrosis of the mandible after extraction of a molar

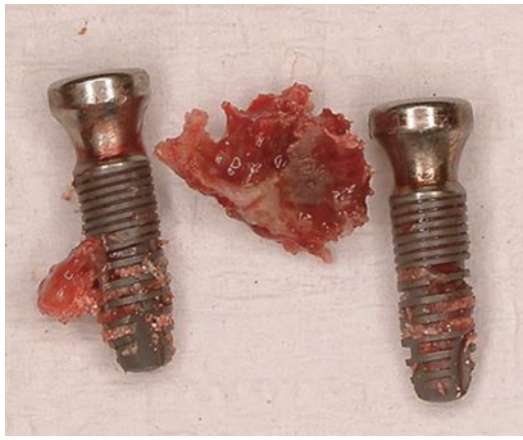


Fig. 6.3 Implants within a medication-related osteonecrosis of the jaws that had to be removed together with the surrounding bone

anti-resoptive drugs such as RANKL inhibitors (e.g., denosumab) leading to the designation of this secondary effect as medication-related osteonecrosis of the jaws [21]. The main risk factors are known to be dento-alveoar trauma, periodontal disease and tooth extraction, though even spontaneous development was reported [22, 23]. Even if the incidence is significantly higher with high dose i.v. bisphosphonate admission, oral intake may also lead to this kind of osteomyelitis, also after placement or removal of dental implants [23]. As this complication is difficult to control and a symptomatic therapy is available only, primary focus is laid on prevention. This implies a comprehensive oral examination and treatment of other risk factors such as periodontitis before considering alveolar surgery.

On this basis, the German S3 guideline concludes as follows [24]:

- In all patients with antiresorptive therapy, the individual risk for development of medication-related osteonecrosis of the jaws should be evaluated before dental implant treatment.
- Indicated dental procedures should be finished before dental implant treatment and the wound healing parameters should be included in individual risk assessment.
- Bone augmentation procedures are not recommended.
- After dental implant placement, the patient should be included in a risk-adopted follow-up program.

6.3.1.4 Rheumatic and Autoimmune Diseases

Rheumatic diseases represent such a heterogeneous picture that a simplifying summary is actually not permitted. Certainly, not only chronic polyarthritis but also rarer diseases such as scleroderma or sarcoidosis make the surgeon aware of possible risks. For therapy, cortisone, chemotherapeutics and, more recently, complex immunotherapeutic medication are often used. At least in the long term, an increased peri-implant bone resorption can be expected [25, 26]. In the current textbooks, long-term cortisone intake is described as critical for bone healing. Clinically and pre-clinically, however, no strong evidence can be found in this statement. Nevertheless, "harmless" non-steroidal anti-inflammatory drugs (e.g., diclofenac) are also commonly used. A highly discussed study has revealed a significantly higher number of implant losses in patients taking non-steroidal anti-inflammatory drugs; in particular, the so-called cluster failures (increased implant losses in a few patients) occurred [26]. Similar data are available from another working group for these drugs [27]. In addition, proton pump blockers are also shown as potentially critical. In brief:

- Rheumatic diseases and autoimmune diseases usually require consultation with the attending physician because of their complexity in diagnosis, course and therapy.

- The question of the negative influence of non-steroidal anti-inflammatory drugs on implant healing requires further attention. So far, a real clinical warning cannot (yet) be pronounced on the basis of two retrospective studies.

6.4 Surgical Complications

6.4.1 Damage to Adjacent Teeth

During implant surgery procedures, damage to adjacent teeth may occur, mostly due to a lack of parallelism of the implant with neighboring teeth or by excessive proximity to the contiguous teeth [28]. Therefore, it is recommended and generally accepted that a minimum distance of 1.5 mm should be respected to each adjacent tooth (Figs. 6.4 and 6.5). If an implant-related damage occurs, the treatment of the respective tooth up to its extraction is mandatory.

6.4.2 Displacement of Dental Implants

Therapeutic goals of dental implant therapy have changed from purely functional purposes to restoration of esthetics using prosthetically driven implant treatment plans. This means that the final location of the restauration decides the implant position and not vice versa [29]. Here, the direction of occlusal loading and not the amount of bone available for the implant determine the implants' bucco-lingual inclination [30].

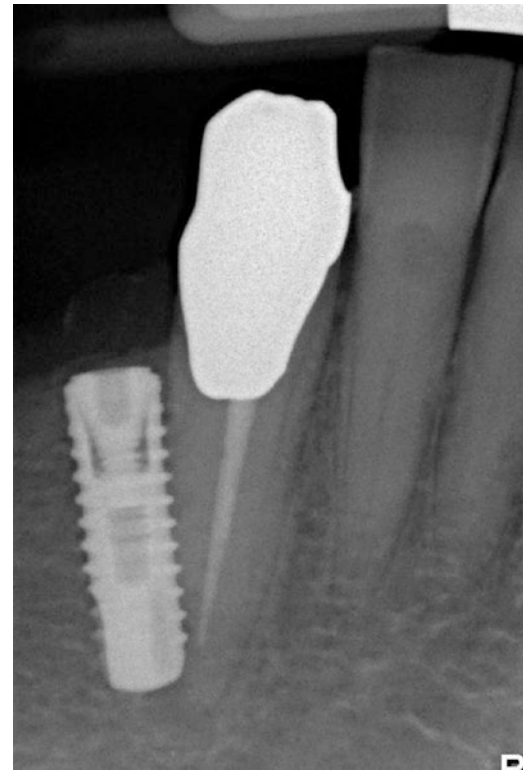


Fig. 6.4 Single-tooth radiograph: implant placed in close proximity to a tooth that received a root canal treatment

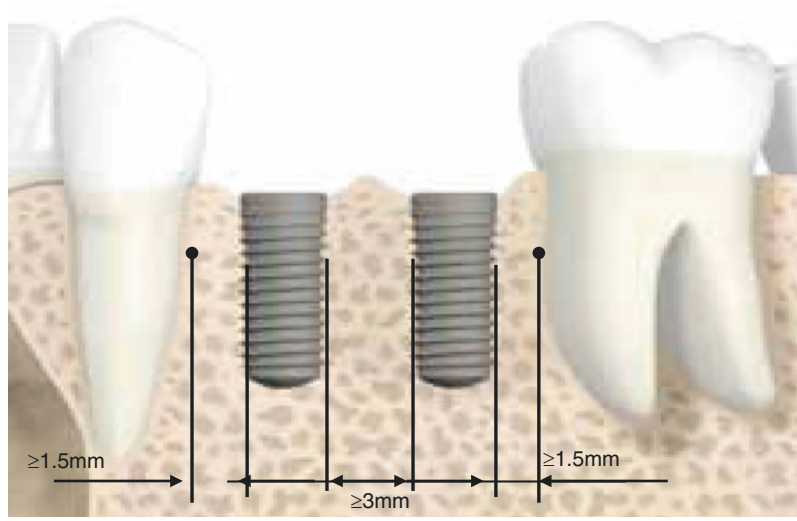


Fig. 6.5 Recommendations for minimum distances between implant and tooth as well as between two implants (from: ITI-Treatment-Guide)

Accidental implant perforation of the cortical bone or even implant migration is not an uncommon problem that might happen either during surgery or even in the healing phase potentially provoking serious adverse events. For example, displacement of an implant into the sublingual space may occur due to reabsorption of the (thin) cortical bone [31]. Also, displacement of dental implants into focal osteoporotic bone marrow defects of the mandible was described [32]. Accordingly, some surgical guides following the ideal prosthetic position without considering underlying anatomic limitations may run the risk of lingual plate perforation, thus leading to severe surgical complications [33]. In the posterior mandible, such lingual bone plate perforation is a surgical complication that is increased in the presence of a lingual concavity (reported in up to 39% of patients [34]). Though the reported incidence of such cases is low [35], it has to be considered that this type of adverse event could have been unnoticed (e.g., because of artifacts around the metallic implants or because of a lack of postoperative 3D radiology) or unreported [36]. Accordingly, when employing virtual implant placement in the posterior mandible, an incidence of lingual plate perforation of up to 1.2% was reported, mostly in sites with lingual concavity [33]. Therefore, it was recommended at least to palpate the lingual ridge of the posterior mandible prior implant treatment to detect lingual concavities. If the finding is positive, a 3D-radiologic scan together with a surgical guide may be indicated [33].

In the maxilla, low bone quality and a high pneumatized maxillary sinus possibly compromise success and survival of dental implants. Here, the implant may also migrate into the paranasal sinus cavities (Fig. 6.6). Potential reasons for this significant complication are inexperience of the surgeon, unexpected perforations during sinus floor elevation, application of heavy force on the low density bone during implant insertion, and dental implant placement without sinus floor elevation of an excessively pneumatized maxillary sinus [37, 38]. If displaced deeper into the craniofacial cavities, further complications such as infection, sinusitis, tissue necrosis and adverse reactions to foreign bodies are possible [39]. Though, migration of an implant into the maxillary sinus is usually symptomless and the implant may even disappear during the follow-up examinations potentially creating the risk for foreign body aspiration together with a life-threatening condition [37].

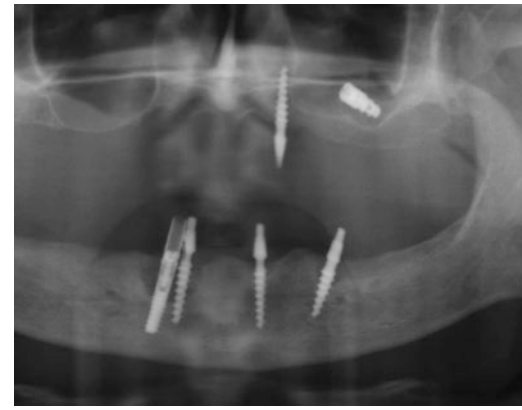


Fig. 6.6 Panoramic X-ray: patient referred for removal of the displaced implant as well as the other implants due to severe peri-implantitis

Once again, 3D radiology offers a tool for implant treatment planning providing high resolution and accurate images of the area of interest even if there is still a significant deviation at entry point and apex when using either computer generated surgical guides or navigation systems [36]. In general, avoidance of any manipulation before surgical removal of the implant can be recommended. The surgical treatment options of foreign bodies in the mandible consist of careful removal of the implant. In the maxilla, an endoscopic approach through the nose or the mouth after atral osteotomy has the advantages of low morbidity, rapid recovery and possible treatment of the affected paranasal cavities [39].

6.4.3 Bleeding, Hematoma and Airway Obstruction

Data from the analyzed CT images revealed intraosseous vessels in the lateral sinus wall visible in 50% of cases. Injury of these structures during osteotomy of the sinus wall, if not taking

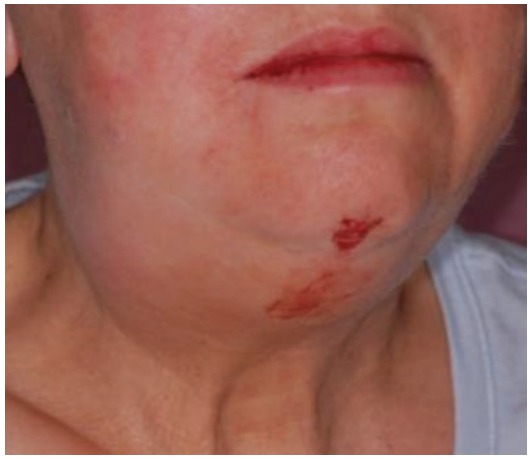


Fig. 6.7 Patient on oral anti-aggregation with severe hematoma after surgical procedure in the posterior mandible

care of, might result in intense bleeding [40, 41]. Besides, severe hemorrhage may occur after iatrogenic damage to the descending or posterior palatin artery. Nevertheless, most bleeding episodes are reported in the mandible. There, in the anterior third/interforaminal region and the floor of the mouth (Fig. 6.7), a rich vascularization by a high number of variable branches and anastomoses of the submental and sublingual arteries (arterial diameter 0.18–1mm with a blood flow of 0.7–3.7 mL/min [42]) is seen. Both these vessels are usually located close to the periosteum of the mandibular lingual cortex. Here, if the lingual periosteum is surgically damaged and/or the lingual cortex is perforated during implant drilling or implant placement, these structures within the sublingual space may be lacerated or transected lading to immediate or delayed bleeding episodes [43]. Also, the respective vessels entering the mandible through bone canals from the lingual side can cause such bleeding when they are injured during host site preparation [44].

Delayed bleeding may be result of the fading vasoconstriction caused by local anesthetics with epinephrine adjunct which can mask the vascular trauma for a certain amount of time. Though, damage to the soft tissue structures in the floor of the mouth (e.g., the anterior belly of the digastric muscle and the mylohyoid muscle) may also induce bleeding without direct vessel damage. Due to the loose tissues in the floor of the mouth, and the high arterial blood flow, spreading of the hematoma to the sublingual area and the space between the lingual muscles is easy. Even if this usually occurs in the area of the anterior mandible as the respective vessels are located close to the lingual plate and the alveolar crest [45], this problem was also reported after placement of implants in the posterior mandible [46, 47]. Usually, the symptoms consist of hematoma formation together with submental swelling, displacement of the tongue, problems while swallowing and/or upper airway obstruction. To avoid this kind of complication, meticulous soft tissue management is essential, and clinicians should exercise maximum care to avoid subperiosteal rupture [46]. As this condition is more likely to occur in cases of severe mandibular atrophy [45] or when needing longer implants, a thorough anatomical knowledge is mandatory for the surgeon. Accordingly, some authors demanded pre-surgical evaluation of mandibular anatomical structures via CT imaging to be an essential part in dental implant planning [43, 44]. It was also recommended to place implants of a length of less than 14 mm in the mandible only [48].

First therapy should be application of bimanual pressure to the floor of the mouth and the lingual mandibular surface as well as application of hemostatic agents into the drilled holes. If not effective, surgical exploration of the site including evacuation of blood clots either under local or under general anesthesia should be carried out. Bleeding vessels need to be ligated or cauterized. In cases direct exploration does not show to be effective, an extraoral approach for ligation procedures of the facial/submental arteries or external carotid angiography together with vascular embolization may become necessary [49, 50]. Evaluation for a possible underlying coagulopathy should be carried out; if the postoperative sublingual hematoma is result of oral anticoagulant medication, surgical exploration and drainage are usually not the treatment options of choice [51]. Compromised upper airways should be always secured in a first management step using intubation narcosis or emergency tracheos-

tomy. To prevent infection in cases of extensive hematomas, antibiotics are recommended [47].

6.4.4 Ingestion/Aspiration of Instruments

Foreign bodies used in dentistry such as drills, implant components and restorations may be ingested swallowed/aspirated during a variety of dental procedures including placement of implants [52]. This ingestion is seen more frequently in children as well as in elderly, mentally impaired of alcoholic patients [53].

Prevention of this complication applies to the identification of patients at risk by means of comprehensive clinical examination including the patients' anamnesis (e.g., medication, wide oropharynx, absence of gag reflex). Next, the patients' position in the dental chair could be adjusted in accordance with the treatment. For example, a supine position in the dental chair will offer direct access to the oropharynx predisposing ingestion of slippery instruments, whereas a sitting position prevents ingestion/aspiration [53, 54]. Placement of a gauze screen across the oropharynx and using ligatures to tie instruments are easy methods for further prevention, of ingestion. If an ingestion of an instrument occurred and the material cannot be removed manually, an immediate radiographic evaluation including the chest, neck and the abdominal region should follow. Though, if the materials lack radiopacity, they may be very difficult to locate. Here, diagnostic bronchoscopy/endoscopy or even 3D radiology might be required [55]. The conservative management of ingested instruments includes serial radiographs and a fiber rich diet. Serious complications (e.g., intestinal perforation, bleeding, obstruction, impactions) occur rarely and instruments that have entered the gastrointestinal tract will most likely pass it without trauma within several days to several weeks. However, if the successive radiographs show the same location of the foreign body or there is a sign of any abdominal tenderness or hemorrhage, invasive removal of the material should be conducted as early as possible

6.4.5 Rhinosinusitis and Sinus Lift Dental Implantation

For placement of dental implants in the posterior maxilla, the maxillary sinus constitutes the main anatomical limitation. Here, when the height of the alveolar bone is limited, sinus elevation can be performed using a variety of different techniques.

The indirect sinus elevation technique (also known as osteotome sinus elevation, crestal or coronal approach [56, 57]) is used for enhancing the maxillary height through an osteotomy prepared in the alveolar crest. Implants may be inserted simultaneously or after a certain time of healing. In general, sinus lift procedures can be considered to be safe. However, several complications have been reported as well such as membrane perforation [58], development of chronic sinusitis [59] (Fig. 6.8), severe bleeding episodes [40] or even paroxysmal positional vertigo [60]. After a certain time, sinus surgery can result in formation of mucocele [61].

Rhinosinusitis (inflammation of the nose and paranasal sinuses) is a very common disease in Western societies having a relevant morbidity. Typical symptoms are nasal blockade and discharge, facial pain and/or pressure, hyposmia, headache, dental and ear pain, halitosis, fatigue and cough [62]. The disease may develop after contamination of the maxillary sinus after dental

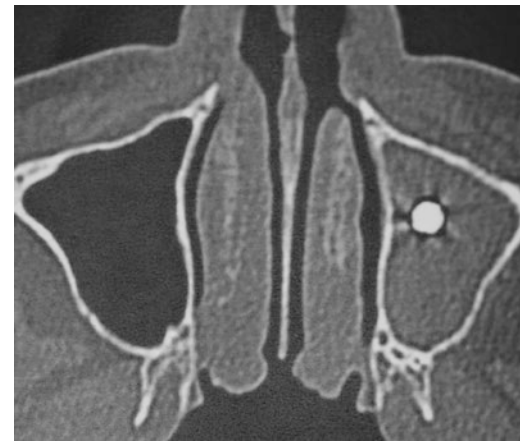


Fig. 6.8 Cone beam tomography showing an implant displaced within the maxillary sinus together with a chronic sinusitis

procedures such as sinus augmentation, especially after large perforations of the maxillary sinus due to the discharge of material fragments. On contrary, small perforations (<2 mm) will often heal spontaneously without development of maxillary sinusitis [63]. Other reasons of rhinosinusitis after dental surgery are named to be ostium obstruction due to postoperative swelling of the maxillary mucosa, blockage of air flow due to diminished intrasinus volume, impaired mucosal activity in the maxillary sinus due to mucosal lacerations as well as implant exposure and extensions [62, 63]. Also, the exposure of dental implants into the nasal cavity may give rise to rhinosinusitis, probably due to the altered nasal airflow [64]. On the other hand, implants' exposure in the maxillary sinus does not seem to cause maxillary sinusitis [65, 66], whereas implant migration into the sinus will lead to severe sinusitis [67]. Accordingly, in between 2 and 4% of cases after sinus surgery, transient or even chronic purulent maxillary sinusitis was reported [62, 63]. For further prevention after sinus elevation surgery, patients are recommended to avoid blowing their nose but also the use of continuous positive airway pressure masks, e.g., because sleep apnea, may result in development of oroantral communications leading to loss of the grafted bone [68]. Though, there may be a latency period of up to a year for augmentative dental surgery-associated maxillary sinusitis and a latency of almost 4 years in implant-associated maxillary sinusitis [69].

If maxillary sinusitis occurs after sinus surgery and/or placement of dental implants into the sinus, immediate treatment is advocated to exclude life-threatening situations such as pansinusitis or even brain abscesses [70]. Odontogenic sinus infections are generally polymicrobial with predominantly anaerobic organisms present in cultures, commonly including peptostreptococcus, prevotella, and fusobacterium [71]. Therefore, conservative treatment should consist of systemic antibiosis with piperacillin or ampicillin in combination with a beta-lactamase inhibitor. For patients with allergy to penicillin, high susceptibility rates were observed with moxifloxacin, ciprofloxacin, and tetracyclines [62, 71]. If the sinus disease is refractory to the respective medication, a surgical approach involving elimination of the underlying dental pathology is aimed for [72].

6.4.6 Mandibular Fracture

In partially edentulous and in totally edentulous patients, the lack of sufficient bone volume for placement of dental implants is a common problem. Reasons may be the presence of the inferior alveolar nerve (IAN), of a submandibular gland fossa, local atrophy due to the lack of teeth at this site or complex surgical techniques, such as the transposition or lateralization of the IAN. Accordingly, the placement of dental implants of these areas may result in reduction of the structural integrity of the mandible leading to a mandibular stress fracture [73–75]. It was discussed controversially, if those implants have to be removed or not [74]. However, this decision remains individually at the time of presentation as there are insufficient data on the outcome of implants in the line of a fracture. Though if the implant is mobile, it should be considered to be a foreign body and thus removed at the time of fracture management. Of course, osteosynthesis of the fractured bone is mandatory even if repair of the fracture via bone graft was described as well [76]. Patients at risk for mandibular fractures should be seen in periodical clinical and radiological follow-ups. Besides, they are supposed to omit occlusal overload during the osseointegration phase.

6.4.7 Nerve Damage

Alteration of the sensation of the IAN or the lingual nerve (LN) after placement of dental implants in the posterior mandible is a serious complication with a reported prevalence of as high as 13% [77, 78]. Interestingly, the IANB is injured significant more commonly as the LN when related to dental implant placement (64.4% versus 28.8%) [79]. The iatrogenic nerve damage most likely occurs due to local

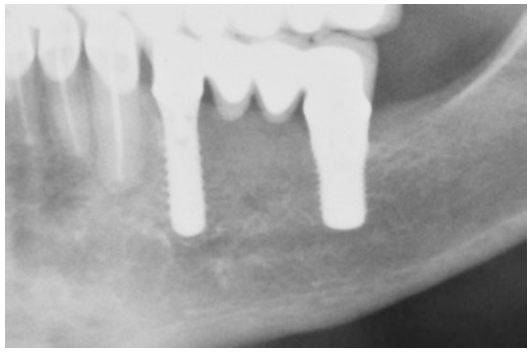


Fig. 6.9 Panoramic X-ray showing implants in the posterior mandible in close proximity to the inferior alveolar nerve

trauma for example after injection of local anesthesia, either due to direct trauma from the (barbed) injection needle, hematoma formation or neurotoxicity of the local anesthetic [80, 81]. Also, injuries caused by implant drills and the implant itself (Fig. 6.9) as well as surgical procedures such as sharp incision at the site of the mental foramen, chemical trauma by special irrigation solutions, thermal injury produced by excessive drill speed, local pressure of soft tissue retraction and surgical hooks or nerve compression caused by suture material may cause an altered nerve sensation [82, 83]. The degree of alteration in sensation that varies from mild paresthesia to complete anesthesia may be transient or even permanent resulting in an affection of many daily functions such as speech, eating, kissing, shaving and drinking. Besides, neuropathic pain after minor perforation of the roof of the mandibular cannal constitutes a reported problem [84]. In accordance, this injury negatively affects the patients' quality of life. Besides, as it is iatrogenic, negative psychological effecty may be exaggerated [83, 85]. Partially, the amount of affection is dependent on the amount of nerve damage (Table 6.1 (from: [86, 87])).

The different types of nerve injury are easily diagnosed with standardized neurosensory examinations also to be performed by the dental practitioner. Before dental treatment, especially in patients with history of alteration of nerval sensory function of associated with previous implant or impacted third molar extraction, examination oft he patient's sensory function is strongly recommended. If nerve damage occurs during surgery, it is of outmost importance to know about the respective possible outcomes and to refer the patient to a specialized center if indicated. Simple neurosensory tests are shown in Table 6.2 (from: [86, 88, 89]). While testing, the patient should be positioned comfortably having her or his eyes closed without distractions.

Table 6.1 Different forms of altered sensation as well as different forms of nerve injuries

Forms of altered sensation	
Paresthesia	Alteration in sensation felt as numbness, burning or prickling sensations, either evoked spontaneous
Dysthesia	Spontaneous or evoked unpleasant abnormal sensation
Analgesia	Loss of pain sensation
Anesthesia	Loss of perception of stimulation by any noxious or non-noxious stimulant
Forms of nerve injuries	
Neuropraxia	Preservation of the continuity of the axon; usually temporary injury
Axonotmesis	Axons disrupted but overall structure and integry of neural tube intact
Neurotmesis	Disruption of the inegrity of the neural tube

Table 6.2 Simple clinical neurosensory tests; for control, the contralateral side is used and the results are documented accurately

Clinical neurosensory tests	
Mechanoceptive	
Static light touch detection	Patient is asked to tell when he/she feels light touch on the face and to point to the exact location
Brush directional discrimination	Patient is asked to tell when he/she feels the brush and to determine the direction of movement
Two-point discrimination	Patient is asked to determine single and 2 points of touch. Theexaminer uses any 2 instruments by which the patient can change the distance between them
Nociceptive	
Pin pressure nociception	Patient is asked to determine the feeling of a pin prick
Thermal discrimination	Patient is asked if he/she feels cold or heat

6.4.7.1 Inferior Alveolar Nerve (IAN)

The IAN is a branch of the posterior division of the mandibular nerve that enters the mandibular foramen and runs in the mandibular canal until leaving the canal through the mental foramen being the mental nerve. In most of the cases, the IAN follows the lingual cortical plate of the mandibular ramus and body (type 1). Though, it may also be located in the middle of the mandibular ramus posterior to the second molar and then runs lingually to follow the lingual plate (type 2) or it may be located near the middle of the ramus and body (type 3 [90]). There is also a low occurrence (0.08–0.95%) of bifid IAN nerve canals enhancing the probability of a nerve damage when placing a dental implant [91–93].

For localization of the IAN during planning of dental implants, several methods are currently used including conventional 2D radiology as well as 3D radiology (cone beam computed tomography (CBCT) or computed tomography (CT)). Even if 3D imaging proves the most accurate and precise method for localization of the IAN, it cannot be considered to be the standard of care due to its cost and radiation exposure. Accordingly, mostly 2D radiology is used which has shown to be sufficient in majority of cases [94]. Though, a safety zone of 1.5–2 mm between the implant body and the mandibular canal is recommended [77, 95], the magnification of the respective machine has to be known in order to calculate the dimensional changes. Also, palpation and bone sounding under local anesthesia are helpful in determining the buccolingual width of the ridge [86]. Surgical exploration of the mental nerve has shown to be of limited value as the nerve exhibits an irregular intraosseous course [96]. Though, even if the exact course of the IAN is known, the surgical procedures should be carried out with outmost care. For example, some implant drills may be longer than the corresponding implants in order to enhance their drilling efficacy. If this detail of the implant system is not known or if the low resistance of the cancellous bone will lead to slippage of the drill, an involuntary damage to the IAN may occur [97, 98]. Besides, when aiming for an immediate implant placement following tooth extraction, efforts to achieve primary stability may also lead to apical extension and nerve injury.

In brief, pre-operative proper localization of the IAN as well as of the local bone is of high importance to prevent IAN injuries. Drill guards attached to the drills or the use of 3D-guided surgery may prevent overpenetration into the bone [98]. Also, the usage of infiltration instead of mandibular posterior block anesthesia can be advocated as the patient will feel pain when the drill or the implant approached the nerve canal [99]. Even so, this technique has not been used in general because the surrounding bone possesses sensitive nerve endings that could cause discomfort during surgery in some cases [100].

Transposition of the IAN before restauration of the posterior mandible with implant supported prostheses could be a treatment option to prevent damage to the IANB. Here, in brief, the mental foramen is corticotomized and repositioned more posteriorly in order to enhance the bone support available for implant placement and to reduce the risk of damage to the IAN [101]. However, such as in all cases for manipulation of the neurovascular bundle, prolonged neurosensory disturbances may occur. Other reported complications are local inflammation leading to osteomyelitis with loss of implant, profuse hemorrhage and mandibular fracture [102–104]. Also the removal of (infected) implants in the posterior mandible after nerve transposition may result in a fracture requiring internal fixation and bone regeneration techniques [104].

6.4.7.2 Prevention and Management of Nerve Damage

Once again, the best way of treatment of nerve damage is prevention. Here, a clear 3D vision of the respective anatomy combining practical knowledge and data from clinical and radiological examination should be mandatory. Determination of the bone mass around the canal of the IAN and at the implants' site and

avoidance of excessive force when approaching the canal are favorable [105]. Next to the usage of 3D-radiology-based intraoperative navigation, the usage of intraoperative periapical radiographs during the drilling sequence will be an inexpensive and somehow reliable tool [106].

Nerve injury during implant placement may be especially suspected after accidental slippage of the drill, implant placement deeper than planned or excessive bleeding out of the drill-hole. Taking radiographs after surgery will help to confirm or exclude if the implant placement caused nerve injury [107]. Next to documentation of intraoperative nerve damage, a neurosensory examination is recommended (Table 6.2) as soon as the anesthetic effect is faded out. Depending on the anesthetic technique and the local anesthetic used, this may take up to several hours [80, 108].

For control of potential inflammatory reactions in the damaged nerve, prescription of steroids or nonsteroidal anti-inflammatory drugs (e.g., 800 mg ibuprofen 3/d for 3 weeks) can be recommended [97]. Any improvement in the patient's condition should be recorded, along with results of a neurosensory examination and the patient's description. If the condition fails to improve within 2 months, referral to a microneurosurgeon (Fig. 6.10) is indicated. Early referral will allow for early management before distant degeneration of the nerve (within 4–6 months of nerve injury) takes place [86].

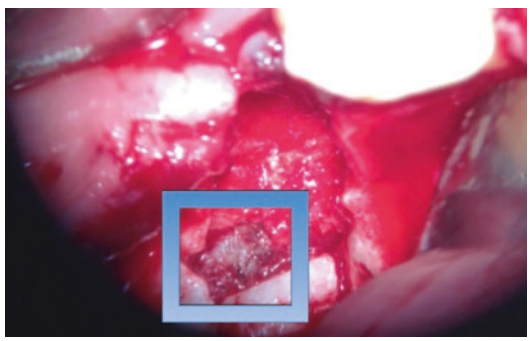


Fig. 6.10 Microsurgical decompression of a damaged inferior alveolar nerve

6.5 Post-surgical Complications

6.5.1 Inflammation and Infection

Postoperative wound infections have a prevalence with figures reaching up to 12%. They are one of the major concerns after implant placement hampering osseointegration and mostly leading to early implant failure (up to 80-fold enhanced risk) [109–111]. Postoperative periimplant infection or chronic stimulation of the nerve can also evoke sensory injury to the IAN even ending up as chronic neuropathy [112]. The treatment itself is complex and infection may even persist until the biomaterial is removed. Traditionally, infections are seen more often in cases affecting the mandible as there is a poor blood supply in the lower jaws together with a thick cortical plate and small cancellous space. In addition, a non-submerged healing phase has shown to decrease the occurrence of postoperative infections. Reasons for this may be that it favors a more aerobic environment together with an enhanced drainage of infectious and inflammatory substances [111]. Prophylactic use of antibiotics has shown limited success as only single-dose oral amoxicillin has shown to be effective at preventing implant failures but did not prevent or reduce the incidence of postoperative infections [113]. As post-surgical infections usually occur several weeks after the surgical procedure, the establishment of a strict early patient follow-up protocol seems necessary to provide adequate and earliest possible treatment. If the infection reached the state of osteomyelitis, its treatment is a long and challenging process requiring long-term systemic antibiosis and—in most of the cases—several surgical approaches [114].

6.5.2 Early Implant Failure

Early implant failure is defined to occur prior prosthetic treatment—e.g., lose implants that have never been integrated into the surrounding bone or implants that experienced early and progressive bone loss (Fig. 6.11)—whereas late fail-

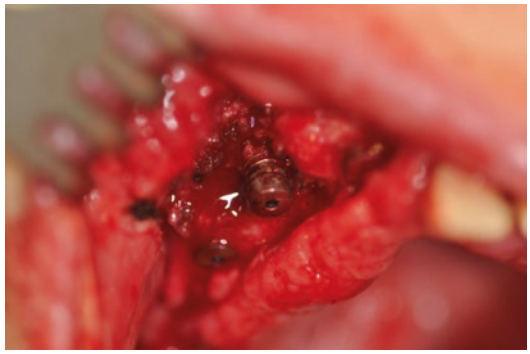


Fig. 6.11 Early implant failure in augmented bone due to lacking osseointegration

ures are seen after prosthetic rehabilitation [115]. Thus, the length of the time period for implants at a risk for early failures is much depending on both choice of surgical loading protocol and when the first radiographic follow-up examination has been performed after prosthesis loading in routine practice [116]. Even if early failures are rather uncommon, incidences between 1 and even 21% are reported [117, 118]. Risk factors are bone quality and implant localization, local and systemic conditions, severity of the surgical trauma as well as surgical technique, infections (most of all history of periodontitis), usage of (immediate) grafting procedures, experience of the surgeon, and smoking habits of the patients [116, 119]. In general, post-surgical foreign body reaction was cited to be a possible reason [120] that may explain the higher rate of early implant failure in cases with additional bone graft usage [118]. Even so, some early failures will occur regardless of operator experience and without any clinically recognizable cause [121]. Progressing bone resorption around the implant was described to be one of the earliest signs of an early implant failure. Accordingly, in a case series of 18 implants that exhibited early bone loss before prosthetic rehabilitation, a timely surgical treatment (curettage, debridement, local antibiosis and re-grafting with bone transplants) could rescue these implants at risk efficiently. Therefore, the author recommended reevaluation visits 4–6 weeks post-implant placement together with diagnostically periapical radiographs to facilitate early treatment [118].

6.5.3 Development of Oroantral and Extra-Oral Fistula

The development of extra-oral fistula is a rather late complication, mostly resulting from peri-implantitis, a chronic inflammatory reaction that causes peri-implant apical radiolucencies. There are a variety of reasons discussed such as overheating of bone during implant drilling procedures, instability, overloading, contamination as well as residual cement and metal particles. Therapy mostly consists of removal of inflamed granulation tissue together with cleaning of the implant surface. In cases of insufficient response to the therapy or severe infections, removal of the implant is advocated to obtain a complete cleaning of the infected tissue including nonviable bone and metallic debris [122].

6.6 Conclusion

Having a safe procedure available for replacement of missing teeth does not mean that everything is safe. If the field of implant dentistry is further advancing offering the clinician various tools and treatment options to facilitate implant placement and to decrease complications in dental implant placement, it has to be taken in mind that the implant is not a screw only and patient inherent variables need significant considerations. Accordingly, a comprehensive medical anamnesis and clinical examination are needed for appropriate case selection and risk assessment. Besides, clinical experience also including the respective implant systems with its advantages and disadvantages as well as radiological exploration should be combined. As for all potential complications, patients' education and informed consent should be aimed for. In addition to an assessment of the respective risk, alternative treatment plans are to be included. Especially in challenging "borderline" situations, a 3D-cross-sectional imaging is advocated for successful implant treatment [123]. So, significant benefits that were also affecting the therapy were seen in the anterior and posterior maxilla when using 2D- and 3D radiology for planning of

dental implant procedures [124]. In terms of perioperative complications (here: Schneiderian membrane perforation, wrong angulation, fenestration, dehiscence, sensory disturbance and infection), 3D radiology enhanced planning of complications with a high degree of prediction when compared to the surgical standard and 2D radiology [125].

References

1. Turrentine FE, Wang H, Simpson VB, Jones RS. Surgical risk factors, morbidity, and mortality in elderly patients. J Am Coll Surg. 2006;203(6):865–77.
2. Pedro RE, De Carli JP, Linden MS, Lima IF, Paranhos LR, Costa MD, et al. Influence of age on factors associated with peri-implant bone loss after prosthetic rehabilitation over osseointegrated implants. J Contemp Dent Pract. 2017;18(1):3–10.
3. Al-Nawas B, Grotz KA. Medically compromised patients in the dental office: demographics and progress in health care. Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz. 2011;54(9):1066–72.
4. Diz P, Scully C, Sanz M. Dental implants in the medically compromised patient. J Dent. 2013;41(3):195–206.
5. Kumar VV, Ebenezer S, Kämmerer PW, Jacob PC, Kuriakose MA, Hedne N, et al. Implants in free fibula flap supporting dental rehabilitation—implant and peri-implant related outcomes of a randomized clinical trial. J Craniomaxillofac Surg. 2016;44(11):1849–58.
6. Schiegnitz E, Al-Nawas B, Kämmerer PW, Grötz KA. Dental implants in irradiated patients: which factors influence implant survival? Clin Oral Investig. 2015;19(7):1691–2.
7. Jimenez M, Hu FB, Marino M, Li Y, Joshipura KJ. Type 2 diabetes mellitus and 20 year incidence of periodontitis and tooth loss. Diabetes Res Clin Pract. 2012;98(3):494–500.
8. Gomez-Moreno G, Aguilar-Salvatierra A, Rubio Roldan J, Guardia J, Gargallo J, Calvo-Guirado JL. Peri-implant evaluation in type 2 diabetes mellitus patients: a 3-year study. Clin Oral Implants Res. 2015;26(9):1031–5.
9. Oates TW Jr, Galloway P, Alexander P, Vargas Green A, Huynh-Ba G, Feine J, et al. The effects of elevated hemoglobin A(1c) in patients with type 2 diabetes mellitus on dental implants: survival and stability at one year. J Am Dent Assoc. 2014;145(12):1218–26.
10. Monje A, Catena A, Borgnakke WS. Association between diabetes mellitus/hyperglycaemia and peri-implant diseases: systematic review and meta-analysis. J Clin Periodontol. 2017;44(6):636–48.
11. Wiltfang J, Naujokat H, Bormann KH, Jakobs W, Wiegner J-U. AWMF S3-Leitlinie Zahnimplantate bei Diabetes mellitus. 2016.
12. Albandar JM, Streckfus CF, Adesanya MR, Winn DM. Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. J Periodontol. 2000;71:1874–81.
13. Pereira ML, Carvalho JC, Peres F, Fernandes MH. Simultaneous effects of nicotine, acrolein, and acetaldehyde on osteogenic-induced bone marrow cells cultured on plasma-sprayed titanium implants. Int J Oral Maxillofac Implants. 2010;25:112–22.
14. Chen H, Liu N, Xu X, Qu X, Lu E. Smoking, radiotherapy, diabetes and osteoporosis as risk factors for dental implant failure: a meta-analysis. PLoS One. 2013;8:e71955.
15. Chrcanovic BR, Albrektsson T, Wennerberg A. Smoking and dental implants: a systematic review and meta-analysis. J Dent. 2015;43:487–98.
16. Alsaadi G, Quirynen M, Komarek A, van Steenberghe D. Impact of local and systemic factors on the incidence of oral implant failures, up to abutment connection. J Clin Periodontol. 2007;34:610–7.
17. Temmerman A, Rasmusson L, Kubler A, Thor A, Quirynen M. An open, prospective, non-randomized, controlled, multicentre study to evaluate the clinical outcome of implant treatment in women over 60 years of age with osteoporosis/osteopenia: 1-year results. Clin Oral Implants Res. 2017;28(1):95–102.
18. Wagner F, Schuder K, Hof M, Heuberer S, Seemann R, Dvorak G. Does osteoporosis influence the marginal peri-implant bone level in female patients? A cross-sectional study in a matched collective. Clin Implant Dent Relat Res. 2017;19(4):616–23.
19. de Medeiros F, Kudo GAH, Leme BG, Saraiva PP, Verri FR, Honorio HM, et al. Dental implants in patients with osteoporosis: a systematic review with meta-analysis. Int J Oral Maxillofac Surg. 2018;47(4):480–91.
20. Kotsakis GA, Ioannou AL, Hinrichs JE, Romanos GE. A systematic review of observational studies evaluating implant placement in the maxillary jaws of medically compromised patients. Clin Implant Dent Relat Res. 2015;17(3):598–609.
21. Neuprez A, Coste S, Rompen E, Crielaard JM, Reginster JY. Osteonecrosis of the jaw in a male osteoporotic patient treated with denosumab. Osteoporos Int. 2014;25(1):393–5.
22. Kanis JA, McCloskey EV, Johansson H, Cooper C, Rizzoli R, Reginster JY, et al. European guidance for the diagnosis and management of osteoporosis in postmenopausal women. Osteoporos Int. 2013;24(1):23–57.
23. Fernandez Ayora A, Herion F, Rompen E, Reginster JY, Magremanne M, Lambert F. Dramatic osteonecrosis of the jaw associated with oral bisphosphonates, periodontitis, and dental implant removal. J Clin Periodontol. 2015;42(2):190–5.
24. Grötz KA, Walter C, Al-Nawas B, Haßfeld S, Sader R, Ullner M. S3-Leitlinie: Zahnimplantate bei medi-

kamentöser Behandlung mit Knochenantiresorptiva (inkl. Bisphosphonate). AWMF online. 2016.
25. Krennmair S, Weinlander M, Forstner T, Krennmair G, Stimmelmayr M. Factors affecting peri-implant bone resorption in four implant supported mandibular full-arch restorations: a 3-year prospective study. J Clin Periodontol. 2016;43(1):92–101.
26. Winnett B, Tenenbaum HC, Ganss B, Jokstad A. Perioperative use of non-steroidal anti-inflammatory drugs might impair dental implant osseointegration. Clin Oral Implants Res. 2016;27(2):e1–7.
27. Wu X, Al-Abedalla K, Abi-Nader S, Daniel NG, Nicolau B, Tamimi F. Proton pump inhibitors and the risk of osseointegrated dental implant failure: a cohort study. Clin Implant Dent Relat Res. 2017;19(2):222–32.
28. Lamas Pelayo J, Penarrocha Diago M, Marti Bowen E, Penarrocha DM. Intraoperative complications during oral implantology. Med Oral Patol Oral Cir Bucal. 2008;13(4):E239–43.
29. Kämmerer PW, Wolf JM, Frerich B, Ottl P. Correction of malpositioned dental implants via LeFort osteotomy. J Oral Maxillofac Surg. 2018 [submitted].
30. Misch CE, Bidez MW. Implant-protected occlusion: a biomechanical rationale. Compendium. 1994;15:1330, 2, 4 passim; quiz 44.
31. Cariati P, Fernandez-Solis J, Marin-Fernandez AB, Valencia-Laseca A, Monsalve-Iglesias F. Accidental displacement of a dental implant into the sublingual space: a case report. J Clin Exp Dent. 2016;8(4):e459–e61.
32. Lee SC, Jeong CH, Im HY, Kim SY, Ryu JY, Yeom HY, et al. Displacement of dental implants into the focal osteoporotic bone marrow defect: a report of three cases. J Korean Assoc Oral Maxillofac Surg. 2013;39(2):94–9.
33. Chan HL, Benavides E, Yeh CY, Fu JH, Rudek IE, Wang HL. Risk assessment of lingual plate perforation in posterior mandibular region: a virtual implant placement study using cone-beam computed tomography. J Periodontol. 2011;82(1):129–35.
34. Watanabe H, Mohammad Abdul M, Kurabayashi T, Aoki H. Mandible size and morphology determined with CT on a premise of dental implant operation. Surg Radiol Anat. 2010;32(4):343–9.
35. Berberi A, Le Breton G, Mani J, Woimant H, Nasseh I. Lingual paresthesia following surgical placement of implants: report of a case. Int J Oral Maxillofac Implants. 1993;8:580–2.
36. Schneider D, Marquardt P, Zwahlen M, Jung RE. A systematic review on the accuracy and the clinical outcome of computer-guided template-based implant dentistry. Clin Oral Implants Res. 2009;20(Suppl. 4):73–86.
37. Damlar I. Disappearance of a dental implant after migration into the maxillary sinus: an unusual case. J Korean Assoc Oral Maxillofac Surg. 2015;41:278–80.
38. Galindo-Moreno P, Padial-Molina M, Sánchez-Fernández E, Hernández-Cortés P, Wang HL, O'Valle F. Dental implant migration in grafted maxillary sinus. Implant Dent. 2011;20:400–5.
39. González-García A, González-García J, Diniz-Freitas M, García-García A, Bullón P. Accidental displacement and migration of endosseous implants into adjacent craniofacial structures: a review and update. Med Oral Patol Oral Cir Bucal. 2012;17:e769–74.
40. Flanagan D. Arterial supply of maxillary sinus and potential for bleeding complication during lateral approach sinus elevation. Implant Dent. 2005;14:336–8.
41. Temmerman A, Hertele S, Teughels W, Dekeyser C, Jacobs R, Quirynen M. Are panoramic images reliable in planning sinus augmentation procedures? Clin Oral Implants Res. 2011;22(2):189–94.
42. Lustig JP, London D, Dor BL, Yanko R. Ultrasound identification and quantitative measurement of blood supply to the anterior part of the mandible. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2003;96(5):625–9.
43. Law C, Alam P, Borumandi F. Floor-of-mouth hematoma following dental implant placement: literature review and case presentation. J Oral Maxillofac Surg. 2017;75(11):2340–6.
44. Kilic E, Doganay S, Ulu M, Celebi N, Yikilmaz A, Alkan A. Determination of lingual vascular canals in the interforaminal region before implant surgery to prevent life-threatening bleeding complications. Clin Oral Implants Res. 2014;25(2):e90–3.
45. Dubois L, De Lange J, Baas E, Van Ingen J. Excessive bleeding in the floor of the mouth after endosseus implant placement: a report of two cases. Int J Oral Maxillofac Surg. 2010;39:412–5.
46. Del Castillo-Pardo de Vera JL, Lopez-Arcas Calleja JM, Burgueno-Garcia M. Hematoma of the floor of the mouth and airway obstruction during mandibular dental implant placement: a case report. Oral Maxillofac Surg. 2008;12:223–6.
47. Isaacson T. Sublingual haematoma formation during immediate placement of mandibular endosseous implants. J Am Dent Assoc. 2004;135(2):168–72.
48. Givol N, Chaushu G, Halamish-Shani T, Taicher S. Emergency tracheostomy following life-threatening hemorrhage in the floor of the mouth during immediate implant placement in the mandibular canine region. J Periodontol. 2000;71(12):1893–5.
49. Remonda L, Schroth G, Caversaccio M, Ladrach K, Lovblad KO, Zbaren P, et al. Endovascular treatment of acute and subacute hemorrhage in the head and neck. Arch Otolaryngol Head Neck Surg. 2000;126:1255–62.
50. Bavitz JB, Harn SD, Homze EJ. Arterial supply to the floor of the mouth and lingual gingiva. Oral Surg Oral Med Oral Pathol. 1994;77:232–5.
51. Gonzalez-Garcia R, Schoendorff G, Munoz-Guerra MF, Rodriguez-Campo FJ, Naval-Gias L, Sastre-Perez J. Upper airway obstruction by sublingual

hematoma: a complication of anticoagulation therapy with acenocoumarol. Am J Otolaryngol. 2006;2:129–32.
52. Cameron SM, Whitlock WL, Tabor MS. Foreign body aspiration in dentistry: a review. J Am Dent Assoc. 1996;127:1224–9.
53. Jain A, Baliga SD. Accidental implant screwdriver ingestion: a rare complication during implant placement. J Dent (Tehran). 2014;11(6):711–4.
54. Barkmeier WW, Cooley RL, Abrams H. Prevention of swallowing or aspiration of foreign objects. J Am Dent Assoc. 1978;97(3):473–6.
55. Knowles JE. Inhalation of dental plates: a hazard of radiolucent materials. J Laryngol Otol. 1991;105:681–2.
56. Summers RB. A new concept in maxillary implant surgery: the osteotome technique. Compendium. 1994;15(152):4–6.
57. Cavicchia F, Bravi F, Petrelli G. Localized augmentation of the maxillary sinus floor through a coronal approach for the placement of implants. Int J Periodontics Restorative Dent. 2001;21:475–85.
58. Zijderveld SA, van den Bergh JP, Schulten EA, ten Bruggenkate CM. Anatomical and surgical findings and complications in 100 consecutive maxillary sinus floor elevation procedures. J Oral Maxillofac Surg. 2008;66:1426–38.
59. Doud Galli SK, Lebowitz RA, Giacchi RJ, Glickman R, Jacobs JB. Chronic sinusitis complicating sinus lift surgery. Am J Rhinol. 2001;15:181–6.
60. Saker M, Ogle O. Benign paroxysmal positional vertigo subsequent to sinus lift via closed technique. J Oral Maxillofac Surg. 2005;63:1385–7.
61. Niederquell B, Brennan PA, Dau M, Moergel M, Frerich B, Kämmerer PW. Bilateral postoperative cyst after maxillary sinus surgery—report of a case and systematic review of the literature. Case Rep Dent. 2016;2016:6263248.
62. Kayabasoglu G, Nacar A, Altundag A, Cayonu M, Muhtarogullari M, Cingi C. A retrospective analysis of the relationship between rhinosinusitis and sinus lift dental implantation. Head Face Med. 2014;10:53.
63. Timmenga NM, Raghoebar GM, Boering G, van Weissenbruch R. Maxillary sinus function after sinus lifts for the insertion of dental implants. J Oral Maxillofac Surg. 1997;55(9):936–9; discussion 40.
64. Raghoebar GM, van Weissenbruch R, Vissink A. Rhino-sinusitis related to endosseous implants extending into the nasal cavity. A case report. Int J Oral Maxillofac Surg. 2004;33(3):312–4.
65. Jung JH, Choi BH, Jeong SM, Li J, Lee SH, Lee HJ. A retrospective study of the effects on sinus complications of exposing dental implants to the maxillary sinus cavity. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2007;103(5):623–5.
66. Zhong W, Chen B, Liang X, Ma G. Experimental study on penetration of dental implants into the maxillary sinus in different depths. J Appl Oral Sci. 2013;21(6):560–6.
67. Quiney RE, Brimble E, Hodge M. Maxillary sinusitis from dental osseointegrated implants. J Laryngol Otol. 1990;104(4):333–4.
68. Anzalone JV, Vastardis S. Oroantral communication as an osteotome sinus elevation complication. J Oral Implantol. 2010;36(3):231–7.
69. Troeltzsch M, Pache C, Troeltzsch M, Kaeppler G, Ehrenfeld M, Otto S, et al. Etiology and clinical characteristics of symptomatic unilateral maxillary sinusitis: a review of 174 cases. J Craniomaxillofac Surg. 2015;43(8):1522–9.
70. Manor Y, Garfunkel AA. Brain abscess following dental implant placement via crestal sinus lift - a case report. Eur J Oral Implantol. 2018;11(1):113–7.
71. Puglisi S, Privitera S, Maiolino L, Serra A, Garotta M, Blandino G, et al. Bacteriological findings and antimicrobial resistance in odontogenic and non-odontogenic chronic maxillary sinusitis. J Med Microbiol. 2011;60(Pt 9):1353–9.
72. Little RE, Long CM, Loehrl TA, Poetker DM. Odontogenic sinusitis: a review of the current literature. Laryngosc Investig Otolaryngol. 2018;3(2):110–4.
73. Karlis V, Bae RD, Glickman RS. Mandibular fracture as a complication of inferior alveolar nerve transposition and placement of endosseous implants: a case report. Implant Dent. 2003;12(3):211–6.
74. Tolman DE, Keller EE. Management of mandibular fractures in patients with endosseous implants. Int J Oral Maxillofac Implants. 1991;6(4):427–36.
75. Boffano P, Roccia F, Gallesio C, Berrone S. Pathological mandibular fractures: a review of the literature of the last two decades. Dent Traumatol. 2013;29(3):185–96.
76. Raghoebar GM, Stellingsma K, Batenburg RH, Vissink A. Etiology and management of mandibular fractures associated with endosteal implants in the atrophic mandible. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2000;89(5):553–9.
77. Bartling R, Freeman K, Kraut RA. The incidence of altered sensation of the mental nerve after mandibular implant placement. J Oral Maxillofac Surg. 1999;57:1408–12.
78. Ellies LG. Altered sensation following mandibular-vimplant surgery: a retrospective study. J Prosthet Dent. 1992;68:664–71.
79. Tay AB, Zuniga JR. Clinical characteristics of trigeminal nerve injury referrals to a university centre. Int J Oral Maxillofac Surg. 2007;36(10):922–7.
80. Kämmerer PW, Adubae A, Buttchereit I, Thiem DGE, Daubländer M, Frerich B. Prospective clinical study comparing intraligamentary anesthesia and inferior alveolar nerve block for extraction of posterior mandibular teeth. Clin Oral Investig. 2018;22:1469–75.
81. Dau M, Buttchereit I, Ganz C, Frerich B, Anisimova EN, Daubländer M, et al. Influence of needle bevel design on injection pain and needle deformation in dental local infiltration anaesthesia—random-

ized clinical trial. Int J Oral Maxillofac Surg. 2017;46(11):1484–9.
82. Hegedus F, Diecidue RJ. Trigeminal nerve injuries after mandibular implant placement—practical knowledge for clinicians. Int J Oral Maxillofac Implants. 2006;21:111–6.
83. Juodzbalys G, Wang HL, Sabalys G. Injury of the inferior alveolar nerve during implant placement: a literature review. J Oral Maxillofac Res. 2011;2(1):e1.
84. Leckel M, Kress B, Schmitter M. Neuropathic pain resulting from implant placement: case report and diagnostic conclusions. J Oral Rehabil. 2009;36(7):543–6.
85. Abarca M, van Steenberghe D, Malevez C, De Ridder J, Jacobs R. Neurosensory disturbances after immediate loading of implants in the anterior mandible: an initial questionnaire approach followed by a psychophysical assessment. Clin Oral Investig. 2006;10(4):269–77.
86. Alhassani AA, AlGhamdi AST. Inferior alveolar nerve injury in implant dentistry: diagnosis, causes, prevention, and management. J Oral Implantol. 2010;36(5):401–7.
87. Seddon HJ, Medawar PB, Smith H. Rate of regeneration of peripheral nerves in man. J Physiol. 1943;102:191–215.
88. Ghali GE, Epker BN. Clinical neurosensory testing: practical applications. J Oral Maxillofac Surg. 1989;47:1074–8.
89. Poort LJ, van Neck JW, van der Wal KG. Sensory testing of inferior alveolar nerve injuries: a review of methods used in prospective studies. J Oral Maxillofac Surg. 2009;67:292–300.
90. Kim ST, Hu KS, Song WC, Kang MK, Park HD, Kim HJ. Location of the mandibular canal and the topography of its neurovascular structures. J Craniofac Surg. 2009;20:936–9.
91. Nortje CJ, Farman AG, Grotepass FW. Variations in the normal anatomy of the inferior dental (mandibular) canal: a retrospective study of panoramic radiographs from 3612 routine dental patients. Br J Oral Surg. 1977;15:55–63.
92. Grover PS, Lorton L. Bifid mandibular nerve as a possible cause of inadequate anesthesia in the mandible. J Oral Maxillofac Surg. 1983;41:177–9.
93. Langlais RP, Broadus R, Glass BJ. Bifid mandibular canals in panoramic radiographs. J Am Dent Assoc. 1985;110:923–6.
94. Vazquez L, Saulacic N, Belser U, Bernard JP. Efficacy of panoramic radiographs in the preoperative planning of posterior mandibular implants: a prospective clinical study of 1527 consecutively treated patients. Clin Oral Implants Res. 2008;19:81–5.
95. Sammartino G, Marenzi G, Citarella R, Ciccarelli R, Wang HL. Analysis of the occlusal stress transmitted to the inferior alveolar nerve by an osseointegrated threaded fixture. J Periodontol. 2008;79(9):1735–44.
96. Anderson LC, Kosinski TF, Mentag PJ. A review of the intraosseous course of the nerves of the mandible. J Oral Implantol. 1991;17:394–403.
97. Kraut RA, Chahal O. Management of patients with trigeminal nerve injuries after mandibular implant placement. J Am Dent Assoc. 2002;133:1351–4.
98. Worthington P. Injury to the inferior alveolar nerve during implant placement: a formula for protection of the patient and clinician. Int J Oral Maxillofac Implants. 2004;19:731–4.
99. Heller AA, WEn S. Alternative to the inferior alveolar nerve block anesthesia when placing mandibular dental implants posterior to the mental foramen. J Oral Implantol. 2001;27:127–33.
100. Heraud J, Orofino J, Trub M, Mei N. Electrophysiologic evidence showing the existence of sensory receptors within the alveolar bone in anesthetized cats. Int J Oral Maxillofac Implants. 1996;11(6):800–5.
101. Jensen O, Nock D. Inferior alveolar nerve reposition in conjunction with placement of osseointegrated implants: a case report. Oral Surg Oral Med Oral Pathol Oral Radiol. 1987;63:263–8.
102. Luna AH, Passeri LA, de Moraes M, et al. Endosseous implant placement in conjunction with inferior alveolar nerve transposition: a report of an unusual complication and surgical management. Int J Oral Maxillofac Implants. 2008;23:133–6.
103. Rosenquist B. Implant placement in combination with nerve transpositioning: experience with the first 100 cases. Int J Oral Maxillofac Implants. 1994;9:522–31.
104. Da Costa Ribeiro R, Luna AH, Sverzut CE, Sverzut AT. Failure of osseointegrated dental implant after alveolar nerve transposition: a report of an unusual complication and surgical management. Implant Dent. 2017;26(4):645–8.
105. Basa O, Dilek OC. Assessment of the risk of perforation of the mandibular canal by implant drill using density and thickness parameters. Gerodontology. 2011;28(3):213–20.
106. Burstein J, Mastin C, Le B. Avoiding injury to the inferior alveolar nerve by routine use of intraoperative radiographs during implant placement. J Oral Implantol. 2008;34(1):34–8.
107. Khawaja N, Renton T. Case studies on implant removal influencing the resolution of inferior alveolar nerve injury. Br Dent J. 2009;206:365–70.
108. Kämmerer PW, Palarie V, Daubländer M, Bicer C, Shabazfar N, Brüllmann D, et al. Comparison of 4% articaine with epinephrine (1:100.000) and without epinephrine in inferior alveolar block for tooth extraction—double blind, randomized clinical trial of anesthetic efficacy. Oral Surg Oral Med Oral Pathol Oral Radiol. 2012;113(4):495–9.
109. Camps-Font O, Figueiredo R, Valmaseda-Castellon E, Gay-Escoda C. Postoperative infections after dental implant placement: prevalence, clinical features, and treatment. Implant Dent. 2015;24(6):713–9.

110. Anitua E, Aguirre JJ, Gorosabel A, Barrio P, Errazquin JM, Roman P, et al. A multicentre placebo-controlled randomised clinical trial of antibiotic prophylaxis for placement of single dental implants. Eur J Oral Implantol. 2009;2(4):283–92.
111. Figueiredo R, Camps-Font O, Valmaseda-Castellon E, Gay-Escoda C. Risk factors for postoperative infections after dental implant placement: a case-control study. J Oral Maxillofac Surg. 2015;73(12):2312–8.
112. Elian N, Mitsias M, Eskow R, Jalbout ZN, Cho SC, Froum S, et al. Unexpected return of sensation following 4.5 years of paresthesia: case report. Implant Dent. 2005;14(4):364–7.
113. Rodriguez Sanchez F, Rodriguez Andres C, Arteagoitia I. Which antibiotic regimen prevents implant failure or infection after dental implant surgery? A systematic review and meta-analysis. J Craniomaxillofac Surg. 2018;46(4):722–36.
114. Schlund M, Raoul G, Ferri J, Nicot R. Mandibular osteomyelitis following implant placement. J Oral Maxillofac Surg. 2017;75(12):2560 e1–7.
115. Esposito M, Hirsch JM, Lekholm U, Thomsen P. Biological factors contributing to failures of osseointegrated oral implants. (I). Success criteria and epidemiology. Eur J Oral Sci. 1998;106:527–51.
116. Antoun H, Karouni M, Abitbol J, Zouiten O, Jemt T. A retrospective study on 1592 consecutively performed operations in one private referral clinic. Part I: Early inflammation and early implant failures. Clin Implant Dent Relat Res. 2017;19:404–12.
117. Chrcanovic BR, Albrektsson T, Wennerberg A. Reasons for failures of oral implants. J Oral Rehabil. 2014;41(6):443–76.
118. AlGhamdi AS. Successful treatment of early implant failure: a case series. Clin Implant Dent Relat Res. 2012;14(3):380–7.
119. Esposito M, Hirsch JM, Lekholm U, Thomsen P. Biological factors contributing to failures of osseointegrated oral implants. (II). Etiopathogenesis. Eur J Oral Sci. 1998;106(3):721–64.
120. Pinholt EM, Haanaes HR, Roervik M, Donath K, Bang G. Alveolar ridge augmentation by osteoinductive materials in goats. Scand J Dent Res. 1992;100(6):361–5.
121. Deas DE, Mikotowicz JJ, Mackey SA, Moritz AJ. Implant failure with spontaneous rapid exfoliation: case reports. Implant Dent. 2002;11(3):235–42.
122. Fujioka M, Oka K, Kitamura R, Yakabe A, Endoh H. Extra-oral fistula caused by a dental implant. J Oral Implantol. 2011;37(4):477–9.
123. Harris D, Horner K, Grondahl K, Jacobs R, Helmrot E, Benic GI, et al. E.A.O. guidelines for the use of diagnostic imaging in implant dentistry 2011. A consensus workshop organized by the European Association for Osseointegration at the Medical University of Warsaw. Clin Oral Implants Res. 2012;23(11):1243–53.
124. Dau M, Edalatpour A, Schulze R, Al-Nawas B, Alshihri A, Kämmerer PW. Presurgical evaluation of bony implant sites using panoramic radiography and cone beam computed tomography-influence of medical education. Dentomaxillofac Radiol. 2017;46(2):20160081.
125. Guerrero ME, Noriega J, Jacobs R. Preoperative implant planning considering alveolar bone grafting needs and complication prediction using panoramic versus CBCT images. Imaging Sci Dent. 2014;44(3):213–20.

Complications of TMJ Surgery

7

Tetsu Takahashi

Contents

7.1 Introduction

Nowadays, although indications of temporomandibular joint open arthroplasty surgery seem to be reduced since minimally invasive temporomandibular surgeries such as arthroscopy or arthrocentesis are more popular compared with conventional open temporomandibular joint (TMJ) surgeries. On the other hand, the total joint replacement of TMJ draws a considerable attention as a last resort in the surgical management of end-stage TMJ diseases. In this chapter, complications of TMJ surgery from minimally invasive surgeries to total joint replacement of TMJ are discussed.

T. Takahashi (✉)
Division of Oral and Maxillofacial Surgery, Department of Oral Medicine and Surgery, Tohoku University Graduate School of Dentistry, Sendai, Miyagi, Japan
e-mail: tetsu@dent.tohoku.ac.jp

7.2 Arthrocentesis

7.2.1 Procedure

In 1991, Nitzan et al. [1] described double-puncture arthrocentesis of the TMJ, in which two needles are introduced into the upper joint space

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_7

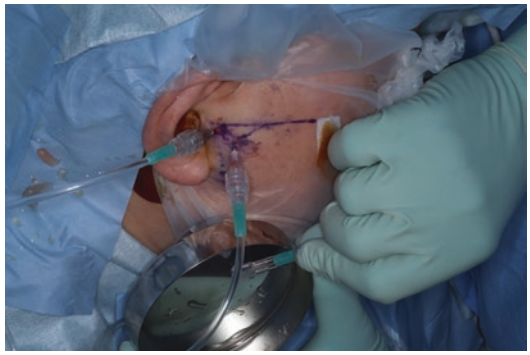


Fig. 7.1 Position of the two cannulas entering the upper joint compartment for an arthrocentesis

(Fig. 7.1). Briefly, two 21-needle applicators are inserted into two guide points and the upper joint space was irrigated with approximately 150 mL of ringer lactate. Finally, sodium hyaluronate and/or corticosteroid was injected into the joint space. The main purpose of this procedure is to flush the upper joint space, lysis and lavage, eliminate inflammatory mediators in the synovial fluid, release disc adhesions, reduce pain, and mobilize the joint.

7.2.2 Complications of Arthrocentesis

The complication rate for TMJ arthrocentesis has not yet been defined, but it is considered to be lower than that for TMJ arthroscopy [2, 3]. Factors related to the complications include the anatomy of the joint, its relation to the surrounding structures, and the method used to penetrate into the upper compartment of the TMJ. Most of the complications of arthrocentesis are minor, and permanent complication rate is few. However, the following complications should be considered:

1. Temporary local swelling
   Temporary swelling of the preauricular tissue is quite often seen due to the extravasation of irrigation fluid. It resolves within 1 day.
2. Otologic complications
   Perforation of external auditory canal, blood clots in the external auditory canal, tympanic membrane injuries, partial hearing loss, a feeling of a blocked ear, and dizziness can be occurred because of the close proximity of the middle ear and cartilaginous ear canal wall of the TMJ. However, those otologic complications are all minor as compared to the arthroscopic surgery.
3. Facial paralysis
   Facial paralysis sometimes occurs due to the infiltration of local anesthetic agent such as lidocaine.
4. Lingual and inferior nerve anesthesia
5. Hemorrhage
6. Tachycardia
7. Syncope
8. Dizziness
9. Severe pain

The above-mentioned complications are all transitory and spontaneously subside within a day or two. Among them, pain and facial paralysis were most common. The only major complication reported has been one case of an extradural hematoma [4].

7.3 Arthroscopic Surgery

TMJ arthroscopic surgery was introduced by Ohnishi in 1974 [5]. Since then, many lines of evidence demonstrated that this procedure was a minimally invasive and safe surgical procedure for the treatment of certain types of internal derangement and osteoarthritis of the TMJ. However, as many advanced surgical procedures have been introduced, more complications have been reported.

7.3.1 Procedures

Usually, surgery is performed under general anesthesia. A tragocanthal line is first established, and the penetration point of the cannula is set at 10 mm anterior to the midtragus and 5 mm caudally to the tragocanthal line. Before penetrating the TMJ capsule, 2 mL of lidocaine is injected into the superior joint capsule, using a 22-gauge needle, to expand the structure. A 5 mL syringe is used so that backpressure can be

felt when the joint space is entered. Epinephrine is not recommended to use for this purpose because it may affect visualization of the synovial vasculature. Puncture is made using a sharp trocar, which introduces a cannula into the superior joint space, using a standard inferio-lateral approach, to a depth of 25 mm. Once penetrating the joint, the sharp trocar is removed, and blunt obturator is inserted to separate the soft tissues within the TMJ. An 18-gauge needle is inserted 5 mm anteriorly and 5 mm caudally from the puncture point of the working cannula as an outflow canal. Diagnostic arthroscopy can be performed by a single puncture technique. However, for the operative arthroscopy, double puncture is necessary (Fig. 7.2). A second puncture is approximately 27 mm anterior and 7 mm inferior to the midpoint of the tragus, which is just anterior and superior to the peak of the articular eminence. A triangulation technique for the second puncture enabled the safety arthroscopic surgical access to the upper joint compartment (Fig. 7.3). The procedure of anterior release and posterior cauterization was introduced for the reliable lysis of adhesions and disc mobilization under arthroscopic direct vision.

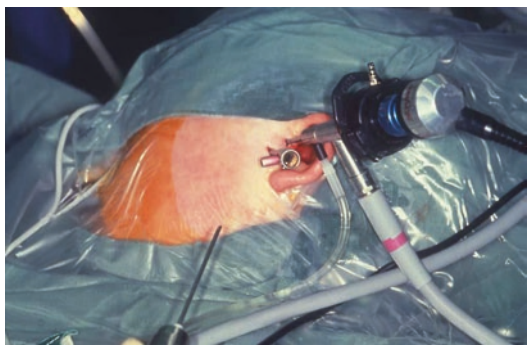


Fig. 7.2 Two cannulas are introduced into the superior joint compartment

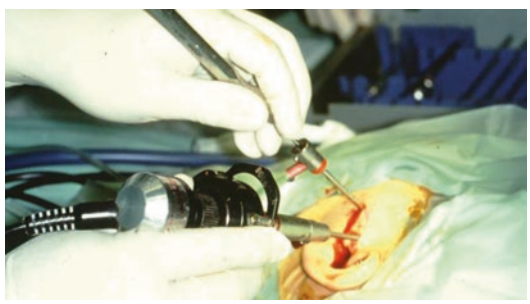


Fig. 7.3 Triangulation technique for arthroscopic surgery

7.3.2 Complications of Arthroscopic Surgery

Although TMJ arthroscopic surgery is a safe surgical procedure, complications could occur according to its surgical procedures. Generally, the more the complicated procedures, the more severe the complications. Therefore, care should be taken during as well as after the arthroscopic surgery. Complications specific to the TMJ arthroscopic surgery include otologic ones and perforations of the middle cranial fossa, and instrument breakage [6, 7]. Most of them took place during or immediately after the surgical procedure.

1. Otologic complications

   Otologic complications detected at perforations includes blood clots in external auditory canal, laceration of external auditory canal, perforation of tympanic membrane, and laceration of external auditory canal. Most of those complications are recognized during the procedure because of a sudden leak of irrigation fluid from the external auditory canal. As the first-aid treatment during the operation, gauze coated with an antibiotic ointment should be packed tightly in the external auditory canal. The gauze would be removed on postoperative day 4. The patient should be referred to otolaryngologists for treatment by irrigation of the external auditory canal and to prescribe antibiotics or hydrocortisone suspension ear drops. The laceration area would be completely healed within a few weeks postoperatively. When tympanic membrane perforation occurs, similar treatment is necessary. Usually, hearing would completely recover, and the tympanic membrane would heal within 1 month after the operation.

   Otologic complications after operation include partial healing loss, ear fullness, and vertigo. When signs and symptoms are found, patients should be referred to otolaryngologists. Most of the partial healing loss occurs

due to postoperative edema of the external auditory canal or middle ear. Usually, the symptoms disappear within 1 month. The case of vertigo should be prescribed an antidinic by an otolaryngologist. Usually, the symptom disappears within a few months.

2. Nerve Injury

Nerve injuries, including injuries of cranial nerve V (trigeminal nerve), VII (facial nerve), and auditory nerve have been reported. The complication rate of the nerve injury is between 0.75% and 3.9% [7].

(a) Cranial nerve V (Trigeminal nerve)

Temporary hypesthesia in the region of the auriculotemporal nerve can be seen. Furthermore, as was the case with arthrocentesis, lingual and/or inferior nerve anesthesia can be seen. Temporary hypesthesia is mainly due to the extravasation of the irrigation fluid.

(b) Cranial nerve VII (Facial nerve)

Temporary paralysis of the zygomatic branch of the facial nerve can be observed. Tsuyama et al. demonstrated that facial nerve deficit was observed in 5 cases in the 301 cases (1.7%) who underwent arthroscopic surgery using the triangulation technique [6]. In most cases, neurologic symptoms were temporary and recovered in a few days. To avoid nerve injuries, the smooth interface of the cannula system, along with a rotational motion on insertion, and careful surgical procures are required. Moreover, excessive extravasation of the irrigation fluid around the nerve tissue should be avoided. To circumvent facial nerve injury, surgeons should perform a safe and gentle puncture and check for continuous flow of the irrigation fluid to avoid excessive irrigation fluid pressure during the surgical procedure.

3. Bleeding

Although most of the previous study reported that there was no direct injury of the superficial temporal vessels by the posterolateral approach using the canthus–tragus line as a guide, a damage to the superficial temporal vessels could occur during the introduction of the cannula into the upper joint compartment. MaCain et al. reported that only one case required arthrotomy for hemostasis in a total of 4831 joints [8]. Although the complication rate of bleeding is low, we should consider the bleeding during arthroscopic procedure. Most cases of the bleeding during arthroscopic procedures can be controlled with vasoconstriction. Placing patient to occlusion often stops bleeding. For severe bleeding, electrocoagulation or insufflation of a Fogarty catheter could be useful. In addition to the bleeding, arteriovenous fistula, pseudoaneurysm, and hematoma are also reported. Those usually appear after a long follow-up period.

4. Infections

Infections after TMJ arthroscopic surgery are very rare ranging from 0% to 1%. However, some case reports showed otitis media, joint infection, and infratemporal space infection.

5. Penetration to the middle cranial fossa

Penetration of the middle cranial fossa has been reported [9]. Sugisaki et al. demonstrated that the mean thickness of the deepest point of the fossa is reported to be 0.9 mm [10]. Therefore, injury to middle cranial fossa can easily occur. Therefore, great care should be taken in the manipulation of instruments near the deepest point of the mandibular fossa. The middle cranial fossa injuries are rare complications. However, if it occurs, it may be a life-threatening complication.

6. Instrumental breakage

Another specific complication of arthroscopic surgery is instrumental breakage because the instruments for TMJ arthroscopic surgery are very small and fine. Therefore, the potential risk of instrumental breakage may be increased by forced or repetitive instrumentation. Specifically, the basket forceps used for a punch biopsy are extremely small and fragile. Once instrumental breakage happens, open surgery is necessary for the removal of instruments.

7.4 Temporomandibular Joint Arthroplasty

The surgical approaches to the TMJ are basically classified as preauricular, endaural, and postauricular. The choice of approach is usually a matter of surgeon's preference. Cosmetic considerations may also influence the choice of approach.

7.4.1 Surgical Anatomic Considerations

The temporoparietal fascia is the most superficial facial layer beneath the subcutaneous fat. It is the lateral extension of the galea and is continuous with the superficial musculoaponeurotic system (SMAS) layer (Fig. 7.4) [11]. It is frequently called the superficial temporalis fascia or the supratympanic SMAS. The blood vessels of the scalp, such as the superficial temporal vessels, run along its superficial aspect close to the subcutaneous fat. On the other hand, the motor nerves, such as temporal branch of the facial nerve, run on the deep surface of the temporoparietal fascia. The temporalis fascia is a tough, fibrous connective tissue structure, substantially thicker than the overlying superficial fascia. Inferiorly, at a variable distance, the fascia splits into two well-defined layers (Fig. 7.4). The outer layer is attached to the lateral margin of the superior border of the zygomatic arch and the inner layer to the medial margin. The zygomatico-orbital branch of the temporal artery and zygomaticotemporal branch of the maxillary nerve are located between the fascial layers.

The temporal nerve branches lie closest to the joint and are the most commonly injured branches during surgery. These nerves are located in a condensation of superficial fascial, temporalis fascia, and periosteum as they cross the zygomatic arch. The most posterior temporal branches lie anteriorly to the post-glenoid tubercle. Their location was measured by Al-Kayat and Bramley as 3.5 ± 0.8 cm from the anterior margin of the bony external auditory canal. Therefore, the two potential sources of facial nerve injury are dissection anterior to the posterior glenoid tubercle, where the temporal branches cross the arch, and aggressive retraction the inferior margin of the flap where the main trunk and temporofacial division are located. To avoid the temporal branch of facial nerve, a flap should be carefully raised including the temporal branch (Fig. 7.5).

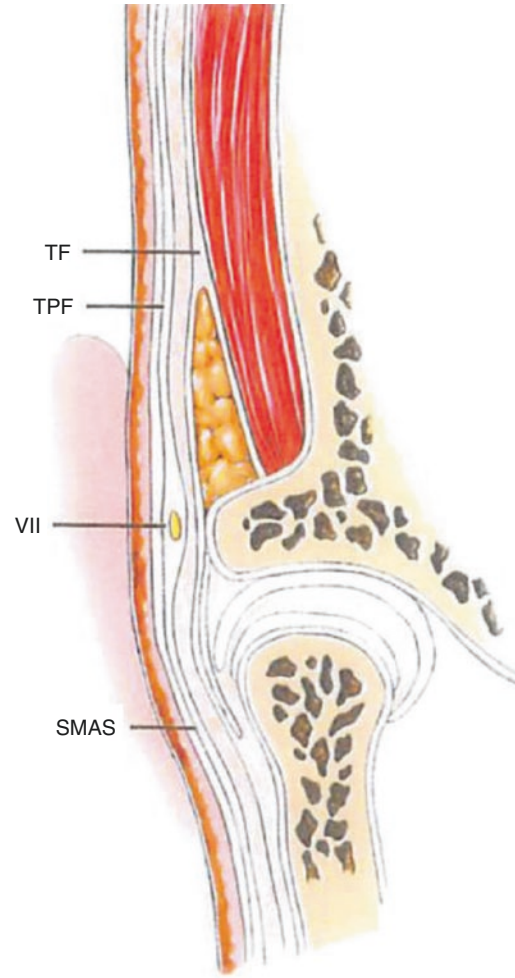


Fig. 7.4 Pre-auricular approach for TMJ arthroplasty. *TF* temporalis fascia (note that it splits inferior to this point into superficial and deep layers), *TPF* temporaretal fascia, *VII* temporal branch of the facial nerve, *SMAS* superficial musculoapnoneruotic system [12]

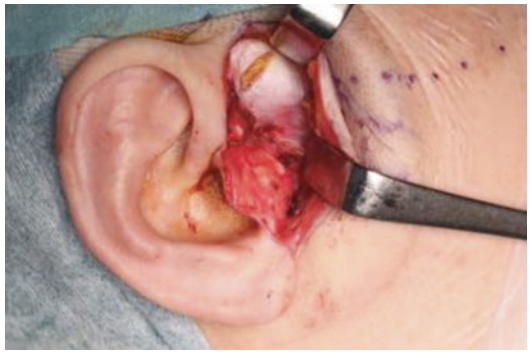


Fig. 7.5 Pre-auricular approach for TMJ arthroplasty. The superficial layer of the temporalis fascia was exposed

7.4.2 Complications of TMJ Open Arthroplasty

Compared to the arthroscopic surgery or arthrocentesis, TMJ open arthroplasty is more invasive and aggressive. The surgical procedures have many steps. Each step has a group of common potential complications, including damaging to adjacent structures of nerves, vessels, the ear, parotid gland, base of the skull, and middle cranial fossa.

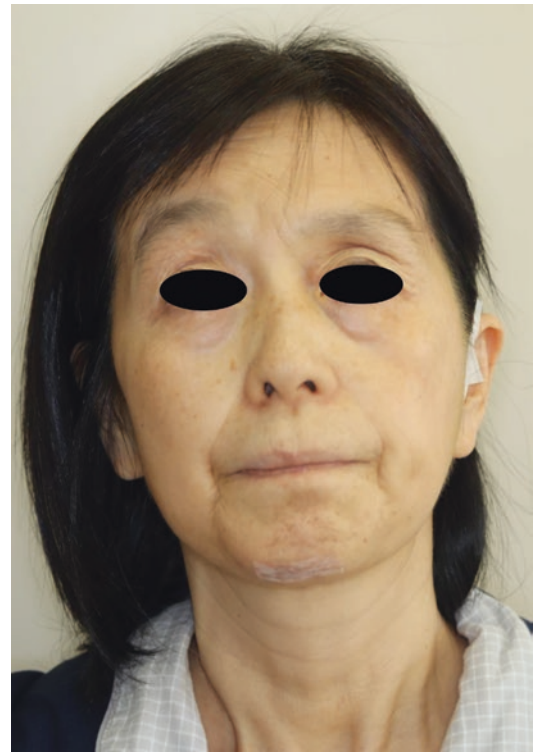


Fig. 7.6 A typical case of the facial nerve damage. The temporal branch of the facial nerve was affected

1. Nerve injuries

   Damage to the nerve is main the result of stretching to gain access to the joint or severing in the dissection. Transient neuropraxia of the temporal branches of the facial nerve occurs in as many as 20–30% of cases. The incidence increases when a separate skin flap is raised. Rarely, the zygomatic branches, even more rarely, the entire temporofacial division may be injured. Most of the cases, it resolves within 3–6 months. However, in a certain amount of cases, damage can be permanent (Fig. 7.6). If the weakness in the frontal branches is observed, cosmetic treatment of the injury can be done with either a forehead lift, or botulinum toxin to the adjacent side to give symmetry. Neuropraxia of the inferior alveolar and lingual nerves may result from clamp placement of the joint manipulation.

   As is the same with arthroscopy and arthrocentesis, when neuropraxia of any nerve injuries is evident after the surgical procedure, hydroxocobalamin acetate (Vitamin B_{12}) and adenosine triphosphate (ATP) were prescribed. The symptoms significantly improved within 2 weeks after surgery.

2. Infections

   Infections from arthroplasty of the joint are in the range of 1–2%. Microorganisms cultured may originate from the skin or external auditory meatus flora. Main routes of the infection include contaminations during the surgical procedure owing to some types of flora from the adjacent structures, immunodeficient patients, and opportunistic infections. Generally, infection can be divided into acute, subacute, and chronic infections. Acute infections could be treated by aggressive wound care and antibiotics. Chronic infections often develop later and can be persistent. Chronic infections can come from a biofilm if an alloplastic material is used. Chronic infections of the joint could lead to chronic osteomyelitis in the joint or spread the infection beyond the joint and into the adjacent structures. When a fistula into the external auditory canal was

found with the foreign body, one could speculate the infection of the joint. The use of Mitek bone anchors (Depuy Mitek, USA) which are the most common foreign body placed in the joint for TMJ disc repositioning surgery has a potential for the infection of the joint. Using periumbilical fat for filling into the joint, care should be taken not to have infections because it is proximal to the umbilics', which is considered part of the dirty field and should be copiously given a sterile preparation.

3. To avoid contamination, an ear packing is avoided as it frequently becomes dislodged during surgery. In addition, the ear is not suctioned during surgery. When the wound is closed, the external auditory canal is irrigated gently with saline via an 18-gauge angiocatheter.
4. Frey's Syndrome

   Frey's syndrome is a well-recognized complication of surgery in preauricular region. It is characterized by unilateral sweating and flushing of the facial skin in the area of parotid gland occurring during meals. The pathophysiology of the syndrome is that the damaged auriculotemporal nerve is invaded and irritated by healing tissue. Although commonly encountered as a complication of total or partial parotidectomy, on rare occasions it follows surgery or fracture of temporomandibular joint. Kryshtalskyj et al. reported that 3 of the 20 patients developed Frey's syndrome who underwent TMJ surgery using a preauricular approach [12].
5. Alloplastic implant

   In 1980s, alloplastic TMJ disc implants such as Teflon/proplast and silicone sheeting have been used for the TMJ reconstruction [13]. The problems with those materials are that they tend to break apart and cause foreign body cell reactions.

7.5 Alloplastic Total Temporomandibular Joint Replacement

Alloplastic total temporomandibular joint replacement (TMJ TJR) has been documented to be a safe and effective option for the management of TMJ pathology in worldwide studies for over two decades [14]. Stock implants such as metal-on-metal and metal-on-ultrahigh molecular weight polyethylene (UHMWPE), or patient-fitted metal-on-UHMWPE are commercially available (Fig. 7.7) [14]. Compared to three aforementioned surgical procedures (arthrocentesis, arthroscopy, and open arthroplasty), TMJ TJR requires the most complex and demanding surgical procedures. Whether the surgeon is using a custom-made or stock joint, the surgical procedures are for the most part the same and the complications are nearly identical. However, since TMJ TJR has alloplastic materials, surgical sterility is extremely important [15].

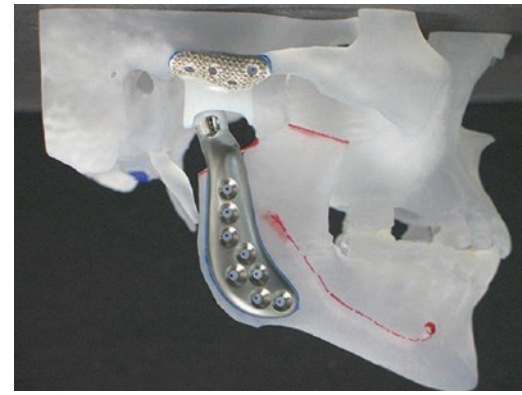


Fig. 7.7 A TMJ TJR system—TMJ concepts from https://tmjconcepts.com

7.5.1 Indications and Procedures

The most common indication is the end stage of TMJ arthritis, such as osteoarthritis, psoriatic, and rheumatoid arthritis. Ankylosis of the TMJ is also an appropriate indication of TMJ TJR. Other indications include damage to the joint by trauma, and a small amount of revision after complications that arise from earlier joint replacement [16]. TMJ TJR requires two incisions. The first incision is identical to the Bramley–Al Kayat technique. The second incision is an upper Risdon (high submandibular) approach to gain access to the lower part of the ramus of the mandibles.

7.5.2 Complications Related to TMJ TJR

1. Nerve injuries

   As mentioned on an TMJ arthroplasty, facial nerve injuries are the most important. Since TMJ TJR requires two incisions (preauricular and retromandibular or submandibular), damage to any one of these branches can result in either a true severing of the nerve or purely a stretching of the nerve. It is possible to damage isolated branches of the facial nerve in the retromandibular or submandibular incision while leaving the main trunk alone. The preauricular incision alternatively can have either the frontal or zygomatic branch damage and/or potentially damage in severe cases the entire trunk of the facial nerve, including all five branches involved. Therefore, careful surgical technique is mandatory. A nerve stimulator or a nerve locator is strongly recommended to avoid facial nerve injuries.
2. Damage to adjacent structures

   The most relevant structures related to TMJ TJR are the three areas around the condyle, including the external and internal structures of the ear, the anatomic areas medial to the joint, and damage to the superior aspect of the glenoid fossa into the intracranial space.
3. Infection

   Postoperative infections of TMJ TJR infections are uncommon (1.5–2.7%) [15]. The orthopedic literature reports a 1–2% incidence of more serious biofilm infections with TJR implants. Therefore, the infection rate of TMJ TJR is almost comparable to general TJR. For the prevention of infection, patients undergoing alloplastic TMJ TJR should receive prophylactic antibiotics coverage within 1 h before the surgical incision. Intraoperative draping and operative technique designed to prevent cross-contamination between the preauricular, and retromandibular surgical sites and oral cavity are essential. The protocol for the management of biofilm infections of custom TMJ TJR devices involves removal of the device components, placement of an appropriate antibiotic spacer in the area of the device, MMF, long-term antibiotic management and reimplantation of the remade device once all signs of infection have resolved [17].
4. Heterotopic bone formation

   When the patients have had previous ankylosis, recurrent ankylosis due to the heterotopic bone formation could occur. Heterotopic bone formation is the presence of bone in the soft tissue surrounding a TMJ TJR where bone normally does not exist. Subsequently, a decrease in joint mobility and/or pain could occur. To prevent it, either a non-steroidal anti-inflammatory drug, such as indomethacin, a diphosphonate such as ethane-1-hydroxy-1, 1-diphosphate, or local radiation therapy, has been recommended. Autogenous fat graft packing around the articulation of the TJR device seems to decrease potential recurrence [14, 18].

References

1. Nitzan DW, Dorwick MF, Martinez GA. Temporomandibular joint arthrocentesis: a simplified treatment for severe, limited mouth opening. J Oral Maxillofac Surg. 1991;49:1663–7.
2. Al-Moraissi EA. Arthroscopy versus arthrocentesis in the management of internal derangement of he temporomandibular joint: a systematic review and meta-analysis. Int J Oral Maxillofac Surg. 2015;44:104–12.
3. Vaira LA, Raho MT, Soma D, Salzano G, Orabona GD, Piombino P, Riu GD. Complications and postoperative sequelae of temporomandibular joint arthrocentesis. Cranio. 2018;36:264–7.
4. Carroll A, Smith K, Jakubowski J. Extradural hematoma following temporomandibular joint arthrocentesis and lavage. Br J Neurosurg. 2000;14:152–4.
5. Onishi M. Arthroscopy of the temporomandibular joint. Kokubyo Gkkai Zasshi. 1975;42:207–13.
6. Tsuyama M, Kondoh T, Sato K, Fukuda J. Complications of temporomandibular joint arthoroscopy: a retrospective analysis of 301 lysis and lavage procedures performed using the triangulation technique. J Oral Maxillofac Surg. 2000;58:500–5.
7. Gonzales-Garcia R, Rodríguez-Campo FJ, et al. Complications of temporomandibular joint arthroscopy: a retrospective analysis study of 670 arthroscopic procedures. J Oral Maxillofac Surg. 2006;64:1587–91.
8. MaCain JP, Sanders B, Koslin MG, et al. Temporomandibular joint arthroscopy: a 6-year multicenter retrospective study of 4,831 joints. J Oral Maxillofac Surg. 1992;50:926–30.

9. Murphy MA, Silvester KC, Chan TYK. Extradural hamatoma after temporomandibular joint arthroscopy: a case report. Int J Oral Maxillofac Surg. 1993;22:332–5.
10. Sugisaki M, Ikai A, Tanabe H. Dangerous angles and depth for middle ear and middle cranial fossa injury during arthroscopy of the temporomandibular joint. J Oral Maxillofac Surg. 1995;53:803–10.
11. Ellis IIIE, Zide MF. Preauricular approach. Surgical approaches to the facial skelton. 2nd ed. Philadelphia: Lippincott Williams and Wilkins; 2005. p. 193–212.
12. Krysthalskyj B, Weinberg S. An assessment for auriculotemporal syndrome following temporomandibular joint surgery through the preauricular approach. J Oral Maxillofac Surg. 1989;47:3–6.
13. Kearn JG, Perrott DH, Kaban LB. A protocol for the management of failed alloplastic temporomandibular joint disc implants. J Oral Maxillofac Surg. 1995;53:1240–7.
14. Mercuri LG. Alloplastic temporomandibular joint replacement: rationale for the use of custom devices. Int J Oral Maxillofac Surg. 2012;41:1033–40.
15. Mercuri LG, Psutka D. Perioperative, postoperative, and prophylactic use of antibiotics in alloplastic total temporomandibular joint replacement surgery: a survey and preliminary guidelines. J Oral Maxillofac Surg. 2011;69:2106–11.
16. Speculand B. Current status of replacement of the temporomandibular joint in the United Kingdom. Br J Oral Maxillofac Surg. 2009;47:37–41.
17. Mercuri LG. Prevention and detection of prosthetic temporomandibular joint infections -update. Int J Oral Maxillofac Surg. 2019;48(2):217–24. https://doi.org/10.1016/j.ijom.2018.09.011.
18. Hoffman D, Pueg L. Complicatoins of TMJ. Oral Maxillofac Surg Clin N Am. 2015;27:109–24.

8 Complications Associated with Treatment of Medication-Related Osteonecrosis of the Jaws (MRONJ)

Suad Aljohani and Sven Otto

Contents

Medication-related osteonecrosis of the jaw is a rare complication of antiresorptive drugs (ARDs), including bisphosphonates (BPs) and denosumab, commonly used in the treatment of osteoporosis and metastatic disease. The treatment of MRONJ poses a real challenge for oral and maxillofacial surgeons not only due to the lack of evidence-based treatment guidelines but also due to the related complications and its tendency to recur after therapy. The progressive nature of MRONJ can lead to inferior alveolar nerve involvement and pathologic fractures in the mandible and maxillary sinus involvement in the maxilla. The management of these complications is generally difficult and still elusive. Great effort should be taken to improve the quality of life by controlling infection, resolving pain, and restoring aesthetics and oral functions.

S. Aljohani
Division of Oral Medicine, Department of Oral Diagnostic Sciences, King Abdulaziz University, Jeddah, Saudi Arabia
e-mail: sraljohani@kau.edu.sa

S. Otto (✉)
Department of Oral and Maxillofacial Surgery, Ludwig-Maximilians-University of Munich, Munich, Germany
e-mail: sven.otto@med.uni-muenchen.de

8.1 Recurrence

The initial clinical recommendations supported the conservative treatment, mainly superficial debridement, long-term antibiotics, and antimicrobial mouthwashes. Nicolatou-Galitis et al. and Hoff et al. reported mucosal healing in only 14.9% and 23% of the cases, respectively, after several months of antibiotics treatment [1, 2]. However, the resulted persistence of exposed bone, progression of necrosis, and recurrent infections could jeopardize chemotherapy and further antiresorptive therapy and compromise

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_8

the quality of life in oncology patients. More importantly, prolonged intake of antibiotics may encourage the proliferation of resistant pathogens and predispose the patients to develop antibiotic resistance. On the other hand, several studies have reported a good healing rate of over 85% after surgical treatment [3–5]. As experience in managing MRONJ has accumulated over the last decade, it is agreed now that complete removal of necrotic bone, rounding of sharp bone edges and tensionless meticulous wound closure in combination with perioperative antibiotic treatment are essential for healing of established MRONJ lesions and for minimizing their recurrence. Utilizing fluorescence with a VELscope fluorescence lamp (LED Dental, Atlanta, Georgia, USA) as a guidance during surgery to distinguish the necrotic bone from the vital bone can allow complete, yet conservative, removal of the necrotic bone (Fig. 8.1). This technique can aid in significant reduction of MRONJ recurrence rate [4, 6]. It is important to keep in mind that surgical management even of early MRONJ stages, stage 0 and 1, is crucial to obtain better treatment outcomes and to decrease MRONJ progression and subsequently the associated complications.

8.2 Pathological Fractures

Pathologic fractures are rare serious complications of MRONJ in the mandible. It was reported to occur in 3–4% of MRONJ patients [7, 8]. There is only very limited data in regards to their management with few published cases so far. Given the progressive nature of MRONJ and its reluctance to therapy, such fractures are expected to be more frequent than before. Mandibular fractures can also occur after complete removal of necrotic bone and can substantially impair quality of life.

Overall, the treatment of these fractures is challenging and the optimal management is still unknown. The reported treatments ranged from conservative treatments to free flap reconstruction. In principle, the general medical status of the patient should be kept in consideration during clinical decision-making of MRONJ patients. In patients with good medical status and good life expectancy, removal of necrotic bone followed by load-bearing osteosynthesis systems is recommended (Fig. 8.2). In patients with fractures reluctant to internal fixation, external fixation could be the treatment of choice. Otto et al. reported three cases treated with open reduction and rigid internal fixation with evident mucosal healing of two of them [8]. In the same case series, one case was reported to be managed with external fixation with a satisfactory outcome of stable pseudoarthrosis and mucosal healing [8]. The patient was reported to be symptomless and able to wear his removable prosthesis. Of note, the screws of the plate should be placed away from the borders of the removed affected bone to allow better integration.

Some authors described the use of microvascular flap reconstruction for reluctant and severe MRONJ [9–12]. Mücke et al. described the use of free flap reconstruction for 20 mandibular MRONJ lesions [13]. Nine defects were reconstructed with fibula flap, five with iliac crest flap and six with radial forearm flap. Recurrence was observed in only one lesion close to a fibula free flap. A systematic review included 6 studies and 31 patients with stage 3 MRONJ reconstructed with osseous free flap has shown a recurrence of only 5% of the cases [14]. Despite the encouraging results, this systematic review was based on case series and case reports, which do not provide a sufficient evidence to establish clinical guidelines. Moreover, such complicated surgical approaches should be considered with extreme caution because of the associated morbidity in elderly patients with many risk factors and comorbidities as well as the risk of developing MRONJ within the flap [15]. In addition, free flap, especially iliac crest flap, should be avoided in multiple myeloma patients due to the risk of transferring malignant bone marrow cells to the jaws. Regardless of the used surgical technique, adequate antibiotic regimen before and after the surgery is of crucial importance.

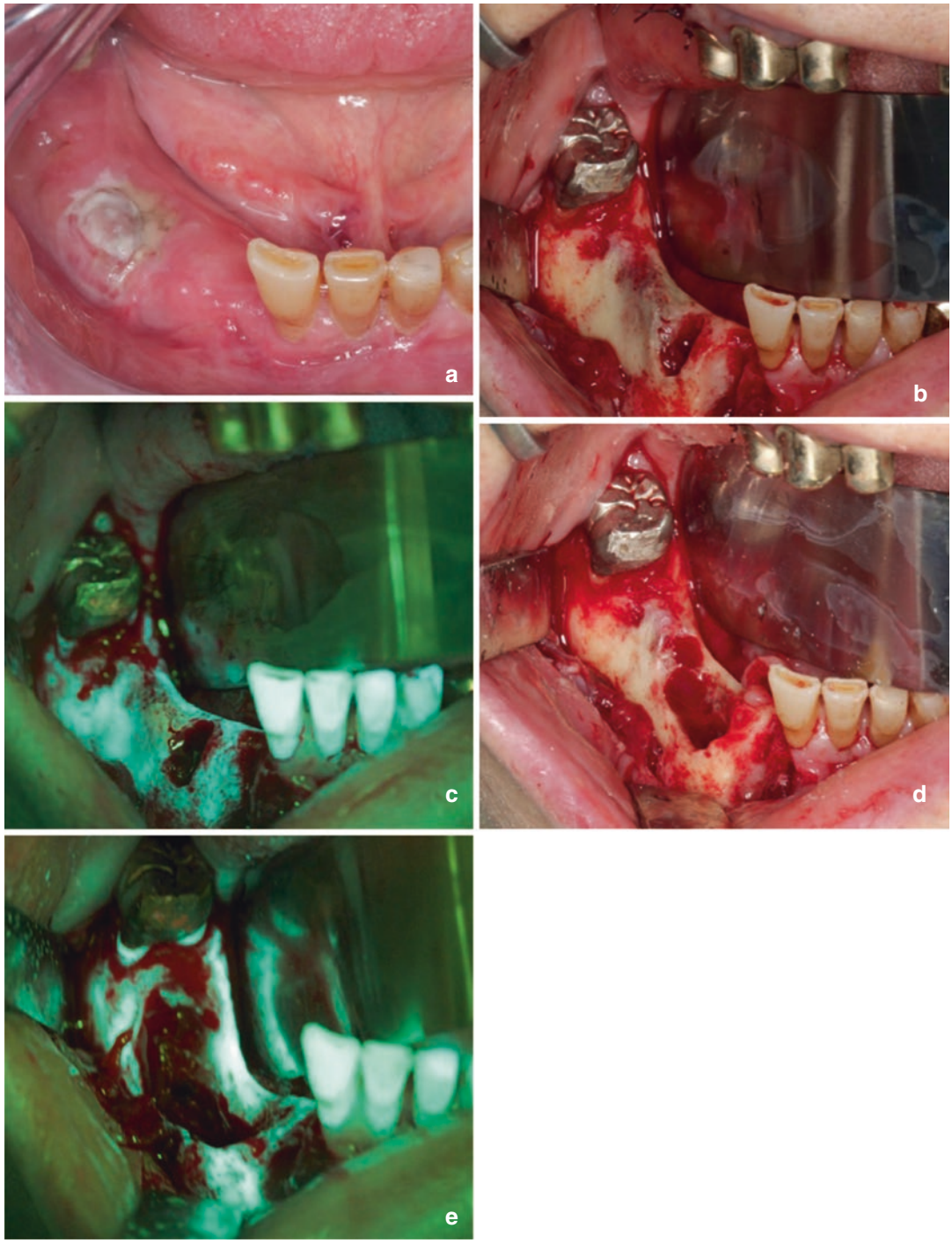


Fig. 8.1 A 78-year-old patient has been treated with denosumab for metastatic prostate cancer: (**a**) clinical presentation of stage 2 MRONJ in the right posterior mandible, (**b**) operative view after elevation of mucoperiosteal flap showed the grayish color of the necrotic bone, (**c**) diminished fluorescence at the area of necrosis, (**d**) the operative view after removal of necrotic bone and smoothening of sharp bone edges, and (**e**) more homogenous and brighter fluorescence was evident after removal of the necrotic bone

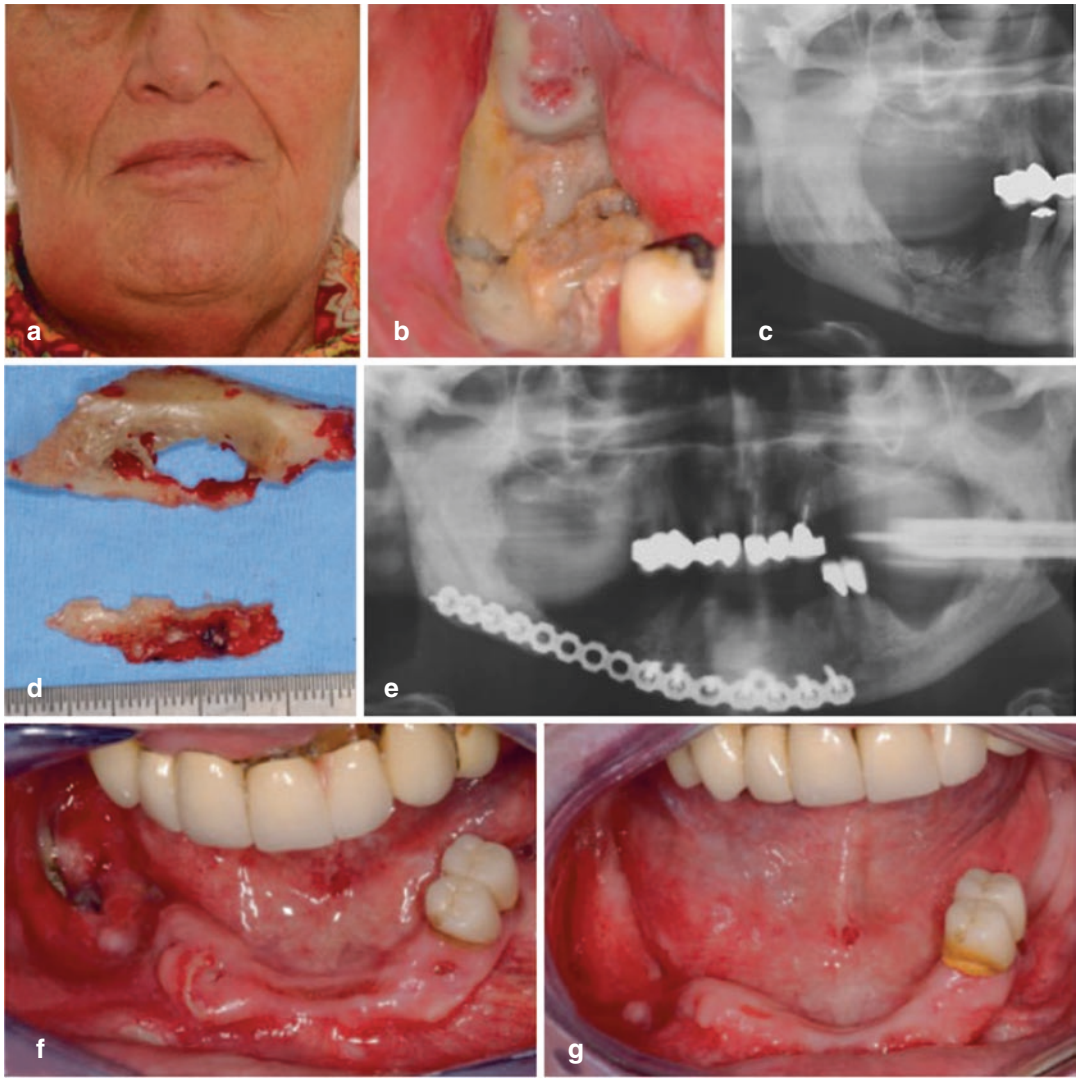


Fig. 8.2 A 65-year-old breast cancer patient had MRONJ as a complication of intravenous administrations of BPs: (**a**) extraoral view with a swelling of the right submandibular area, which was painful on palpation, (**b**) intraoral view with a large area of exposed necrotic bone and signs of massive superinfection (swelling, pus) and a visible fracture of the mandible with mobile segments, (**c**) panoramic radiograph with a mixed radiolucent and radiopaque appearance and a visible fracture line located in the right mandibular body, (**d**) large bone sequesters that could be removed in the course of the treatment including segmental resection of the mandible, rigid internal fixation using a Synthes 2.4 unilock plate (DePuy Synthes, Germany), and complete closure of the wound, (**e**) postoperative panoramic radiograph showing the resected area of the right mandible and the rigid internal fixation (Synthes 2.4 unilock plate), (**f**) intraoral view 3 weeks postoperatively with a late dehiscence and plate exposure in region 47/48, and (**g**) intraoral view 4 months postoperatively with complete mucosal healing after local disinfectant measurements using disinfectant mouth rinses and activated photodynamic therapy (Reprinted with kind permission of © Georg Thieme Verlag KG) [8]

A case of MRONJ-induced fractured mandible in osteoporosis patients with concomitant vertebral compression fracture was reported to be managed with sequestrectomy, teriparatide (56.5 μg/week), and antibiotic therapy (for 8 months) [16]. After 18 months of teriparatide administration, improvement in bone generation and mandibular continuity was evident. Nevertheless, teriparatide is contraindicated in metastatic cancer patients who represent the majority of MRONJ patients.

8.3 Involvement of Inferior Alveolar Nerve

Vincent symptom or numb chin syndrome can be one of the early symptoms of MRONJ [17]. It can be also a clinical manifestation of advanced MRONJ. It represents an impairment of inferior alveolar nerve evidenced by numbness of the chin, lower lip, and lower anterior teeth and gingiva. Painful trigeminal neuropathy was also reported in relation to long-standing untreated MRONJ and resolved after a combination of surgical and antibiotic therapy [18]. Local infection, sequestration, and pathological fractures are thought to alter the nerve function. However, the exact etiopathogenesis remains to be elucidated. A recent study has shown that BPs itself can induce degeneration of the inferior alveolar nerve fibers [19]. In general, this condition tends to resolve after MRONJ surgical and antibiotic treatment, although it may also arise as a postoperative complication. Only one retrospective study of three patients reported allogeneic nerve grafting after MRONJ resection with successful outcomes [20].

8.4 Oroantral or Oronasal Communications

As MRONJ is detected less frequently in the maxilla than in the mandible, proper investigations of the clinical course and the treatment outcomes of maxillary MRONJ are still lacking. There are only few studies so far analyzing the complications and the treatment of MRONJ of the upper jaw [21, 22]. Maxillary sinus involvement was reported in 44% of upper jaw MRONJ cases [23]. Maxillary sinusitis was detected in 43.6% of the cases, while oroantral communications were identified in 36% of the cases [23]. Even in absence of maxillary sinus involvement prior to treatment, it could be induced after complete removal of the necrotic bone especially in the posterior maxilla. The management of oroantral communications is crucial not only to improve patients' quality of life but also to avoid involvement of other paranasal sinuses and other nearby vital structures. Nasal septal abscess, orbital cellulitis, skull base necrosis, and brain abscess were reported in relation to MRONJ of the maxilla [24–28]. Such life-threatening complications although are extremely rare, they tend to occur particularly in immunocompromised and cancer patients. Therefore, special attention should be given to the optimal management of maxillary MRONJ.

The gold standard for treatment of stage 3 maxillary lesions is complete necrotic bone removal followed by smoothening of sharp bony edges and meticulous wound closure with adequate perioperative antibiotic treatment. The sinus lining has to be approached carefully during necrotic bone removal to avoid opening of the maxillary sinus unless signs of sinus empyema are present. In case of lesions with medium to large oroantral communication, tensionless wound closure with double-layer wound closure, mainly buccal fat pad flap covered by mucoperiosteal flap, is recommended (Fig. 8.3). This technique is reliable and easy to apply. Buccal fat pad flap provides mechanical protection, adequate vascularization of the underlying bone and adipose tissue-derived stem cells and thus can promote healing [29]. Nasolabial flap is another surgical option to cover extended oroantral communications [30]. However, this flap necessitates vestibuloplasty and pedicle division prior to dental prosthetic rehabilitation.

In fact, the management of stage 3 maxillary MRONJ is complex and the treatment outcomes are hard to predict. A second surgical attempt to manage persisting oroantral communication is not always feasible particularly in elderly patients with several comorbidities. Therefore, prosthetic rehabilitation with obturators can be the management of choice in extended cases as well as refractory cases with favorable anatomy in absence of suppuration and infection (Fig. 8.4) [31]. Obturator prostheses were evaluated extensively for rehabilitation after oral cancer resections and were shown to be effective in restoring esthetic and masticatory function and in preventing nasal fluid leakage [32]. Nevertheless, few cases with obturators prostheses in MRONJ patients were published so far. These studies reported encouraging results and found them to be well-tolerated and uncomplicated [31, 33]. Rehabilitation with obturators is, however, not an alternative to complete surgical removal of necrotic bone which is essential to suppress MRONJ progression.

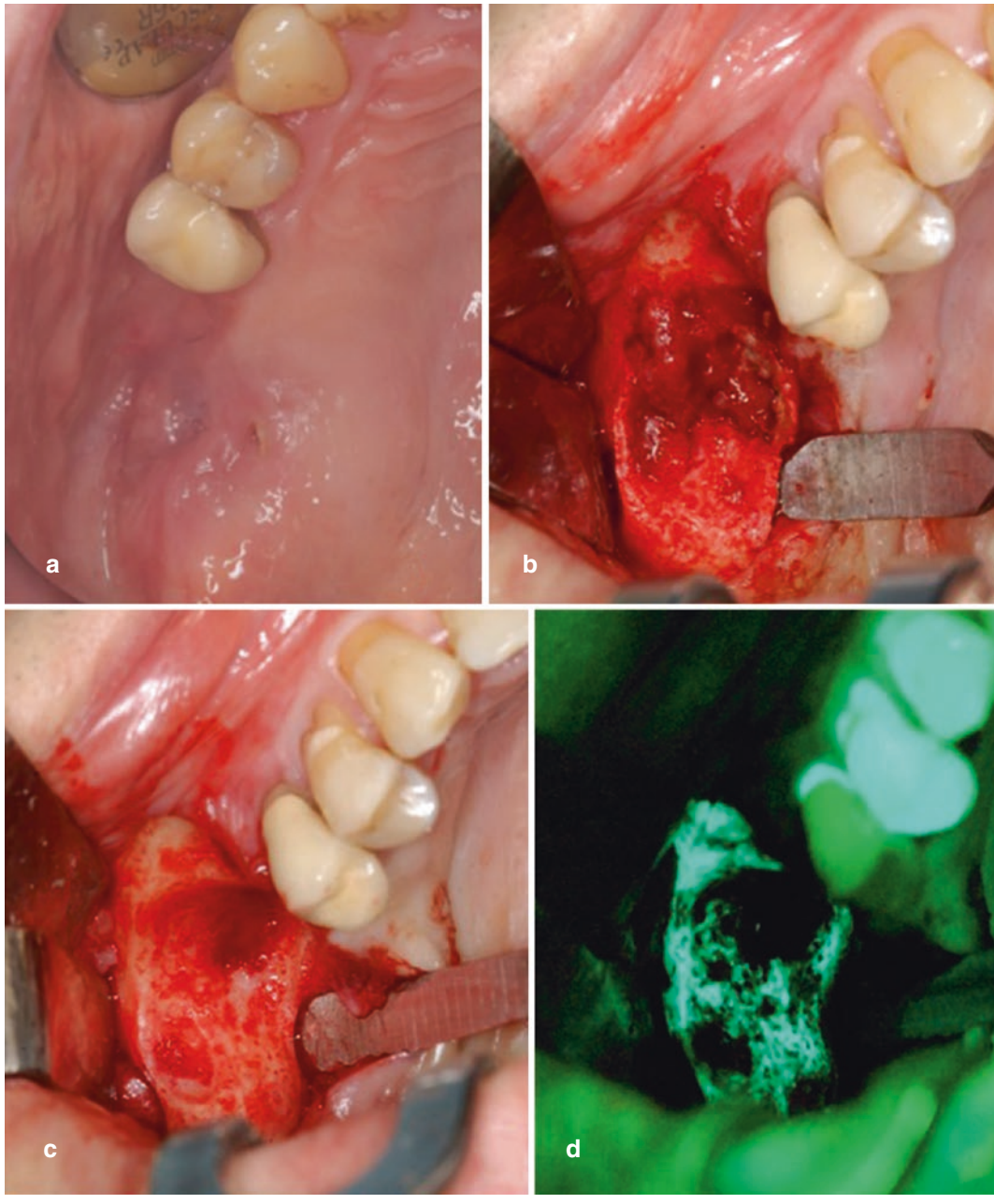


Fig. 8.3 A 70-year-old male patient after a single dose of denosumab and previous intake of zoledronate (for 43 months) for metastatic prostate cancer: (**a**) stage 2 MRONJ lesion of the upper right maxilla, (**b**) the necrotic bone was obvious after reflection of mucoperiosteal flap, (**c**) and (**d**) intraoperative view after complete removal of necrotic bone using fluorescence-guided surgery: the fluorescence was homogenously green except for areas of exposed sinus mucosa which was left uninterrupted, and (**e**) buccal fat pad flap was used to cover the defect followed by coverage with mucoperiosteal flap and tension-less meticulous wound closure to minimize the risk of maxillary sinusitis

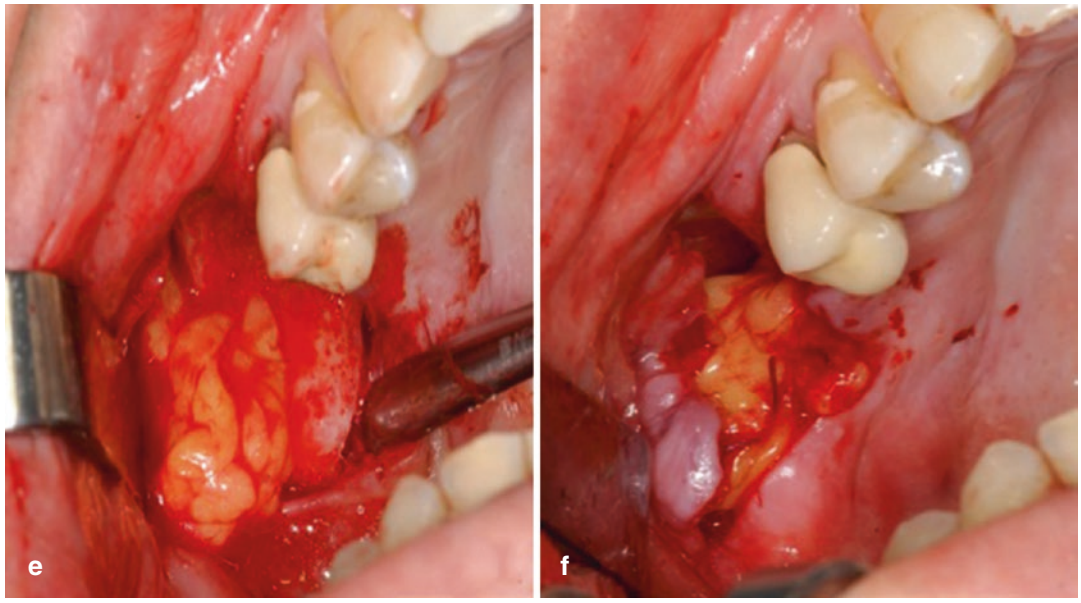


Fig. 8.3 (continued)

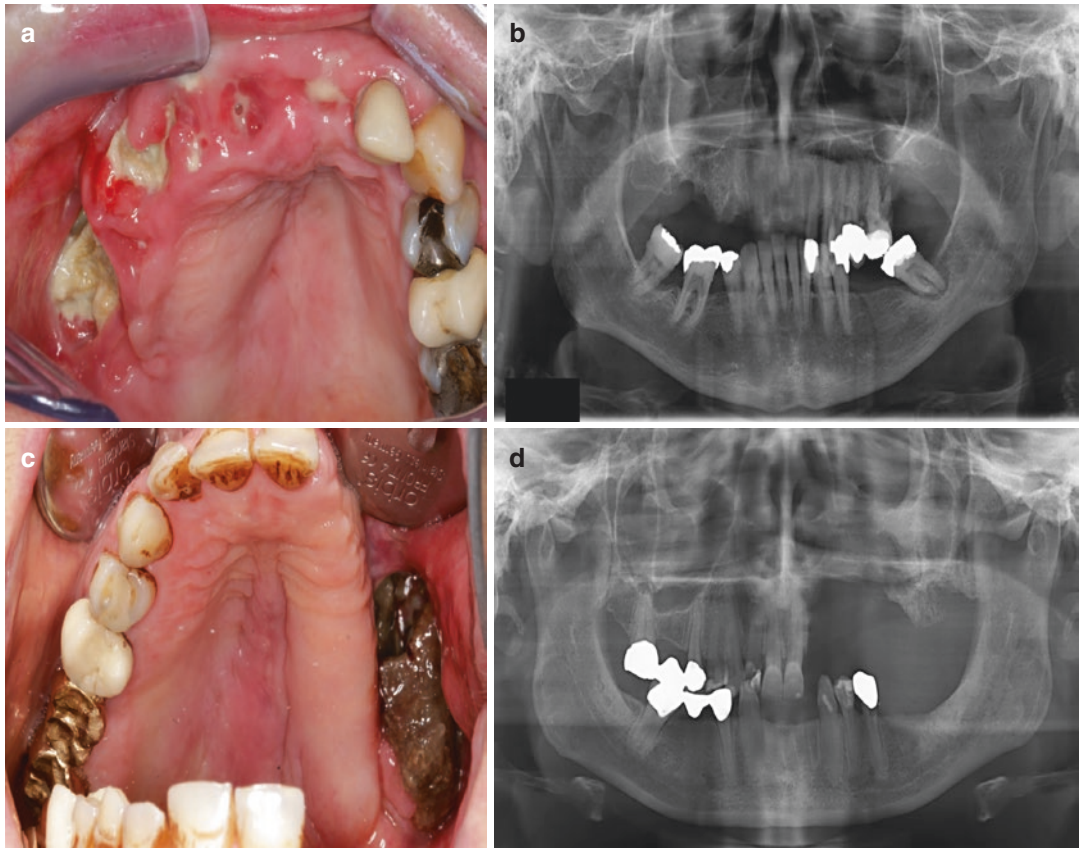


Fig. 8.4 (**a**) and (**b**), (**c**) and (**d**). Preoperative clinical pictures and panoramic radiographs of two extended MRONJ cases managed with obturators. Obturator prosthesis is a relevant option for such cases

References

1. Hoff AO, Toth BB, Altundag K, Johnson MM, Warneke CL, Hu M, Nooka A, Sayegh G, Guarneri V, Desrouleaux K, Cui J, Adamus A, Gagel RF, Hortobagyi GN. Frequency and risk factors associated with osteonecrosis of the jaw in cancer patients treated with intravenous bisphosphonates. J Bone Miner Res. 2008;23:826–36.
2. Nicolatou-Galitis O, Papadopoulou E, Sarri T, Boziari P, Karayianni A, Kyrtsonis MC, Repousis P, Barbounis V, Migliorati CA. Osteonecrosis of the jaw in oncology patients treated with bisphosphonates: prospective experience of a dental oncology referral center. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2011;112:195–202.
3. Carlson ER, Basile JD. The role of surgical resection in the management of bisphosphonate-related osteonecrosis of the jaws. J Oral Maxillofac Surg. 2009;67:85–95.
4. Otto S, Ristow O, Pache C, Troeltzsch M, Fliefel R, Ehrenfeld M, Pautke C. Fluorescence-guided surgery for the treatment of medication-related osteonecrosis of the jaw: a prospective cohort study. J Craniomaxillofac Surg. 2016;44:1073–80.
5. Ristow O, Otto S, Troeltzsch M, Hohlweg-Majert B, Pautke C. Treatment perspectives for medication-related osteonecrosis of the jaw (MRONJ). J Craniomaxillofac Surg. 2015;43:290–3.
6. Ristow O, Otto S, Geiss C, Kehl V, Berger M, Troeltzsch M, Koerdt S, Hohlweg-Majert B, Freudlsperger C, Pautke C. Comparison of autofluorescence and tetracycline fluorescence for guided bone surgery of medication-related osteonecrosis of the jaw: a randomized controlled feasibility study. Int J Oral Maxillofac Surg. 2017;46:157–66.
7. Abu-Id MH, Warnke PH, Gottschalk J, Springer I, Wiltfang J, Acil Y, Russo PA, Kreusch T. "Bis-phossy jaws"—high and low risk factors for bisphosphonate-induced osteonecrosis of the jaw. J Craniomaxillofac Surg. 2008;36:95–103.
8. Otto S, Pautke C, Hafner S, Hesse R, Reichardt LF, Mast G, Ehrenfeld M, Cornelius CP. Pathologic fractures in bisphosphonate-related osteonecrosis of the jaw-review of the literature and review of our own cases. Craniomaxillofac Trauma Reconstr. 2013;6:147–54.
9. Engroff SL, Kim DD. Treating bisphosphonate osteonecrosis of the jaws: is there a role for resection and vascularized reconstruction? J Oral Maxillofac Surg. 2007;65:2374–85.
10. Hanasono MM, Militsakh ON, Richmon JD, Rosenthal EL, Wax MK. Mandibulectomy and free flap reconstruction for bisphosphonate-related osteonecrosis of the jaws. JAMA Otolaryngol Head Neck Surg. 2013;139:1135–42.
11. Nocini PF, Saia G, Bettini G, Ragazzo M, Blandamura S, Chiarini L, Bedogni A. Vascularized fibula flap reconstruction of the mandible in bisphosphonate-related osteonecrosis. Eur J Surg Oncol. 2009;35:373–9.
12. Seth R, Futran ND, Alam DS, Knott PD. Outcomes of vascularized bone graft reconstruction of the mandible in bisphosphonate-related osteonecrosis of the jaws. Laryngoscope. 2010;120:2165–71.
13. Mucke T, Jung M, Koerdt S, Mitchell DA, Loeffelbein D, Kesting MR. Free flap reconstruction for patients with bisphosphonate related osteonecrosis of the jaws after mandibulectomy. J Craniomaxillofac Surg. 2016;44:142–7.
14. Vercruysse H Jr, Backer TD, Mommaerts MY. Outcomes of osseous free flap reconstruction in stage III bisphosphonate-related osteonecrosis of the jaw: systematic review and a new case series. J Craniomaxillofac Surg. 2014;42:377–86.
15. Pautke C, Otto S, Reu S, Kolk A, Ehrenfeld M, Sturzenbaum S, Wolff KD. Bisphosphonate related osteonecrosis of the jaw—manifestation in a microvascular iliac bone flap. Oral Oncol. 2011;47:425–9.
16. Yao M, Shimo T, Ono Y, Obata K, Yoshioka N, Sasaki A. Successful treatment of osteonecrosis-induced fractured mandible with teriparatide therapy: a case report. Int J Surg Case Rep. 2016;21:151–3.
17. Otto S, Hafner S, Grotz KA. The role of inferior alveolar nerve involvement in bisphosphonate-related osteonecrosis of the jaw. J Oral Maxillofac Surg. 2009;67:589–92.
18. Zadik Y, Benoliel R, Fleissig Y, Casap N. Painful trigeminal neuropathy induced by oral bisphosphonate-related osteonecrosis of the jaw: a new etiology for the numb-chin syndrome. Quintessence Int. 2012;43:97–104.
19. Dietrich EM, Theodora P, Antonia S, Georgios K, Esthelle N. Ultrastructural alterations of the inferior alveolar nerve in wistar rats after alendronate administration per os: hypothesis for the generation of the "numb chin syndrome". J Histol Histopathol. 2015;2:24.
20. Tolomeo PG, Loparich A, Konicki WS, Fleisher KE. Nerve reconstruction for patients with medication-related osteonecrosis of the jaw. J Oral Maxillofac Surg. 2017;75:e401–2.
21. Berrone M, Florindi FU, Carbone V, Aldiano C, Pentenero M. Stage 3 medication-related osteonecrosis of the posterior maxilla: surgical treatment using a pedicled buccal fat pad flap: case reports. J Oral Maxillofac Surg. 2015;73:2082–6.
22. Voss PJ, Vargas Soto G, Schmelzeisen R, Izumi K, Stricker A, Bittermann G, Poxleitner P. Sinusitis and oroantral fistula in patients with bisphosphonate-associated necrosis of the maxilla. Head Face Med. 2016;12:3.
23. Mast G, Otto S, Mucke T, Schreyer C, Bissinger O, Kolk A, Wolff KD, Ehrenfeld M, Sturzenbaum SR, Pautke C. Incidence of maxillary sinusitis and oroantral fistulae in bisphosphonate-related osteonecrosis of the jaw. J Craniomaxillofac Surg. 2012;40:568–71.

24. Khan AM, Sindwani R. Bisphosphonate-related osteonecrosis of the skull base. Laryngoscope. 2009;119:449–52.
25. Maeda M, Matsunobu T, Kurioka T, Kurita A, Shiotani A. A case of nasal septal abscess caused by medication related osteonecrosis in breast cancer patient. Auris Nasus Larynx. 2016;43:93–6.
26. Malik R, Fernando BS, Laitt RD, Leatherbarrow B. Bisphosphonate-induced osteonecrosis of the maxilla presenting as a cicatricial ectropion. Orbit. 2014;33:369–71.
27. Matsushita A, Kamigaki S, Nakamura Y. A case of brain abscess secondary to bisphosphonate-related osteonecrosis of the jaws in metastatic bone lesions from breast carcinoma. Gan To Kagaku Ryoho. 2013;40:631–3.
28. Yamagata K, Nagai H, Baba O, Uchida F, Kanno N, Hasegawa S, Yanagawa T, Bukawa H. A Case of brain abscess caused by medication-related osteonecrosis of the jaw. Case Rep Dent. 2016;2016:7038618.
29. Burian E, Probst F, Palla B, Riedel C, Saller MM, Cornelsen M, Konig F, Schieker M, Otto S. Effect of hypoxia on the proliferation of porcine bone marrow-derived mesenchymal stem cells and adipose-derived mesenchymal stem cells in 2- and 3-dimensional culture. J Craniomaxillofac Surg. 2017;45:414–9.
30. Lemound J, Muecke T, Zeller AN, Lichtenstein J, Eckardt A, Gellrich NC. Nasolabial flap improves healing in medication-related osteonecrosis of the jaw. J Oral Maxillofac Surg. 2018;76(4):877–85.
31. Troeltzsch M, Probst F, Troeltzsch M, Ehrenfeld M, Otto S. Conservative management of medication-related osteonecrosis of the maxilla with an obturator prosthesis. J Prosthet Dent. 2015;113:236–41.
32. Chigurupati R, Aloor N, Salas R, Schmidt BL. Quality of life after maxillectomy and prosthetic obturator rehabilitation. J Oral Maxillofac Surg. 2013;71:1471–8.
33. Gollner M, Holst S, Fenner M, Schmitt J. Prosthodontic treatment of a patient with bisphosphonate-induced osteonecrosis of the jaw using a removable dental prosthesis with a heat-polymerized resilient liner: a clinical report. J Prosthet Dent. 2010;103:196–201.

Part III

Trauma

9 Complications in Cranio-Maxillofacial Trauma

Robert Gassner

Contents

9.1 Introduction

Cranio-maxillofacial trauma (CMFT) deals with facial bone fractures, dental trauma, soft tissue injuries and lesions of its vessels and cranial nerves. CMFT is also often associated with concomitant injuries. Due to the complexity of facial structures and features, CMFT occurs, in addition or together with other trauma creating situations that turn the condition harder to treat, repair or heal [1–10].

This chapter on complications in CMFT is an attempt to display the plethora of problems accompanying patients who suffered from CMFT.

R. Gassner (✉)
Department of CMF and Oral Surgery, Medical University of Innsbruck, Innsbruck, Austria
e-mail: robert.gassner@tirol-kliniken.at

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_9

Five major causes of CMFT exist: work, traffic, assault, sport, and activities of daily life. The main mechanisms of CMFT are falls, collisions with others, collisions with stationary objects, failure of equipment, and/or failure of proper equipment use among others.

Cranio-maxillofacial surgery is a specialty which focuses on treating CMFT in a unique way that complications are avoided or at least solved over time using regenerative and wound healing potential. Usually, complications arise when necessary surgical procedures are not done or postoperative care is insufficient or lacking. But sometimes surgery may cause complications itself and it may be minimized with reconstructive procedures which in turn may cause donor site morbidity itself.

This chapter deals with CMFT-related complications which bother patients by affecting their quality of life in a highly developed health care system. Some patients may suffer from more than one complication. From a logistic standpoint of view as an outside observer, it seems to be necessary to describe in a first step what to do to avoid the complication and in a second step how to get treatment back on a good track. But in reality, the sequence of an accident, a patient's medical history, diagnostic evaluation, and surgical and/or conservative treatment with follow-up care has the main goal to avoid complications per se and allow a recovery to normal function as fast as possible. Therefore, complications are dissected in an orderly fashion. Most of them never occur, are avoided in the beginning, or in case they are present they are at least mitigated.

9.2 Complication—Loss of Sensation: Supra-, Infraorbital or Mental Nerve Damage

This complication is often part of a facial bone fracture occurring at the time of the accident. Nerves are squeezed between fracture fragments and open reduction, and gentle anatomic repositioning allows recovery of the damaged axons and sensitivity recovers within weeks, months, and sometimes in years.

First: what to do to avoid a permanent complication:

It is very important to **avoid additional harm to nerval structures** in proximity to the supraorbital, infraorbital and mental foramen during facial bony reconstruction with plates and screws—staying away from the infraorbital foramen or avoiding to drill screws into the infraalveolar canal. But it is necessary to apply a stable fixation with at least three screws on each side of a fracture below the inferior alveolar canal or apply two short screws above (to avoid damage to the roots of teeth) and two screws below the inferior alveolar canal.

Second: how to get treatment back on a good track:

There are scientific reports regarding microscopic nerve repair in case of iatrogenic damage. The success rate of complete recovery is fragile as not only hyp- and paresthesia but also dys- and hyperesthesia are reported [11, 12].

Patients who suffer from injuries affecting sensory trigeminal nerve endings such as **the supra-, infraorbital or mental nerve** which in some cases are sensed as disturbance of the **occlusion,** although no hindrance is present after fixing CMFT but which in turn is sometimes seen as the onset and awareness of TMJ disorders.

Case 1

A female mountain biker, 40 years of age, rode down a mountain trail. She was suddenly airborne over a gate when she used her front brakes [13–15]. She crashed on dirt with head and face. Following initial unconsciousness, she went on riding down the trail. She was immediately taken to the hospital by ambulance from her family practitioner. Clinical evaluation in the emergency room and a 3D CT scan of her facial skeleton revealed no facial fractures (Fig. 9.1a).

Due to the pain and sensory loss in her left cheek, she was reassessed on the third day after the accident, and a zygoma fracture with fracture of the lateral maxillary sinus wall and orbital floor fracture was diagnosed in the plain radiograph (Fig. 9.1b).

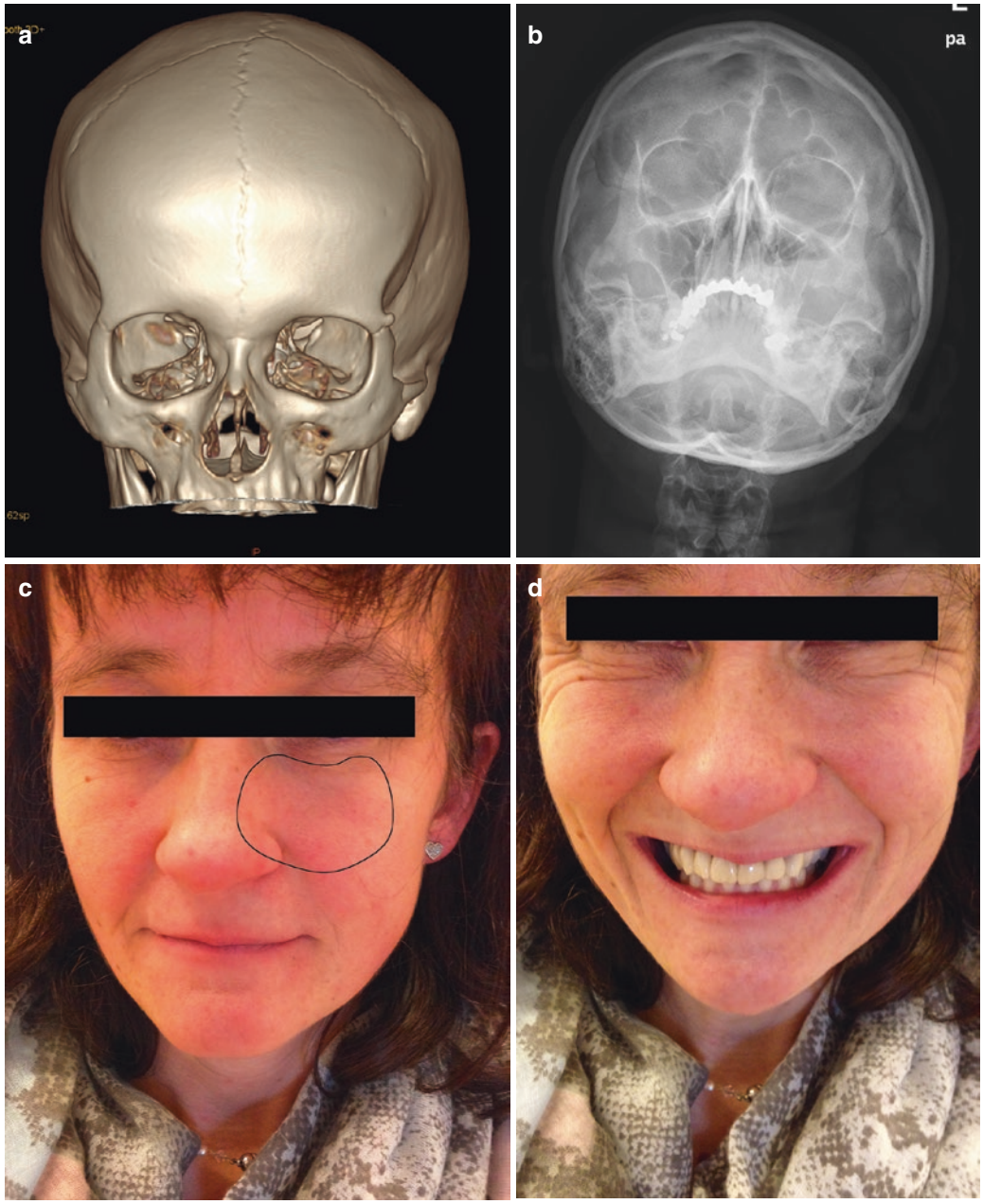


Fig. 9.1 (**a**) 3D CT scan without facial bone fractures. (**b**) Plain radiograph: maxillary sinus wall and orbital floor fracture left side. (**c**) Persisting dysesthesia after orbital floor repair. (**d**) Entropion of left lower eyelid

Surgery for orbital floor repair was uneventful. But as a permanent complication of the accident the patient complains suffering from sensitive disorder of her left cheek (Fig. 9.1c) and malfunction of her left lower eyelid scratching the cornea of maligned hairs (entropion) (Fig. 9.1d).

Case 2

A female cyclist, 58 years of age, collided with a car [13–15]. She sustained mandible fractures of the right condyle and left mandibular body including a complicated fracture of the left lower first premolar tooth (Fig. 9.2a).

She underwent successful surgical treatment of her mandible fractures including tooth removal (Fig. 9.2b) and bone wound healing occurred undisturbed (Fig. 9.2c).

Two years after the accident, she still suffers from persisting complications such as sensory dysfunction of her left lower lip and chin showing dysesthesia [16] on touching (Fig. 9.2d) which persisted also after hardware removal 1 year after the accident (Fig. 9.2e).

Case 3

A physician, 54 years of age, sustained a zygoma fracture on the right side (Fig. 9.3a), mandible fractures of the right ascending ramus and left condyle (Fig. 9.3b), and a clavicular (collar) fracture on the left side due to a ski accident (Fig. 9.3c) [17–22].

All fractures were treated successfully with open reduction and rigid fixation using plates and screws on the third day after the accident (Fig. 9.3d).

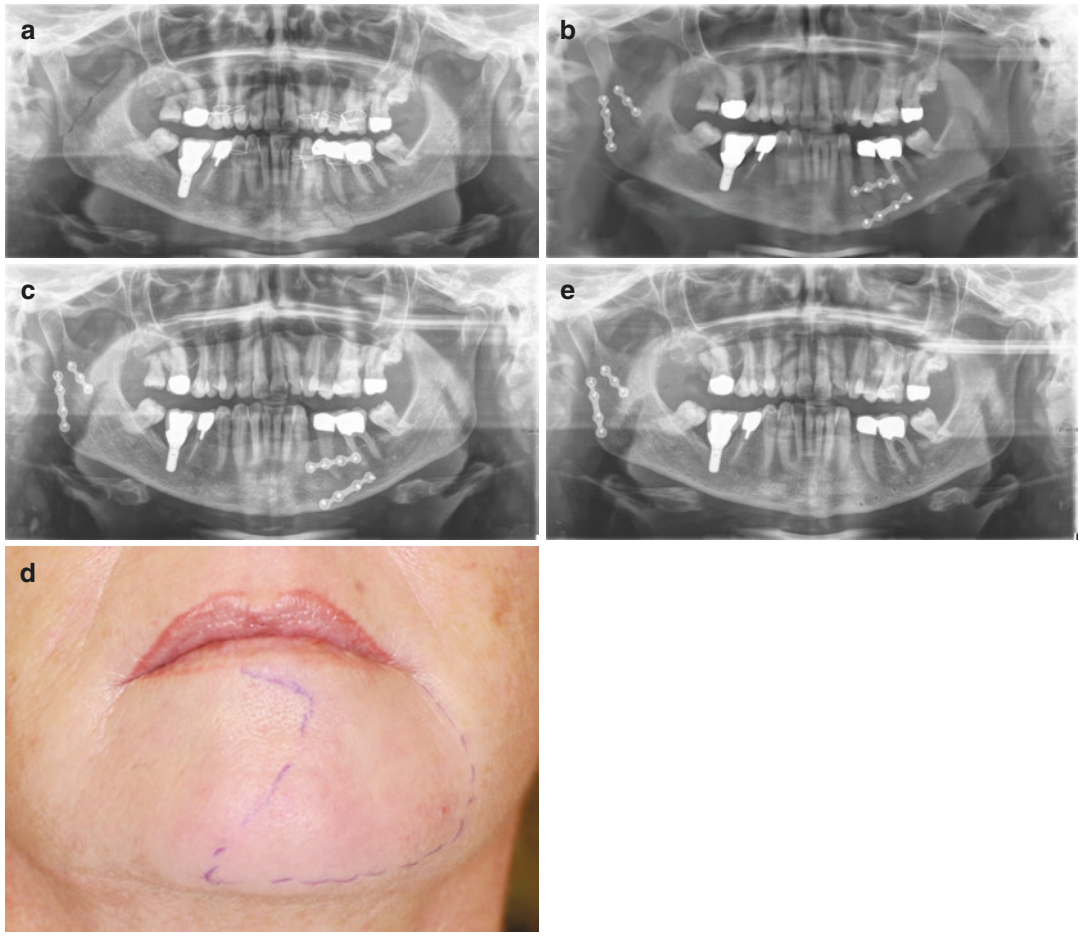


Fig. 9.2 (**a**) Panoramic radiograph with fracture of the right condyle and left mandibular body and root fracture of left lower first premolar. (**b**) Panoramic radiograph showing successful open reduction and osteosynthesis including tooth removal of fractured tooth. (**c**) Panoramic radiograph showing uneventful bone healing of fractures. (**d**) Dysesthesia of left lower lip area. (**e**) Persisting dysesthesia of left lower lip area 1 year after hardware removal

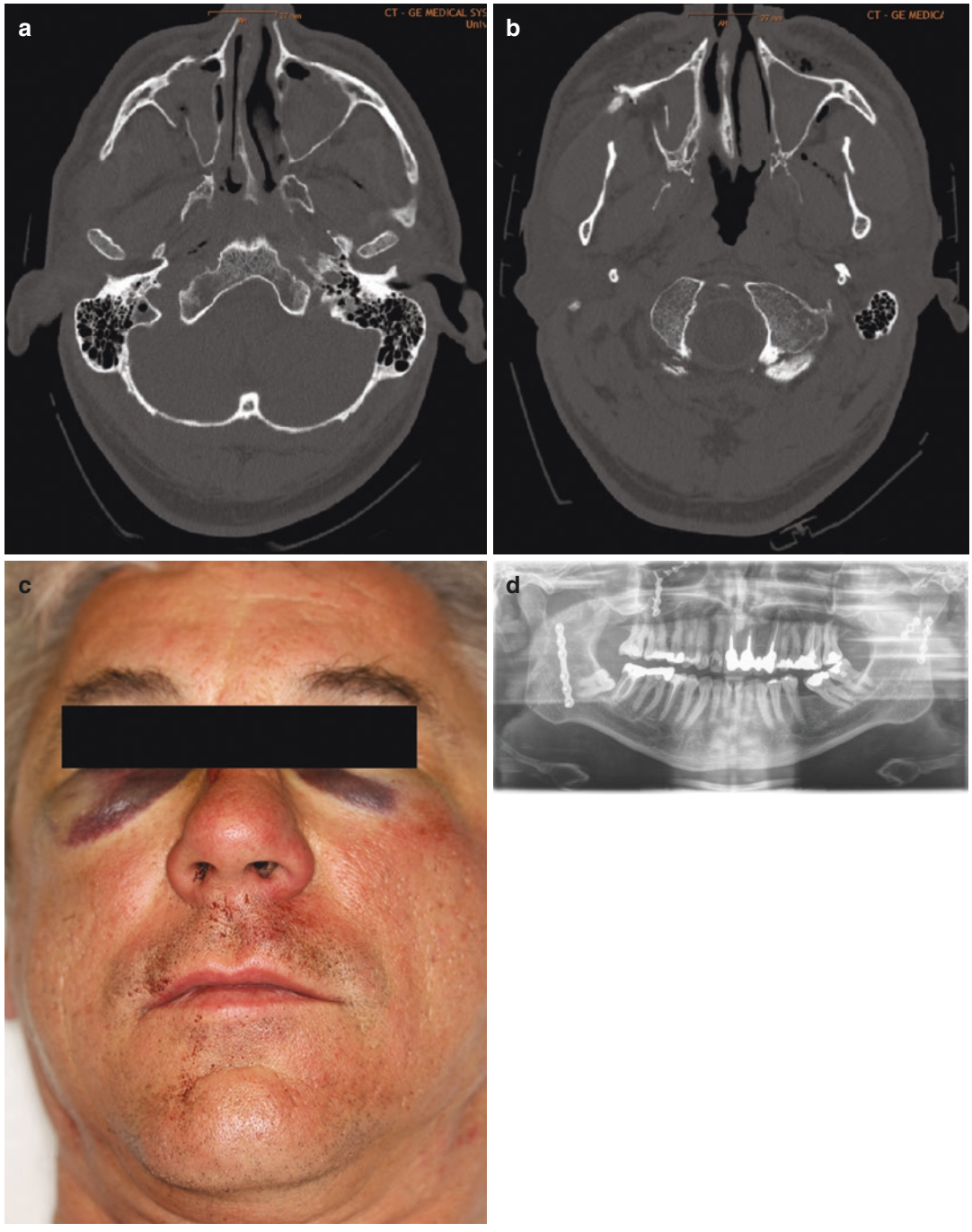


Fig. 9.3 (**a**) Axial CT scan displaying zygoma fracture in the right side panoramic radiograph with radiogenic-induced trism. (**b**) Axial CT scan with left condyle and right ramus fracture of the mandible. (**c**) Binocular hematoma of patient. (**d**) Panoramic radiograph showing successful open reduction and osteosynthesis of mandible and zygoma fractures

Although clinical evaluation shows a stable good occlusion 2 years later he still suffers from complications from his accident, such as sensitive disorders in the area of his right cheek and lower lip including occlusal problems during chewing with TMJ clicking on the left side.

9.3 Complication: Loss of Facial Motion: Facial Nerve

Damage of the facial nerve at the time of the accident is rare even in facial soft tissue trauma. Observing one-sided facial palsy is a clinical sign for the presence of a skull base fracture, especially with otorrhea.

Knowing the anatomy of the facial nerve during surgical access to different parts of the bony mandible is a requirement to avoid damage to the facial nerve. The position of the marginal ramus of the facial nerve needs to be identified, especially during extraoral access to the mandibular body and angle. The presence of postoperative loss of function of the marginal branch of the **facial nerve** is usually attributable to nerve injury during extraoral access for mandibular body fracture repair. A further important complication becomes obvious as a mostly temporary loss of function of the **facial nerve** following condyle fracture repair due to tension caused by hooks to expose the condylar neck intraoperatively. Usually recovery of nerve function takes 1–6 months [23].

Case 4

A 61-year-old female e-biker [13–15] fell into a dip along the road and sustained a high condyle fracture of the mandible on the left side (Fig. 9.4a). Following surgical intervention to reduce the fracture (Fig. 9.4b) and perform rigid

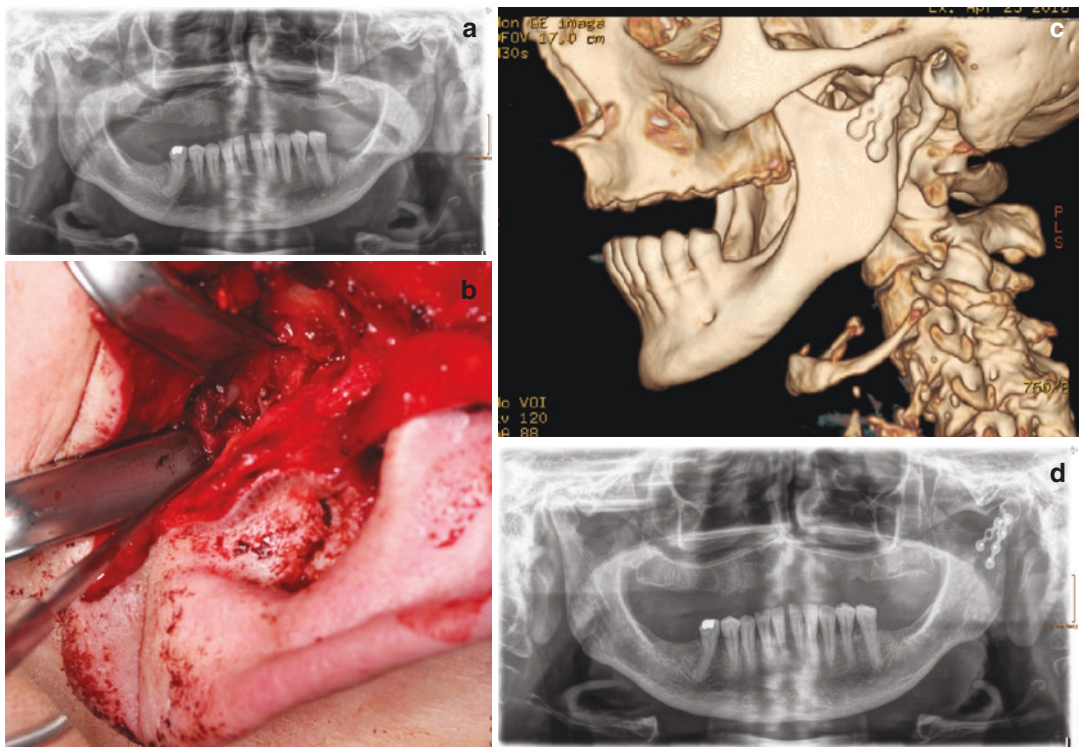


Fig. 9.4 (**a**) Panoramic radiograph displaying left high condyle fracture. (**b**) Intraoperative view of access to the condyle. (**c**) 3D CT image of condyle fracture repair and osteosynthesis. (**d**) Panoramic radiograph with two plates in typical position for condyle fracture repair

fixation with two miniplates, the patient suffered from temporary facial palsy on the left side due to manipulation near the facial nerve. Bone wound healing itself was uneventful (Fig. 9.4c, d).

9.4 Complication: Loss of Teeth and Alveolar Bone

While bone fractures usually heal following a dynamic path without leaving scars, the dento-alveolar process with fractures and luxations of teeth are delicate areas and prone to leaving complications patients may deal all their life. In case of an uncomplicated fracture of a tooth, the pulp is not affected and the tooth can be repaired and restored. A complicated tooth fracture requires removal of the tooth pulp and root channel treatment. A tooth fracture reaching below the gingiva necessitates the removal of the tooth because tooth repair needs dry conditions during tooth conditioning for sealants. Tooth fractures in the apical third of the root may heal.

Tooth luxations include injuries to the alveolar bone socket disturbing the integrity of the periodontal ligament. While contusions of teeth may heal without complications, luxations of teeth require root channel treatment to avoid color changes of the crown. A consequence of successful root channel treatment is loss of the unique plasticity of the tooth. Destruction of the periodontal ligament leads to tooth ankylosis, and bone homeostasis and bone turn over replace dentin with bone leading finally to resorption and loss of teeth.

Severe dental trauma with tooth loss in children and adolescents is usually replaced with removable prosthetic partial dentures as long as growth is occurring in adulthood because inserted implants are known to stay behind while the occlusal plane is changing during growth. The complication of tooth loss in childhood requires as a young adult in a first step the restoration of the alveolar process with bone augmentation and in a second step insertion of implants and implant-borne crown and bridge work as a time-consuming and a financial intensive therapy.

The traumatic loss of teeth and alveolar bone is a severe complication of CMFT in growing patients because implant insertion for full dental rehabilitation has to wait until adulthood [3, 22]. Implant insertion in children and adolescents causes the complication of mismatch of occlusal levels as implants stay in place while surrounding teeth are constantly changing positions due to bone growth.

Case 5

A scooter pilot, 15 years of age, sustained a life-threatening polytrauma including head trauma, blunt thoracic and abdominal with hemodynamic spleen injury, left upper arm fracture, laceration of left upper leg and right knee, and nasal, midface and maxilla fractures including loss of alveolar bone and all four upper incisors with perforation of the lower lip crashing into a truck (Fig. 9.5a) [3, 24, 25].

Following immediate splenectomy and stable osteosynthesis of the upper arm (Fig. 9.5b), fracture facial lacerations and wounds were sutured under general anesthesia (Fig. 9.5c). A removable prosthetic device for replacement of four upper incisors and missing alveolar bone is a complication of the accident until adulthood (Fig. 9.5d). Then, bone reconstruction for the maxilla and subsequent implant placement with implant based crowns allow rehabilitation of the patient in the upcoming years. Scars are visible remains from upper arm trauma (Fig. 9.5e) and splenectomy (Fig. 9.5f).

Case 6

A 28-year-old female stumbled down the staircase and sustained facial bone fractures of the mandible such as condylar head fracture on the right side with dislocation, symphyseal fracture of the mandible, condyle fracture on the left side with displacement, dental trauma with avulsion of all four upper incisors and subluxation

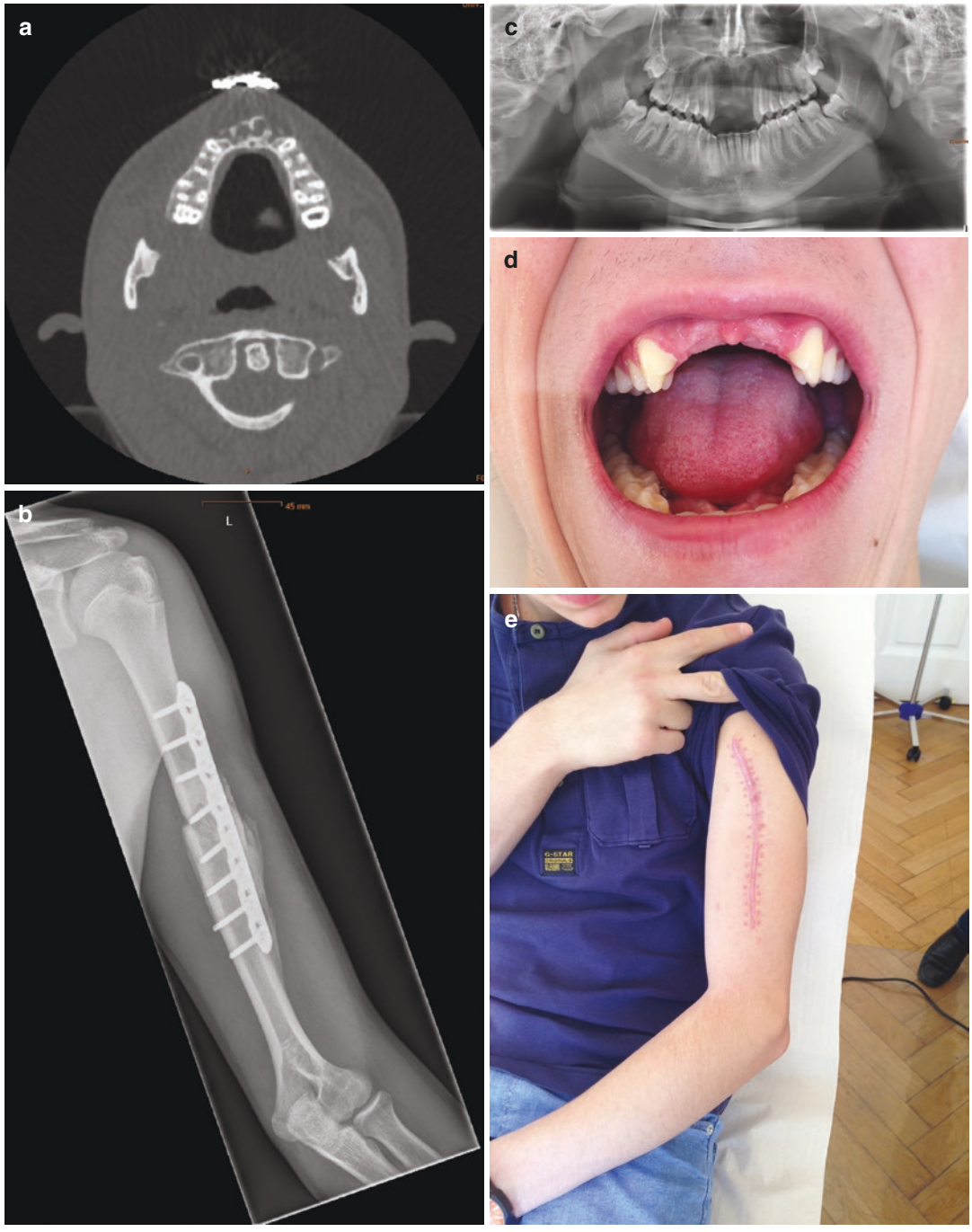


Fig. 9.5 (**a**) Axial CT scan depicting maxillary alveolar bone fractures and avulsion of four permanent incisors. (**b**) Radiograph of left upper arm with osteosynthesis of humerus. (**c**) Panoramic radiograph with missing alveolar bone and upper incisors. (**d**) Clinical view of the adolescent patient. (**e**) Scars of upper arm fracture. (**f**) Abdominal scar after splenectomy

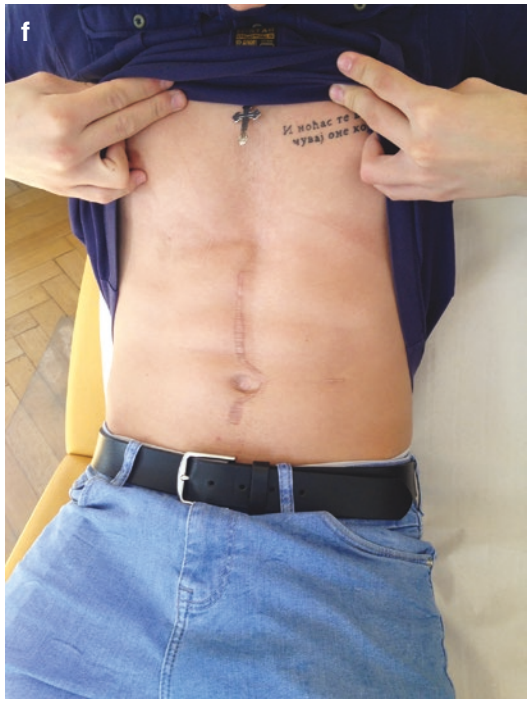


Fig. 9.5 (continued)

of upper right canine, and soft tissue injuries including lacerations of the chin and lower lip (Fig. 9.6a–d).

All mandible fractures were addressed with open reduction and rigid fixation following imprints for upper jaw and lower jaw. All upper incisors had to be excluded from refixation in the maxilla, while the canine was stabilized with a dental splint. Soft tissue injuries were cleaned, rinsed and sutured (Fig. 9.6e–g).

Following surgery, the patient suffered from facial palsy on the left side due to retraction forces unintentionally applied during condyle fracture repair as a temporary complication of surgery (Fig. 9.6h). Facial nerve function fully recovered 3 months later.

To avoid an unsatisfactory outcome of the repair of the condylar head fracture, approximately 3 weeks after surgery guided opening of the mandible was started with an 'activator' on a daily base. Regular recalls every 3 weeks helped to prevent the development of TMJ dysfunction and especially ankylosis (Fig. 9.6i).

The complication of the injury with permanent loss of all four upper incisors needs the use of a removable prosthetic device. Insertion of implants and implant-based crowns is planned.

9.5 Complication: Infection of Fracture Site

Infection of CMFT was a big problem in earlier times. Even today the **infection as a complication** at the fracture site can be starting point of osteomyelitis and pseudarthrosis [26, 27].

First: what to do to avoid the complication:

The occurrence of a fracture always includes the damage of blood vessels at least capillaries. Free blood leads to hematoma formation and is an ideal place for bacteria to proliferate. The oral cavity and its interface with the outside world along the gingival border of teeth are places where a plethora of different bacterial species exists. Facial bone fractures in tooth-bearing areas are known to be prone to infection due to severe bacterial proliferation. Therefore, antibiotic prophylaxis is indicated in all cases. There is evidence that single-shot therapies are correct and sufficient as treatment; still the debate is ongoing [1–10]. Mandibular angle fractures communicating with the oral cavity along partially retained third molars are prone to infection. The debate is long whether existing inflammation such as gingivitis and parodontitis requires tooth removal to reduce the risk of infection of the fracture site [1–10].

Second: how to get treatment back on a good track:

Not every patient is seeking a physician or dentist just because of a fracture. In case of an infected fracture, patients usually seek help and receive treatment. Antibiotic treatment based on an antibiogram and then in addition stable rigid

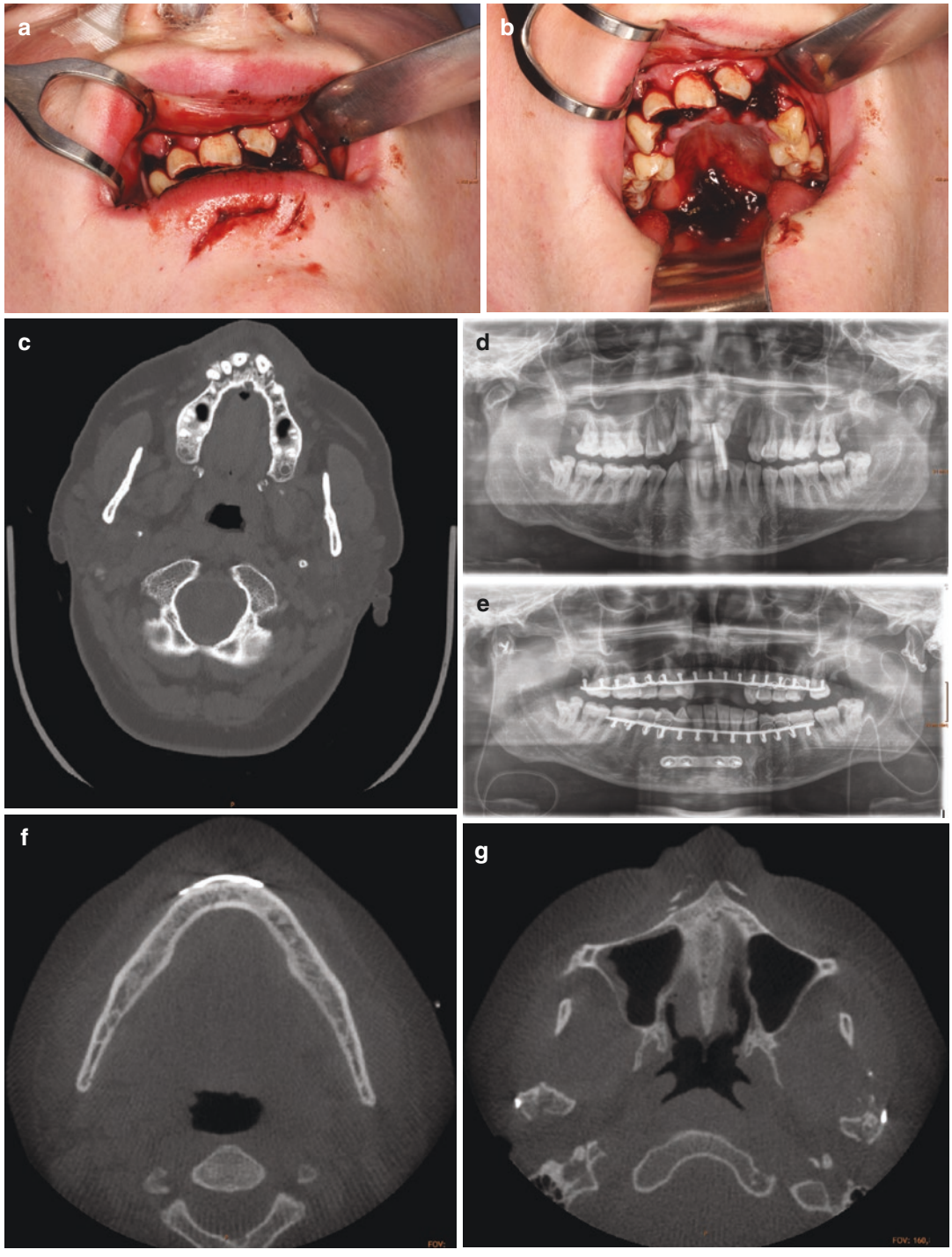
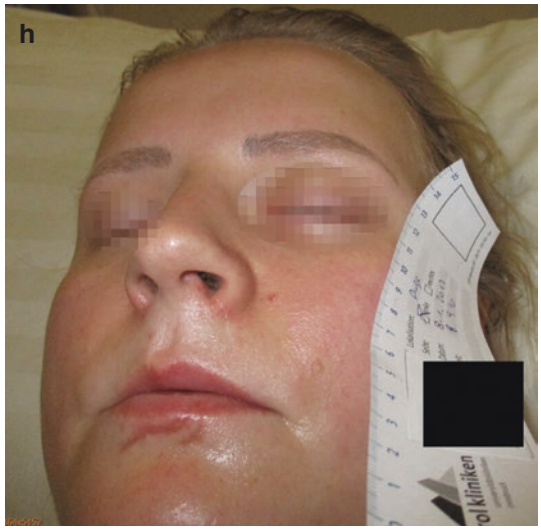


Fig. 9.6 (**a**) Clinical view of dentoalveolar trauma and lower lip laceration. (**b**) Clinical view of dentoalveolar trauma. (**c**) Axial CT scan of dentoalveolar trauma. (**d**) Panoramic radiograph of mandible fractures of symphysis and both condyles and dentoalveolar trauma. (**e**) Postoperative panoramic radiograph of fracture treatment with arch bars, plates and screws. (**f**) Postoperative axial CT scan of mandible with plate in symphyseal region. (**g**) Postoperative CT scan after condyle fracture repair. (**h**) Postoperative temporary facial palsy on left side. (**i**) Panoramic radiograph with functional treatment guiding condyle fracture healing



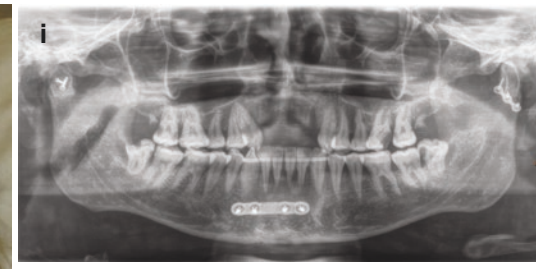


Fig. 9.6 (continued)

fixation is the appropriate treatment. At least, three screws on each side of the fracture line are recommended to stabilize the site.

Case 7

A 48-year-old male mountain biker sustained mandibular fractures of the left condyle and right angle. Following open reduction with rigid fixation of the fractures under general anesthesia, (1) at an outside institution, the patient developed an infection at the right angle 10 days later which was treated with incision and drainage (2). Two months later osteosynthesis material and wisdom tooth on the right side were removed, (3) and intermaxillary fixation was applied to treat the pseudarthrosis using elastic rubber bands for 1 month. Another 14 days later, open reduction and cancellous bone from the iliac crest was inserted into the right angle fracture (4). Four days later, revision surgery of the plate with a screw sticking into the inferior alveolar canal was performed (5).

Finally—2 weeks later—the patient was admitted to our institution (Fig. 9.7a). The patient suffered on his right side from the following complications: pain, loss of motor function of the marginal branch of the facial nerve, loss of sensory function of the lower lip and chin, and facial asymmetry. Despite severe parodontitis, dental decay and calculus, the patient showed a severely reduced dentition (Fig. 9.7b,c). He was a heavy smoker. While it was possible to reduce infection and pain, remove tooth decay and parodontitis via dental extractions and antibiotic therapy and even undergo behavioral changes regarding his smoking habits, the complications such as loss of facial nerve and inferior alveolar nerve function are permanent consequences for the patient (Fig. 9.7d, e).

Case 8

A 53-year-old male was admitted to the hospital with severe swelling and redness of the right orbit (Fig. 9.8a, b) [26, 28, 29]. Radiological evaluation revealed an infected and untreated zygoma fracture with minimal displacement of the orbital floor on the right side and a severely deviated septum (Fig. 9.8c, d). After incision and drainage, drainage of the maxillary sinus, and systemic antibiotic treatment, the zygoma fracture was treated without further surgical intervention or orbital floor repair (Fig. 9.8e).

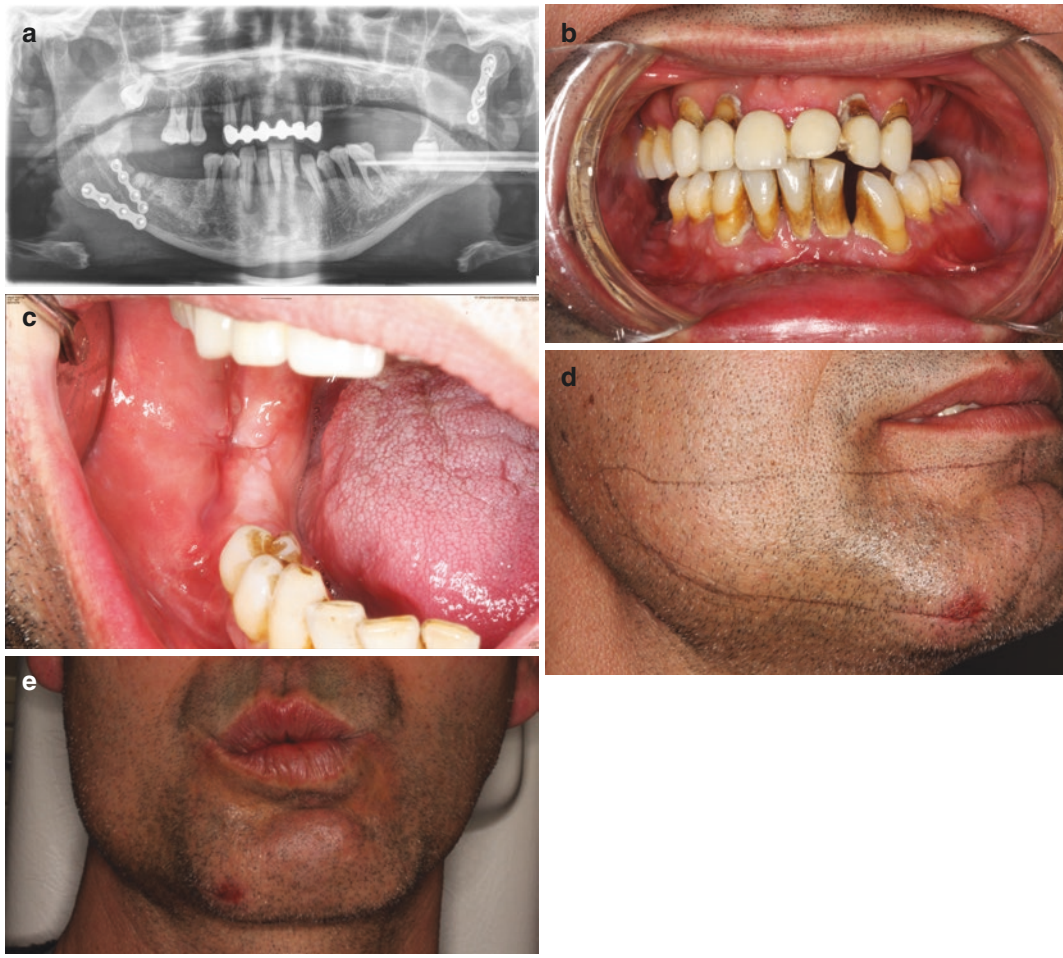


Fig. 9.7 (**a**) Panoramic radiograph after revision surgery for delayed fracture healing. (**b**) Occlusal situation of the patient. (**c**) Intraoral view. (**d**) Dysesthesia of right lower lip and cheek area. (**e**) Loss of marginal facial nerve functions moving the right angle of the mouth

9.6 Complication: Fragment Dislocation Due to Screw Loosening

First: what to do to avoid the complication:

Extensive research and experience exist regarding correct placement of plates and screws. Following open reduction of fractured bones, stable rigid fixation allows function without load. During the postoperative period, there is a permanent risk that patients overload their plated fractures in the healing period. Fluid and soft diet is necessary in the first weeks after surgery. Condyle fractures tend to show slight dislocations in the healing period. Therefore, usually two plates are inserted along the posterior border and the anterior edge toward the coronoid process.

Second: how to get treatment back on a good track:

In case of clinical or radiological signs of dislocations following successful open reduction

and rigid fixation of condyle fracture, the use of intermaxillary fixation either with IMF screws or arch bars for a month stabilizes function.

Because the main growth center of the mandible is represented in the condyle, it is possible to guide children to normal TMJ and mandibular function even in the severe condylar neck and head trauma. After a short period of around 14 days of consolidation of the traumatized area, a specially formed activator restores TMJ function to avoid TMJ ankylosis. To avoid complications in patients, they are on recall for suture

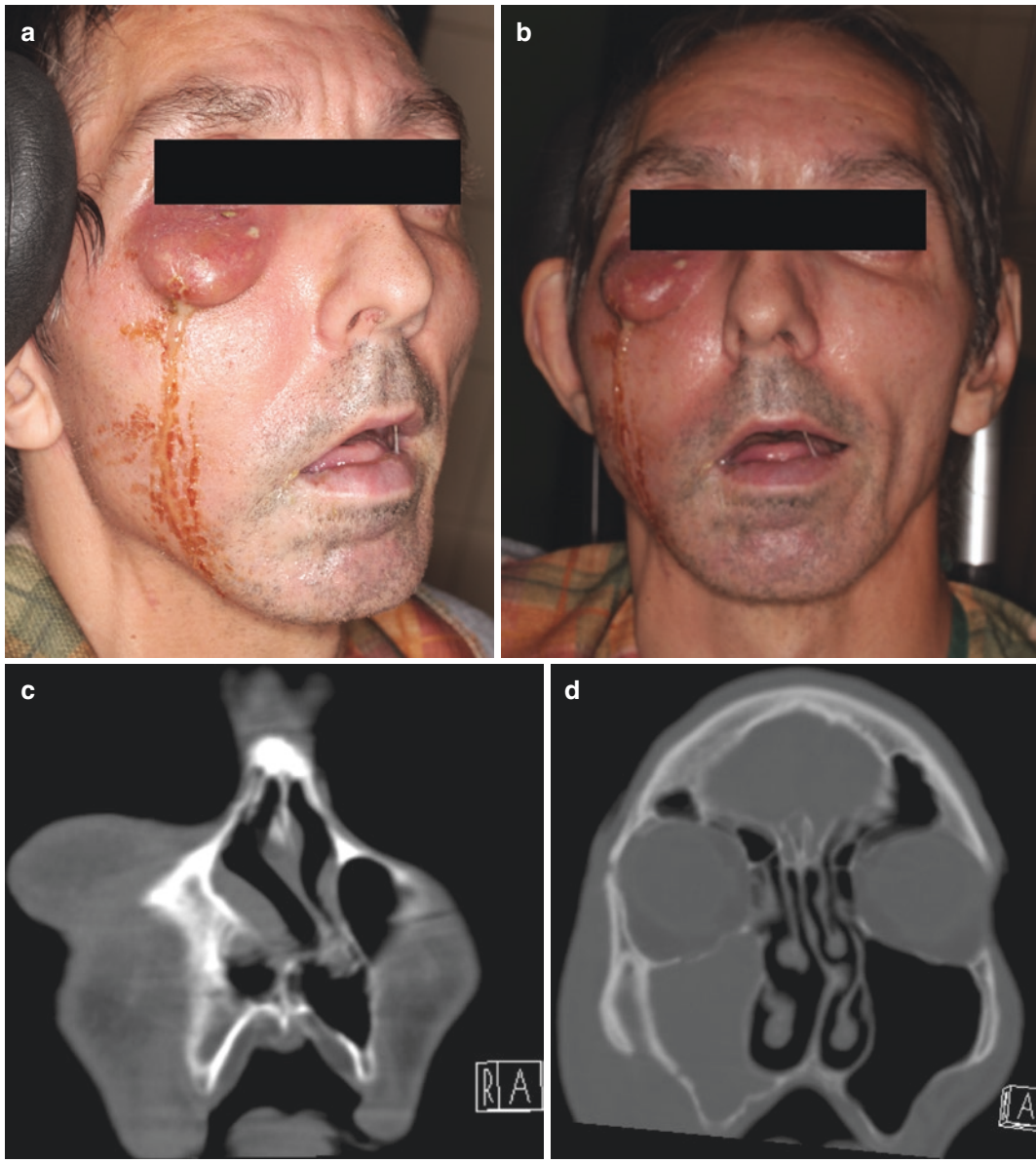


Fig. 9.8 (**a**) Side view of infected zygoma fracture. (**b**) Frontal view of infected zygoma fracture. (**c**) Coronal CT scan: soft tissue swelling empyema of maxillary sinus. (**d**) Coronal CT scan: empyema of maxillary sinus. (**e**) Infraorbital and nasal drainage of maxillary sinus

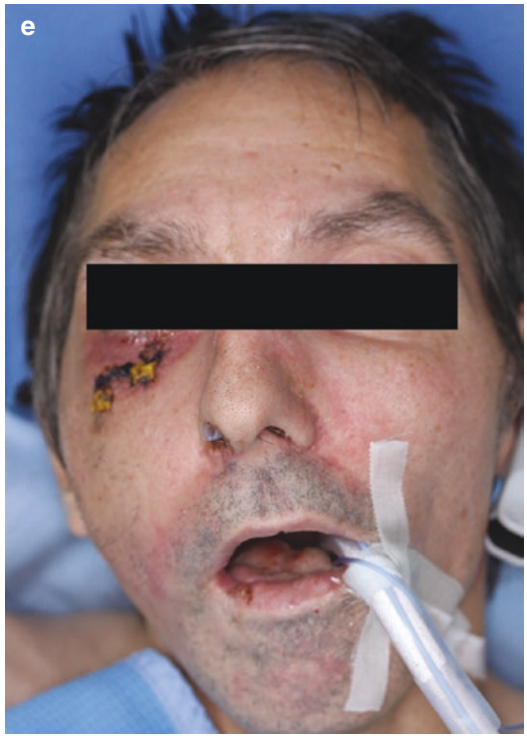


Fig. 9.8 (continued)

removal, then after 3 weeks, 6 weeks, 3 months, 6 months and 1 year after trauma to allow healing without complications.

While condyle fractures are successfully treated with two miniplates, a four hole plate at the posterior border and a three hole plate at the ventral border, there is still a risk of **dislocating the condyle** slightly **during the healing process due to screw loosening**. Therefore, postoperative recalls after 6 weeks, 3 months, 6 months, and 1 year after ORIF are highly recommended to deal with and avoid occlusal or TMJ-related problems in every trauma case.

Case 9

A male patient, 48 years of age, fell down the staircase [26, 27]. The panoramic radiograph revealed a dislocated condyle fracture above a healed mandibular angle fracture (Fig. 9.9a). He was surgically treated with open reduction and rigid fixation using a three hole miniplate with two screws anteriorly and a four-hole miniplate with four screws posteriorly in correct anatomic position and at the same time the osteosynthesis plate at the left mandibular angle was removed (Fig. 9.9b).

A year later he complained that he suffers frequently from pain on the left side. The panoramic radiograph showed a reduction of condylar height on the left side, screw loosening of the anterior upper screw, and in addition a tiny metallic part (Fig. 9.9c) which was not present before. So the plates were removed (Fig. 9.9d), but the tiny metallic part stayed, being visible within the previous fracture line.

Case 10

Walking his dog a male person 59 years of age stumbled when the animal suddenly pulled its leash. He fell on his mandible and sustained a left condyle and a right mandibular body fracture (Fig. 9.10a). Following successful surgical treatment of the fractures, the patient was discharged the third postoperative day (Fig. 9.10b).

Despite the recommendation of staying on a soft diet, the patient complained that he is now suffering from pain in his left condyle 2 months later. Radiologic evaluation confirmed the complication of displacement of the reduced condyle due to screw loosening of inserted screws (Fig. 9.10c). The patient was treated with intermaxillary maxillomandibular rubber bands to achieve condyle fracture healing (Fig. 9.10d).

The bone healing of the left condyle resulted in a slightly reduced height compared to the original length showing a sliding of 1 mm occurred between initial touching of opposing teeth and the final position of occlusion (Fig. 9.10e, f). TMJ clicking is present, causing sudden sensations of pain during motion. In addition, the patient suffers from reduced sensitivity in the right lower lip [16].

9.7 Complication: Hardware Failure

Case 11

A 20-year-old patient had suffered a right condyle and symphyseal fracture of the mandible which was surgically addressed with two plates

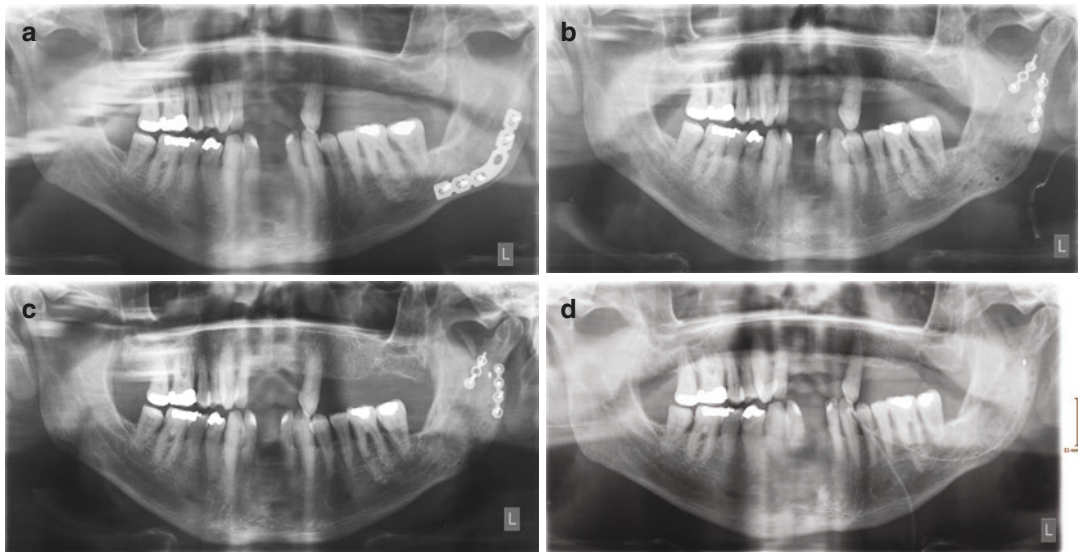


Fig. 9.9 (**a**) Panoramic radiograph of a dislocated condyle fracture in a healed mandibular angle fracture (left side). (**b**) Panoramic radiograph after open reduction and rigid fixation of the condyle fracture and plate removal of healed angle fracture site. (**c**) Panoramic radiograph displaying loss of condylar height due to screw loosening and metallic artifact 1 year later. (**d**) Panoramic radiograph after plate removal and persistence of metallic artifact

in the right condyle region from a preauricular approach and another plate in the midline of the mandible from an intraoral approach (Fig. 9.11a).

Three years later, the patient showed mandible fractures affecting the left condyle and again symphyseal fracture of the mandible, including loss of the middle lower left incisor (Fig. 9.11b). A tongue piercing is also depicted.

The treatment was open reduction and internal rigid fixation with two plates on the left condyle and two plates in the midline of the mandible (Fig. 9.11c).

Despite successful fracture healing, root channel treatment of the middle upper central incisors is visible 1 year later (Fig. 9.11d).

Again the now 28-year-old patient was admitted to the hospital the third time. A panoramic radiograph revealed three fracture sites of the mandible where years ago already fractures in similar locations were surgically treated. Despite fracture lines, also fractured plates are visible (Fig. 9.11e). Moreover, he showed a neglected dentition with multiple lesions with caries. As a drug-addicted person, he did not accept to take necessary medication to avoid seizures. Subluxation of his frontal teeth complicated the situation (Fig. 9.11f).

In addition, reoperation was not feasible as he did not obey the required avoidance of food intake prior to anesthesia. So based on an imprint of the mandible, an individual model was created and lingual and buccal individual splints were inserted to stabilize the mandible fracture in the midline. Upper teeth were splinted with a customary splint (Fig. 9.11g, h).

Panoramic radiographs document the healing process (Fig. 9.11i, j).

9.8 Complication: Intra- and Periorbital Region, Temporary Vision Loss [30–35]

First: what to do to avoid the complication:

The orbital socket is housing extremely important structures for an individual's full function. A force intruding the orbit often results in an orbital floor fracture to avoid damage to the eyeball. In children, the elastic bone may entrap the inferior

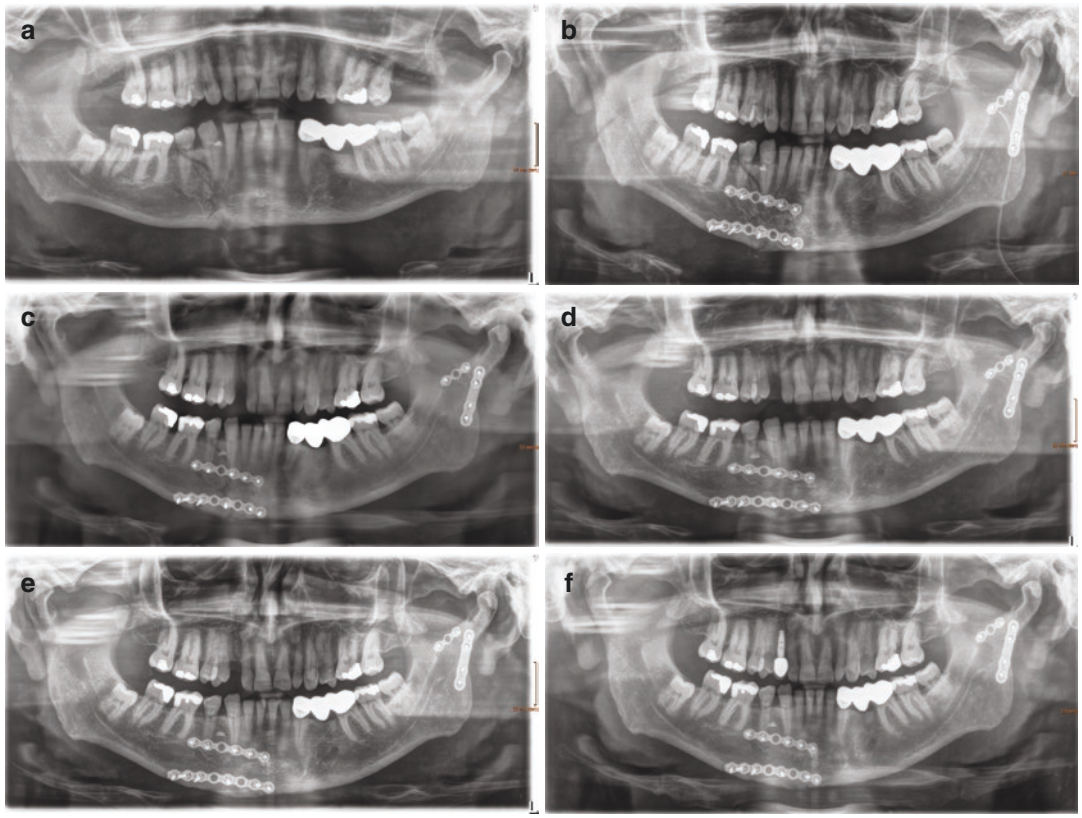


Fig. 9.10 (**a**) Panoramic radiograph with left condyle and right mandibular body fracture. (**b**) Panoramic radiograph after open reduction and rigid fixation of fractures. (**c**) Panoramic radiograph with reduced condylar height due to screw loosening 2 months later. (**d**) Panoramic radiograph with insufficient fracture healing despite reduced condylar height. (**e**) Panoramic radiograph with gradual improvement of bone healing with reduced condylar height. (**f**) Panoramic radiograph in healed position and reduced condylar height

eye muscle causing double vision. This situation requires immediate release of the entrapped muscle. Despite the eye, its muscles and nerves may be hampered due to injury. Especially, the development of a retrobulbar hematoma may lead to vision loss and needs to be ruled out in any fracture involving the orbital socket. Exophthalmos, hard globe, and severe pain are clinical signs to be ready for immediate enlargement of the retrobulbar space with downfracturing of the orbital floor to save eye vision.

Case 12

A 22-year-old male was hit by a water filled airborne missile causing a massive injury of the right orbit and globe. He sustained a severe orbital floor and medial orbital fracture (Fig. 9.12a).

He underwent orbital floor repair with a resorbable patch (Fig. 9.12b, c).

In the postoperative CT evaluation, it was clear to restore the orbital socket using 3D navigation (Fig. 9.12d). CT data were analyzed and a mirror image of the left orbital socket was created to realign the right orbital socket in correct dimensions. An individually shaped titanium mesh was inserted (Fig. 9.12e–g). Postoperative CT shows the correct positioning of the titanium mesh (Fig. 9.12h, i).

Initial diplopia improved within weeks. Eye movements in all directions returned normal (Fig. 9.12j–m). As a permanent complication of the incident a traumatic widening of the right pupil and a slightly higher positioning of the right globe remain due to the inserted mesh (Fig. 9.12n, o).

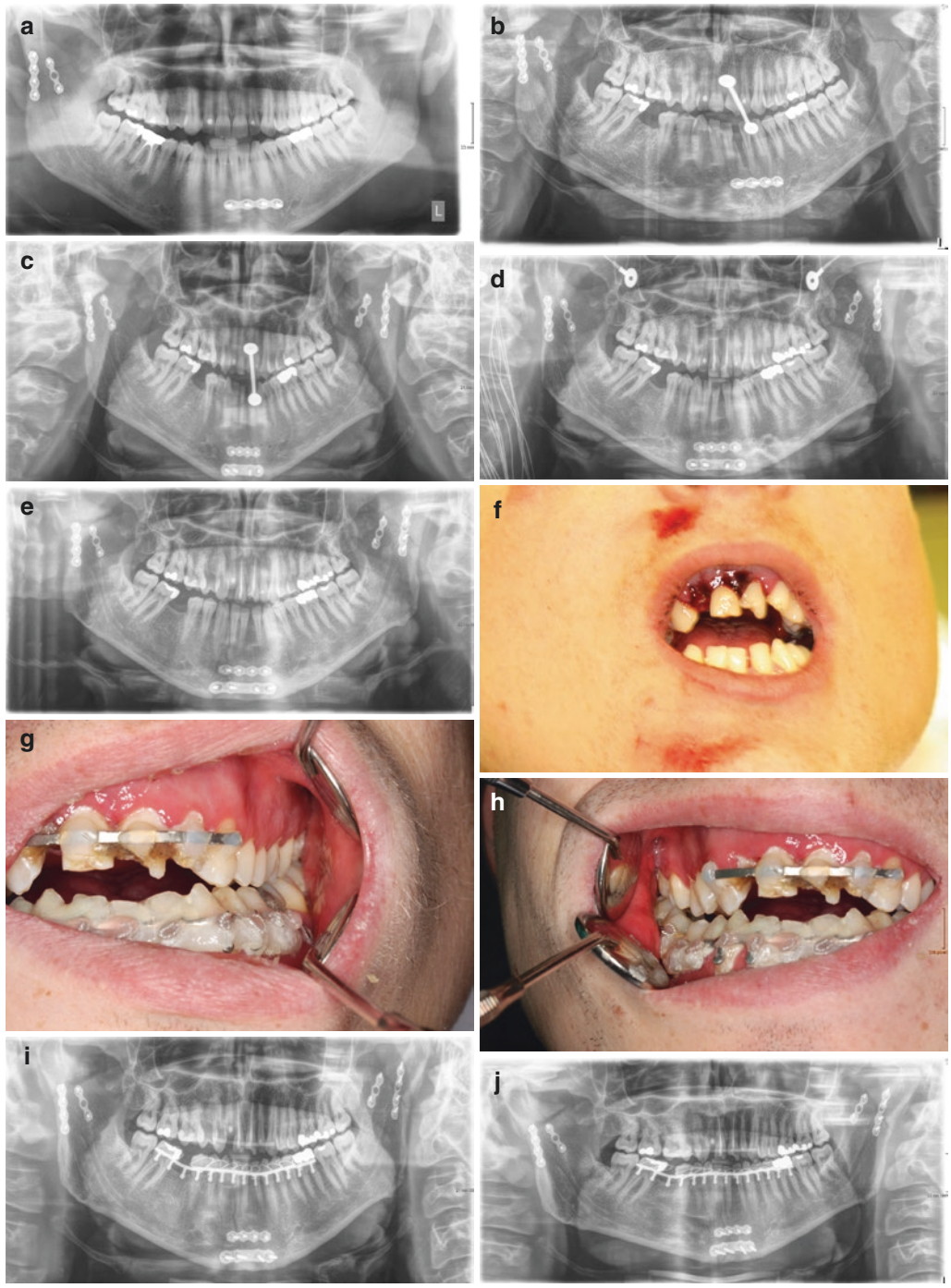


Fig. 9.11 (**a**) Panoramic radiograph after open reduction and rigid fixation of a right condyle and left parasymphyseal body fracture. (**b**) Panoramic radiograph with left condyle and symphyseal body fracture 3 years later. (**c**) Panoramic radiograph after open reduction and rigid fixation of a left condyle and symphyseal body fracture. (**d**) Panoramic radiograph showing successful bone healing 1 year later. (**e**) Panoramic radiograph again reveals three fractures sites with fractured plates. (**f**) Clinical view showing subluxation of upper frontal teeth. (**g**) Clinical view of splinted upper frontal teeth and arch bar in the mandible for non-operative treatment (left side). (**h**) Clinical view of splinted upper frontal teeth and arch bar in the mandible for non-operative treatment (right side). (**i**) Panoramic radiograph of splinted upper frontal teeth and arch bar in the mandible. (**j**) Panoramic radiograph of splinted upper frontal teeth and arch bar in the mandible 8 weeks later

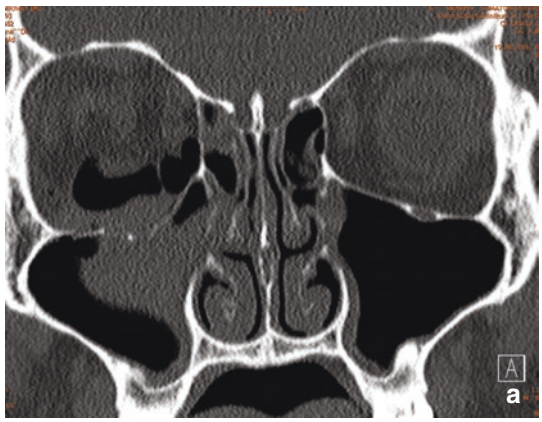
Case 13

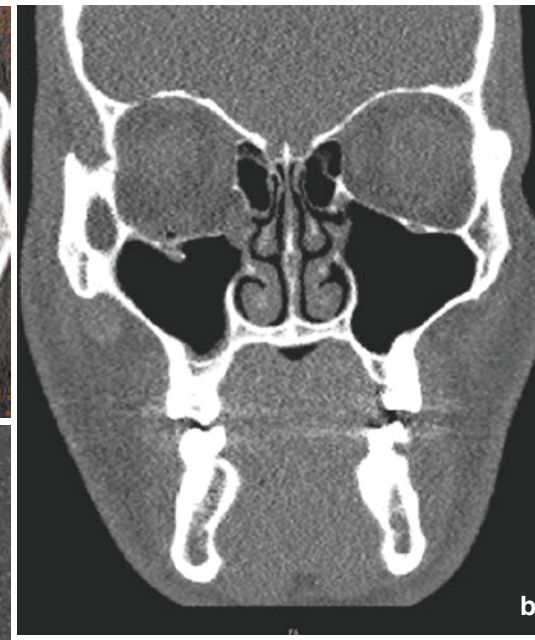
A 25-year-old male was exposed to an act of violence with a fist and sustained a zygoma and orbital floor fracture on his left side. CT scans also show an old orbital floor fracture on the right side (Fig. 9.13a–d). On the third day after the incident, he was treated with open reduction and restoration of the orbital floor using a resorbable patch. In the recovery room, he reported as a major complication complete loss of vision of his left eye. He was immediately taken back to the operating room. The inserted patch was removed to release hematoma formation and lateroorbital and infraorbital closed incisions were Kept open and drained (Fig. 9.13e, f).

The patient recovered and his vision of his left eye was rescued. Bone wound healing itself was uneventful (Fig. 9.13g).

Case 14

A 19-year-old male quad pilot sustained a severe traumatic brain injury with epidural bleeding, cranial vault fracture, and retrobulbar hematoma of the left orbital socket. At admittance, he





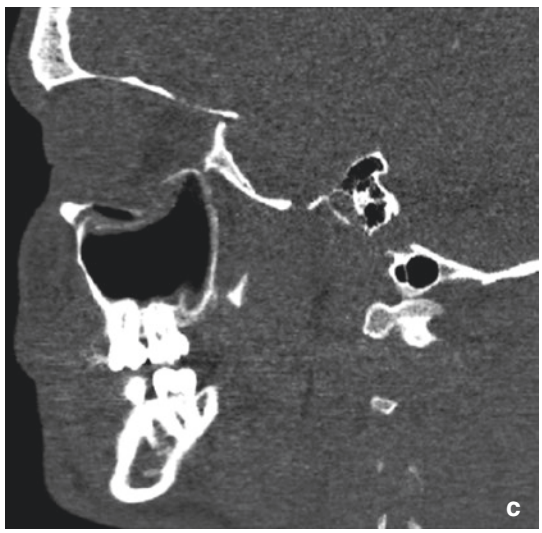


Fig. 9.12 (**a**) Coronal CT scan: right orbital floor fracture with severe diplopia. (**b**) Coronal CT scan: unsuccessful repair of the orbital floor with resorbable patch. (**c**) Sagittal CT scan: unsuccessful repair of the orbital floor with resorbable patch. (**d**) 3D Navigation planning. (**e**) Surgical approach. (**f**) Removal of resorbable mesh. (**g**) Insertion of titanium mesh. (**h**) Coronal CT scan: anatomic repair of the orbital floor with titanium mesh. (**i**) Sagittal CT scan: anatomic repair of the orbital floor with titanium mesh. (**j**) Upward eye movement. (**k**) Eye movements to the left. (**l**) Downward eye movement. (**m**) Eye movements to the right. (**n**) Trauma-induced wider right pupil. (**o**) Higher positioning of right globe

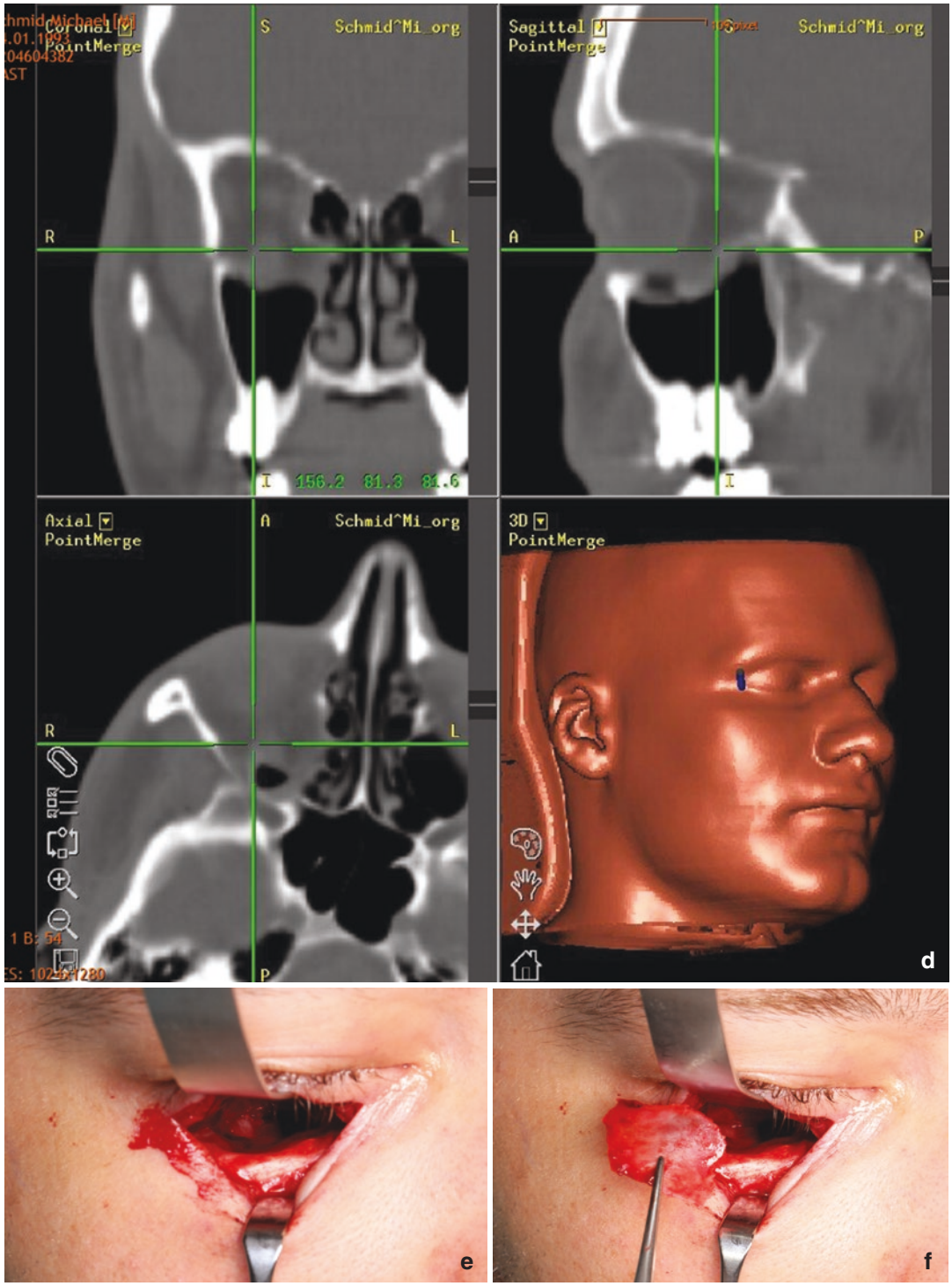


Fig. 9.12 (continued)

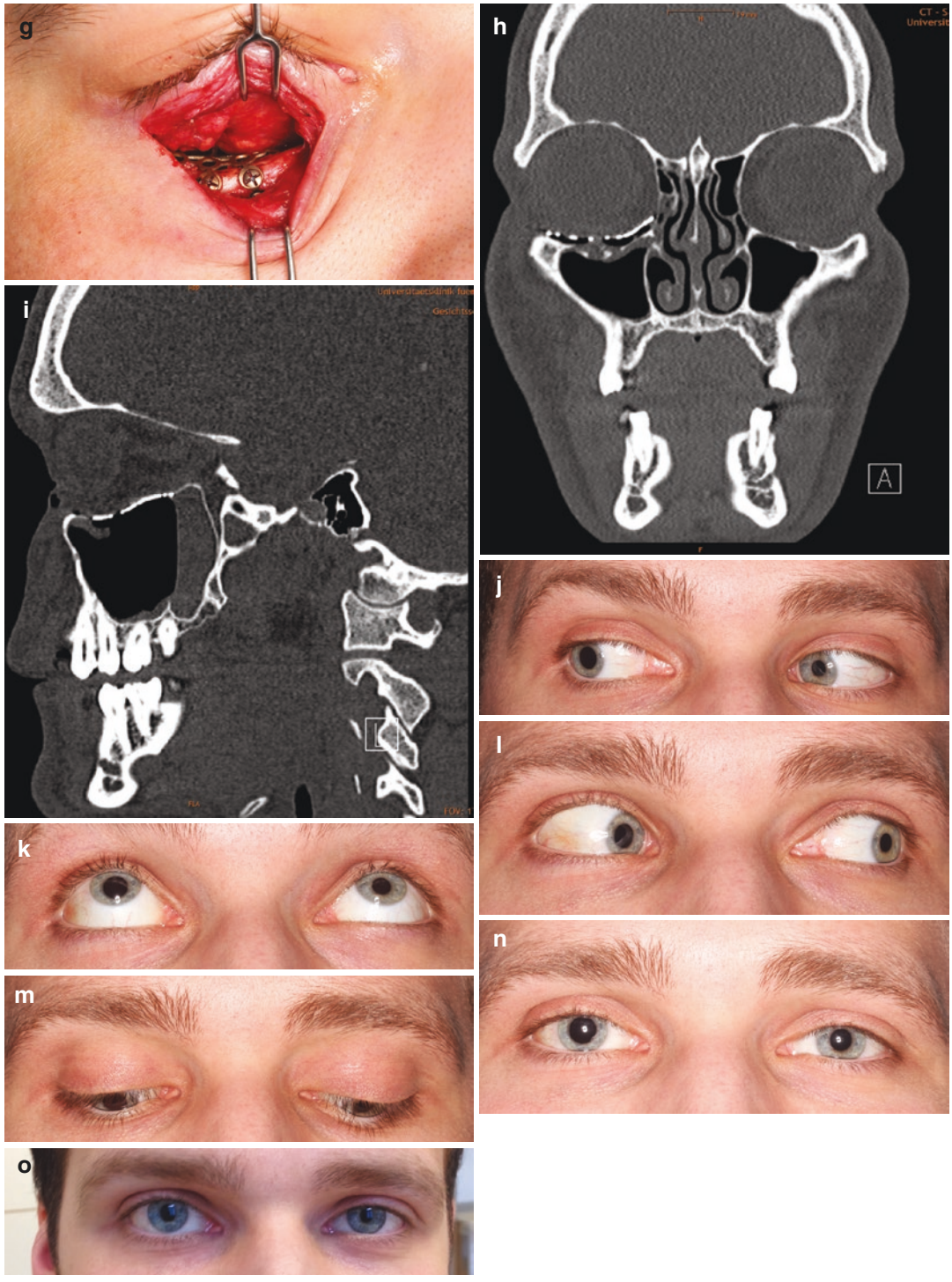


Fig. 9.12 (continued)

showed increasing dizziness and double vision. Despite craniotomy to reduce intracranial pressure, decompression of the left orbit was performed to avoid loss of vision due to the stretched ocular muscles as an alarming sign of impairment (Fig. 9.14).

Case 15

A 20-year-old male farmer was hit by a 500 kg heavy hey pellet creating a head brain trauma with intracranial hemorrhage, skull base fracture including a complex fracture of the orbital roof, comminuted fractures of the posterior wall of the

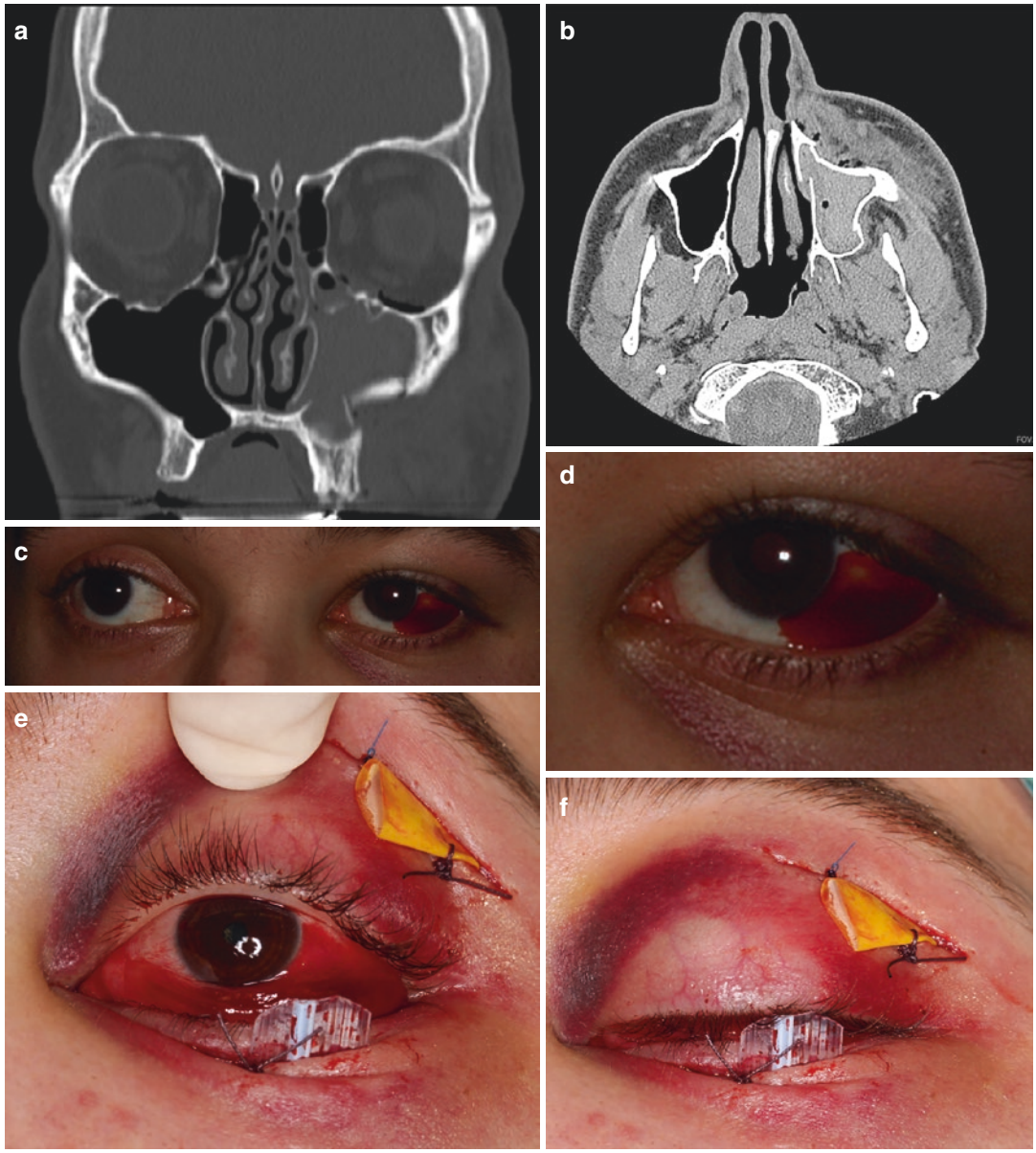


Fig. 9.13 (**a**) Coronal CT scan: left zygoma and orbital floor fracture and old orbital floor fracture. (**b**) Axial CT scan: left zygoma fracture. (**c**) Panoramic radiograph with radiogenic-induced trism. (**d**) Clinical image of both eyes. (**e**) Clinical image of affected eye. (**f**) Drainage of intraorbital hematoma lateroorbital and infraorbital (eye open). (**g**) Drainage of intraorbital hematoma lateroorbital and infraorbital (eye closed)

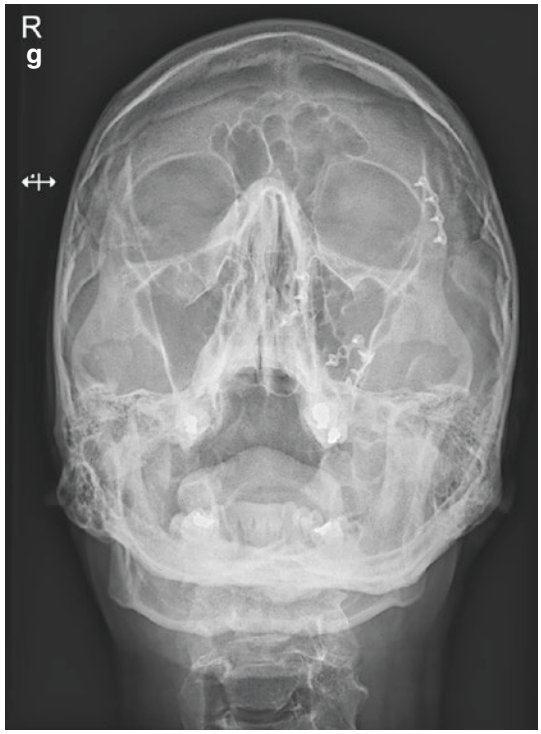


Fig. 9.13 (continued)

frontal sinus sphenoid fracture and intraorbital hamorrhage on the left side which deteriorated in the first hours upon arrival at the intensive care unit (Fig. 9.15a, b).

Due to the growing retrobulbar hematoma with a clear reduction of the activity of the oculomotorius, nerve enlargement of the orbital socket was mandatory to reduce the pressure to the globe with resulting loss of vision. Blood drainage to the maxillary sinus due to the creation of an orbital floor fracture allowed to stabilize the patient's visual function.

Ten days later, orbital floor repair with a resorbable patch led to full recovery of vision for the left eye.

9.9 Complication: Vision Loss [30–35]

Case 16

A 43-year-old male sustained a severe orbital trauma due to a metallic foreign body ejected from a chisel which was hit from a hammer at work (Fig. 9.16a–c). The globe itself was also injured and resulted in loss of vision as a permanent complication due the injury (Fig. 9.16d). The orbit was explored, and the foreign body was removed. An artificial lens was inserted and the retina was coagulated using a laser.

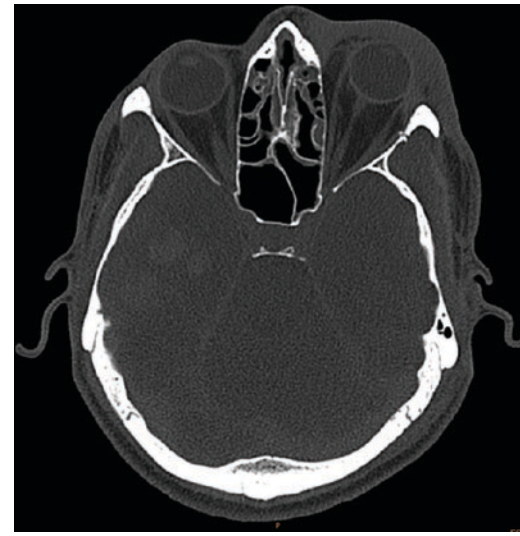


Fig. 9.14 Axial CT scan depicts typically stretched ocular muscles of left orbital socket

Case 17

A 18-year-old male sustained a self-inflicted gunshot to his right orbit (Fig. 9.17a, b). Although surgical intervention occurred in a rapid fashion (Fig. 9.17c), the complication of vision loss to his right eye is permanent (Fig. 9.17d–f).

9.10 Complication: Skull Base Fracture and Intracranial Hemorrhage [5, 9, 36–42]

First: what to do to avoid the complication:

Every patient who sustained head trauma needs to be observed after the incident. Nausea, vomiting, and seizures are clear clinical signs hinting on intracranial injury such as bleeding around and within the brain. CT scan and control CT scans 24 h later are known to rule out the consequences of skull base fractures and intracranial bleeding avoiding complications such as brain damage and even death.

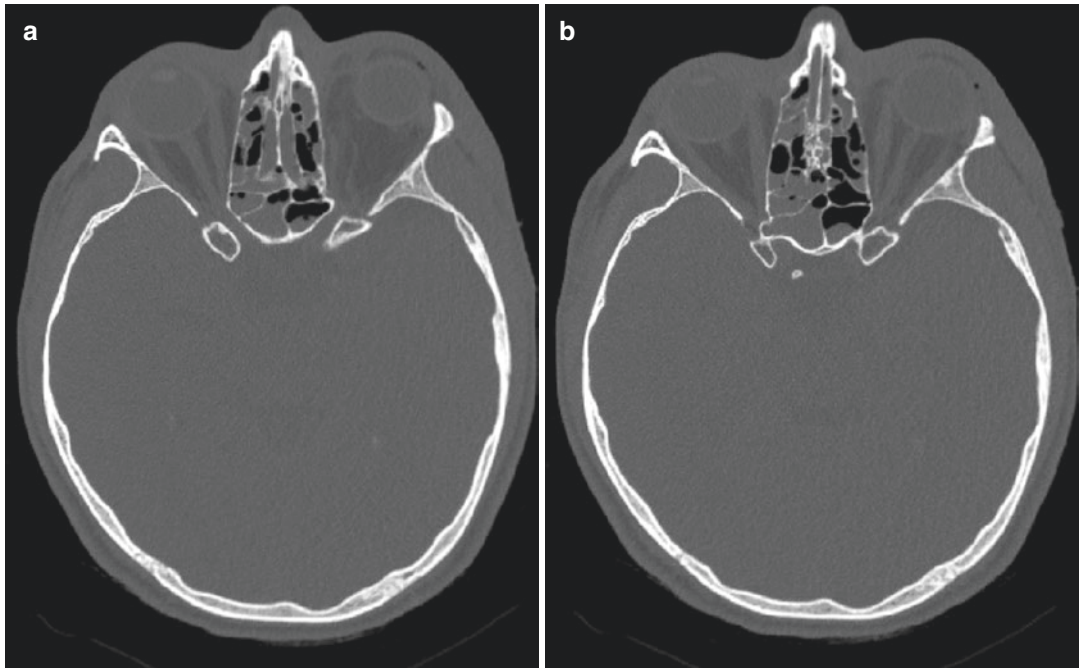


Fig. 9.15 (**a**) Axial CT scan depicts retrobulbar hematoma of the left orbital socket. (**b**) Axial CT scan depicts typically stretched ocular muscles of left orbital socket

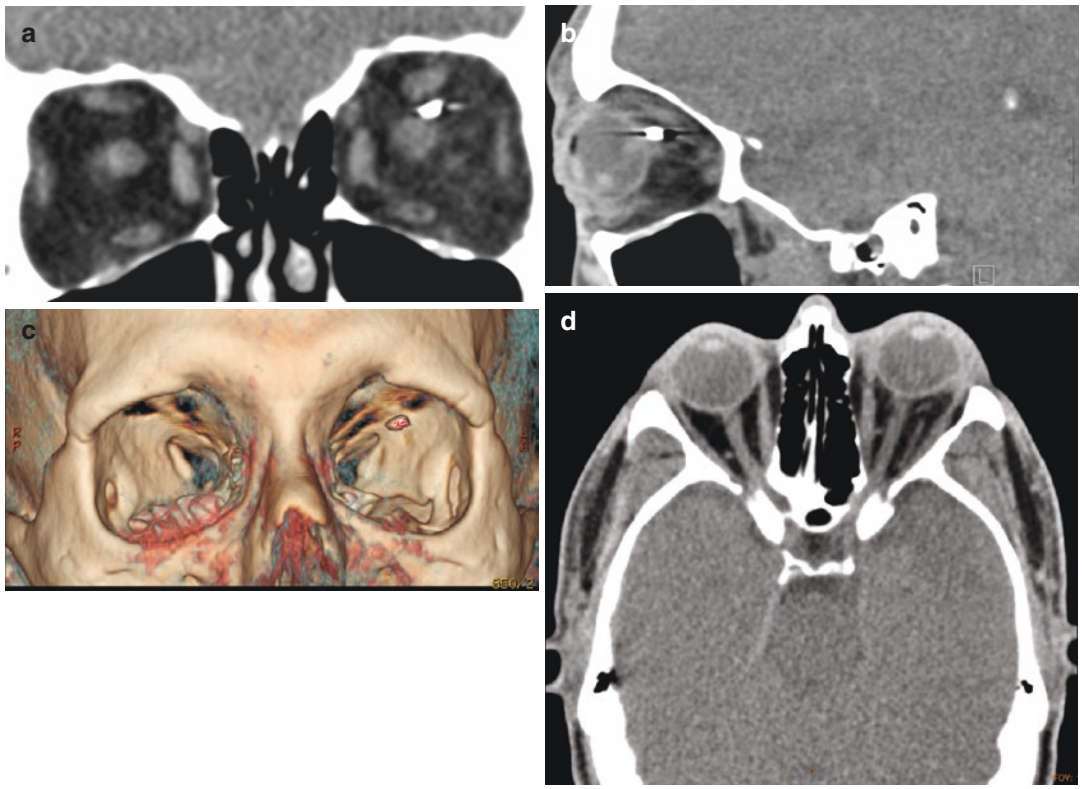


Fig. 9.16 (**a**) Coronal CT scan: metallic foreign body in the left orbital socket. (**b**) Sagittal CT scan: metallic foreign body behind the globe. (**c**) 3D image of the metallic foreign body in the orbital socket. (**d**) Axial CT scan after removal of foreign body

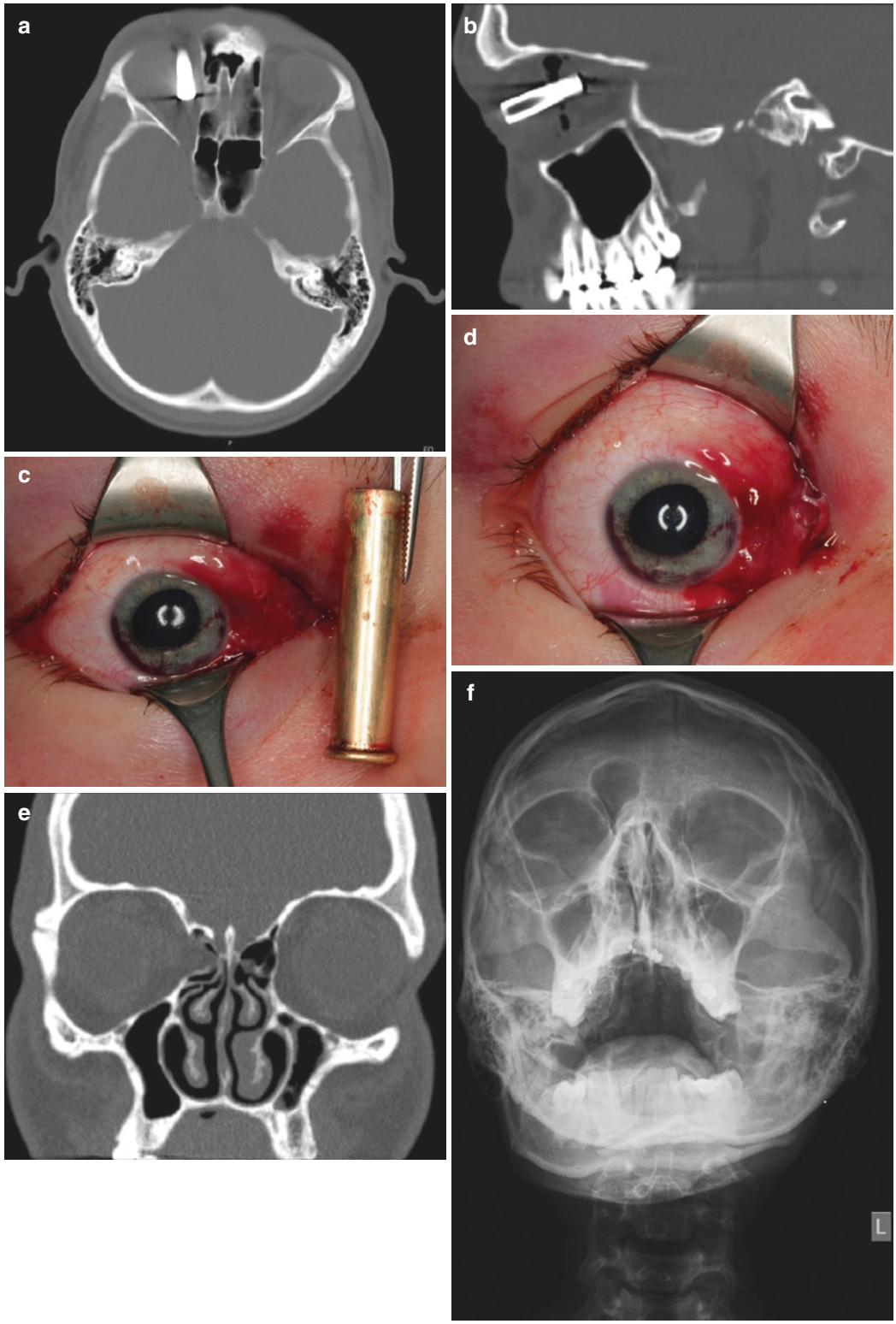
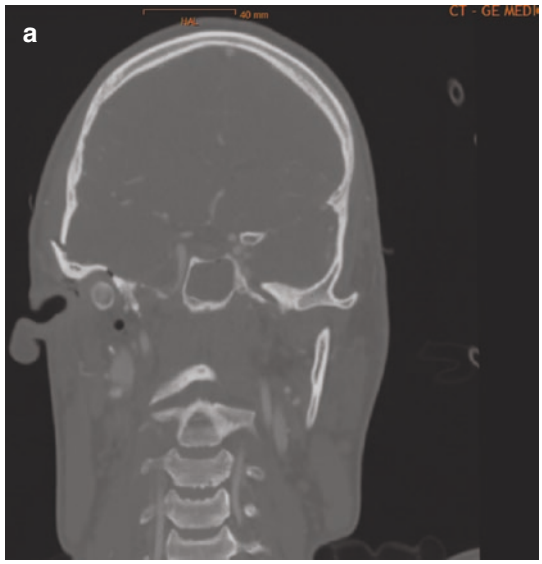


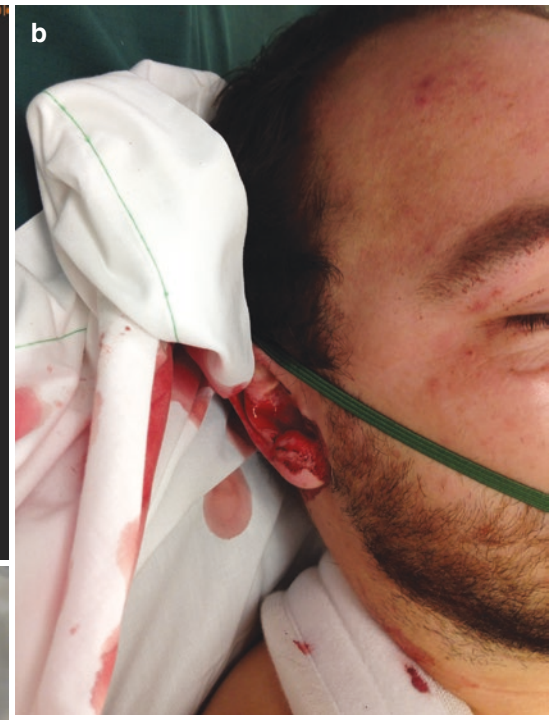
Fig. 9.17 (**a**) Axial CT scan with foreign body (bullet) in the right orbital socket. (**b**) Sagittal CT scan with foreign body (bullet) in the right orbital socket. (**c**) Clinical image with bullet retrieved. (**d**) Clinical image without bullet. (**e**) Postoperative coronal CT scan with medial orbital wall fracture. (**f**) Plain radiograph of head

Second: how to get treatment back on a good track

Case 18

A 24-year-old male was in ApreSki mood and jumped down a wall of 3 m. He sustained a fracture of the glenoid fossa of the TMJ including the skull base and a phase of unconsciousness due to intracranial hemorrhagic signs (Fig. 9.18a). He also showed otorrhea and ear bleeding from the right ear as clinical sign (Fig. 9.18b). Due to a good occlusion including the molar areas, conservative treatment was the best option (Fig. 9.18c). Initial right facial palsy as a complication from the skull base fracture resolved during his stay at the recovery unit for 2 weeks.





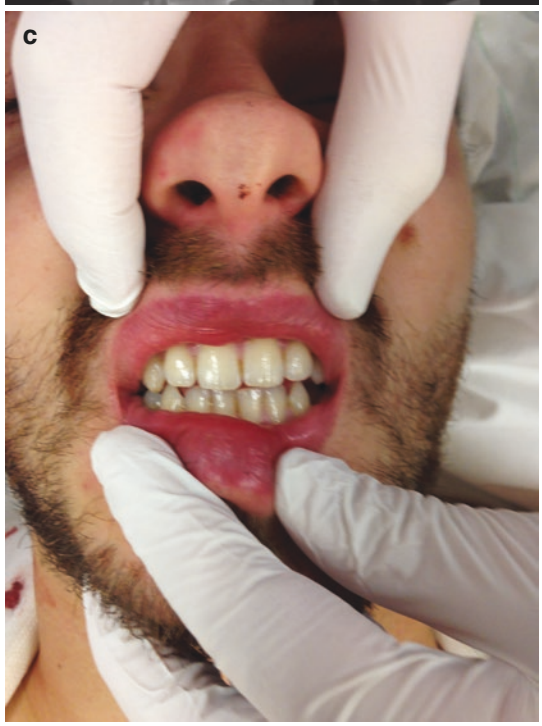


Fig. 9.18 (**a**) Coronal CT scan depicting fracture of the glenoid fossa of the TMJ. (**b**) Clinical sign of otorrhea. (**c**) Occlusal situation

Case 19

A 58-year-old male fell with his bicycle and sustained multiple facial soft tissue injuries (Fig. 9.19a–c), dental trauma (Fig. 9.19d, e), and a 10 cm long laceration along the right sternocleidomastoid muscle (Fig. 9.19f). Radiologic evaluation revealed a dissection of the right internal carotid artery resulting in an aneurysm

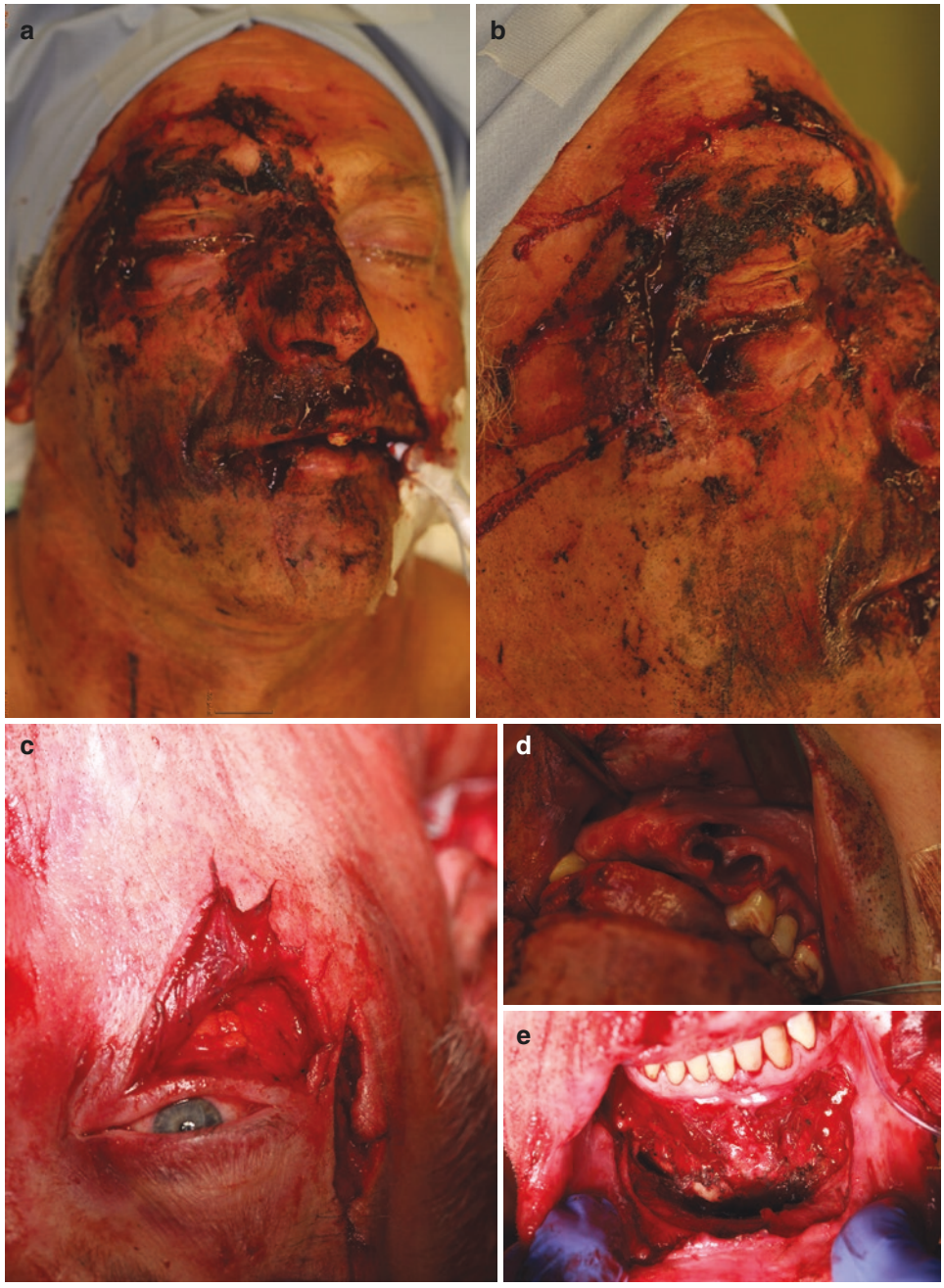
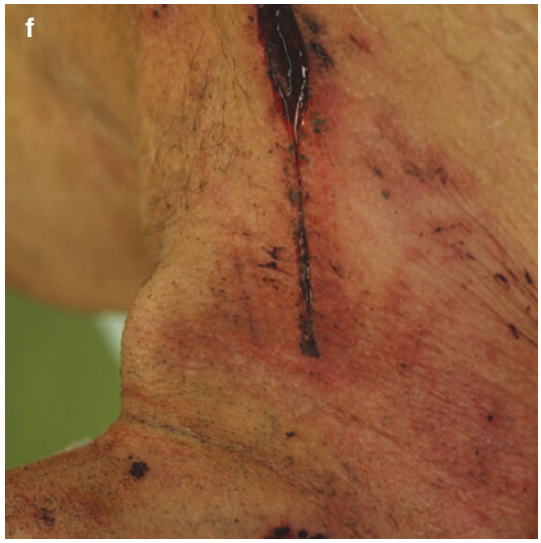
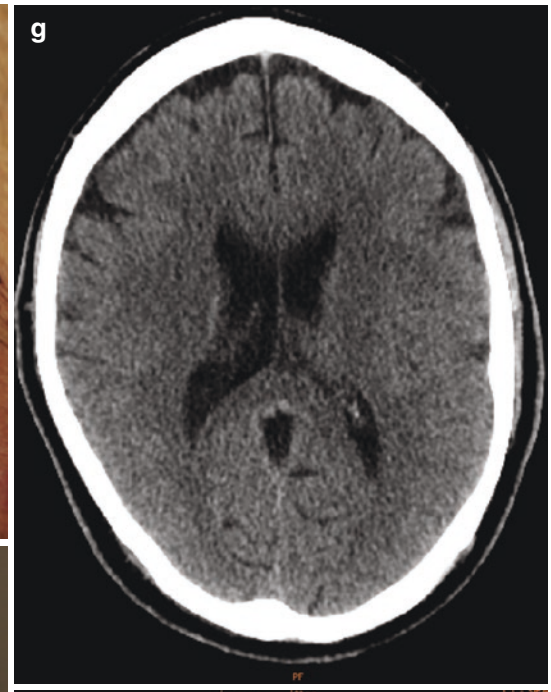
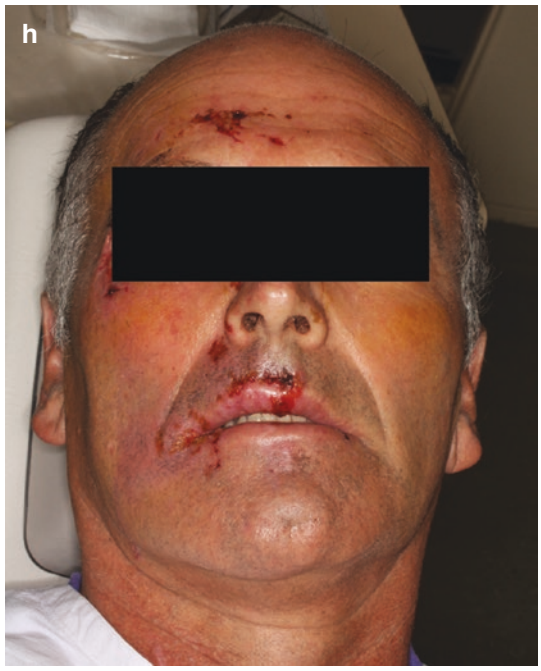


Fig. 9.19 (**a**) Clinical image: soft tissue trauma. (**b**) Clinical image: soft tissue trauma. (**c**) Clinical image: periorbital lacerations. (**d**) Clinical image: dental trauma. (**e**) Clinical image: intraoral laceration. (**f**) Clinical image: laceration along sternocleidomastoid muscle. (**g**) Axial CT scan: dissection of internal carotid artery created aneurysm. (**h**) Clinical image: at discharge after 1 week. (**i**) Axial CT scan: chronic subdural hematoma. (**j**) Axial CT scan: chronic subdural hematoma. (**k**) Axial CT scan: chronic subdural hematoma. (**l**) Axial CT scan: drainage with parietal burr holes. (**m**) Clinical image of recovered patient 18 months later







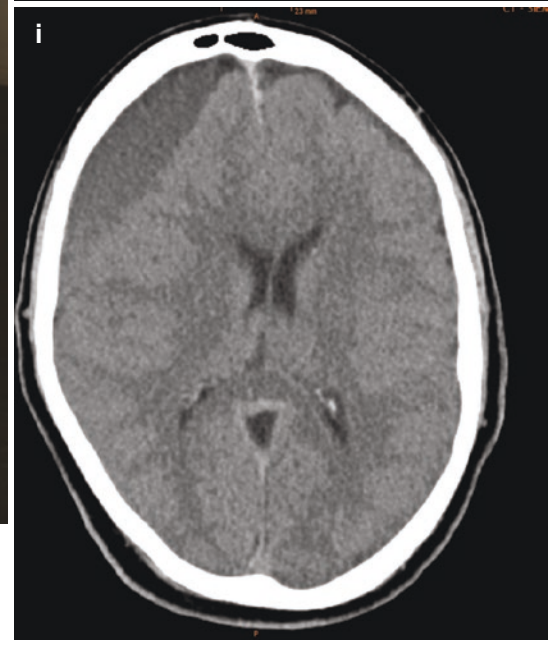


Fig. 9.19 (continued)

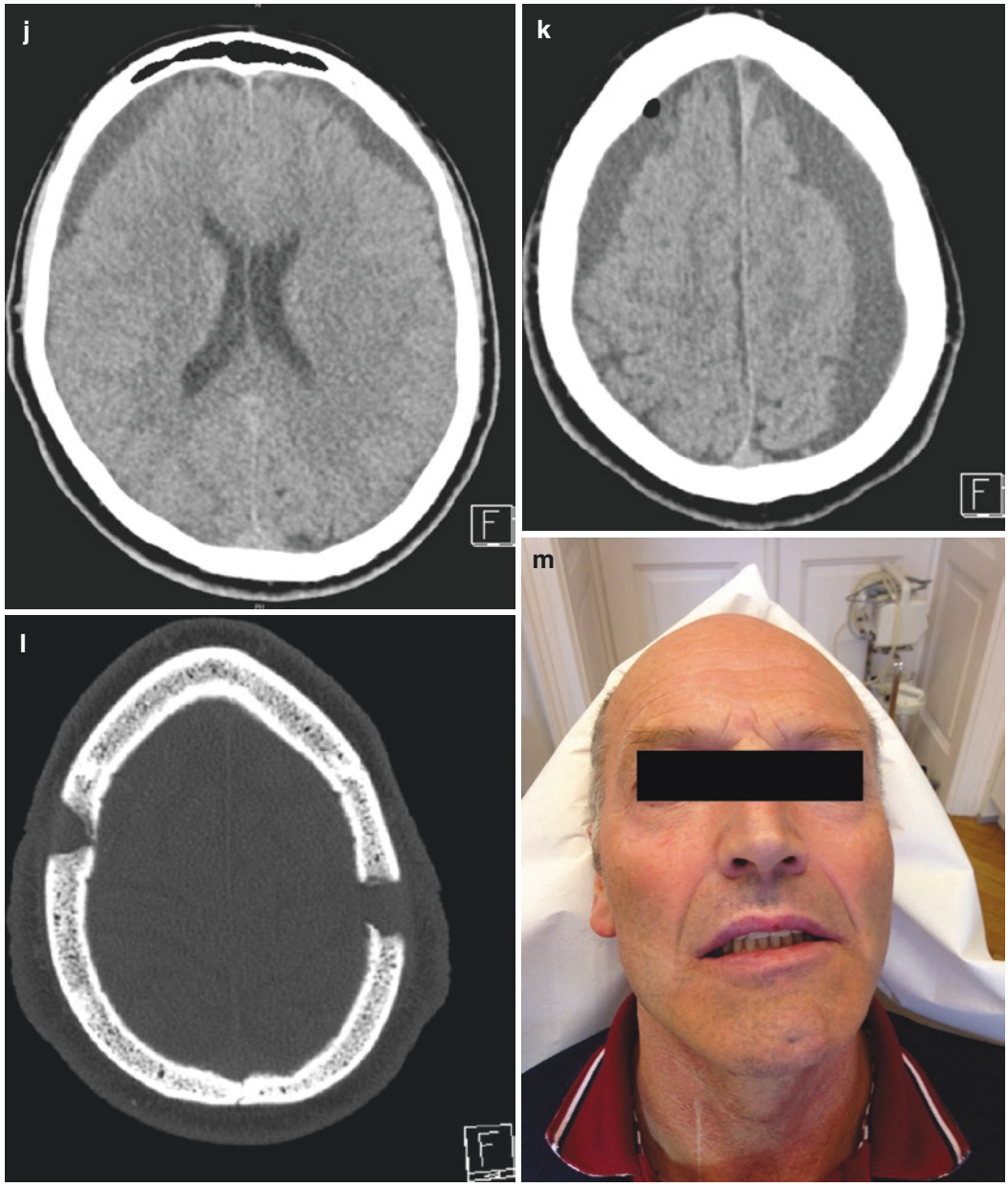


Fig. 9.19 (continued)

(Fig. 9.19g). To avoid blood clotting, the patient was given antithrombotic treatment despite suturing his facial wounds and repairing his dental trauma. He was discharged from the hospital 1 week later (Fig. 9.19h).

Due to persisting and even aggravating headache, he was reassessed 1 month later. As a complication, CT scans of the head showed severe chronic subdural hematoma (Fig. 9.19i–k) which had to be drained with burr holes on both sides of the parietal bones (Fig. 9.19l). One week later, he was released from the hospital.

Evaluation of the patient 18 months later revealed good recovery from his complex injuries (Fig. 9.19m).

Case 20

A 30-year-old male lumber worker was hit by a falling tree. His head was squeezed between trunks and resulted in skull base and cranial fault fractures including the right orbit (Fig. 9.20a–e). While the complication of amaurosis and loss of eye movement due to injuries to the cranial nerves III, IV and VI on the right side remain permanently, facial palsy on the left side of the face recovered 1 month later. Postoperative 3D CT evaluation shows zygoma and orbital bone positions (Fig. 9.20f).

Case 21

A 75-year-old male fell 6 m from a scaffold and sustained life-threating injuries including intracerebral bleeding with midline shift, skull base fracture, midface fractures, and cervical spine fractures (Fig. 9.21a–d). The mandible did not show any injury. Nevertheless, the patient recovered suffering from vision loss of his right eye as the remaining complication of his accident (Fig. 9.21e).

9.11 Complication: TMJ Ankylosis

First: what to do to avoid the complication:

Recognition of mandibular condylar neck and head trauma is a first step preventing TMJ ankylosis. In case of limited mouth opening after facial trauma, condylar neck and head fracture need to be excluded. When operative interventions are not possible, initial stabilization with intermaxillary fixation for 14 days in case of condylar head fractures and 28 days in case of condylar neck fractures is important following an even more important motion therapy to prevent ankylosis within the TMJ. When surgical reduction and stable fixation in anatomic correct position are possible, postoperative motion can be started immediately after surgery without loading forces.

Case 22

Undetected TMJ trauma is the major cause of TMJ ankylosis. Forces against the chin, e.g., falls or violence, are well-known risks for fractures or lesions of the TMJ which turn untreated in TMJ ankylosis. A 13-year-old child was severely handicapped in daily life as he was unable to move his mandible due to extensive TMJ ankylosis (Fig. 9.22a–c). Bony fusion of the TMJ had to be released to restore normal TMJ spaces (Fig. 9.22d–h). Postoperative mouth opening allowed a movement range of 5 cm interincisal distance (Fig. 9.22i). Protrusion and laterotrusion on both sides were also established (Fig. 9.22j–l).

9.12 Complication: Gun Shot

Case 23

A farmer 73 years of age was found in his wood due to a self-inflicted gunshot wound destroying mandible, maxilla, and nose (Fig. 9.23a). Following CT evaluation (Fig. 9.23b) and stabilization in the ER room, separation of the big communication between oral and nasal areas was most important (Fig. 9.23c). Miniplates and a reconstruction plate were inserted to stabilize the scattered mandible and maxilla where several bony parts were missing (Fig. 9.23d).

During the next weeks, it was necessary to change the fixation of the mandibular parts to another plate as depicted in Fig. 9.23e–i. The patient recovered and when he was offered to allow him to wear dentures he declined as he felt so well and assured us he chews everything with his gums (Fig. 9.23j, k).

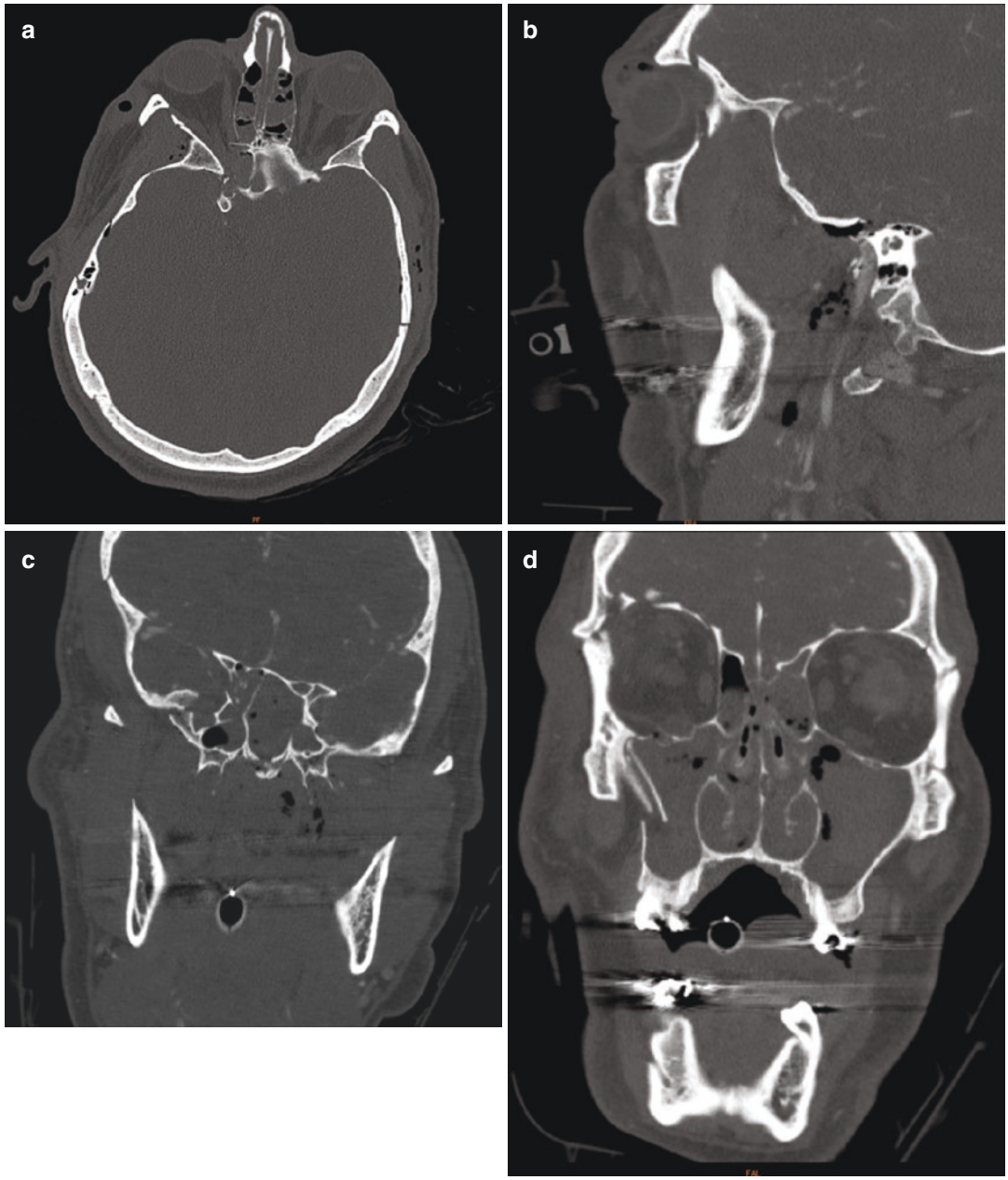


Fig. 9.20 (**a**) Axial CT scan: skull base and cranial fault fractures. (**b**) Sagittal CT scan: orbital and skull base fractures. (**c**) Coronal CT scan: skull base fractures. (**d**) Coronal CT scan: midface fractures. (**e**) Coronal CT scan: skull base fractures. (**f**) 3D CT scan: orbital and cranial vault fracture repair with plates and screws

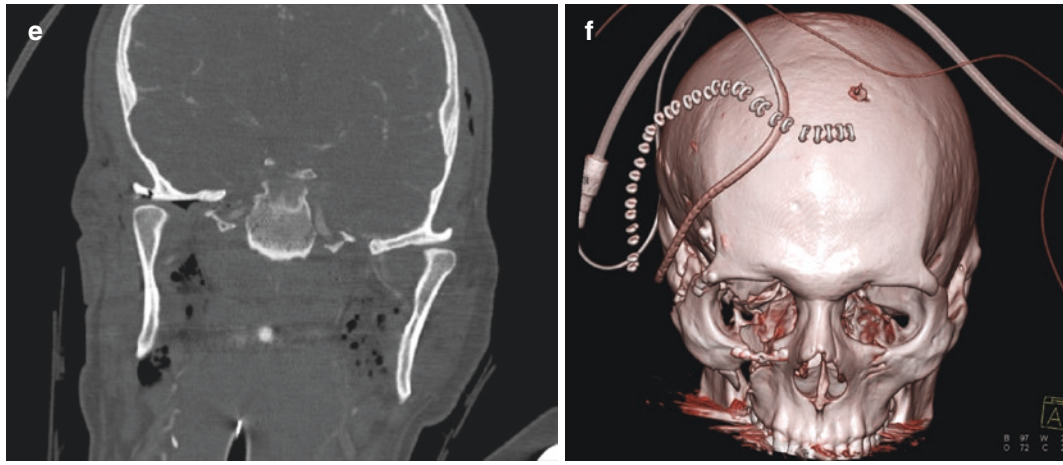


Fig. 9.20 (continued)

Case 24

A 21-year-old male was referred to our institution suffering from an untreated war injury with infected comminuted mandible fractures (Fig. 9.24a), with pus pouring from his right cheek (Fig. 9.24b).

Under intravenous antibiotic treatment intermaxillary fixation was established and the severely infected and scattered mandibular bone was removed. A stereolithography model showed the size of the defect (Fig. 9.24c) before inserting a reconstruction plate 1 month later (Fig. 9.24d). The mandible was reconstructed using an iliac crest free flap (Fig. 9.24e) and postoperative X-rays in two dimensions showed stable position of the neomandible (Fig. 9.24f, g).

9.13 Summary

Taken together, there are numerous possibilities for causes of complications due to CMFT [43–45]. Most common complications are temporary and in rare situations permanent involvements of cranial nerves in patients with fractures, especially when they are dislocated. Numbness of a facial region may recover completely or only in part with hypesthesia, dysesthesia, or even hyperesthesia.

The occlusion can be altered due to incomplete reduction of CMFT and in addition when sensory trigeminal nerve dysfunction mimicks a disturbance of the **occlusion** although no hindrance is present. CMFT in turn is sometimes the starting point of onset and awareness of TMJ disorders. Following surgical access for condyle fracture repair or mandibular body fracture reduction, mostly temporary loss of function of the **facial nerve** is visible. Injury to the third, fourth or sixth cranial nerve results in diplopia. **Infection as a complication** at the fracture site can be starting point of osteomyelitis and pseudarthrosis and can lead to screw loosening and hardware failure. Even with proper handling and correct use of titanium plates for open reduction and rigid fixation, the plate itself may break months and years after successful bone healing. In children and adolescents, the replacement of traumatic loss of teeth and alveolar bone has to be postponed until adulthood as inserted implants in restored bone areas stay in place while surrounding structures including teeth and bone keep on growing and changing in all three dimensions in children and adolescents.

Orbital floor fracture repair is a routine procedure at this institution. To avoid any danger to the optical nerve and its artery of the retina,

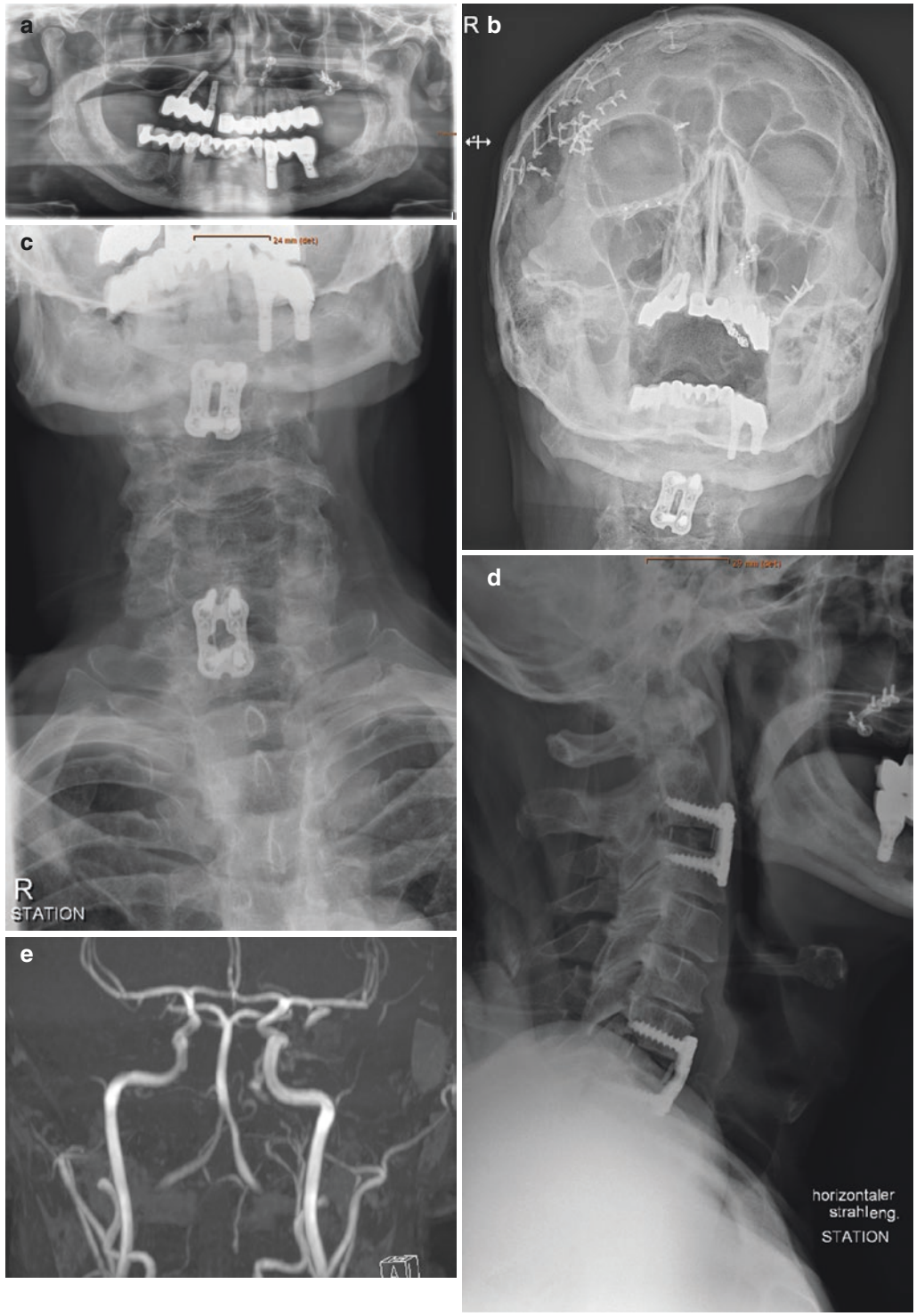


Fig. 9.21 (**a**) Postoperative panoramic radiograph after open reduction and rigid fixation of midface fractures with plates and screws. (**b**) Postoperative plain radiograph of head after open reduction and rigid fixation of cranial vault, cervical spine and midface fractures with plates and screws. (**c**) Anteroposterior radiograph of cervical spine after open reduction and rigid fixation of cervical spine fractures with plates and screws. (**d**) Lateral radiograph of cervical spine after open reduction and rigid fixation of cervical spine fractures with plates and screws. (**e**) Postoperative angiography

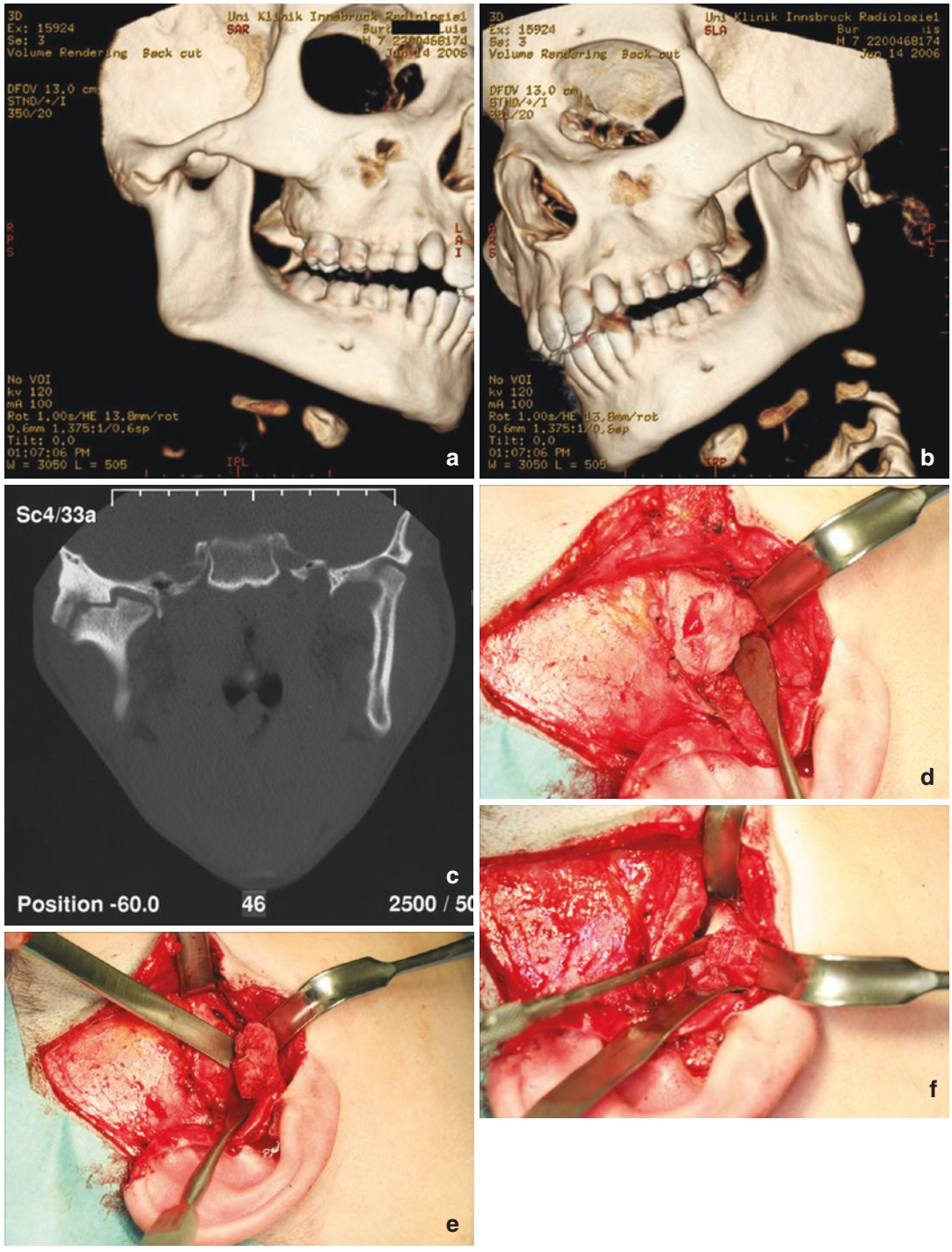


Fig. 9.22 (**a**) 3D CT scan: ankylosis of right TMJ. (**b**) 3D CT scan: ankylosis of left TMJ. (**c**) Coronal CT scan: ankylosis of both TMJs. (**d**) Intraoperative image: right side. (**e**) Intraoperative image: right side. (**f**) Intraoperative image: right side. (**g**) Intraoperative image: right side. (**h**) Intraoperative image: closure of right side. (**i**) Clinical image: postoperative mouth opening. (**j**) Clinical image: protrusion. (**k**) Clinical image: lateral shift to the right. (**l**) Clinical image: lateral shift to the left

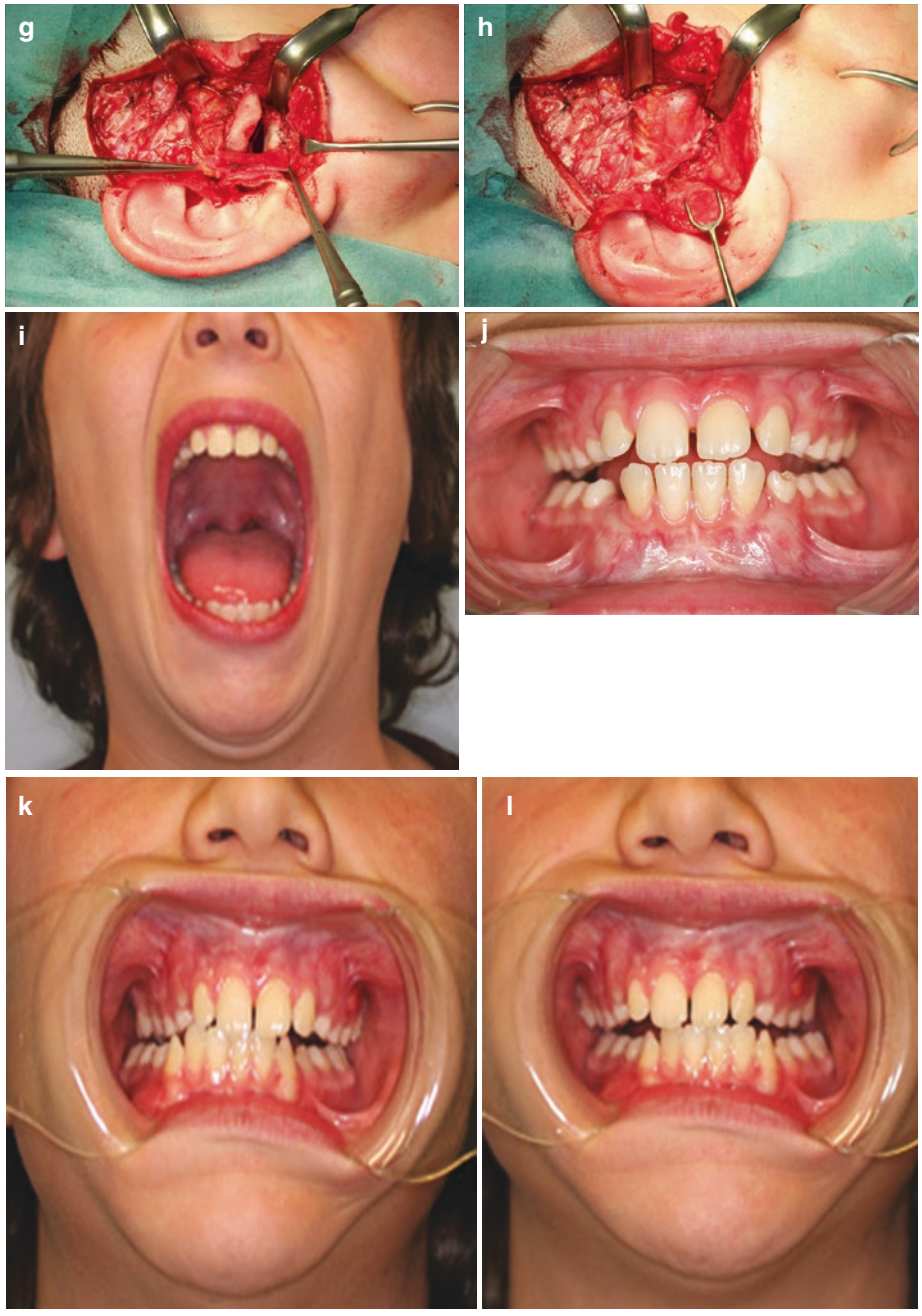


Fig. 9.22 (continued)

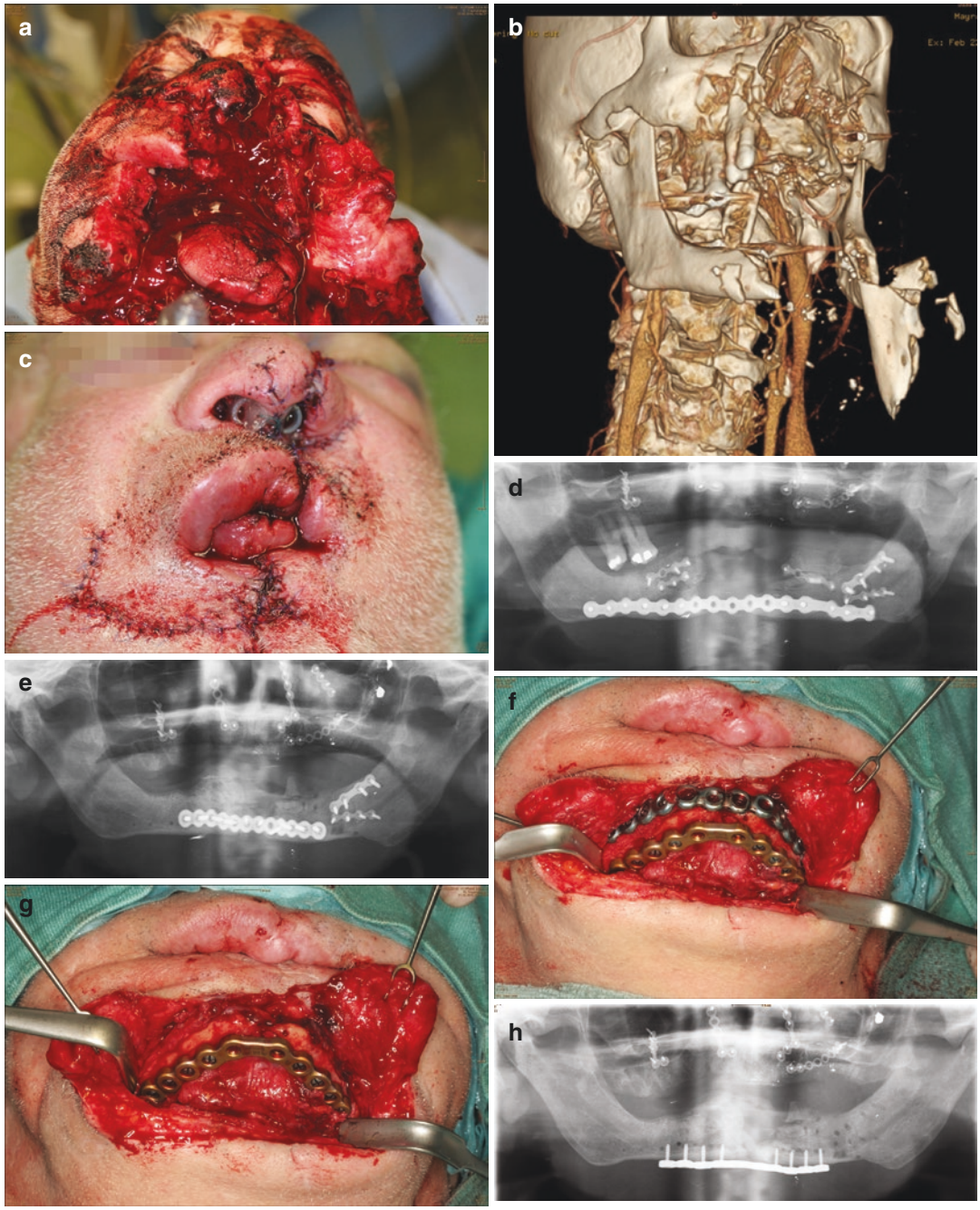


Fig. 9.23 (**a**) Clinical image: Gun shot wound destroyed mandible, maxilla and Nose. (**b**) 3D CT scan of clinical image. (**c**) Clinical image: separation oral and nasal cavity with nasal splints. (**d**) Panoramic radiograph with multiple plates and screws in midface and mandible. (**e**) Panoramic radiograph with reconstruction plate replacement after demarcation of nonvacularized bone. (**f**) Clinical image of plate replacement. (**g**) Clinical image of atypically placed reconstruction plate. (**h**) Panoramic radiograph with atypically placed reconstruction plate replacement. (**i**) Panoramic radiograph after plate removal/fibrous nonunion of mandibular midline. (**j**) Frontal view of happy patient 5 years later. (**k**) Side view of happy patient 5 years later

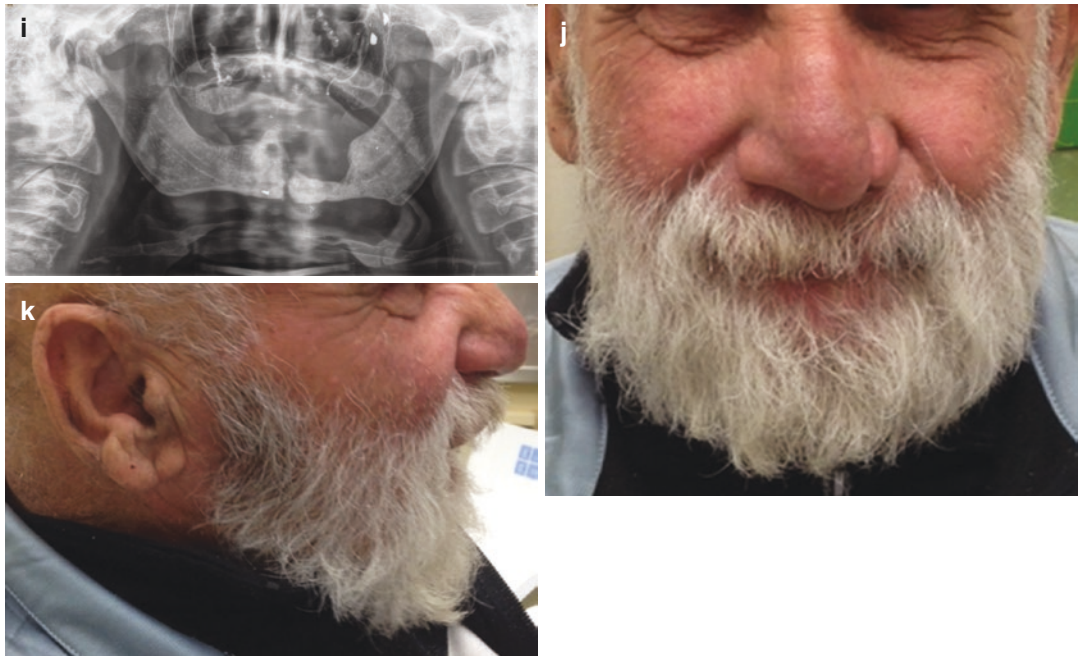


Fig. 9.23 (continued)

operative procedures necessitate careful planning and atraumatic preparation. Postoperative vision control initially every hour is important in case of retrobulbar hematoma formation. Immediate hematoma release definitely prevented vision loss in several cases of the last years when the sudden protrusion of the globe was observed. Due to 3D postoperative evaluation, physicians tend intraoperatively to perfection in orbital floor reconstruction for a great postoperative image putting a lot of stress on the swollen orbital structures. 3D Navigation is a great asset in the surgical armamentarium put insertion of a titanium-mesh may lead to overcorrection of the orbit.

Severe CMFT is frequently accompanied by concomitant intracranial, skull base, and cervical spine injuries complicating the situation. Tracheostomy or submental intubation is sometimes required to provide a stable breathing function [43].

In conclusion, this chapter on CMFT is only giving a small insight into the huge field of possible complications. Depending on one's perspective, the term consequences or sequelae may also be appropriate instead of the term complication. But usually situations or details in the course of CMFT complicate the injury and make it difficult to treat or heal. Sometimes secondary diseases or conditions develop in the course of CMFT which are not discussed in this chapter.

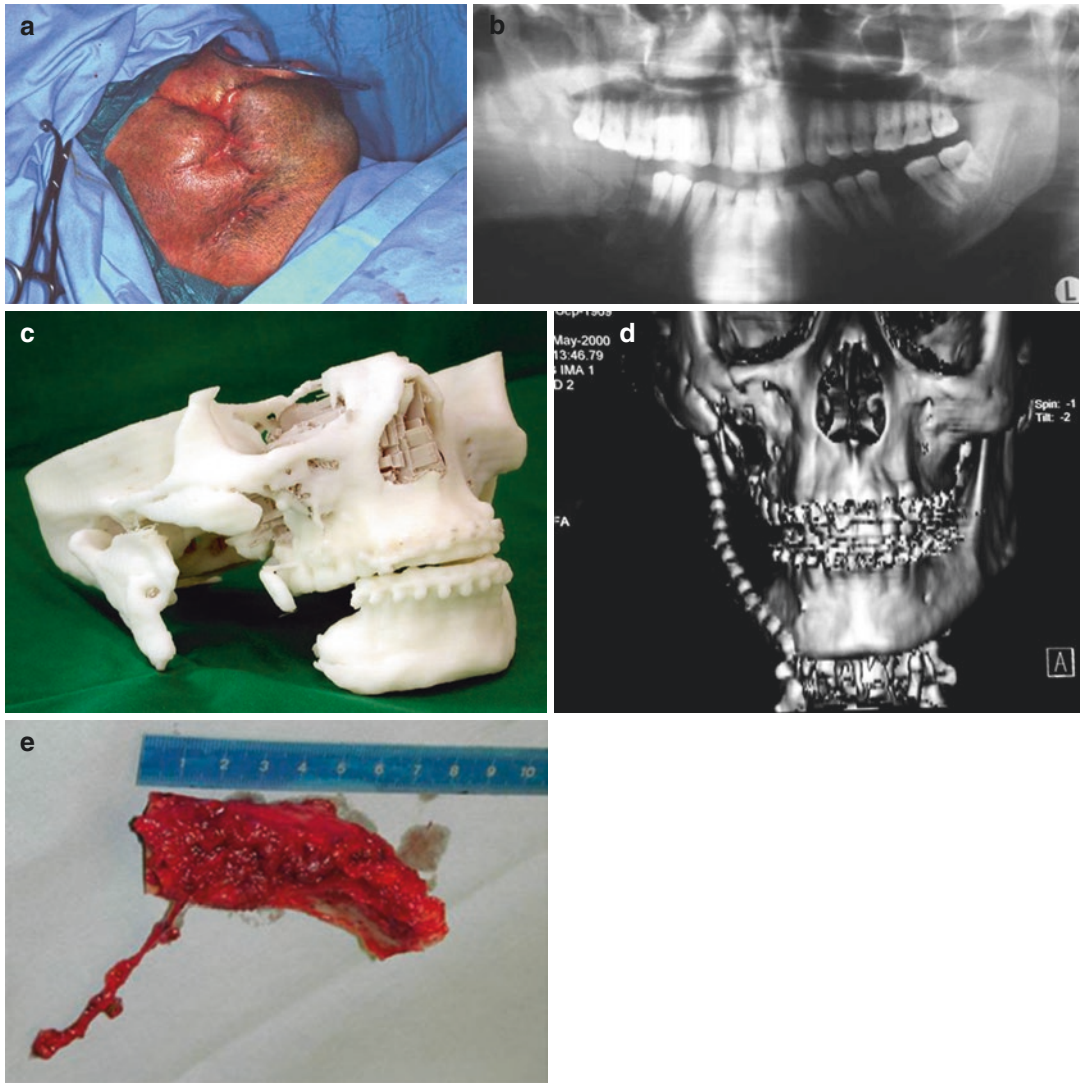


Fig. 9.24 (**a**) Clinical image of infected comminuted mandible fractures. (**b**) Preoperative panoramic radiograph depicting comminution of right mandible. (**c**) Stereolithographic model showing the defect size. (**d**) 3D image of reconstruction plate on the right side of the mandible. (**e**) Free iliac crest bone flap with circumflex profound vessels. (**f**) Plain radiograph of head with inserted flap and reconstruction plate. (**g**) Plain radiograph of mandible with inserted flap and reconstruction plate

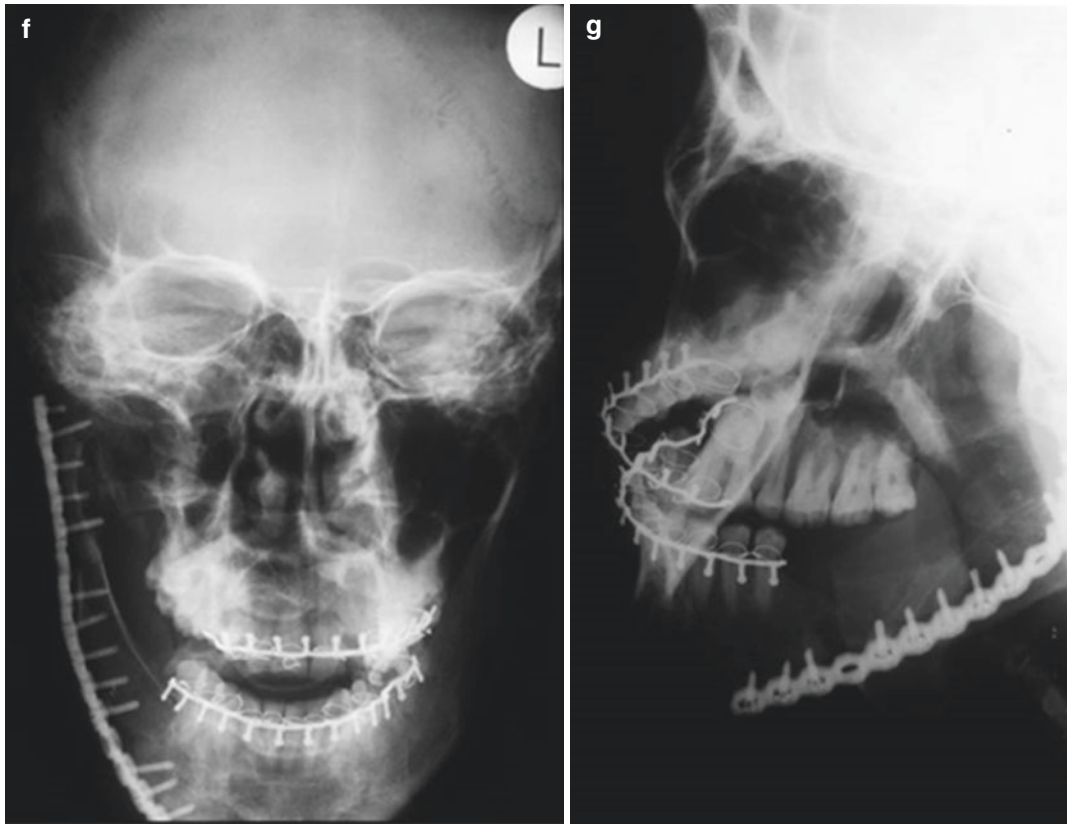


Fig. 9.24 (continued)

References

1. Abramowicz S, Allareddy V, Rampa S, et al. Facial fractures in patients with firearm injuries: profile and outcomes. J Oral Maxillofac Surg. 2017;75:2170–6.
2. Gassner R, Tuli T, Hächl O, et al. Cranio-maxillofacial trauma: a review of 9543 cases with 21, 067 injuries in 10 years. J Craniomaxillofac Surg. 2003;31:51–61.
3. Gassner R, Tuli T, Hächl O, et al. Cranio-maxillofacial trauma in children: a review of 3385 cases with 6060 injuries in 10 years. J Oral Maxillofac Surg. 2004;62:399–407.
4. Haechl O, Tuli T, Schwabegger A, et al. Maxillofacial trauma due to work-accidents. Int J Oral Maxillofac Surg. 2002;31:90–3.
5. Hohlrieder M, Hinterhölzl J, Ulmer H, et al. Traumatic intracranial hemorrhages in facial fracture patients: review of 2195 patients. Intensive Care Med. 2003;29:1095–100.
6. Kraft A, Abermann E, Stigler R, et al. Craniomaxillofacial trauma: synopsis of 14, 654 cases with 35, 129 injuries in 15 years. Craniomaxillofac Trauma Reconstr. 2012;5:41–50.
7. Missmann M, Tauscher T, Jank S, et al. Impaled head. Lancet. 2010;375:317.
8. Mundinger GS, Dorafshar AH, Gilson MM, et al. Blunt-mechanism facial patterns associated with internal carotid artery injuries: recommendations for additional screening criteria based on analysis of 4,398 patients. J Oral Maxillofac Surg. 2013;71:2092–100.
9. Obwegeser AA, Rieger M, Baubin MA, et al. Interdisziplinäre Versorgung und Outcome komplexer frontobasaler Frakturen mit Mittelgesichtsbeteiligung. J Neurol Neurochir Psychiatr. 2012;13:12–9.
10. Skjelbred P, Maron G, Gassner R. Facial injuries. In: Bahr R, McCrory P, LaPrade R, Meeuwisse W, Engebretsen L, editors. The Olympic Manual of Sports Injuries. IOC Medical Commission series entitled Encyclopedia of Sports Medicine and Handbooks of Sports Medicine and Science. Hoboken, NJ: Wiley-Blackwell; 2012. p. 78–94.
11. Bagheri SC, Meyer RA, Khan HA, Steed MB. Microsurgical repair of peripheral trigeminal nerve injuries from maxillofacial trauma. J Oral Maxillofac Surg. 2009;67:1791–9.
12. Yampolsky A, Ziccardi V, Chuang SK. Efficacy of acellular nerve allografts in trigeminal nerve reconstruction. J Oral Maxillofac Surg. 2017;75:2230–4.

13. Gassner R, Tuli T, Emshoff R, et al. Mountainbiking—a dangerous sport: comparison with bicycling on oral and maxillofacial trauma. Int J Oral Maxillofac Surg. 1999;28:188–91.
14. Gassner R, Hackl W, Tuli T, et al. Differential profile of facial injuries among mountainbikers as compared to bicyclists. J Trauma. 1999;47:50–4.
15. Kloss FR, Tuli T, Haechl O, et al. Trauma Injuries sustained by cyclists. Trauma. 2006;8:77–84.
16. Tay A, Lai J, Lye K, et al. Inferior alveolar nerve injury in trauma-induced mandible fractures. J Oral Maxillofac Surg. 2015;73:1328–40.
17. Gassner R, Traugott D, Röthler G, et al. Epidemiology of facial injuries sustained in Alpine Skiing. American Society for Testing and Materials (ASTM). In: Skiing trauma and safety. STP 1266, vol. 10; 1996. p. 76–81.
18. Gassner R, Hackl W, Tuli T, et al. Facial injuries in skiing: a retrospective study of 549 cases. Sports Med. 1999;27:127–34.
19. Gassner R, Ulmer H, Tuli T, et al. Incidence of oral and maxillofacial injuries due to different injury mechanisms in Alpine skiing. J Oral Maxillofac Surg. 1999;57:1068–73.
20. Gassner R. Oral and maxillofacial-associated winter sport injuries. Dent Clin N Am. 2000;44:157.
21. Gassner R, Garcia JV, Leja W, et al. Traumatic dental injuries in Alpine skiing. Endodont Dent Traumatol. 2000;16:122–7.
22. Tuli T, Hächl O, Berger N, et al. Facial trauma: how dangerous is skiing and snowboarding? J Oral Maxillofac Surg. 2010;68:293–9.
23. Kadakia S, Helman S, Saman M, Cooch N, Wood-Smith D. Concepts in neural coaptation: using the facial nerve as a paradigm in understanding principles surrounding nerve injury and repair. J Craniofac Surg. 2015;26:1304–9.
24. Gassner R, Bösch R, Tuli T, et al. Prevalence of dental trauma in 6000 patients with facial injuries: implications for prevention. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1999;87:27–33.
25. Perry M, Morris C. Advanced trauma life support (ATLS) and facial trauma: can one size fit all? Part 2: ATLS, maxillofacial injuries and airway management dilemmas. Int J Oral Maxillofac Surg. 2008;37:309–20.
26. Kloss F, Tuli T, Haechl O, et al. The impact of aging on cranio-maxillofacial trauma—a comparative investigation. Int J Oral Maxillofac Surg. 2007;36:1158–63.
27. Kloss F, Stigler R, Brandstaetter A, et al. Complications related to midfacial-fractures—operative versus conservative treatment. Int J Oral Maxillofac Surg. 2010;40:33–7.
28. Zosa BM, Elliott CW, Kurlander DE, Johnson F, Ho VP, Claridge JA. Facing the facts on prophylactic antibiotics for facial fractures: one day or less. J Trauma Acute Care Surg. 2018;85(3):444–50. https://doi.org/10.1097/TA.0000000000002009.
29. Hammond D, Parmar S, Whitty J, McPhillips M, Wain R. Prescription of antibiotics: does it alter the outcome for patients who have fractures of the angle of the mandible? Br J Oral Maxillofac Surg. 2017;55:958–61.
30. Jamal B, Pfahler S, Lane K, et al. Ophthalmic injuries in patients with zygomaticomaxillary complex fractures requiring surgical repair. J Oral Maxillofac Surg. 2009;67:986–9.
31. Brandt M, Haug R. Traumatic hyphema: a comprehensive review. J Oral Maxillofac Surg. 2001;59:1462–70.
32. He D, Blomquist P, Ellis E III. Association between ocular injuries and internal orbital fractures. J Oral Maxillofac Surg. 2007;65:713–20.
33. Ansari M. Blindness after facial fractures: a 19-year comprehensive study. J Oral Maxillofac Surg. 2005;63:229–37.
34. Kumar P, Sing V. Reconstructive dilemma after blindness. J Maxillofac Oral Surg. 2015;14:271–4.
35. Urolagin S, Kotrashetti S, Kale T, et al. Traumatic optic neuropathy after maxillofacial trauma: a review of 8 cases. J Oral Maxillofac Surg. 2012;70:1123–30.
36. Hohlrieder M, Hinterhoelzl J, Ulmer H, et al. Maxillofacial fractures masking traumatic intracranial hemorrhages. Int J Oral Maxillofac Surg. 2004;33:389–95.
37. Kloss F, Laimer K, Hohlrieder M, et al. Traumatic intracranial hemorrhage in conscious patients with facial fractures—a review of 1959 cases. J Craniomaxillofac Surg. 2008;36:372–7.
38. Tsang K, Whitfield P. Traumatic brain injury: review of current management strategies. Br J Oral Maxillofac Surg. 2012;50:298–308.
39. Chegini S, Gallighan N, Mcleod N, et al. Outcomes of treatment of fractures of the frontal sinus: review from a tertiary multispecialty craniofacial trauma service. Br J Oral Maxillofac Surg. 2016;54:801–5.
40. Hackl W, Ulmer H, Hausberger K, et al. Incidence of combined cervical spine injuries and facial trauma. J Trauma. 2001;50:41–5.
41. Reich W, Sorov A, Eckert A. Maxillofacial trauma—underestimation of cervical spine injury. J Craniomaxillofac Surg. 2016;44:1469–78.
42. Hackl W, Hausberger K, Sailer R, et al. Prevalence of cervical spine injuries in patients with facial trauma. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;92:370–6.
43. Kaiser A, Semanoff A, Christensen L, et al. Submental intubation: an underutilized technique for airway management in patients with panfacial trauma. J Craniofac Surg. 2018;29(5):1349–51. https://doi.org/10.1097/SCS.0000000000004496.
44. Rothweiler R, Bayer J, Zwingmann J, et al. Outcome and complications after treatment of facial fractures at different times in polytrauma patients. J Craniomaxillofac Surg. 2018;46:283–7.
45. Vatsala R, Elavenil P, Saravanan C, et al. Evaluation of depression associated with post-traumatic stress disorder after maxillofacial injuries—a prospective study. J Oral Maxillofac Surg. 2018;76(6):1282.e1–9. https://doi.org/10.1016/j.joms.2018.02.011.

10 Should Osteosynthesis Material in Cranio-Maxillofacial Trauma be Removed or Left In Situ? A Complication-associated Consideration

Andreas Kolk

Contents

10.1 Introduction

For 60 years, open reduction and internal fixation (ORIF) with various metallic osteosynthetic materials (OM) has been the standard treatment for maxillofacial fractures [1]. Since some metals and alloys such as stainless steel or Vitallium [2, 3] are thick and have shown adverse effects such as corrosion and local tissue reactions, they have to be removed as a mandatory part of complete fracture treatment [4]. Nowadays, titanium (Ti) is generally accepted as being a relatively pure material for use as an OM; it has proven its biocompatibility and corrosion resistance and has exhibited only minimal or even no tissue reactions over the long term [5, 6]. Ti is usually commercially distributed as Ti-6Al-4V or Ti-6Al-7Nb, i.e. as an alloy composed of 6% aluminium and 4% vanadium or alternatively 7% niobium. In addition, compared with other implanted metallic materials, the use of Ti has led to OM having an elastic modulus relatively similar to that of bone [7, 8]. Multiple analyses have demonstrated that even local macrophage responses to Ti material are so low long term that, from a biocompatibility point of view, Ti could remain in situ indefinitely. On the other hand, even asymptomatic Ti-6Al-4V alloys in situ long term can result in the systemic dissemination of Ti and vanadium into hair and nails [9]. A further indication of the biocompatibility of Ti is provided by the widespread use of dental implants, which are regularly planned to

A. Kolk (✉)
University Clinic of Oral and Maxillofacial Surgery, Medical University of Innsbruck, Innsbruck, Austria
e-mail: andreas.kolk@i-med.ac.at, andreas.kolk@tirol-kliniken.at

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_10

be left in the human body for the remaining lifetime of the patient or for as long as possible.

In addition to these basic material-associated considerations, the question of leaving or removing Ti mini-, midi- or microplates or various types of screws in maxillofacial trauma patients after ORIF has been the subject of debate for many years [4, 7, 10, 11].

Several aspects influence such considerations. Prior to a detailed analysis of the pros and cons concerning the removal versus the permanent presence of OM, mention should be made that this discussion focuses only on asymptomatic material. In case of loosening or even infection with delayed healing of the underlying bone, OM should be removed as soon as possible (Fig. 10.1a, b). In contrast to the "permanent" subgroup, which comprises most of the patients after ORIF, one relative and a few strong indications should be noted regarding the elective removal of OM.

The goals of this chapter are (1) to elucidate the long-term discussions concerning the elective removal of OM, (2) to demonstrate current opinion concerning the frequency of the leaving of asymptomatic material in situ and the handling of symptomatic plates, and (3) to explain the few strong indications that require early hardware removal.

OM is designated asymptomatic if it does not show any radiological and clinical signs of loosening, the previous fracture line adjacent to the material has healed completely (Fig. 10.2), and the patient does not complain about any form of discomfort such as thermal or local skin sensitivity or palpability through the overlying skin (Fig. 10.3a, b). "Asymptomatic" OM affects 80–90% of all patients [12, 13]. In the latter group, most of the surgeons worldwide would leave the material for the duration of the patient's life to prevent any undue risk to the patient.

10.2 What are the Arguments Against the Elective Removal of OM?

1. Titanium has proven its biocompatibility and corrosion resistance and so can permanently stay in contact with local tissue.

   Localized or general corrosion is one of the main problems and, therefore, one of the most important parameters in terms of the biocompatibility of any metallic implant. Released ions lead to a reaction between body fluids and the metallic OM and are responsible for various problems such as inflammation or foreign body reaction through giant cells [14] and finally are liable for the loosening of the implanted material from the bone. Because of the strong passive oxide film formation on a Ti surface, largely consisting of TiO_2, Ti-based OM are highly corrosion resistant via a local protective shield of the OM and are chemical inert. Apart from these convincing properties of Ti, one has to keep in mind that metal release can also be induced via plate bending or the screw driving process during the fixation of the plate [15].

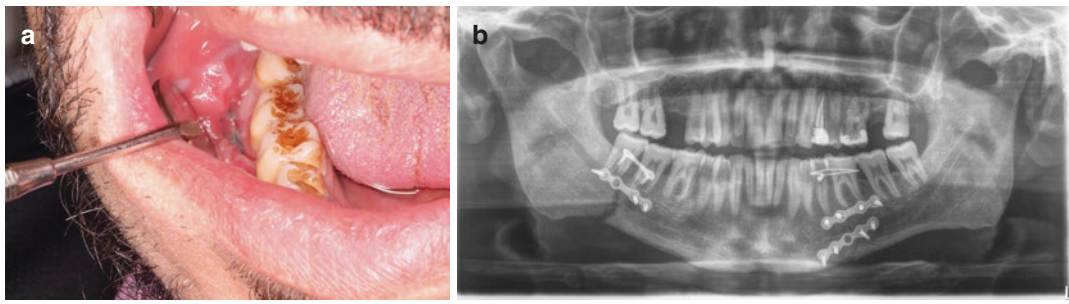


Fig. 10.1 (**a**, **b**) Indication for non-elective immediate removal of OM and surgical revision. Intraorally exposed miniplate (**a**) due to their radiologically proven insufficient stability and position with consecutive screw loosening and inadequate fracture healing (**b**)

Most OM infections occur within the first year. If, for example, a Ti plate is clinically and radiologically asymptomatic for 12 months or longer, secondary loosening attributable to Ti-ion-induced bone-resorbing cytokines, such as IL-6, -17 and TGF-β [16, 17] at a later time point, is unlikely. The risk of late bone resorption around a loosened and subsequently infected plate is minimal.

2. In some areas of the facial skeleton, such as the condylar neck region, a second procedure is sometimes associated with an even higher approach-related risk of side effects, if the initial ORIF has been performed via an extraoral (e.g. retromandibular) approach. An elective second surgery for hardware removal through the pre-existing retromandibular scars can occasionally significantly increase the risk of facial nerve palsy because the identification of certain facial nerve branches is much more difficult in the pre-operated scar tissue. As an alternative, hardware removal can principally be performed via an intraoral approach with a 90°-angled screw driver, but this is sometimes difficult if one of the plates has been fixed at the posterior border of the ascending ramus (Fig. 10.2a, b). In addition, a second extraoral approach would probably increase the risk of salivary fistulas by cutting the parotid gland and would reduce the aesthetic appearance of the facial scar of the former approach.
3. Predominantly in younger patients, osseointegration of the material, e.g. not only positional or lag screws, but also midi- and microplates, can progress so quickly that, even 6 months after ORIF, complete hardware removal cannot be guaranteed if the situation has not been evaluated intraoperatively. The initiation of surgery under those unfavourable starting conditions, with the risk of only partial

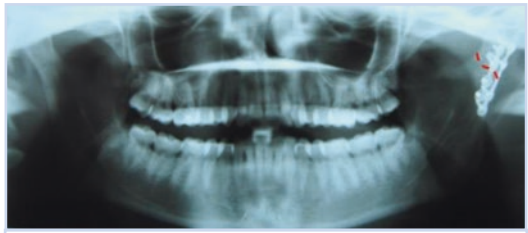


Fig. 10.2 ORIF of a middle to high condylar neck fracture on the left side via a retromandibular approach. Note the height of the cranial screws of the two miniplates above the red-marked fracture line close to the high condylar neck. The posterior plate is located on the dorsal part of the ascending ramus. Removal of the OM will be difficult either via an intraoral approach with an angled screw driver or via the former retromandibular approach through scar tissue

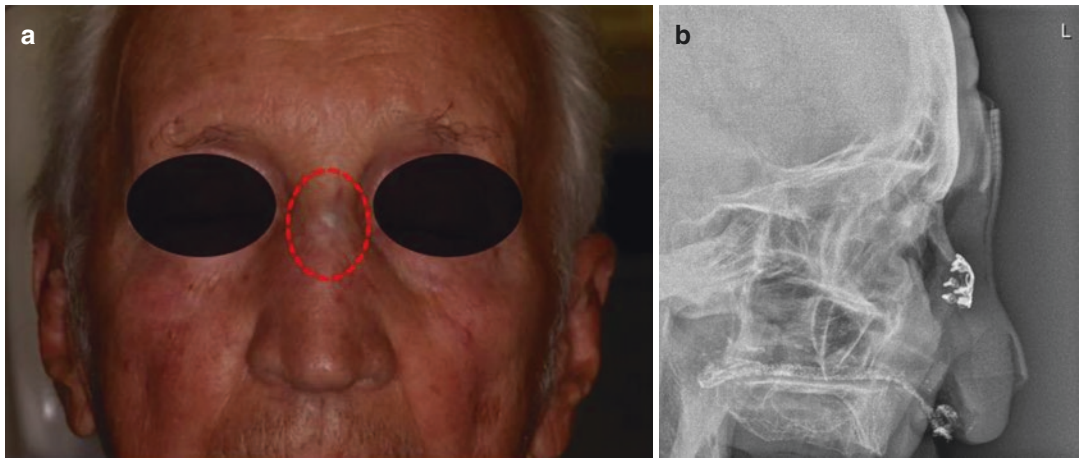


Fig. 10.3 (**a**, **b**) Regularly healed ORIF of a multifragmented nose bone fracture 4 years ago as seen in the lateral head image (**a**). Due to the exposed underlying position of the OM and the thin skin at the nasal dorsum, the patient complained problems due to permanent compression by regularly wearing his glasses. So he insisted of elective removal of the OM

removal of the OM, is not acceptable. In these cases, leaving the OM is definitively the better recommendation for the patient.

10.3 What do the Protagonists of Elective Hardware Removal Argue?

1. OM removal is an easy and low-risk procedure that normally takes less time compared with the initial surgery for ORIF. In addition, elective surgical cases are ideal training procedures for young resident surgeons.
2. If elective removal of the OM is performed after 6–12 months, it is a safe procedure and prevents any potential long-term complications such as late sensitivity feelings, palpability through the skin, or inflammatory responses. Life expectancy permanently increases. Trauma patients undergoing surgery for ORIF are mainly young. However, no data are available indicating the way in which Ti behaves after 30 or 40 years.

   When compared with other metals, such as nickel and chromium, sensitivity to Ti is relatively rare. Mostly, this sensitivity is related to complications such as the skin perforation of the OM, the loosening of total joint replacements, or the corrosion of the OM. In the last-mentioned situation, Ti stimulates immunological responses. Activated CD4+ T-lymphocytes have been found close to the OM surface in the head and face region as a sign of a specific immunological response [18, 19]. This slow development takes a minimum 6–12 months unless such a type IV reaction to Ti occurs. When the elective removal of OM is performed within 6–12 months, the risk of developing material sensitivity can thus be excluded.

   Ti alloys such as Ti-6Al-4V are significantly less toxic than Co-Cr particles, which lead to an early activation of phagocytosis with cell death. Ti particles pass slowly into the surrounding tissue over time [19]. In contrast to an acute inflammatory reaction occurring together with mast cell degradation and the activation of polymorphonuclear leukocytes, a Ti-particle-induced inflammatory long-term process activates phagocytosing macrophages via blood-borne monocytes that differentiate to lymphocytes. Under the influence of IL-4 and IL-13, these cells convert to fused macrophages that, in turn, become foreign body giant cells [14]. These subsequently express pro-inflammatory cytokines, such as IL-6, TNF-α and IL1-β, which are related to a chronic inflammatory response resulting in an osteolytic process [20]. The normal balance between bone-depleting and bone-protecting cytokines is disturbed in favour of a chronic bone resorption process. As a standard tissue reaction adjacent to the OM, these monocytes lead to granulation tissue and, finally, to fibrous capsule formation around the implanted foreign material. As a consequence, this mild inflammatory response related to Ti-particle dissemination results in the accumulation of granulation tissue and the loosening of OM attributable to bone resorption. Such an inflammatory sequence has been found in the surrounding tissue of removed craniomaxillofacial OM [18]. Even though only case reports and no epidemiological studies have been published about the association of Ti implants and mutagenicity [21], a comparable theoretical context cannot be excluded for 100% in the case of OM in the craniomaxillofacial region. Compared with the number of applied Ti–OM, cases of directly related soft tissue malignancies are extremely rare, so that the latter cannot count as an argument for an elective removal of OM [7]. The majority of experimental cases have shown corrosion [22] or wear debris [21] as being the most probable underlying cause of malignant development in orthopaedic alloy implants. In current publications, elevated blood concentrations of metal ions induced by abnormal wear and corrosion of the metal-on-metal implants have been proposed as being responsible for this improbable association of orthopaedic implants and carcinogenesis [23–25]. Because of wear debris after metal-on-metal hip replacement, elevated levels of metal ions

in the skin, together with ultraviolet radiation, could possibly cause DNA damage [26]. These potential associations are not applicable for Ti-based OM in the head and neck region and so the discussion concerning cancerogenity of permanent OM remains theoretical.

3. The quality of computer tomography or magnetic resonance imaging is increasing permanently and new artefact-suppressing sequences have been developed [27–29]. Moreover, Ti–OM does not induce the same amount of imaging artefacts and the relatively insignificant scattering that other metals do [30]. Nevertheless, especially in cases with multiple plates in the frontal bone or midface region, permanent metallic artefacts can sometimes make the imaging interpretation of certain findings impossible [31]. In early cases involving therapeutic radiation, concerns about back scattering were worrying. Nowadays, as intensity-modulated radiation therapy is the standard for the head and neck region, this is no longer relevant [32]. If the removal of the OM is desired for a better evaluation of repeated imaging, it is almost impossible or causes significantly more collateral damage, sometimes after many years.
4. Many different health insurance systems exist around the world. Whereas in countries such as the US, the elective removal of OM is not covered by the standard insurance system, European countries such as Germany, Austria, Belgium or Sweden, which have a government-controlled health system, profit issues might play a role in decision-making for elective hardware removal.
5. A further argument exists for elective OM removal: the patient's choice. Some patients insist on the elective removal of asymptomatic OM, because they feel uncomfortable with the remaining OM in situ in some situations (Fig. 10.3a, b), they are afraid of developing late complications such as certain forms of discomfort or chronic infection or they just do not want to have any artificial material inside their body unless it is absolutely necessary.

10.4 Indication for Elective Removal of Asymptomatic OM

One relative and a few strong indications exist for the elective removal of OM.

10.4.1 Strong Indications

1. In the growing skeleton, bone healing is usually excessive and subsequently leads to growth restriction [33] because of fixed OM. Removal should, therefore, occur 3 months after ORIF. Biodegradable materials might be an alternative [34].
2. Highly atrophic fractures of the lower jaw usually require bone plating for fixation. Because of the missing height of the alveolar process and any ongoing atrophic degeneration of the bone, these plates may become exposed over time and prohibit the ability to wear a denture. These plates have to be removed immediately after fracture consolidation.
3. ORIF of condylar head fractures is increasingly coming into the realm of experienced surgeons. To gain an optimal functional and anatomic long-term outcome and to prevent scaring within the joint, the OM should be fixed from outside the joint capsule. This is mostly carried out with Ti positional screws [35], which should be removed after 3 months. Subsequent to that time, the bone directly adjacent to the screw head is resorbed and the bolt head thus freed from the underlying bone. This can lead to the functional impairment of the temporomandibular joint (TMJ) (Fig. 10.4a, b). As a positive side effect of this second look procedure, potential fibrous adhesions in the lower joint space can easily be removed.

10.4.2 Relative Indication

Apart from wound dehiscence, loose or broken OM hardware can become symptomatic because of microbial biofilm-related infections [36] as the

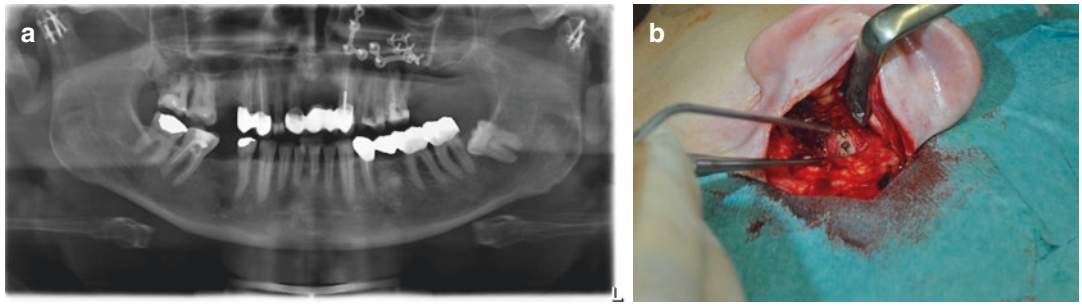


Fig. 10.4 (**a**, **b**) ORIF of a multi-fragmented bilateral condylar head fracture using a combination of microplates and positional screws 2 months previously. The condylar head fractures have healed (**a**: OPG). The OM is now non-functional and the screw heads (**b**: situation in vivo) are exposed to the lateral joint capsule resulting in a reduced TMJ function

most common cause [37]. Compared with other maxillofacial areas, the mandibular body is one of the most often involved locations affiliated with infection problems after ORIF [38] (Fig. 10.1a, b). Within this subgroup, a plate located in the area of mandibular angle, especially in the presence of an unerupted or partially erupted wisdom tooth, should be removed routinely to prevent secondary infection attributable to the mobile mucosa covering the plate but without stable attachment to the wisdom tooth. Generally, wisdom teeth can develop pericoronitis that would interfere with the plate. Therefore, the easy and quick removal of both tooth and plate under local anaesthesia can be recommended [39].

10.5 Conclusion

No international consensus has been reached in the discussion of the elective removal of asymptomatic Ti plates and screws in the craniomaxillofacial area versus the permanent placement of OM, because of the lack of scientifically established data supporting either decision. Elective removal of asymptomatic OM implies the prevention of any undue risk to the patient, but this statement has not yet been supported in the sense of a guideline. Nevertheless, the reopening of a previously used retromandibular approach to regain access to the condylar neck could itself be an undue risk if any new damage of the facial nerve branches or the parotid gland with salivary fistulas occurs or if the cosmetic result of the extraoral scar deteriorates. Therefore, other than in Western European countries, asymptomatic plates and screws are not removed in the majority of areas around the world. Apart from the limited availability of general anaesthesia in these medical systems, cost reasons are of huge influence on the decision to leave the hardware in situ.

Independently of these arguments, some indications exist for the elective removal of asymptomatic OM, such as the growing skeleton in paediatric traumatology, hardware lying close to the alveolar process of the atrophic edentulous mandible to regain prosthetic ability, the fixation of condylar head region fractures after ORIF with positional screws or just the patient's wish e.g. because of fear of any foreign material left in their body for the whole life time.

References

1. Thoma KH, Holland DJ Jr, et al. Fracture cases treated by means of internal fixation. Oral Surg Oral Med Oral Pathol. 1948;1:90–7.
2. Luhr HG. On the stable osteosynthesis in mandibular fractures. Dtsch Zahnarztl Z. 1968;23:754.
3. Becker R, Machtens E, Lenz J. Possibilities and limitations of compression osteosynthesis. Fortschr Kiefer Gesichtschir. 1975;19:87–91.
4. Alpert B, Seligson D. Removal of asymptomatic bone plates used for orthognathic surgery and facial fractures. J Oral Maxillofac Surg. 1996;54:618–21.
5. Trevisan F, Calignano F, Aversa A, et al. Additive manufacturing of titanium alloys in the biomedical

field: processes, properties and applications. J Appl Biomater Funct Mater. 2018;16:57–67.
6. Ottria L, Lauritano D, Andreasi Bassi M, et al. Mechanical, chemical and biological aspects of titanium and titanium alloys in implant dentistry. J Biol Regul Homeost Agents. 2018;32:81–90.
7. Haug RH. Retention of asymptomatic bone plates used for orthognathic surgery and facial fractures. J Oral Maxillofac Surg. 1996;54:611–7.
8. Munuera C, Matzelle TR, Kruse N, et al. Surface elastic properties of Ti alloys modified for medical implants: a force spectroscopy study. Acta Biomater. 2007;3:113–9.
9. Bozkus I, Germec-Cakan D, Arun T. Evaluation of metal concentrations in hair and nail after orthognathic surgery. J Craniofac Surg. 2011;22:68–72.
10. Cahill TJ III, Gandhi R, Allori AC, et al. Hardware removal in craniomaxillofacial trauma: a systematic review of the literature and management algorithm. Ann Plast Surg. 2015;75:572–8.
11. Kim YK, Yeo HH, Lim SC. Tissue response to titanium plates: a transmitted electron microscopic study. J Oral Maxillofac Surg. 1997;55:322–6.
12. Thoren H, Snall J, Kormi E, Lindqvist C, Suominen-Taipale L, Tornwall J. Symptomatic plate removal after treatment of facial fractures. J Craniomaxillofac Surg. 2010;38:505–10.
13. Nagase DY, Courtemanche DJ, Peters DA. Plate removal in traumatic facial fractures: 13-year practice review. Ann Plast Surg. 2005;55:608–11.
14. Anderson JM, Rodriguez A, Chang DT. Foreign body reaction to biomaterials. Semin Immunol. 2008;20:86–100.
15. Galante JO, Lemons J, Spector M, Wilson PD Jr, Wright TM. The biologic effects of implant materials. J Orthop Res. 1991;9:760–75.
16. Kitaura H, Kimura K, Ishida M, Kohara H, Yoshimatsu M, Takano-Yamamoto T. Immunological reaction in TNF-alpha-mediated osteoclast formation and bone resorption in vitro and in vivo. Clin Dev Immunol. 2013;2013:181849.
17. Fox SW, Fuller K, Bayley KE, Lean JM, Chambers TJ. TGF-beta 1 and IFN-gamma direct macrophage activation by TNF-alpha to osteoclastic or cytocidal phenotype. J Immunol. 2000;165:4957–63.
18. Katou F, Andoh N, Motegi K, Nagura H. Immunoinflammatory responses in the tissue adjacent to titanium miniplates used in the treatment of mandibular fractures. J Craniomaxillofac Surg. 1996;24:155–62.
19. Nautiyal VP, Mittal A, Agarwal A, Pandey A. Tissue response to titanium implant using scanning electron microscope. Natl J Maxillofac Surg. 2013;4:7–12.
20. Souza PP, Lerner UH. The role of cytokines in inflammatory bone loss. Immunol Invest. 2013;42:555–622.
21. Sunderman FW Jr. Carcinogenicity of metal alloys in orthopedic prostheses: clinical and experimental studies. Fundam Appl Toxicol. 1989;13:205–16.
22. Vahey JW, Simonian PT, Conrad EU 3rd. Carcinogenicity and metallic implants. Am J Orthop (Belle Mead NJ). 1995;24:319–24.
23. Brewster DH, Stockton DL, Reekie A, et al. Risk of cancer following primary total hip replacement or primary resurfacing arthroplasty of the hip: a retrospective cohort study in Scotland. Br J Cancer. 2013;108:1883–90.
24. Makela KT, Visuri T, Pulkkinen P, et al. Risk of cancer with metal-on-metal hip replacements: population based study. BMJ. 2012;e4646:345.
25. Smith AJ, Dieppe P, Porter M, Blom AW, National Joint Registry of England and Wales. Risk of cancer in first seven years after metal-on-metal hip replacement compared with other bearings and general population: linkage study between the National Joint Registry of England and Wales and hospital episode statistics. BMJ. 2012;344:e2383.
26. Makela KT, Visuri T, Pulkkinen P, et al. Cancer incidence and cause-specific mortality in patients with metal-on-metal hip replacements in Finland. Acta Orthop. 2014;85:32–8.
27. Mathew CA, Maller S, Maheshwaran. Interactions between magnetic resonance imaging and dental material. J Pharm Bioallied Sci. 2013;5:S113–6.
28. Hung SC, Wu CC, Lin CJ, et al. Artifact reduction of different metallic implants in flat detector C-arm CT. Am J Neuroradiol. 2014;35:1288–92.
29. Hargreaves BA, Worters PW, Pauly KB, Pauly JM, Koch KM, Gold GE. Metal-induced artifacts in MRI. Am J Roentgenol. 2011;197:547–55.
30. Eppley BL, Sparks C, Herman E, Edwards M, McCarty M, Sadove AM. Effects of skeletal fixation on craniofacial imaging. J Craniofac Surg. 1993;4:67–73.
31. De Crop A, Casselman J, Van Hoof T, et al. Analysis of metal artifact reduction tools for dental hardware in CT scans of the oral cavity: kVp, iterative reconstruction, dual-energy CT, metal artifact reduction software: does it make a difference? Neuroradiology. 2015;57:841–9.
32. Shimamoto H, Sumida I, Kakimoto N, et al. Evaluation of the scatter doses in the direction of the buccal mucosa from dental metals. J Appl Clin Med Phys. 2015;16:5374.
33. Eppley BL, Platis JM, Sadove AM. Experimental effects of bone plating in infancy on craniomaxillofacial skeletal growth. Cleft Palate Craniofac J. 1993;30:164–9.
34. Kolk A, Kohnke R, Saely CH, Ploder O. Are biodegradable osteosyntheses still an option for midface trauma? Longitudinal evaluation of three different PLA-based materials. Biomed Res Int. 2015;2015:621481.
35. Kolk A, Neff A. Long-term results of ORIF of condylar head fractures of the mandible: a prospective 5-year follow-up study of small-fragment positional-screw osteosynthesis (SFPSO). J Craniomaxillofac Surg. 2015;43:452–61.
36. Jhass AK, Johnston DA, Gulati A, Anand R, Stoodley P, Sharma S. A scanning electron microscope characterisation of biofilm on failed craniofacial osteosynthesis miniplates. J Craniomaxillofac Surg. 2014;42:e372–8.

37. Bhatt V, Chhabra P, Dover MS. Removal of miniplates in maxillofacial surgery: a follow-up study. J Oral Maxillofac Surg. 2005;63:756–60.
38. Rallis G, Mourouzis C, Papakosta V, Papanastasiou G, Zachariades N. Reasons for miniplate removal following maxillofacial trauma: a 4-year study. J Craniomaxillofac Surg. 2006;34:435–9.
39. Yamamoto K, Matsusue Y, Horita S, Murakami K, Sugiura T, Kirita T. Routine removal of the plate after surgical treatment for mandibular angle fracture with a third molar in relation to the fracture line. Ann Maxillofac Surg. 2015;5:77–81.

11 Complications and Facial Pain in Cranio-Maxillofacial and Oral Surgery

Wilhelm Eisner and Sebastian Quirbach

Contents

11.1 Introduction

Pain is an integral part of endogenous protective reaction to external or internal effects. The goal is to maintain the integrity of the tissue by early detection of harmful influences. In medicine, pain plays a key role; pain can be seen as a specific symptom, e.g., in acute appendicitis. Furthermore, pain can be identified as a chronic disease. In both cases, the need for specific, formal identification,

W. Eisner (✉) • S. Quirbach
Department of Neurosurgery, Medical University Innsbruck, Innsbruck, Austria
e-mail: wilhelm.eisner@i-med.ac.at

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_11

as well as specialized therapy exists. According to the WHO, pain therapy should treat all forms of pain effectively. It is a well-known fact that this ultimate goal is difficult to reach.

New possibilities of interference with neuronal functions without damaging the organism have arisen with the development of neuromodulation over the course of the past 40 years. The main field of action for neuromodulation is neuropathic pain as well as select forms of nociceptive pain which do not correspond to conventional pain therapy according to WHO recommendations. Due to its high level of invasiveness, neurosurgical pain therapy is always considered as the last step in a multimodal therapy setting.

It is not uncommon that patients suffering from major chronic pain never reach this last step, and physicians who treat them generally resign to accept an untreatable, chronic form of pain. Since pain is almost invisible to others, empathy with these patients is very difficult. Other factors that may complicate adequate treatment of chronic pain are competition between medical disciplines and cultural idiosyncrasies, as well as the feeling of a personal failure in case a doctor must admit that a patient suffers from chronic pain.

In this article, we present an overview of neurosurgical pain therapy for neuropathic facial pain following maxillofacial surgery.

11.2 Basics

11.2.1 Nociceptive Pain

Pain can be caused by mechanical, thermal, or chemical damage to tissue. Pain can be classified as nociceptive, neuropathic, or functional types. In the following section, we briefly describe anatomic and physiological properties of pain sensation and processing, since these are the basics for neurosurgical pain therapy. Chronification of pain, pain memory, the processing of chronic pain, neurotransmitters, neuromodulators, first messenger and second messenger systems of microglia, and astrocytes have been addressed in earlier manuscripts.

In nociceptive pain, stimulus reception occurs in peripheral receptors, i.e., end bulbs of Krause and Ruffini corpuscles and free nerve endings. Afferent C- and A-delta fibers then transmit the stimulus to the dorsal column of the spinal cord, i.e., nociceptive synapses in the substantia gelatinosa [1]. From there, the signaling pathway follows via ascending spinal cord neurons through the spinothalamic and the spinoreticular tract to the brainstem, on to the thalamus and finally to the cerebral cortex. Studies based on functional imaging positron emission tomography (PET) demonstrated that several structural centers perform pain processing and sensation, i.e., thalamus, basal ganglia, mesencephalon, periaqueductal gray, anterior cingular cortex, insula, primary sensory cortex I+II, motor cortex, perimetric cortex, supplementary motor area (SMA), prefrontal cortex, posterior parietal cortex, and cerebellum (1).

The lateral pain conduction mainly describes sensory-discriminative aspects of pain processing; the medial system mostly motivational-affective and cognitive-evaluative aspects. Pain memory seems to be contained within the medial system, as well as autonomous and endocrinal reactions. The lateral system conducts signals through the spinothalamic tract and the ventrobasal thalamus to the primary (I) and secondary (II) sensor cortical areas, the parietal operculum, and the insular cortex. In the medial system, signaling pathways run through the spinothalamic tract and the intralaminar and medial thalamic cores to the anterior cingular cortex, the amygdala, hippocampus, and hypothalamus as well as spinoreticular projections to the nucleus parabrachialis and locus coeroleus and via spinomesencephalic projections to the periaqueductal gray [2].

11.2.2 Neuropathic Pain

Contrary to nociceptive pain, neuropathic pain is caused by direct injury to the nervous system itself by traumatic, inflammatory, or toxic damage to a peripheral nerve, the plexus, the nerve root, or the central nervous system (infarct or intraparenchymal bleeding) or in the present topic the trigeminal nerve or its branches. This damage leads

to a loss of information within the central nervous system and is accompanied by a hypoesthetic area and burning pain. Damage or dysfunction of nociceptive fibers leads to abnormal or even loss of impulse generation, thus creating neuropathic degenerations to the axon, glia tissue, and the surrounding tissue. A decrease in concentration of substance P and calcitonin generated peptide as well as an increase in galanine and neuropeptide Y in afferent neurons lead to hyper excitability and ectopic neural pacemaker function. Furthermore, changes to the integrity of neural cell membranes take place. Endogenic suppression of pain with descending effects can terminate nociceptive input to the myelon by releasing noradrenalin and serotonin. In patients with chronic pain, the activability of this descending inhibition is reduced. Activation of glial cells caused by injury to a peripheral nerve, inflammation, or a strong pain stimulus promotes hyperalgesia. On the other hand, inhibition of glial cells prevents hyperpathia. Propentofylline may inhibit glial cells and, therefore, prevents hyperpathia. For further information on this topic, see current basic research of pain physiology. As mentioned above, neuropathic pain is associated with a loss of information in the central nervous system and activation of ectopic generators. The thalamus receives little or no information. Thalamic nuclei involved in pain processing are the ventral posterolateral nucleus, the ventral posteromedial nucleus, the ventral posterior internal nucleus, the posterior nuclei, and selected inter laminar nuclei. Pain is processed via bursts that are forwarded via sensor and/or nociceptive fibers from the peripheral system to the brain. If this information is missing, a sensory deficit, i.e., hypesthesia, occurs. In neuropathic pain, the missing information is replaced with dysesthesia, or more commonly, allodynia.

11.2.3 Neurosurgical Therapy

11.2.3.1 Neuromodulation and Stereotactic Surgery

Neuromodulation can be described as a reversible electrical interference of neural structures. As early as 60 B.C., the Roman physician Scribonius Largus used torpedo fish and electric eels to treat arthritic joint pain and headache [3]. After the development of the first cardiac pacemaker in Stockholm in 1958, evaluation of implantable neural stimulators began. Over the past 50 years, persistent stimulation of the spinal cord and the brain has proved to be a viable option for the treatment of neuropathic pain. In 1967, Shealy and Mortimer published the first report of subdural spinal cord stimulation for treatment of neuropathic leg pain [4]. In the 1960s, deep brain stimulation was developed and performed as an experimental treatment of chronic pain [5–17]. Since fully implantable stimulation systems were not available until the 1970s, deleting surgery and lobotomies were performed. In 1974, the first fully implantable, monopolar stimulation electrode became available, and in 1981, the first prototype of an integral stimulator was introduced. The battery lifetime of earlier systems was initially limited to a maximum of 6 years; since 2009, rechargeable systems with unrestricted lifetime are available. By using stereotactic devices intraoperatively, it is possible to perform minimally invasive surgery via small trepanations in order to treat dyskinetic and psychic disorders.

Stereotactic devices are highly sophisticated mechanical aids that have been developed between 1880 and 1960. In stereotactic surgery, a rigid circular frame is attached directly to the patient's head. Geometric localizer plates that are visible in CT or MRI scans are fixed to the frame. The acquired imaging data are then transformed into a virtual three-dimensional stereotactic space related to the frame and every single point within the patient's brain can then be addressed by space coordinates. For accurate, minimally invasive trepanation, an insertion guide is attached to the frame. The cerebral structure that is intended to be treated can then be stimulated by electrodes with different amplitudes and currents. The depth of penetration can be varied by using increasing currents; thus, inadvertent damage to adjacent structures can be avoided.

By stimulating cerebral structures before therapeutic sclerotherapy, it is possible to relocate functional areas accurately. As mentioned before, field current plays an important role in stereotactic surgery; however, by modulating the fre-

quency, different effects on brain tissue can be achieved.

Simulations with low frequency (1–80 Hz) cause neuronal activation or excitation; higher frequencies above 100 Hz cause blocking or inhibition of neuronal activity. As an example, stereotactic treatment of Parkinson's disease is performed by stimulation of the subthalamic nucleus. The varying degree of low frequency excitation and high frequency inhibition leads to an equalizing of all cerebral structures (basal ganglia) involved.

Over the past 25 years, deep brain stimulation has proven to be an effective treatment for dyskinetic disorders [18], while traumatizing procedures such as neurotomies and amputations have been performed for over 2000 years.

Neuromodulation as treatment for chronic pain has been available for 50 years, signifying a departure from destructive procedures. These surgical procedures can be separated into peripheral and central procedures. Peripheral neurostimulation or spinal cord stimulation is performed by all surgical subgroups such as neurosurgery, anesthesia, general medicine, plastic surgery, vascular surgery, orthopedics, and traumatology. Surgery on the central nervous system itself, especially the brain, should be performed only by specialized and highly experienced neurosurgeons. For neurosurgeons without a great deal of experience, deep brain stimulation and stereotaxy may be too challenging. Therefore, it is recommended to maintain the number of neurosurgeons specializing in this area at a low level.

11.3 Atypical Trigeminal Pain: Neuropathy of the Trigeminus Nerve

Neuropathic pain originating from the trigeminus nerve may be caused by direct trauma to the nerve in its central course, the ganglion gasseri, or to the peripheral branches (orbital nerve, maxillary nerve, and mandibular nerve). Trauma may occur by traumatic head injury/traumatic brain injury or by iatrogenic impairment during surgical procedures on the head and face.

Patients affected initially present dysesthesia and allodynia which do not regress completely. Complete disruption of afferent fibers may lead to one of the most painful conditions, analgesia dolorosa. It encompasses a complete loss of sensation together with permanent allodynic pain. This condition presents an extremely high grade of impairment and is accompanied by a high suicide rate among afflicted patients. Pain medication such as NSAIDs and opioids has only a very limited effect, even when administered in high doses. Opioids may lead to sedative effects which can be desirable as palliative measures. Even though neuroleptic medication and tricyclic antidepressants such as pregabaline, gabapentine, or amitriptyline may show better results, administration of this medication must often be reduced or terminated due to undesired side effects associated with long-term usage. For further information, see the literature on conservative treatment of neuropathic pain [19]. The following section considers surgical treatment of trigeminal neuropathy [20].

Until the 1970s, trigeminal neuropathy was not considered as an entity separate from other neuropathies despite different underlying pathologic conditions. Therefore, treatment options were rhizotomies and tractotomies (medullar, pontine, mesencephal, and spinothalamic tracts). Morbidity and mortality rates were comparatively high, as was the rate of recurrence. Beginning in 1947, stereotactic mesencephalotomies were introduced. Less invasive methods included thermocoagulation or rhizotomy of the gasseri ganglion with glycerol. Prof. William Sweet performed thermocoagulations of the ganglion; he observed that patients already reported pain relief during probative stimulation of the ganglion for correct position of the probes.

Patients suffering from typical trigeminal *neuralgia* report zapping pain, usually without loss of sensation. The suspected underlying cause is a pathologic contact between nerve and vessels in the trigeminal root entry zone, i.e., a short span of the trigeminal nerve before the entrance into the brainstem. In this area, the neural sheath consists of vulnerable and sensible oligodendroglia instead of myelin. Pulsations in a

circular section of either the anterior inferior cerebellar artery (AICA) or posterior inferior cerebellar artery (PICA) lead to focal demyelination in the root entry zone and consecutively, sensible afferences (e.g., contact, talking, chewing, draft,…) develop into nociceptive afferences, thus causing pain. Therapeutic approaches include pharmaceutical means such as carbamazepine; surgical options include microvascular decompression (Jannetta technique), rhizotomy with glycerol, or balloon decompression of the ganglion gasseri. Furthermore, gamma knife techniques can be applied. Complications related to surgery may cause damage to nerve, associated with the onset of permanent pain. As mentioned before, the occurrence of a sensory deficit in conjunction with burning pain is characteristic for trigeminal neuropathy. A former tutor of mine, Prof. Ulrich Steude from Munich University [21], developed a method for stimulation of the ganglion gasseri; he reported that 2 out of 3 patients showed an improvement of more than 60% of their painful sensations. He described the importance of neural stimulation between the damaged structure (the nerve) and the final processing entity (the brains cortex – the consciousness of humans) [22–29]. This anatomical prevalence differs from transcutaneous electric nerve stimulation (TENS) as well as subcutaneous electric stimulation, where stimulation ensues distal to the lesion.

The following case reports demonstrate the historic development in the therapy of neuropathic facial pain over the course of the past 40 years. This timespan was chosen deliberately, since the revolution in imaging took place in the 1970s with the advent of computed tomography, leading to revolutionary new techniques in neurosurgery.

Description of the implantation of electrodes into the ganglion gasseri in a 18-year-old female suffering from pharmacoresistant neuropathic facial pain after extraction of a wisdom tooth maxillary right.

The following section gives a detailed description of ganglion gasseri stimulation, first described by my teacher Prof. Ulrich Steude, Neurosurgical Department, Ludwig Maximilian University of Munich, Germany [21]. Patient treated with this method initially receive a testing electrode implanted through the foramen ovale directly to the ganglion gasseri. Similar to thermocoagulation of the ganglion, this takes place under short general anesthesia. In a stationary setting, the effect of this stimulation is experienced over the course of a few days. A positive effect is achieved when the painful area now includes paresthetic sensations caused by stimulation, and the pain is reduced by at least 60%. Four weeks later, the definitive implantation of a stimulation electrode is performed. For definite implantation, a monopolar electrode containing a bullet contact on the tip of the electrode is used and wired to the stimulator which is similar to a pacemaker. Several aspects during the intervention are crucial for the therapeutic effect. First, proper positioning of the patient must be ensured. The patient's head should be reclined and facing the surgeon directly. Thus, the foramen ovale lies in a straight line to the surgeon and its entire diameter is available for the insertion probe. Tangential insertion of the probe in the foramen may not be successful since the insertion probe consists of an 18G needle. The next step is to scrub the skin, starting at the corner of the patient's mouth. Due to the positioning, the eyes may be affected by disinfecting agents. This should be avoided by all means since the patient is awake and may not tolerate this irritation, possibly aborting the intervention. The insertion of the probe should not be performed too low in the relation of the corner of the mouth or to the horizontal extension of the lips, which may lead to displacement of the electrode on opening the mouth. If such misplacement has occurred, the electrode may be dislocated distally by the opening of the foramen ovale; thus, dysesthesia sensations caused by stimulation may be transferred into areas that are primarily not affected by pain. In one of our cases, a patient was treated successfully with stimulation but her frequent usage of chewing gum led to deterioration of the symptoms due to lateral bending of the cheek.

The ideal trajectory follows a straight line to the pupil at frontal view until crossing at halfway point of an imaginary line between external

acoustic meatus and lateral canthus. Puncture too far laterally may lead the probe into the foramen spinosum and may cause puncture of the medial meningeal artery. A significant hematoma without loss of function may occur.

After correct placement of the probe within the foramen ovale, the mandrin is replaced by a standard stimulation electrode. Then, the generator is set to zero amps and a rectangular impulse at 2 Hz is selected. Stimulation intensity is increased gradually until rhythmic fasciculation of the patient's mouth, i.e., chewing movements are observed. A flush within the stimulated trigeminal areas may occur, especially at an intensity of 50 Hz. It is important to instruct the patient now to keep his head still. For definite testing, intensity is reduced once again to zero and is then increased very slowly. Then, the patient is asked where he senses paresthesia, i.e., jaw, cheek, ala of the nose, forehead, or around the eye. According to the stimulated area, the position of the electrode is corrected. When sufficient stimulation has been reached, the testing electrode is removed and replaced by a permanent electrode, either for further testing or for definite stimulation. For replacement, the cannula for puncture is inserted over the electrode; caution must be exercised when removing the cannula in order not to remove the electrode. Lateral fluoroscopy is recommended for verification of correct placement of the electrode. The electrode should be fixated to skin by small stitches and steri strips. The patient is the transferred to the ward and receives instructions for the test stimulation. Different stimulation settings should be tried over the course of a few days in order to find the best result. In our experience, definite implantation of a stimulating electrode and the pacemaker should not be performed immediately after the testing phase. Due to the percutaneous channeling of the electrode, bacterial contamination of the operating area must be avoided. Bacteria may ascend by 0.3 mm per day; the tunneling of test electrodes on the face should only be performed over the course of a few centimeters, while definite electrodes may be tunneled percutaneously over a longer distance. Therefore, in a test setting, the risk of wound infections is relatively high. Furthermore, due to time pressure when the patient awaits the definite implantation, expectations regarding the therapeutic effect may be interpreted erroneously and placebo-like effects may occur. Therefore, we recommend implantation of the definite system at least 4 weeks after the testing phase. The test electrode can be removed without any anesthesia. The electrode is pulled out after removing the fixating steri strips.

Implantation of the definite system is performed similar to the testing system. An additional skin incision must be made with a thin nonabsorbable suture loop; puncture of the foramen ovale is then performed in the same way as in the test stimulation, and the suture is then tightened for fixation. The electrode is than tunneled subcutaneously to the jaw angle and via connectors to the ipsilateral infraclavicular area. There, the neurostimulator is inserted subcutaneously and connected to the electrode. The stimulator is programmed percutaneously. Outpatient follow up should occur every 3 months initially, and then once a year.

11.4 Stereotactic Implantation of Electrodes into Basal Ganglia

This chapter explains central modulation of neural structures. Stimulation of certain areas of the sensory thalamus (lateral and medial ventral posterior nucleus) as well as the posterior limb of the internal capsula or other structures of the pain-processing areas described previously may reduce but not eliminate neuropathic facial pain. If an additional nociceptive component exists, stimulation of the periaqueductal or periventricular gray may lead to release of endogenous opioid peptides; therefore, an activation of inhibiting descending tracts occurs.

A meta-analysis performed by the European society of functional neurosurgery in 2007 [30] demonstrated that deep brain stimulation achieved better results against nociceptive pain (63%) than against neuropathic pain (47%). This demonstrates the low level of quality that used to be achieved by stereotactic surgery. The main

principle of applying an electrode into a target area followed by regulation via a stimulator requires a high level of accuracy in surgical planning; only then is it possible to achieve good results and few undesired side effects with a low amount of voltage. The meta-analysis showed that stimulation of the physically largest target area, the periaqueductal gray, had better effectivity than stimulation of the smaller target structures within the thalamus (size of 1–2 mm). Nevertheless, interpretation of these results is variable. Therefore, further considerations in order to treat neuropathic pain were taken into account.

The next step was to reintroduce the posterior limb of the capsula interna as a target area. We learned that electrode placement had been performed too far dorsally in all international centers in the past; therefore, it was not possible to exploit the full potential of this technique. The combination of stimulating both the sensory thalamus and the dorsal limb of the capsula interna may result in complete reduction of neuropathic facial pain, even with analgesia dolorosa. It is also possible to achieve satisfying results in treatment for allodynia, hyperpathia, and dysesthesia. If an additional nociceptive component exists, it is possible to stimulate the periventricular or periaqueductal gray and, therefore, activate inhibiting descending tracts through release of endogenic opioid peptides. In conclusion, we were able to develop stereotactic surgery for the use of neurosurgical pain therapy over the course of the past 25 years; today, we are able to eliminate pain entirely in patients suffering from the severest forms of chronic pain conditions.

11.4.1 Case Report: Atypical Facial Pain Following Maxillofacial Surgery

A 36-year-old female patient with congenital hemi-maxilla hypoplasia right side without neurological deficit especially sensory deficit in the second trigeminal branch on both sides and without pain in the face was treated with bimax osteotomy in 2015 by maxillofacial surgery in another university hospital. She was suffering from face asymmetry with chin pointed to the right and tilt of the occlusal plane. A bimax osteotomy, a genioplasty, onlay with bone from the iliacal crest, and a nose train's lung were performed. On the right side, the maxila was mobilized 5 mm caudaly and on the left side 2 mm cranialy correcting the occlusal plane tilt. The mandibula got medialy cut and mobilized. Following surgery and removal of the osteosynthesis instrumentation in 2016, the patient had a severe sensory deficit in the face on the left side. She has a permanent sensory deficit with some unpleasant pressure sensation in the left cheek including sensory deficit in the anterior left side of the tongue. A tormenting mouth dryness and a constant burning pain was treated by NSAR and opiods, followed by antiepileptic drugs and triciclic antidepressants with no reduction of the constant radiating pain in the area of the maxillary nerve on the left side under VAS 6 ranging up to VAS 8 resulting in body weight loss of 15 kg in a slim patient. In the end, she gained a light pain reduction by pregabaline 100 mg 1-0-1, tramabene 50 mg 1-0-1, and trittico 150 mg 0-0-1. Increasing the dosage was resulting in more side effects and in no further pain reduction. The patient was able to sleep 2 h in the night, and then she woke up and had to do things like going around or cleaning the house to get some reduction in pain. By doing so she was able to sleep 2 more hours after these 2 h of doing something. In September 2017, she got introduced to me by a former pupil and collegue. After realizing that we have no further pharmacological possibilities I explained all surgical methods to her and favorizing deep brain stimulation of the sensory thalamus and the posterior limp of the internal capsule ipsilateral to the painful side according to my development followed by ganglion gasseri stimulation on the left side and the last option epidural motor cortex stimulation. The hirachial order was determined by the fact that for ganglion gasseri stimulation we had to perform the surgery through the maxillofacial surgery field which is associated with a higher risk of malfunction of the procedure. The reason for becoming last option for motor cortex

stimulation is that the patient is facing a high risk of dysfunction of the method following 2 to 3 years of therapy according to scare tissue between dura and electrode [31]. In 25 October 2017, we implanted stereotactically under general sedation analgesia two electrodes into the left cerebral hemisphere. The sensory thalamic electrode was applied by a pre-coronal approach and the internal capsule electrode by a parietal postcentral approach. The two electrodes were extended by regular extension cables which got externalized percutaneously to the midline where it got sutured and fixed by a tobacco pouches suture. Following a percutaneous test trail period of 7 days, we all understood that both electrodes were treating the patient perfect. A cessation of the burning pain in the sensory deficit area including the wind up phenomenon resulted and the stimulation system got internalized. Stimulation settings were in electrode 1 in sensory thalamic neurons a bipolar setting with contact polarity of contact 1 negative and contact 2 positive, intensity 0.4 mA, pulse widths 50 μs and 50 stimuly per second. In electrode 2, in the posterior limp of the internal capsule electrode contact polarity was a bipolar setting with contact 1 negative and contact 3 positive, 0.4 mA intensity, 50 μs pulse widths, 50 Hz. One and a half months later, the microthalamotomy effect ceased and the patient had a deep pressure in the cheek and some pain not as severe as before surgery but more as she went home from our hospital. We increased the intensity of the thalamic electrode from 0.4 mA to 0.8 mA and in the posterior internal capsule limp electrode from 0.4 mA to 0.9 mA and all symptoms disappeared and she felt an almost normal sensation in the previous painful area including the tongue. The morphine medication got washed out and was stopped. Six weeks later, we increase the thalamic electrode to 1.9 mA and the internal capsule electrode to 0.95 mA with very good pain treatment. The patient gained already 7 kg weight. Three months later, the intensity was increased on both electrodes to 2.7 mA. After all that the patient had to undergo 10 dental treatments to restaurate her teeth causing more pain. Two years after our operation we increased the stimulation area by adding one contact by activation in the thalamic electrode and 2 contacts in the internal capsule electrode with 2.95 mA intensity, 50 μs pulse widths, 50 Hz frequency and we were able to stabilize our excellent results. We allowed the patient to increase and decrease intensity in a certain frame from 0.3 mA to 3.5 mA according to her needs. With our new method we were able to manage 2 1/2 years of a new quality in life of a severe neuropathic pain patient.

11.4.2 Case Report: Fracture of the Left Maxillary Bones

A female patient got her left midface bones, maxillary bone, maxillary sinus, and orbital floor broken by a stroke of her horse behind hand hoof. Immediately she felt a sensory deficit and a numbness in her left cheek. Maxillofacial surgery was performed at another university hospital. Instrumentation and reconstruction of the broken bones from the orbital floor to the maxillary bone were performed in 2006. Because of severe neuropathic pain within the sensory deficient area the instrumentation got removed in 2012 without positive effect on her pain situation. Medication with Neurontin 900 mg 1-1-1, Hydal 12 mg 1-0-1, Dronabinol 13 trops 1-1-1 kept the pain intensity between VAS 6 and 8 of ten. In 2015, a test trial with ballon compression of the gasserian ganglion by utilizing a fogarty catheter was performed. In 2017, we performed a deep brain test stimulation in the ipsilateral sensory thalamic region of the ventrolateral thalamus and the dorsal limp of the internal capsule. Electrophysiological examinations by quantative sensory testing revealed a severe hyperpathy for cold, warm, and sharp sensation with a clear wind up phenomenon on repeated non-painful sensory stimulation. Within 6 days, a significant pain reduction could be achieved resulting in an internalisation of the stimulation system. Stimulation parameters of the left thalamus were contact 1 negative and contact 2 positive, 60 μs, 50 Hz, 1.6 V. The stimulation parameters in the internal capsule were

contact 0 negative, contact 1 positive, 60 μs, 50 Hz, and 0.8 V. One week after surgery the sensory testing revealed no difference between the right and the left sides of her face. The pain got completely extinguished. Our new method against severe pharmacolgical nonresponsive neuropathic pain syndromes is high effective and stable since 2012.

11.5 Epidural Motor Cortex Stimulation

Epidural stimulation of the motor cortex [30–34] has been abandoned by our group after 5 years of application. All of our patients reported a significantly diminished effect or even a loss of effect after 2 years or more. Even with a reduction of pain up to 30–60%, the persisting pain was still reported as unacceptable. In the early stages of the therapy, an increased intensity of stimulation led to increased muscular tone in the associated areas; therefore, we concluded that the positioning of the electrodes on the motor cortex was correct. Nevertheless, the pain reduction decreased continuously. After more than 2 years of stimulation and confirmed integrity of the system, proven by correct resistances within the system, positive effects could not be reproduced. Revision surgery was performed in a few patients; in two cases, we found thickening of the dura, and in one patient a calcified layer between dura and electrodes was found. In our hypothesis, these alterations are due to a shift in pH and precipitation of calcium, caused by high electrical intensity. This biochemical reaction can be observed in all applications of external electrical fields, i.e., plate electrodes in deep brain stimulation or spinal stimulation as well as in generators of deep brain stimulation or cardiac pacemakers. Around the source of electric current, i.e., the electrode, a tough, crude coating is formed; this fibrotic or even calcified coating leads to isolation of the electric current in terms of an endogenous defense reaction. These observations led to abandoning of epidural motor cortex stimulation in favor of deep brain stimulation for neurosurgical pain therapy.

11.5.1 Cluster Headache

11.5.1.1 Etiology and Therapy of Cluster Headache

The following section describes the etiology as well as neurosurgical therapy of cluster headache that has proven not to respond to pain medication [35, 36]. This painful facial condition is described as one of the most affecting pain syndromes. It always occurs unilaterally on the same side of the face, switches to the contralateral side are not described. Van Swieten was the first to perform research on this entity and published his findings as early as 1745. The current guidelines regarding this topic derive from International Headache Society, including a classification of headache and facial pain syndromes as well as diagnostic criteria. Cluster headache consistently affects the first trigeminal branch and the ophthalmic nerve. Pain is accompanied by vegetative symptoms such as ipsilateral lacrimation, nasal secretion, conjunctival injection, myosis, ptosis, blepharedema, and activation of the parasympathicus. Characteristic attributes are cyclic occurrence of the symptoms, as well as seasonal variations, exact circadian rhythm, and punctuality. Initially, it was assumed that inflammatory processes at the sinus cavernosus, afferent veins, or the sympathic plexus around the internal carotid artery cause these symptoms. This theory was supported by the fact that vasodilatation by nitroglycerine causes the onset of pain attacks. On the contrary, the strict one-sidedness as well as the circadian rhythm may be indicative for a central nervous process. Today, the vascular alterations are assumed to be epiphenomena of an activation of the trigeminovagal system.

During attacks of cluster headache, the plasma level of testosterone varies; furthermore, a decreased sensitivity for thyreotropic releasing hormone and a loss of the circadian melatonin rhythm accompanied by nocturnal serum level peaks occurs. These factors support the hypothesis of hypothalamic participation. Circadian processes are known to be controlled by the ventral hypothalamus, i.e., oscillators in the suprachiasmatic nuclei, and furthermore, influences caused by brightness occur via retinohypothalamic tracts;

therefore, it is assumed that the trigger for cluster headache must be located within the hypothalamus or its immediate vicinity. Due to the possibility of provoking the attacks with nitroglycerine and terminating the attacks with sumatriptane, an examination of these phenomena by means of a PET scan (positron emission tomography) was indicated; here, a radioactive water molecule with a short half-life period (H2O15, 7 min) was used. Researchers at the Department for clinical neurology and cognitive neurology, Queens Square University Hospital, London, UK [36] were able to reproduce activities typical for facial pain in the anterior cingular cortex bilaterally, the insula, the thalamus, the cerebellar hemispheres, and the vermis cerebelli without any activation of the brainstem as seen in migraine, by using nitroglycerine and sumatriptane. Interestingly, ipsilateral activation of the hypothalamus and the hypothalamic gray in patients affected by cluster headache was detected.

Therefore, it was concluded that deep brain stimulation, similar to treatment of dyskinetic disorders, could be a viable therapeutic approach. A platinum electrode is placed within the active area of the hypothalamus and inhibitory high-frequency stimulation is performed.

The outcome of this approach is excellent; hundreds of patients have been treated successfully. A limitation of this method, however, is the fact only few patients suffering from this diagnosis do not respond to classic medication and, therefore, are eligible for deep brain stimulation.

11.5.2 Neurodestructive Interventions

Destructive surgery such as chordotomy, tractotomy, or exheresis is no longer performed by our study group. These interventions should only be performed in case of extreme pain originating from malignant diseases that do not respond to other therapeutic means and when only a short life expectancy is given. It has been proven that due to the surgical deafferentation, even more intense pain may occur.

Physicians who are not specialized in functional neurosurgery and neurosurgical pain therapy may ignore these facts. It can often be observed that patients who seemingly do not respond to surgical therapy are transferred to psychiatrists; however, it is the responsibility of all specialists in pain management to handle even the most difficult cases and increase the quality of life of our patients.

11.5.2.1 Case Report: Trigeminal Neuralgia/Neuropathy, Deep Brain Stimulation—Novel Therapeutical Concepts, Radiosurgery

The following section describes treatment of a difficult case of failed therapy in chronic trigeminal pain; step-by-step, all therapeutic options had to be considered in order to achieve satisfying pain reduction and quality of life.

In 1999, a female in her 30s started to develop acute, lancinating pain in the second and the third trigeminal branch on the left side. NSAIDs showed no significant effect on both pain intensity and frequency of pain attacks. Administration of carbamazepine at up to 2.5 g per day as well as high-dosed amitriptyline and pregabaline could reduce the intensity, but the results were still not satisfying. From 2002 onwards, the patient developed trigeminal pain also on the right side. In 2006, after more than 6 years of unsatisfying conservative treatment, microvascular surgical decompression of the "root entry zone" (entry point of the trigeminal nerve in the brainstem) on the left side was performed. Intraoperatively, a loop of the anterior inferior cerebellar artery (AICA) was found to compress the root entry zone; the vessel loop was removed, and a Teflon sponge was used as an interponate. Half a year later, the same procedure was performed on the right side. The initial results were good; the patient was pain free. Within 1 year, however, the symptoms recurred on the left side and then also on the right side. Conservative treatment now showed better results than in the initial treatment of the patient, but the results remained unsatisfying. In 2010, surgical explo-

ration and re-decompression of the right trigeminal nerve was performed; this procedure, however, showed no effect on pain intensity and frequency of pain attacks. Again, conservative treatment showed no improvement; after interdisciplinary review, a rhizotomy of the trigeminal nerve was considered. By the end of 2012, the patient was still suffering from unbearable chronic pain of neuropathic character accompanied by partial hypoesthesia on the right side of the face and additional intermittent neuralgic pain attacks. Therefore, a selective sensory rhizotomy of the sensory branch of the trigeminal nerve was performed. Postoperatively, the patient described a complete loss of touch sensitivity on the right side of the head and neck. Subjectively, the patient felt a hot, burning form of dysesthesia; small drafts of air or slight touching lead to unbearable pain. Gabapentine, amitriptyline, and pregabaline were administered in the highest dosage possible. After removal of the stitches, the patient was discharged. The patient then had to tolerate chronic neuropathic pain with abnormally intensified touch sensitivity and dysesthesia. She could not tolerate breezes of air or moderate levels of noise. Bright light, such as reflections in the snow were also unbearable. The additional administration of clomipramine resulted in a slight improvement if the patient could reduce external disturbances.

After rhizotomy on the right side, the patient developed recurring neuralgia on the left side with lancinating pain in the nasolabial sulcus and around the corner of the mouth to the left; high dosage of carbamazepine and other specific medication showed no effect. The patient was admitted to the gamma knife center at the general hospital (AKH) in Vienna; in April 2014, radiosurgery on the left trigeminal nerve was performed. Afterwards, pain on the right facial side including the eye was increasing again; the patient was finally admitted to our department for evaluation of further therapeutic options. We decided on application of our modified therapeutic concept for chronic neuropathic pain, i.e., test stimulation of the sensory thalamus (ventro-postero-medial nucleus) and the posterior limb of the capsula interna ipsilateral to the affected side of the face.

In May 2014, an electrode for deep brain stimulation was implanted into the right sensory thalamus and another electrode into the posterior limb of the right capsula interna (in general anaesthesia). The electrodes were temporarily tunneled subcutaneously, thus enabling activation through the skin on the head. After the implantation, the patient reported improvement regarding the burning pain sensation; this was seen as the microthalamotomy effect after correct implantation of electrodes. Over the course of the next 3 days, all contacts of the 4-pole platinum electrode were repeatedly test stimulated; the neuropathic, chronic pain sensations as well as the intense, painful touch sensitivity had disappeared. Even the preexisting allodynia had disappeared; the patient was able to touch her face again without pain. In the next step, the definite stimulating system was implanted and the pulse generator was activated. The stimulation intensity was programmed in way that the patient was pain free on the right side of the face when she did not move. Under physical strain, the patient still felt a sensation of flush on the right side; this was reported as unpleasant, but not as painful as it was before the implantation of the electrodes. Sometime later, the patient developed a light tingling, i.e., intermittently appearing lancinating pain on the left side of the face. Pharmaceutical treatment improved these sensations; the patient regularly visits our specialized outpatient department for treatment of chronic pain. At the moment, the patient reports a high quality of life with little to no pain.

This case report demonstrates the importance of surgical treatment of chronic pain. Limitation of treatment options solely to pharmaceutical means is a common mistake. Nevertheless, it is important to evaluate invasive therapy under critical consideration of potential risks and undesired side effects.

The Innsbruck Algorithm of invasive intracerebral pain treatment (W. Eisner).

Preconditions:
- Failure of at least three different conservative therapeutic attempts performed by specialists
- Last surgical intervention over 12 months in the past
- No contraindication against surgery

Preoperatively:
- Anamnestic definition of the underlying type of pain: neuropathic, nociceptive, or mixed type
- Stereotactic MRI scan for surgical planning
- Neuropsychological testing
- Administration to our outpatient department
- Neurophysiological exams for quantitative follow up
  - Step Ia: Neuropathic pain
    - Implantation of electrodes into the sensory thalamus and the posterior limb of the capsula interna in general anesthesia
  - Step Ib: Nociceptive pain or mixed type
    - Implantation of electrodes into the sensory thalamus, the posterior limb of the capsula interna, and the periventricular/periaquaeductal gray
  - Step II: After failure of Step I, after removal of test electrodes
    - Revision surgery with combined local anesthesia and intravenous analgosedation, 5-channel-macrostimulation via preexisting burr holes while the patient is awake, reevaluation of the individual functionality of the selected target area
  - Step III: After failure of Step II, continuing unbearable pain, all pain types
    - Implantation of electrodes into the anterior cingulate cortex bilaterally
  - Step IV: After failure of Step III or in addition to Step III, all pain types
    - Intracerebral intraventricular administration of morphine after positive testing via port system with implanted pump

11.5.3 Special Notes on Deep Brain Stimulation

Deep brain stimulation has been performed by our study group in Munich as well as in Innsbruck since the early 1990s. No severe complications have been observed in our patient population. We have treated over 1000 patients; more than 100,000 patients have been treated worldwide. DBS can be regarded as a safe and clinically reliable method. The low number of surgeons performing this method is caused by its complexity.

One special focus should include the consequent avoiding of bacterial infections such as pneumonia or urinal tract infections since ascending infections of the implants are common. Prophylactic antibiotics should be administered long enough in order to prevent the need of implant removal and, therefore, continuing pain.

11.5.4 Relevance for Practice

Cases similar to those we described here can be found in numerous medical facilities, hospitals, as well as ambulatory offices. We intend to demonstrate alternate "exit strategies" in seemingly desperate cases of chronic pain. Furthermore, we plea for understanding that ups and downs of therapeutic results should not be accounted as failures of others instantly. Rather, we intend to achieve a form of relief for other participating entities with potential benefit for the patient. In Austria, we intend to achieve complete documentation of all therapeutic interventions described in this manuscript, and we plea for consequent teaching in neurosurgical pain therapy. Facial pain must be regarded as an interdisciplinary entity; it encompasses odontology, maxillofacial surgery, neurology, anesthesiology, and neurosurgery.

Conflict of Interest The author is lecturer, trainer, and instructor at the "International Young Neurosurgeons Training Program" by Medtronic™.

References

1. Melzack R, Wall PD. Pain mechanism: a new theory. Science. 1965;150:971–8.
2. Kupers R, Gybels J. What have PET studies taught us about cerebral mechanisms involved in analgesic effect of DBS? In: Lozano AM, Gildenberg PL, Tasker RR, editors. Textbook of stereotactic and func-

tional neurosurgery. Berlin: Springer-Verlag; 2009. p. 2217–32.
3. Scribonius L, Helmreich G, editors. Compositiones medicamentorum. Leipzig: Teubner; 1887.
4. Shealy NC, Mortimer TJ, Reswick JB. Electrical inhibition of pain by stimulation of the dorsal column, anesthesia and analgesia. Anesth Analg. 1967;46:489–91.
5. Heath RG, Mickle WA. Evaluation of seven years experience with depth electrode studies in human patients. In: Ramey ER, O'Doherty DS, editors. Electrical studies on the unanesthetized human brain. New York: Paul B. Hoeber; 1960. p. 214–47.
6. Mazars G, Merienne L, Ciolocca C. Intermittent analgesic thalamic stimulation. Preliminary note. Rev Neurol (Paris). 1973;128:273–9.
7. Mazars G, Merienne L, Cioloca C. Treatment of certain types of pain with implantable thalamic stimulators. Neurochirurgie. 1974;20:117–24.
8. Mazars GJ. Intermittent stimulation of nucleus ventralis posterolateralis for intractable pain. Surg Neurol. 1975;4:93–5.
9. White JC, Sweet WH. Pain and the neurosurgeon: a 40-year experience. Springfield, IL: Charles C. Thomas; 1969.
10. Hosobuchi Y, Adams JE, Rutkin B. Chronic thalamic stimulation for the control of facial anesthesia dolorosa. Arch Neurol. 1973;29:158–61.
11. Hosobuchi Y, Adams JE, Rutkin B. Chronic thalamic and internal capsule stimulation for the control of central pain. Surg Neurol. 1975;4:91–2.
12. Adams JE, Hosobuchi Y, Fields HL. Stimulation of internal capsule for relief of chronic pain. J Neurosurg. 1974;41:740–4.
13. Fields HL, Adams JE. Pain after cortical injury relieved by electrical stimulation of the internal capsule. Brain. 1974;97:169–78.
14. Richardson DE, Akil H. Long-term results of periventricular gray self-stimulation. Neurosurgery. 1977;1:199–202.
15. Richardson DE, Akil H. Pain reduction by electrical brain stimulation in man. I. Acute administration in periaqueductal and periventricular sites. J Neurosurg. 1977;47:178–83.
16. Hosobuchi Y, Adams JE, Linchitz R. Pain relief by electrical stimulation of the central gray matter in humans and its reversal by naloxone. Science. 1977;197:183–6.
17. Dieckmann G, Witzmann A. Initial and long-term results of deep brain stimulation for chronic intractable pain. Appl Neurophysiol. 1982;45:167–72.
18. Deuschl G, Schade-Brittinger C, Krack P, et al. A randomized trial of deep-brain stimulation for Parkinson's disease. N Engl J Med. 2006;355:896–908.
19. Baron R, Sommer C, Tölle TR, et al. Diagnostik und Therapie neuropathischer Schmerzen. In: Diener HC (Hrsg.). Leitlinien für Diagnostik und Therapie in der Neurologie. 3. aktuelle und erweiterte Auflage. Stuttgart: Georg Thieme Verlag; 2005. p. 531–44.
20. Barker FG, Jannetta PJ, Bissonette DJ, et al. The long-term outcome of microvascular decompression for trigeminal neuralgia. N Engl J Med. 1996;334:1077–83.
21. Steude U. Percutaneous electro-stimulation of the trigeminal nerve in patients with atypical trigeminal neuralgia. Acta Neurochir. 1978;21:66.
22. Meyerson BA, Hakanson S. Allevation of atypical trigeminal pain by stimulation of the Gasserian ganglion via an implanted electrode. Acta Neurochir Suppl (Wien). 1980;30:303–9.
23. Steude U. Radiofrequency electrical stimulation of the Gasserian ganglion in patients with atypical trigeminal pain. Methods of percutaneous temporary test-stimulation and permant implantation of stimulation devices. Acta Neurochir Suppl (Wien). 1984;33:481.
24. Steude U. Percutaneous electrical stimulation of the Gasserian ganglion in patients with atypical trigeminal neuralgia. A new technique for percutaneous test stimulation and permanent implantation of a stimulation device. In: Erdmann W, editor. The pain clinic I. Utrecht: VNU Science Press; 1985. p. 239.
25. Meglio M. Percutaneously implantable chronic electrode for radiofrequency stimulation for the Gasserian ganglion: a new perspective in the management of trigeminal pain. Acta Neurochir Suppl (Wien). 1984;33:521–5.
26. Young RF. Electrical stimulation of the trigeminal nerve root for the treatment of chronic facial pain. J Neurosurg. 1995;83:72–8.
27. Raab WHM, Kobal G, Steude U, et al. Die elektrische Stimulation des Ganglion Gasseri bei Patienten mit atypischem Gesichtsschmerz: klinische Erfahrung und experimentelle Kontrolle durch elektrische Pulpareizung. Dtsch Zahn Mund Kieferheilk Zentralsl. 1987;42:793–7.
28. Steude U, Fritsch H, Kobald G. Therapeutic electrostimulation of the trigeminal ganglion in patients with atypical trigeminal neuralgia and the response on real pain evoked potentials. Mod Neurosurg. 1989;2:305–10.
29. Steude U, Stodiek S, Schmiedek P. Multiple contact foramen ovale electrode in the presurgical evaluation of epileptic patients for selective amygdalaohippocampectomy. Acta Neurochir Suppl (Wien). 1993;58:193–4.
30. Cruccu G, Aziz TZ, Garcia-Larrea L, Hansson P, Jensen TS, Lefaucheur JP, Simpson BA, Taylor RS. EFNS guidelines on neurostimulation therapy for neuropathic pain. Eur J Neurol. 2007;14(9):952–70.
31. Slotty Philipp J, Wilhelm E, Honey Christopher R, Christian W, Jan V. Long-term follow-up of motor cortex stimulation for neuropathic pain in 23 patients. Stereotact Funct Neurosurg. 2015;93:199–205.
32. Tsubokawa T, Katayama Y, Yamamoto T, et al. Chronic motor cortex stimulation for the treatment of central pain. Acta Neurochir Suppl (Wien). 1991;52:137–9.
33. Tsubokawa T, Katayama Y, Yamamoto T, et al. Chronic motor cortex stimulation in patients with thalamic pain. J Neurosurg. 1993;78:393–401.

34. Meyerson BA, Lindblom U, Linderoth B, et al. Motor cortex stimulation as treatment of neuropathic trigeminal pain. Acta Neurochir Suppl (Wien). 1993;58:150–3.
35. Katayama Y, Tsubokawa T, Yamamoto T. Chronic motor cortex stimulation for central deafferentation pain: experience with bulbar pain secondary to Wallenberg syndrome. Stereotact Funct Neurosurg. 1995;34:42–8.
36. May A, Bahra A, Büchel C, et al. Hypothalamic activation in cluster headache attacks. Lancet. 1998;352:275–8.

Wilhelm Eisner Born in lower Bavaria, Germany, study in chemistry in Cologne and Mainz, Germany, degree in medicine in Munich, Germany, 1990 to 1999 neurosurgical residency at the Ludwigs-Maximilian University at Grosshadern, Munich, neurosurgical consultant in 1998, since 1999 Department of Neurosurgery, Medical University Innsbruck, Austria, habilitation in 2001; hospitations and fellowships: 1991, Inselspital Berne, Switzerland, (UD Schmid and K Rössler), 1992 Childrens Hospital Pittsburgh, PA, USA (P Jannetta, A Moller, LD Lunsford), 1998 University of Cologne, Germany, Department of Stereotaxy and Functional Neurosurgery; spokesman of the neurosurgeons in Tyrol at the Austrian medical chamber, spokesman of the working group on functional neurosurgery and stereotaxy, pain therapy, *radiosurgery*, and intraoperative electrophysiology of the Austrian Society of Neurosurgery (ÖGNC), counselor at the Austrian Society of functional MRI, board member of the Austrian Pain Society, vice president of the German Society of Neuromodulation, editorial board member of the Journal of Interdisciplinary Neurosurgery (USA), authorized and certified court expert advisor.

Part IV

Tumor

12 Complications in Skull Base Surgery

Álvaro Rivero Calle
and Gregorio Sánchez Aniceto

Contents

Anterior craniofacial resection for tumors affecting the skull base was first described in the medical literature around 1954 (Klopp), with further patient series published in the 1960s (Ketcham). After these first experiences, the procedure was almost forgotten for almost three decades. The initial reluctance to universally accept skull base surgery by the medical community was based in the high complication rate and perioperative mortality, above the standard numbers of any other oncological surgery procedure. This is especially because of the exposure of the delicate structures in the intracranial space to the upper nasal airway, the complex defects created, and difficulties for optimal safe reconstruction at those initial times. With the principles of pediatric craniofacial surgery established by Paul Tessier in the 1970s, then applied to oncological surgery by Ian Jackson in the 1980s, and then consolidated by Sekar and Janecka in the 1980s and 1990s, craniofacial

Á. Rivero Calle · G. Sánchez Aniceto (✉)
Oral and Maxillofacial Surgery Department, 12 de Octubre University Hospital, Madrid, Spain
e-mail: alvaro.rivero@salud.madrid.org; gsaniceto@meytel.net

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_12

resection became a standard procedure in head and neck oncological surgery with good results in terms of tumor control and patient survival. While expected perioperative mortality has been decreased to 4.5%, complication rate is still high (30–35%), and transnasal endoscopically assisted craniofacial resection seems to minimize the incidence of approach-related complications while still obtaining good tumor control. We may classify complications as follows:

1. Intracranial complications.
2. Complications related to the surgical wound.
3. Systemic complications.
4. Ocular complications.

12.1 Intracranial Complications

12.1.1 Neurological

12.1.1.1 Brain Edema and Contusion

Brain edema and contusion are direct results of extent and duration of brain retraction as well as certain degree of perilesional edema (e.g., with some meningiomas). Compromising and elimination of draining veins during tumor resection may aggravate the situation.

It can be prevented by performing wider skull base bone removal instead of brain retraction. It can be time consuming (approach) but there will be a better surgical field and less brain retraction-related complications. Minimizing the craniotomy size and "access angle" by using the subcranial approach instead of the standard subfrontal (Derome) or extended subfrontal (Sekhar) ones will provide with a good surgical field with less brain retraction needed.

Perioperative spinal fluid drainage can help (lumbar subarachnoid catheter) in selected cases but we have to take into consideration main drainage veins and collateral drainage channels.

We must choose our approach carefully in order to avoid prolonged and excessive brain retraction. Routine CT scan, usually within the first 24 h, helps in an early diagnosis of edema and contusion, as well as pneumocephalus or hematoma. Significant pneumocephalus is either related to the use of postoperative CSF drain or a suboptimal skull base seal or both.

Edema management includes several actions as the use of preoperative steroids if the edema is noted on the preoperative imaging, modify patient position (Head 30º above heart level, avoid twisting the neck intraoperatively), continuous observation of the patient in an ICU environment, fluid restriction, osmotic agents and hyperventilation will help. Surgical decompression may be necessary in removing bone or performing cerebrospinal fluid (CSF) release from a cistern or from a lumbar drain.

12.1.1.2 Intradural and Extradural Hematoma

This may arise from inadequately surgical bleeding control from afferent or intratumor vessels; it is thus important to have excellent exposure and control of the operative area during surgery so that meticulous hemostasis may be maintained. Preoperative embolization is also of significant help since it reduces intraoperative bleeding providing a clearer working area, specially in some highly vascularized tumors. The use of ultrasonic ablation systems and bipolar-ultrasonic scalpel is also helpful. Surgical reexploration is necessary in most circumstances when significant postoperative blood collection exists. In an excessive bleeding scenario during surgery the presence of unnoticed coagulopathy should be kept in mind.

12.1.1.3 Cranial Nerve Dysfunction

Temporary or permanent dysfunction is a common finding after skull base surgery due to the proximity and involvement of cranial nerves by skull base lesions. Intraoperative monitoring and gentle surgical manipulation will reduce the incidence of this complication. Sensitive nerves are weaker to function loss than motor nerves. There are less chances of recovery when the function is impaired prior to surgery.

Nature of the lesion can act as a predictor of dysfunction because of the extension of dissection required to perform the excision (meningiomas require greater dissection than other tumors like neurilemomas); also the location of the tumor is a key factor jeopardizing different nerves (e.g., petrous apex, cavernous sinus).

Since the III oculomotor nerve innervates several muscles the recovery is less satisfactory when a total paralysis exists after surgery. In contrast, management of IV and VI nerve dysfunction is easier since they innervate single muscles.

Damage to the first V cranial nerve division could lead to serious eye complications and even blindness due to corneal anesthesia, so patients should be instructed in eye care to avoid that.

Facial nerve (VII) injuries cause significant functional and psychological impairment and may arise from resection of lesions at the cerebellopontine angle and temporal bone or extensive dissection and mobilization in a lateral approach. Intraoperative EMG is recommended to minimize the risks of injury and immediate repair should be done if intraoperative nerve section is observed. It may be necessary the use of cable grafts or drilling of the mastoid segment in order to achieve direct coaptation of the ends. "Babysitter" anastomosis between XII cranial nerve ramus and VII should be considered when the proximal facial nerve stump is not available (Figs. 12.1, 12.2, 12.3, 12.4, 12.5, 12.6, 12.7, 12.8 and 12.9).

Clinical Case 1

- 43-year-old female
- Sphenoid meningioma. Orbital dystopia.
- Combined approach (note facial nerve controlled in a vessel loop and orbital reconstruction using bone grafts).
- Immediate facial palsy due to operative traction. Recovery after a few weeks. Post-op CT.

Coexistence of facial and trigeminal nerve damage is a situation of great concern and requires operations on lateral or medial canthal tendons or upper lid (gold weight) to facilitate eye closure and protection.

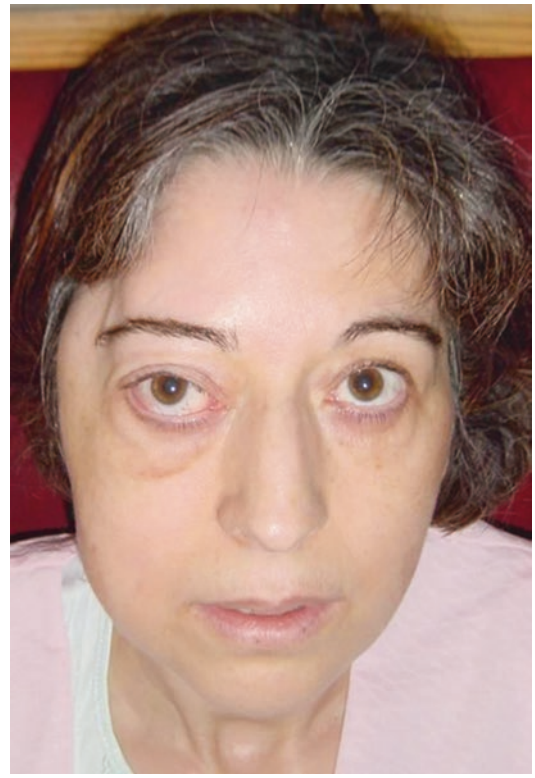


Fig. 12.1 Right orbit dystopia

IX, X, and XII cranial nerves are in danger when the lesion arises on the clivus and foramen magnum area. If nerve dysfunction is present preoperatively, usually there is a compensation phenomenon from the non-affected side. However in patients with no loss of function before the operation the need of performing a tracheostomy or gastrostomy should be warned. These two actions help to avoid aspirations during the postoperative period and lead to an adequate nutrition and can be removed once the patient is recovered. Vocal cord augmentation could be considered if a complete vagus nerve paralysis is expected.

Spinal accessory nerve palsy can lead to disabling chronic shoulder joint arthropathy so that should be repaired if it is intraoperatively transected.

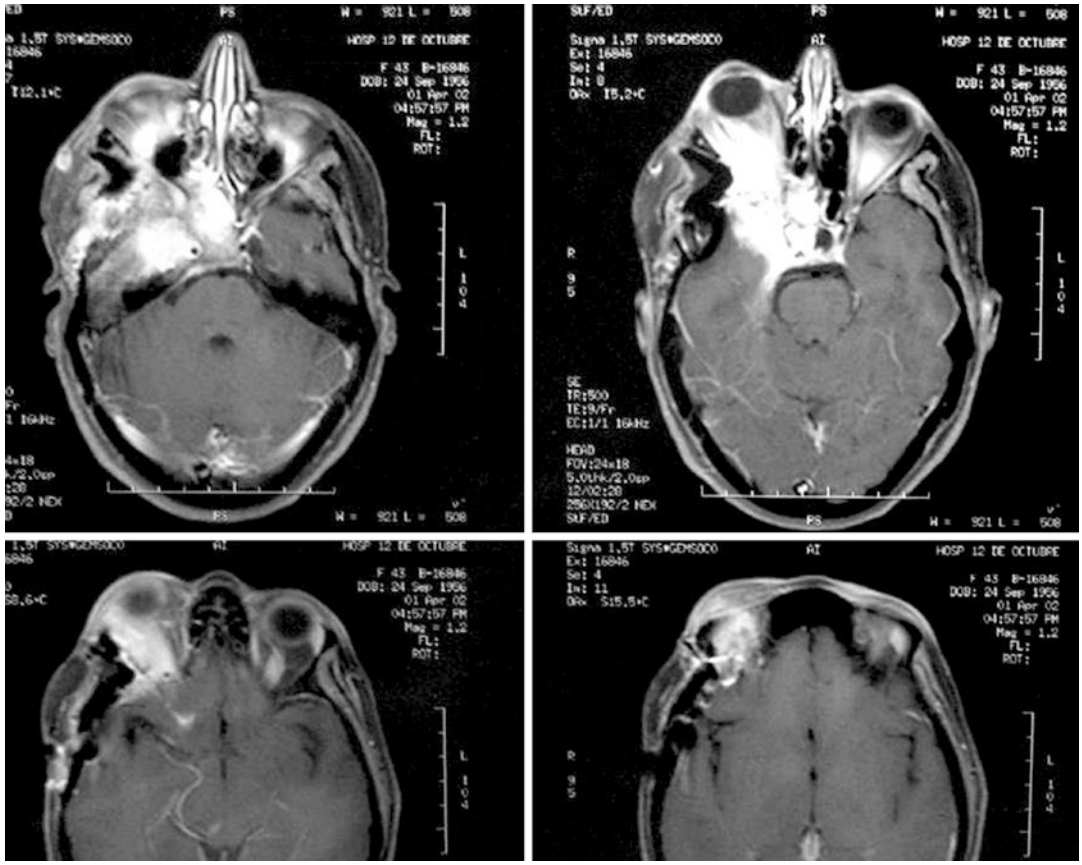


Fig. 12.2 MRI. Sphenoid meningioma

12.1.1.4 Impairment of Brain Function

This may be a consequence of brain retraction and manipulation or damage to the vascular supply of a certain area. Pituitary dysfunction is sometimes observed in tumors dissected from the cavernous sinus or suprasellar areas and pituitary function tests may be necessary.

Hypothalamic dysfunction can be observed in tumors arising from the third ventricle.

12.1.1.5 Intracranial Infection

Intracranial infection is usually associated with CSF leak and inadequate closure of the skull base defect (see 2.1./2.2). Early postoperative infections are frequently bacterial and associated to staphylococcus aureus, pseudomonas, or other nosocomial germs. Pneumococcus is usually responsible for late meningitis.

12.1.2 Vascular

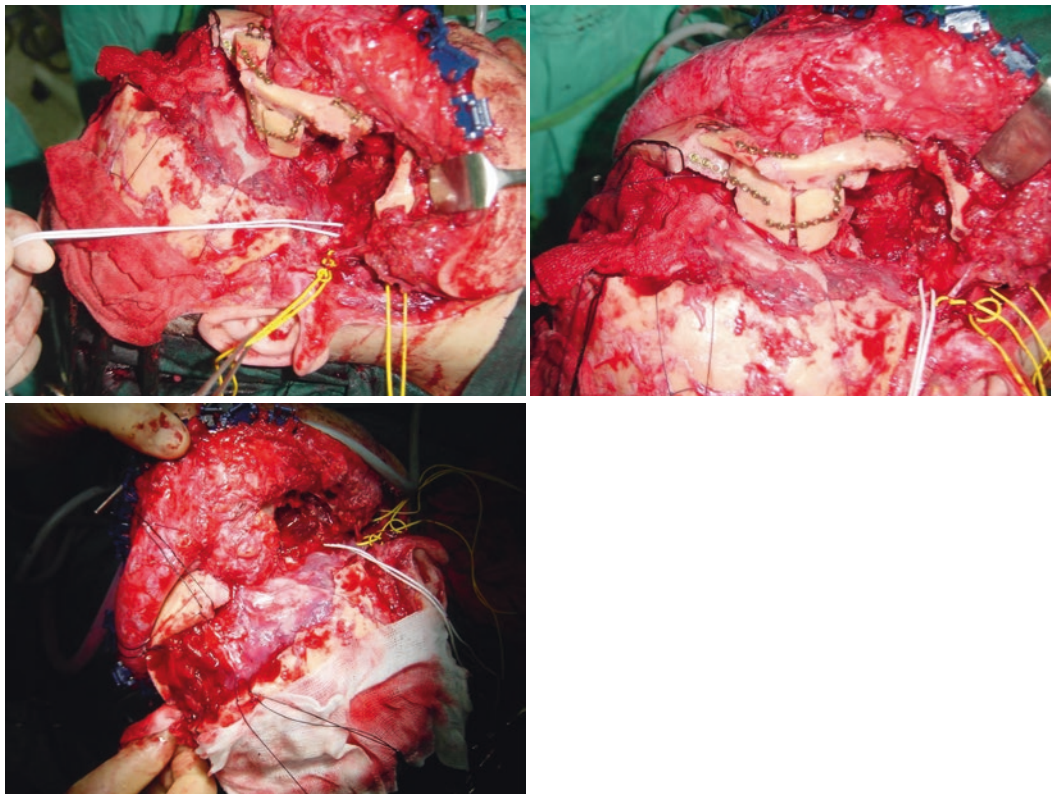
Lesions may compromise the carotid artery or vertebrobasilar system, including their respective branches. Also the approaches used to resect these tumors may jeopardize that vessels. Vessel injury is manifested as intraoperative or postoperative hemorrhage or infarction in the territory of its supply.

Prevention is key in avoiding these complications. Careful evaluation of preoperative

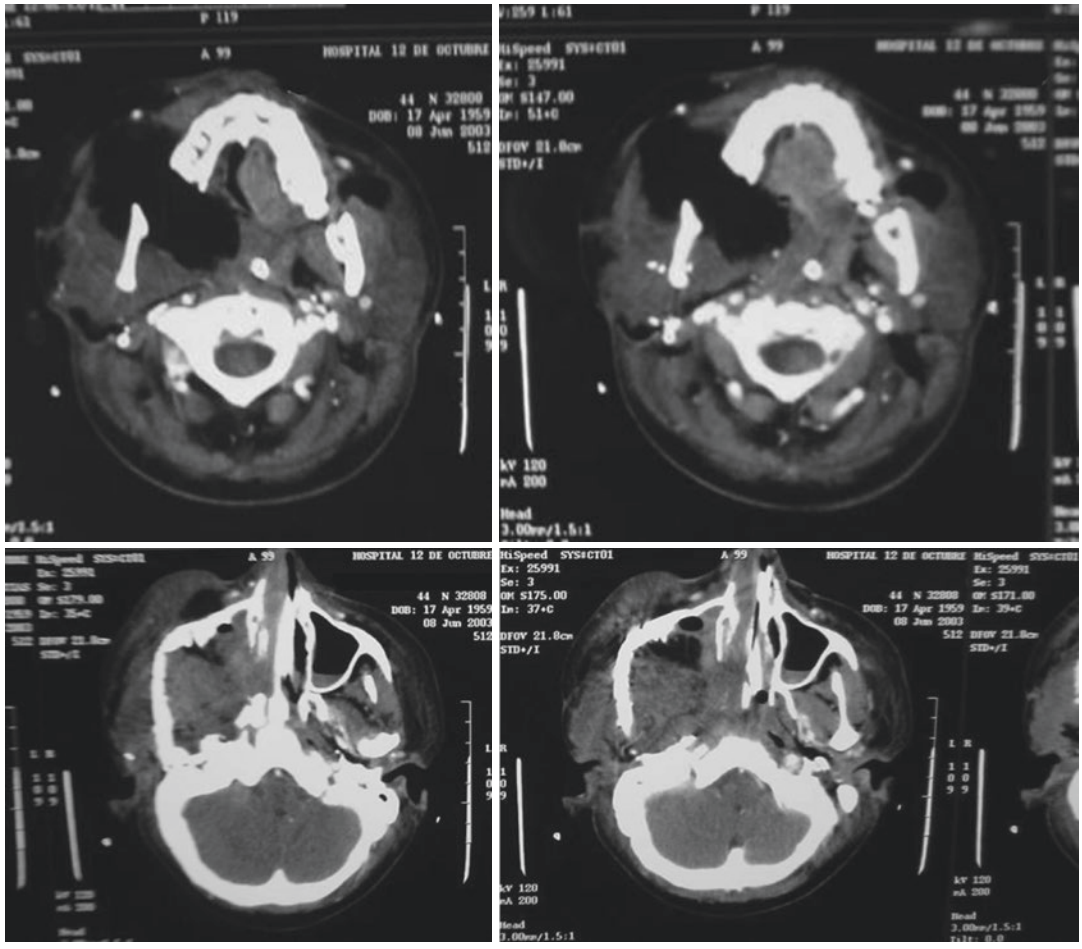
images should be done. MRI helps us in assessing the length and circumference of vessel involvement while angiography reveals vessel narrowing. Previous history of surgery or radiation therapy will indicate the ease or difficulty of vessel dissection. When a potential problem is identified a balloon occlusion test should be performed to assess the contralateral circulation. Spect-CT has been traditionally associated to improve test sensitivity. These tests have been very reliable in relation to the carotid system but similar tests for the vertebrobasilar system are not available.

As a general rule we must have control of both proximal and distal aspects of the vessel. Separation of tumors from the vessel should be carried out sharply to avoid irregular tears, which are much more difficult to repair. When a major vessel is exposed to the paranasal sinuses or the pharynx, it is imperative to reestablish isolation of the vessel from the contaminated areas using vascularized tissue to avoid infection and rupture of the vessel.

Lacerations can be repaired with direct sutures if the tear is clean and produced by sharp dissection. In some cases vein patches or vein grafts are used to repair the vessel wall. If the vessel cannot be repaired direct interposition grafts or revascularization should be carried out. Time of occlusion must be as short as possible and brain



Figs. 12.3–12.5 Combined approach (note facial nerve controlled in a vessel loop and orbital reconstruction using bone grafts)



Figs. 12.3–12.5 (continued)

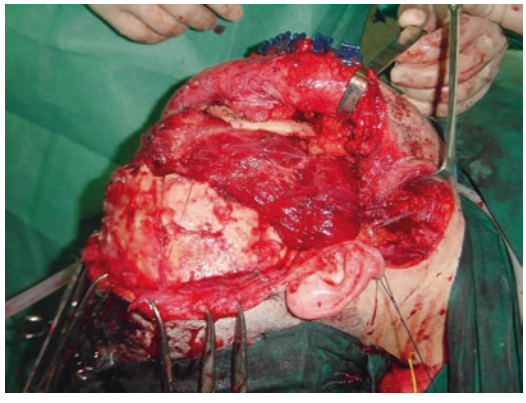


Fig. 12.6 Rectus abdominis flap in place

protection maneuvers such as using etomidate or hypothermia are convenient. Close surveillance must be continued during the postoperative period to detect developing possible new neurological deficit early (Figs. 12.10, 12.11, 12.12 and 12.13).

Clinical Case 2

- Neglected SCC carcinoma skin.
- Favorable carotid occlusion test.
- Tumor resection and free flap recon.
- Postoperative ICTUS.

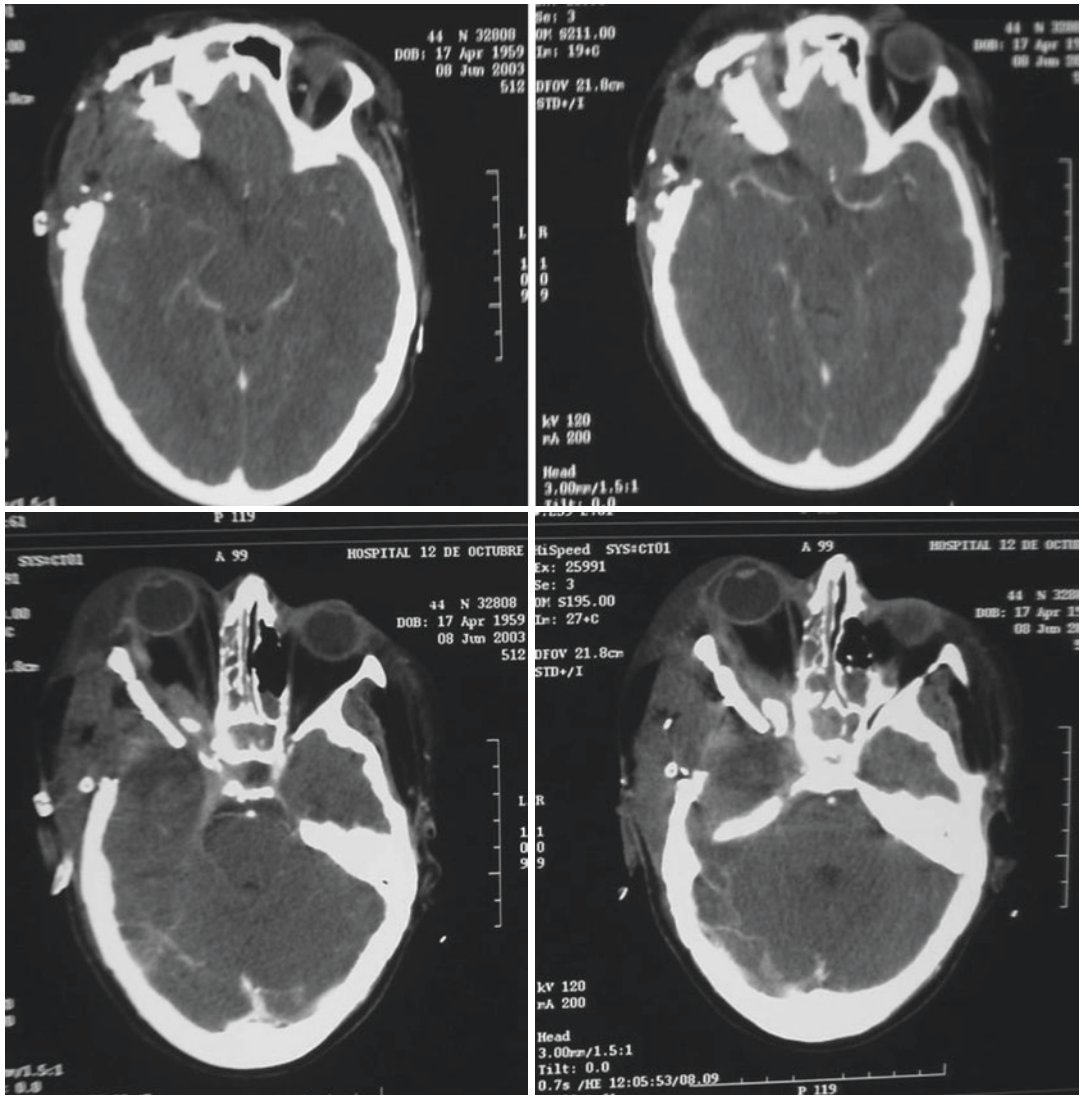


Fig. 12.7 Post-op CT

12.2 Complications Related to Surgical Wound

12.2.1 Cerebrospinal Fluid Leak

Prevention by using an appropriate surgical approach and reconstruction planning is the best way to avoid this complication. Postoperative CSF leak usually arises in the early postoperative period while the patient is hospitalized. We can anticipate a high risk of this complication if a large central anterior skull base defect is produced; involvement of paranasal sinus walls is present in the preoperative imaging, when the CSF basal cisterns are close to the resection area or a large opening of the sinuses is anticipated.

We should not compromise a complete tumor resection fearing a CS leak; on the contrary we

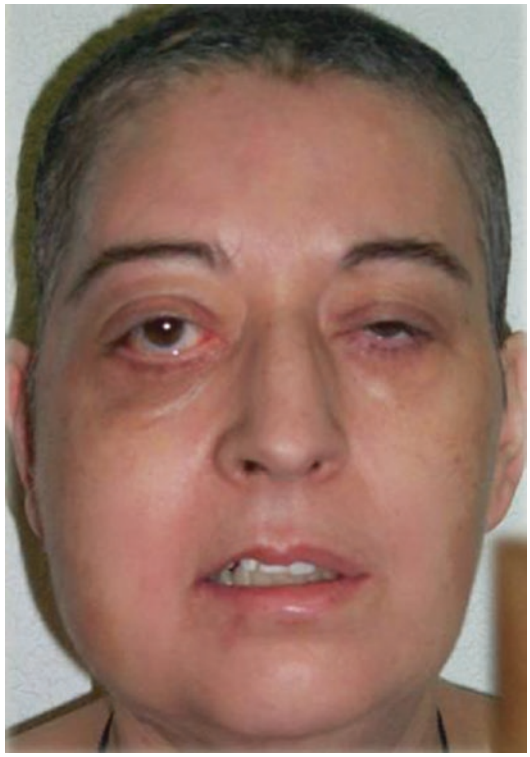


Fig. 12.8 Immediate post-op facial palsy

Fig. 12.9 6 months post-op

should perform an adequate reconstruction to that resections.

Previous history of radiation therapy is a main cause of postoperative leakage and using vascularized tissue (pericranial flap, galeal-pericranial, temporalis muscle, vascularized free flaps, etc.) for the reconstruction is mandatory in those cases, also when postoperative radiation therapy is planned (Fig. 12.14).

Diagnostic methods include direct interrogation during the daily rounds, presence of CSF in the drainage that can be confirmed by β2 transferrin test, Glucostix test (fluorescein), and CT or MRI scans to depict the 3-D imaging of the situation. CT scans with 0.5 mm define the bone defect; MRI will frequently show the CSF leak happening during Valsalva at the hyperintense T2 sequence.

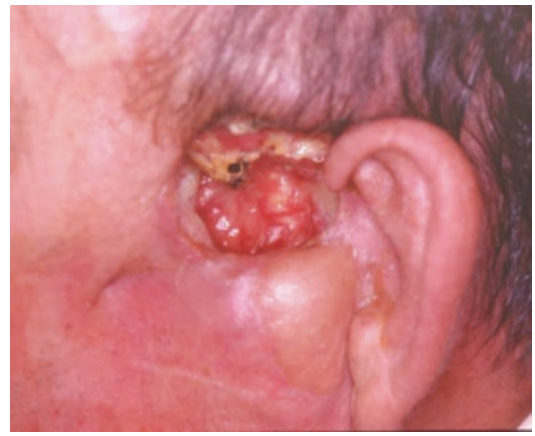


Fig. 12.10 Preauricular SCC

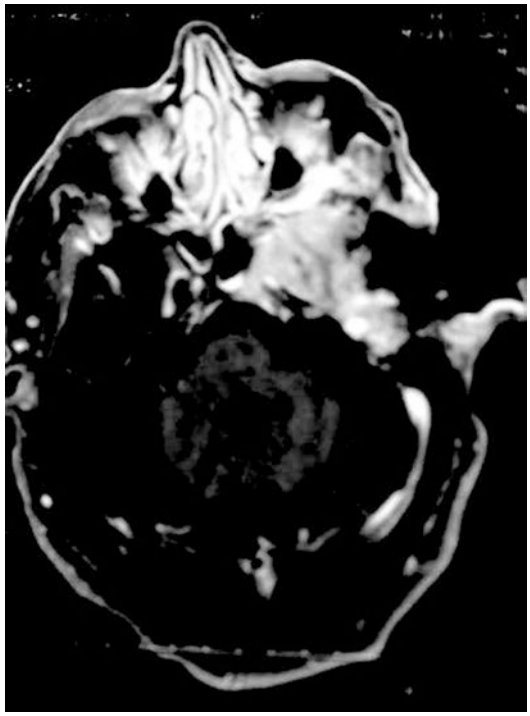


Fig. 12.11 MRI

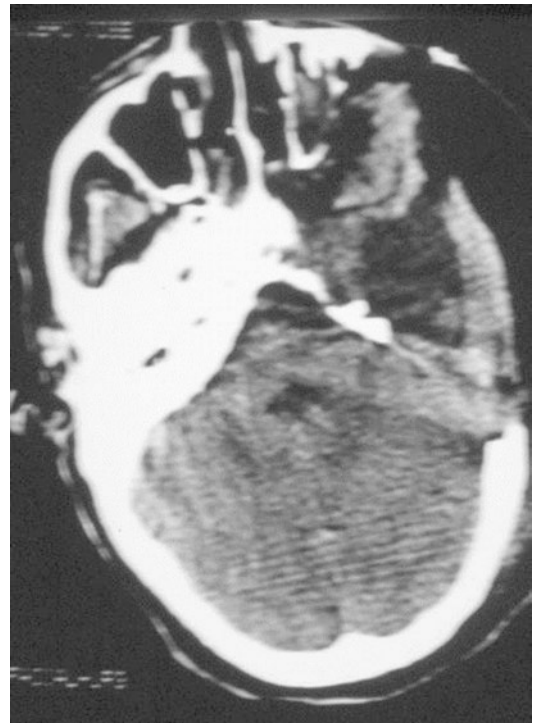


Fig. 12.13 CT ictus

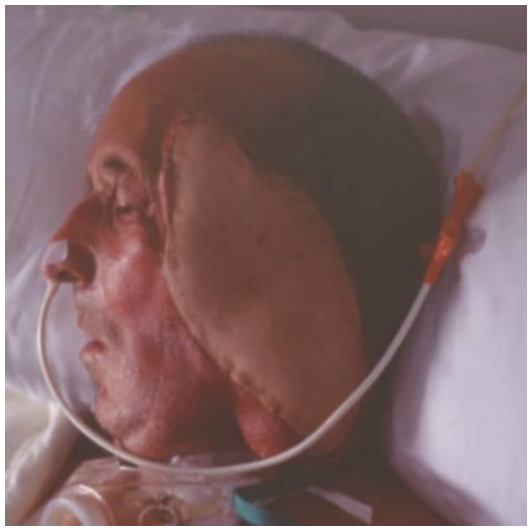


Fig. 12.12 Flap reconstruction

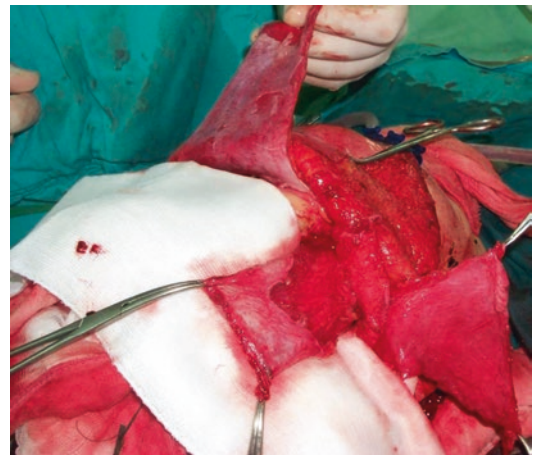


Fig. 12.14 Local flaps. Galea-pericranium flap. Temporoparietal flap. Temporalis muscle flap

Small leak initial management includes bed rest and maybe a CSF lumbar drainage (30–50 mL/h in order to avoid pneumoencephalus). This is continued during 4 or 5 days and then reevaluated. If persistent leakage or a large leak is present or imaging shows a significant defect, surgical exploration is mandatory. Closure is performed using the same surgical approach as for resection, and the "reconstructive ladder" principle applied (Figs. 12.15, 12.16, 12.17, 12.18, 12.19, 12.20, 12.21, 12.22, 12.23, 12.24, 12.25, 12.26, 12.27, 12.28 and 12.29).

Clinical Case 3

- 30-year-old male. Radio-induced leiomyosarcoma
- Underwent resection (maxilla, ethmoid, anterior cranial fossa and dura) and primary reconstruction using a free flap (ALT).
- Inadequate isolation and dead space. Leading to CSF leak and empyema and bone flap loss.
- Reoperation: Debridement, hardware and bone flap withdrawal, upper aerodigestive tract-CNS communication. Omental free flap.
- Reentry a few months later. PSI cranioplasty.

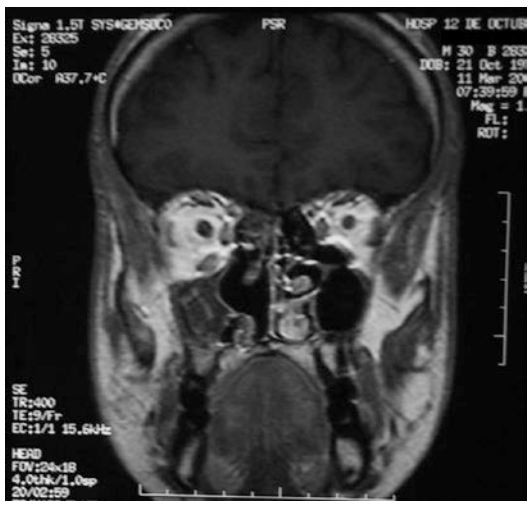


Fig. 12.15 MRI. Radio-induced leiomyosarcoma

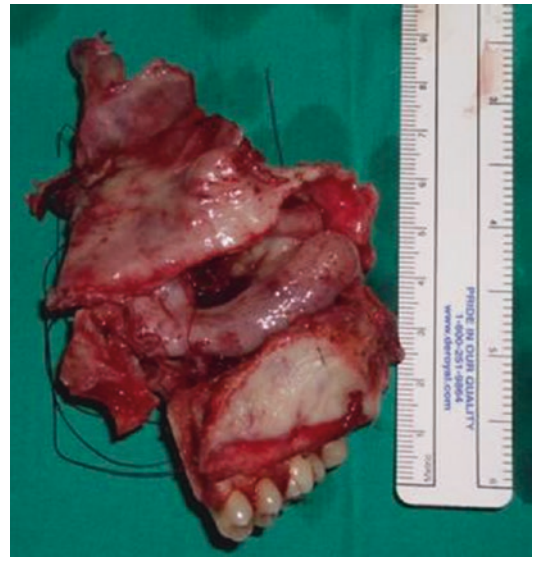
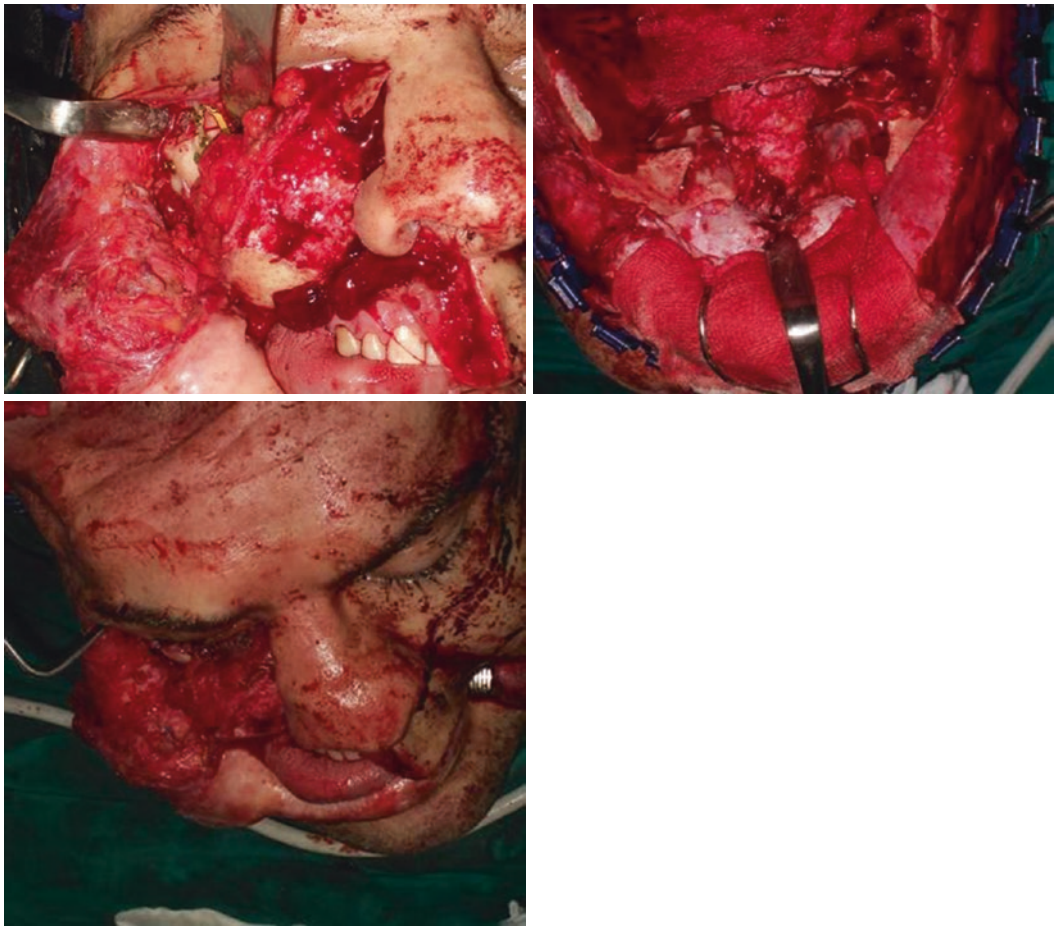


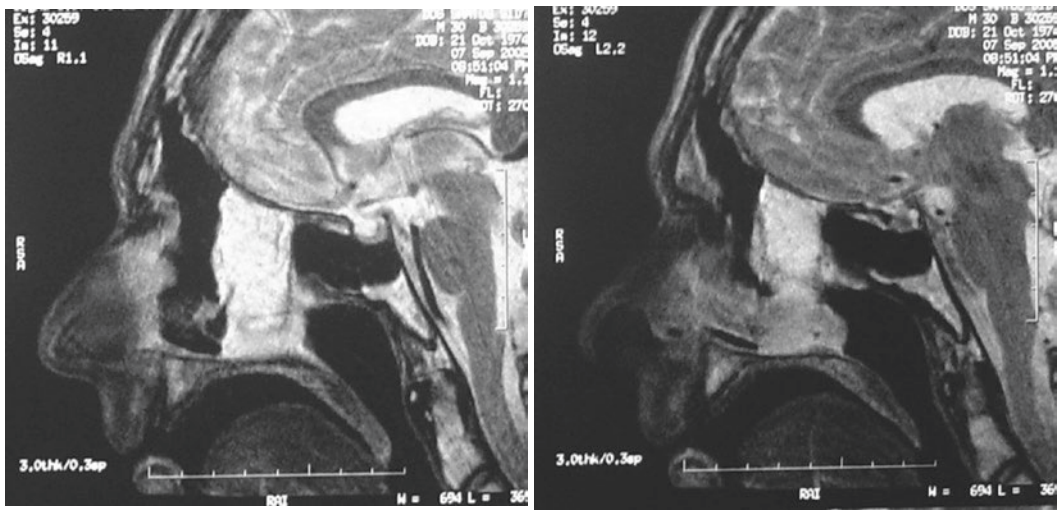
Fig. 12.16 Surgical specimen

12.2.2 Infection

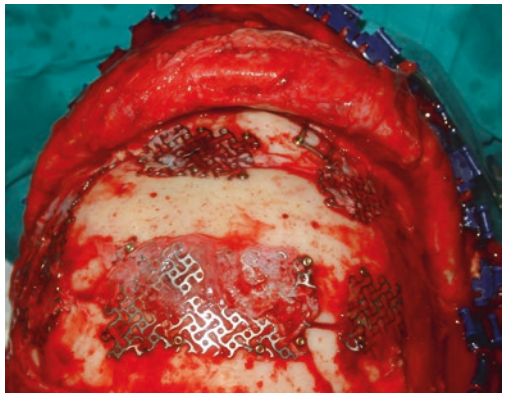
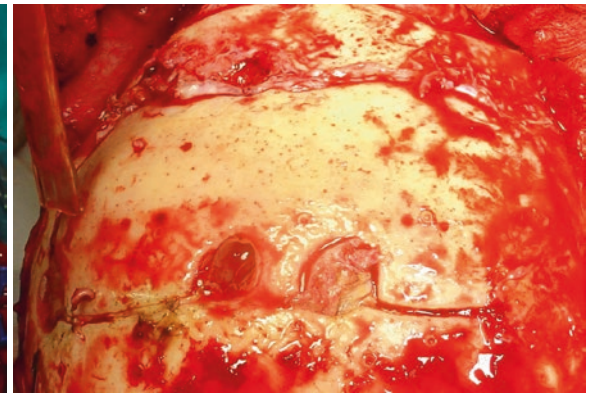
Cranial base surgery frequently leads to broad communications between the central nervous system and the upper aerodigestive tract. Infections may manifest as extradural or intradural abscess or meningitis. Length of operation and proper tissue handling avoiding desiccation are important factors to take into account. Intraoperative antibiotics are used routinely (ceftriaxone or aminoglycoside and vancomycin) and drains should be removed as soon as they are not necessary. Intradural and extradural portions of the intervention may be carried out in different time whenever possible. A key factor is to reestablish proper isolation to SNS from the skin and aerodigestive tract. Therefore skin incisions should be carefully placed to assure good blood supply to the skin flaps and dead space must be avoided. Reconstruction may be performed by using local flaps, regional flaps, or free flaps. In cases of prior surgeries or radiation therapy vascularized tissue reconstruction is mandatory.



Figs. 12.17–12.19 Combined approach and primary reconstruction using free flap (ALT)



Figs. 12.20 and 12.21 MRI. Inadequate anterior cranial fossa isolation and dead space. Leading to CSF leak and empyema and bone flap loss

Figs. 12.22 and 12.23 Reoperation: Debridement, hardware, and bone flap withdrawal

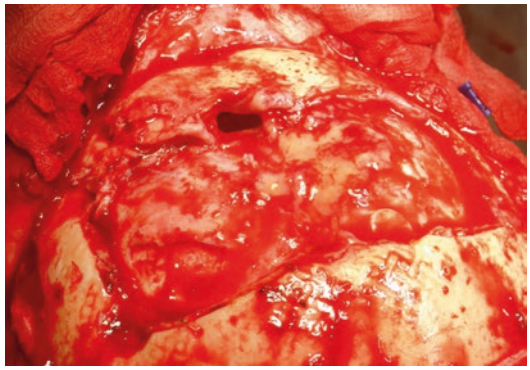


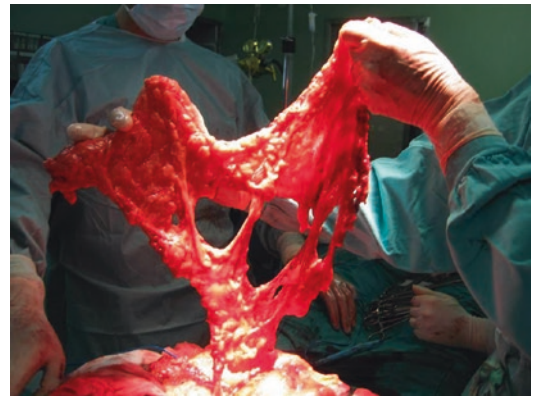
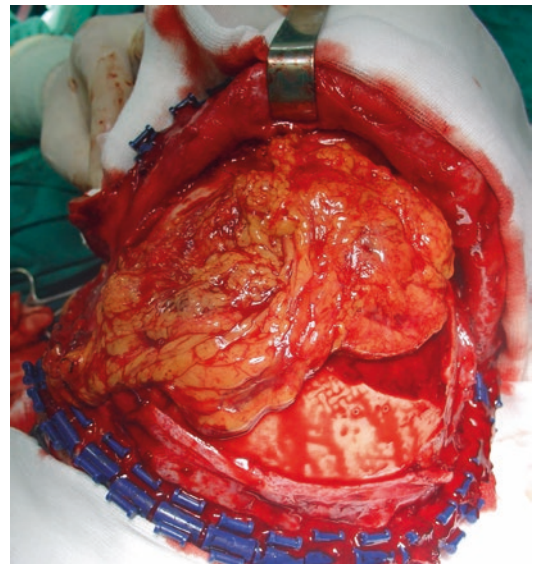
Fig. 12.24 Reoperation. Upper aerodigestive tract-CNS communication

CSF leakage is a common cause of meningitis and must be rapidly addressed.

Treatment includes specific parenteral antibiotics determined by the spinal fluid analysis. CSF leak must be repaired if present. Extradural abscesses need to be explored and thoroughly debrided. Shaving the patient's head with blades is not recommended because it causes microwounds breaking the skin barrier and increasing the risk of wound infection.

12.2.3 Mucocele

When access to the anterior skull base includes transecting the frontal sinus (extended subfrontal or subcranial approaches), sinus function preser-

Figs. 12.25 and 12.26 Omental free flap harvesting and insetting

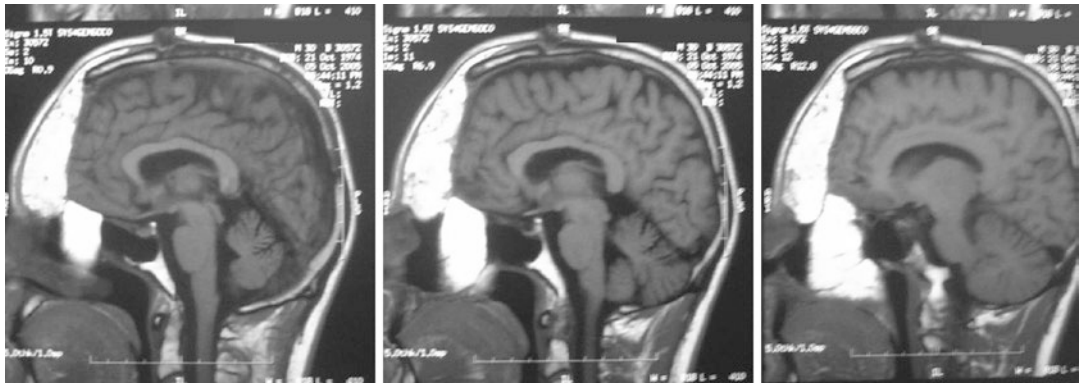
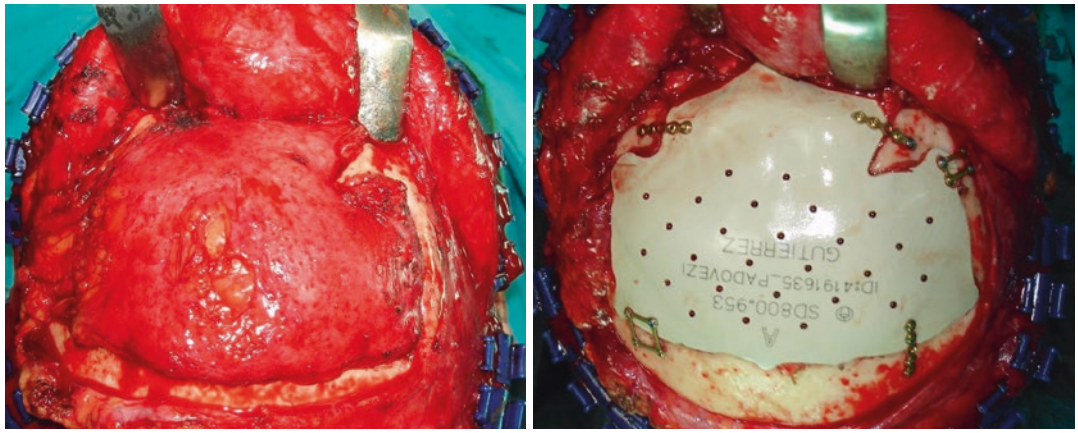


Fig. 12.27 Post-op MRI. Adequate ACF isolation



Figs. 12.28 and 12.29 Reentry a few months later. PSI cranioplasty

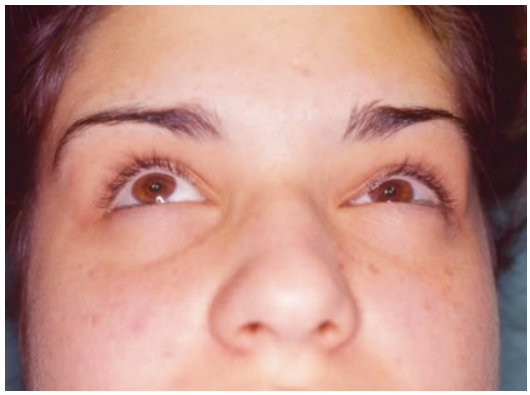


Fig. 12.30 Orbit deformity

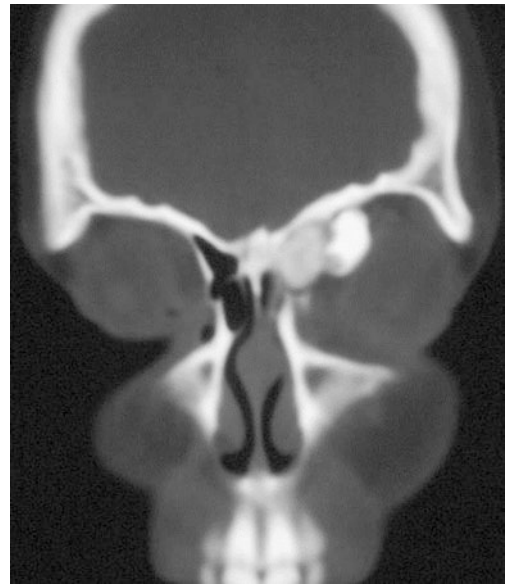
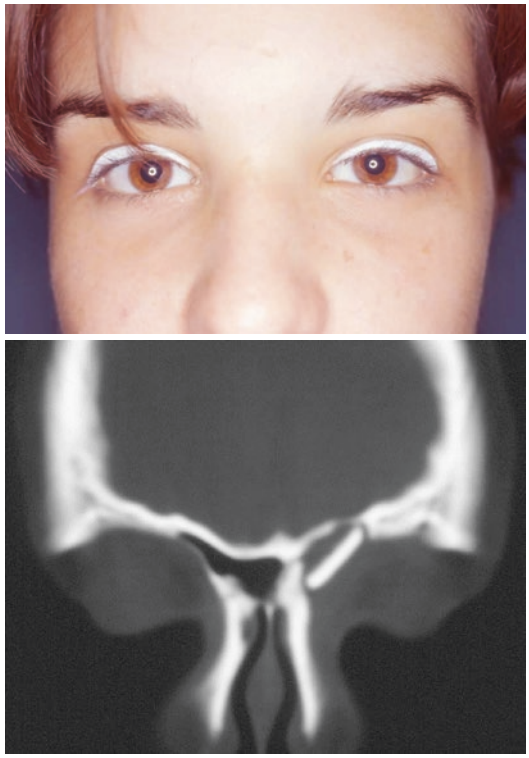


Fig. 12.31 CT. Osteoma affecting nasofrontal outflow duct

vation is usually not an option, and either cranialization or sinus obliteration (depending on the amount of posterior wall left intact) is usually needed. Mucocele and mucopyocele may arise many years after the surgical procedure



Figs. 12.32 and 12.33 Clinical appearance and CT after resection and bone graft

(Figs. 12.30, 12.31, 12.32, 12.33, 12.34, 12.35, 12.36 and 12.37).

Clinical Case 4

- Osteoma/mucocele.
- First intervention.
- Years later mucocele.
- Frontal sinus obliteration.

12.2.4 Osteonecrosis

Extensive craniotomies, specially in association with large dural resections and dead space, sometimes lead to postoperative bone flap necrosis. In irradiated patients, incidence is higher. Minimizing craniotomy size, filling dead space with vascularized tissue, and proper bone fixation with microplates and screws will be key for preventing this complication. When it occurs, secondary cranioplasty will be needed, sometimes in association with a soft tissue free flap to revascularize the area and fill dead space.

12.3 Systemic Complications

Proper assessment of comorbidities and patient preparation is highly relevant as in any major head and neck surgical procedure. Systemic complications may arise as a result of the length of the operation or directly as a result of another complication of the surgery such as cranial nerve or brain compromise. Fluid imbalance and lung atelectasis are less frequent despite the length of intervention. Intermittent compression devices, avoidance of hypovolemia, lower extremity stockings, and subcutaneous heparin have reduced the incidence of deep venous thrombosis (DVT) and subsequent pulmonary embolism.

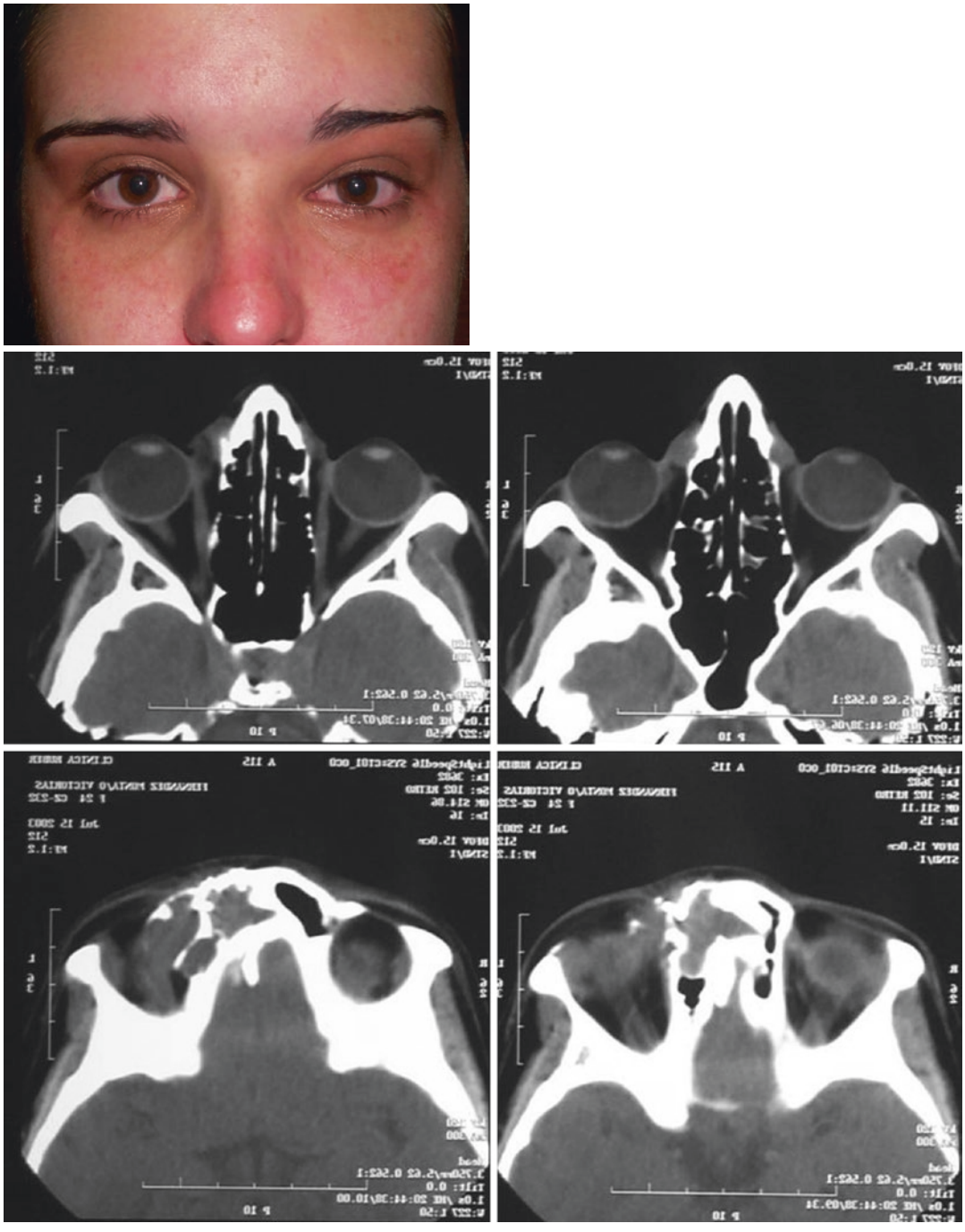
Respiratory complications are frequent in those patients with lower cranial nerve dysfunction and low consciousness level. Temporary tracheostomy may be necessary in these patients with airway protection difficulties.

When swallowing or airway self-protection difficulties are anticipated an adequate nutrition should be established with nasogastric tube or a temporary gastrostomy or jejunostomy.

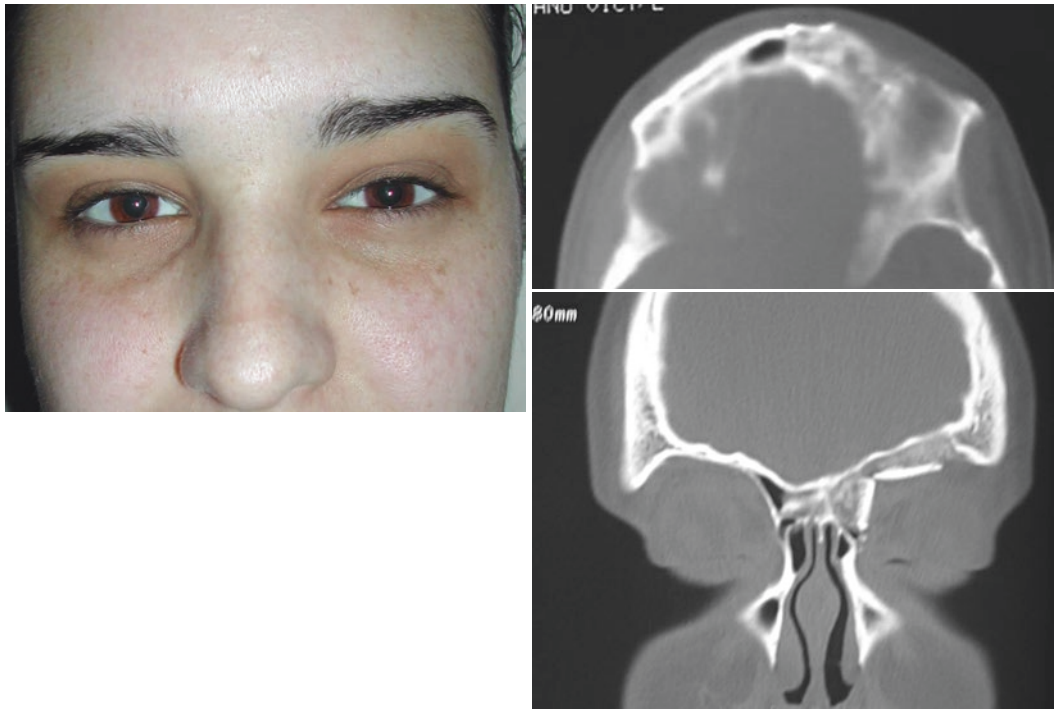
12.4 Ocular Complications

Standard intraoperative corneal protection is mandatory; most patients needing external approaches undergo eyelid stitching. Transient or permanent tarsorrhaphy is sometimes needed. Gold weights and tarsal strip and tarsal sling procedures are considered when significant or permanent deficits in eyelid closure exist.

Postoperative enophthalmos occurs when orbital walls have been resected or mobilized or both and adequate 3-D orbital reconstruction has not been achieved.



Figs. 12.34 and 12.35 Years later the patient developed frontal mucocele. Clinical appearance and CT



Figs. 12.36 and 12.37 Clinical appearance and CT after revision surgery and frontal sinus obliteration

Suggested Reading

Fliss D, Gil Z. Atlas of surgical approaches to paranasal sinuses and the skull base. Berlin: Springer; 2016.

Day JD, Koos WT, Matula C, Lang J. Color atlas of microneurosurgical approaches. Stuttgart, New York: Thieme; 1997.

Donald P. Surgery of the skull base. Philadelphia: Lippincott-Raven; 1998.

Sekhar LN, Fessler RG. Atlas of neurosurgical techniques. New York: Thieme; 2006.

Di Leva A, Lee JM, Cusimano MD. Handbook of skull base surgery. New York: Thieme; 2016.

Deopujari CE, Karmarkar VS. Textbook of operative neurosurgery, vol. 2. 1st ed. New Delhi: B. I. Publication; 2005.

Ducic Y. Complications of skull base surgery. Semin Plast Surg. 2017;31:227–30.

Nibu K. Complications in salvage surgery for nasal and paranasal malignant tumors involving the skull base. J Neurol Surg B. 2018;79:224–8.

Raza S. Complication avoidance in endoscopic skull base surgery. Otolaryngol Clin North Am. 2016;49:227–35.

Solares C. Panorama of reconstruction of skull base defects: from traditional open to endonasal endoscopic approaches, from free grafts to microvascular flaps. Int Arch Otorhinolaryngol. 2014;18:S179–86.

Ganly I, Patel SG, Singh B, Kraus DH, Bridger PG, Cantu G, Cheesman A, De Sa G, Donald P, Fliss DM, Gullane P, Janecka I, Kamata SE, Kowalski LP, Levine PA, Medina Dos Santos LR, Pradhan S, Schramm V, Snyderman C, Wei WI, Shah JP. Craniofacial resection for malignant paranasal sinus tumors: report of an international collaborative study. Head Neck. 2005;27(7):575–84.

Borg A, Kirkman MA, Choi D. Endoscopic endonasal anterior skull base surgery: a systematic review of complications during the past 65 years. World Neurosurg. 2016;95:383–91.

Complications in Maxillofacial Tumor Surgery

13

Bernhard Frerich

Contents

B. Frerich (✉)
Department of Oral and Maxillofacial Surgery, Facial Plastic Surgery, Rostock University Medical Centre, Rostock, Germany
e-mail: bernhard.frerich@med.uni-rostock.de

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_13

13.1 Introduction

Complications in maxillofacial tumor resection may be caused during planning, surgical resection, and postoperative care. Surgical tumor resection may include a lot of severe side effects and functional impairments due to scars and the resection of nerves and muscular tissue, which are inevitably linked to the treatment. Thereby the boundaries between expectable and indispensable morbidity associated with the therapy and true avoidable complications are merging. The following description of adverse events in the context with oral tumor surgery is by no means complete but contains true complications as well as measures for the avoidance of functional impairment due to surgery, the implementation of which is not always considered a complication.

Generally, in malignant diseases, complications and treatment-associated morbidity are considered less absolute. Morbidity and a higher risk of complications are accepted rather by the patients to be cured of potential life-threatening disease. On an individual basis, it may be difficult to balance the benefit of surgical resection against its unavoidable sequelae and the risk of complications in order to gain an advantage in life expectancy.

In the case of tumors, in which surgical treatment is the only curative modality, e.g., sarcoma or malignant melanoma of the oral cavity, the decision for resection may be rather clear and also the assumption of the risk of complications. In squamous cell carcinomas on the other hand, in which alternative non-operative treatment modalities are available, the decision for or against a surgical treatment may be more difficult, and the rate of complications has to be offset against the success rate of surgical treatment, either alone or as part of a multimodality approach. Given the possible equivalence of surgery and radiotherapy for the cure of early squamous cell carcinomas, the justification for surgical treatment results from the improved functional outcome as compared to radiotherapy in these tumors. That determines the benchmark, and any functional outcome significantly deviating from this may be considered an adverse event albeit not a complication.

13.2 General Complications of Tumor Surgery

The incidence of postoperative complications after head and neck surgery is high. The 30-day complication and mortality rates are 20% and 1%, respectively [1–3]. With regard to all head and neck tumor cases, even 3% mortality and a rate of 33% of patients who experience at least one complication were found in large surveys [4]. Also in patients with free flap reconstruction, the rate of major complications is 30% [5] and up to 4% mortality was described in selected high-risk patient groups requiring free flap reconstruction within 30 days postoperatively [6]. In addition to surgical complications, such as hemorrhage, hematoma, wound dehiscence, and flap failure, medical complications such as pneumonia, sepsis, cardiac failure, and delirium are common. In particular, the occurrence of medical complications (specifically sepsis, renal failure, respiratory infections) increases the risk of in-hospital mortality [4]. Complications may critically delay the receipt of adjuvant therapy, and consequently, the advent of complications correlates negatively with survival. Patients experiencing complications have a significantly longer postoperative hospital stay [3] and a poorer prognosis.

13.2.1 Upper Airway Compromise

Respiratory management and safety measures for the protection of the upper airways are of uppermost importance. Airway obstruction in non-tracheotomized patients due to postoperative edema and hematoma is one of the most serious acute complications in oral cancer surgery and particularly relevant after microsurgical tissue transfer into the oral cavity. It can also occur after resection of the frontal parts of the mandibular arch if the suspension of the tongue (e.g. genioglossus muscles) and the other muscles of the floor of mouth are not refixed. Likewise, after reconstruction of the anterior mandible and the floor of mouth with osteosepto- or osteomyocutaneous flaps, a proper refixation of the anterior digastric muscles is hampered, and this adds additional risk

of airway obstruction. The safeguarding of the upper airways by temporary tracheostomy therefore is strongly recommended in patients who receive reconstruction with microvascular free flaps in the lower oral cavity [7]. The tendency of the flap to swell must also be kept in mind during maxillary reconstructions, where the risk is occasionally underestimated. In principle, a temporary tracheotomy is safer and the risks and complications of a correctly performed tracheotomy are low or even essentially event-free [8] and sometimes overemphasized. Postoperative pneumonia is usually not a result of tracheostomy, but of the duration of surgery, blood loss of patients with corresponding comorbidities, especially COPD. In fact, the risk of pneumonia is higher in patients who are not tracheotomized [9]. Also, decannulation failure cannot be attributed to the tracheostomy as it is mainly caused by dysphagia and aspiration due to resection or reconstruction and not by the tracheostomy. Rather, these patients require tracheostomy to avoid aspiration pneumonia. There are some rules which have to be followed in the ward in order to avoid complications like cannula obstruction and displacement. That includes proper training of the staff in the care and the management of tracheotomized patients. In these patients, we change the tracheal cannula at least every 2nd day from the third postoperative day. Tumor patients often suffer from chronic obstructive pulmonary diseases with corresponding bronchial secretion. In case of insufficient care, there is a risk that drying secretions lead to obstruction of the cannula. This can be prevented by regularly changing the cannula, which promotes expectorations, also contributing to pneumonia prophylaxis. For the same reason, the puncture tracheotomies were abandoned. Although the long-term esthetic and functional results are excellent [10], frequent and even emergency cannula changes are not possible, so that they are not suitable for the special clientele of head and neck tumor patients.

In smaller tumors, in which defects are reconstructed by local measures, tracheostomy is not necessary as a general rule.

In any case of subjective dyspnea and in any case of objective signs of dyspnea, a rapid tracheotomy must be considered, since the time from the first dyspnea signs to the urgent need of resuscitation may be very short.

13.2.2 Postoperative Pneumonia

Pneumonia is, as mentioned above, the most common medical complication in the resection of oral squamous cell carcinoma and its occurrence is associated with significantly increased mortality [5]. Patient-related risk factors for the development of pneumonia are age, ASA classification, and pre-existing COPD.

The risk of postoperative pneumonia increases in various investigations with longer duration of surgery, intraoperative blood loss, longer-term tracheostomy, and a longer stay in intensive care [11–13]. In particular, delayed mobilization (>4 days) is a risk factor for the occurrence of pneumonia [11]. Therefore, the operation must be planned in such a way that, depending on the individual risk profile of the patient, mobilization is achieved as quickly as possible and longer-term ventilation or intensive care stays are avoided. This, in turn, makes elective tracheostomy necessary if there is a risk of airway obstruction in order to avoid prolonged naso- or orotracheal intubation. Prolonged antibiosis over 5 days had no influence on the incidence of postoperative pneumonia in a prospective study in oral cancer patients in comparison with single-shot prophylaxis but on the rate of surgical site infections [14]. The single-shot antibiotic regimen in this study consisted of the administration of a broad-spectrum antibiotic from 30 min before surgery, which was repeated intraoperatively every 3 h. Additionally intensified antiseptic oral health care is recommended to avoid pulmonary infections.

13.2.3 Intra- and Postoperative Bleeding

Blood loss during tumor surgery is a significant risk factor for developing postoperative complications [15]. With this in mind, a blood-saving

surgery reduces the risk of perioperative complications, including postoperative pneumonia [13], the need for flap revision surgery including flap loss, and further medical and surgical complications. Besides the diligence and the skill of the surgeon, this depends also on some device-related requirements, for example, the use of bipolar electrocautery scissors or piezosurgery devices.

Postoperative bleeding especially in the tongue may cause postoperative tongue edema and obstruction of the oral cavity and the pharyngeal respiratory passage by swelling of the tongue body. Bleeding into the tongue body after the closure of the sutures requires reopening of the wound and revision of the bleeding areas. If necessary, a tracheotomy may be needed at the latest, all interventions with a higher risk of swelling should better be planned with a primary tracheotomy.

In addition, it is self-evident that hematomas and postoperative bleeding must be removed or stopped immediately. The risk of postoperative bleeding is, of course, also associated with anticoagulation or thrombocyte aggregation inhibition after microsurgical flap surgery. However, with careful hemostasis, even full heparinization in combination with thrombocyte aggregation inhibition is largely safe, if it is started 2–3 h after the end of the operation. It is important that the wound surfaces are dry during wound closure and are placed against each other via a Redon suction drainage.

If excessive blood flow into the Redon occurs immediately after surgery, e.g., after lengthy procedures with major blood loss, especially in connection with microsurgical bone grafts, or if this is feared, it may be helpful to open the Redon intermittently. The Redon is opened and closed every 30 min or only once per hour for 5 min. At the same time, the neck and chin area are gently compressed by not too tight but evenly soft head bandage. It should still be possible to place the finger lightly between the bandage and the neck. After 24 h, when coagulation has normalized, sometimes also a problem with low body temperature, the Redon suction drainage can be fully opened. It is not necessary to say that this procedure is advisable only for intubated or tracheotomized patients.

13.2.4 Tissue Necrosis and Wound Healing Disturbance

Wound infection is a common complication after oral cancer surgery and may result in significant functional morbidity, poor cosmetic results, and prolonged hospitalization [16]. The sources of wound healing disorders are manifold. Most of the wound infections are flap related and secondary to insufficient reconstructions [16]. Many tumor patients have limited oral hygiene, which promotes healing disorders and wound infections as well as reduced general condition or metabolic disorders. Long operation times and blood loss also promote wound healing disorders [17]. In addition, recumbent, sedated or ventilated patients tend to suffer from wound healing disorders due to the accumulation of secretion in the back of the oral cavity. Insofar a careful surgical technique and a straightforward surgery, resulting in early mobilization, contribute considerably to undisturbed wound healing. Excessive use of electrosurgery can also be an important reason for wound healing disturbance. In contrast to occasional reports, early secondary suturing of dehiscent wounds is important, and therefore, regular wound monitoring and oral hygiene, e.g., with chlorhexidine, are necessary. In a prospectively controlled study, it was shown that the use of oral health care could significantly reduce the rate of wound infections after tumor surgery [17]. The significance of perioperative antibiotic prophylaxis remains controversial. In a prospective trial with oral cancer patients, a prolonged antibiosis (5 days) contributed to a reduction in surgical site infections, as compared to single-shot prophylaxis (30 min pre-op and every 3 h during operation, but discontinued directly after operation), whereas in a retrospective survey of over 900 patients covering all maxillofacial diagnoses, there was no difference between single-shot prophylaxis and prolonged antibiosis, also not in the segment of tumor patients [18]. However, given

the prospective data, there is some evidence of an advantage of a prolonged antibiotic prophylaxis of up to 5 days compared to a strictly perioperative antibiotic prophylaxis at least in the larger tumor operations. Predilection sites for wound healing disorders are the transition zone from a resection defect to toothed segments of the mandible or reconstructions of the soft palate. In these regions, two-layered suturing and use of (vertical) mattress sutures are helpful. In particular, at the mandibular stump in resection defects, sutures may be fixed to adjacent teeth and should not be removed too early(12–14 days minimum).

The reason for wound healing disorders in the soft palate mostly is an insufficient suturing or tightness against the nasal/epipharyngeal space. For specific aspects in reconstruction of velar defects, see chapter "Resection of the soft palate". In case of dehiscence, all wound edges have to be excised, and the secondary suturing has to take into account covering of the nasal side as well as of the oral side of the defect according to the rules for velar reconstruction, which are described there.

Tissue necrosis due to compromised vascularization after resection affects in particular the tongue. The tissue of the tongue tends to necrosis, when the muscle tissue is pedicled too scarcely and vascularization is hampered. Intraoperative livid discoloration of the tongue mucosa is a sign of tongue sections that do not recover postoperatively. If an attempt is nevertheless made to preserve them, early debridement of postoperative necrosis is mandatory (specific further aspects see chapter "Tongue and floor of the mouth resection").

The tongue tissue and its blood circulation are difficult to assess for the first days and sometimes appear misleadingly intact by superficial inspection (Fig. 13.1a). The mucosa fades but in reality is no longer supplied with blood (Fig. 13.1b). The non-vascularization of the tongue can be assessed with a cannula (e.g., 16G). Only a few days later, dehiscence and tissue breakdown occur.

If the sutures are too dense and too tight, they can also lead to wound edge necrosis and dehiscence with pronounced tongue edema.

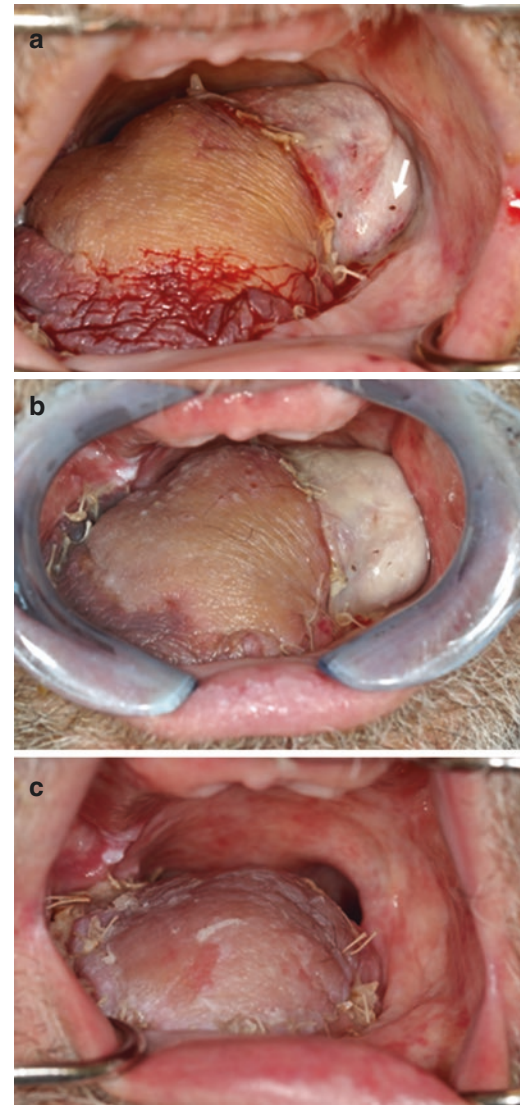


Fig. 13.1 Case study 1, partial tongue necrosis. (**a**) Situation after partial resection of the tongue in a recurrence of a tongue carcinoma, pre-irradiated situation. 4. postoperative day. The parts of the tongue which appear to have been congested immediately after the operation have become paler, only a little reddish fluid can be seen on the puncture (arrow), in contrast to the puncture site at the frontal border of the radial forearm flap. (**b**) Eighth postoperative day, tongue tissue strongly pale, necrosis clearly visible, puncture sites are empty. (**c**) Situation after debridement and secondary suturing

Wound healing disorders on the floor of the mouth are mostly caused by graft complications. The main risk is a migration of the inflammation

into the neck area, especially after neck dissection, where it can lead to further complications (e.g., abscess in the neck loges, cervical skin necrosis, oral floor fistula, see case study 3). If at all possible, it should therefore be prevented that secretion and saliva can sink into the deeper areas of the floor of the mouth. An early secondary closure should therefore be performed. If teeth are present, it makes sense to make a dressing plate that covers and seals the defect. We manufacture dressing plates with a shield with rounded edges, which cover the area of impaired wound healing. Some space is blocked out for a fat-based ointment tamponade, which is changed once or twice a day. If this is carried out consistently, the area of the disturbed wound healing is reliably sealed so that granulation from the depths leads to healing (Fig. 13.2a, b). Case study 2 shows the procedure.

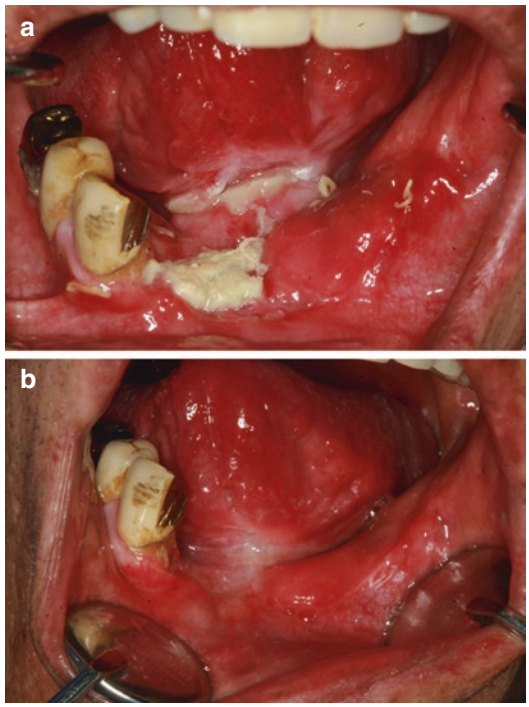


Fig. 13.2 (**a**) Case study 2: FAMM flap with necrotic tip after partial resection of the floor of mouth together with marginal mandibulotomy. (**b**) Situation after 4-week treatment with a dressing splint

13.2.4.1 Case Study 1: Partial Tongue Necrosis

A 62-year-old patient after resection of a recurrence of a tongue carcinoma that had originally been treated surgically and adjuvant radiation was reported. The recurrent tumor was resected, and the defect was covered with a radial forearm flap. On the first postoperative day, a revision of the radial flap was necessary due to venous congestion. The parts of the remaining tongue that had initially been congested showed a slight pale on the fourth postoperative day, with minimal bleeding visible on puncture (Fig. 13.1a). Attempts at preservation were made with full heparinization and thrombocyte aggregation inhibitors. Nevertheless, progressive necrosis, which is clinically obvious 4 days later (Fig. 13.1b). In the present case, early full heparinization with the most intensive (probably double) thrombocyte aggregation inhibition possibly would have shown better success. In the current situation (Fig. 13.1b), the tissue must be removed; functionally, this is devastating in this situation, however, healed further uneventful (Fig. 13.1c).

13.2.4.2 Case Study 2: Necrosis of a FAMM-Flap in the Floor of the Mouth

A 57-year-old patient with T1 carcinoma on the lingual side of the left alveolar process was reported. Partial resection of the floor of the mouth was performed with lingual marginal mandibulotomy. The defect and especially the alveolar ridge area were covered with a FAMM flap from the left side. This became necrotic at the tip, which was particularly problematic with regard to the underlying mandibular rim. A dressing plate was made, which was changed twice a day with ointment dressings by the patient. The necrotic flap tip remained clean, and granulation and secondary covering of the basal rim occurred underneath (Fig. 13.2a), so that no further intervention was necessary. Function, swallowing, and speaking were flawless even afterward (Fig. 13.2b).

13.2.5 Extraoral Wound Healing Disorder

Necrosis of the skin of the neck can occur if the wound edges dry during a prolonged surgery, preferably in patients with pre-existing vascular disorders. This complication is also more frequent in triangular flap formation compared to linear incisions (e.g., McFee). Further risks are thin skin preparation, missing of underlying platysma (e.g., in the midline). The following problems are associated with this wound healing disorder: Delay of adjuvant radio- or radiochemotherapy with the risk of prognosis deterioration, risk of bleeding, and persistent pharyngeal fistula. If a tight closure exists or can be created after intraoral closure, the defect can be conditioned externally with a VAC (vacuum-assisted closure) dressing (Fig. 13.3) and subsequently treated with a full or split skin graft or even sutured secondarily (Fig. 13.3e, f).

13.2.5.1 Case Study 3: Infection and Wound Healing Disorders Intra- and Extraoral

A patient 59 years of age with a T4 tumor of the floor of the mouth infiltrating the tongue was reported (Fig. 13.3a). From a functional point of view, the reconstruction was planned and performed with radial forearm flap, neurovascular pedicled infrahyoidal muscle flap bilaterally, and a long-distance marginal mandibulotomy. Postoperatively there was a need for revision of the radial forearm flap due to venous congestion. Later, circular partial loss at the periphery of the radial flap was found along with the formation of fistula of the floor of mouth to the submental skin. The cervical skin turned partially necrotic.

To treat this complication, the wound edges of the radial flap were excised and it was possible to reattach them tightly into the floor of the mouth (Fig. 13.3b). The necrotic cervical skin was excised, partially covered with skin split-thickness graft and the submental cavity was sealed with vacuum drainage and in this way reduced in size (Fig. 13.3c, d). The remaining defect was finally covered with a split-thickness graft.

13.2.6 Non-In-Sano Resection

Generally, the non-in-sano resection is considered a risk rather than a complication. However, there are specific precautions which have to be respected in order to avoid incomplete resection and unnecessary radicality. The first is to plan the resection thoroughly. That pertains to the involvement of the bone (mandible), the extent of tongue resection, and the involvement of parapharyngeal space, neighborhood to functional or vital relevant structures (vascular bundle, nerves). The second is to perform resection under continuous hemostasis, in order to be able to see, to feel, and to palpate tumor borders. That perhaps is most important. The third is to keep the overview by an appropriate surgical approach. Last but not least, the no-touch-rule must be followed, i.e., that no part of the tumor gets in touch with the healthy tissue in order to avoid contact with metastasis. Sometimes, a difficult step is the pull-through into the neck. It is easier in case of diverging vertical osteotomy lines. During the pull-through, the tumor has to be pushed dorsally and then inferiorly, so that the tumor can be delivered without touching the neck. We cover the tumor intraorally with compresses to prevent an unintentional touch to healthy tissue during pull-through.

13.3 Complications of Specific Surgical Maneuvers

13.3.1 Mandibular Resection

Mandibular resection may be a stand-alone surgical procedure for benign and malignant neoplasm of the bone as well as a part of the resection of tumors originating from the oral mucosa. In latter, mandibular resection is intended to achieve

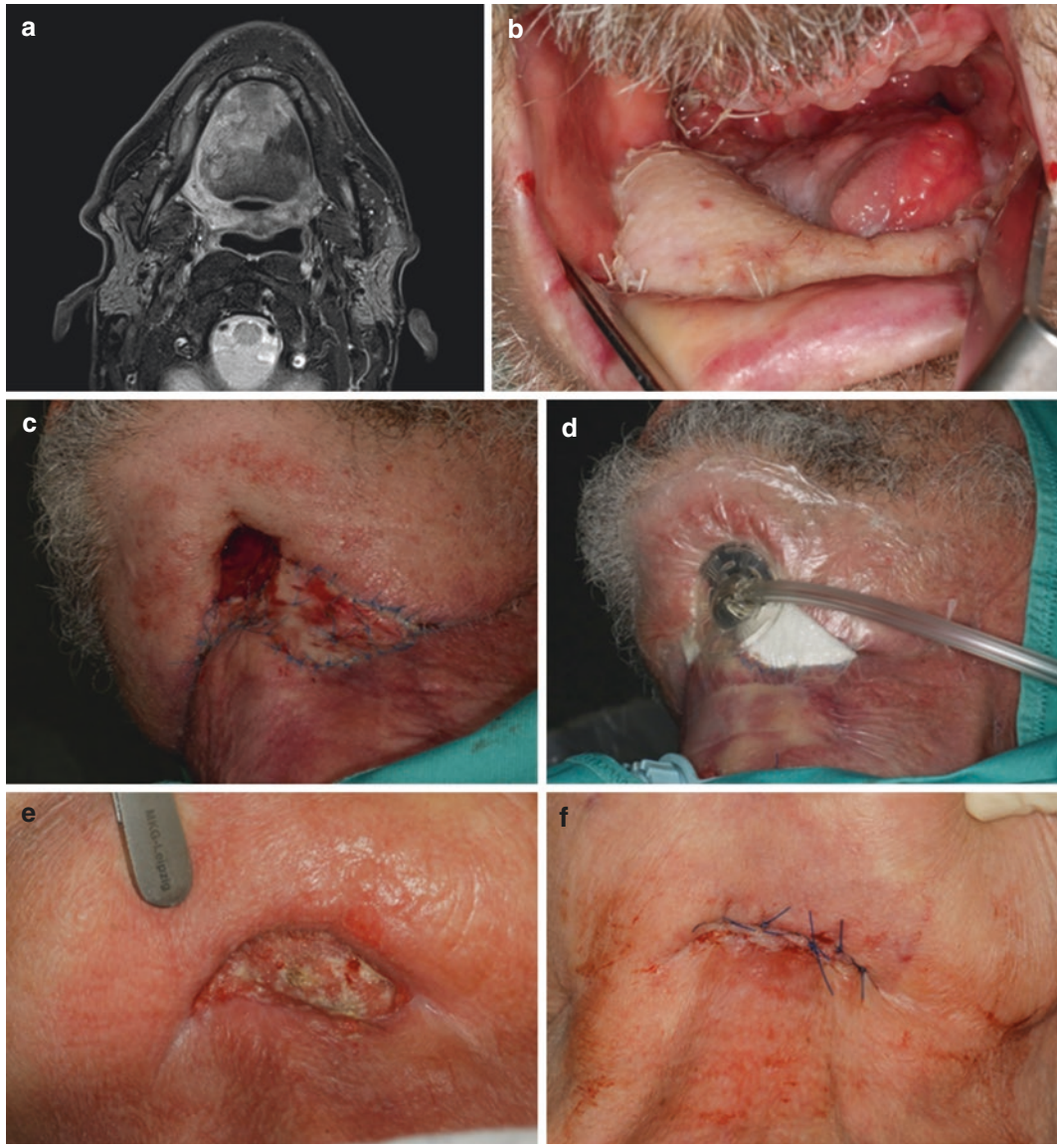


Fig. 13.3 Case study 3, infection and wound healing disorders intra- and extraoral. (**a**) MRI of a carcinoma of the floor of mouth with infiltration of the root of the tongue. The tumor was resected together with long-distant marginal mandibular osteotomy. (**b**) Situation after flap revision and tight re-attachment of the marginal necrotic and detached flap. (**c**) At the same time, a cervical skin necrosis developed, which was removed and partially covered with split thickness skin graft. (**d**) The large cavity between the remaining mandibular rim, radial forearm flap, and submental skin was reduced by vacuum drainage. (**e**) Reduction and granulation of the defect after vacuum drainage treatment and (**f**) closure by suture in another patient

adequate safety margins in tumors originating from the floor of the mouth or tongue, neighboring or involving the mandible. In both scenarios, partial resections, preserving the continuity of the mandible, as well as segmental resections are in use, which all display a specific range of complications.

13.3.2 Marginal Rim Resections with Preservation of Mandibular Continuity

The various types of rim resection differ in the tilt of the osteotomy plane. The classical rim resection with an almost horizontal osteotomy plane is

mainly performed in case of benign osseous lesions of the alveolar process, which need a safety margin against the healthy bone, or small gingival carcinomas, which are more common in elder patients. For tumors of the floor of the mouth adjacent to the mandible but not penetrating the cortical plane, the marginal resection with oblique or sagittal osteotomy plane is used. For tumors of the buccal mucosa, also external marginal resections are possible.

The typical complication of marginal or rim resection is an inadvertent fracture. A fracture of the residual mandible may occur during the osteotomy as well as in the postoperative course. Intraoperative fractures are a result of an underdesigned residual mandibular rim or insufficient preparation of the cortical osteotomies. Hence, the osteotomy is forced with too much effort.

The classical surgical technique of marginal osteotomy formerly consisted of marking the resection borders by a continuous series of cortical drillings with the rose drill, which are then connected with a Lindemann bur or a compass saw. This isolates the affected piece of bone and allows it to be released using chisels indirect impact in order to cut through or disrupt bone bridges that cannot be reached directly. The osteotomies should be arranged diverging from the direction of removal.

The use of an oscillating saw alone is more efficient but not necessarily more precise. The use of a flexible blade is recommended to saw curved lines. Generally, the risk of fractures is higher in sagittal osteotomies crossing the midline, because the arch is more narrow, complicating the mobilization of the segment, and the lingual cortical plate is of particular thickness in the midline region. In appropriate situations, it is advisable to cut the mandible holding the tip of the saw blade toward the basal-lingual side which is controlled by palpation. Elsewise a lingual basal corticotomy may be performed also with the saw (or a burr) from the neck. When all osteotomy lines are properly connected (especially the lingual basal with the vertical osteotomy lines) and when all osteotomy lines diverge lingually, the split can be performed safely with an osteotome.

The use of piezoosteotomy allows today a much more targeted osteotomy and safer protection of the inferior alveolar nerve [19]. It works in a very clean and bloodless way. The time-consuming procedure is outweighed by a higher precision and predictability of the osteotomy. In particular, the osteotomy from the basal-lingual side is much more precise than with the saw, which is more difficult to keep in the line and tends to slide away. With the use of piezoosteotomy, very long marginal resections spanning both sides are safely possible.

13.3.2.1 Avoidance of Inadvertent Fracture

Observing some rules helps to avoid fracture:

- the osteotomy lines should be curved and edges should be avoided for larger osteotomies [20].
- In general, a minimum of 1 cm basal bone is believed to be sufficient for the stability of the remaining mandibular arch [20].
- Preparation of all cortical osteotomies over the complete circumference of the planned rim. This includes the lingual aspect and especially the transitions between vertical osteotomies with the horizontal cortical osteotomies at the edges of the rim
- Strict avoidance of undercuts, osteotomies have to diverge in direction of the mobilization (mostly lingual)
- that is especially true when a sagittal resection is planned from the lingual aspect of the anterior mandibular arch, whereas in edentulous patients with sufficient height of the mandible, an oblique marginal osteotomy might be easy and without risk.

In case of lower bone heights and in any case in which insufficient stability of the remaining bone cross section is suspected, the reinforcement of the osteotomized area with a reconstruction plate sometimes is recommended. However, the benefit remains equivocal. In case of benign tumors, in which the application of adjuvant radiotherapy can be ruled out, this is possible and recommended. In these situations, the floor of the mouth still inserts at the remain-

ing lingual cortex delivering vascularization. In malignant tumor resection, it carries perhaps more risks than advantages. For the application of a reconstruction plate, the outer periosteum has to be detached. That results in de-vascularization of the lower rim, leading to avascular necrosis or osteoradionecrosis in the case of postoperative radiation. The central vascular nerve bundle does not ensure the vascular supply of the mandible alone, but the mandible is mainly supplied by the buccal periosteum coming from the facial artery system. The main prophylaxis for complications in the sense of avascular necrosis or osteoradionecrosis is to leave the soft tissue periosteal bedding outside the immediate osteotomy area, especially to the base of the mandible. For the same reason, a rim resection should never combine with an access osteotomy.

Damage to the inferior alveolar nerve cannot always be safely avoided. The safety of tumor removal has primacy. In suitable cases, the inferior alveolar nerve can be reconstructed with a nerve transplant.

13.3.2.2 Management of Inadvertent Fracture in Marginal Osteotomy

If a fracture has occurred, it must be treated in a stable way, usually with a bridging plate. The management depends on the dignity of the tumor, the expected adjuvant treatment (radiation), and the kind of concomitant soft tissue resection and the soft tissue coverage has to be respected.

In case of benign tumors, the handling of this complication is quite clear. In most of these cases, the mandibular nerve has been preserved, and the aim is to preserve the continuity of the basal mandibular arch, irrespectively of a potential additional osseous reconstruction. The treatment of choice will be stable oseosynthesis. Vascularization of the remaining bone is not severely compromised and the application of osteosynthesis material at the outer cortex without higher risk. If soft tissue covering is sufficient, a simultaneous bone grafting is possible. This may also be done in a second stage.

If a fracture of a thin residual lower margin has occurred in a malignant tumor and postoperative radiotherapy or chemoradiation may follow, a segmental resection and bridging with a reconstruction plate is a better choice in order to avoid further complications. A fracture of the lower border, even if stabilized, ends often up in avascular necrosis and often in later exposure of the bone, since the application of the plate needs subperiostal dissection/stripping of the outer mandibular cortex and disturbs vascularization of the remaining arch. This is especially true if the mandible is also skeletonized on the lingual side (in malignant floor of mouth neoplasms) and must be taken into account in the information of patients with oral cancer and planned deep marginal osteotomy.

13.3.2.3 Avascular Necrosis

Avascular necrosis preferentially occurs after osteotomy of large rims with a thin remaining mandible and subsequent radiotherapy. The main cause is insufficient vascularization. It can be avoided by preserving the thick fibrous tissue at the lower lateral border of the mandible. Vascularization of the mandible is provided either by the central neurovascular bundle but even more by the system of the facial artery, which enters the bone via the lateral periosteum, preferentially at the lateral border of the mandible. Therefore, the mandible should not be skeletonized during a rim resection and lateral soft tissue layers should remain on the outer aspect of the mandible. An example, where this issue leads to complications, is shown in Fig. 13.4a.

Hence, the lateral aspect of the mandible is freed from soft tissue only in the level of the planned osteotomy, whereas the lower lateral periosteum remains fixed. This prevents also postoperative osteoradionecrosis of the residual mandible.

Avascular necrosis becomes evident by exposure of the afflicted bone extra- or intraorally. The only measure with the prospect for success is

resection alone or with microvascular bone transplantation. The bridging with reconstruction plates alone will lead to fistula formation again and to further loss of tissue.

Case Study 4: Avascular Necrosis

At the time of the takeover of treatment of a 48-year-old patient, he presents with the fourth relapse of squamous cell carcinoma originally of

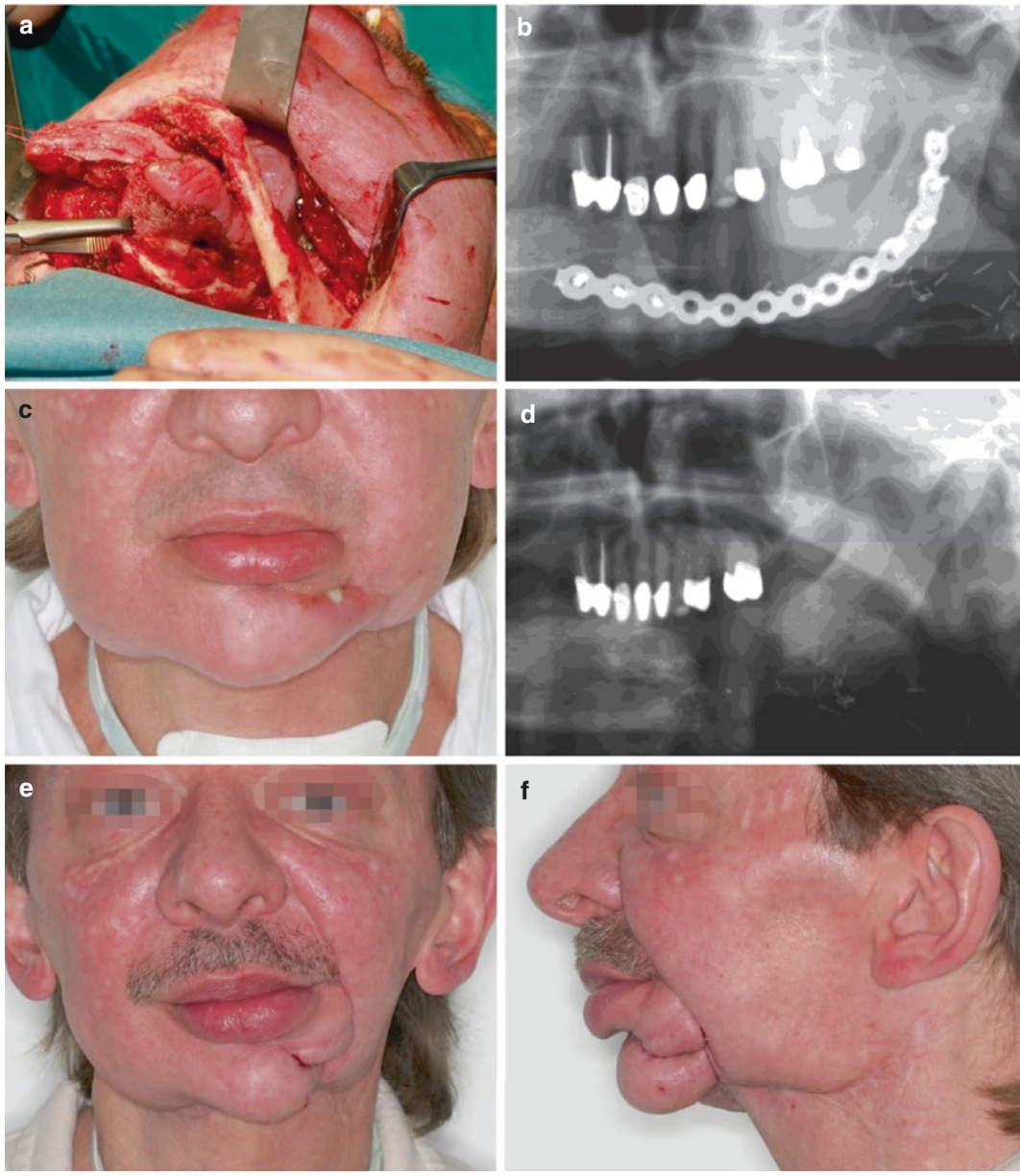


Fig. 13.4 Case study 4, avascular necrosis. (**a**) Resection of a fourth recurrence of a carcinoma of the floor of the mouth by pulling through with edge resection. The outer surface of the mandible was skeletonized and (**b**) reinforced with a reconstruction plate. (**c**) Purulent fistula formation 5 months later based on avascular necrosis. (**d**) Unsuccessful attempt of multiple debridement, then removal of remaining margin and reconstruction plate. (**e**, **f**) Situation after healing with fistula and mandibular deviation. (**g**, **h**) After reconstruction with microsurgical osteomyocutaneous iliac crest graft and flap thinning. (**i**, **j**) Implant-based prosthetic rehabilitation

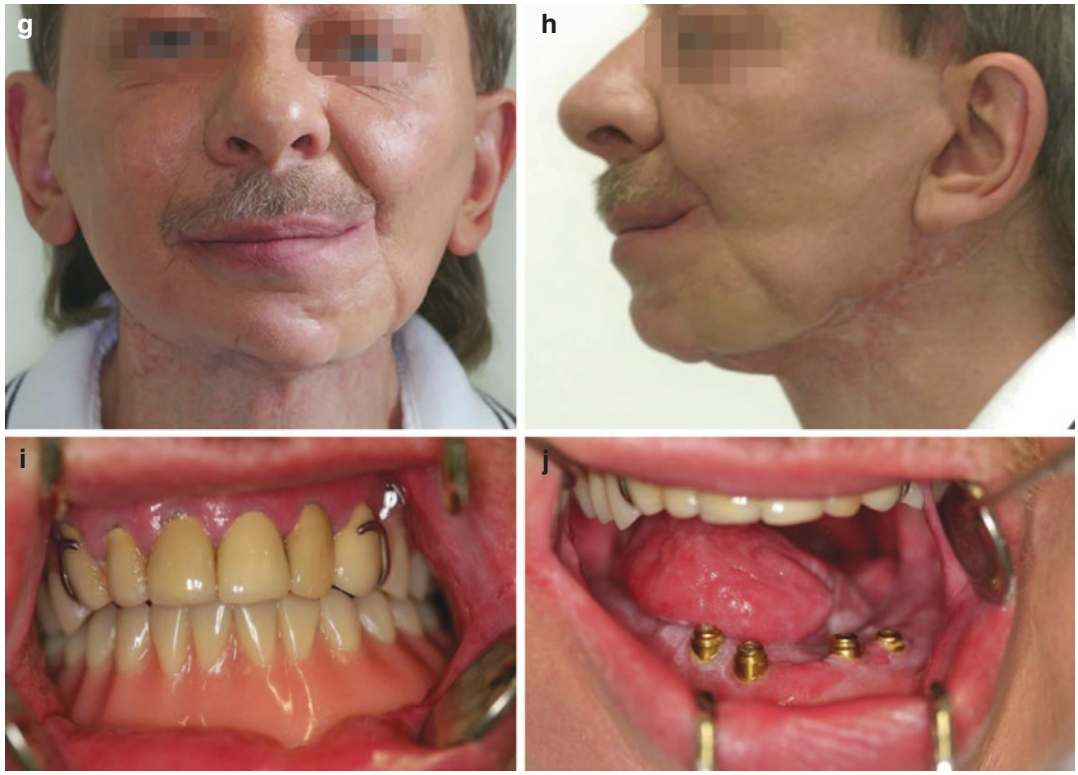


Fig. 13.4 (continued)

the floor of the mouth. The tumor had been initially treated by laser surgery, the first recurrence also by laser surgery, then by radiotherapy, and then by surgery with superficial resection of the alveolar process. The patient now presented with a fourth recurrence or tumor residue in the area of the floor of the mouth and adjacent left alveolar process. Due to the history of radiation, radical resection and subsequent brachytherapy were planned.

Resection was performed with pull-through, the mandible was skeletonized on the left side (Fig. 13.4a) and reinforced with a reconstruction plate (Fig. 13.4b), which turned out to be detrimental. Five months postoperatively, a purulent fistula formation occurred intra- and extraorally (Fig. 13.4c). This was followed by an attempt of debridement with plastic surgical covering, followed by removal of the remaining mandibular border and finally of the reconstruction plate (Fig. 13.4d). This resulted in a strong deviation of the mandible; however, the situation healed (Fig. 13.4e, f). Three months later, the reconstruction was performed with an osteomyocutaneous iliac crest graft. Figure 13.4g, h show the situation after thinning and aligning of the flap, which was initially inserted in excess, and after lifting and tightening of the corner of the mouth. Finally, an implant-supported dental rehabilitation was performed (Fig. 13.4i, j). Since then, the patient has been free of disease and recurrence for 8 years.

13.3.3 Segmental Mandibular Resection

Typical complications are damage to the marginal branch of the facial nerve and plate-associated problems when using a bridging plate without bone graft. These can occur early as mucosal dehiscence during wound healing, or later as intra- or extraoral perforation, not only after irradiation.

Primary wound healing disturbances usually occur between the 4th and 8th postoperative day. An important cause of intraoral mucosal dehiscence is not carefully smoothed cortical margins and tension of sutures, whether due to insufficient soft tissue mobilization or an insufficient flap. The most critical point is the transition zone to the toothed mandibular stump, where a very careful suturing technique must be used. It requires constant local wound care in addition to intensive oral hygiene and food supply through a nasal or percutaneous gastric tube over a longer period of time. The prospects of a secondary soft tissue closure depend on the condition of the soft tissues, and the mobilization reserves from the surrounding area. However, with a weak soft tissue covering, secondary perforations can also occur at a later time, which almost always lead to plate removal in the course of some months.

The long-term exposure of the plate after extraoral migration through the skin and subcutis is supported by postoperative radiation, thin soft tissue coverings, and overcontouring of the plate. It occurs particularly in convex plate sections. Measures for prevention are the covering with the masseteric muscle in the mandibular angle area, the attachment of which should be fixed to the plate in such a way that it surrounds the plate edge, and the avoidance of too thin tissue layers in the front. If available, the wrapping into the muscular part of myocutaneous flaps, the internal fixation of the chin soft parts to the plate, and the avoidance of void cavities under the plate are helpful. This is achieved by suture fixation of the stumps of the floor of mouth muscles and external tongue muscles suspension on the plate as well as the use of voluminous muscular flap plastics (e.g., Lat. dorsi). The undercontouring of the plate is particularly important, which in principle is possible by freehand bending but is better enabled with preoperative computer planning [21]. The most reliable way to prevent plate-associated complications is the use of primary vascularized bone grafts.

Despite all precautionary measures, up to 50% of the plates are lost within the first 2 years, whether due to material (fatigue) fracture, screw loosening, or even perforation and extraoral or intraoral exposure [22]. Then, the removal of the plate and replacement by a (vascularized) bone graft is the method of choice (case study 5, Fig. 13.5d–f). If bone grafting is not feasible due to medical-related reasons, the plate is removed without replacement, accepting a deviation of the mandible. The deviation is limited by the intermediate scarring fixation of the mandibular stumps.

If bridging of a mandibular defect lacks on the long term, this leads to severe dysfunctions: Shortening of the masticatory muscles and loss of mandibular mobility, loss of interocclusal distance, loss of lip closure, and "drooling," endangerment of the respiration due to missing suspension of the floor of the mouth and the genioglossus muscles, aspiration, and swallowing disorders due to loss of laryngeal elevation, phonetic and masticatory dysfunction. Hence, an unbridged mandibular defect must be avoided with all available means.

13.3.3.1 Plate-Associated Complications

The typical complications of reconstruction plates are fractures, screw loosening or plate fractures, and intra- or extraoral plate extrusion. This affects around 50% of the reconstruction plates after 2 years [22]. Insofar a reconstruction plate has to be considered a temporary measure for bridging a mandibular defect and intentionally should be replaced by a bone transplant 1–2 years after tumor resection.

Nevertheless, some of the reconstruction plates last much longer, and this depends on the location of the defect and on technical and procedural details. Firstly, extrusion mostly happens with long plates or at convex parts of the plate, e.g., the chin and the paramedian and lateral parts ventral to the muscle sling. The mandibular angle region and the region of the ascending ramus is protected from plate exposure by the envelope of the masticatory muscles. It is important to suture the muscles or their remnants around the plate so that it is reliably covered. The plate may be shortened/undercontoured in the angle region. In this way, long-lasting plates are possible.

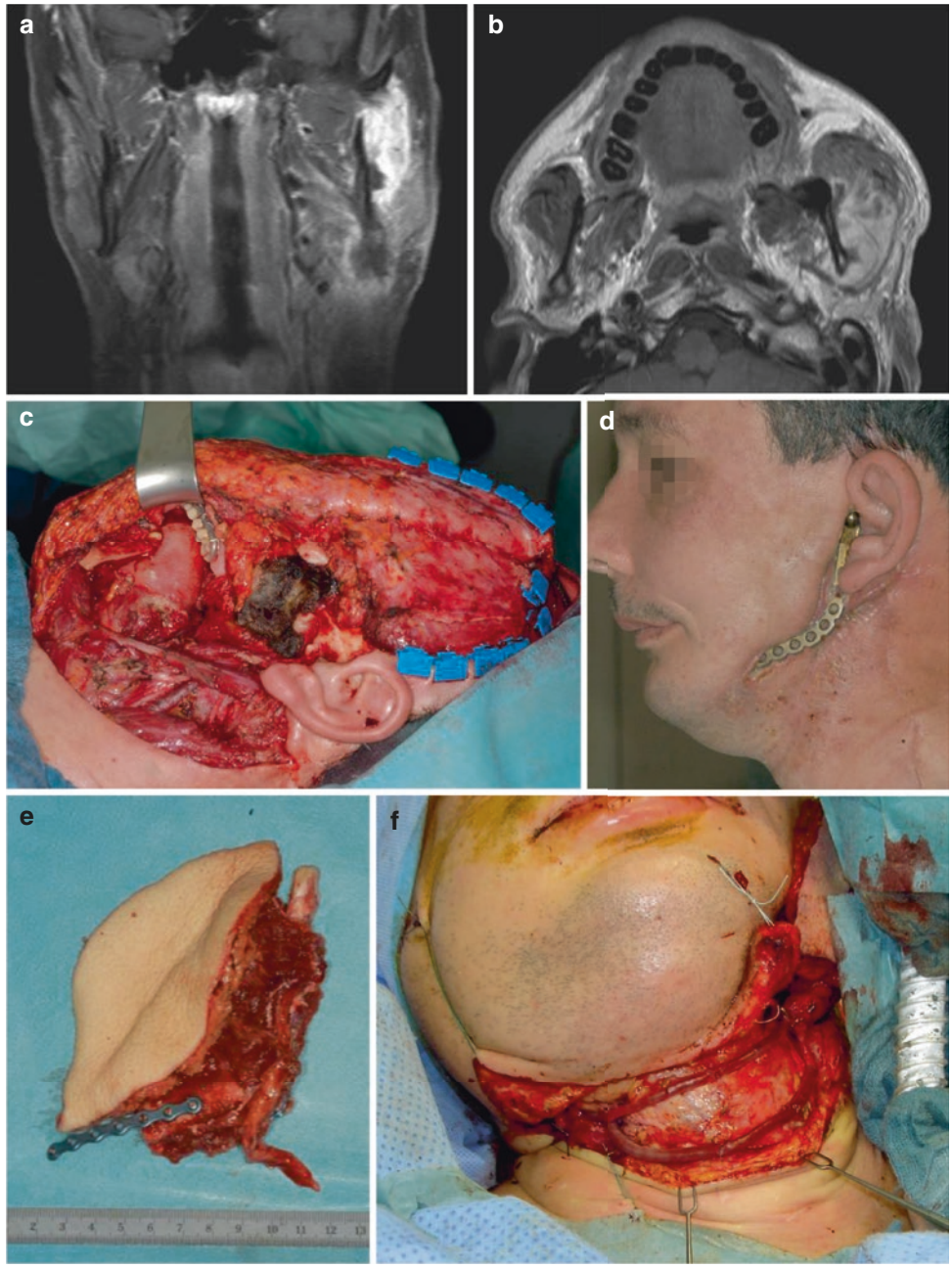


Fig. 13.5 Case study 5, exposed reconstruction plate, TMJ reconstruction, ischemic insult. (**a**, **b**) MRI of at the time of diagnosis a 36-year old patient with an adenoid cystic carcinoma of the left parotid gland with infiltration of the glenoidal fossa. A misdiagnosis as osteomyelitis had preceded. (**c**) Resection in the form of a radical parotidectomy with resection of the facial nerve and partial mandibular resection including the Fossa articularis and anterior parts of the auditory canal, primary suspension of the mimic musculature with fascia lata in remaining parts of the temporal muscle. A reconstruction of the N. facialis was not performed due to planned HDR brachytherapy for prognostic reasons. Reconstruction of the defect with latissimus dorsi flap and reconstruction plate with joint head, postoperative HDR brachytherapy. (**d**) Exposed reconstruction plate 14 months after primary surgery. (**e**) Removal of the reconstruction plate and reconstruction with osteoseptocutaneous fibular graft, joint replacement with rib graft from osteocartilaginous transition zone. (**f**) Bridging with V. saphena conduit. (**g**, **h**) Situation after lid loading with gold weight. (**i**) View after reconstruction

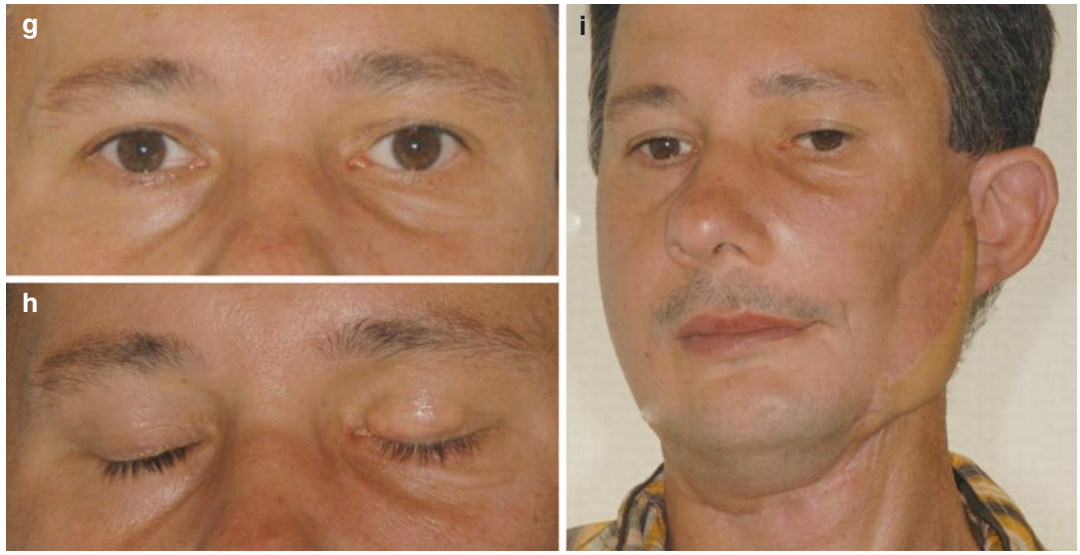


Fig. 13.5 (continued)

A further point of importance is proper fixation. Generally, 3–4 screws are recommended, the first 0.5 cm from the stump. That is true for the mandibular body. However, at the ascending ramus, it may be advisable to insert 4 or more screws, since the bone is much thinner and it is important to place the plate exactly above the dorsal rim of the ascending ramus.

The third point for plates in the mandibular body region is undercontouring at the site of the defect. This may be reached by individual bending or by use of CAD-CAM.

Case Study 5: Exposed Reconstruction Plate, TMJ Reconstruction

In this complex case, the adverse events included misdiagnosis, exposure of a reconstruction plate, and an ischemic insult. At the time of diagnosis of an adenoid cystic carcinoma T4a with perineural spreading in the left parotid gland, the patient was 36 years old. Prior to this, a misdiagnosis of osteomyelitis of the left ascending ramus and a decortication had been performed on the background of pain lasting several months. Therefore, the resection of the tumor had to be performed including the mandibular angle and the ascending ramus, and due to the extent of the tumor (Fig. 13.5a, b), including articular fossa and anterior parts of the external auditory canal (Fig. 13.5c). In order to be able to apply an excessively high radiation dose, HDR-brachytherapy was planned and the brachytherapy catheters were inserted with the primary resection. The defect was provided with a latissimus dorsi flap, and the mandible was bridged with a ready-made reconstruction plate with joint head. A primary bone graft was rejected for several reasons: First, the large-volume defect could not have been reliably sealed. Second, the insertion of the HDR brachytherapy catheters would not have been reliably possible, especially in the critical auditory canal area and in the resected fossa articularis area.

Due to complete resection of the facial nerve, a primary suspension of the mimic musculature with fascia lata was performed, which was fixed semi-dynamically to the residues of the temporal muscle. The lagophthalmus was treated with lid loading later (Fig. 13.5g, h). From about 1 year after the treatment, the reconstruction plate was progressively exposed until the joint head was included (Fig. 13.5d). The reconstruction was planned with an osteoseptocutaneous fibular graft, the joint part itself with a rib graft from the osteocartilaginous transition zone (Fig. 13.5e). In order

to connect the microvascular anastomosis of the fibular graft under the difficult scar conditions, saphenous vein grafts were interposed with a connection on the opposite side, about one and a half years after the primary operation (Fig. 13.5f). The removal of osteosynthesis material and a thinning of the skin paddle, together with surgical widening of the restricted mouth opening 8 months later, resulted in another unexpected and rare complication: The mandible had been forced open intraoperatively for about 20 min. Postoperatively, the patient woke up protracted and had aphasia. The MRI showed a limited infarct in the media flow area. It was recapitulated that the intraoperative forced opening together with the scars in the resection area, which extended to the internal carotid artery had led to an intraoperative closure of the internal carotid artery and treatment with platelet aggregation inhibitor. As a result, a brain abscess developed which was drained neurosurgically. Fortunately, the patient recovered completely without significant neurological consequences and the aphasia disappeared. The patient (Fig. 13.5i) is now free of tumor and recurrence for 15 years.

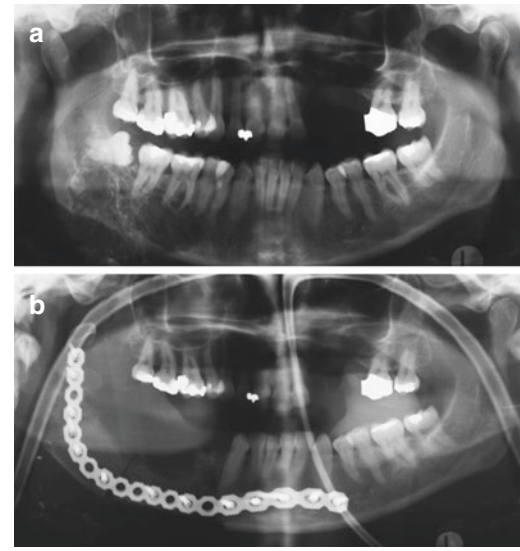


Fig. 13.6 Case study 6, malposition of condylar fragment in mandibular resection and primary reconstruction. (**a**) Odontogenic myxoma right mandibular angle and ascending ramus. (**b**) Luxation of the condylar fragment and malposition

13.3.3.2 Complications of Exarticulation, TMJ Resection/Reconstruction

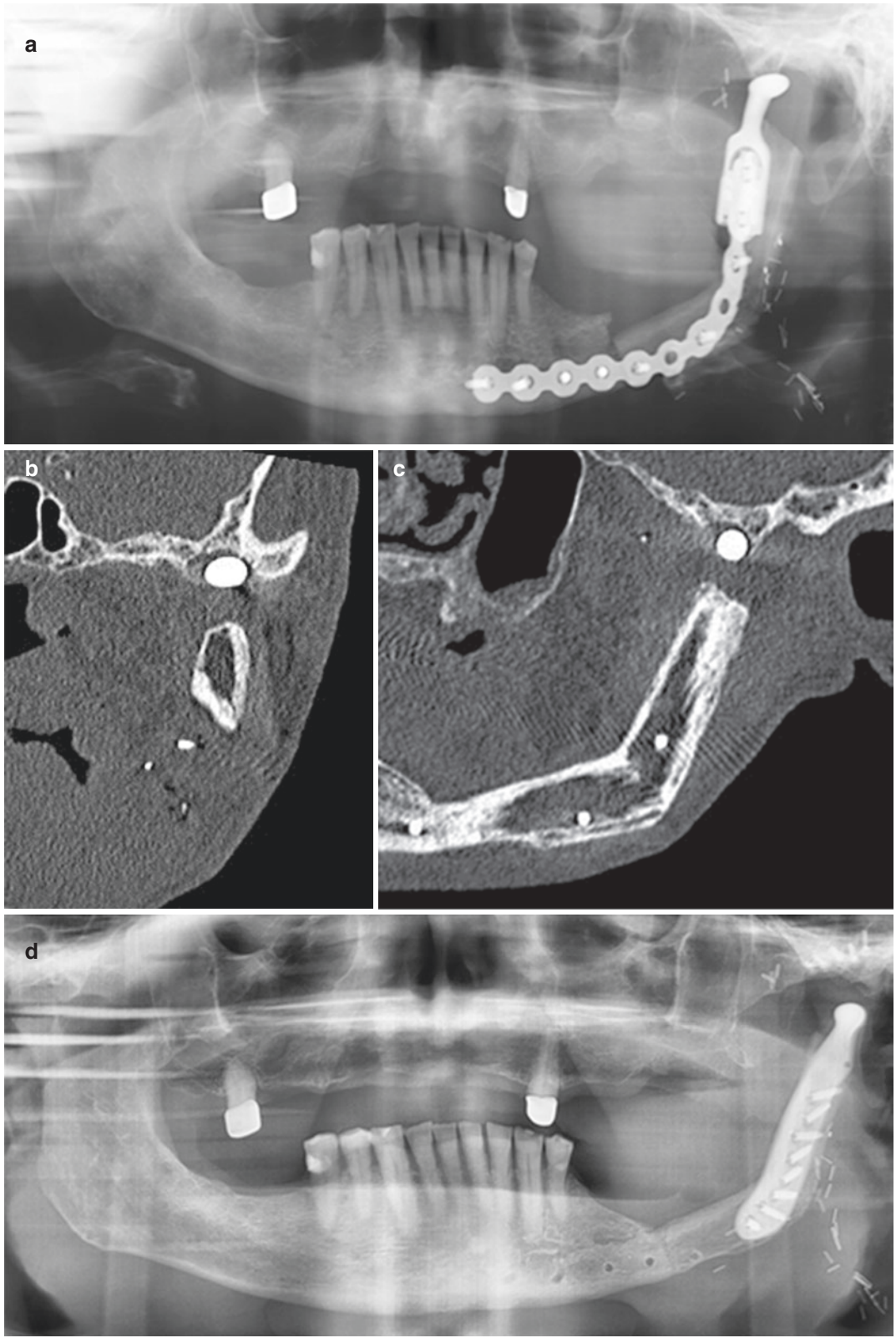
A typical risk of segmental resection and bridging with a reconstruction plate or a bone transplant is dislocation or luxation of the temporo-mandibular joint, in particular when the remaining condylar fragment is small and has lost guidance by the muscle sling. Repositioning the joint must be done immediately, later it is difficult and hardly worth the effort (Case study 6, Fig. 13.6a, b). Otherwise, a functional follow-up treatment is better in order to treat the deviation in mouth-opening conservatively.

Care should also be taken with the positioning of the reconstruction plates with ready-made condyle. Correct positioning in the cartilage-lined fossa articularis reduces risk of bony resorption. In case of incorrect positioning on bony structures, e.g., on the eminentia articularis, the risk of pressure-induced bone resorption is considerably higher (Fig. 13.7) and consequently also the entry into the middle cranial fossa. A total endoprosthesis provides a remedy.

In addition to the complications mentioned above, damage of the facial nerve needs to be noted as well as the already mentioned positioning errors of joint replacement. Facial nerve damage can also occur secondarily if the plate or prosthesis is exposed subauricularly or extraorally in the jaw angle region. Also parts of the CAD-CAM prostheses can lead to secondary pressure damage (e.g., forehead-eye-branch by the acetabular part of the TEP).

One should be aware that the joint and the ascending mandibular ramus are located near the internal carotid artery. A fortunately rare complication is described in case study 5, in which after joint reconstruction in connection with severe scarring, a blockage of the mouth opening led to an ischemic insult (by compression of the internal carotid artery).

Fig. 13.7 (**a**) Situation after primary reconstruction of a mandibular defect after resection of a large ameloblastoma with pathologic fracture. The condyle of the reconstruction plate is malpositioned on the tip of the eminentia articularis. (**b**) Frontal and (**c**) Lateral CT view showing bony resorption 2 years later. (**d**) The plate was replaced by a total endoprosthesis with alloplastic socket


a
b
c
d

Case Study 6

Condylar malposition is a risk with immediate reconstruction after partial mandibular resections, especially with high condylar resection. In the case of resection of an odontogenic myxoma in the right mandibular angle and ascending ramus treated some time ago, the condylar fragment was luxated presumably during preparation and resection, and as a result, the condyle was misplaced in the reconstruction plate. There was a pre-existing subluxation. The preparation had been performed exclusively via submandibular approach. Postoperatively, a deviation of the midline occurred to the left side and was treated with elastics.

However, the conservative treatment approach did not lead to repositioning, and so it was decided to move the mandible via corrective osteotomy and Obwegeser-Dal Pont osteotomy on the opposite side. This was only partially successful, but the symmetry of the mouth opening was improved afterward. Later, the condyle slipped in front of the eminentia again.

Looking back, it must be stated that malpositions of the condyle must be corrected immediately. In pre-existing class 2 situations, there is a higher risk of dislocation of small condylar fragments. If positioning via a submandibular approach is not safe, one should switch to a preauricular approach or a revision should include a preauricular approach. A current solution would also include a total endoprosthesis.

13.3.4 Access Osteotomy

Paramedian access osteotomies can significantly facilitate access to dorsal tumors, especially at the base of the tongue and also at the lateral oropharynx. Classically, they are part of the lip split, and actually the bony access osteotomy can only be fully used together with lip split. If lip split is to be avoided, however, it also provides a satisfactory overview and freedom of action for the base of the tongue in combination with a visor flap, although not as spacious as with lip split. The paramedian access osteotomy, which is classically ventral to the mental foramen, is virtually low in complications. The only and essential complication is pseudarthrosis, especially in association with postoperative radiation. Stepped and angled osteotomies have been described to avoid this. From the author's point of view, the essential aspect is the use of stable osteosynthesis systems in reconstruction plate format. The plate is adjusted before osteotomy, and the holes are predrilled. After tumor resection and flap suturing, stable osteosynthesis is performed with the prepared plate. If used correctly, no pseudarthrosis occurs, not even after radiation. However, an access osteotomy should not be combined with a marginal mandibulotomy and no exposure of the vestibular mandibular cortex should be performed. Both are detrimental to blood flow to the mandibular stumps and must be avoided in conjunction with an access osteotomy.

13.3.5 Maxillary Resection

Apart from the risk of bleeding, resections on the maxilla are actually relatively uncomplicated. The essential risks and the high complexity of maxillary defects are only revealed in the reconstruction (see Chap. 15). Complications arise above all with the involvement of the dorsal parts of the maxilla, especially the pterygoid. The risk of bleeding from branches of the maxillary artery and from the venous pterygoid plexus, which can impair the clarity of the surgical site, is particularly high. A rare but relevant complication in high dorsal resections is amaurosis, either by direct injury to the optical canal or by atypical fractures from the pterygoid into the sphenoid bone. The use of a navigation device is helpful in this localization. In case of en bloc resection of the pterygoid process, this should be cut or weakened preferentially with a piezosurgical device before mobilizing the maxilla with osteotomes.

The use of piezosurgery significantly reduces the risk of bleeding. All bone incisions and accessible soft tissue margins should be prepared before mobilizing the maxilla.

13.3.6 Tongue and Floor of the Mouth Resection

The complications of resection of the floor of the mouth and the tongue are described together, as they merge into each other due to their anatomical

proximity. Typical complications in the area of the tongue, floor of the mouth, and cheeks include the injury of the various functional relevant structures, from the sensory trigeminal branches to the excretory ducts of the large salivary glands.

Some of these injuries are inevitably necessary due to the expansion of the tumor, others happen unintentionally as a complication

N. lingualis: In most larger tumors of the floor of mouth, the lingual nerve has to be resected due to his proximity to the tumor. The lingual nerve is preserved in smaller tumors in the anterior floor of mouth and in tumors of the tongue, which extend not too much laterally. In latter, he is identified from submandibular or intraoral after incision in the lateral floor of the mouth and followed dorsally. If an appropriate distance to the tumor can be achieved, it is held away laterally.

N. hypoglossus: In order to avoid injuries to the trunk of the hypoglossal nerve, it should be identified from the cervical side right at the beginning of a resection in the anterior or lateral floor of the mouth. The muscles of the floor of the mouth, especially the mylohyoid muscle, are severed parallel to the mandible at an appropriate safety distance from the palpable tumor. The hypoglossal nerve is immediately behind them. The resection plane is followed at a sufficient distance from the tumor but laterally/cranially to the course of the hypoglossal nerve. He radiates into the tongue musculature with a medially located fan in parasagittal alignment, so that this usually works well with small- and medium-sized tumors of the lateral tongue or floor of mouth. This fan may be used as a preparation plane, thus protecting the branches in a targeted manner. Since the hypoglossal nerve radiates in several portions from dorsal to ventral into the tongue muscles, the ventral parts can be removed and the dorsal parts important for the second phase of the swallowing still function. This is particularly relevant for carcinomas of the anterior floor of mouth infiltrating from ventral into the root of the tongue. However, it is important that the nerve stem and the branches running parasagittally from here are preserved as far ventrally as possible. Therefore the exposure right at the beginning of the resection is important. A further source of complications is the injury of the accompanying vein. If it bleeds and coagulates, this can also lead to postoperatively relevant hypoglossal damage. Care should therefore be taken to protect them.

Whartons duct: Injuries or resections are only playing a role with resection of small squamous cell carcinomas or leukoplakia of the anterior floor of the mouth, when no neck dissection is performed. A marsupialization extending far dorsally is recommended in order to avoid a stricture. In the case of a late stricture of the Wharton's duct with consecutive sialadenitis, complication management consists of renewed marsupialization, or in case of failure, submandibulectomy.

Wound healing disorders should be treated as quickly as possible to avoid cervical fistulas (see chapter “Tissue necrosis and wound healing disturbance”).

Also, postoperative bleeding and tongue edema are main complications of tongue surgery, which are addressed in chapter “intra- and postoperative bleeding.” Both cause an obstruction of the oral cavity and the pharyngeal respiratory passage by swelling of the tongue body. Bleeding into the tongue body after the closure of the sutures requires reopening of the wound and revision of the bleeding areas. If necessary, a tracheotomy may be necessary at the latest, and all interventions with a higher risk of swelling should better be planned with a primary tracheotomy.

If the muscular bulk of the pedicle remains too narrow in the middle and dorsal third of the tongue during a tongue resection, this may lead to a circulatory disorder and subsequent necrosis of the tip of the tongue. Parts of the tongue with a strong livid discoloration intraoperatively or immediately postoperatively do not usually recover (see chapter “Tissue necrosis and wound healing disturbance,” case study 1/Fig. 13.1).

Specific aspects of vascularization and preservation of the tip of the tongue despite resection in the base or root area of the tongue: In the case of tumor infiltrations of the base of the tongue or the root of the tongue close to the midline or crossing the midline, there is a risk that the anterior third of the tongue, which is not tumor infiltrated, succumbs to necrosis or cannot be preserved. In general, a primary non-surgical procedure, i.e., radiochemotherapy, should be considered for tumors in these localizations. In salvage situations, however, the question of the indication for

surgery may arise as well as the question of the preservation of the residual tongue. In suitable cases, the anterior part of the tongue can still be preserved and remain functional, even if limited. For this purpose, it is important to identify the winding course of the lingual artery of the opposite side of the imaging and to preserve it specifically. The distance of the lingual artery of the opposite side from the tumor is thus the essential criterion for the functional resectability of the tumor. The venous outflow can be maintained better by leaving the opposite side broadly pedicled in the mucosa and by initially avoiding neck dissection on the opposite side, which can be done later if necessary.

In patients, in which tongue has been resected, or even has been lost, reconstruction is the second choice, which is illustrated with the following case study.

13.3.6.1 Case Study 7

A male patient presented with complete loss of the floor of the mouth and the tongue, the mandible shortened and deviated, on the left side a retracted latissimus dorsi flap in situ (Fig. 13.8a–d). Anamnestically an oropharyngeal carcinoma including continuity of the mandible and parotidectomy had been resected 2 years ago at age of 52. During a later attempt to reconstruct the mandible, the tongue and the floor of the mouth were lost, presumably due to a bilateral ligation of the lingual vessels.

The reconstruction was performed in several steps. The first step was a tongue reconstruction with a neurovascular anastomosed latissimus dorsi flap and the bridging of the mandibular defect with a reconstruction plate (Fig. 13.8e). In the second step, the reconstruction of the mandible and floor of mouth with an osteoseptocutaneous fibula graft was planned. Due to the difficult vascular conditions, the vascular anasomosis was performed with saphenous conduits arterial end-to-end to the external carotid artery and venous end-to-end to the jugular vein of the opposite side.

Two days after the operation, venous congestion occurred in the skin flap of the fibular graft, so that the indication was put for revision. Thrombi appeared in all sections of the venous system of the conduits and the fibula flap. The thrombi were removed with Fogarthy catheters, and the veins of the flap were almost completely opened via incisions and exposed under microscopic view. The venous conduit was renewed and all anastomoses revised. This was followed by intensive antithrombotic treatment with dual platelet aggregation inhibition and anticoagulation (full heparinization), regular sonographic Doppler monitoring of the saphenous conduits. The skin island was lost superficially, but the subepidermal parts were preserved, as was the bone. Fourteen days later, the area above the fibula graft was covered with split skin graft.

Later, after healing, a CAD-CAM total temporomandibular joint endoprosthesis was implanted (Fig. 13.8f, g). The pre-existing facial paralysis was treated by suspension of the lips and the lower face soft tissues including the flap with fascia to the temporal muscle (Fig. 13.8h–j). The patient received three repeated contour corrections by lipofilling. The swallowing could be improved by increasing the volume of the neotongue also with repeated lipofilling. The patient swallows without aspiration.

13.3.7 Resection of the Soft Palate

A resection of the soft palate affects usually also the lateral wall of the oropharynx (if the soft palate is not even considered part of the oropharynx). Larger tumors also require resection of the tuber maxillae. With these en bloc resections directed dorsally upward, there is a considerable risk of intraoperative bleeding. If the extension is caudal, the base of the tongue and the posterior lateral floor of the mouth are affected, and laterally the mandible. A simultaneous segmental resection of the mandible makes the procedure considerably easier, as the access is then very straightforward. If a mandibular segment resection is not indicated, there are two possibilities: Delivery to the neck or access mandibulotomy. Delivery into the neck may require a partial lingual release in order to obtain sufficient visibility and accessibility from caudal. However, it is important to prepare from the caudal side, i.e., from the neck side and upward as far as possible along the dorsal circumference of the tumor, in order to be able to carry out the preparation on

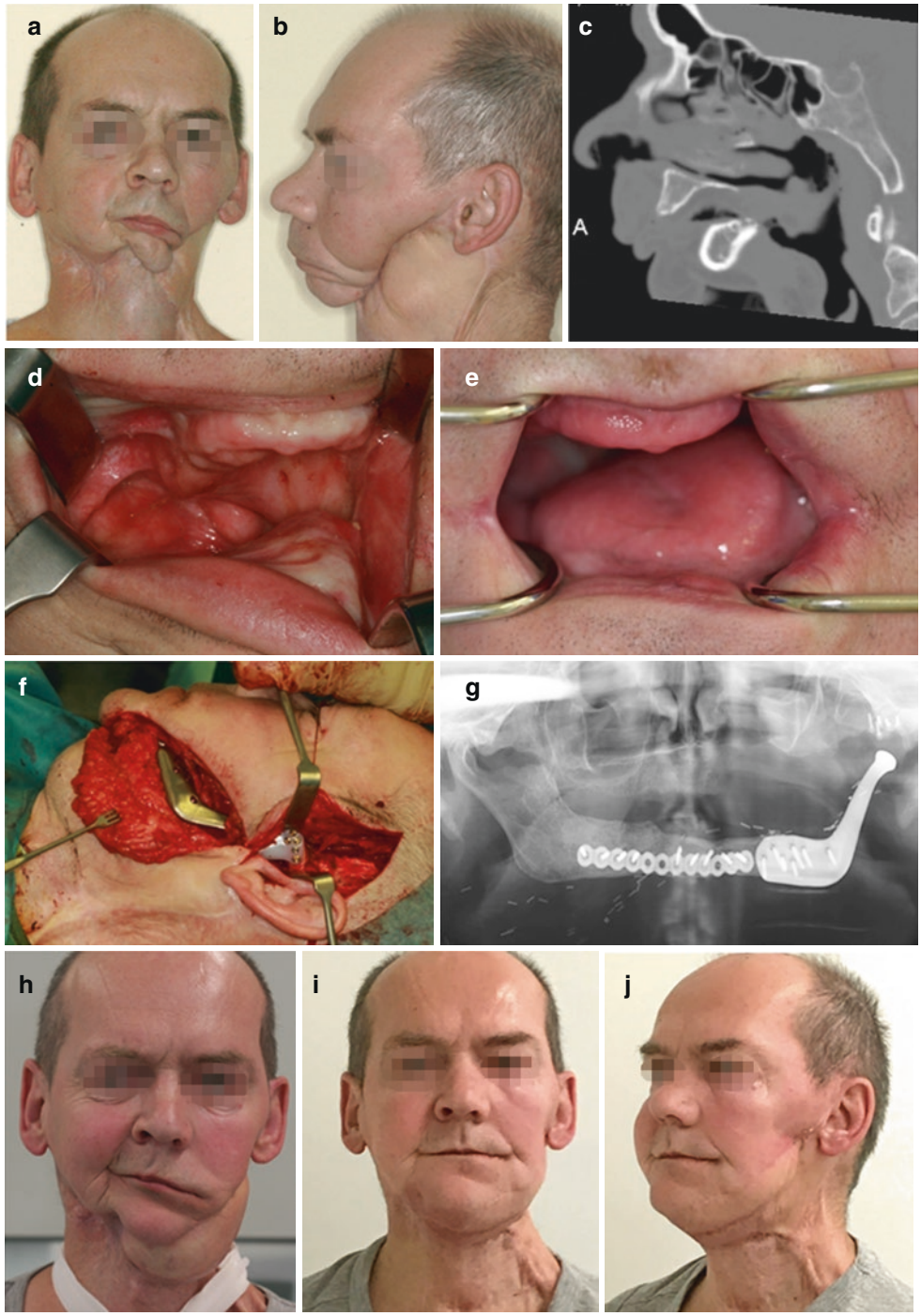


Fig. 13.8 Case study 7, patient with complete loss of tongue and floor of mouth, facial paralysis left lower face. (**a**, **b**) First presentation. (**c**) CT imaging. (**d**) Intraoral view. (**e**) In the first step, the tongue was reconstructed with a neurovascular anastomosed latissimus dorsi flap, and the mandibular defect with a reconstruction plate and ready-made joint head. (**f**) The next steps comprised mandibular reconstruction with a fibula graft, which proved to be difficult due to revision surgery, including the saphenous vein conduits (not shown, see text). It was followed by two-stage flap thinning and joint reconstruction with custom-made total endoprosthesis. (**g**) Panoramic view after fibular graft and custom-made TEP. (**h**) Frontal view after mandibular reconstruction, prior to facial suspension. (**i**, **j**) Situation after suspension of mimic musculature with fascia lata, lid-loading, and twice lipofilling (chin and neck right side, left preauricular region). Also intraoral lipofilling performed, satisfactory swallowing function remained, despite loss of volume in the neo-tongue, also in the wake of the revision surgery of the fibula (see text)

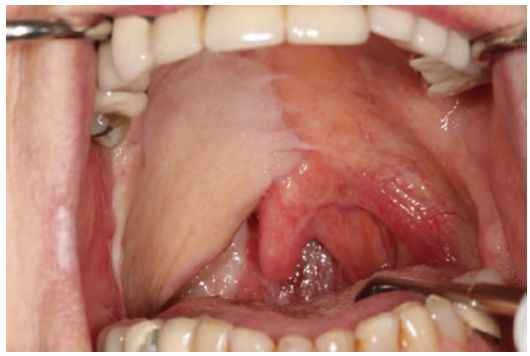


Fig. 13.9 Example of the approach for the reconstruction of the soft palate and the intermaxillary fold with the radial flap. The flap is folded and doubled, sutured to the wound edges of the posterior pharyngeal wall and the nasal surface of the soft palate, forming a tight epithelial barrier to the upper pharyngeal cavity

the dorsal aspect of the maxilla as the last step (because of bleeding risk).

The reason for wound healing disorders in the soft palate mostly is an insufficient suturing or tightness against the nasal/epipharyngeal space. When reconstructing with a radial forearm flap, the flap must be folded to achieve an epithelial seal to the pharyngeal/epipharyngeal space as well as to the oral cavity. The backmost margin of the flap has to be sutured with the mucosa of the pharyngeal wall and of the nasal side of the velum. The flap is then folded along the caudal edge of the velum to be reconstructed and the anterior margin is sutured in the oral side of the defect (Fig. 13.9). It is of importance that the reconstruction is tightly epithelium-covered on the nasal as well as on the oral aspect. When the reconstruction of the nasal surface is disregarded, the load with secretion leads to bacterial transmigration from the nasal side and ends up with dehiscence of the velar reconstruction.

Possible complications are above all injuries of the major vessels, nerves and further anatomically functionally relevant structures. Their injury is circumvented in detail by the above-mentioned planned approach as follows:

Large cervical vessels: If the tumor expands dorsally medially into the vicinity of the large vessels, they are first prepared as far as possible cranially near the base of the skull, and branches (e.g., of the external carotid artery) are ligated and severed as required, so that the resection plane to the vessels is clarified before mobilization of the tumor.

N. lingualis: The lingual nerve can only be preserved in rare cases of these tumors. Procedures for its protection correspond to what has already been described in the chapter “Tongue and floor of mouth resection”.

N. hypoglossus: The protection of the hypoglossal nerve is simple in principle. It has already been identified during the removal of the neck and is the first to be presented cervically during tumor resection. The floor of the mouth is cut laterally to the tumor at an appropriate safety distance. In the further course, the hypoglossus nerve together with the tongue is held away medially and is thus out of the resection area.

Glossopharyngeal nerve. The glossopharyngeal nerve is rarely endangered, since it runs far medial to the vessels, in the upper part laterally to the carotid artery and is therefore spared in itself. His injury in the area close to the stem, especially the ganglia, condemns severe swallowing disorders and should be avoided. However, the risk only exists of very large tumors or salvage resections.

The auditory tube is occasionally located in the resection area in larger tumors reaching epipharyngeally. If the tube bead is located in the marginal area of the resection, it must be remembered that postoperative swelling can lead to obstruction. Prophylaxis with decongestant nasal drops can be attempted. A paracentesis is indicated in the case of an obstruction but anyway in the case of resection and covering with a flap.

13.4 Functional Impairments

13.4.1 Articulation

Resections in the area of the tongue, the floor of the mouth, and the soft palate affect articulation. There are some points to be considered:

Oral tongue defects up to about one-third of the muscle volume may be closed by direct closure and/or rotation of the tongue body as long as the floor of the mouth is not affected. The defect is covered by mobilizing the mucosa from the floor of the mouth longitudinally, as long as the tongue body does not become too narrow, with V-Y-like shortenings of the resection margin. The tip of the tongue can also be rotated transversally into the defect, but strong rotation to the defect side is unfavorable for articulation. Strong (thickness 3-0 or 2-0), sufficiently deep and not too tightly knotted single sutures are used for the sutures on the tongue body. Larger defects benefit, depending on the location, from the use of flaps, but these must not be oversized. In the case of losses of up to 50% of the volume of the oral tongue, the tongue function does not remain significantly disturbed in the long term as long as the oral floor mucosa is preserved and mobility is provided by reserve mucosa or flap reconstruction.

It is important to reconstruct the soft palate as explained above in order to avoid velopharyngeal insufficiency. The velum must have some volume, which is delivered by the doubled radial forearm flap. Dehiscence must be avoided as mentioned above, because secondary sutures are often unsuccessful.

13.4.2 Dysphagia and Aspiration

Dysphagia is a greater problem. In addition to the loss of muscle mass and thus of mobility and strength, it is aspiration which, after resection and reconstruction in the dorsal sections of the lower oral cavity floor, leads to swallowing disorders and to decannulation failure (or even to pneumonia or death).

The aspiration can be intradeglutitive, i.e., take place during the pharyngeal phase of the swallowing process, e.g., due to insufficient laryngeal elevation, or postdeglutitive, i.e., food components slide into the glottis area after the swallowing process, which is often related to sensory disturbances in the area of the swallowing road, as bolus retentions are not noticed and then slide into the laryngeal entrance after the actual swallowing process. This has consequences for resection and reconstruction:

1. Flaps in the transition zone from the lateral floor of the mouth to the pharynx or the base of the tongue should be avoided, as they form an insensitive ramp over which bolus retentions slide unnoticed into the larynx. The base of the tongue and the pharyngeal ring area should be reconstructed with local tissue as possible. This can be achieved in the area of the base of the tongue, e.g., by dorsal displacement of the residual tongue body and primary suturing, and above this, the lateral oropharyngeal wall may reconstructed with the flap. Bulky myocutaneous flaps are much more problematic than thin flaps, e.g., radial forearm flaps, which also regain sensitivity after some time.
2. The preservation of laryngeal elevation is essential for swallowing. In particular, in association with primary osseous reconstruction spanning the anterior mandibular arch, a suspension of the hyoid is difficult to achieve. However, this function of the suprahyoid muscles is essential for the swallowing process; otherwise, the laryngeal elevation cannot take place and aspiration threatens. It is possible to fix the suture on the remaining lateral mandibular stumps. In all standard resections, we ensure that the remaining suprahyoid muscles are refixed to the mandibular arch.

13.4.3 Facial Paralysis

The risk of damaging the marginal branch of the facial nerve is high when incisions of skin and platysma are running along or to near by the horizontal border of the mandible, because layer preparation on the submandibular gland is not longer possible. Even with correct incision it is a known problem of submandibular approach to the neck and to the mandible in the resesction of oral cavity tumors. The problem for the patients after damage of the marginal branch of the facial

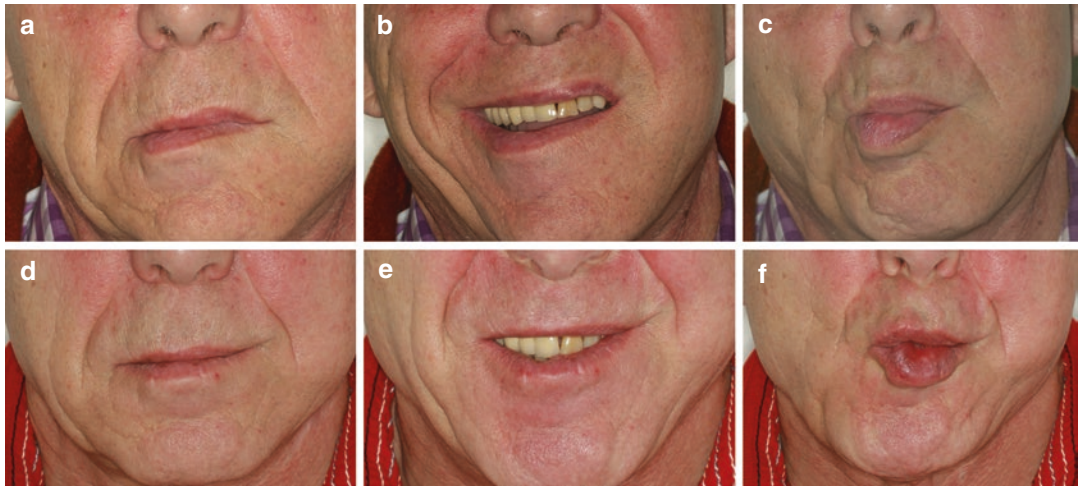


Fig. 13.10 Example of a patient with paralysis of the mandibular branch of the facial nerve after resection of a basal cell carcinoma and a cheek rotation flap, but situation is also transferable to patients with this paralysis after resection of intraoral tumors. The disfigurement is noticeable under function (**a**. at rest, **b**. smiling, **c**. pursing of the lips). After insertion of a Gore-Tex band, the lower lip is stabilized, and the asymmetry is less unpleasant even under function (**d**. at rest, **e**. smiling, **f**. pursing)

nerve is that the affected side of the lower lip is stretched under function (pursing the lips or smiling). The disfigurement can be improved, albeit imperfectly, by inserting a Gore-Tex band or fascia under the vermilion, which restrains this stretching. The band may be fixed at the next functioning muscle or in the level of the SMAS (see example Fig. 13.10).

In complete resections of the facial nerve, where immediate nerve reconstruction is not indicated or adequate (which however should remain the first choice), there are good experience with reining techniques and other secondary procedures. This includes fixation directly at the temporal muscle (case study 5 and 7) or in the McLaughlin-technique. If applicable, an additional SMAS facelift is helpful. Gore-Tex bands should not be used in irradiated tissue, and one should use fascia lata for this purpose. Lid loading is very helpful (case study 5). "Les petites gestes supplémentaires" form facelift and lipofilling (see case study 7), which proves to be permanent after two to three sessions in the lateral facial and temporal region.

References

1. Luryi AL, Chen MM, Mehra S, Roman SA, Sosa JA, Judson BL. Hospital readmission and 30-day mortality after surgery for oral cavity cancer: analysis of 21,681 cases. Head Neck. 2016;38(Suppl 1):E221–6.
2. Awad MI, Shuman AG, Montero PH, Palmer FL, Shah JP, Patel SG. Accuracy of administrative and clinical registry data in reporting postoperative complications after surgery for oral cavity squamous cell carcinoma. Head Neck. 2015;37(6):851–61.
3. Schwam ZG, Sosa JA, Roman S, Judson BL. Complications and mortality following surgery for oral cavity cancer: analysis of 408 cases. Laryngoscope. 2015;125(8):1869–73.
4. Nouraei SA, Middleton SE, Hudovsky A, Darzi A, Stewart S, Kaddour H, et al. A national analysis of the outcome of major head and neck cancer surgery: implications for surgeon-level data publication. Clin Otolaryngol. 2013;38(6):502–11.
5. Patel RS, McCluskey SA, Goldstein DP, Minkovich L, Irish JC, Brown DH, et al. Clinicopathologic and therapeutic risk factors for perioperative complications and prolonged hospital stay in free flap reconstruction of the head and neck. Head Neck. 2010;32(10):1345–53.
6. Pohlenz P, Klatt J, Schmelzle R, Li L. The importance of in-hospital mortality for patients requiring free tissue transfer for head and neck oncology. Br J Oral Maxillofac Surg. 2013;51(6):508–13.

7. Goetz C, Burian NM, Weitz J, Wolff KD, Bissinger O. Temporary tracheotomy in microvascular reconstruction in maxillofacial surgery: benefit or threat? J Craniomaxillofac Surg. 2019;47(4):642–6.
8. Halfpenny W, McGurk M. Analysis of tracheostomy-associated morbidity after operations for head and neck cancer. Br J Oral Maxillofac Surg. 2000;38(5):509–12.
9. Meier J, Wunschel M, Angermann A, Ettl T, Metterlein T, Klingelhoffer C, et al. Influence of early elective tracheostomy on the incidence of postoperative complications in patients undergoing head and neck surgery. BMC Anesthesiol. 2019;19(1):43.
10. Adam H, Hemprich A, Koch C, Oeken J, Schmidt H, Schramek J, et al. Safety and practicability of percutaneous translaryngeal tracheotomy (Fantoni technique) in surgery of maxillofacial and oropharyngeal tumours—own results and review of the literature. J Craniomaxillofac Surg. 2008;36(1):38–46.
11. Yeung JK, Harrop R, McCreary O, Leung LT, Hirani N, McKenzie D, et al. Delayed mobilization after microsurgical reconstruction: an independent risk factor for pneumonia. Laryngoscope. 2013;123(12):2996–3000.
12. Nkenke E, Vairaktaris E, Stelzle F, Neukam FW, St Pierre M. No reduction in complication rate by stay in the intensive care unit for patients undergoing surgery for head and neck cancer and microvascular reconstruction. Head Neck. 2009;31(11):1461–9.
13. Xu J, Hu J, Yu P, Wang W, Hu X, Hou J, et al. Perioperative risk factors for postoperative pneumonia after major oral cancer surgery: a retrospective analysis of 331 cases. PLoS One. 2017;12(11):e0188167.
14. Bartella AK, Kamal M, Teichmann J, Kloss-Brandstatter A, Steiner T, Holzle F, et al. Prospective comparison of perioperative antibiotic management protocols in oncological head and neck surgery. J Craniomaxillofac Surg. 2017;45(7):1078–82.
15. Wang C, Fu G, Liu F, Liu L, Cao M. Perioperative risk factors that predict complications of radial forearm free flaps in oral and maxillofacial reconstruction. Br J Oral Maxillofac Surg. 2018;56(6):514–9.
16. Belusic-Gobic M, Car M, Juretic M, Cerovic R, Gobic D, Golubovic V. Risk factors for wound infection after oral cancer surgery. Oral Oncol. 2007;43(1):77–81.
17. Sato J, Goto J, Harahashi A, Murata T, Hata H, Yamazaki Y, et al. Oral health care reduces the risk of postoperative surgical site infection in inpatients with oral squamous cell carcinoma. Support Care Cancer. 2011;19(3):409–16.
18. Bartella AK, Lemmen S, Burnic A, Kloss-Brandstatter A, Kamal M, Breisach T, et al. Influence of a strictly perioperative antibiotic prophylaxis vs a prolonged postoperative prophylaxis on surgical site infections in maxillofacial surgery. Infection. 2018;46(2):225–30.
19. Wagner ME, Rana M, Traenkenschuh W, Kokemueller H, Eckardt AM, Gellrich NC. Piezoelectric-assisted removal of a benign fibrous histiocytoma of the mandible: an innovative technique for prevention of dentoalveolar nerve injury. Head Face Med. 2011;7:20.
20. Barttelbort SW, Bahn SL, Ariyan SA. Rim mandibulectomy for cancer of the oral cavity. Am J Surg. 1987;154(4):423–8.
21. Essig H, Rana M, Kokemueller H, von See C, Ruecker M, Tavassol F, et al. Pre-operative planning for mandibular reconstruction—a full digital planning workflow resulting in a patient specific reconstruction. Head Neck Oncol. 2011;3:45.
22. Kämmerer PW, Klein MO, Moergel M, Gemmel M, Draenert GF. Local and systemic risk factors influencing the long-term success of angular stable alloplastic reconstruction plates of the mandible. J Craniomaxillofac Surg. 2014;42(5):e271–6.

Complications in Neck Dissection

14

Volker Hans Schartinger

Contents

14.1 General Considerations

In primary treatment of the clinically positive neck, the extent of neck dissection was clearly reduced since Crile first referred to this surgical procedure in 1906 as radical neck dissection (RND) [1]. Today, modified radical neck dissection (MRND) is considered to be the reference treatment in this situation. However, even selective neck dissection (SND) seems to provide reliable regional control rates in selected cases with single nodal positivity [2].

In the clinically negative neck, there is growing evidence that performing an elective SND is superior to a wait-and-see strategy [3]. Level V is never dissected in these cases, because the risk for occult lymph node metastases is very low and the branches of the spinal accessory nerve (SAN) innervating the trapezius muscles typically pass this level with a long course. Additional reduction of dissected levels should avoid further typical complications. For example, to spare dissection of level IIB further reduces the risk of injuries of the SAN and leaving level IV untouched should reduce the rate of chyle leak [4].

V. H. Schartinger (✉)
Department of Otorhinolaryngology, Medical University of Innsbruck, Innsbruck, Austria
e-mail: volker.schartinger@i-med.ac.at

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_14

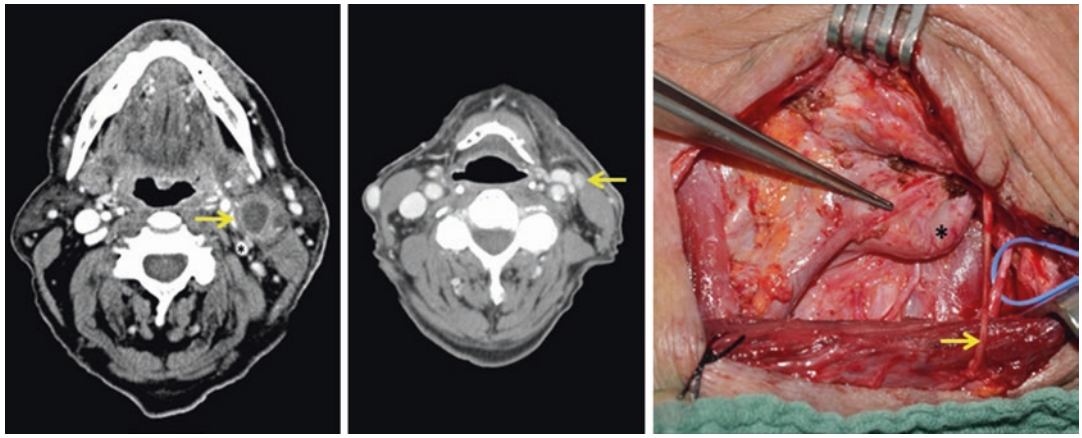


Fig. 14.1 A patient before and after a concomitant radiochemotherapy of a p16 positive squamous cell carcinoma of the oropharynx (cT1N1M0 according to TNM classification 8th edition): (left) bulky nodal disease in level II with compression of the vena jugularis interna (arrow) and kinking of the internal carotid artery (∗) before treatment; (middle) restaging showed a small contrast enhancing lymph node (arrow) at the border to level III suspicious for residual disease and patient was admitted to salvage surgery. In this case, extent of the neck dissection was reduced to level II and III; (right) intraoperative situs with sparing of the spinal accessory nerve (marked with vessel loop) and the great auricular nerve (arrow). The forceps points to a thin and compressed vena jugularis interna near to a kinking internal carotid artery (∗)

After treatment with combined radiochemotherapy, a planned neck dissection is not considered standard of care anymore. If there is evident residual disease, the type of the neck dissection may range from a radical to a selective and even superselective neck dissection (Fig. 14.1). This critically depends on the extent of the disease and the infiltrated structures, which has to be carefully assessed preoperatively [5, 6]. Furthermore complications can be minimized by choosing the right time window for the neck dissection which is approximately 4–12 weeks after initial treatment [7].

Although reduction of dissected levels is advisable in adequately selected patients, minimal invasive approaches are not recommended. Neck dissection should be performed by accurate identification of anatomic landmarks and accurate exposition of vital structures to prevent complications.

Summarized, it appears conclusive that reducing frequency and extent of a surgical procedure results in less complications.

Bulky nodal disease has to be treated with adequate invasiveness in order to cure the patient in any way. In contrast, elective SND should be a safe procedure and permanent consequences should be avoided.

14.2 Severe and Life-Threatening Complications

14.2.1 Sudden Death

Sudden death is the most severe complication in all surgical procedures. In neck dissection, these events are reported to be 0.5% and 1.3% during the first 3 and the first 30 postoperative days, respectively. Early sudden deaths are frequently associated with thromboembolic events, whereas later ones are more caused by previous other postoperative complications. Venous thrombosis prophylaxis should be provided to all hospitalized cancer patients, especially to prevent early sudden death [8, 9].

14.2.2 Stroke

Stroke is a rare complication in neck dissection but strongly associated with 30-day mortality. The reported incidence rates of a perioperative stroke after neck dissection are clearly below 1%, even below 0.1% in some studies. The benefit of the operation outweighs the risk of a stroke by far. Nevertheless, there is higher risk in older

patients and/or patients with comorbidities associated with carotid stenosis. These patients have a significantly higher risk, especially if bilateral neck dissection is performed. This may influence the general decision-making process how to treat patients with multiple comorbidities [10].

14.2.3 Regional Failure

Regional failure after treatment is an important complication and associated with poor survival. A minimum nodal yield is required for each type of neck dissection and the likelihood of detection of occult metastases increases with the number of dissected lymph nodes [11, 12].

14.2.4 Injury or Ligation of the Internal Jugular Vein (IJV)

Unintended injury of the IJV can be associated with severe blood loss and/or air embolism. Air embolism is rare but can lead to severe anesthesiological complications with sudden falls of end-tidal carbon dioxide levels and blood pressure. These patients should be immediately placed to Trendelburg position and rotated to the left side. Aspiration of air from the right side of the heart may be required.

Limited lacerations of the IJV can be recomposed by sutures. Sacrificing one IJV is an integral part of a RND and leads to no severe functional consequences. To avoid intraoperative life-threatening events, anatomy and infiltration of the IJV should be carefully studied preoperatively and if necessary, intraoperatively marked with vessel lopes.

In cases of bilateral neck dissection, thrombosis may occur on the contralateral side where the vein is preserved in up to 30% of the cases. Bilateral occlusion of the IJV has to be avoided. Extensive prolonged edema, dangerous increase of intracranial pressure, and even blindness and death may occur in these patients. If one IJV had to be sacrificed, at least 1 month has to pass before operating the contralateral side is reasonable [13, 14].

14.2.5 Carotid Blow Out

Unintended injury of the carotid artery in the untreated neck in elective SND is and should be a very rare event. On the other hand, previous irradiation of the neck with or without residual disease may result in a confusing situs and dissection can be very difficult, especially when advanced fibrosis has occurred years after initial radiotherapy.

Interestingly, sometimes even bulky disease can be easily cleared of the vessel without any complication. Preoperative assessment of the anatomic course, the structure and the involvement of the carotid artery makes intraoperative complications unlikely.

Surgeons should be aware of kinking of the internal carotid artery in level II (Fig. 14.1) and should develop a strategy if the tumor cannot be cleared from the carotid artery. Ligation is only necessary in very rare cases. If a ligation of the carotid artery is required, a temporary balloon-occlusion test has to be performed before. Prolonged temporary or permanent interruption of the carotid's blood flow without compensatory circulation leads to contralateral hemiparesis.

Vascular prostheses and vessel interpositions can be applied in selected case, but this combination is usually associated with a poor prognosis. The common carotid artery bleeding can be controlled by compression against bony prominences (e.g., transversus process of the sixth cervical vertebra). The external carotid artery can be sacrificed on one side without any complications [13–15].

14.2.6 Flap Loss and Wound Healing After Treatment

Especially in previously irradiated patients with advanced fibrosis, delayed wound healing is to be expected and sometimes primary wound closure is not possible even without resection of the platysma-skin-flap. In combination with wound infection and/or fistula formation, critically prolonged healing processes may lead to arrosion of large vessels with life-threatening bleedings.

Sometimes all surgical and reconstructive attempts fail due to poor general conditions of the patients combined with a tissue breakdown [15, 16].

14.2.7 Chyle Leak

Six to seven hundred lymph nodes are drained by the thoracic duct and the right lymphatic duct. The thoracic duct is usually the main vessel transporting chyle and lymph fluid from below the diaphragm ending in a lympho-venous junction between the IJV and the subclavian vein on the left side. The right lymphatic duct, if present, transports solely lymph from the cervical and thoracic regions. However, this is not always the case, because a wide range of variations of the cervical part of the thoracic duct is reported. Only one-third of the patients have a single-sided trunk with one opening to the venous system. Multiple openings or a lymphatic plexus with one or multiple openings represent pitfalls for head and neck surgeons.

Although nearly all variations in the course and side of the thoracic duct are possible, the ending of the thoracic duct on the right side is reported to be between 1% and 5% and is even neglected in some studies [17].

Nevertheless, an unintentional and unnoticed intraoperative injury of the thoracic duct can occur in neck dissections including levels IV and V. On the one hand, care should be taken while dissecting these levels, and on the other hand, identifying of the thoracic duct is not recommended. Intraoperative abdominal compression or placing the patient in the Trendelburg position combined with temporary disconnection of the tracheal tube may help to detect chyle leak [14, 18]. The incidence of chyle fistula is below 5% in neck dissections including level IV. Chylothorax is rarer but even may occur bilaterally [19, 20].

Chyle leak can be categorized in low- or high-output fistulas. They may lead to potentially life-threatening hypovolemia, hypoalbuminemia, or electrolyte abnormalities.

If a leak is detected intraoperatively, restoration is frequently ineffective and therefore application of fibrin glue with or without muscular flaps is recommended. If the chyle leak is detected postoperatively, the therapy is usually stepwise and dominated by conservative approaches. Nutrition modification including “fat-free” or “low-fat” diet supplements, enteral products with high percentage of medium-chain triglycerides or total parenteral nutrition should initially be applied. Suction may be continued, discontinued, increased, or decreased. Additionally, octreotide (a synthetic somatostatin analog) can be administered subcutaneously [19, 21–23]. At which time surgical intervention may be considered critically depends on different parameters, such as medical condition, peak drain production, and plasma disorders. Up to 30 days may pass between the initial diagnosis of chyle leak and the surgical intervention.

If surgical intervention is required, identification and ligation of the thoracic duct alone may be difficult and additional application of fibrin glue is recommended. Moreover, some authors propose the additional us of muscle flaps. In refractory cases, thoracic duct embolization can be considered. In this approach, a contrasting agent is injected ultrasound-guided in the inguinal lymph node and a lymphangiography is performed. Afterwards, the thoracic duct is punctuated transhepatically under computer tomographic control and an embolization can be performed via catheter. This procedure is sometimes difficult, but if successful, more aggressive surgical procedures can be avoided [24, 25].

14.3 Neural Structures

14.3.1 Spinal Accessory Nerve

The spinal accessory nerve (SAN) is an important anatomic landmark in level II and level V and divides both levels in sublevel A and B. In level II, the nerve may be identified easily but frequently has contact to prominent or pathological enlarged lymph nodes (Fig. 14.1). In the posterior triangle (level V), the anatomic course is frequently more superficial than anticipated by most surgeons. Moreover, the relationship of the SAN to the sternocleidomastoideus muscle can differ

considerably. The branches of the SAN innervating the trapezius muscle can leave sternocleidomastoid muscle after an intramuscular course or dorsally pass the muscle. Both is usually cranially from the Erb point with a distance range between the Erb point and the SAN's cranial passing from 0 to 3.8 cm as reported in an anatomic study. In another anatomic study, the distance between the SAN's cranial passing and the clavicula was measured. A distance of 8.2 cm ± 1.01 cranially was observed [26, 27].

Level II is part of every neck dissection and the SAN is at risk in every procedure. Sacrifice or injury may lead to a temporary or permanent shoulder syndrome which can be painful and is characterized by: (1) drooping of the shoulder, (2) limited forward flexion of the shoulder, (3) limited lateral abduction, (4) rotation of the scapula, and (5) electromyographic changes (Fig. 14.2). Interestingly, shoulder syndrome does not occur in all RNDs where per definition the SAN is resected. Severe clinical problems can be observed in 60–80% after RND. Others describe rates of up to 25% of normal shoulder function after RND by electromyography (EMG) [28, 29]. This discrepancy is one of the reasons, why the innervation of trapezius muscle is still discussed controversial. Some authors consider the SAN as the only motor supply to the trapezius muscle. However, a normal shoulder function after RND may also be explained by branches of the cervical plexus running to the transverse and ascending part of the trapezius muscle. Various courses of these cervical plexus branches were previously described. Frequently two, more seldom three branches run subfascially to the ventral surface of transverse part of the trapezius muscle and merge with the SAN. Sometimes this union occurs more caudal to this description. Additionally, in the same study also only one single branch of the SAN without anastomosis was found to run to the descending part of the trapezius muscle [26, 30].

In nerve-sparing operations, it is plausible that the rate of shoulder syndrome is higher if the SAN is completely exposed in level V. Clinically evident shoulder droop occurs in about 5% in SND without level V and in 30% in nerve-sparing operations with dissection of level V. However, in the same study, changes in the EMG without clinically evident shoulder syndrome are reported to be much higher. This was 40% in the group without dissection of level V and 85% in the group with dissection of level V, respectively [31]. Some studies report exceptionally better outcomes in nerve-sparing MRND, whereas other report a higher rate of shoulder complication in SND. For adequate interpretation of these studies, it is important to take the differently defined outcome parameters into consideration.

Fig. 14.2 A patient 2.5 years after the treatment of a cT2N2bM0 (TNM classification 7th edition) squamous cell carcinoma of the oral cavity including a radical neck dissection with persisting palsy of the right spinal accessory nerve: (left) drooping of the shoulder and atrophy of the pars superior of the trapezius muscle; (middle) limited lateral abduction below 90°; (right) limited forward flexion of the shoulder

True severe shoulder impairment with arm abduction below 90° with corresponding total denervation is comparable low in almost all studies. Other clinical signs and different categorization of mild impairment lead to varying results. These should not be compared, because reports about single symptoms such as shoulder pain and results of questionnaires can differ considerably [28, 29, 32].

Traction, skeletonization, devascularization, and frequent electrostimulation of the SAN should be avoided. If the nerve is injured unintentionally or as a clinical requirement, the risk for a shoulder syndrome should be minimized by preserving the branches from the cervical plexus serving the trapezius muscle. Grafting with nerve interpositions (e.g., great auricular nerve) can be considered in selected cases [29, 33].

Once paresis of the SAN is evident, the time until nerve recovery occurs critically depends on the type of damage. Neurotmesis has the poorest potential for recovery. Axonotmesis represents different degrees of axonal injury and may recover within 18 months. Posttraumatic demyelination results in neurapraxia and a short-term dysfunction with recovery after weeks is likely [34].

Most authors state that physiotherapy should be applied immediately to patients when paresis of the SAN is evident. Questionnaires and clinical tests are recommended in this situation. Although physiotherapy is well tolerated in these patients, its beneficial effect remains unclear due to lacking prospective clinical trials [29, 34].

14.3.2 Other Neural Structures

In extended neck dissection (END) per definition, additional structures to that already sacrificed in RND (IVJ, SAN, sternocleidomastoideus muscle) are resected. This involves particularly all other caudal cranial nerves and depends on the extent of disease. If END is necessary, it is usually decided intraoperatively and cannot be assessed before by common imaging modalities. However, EMG changes or clinically evident palsy make an infiltration of nerval structures more likely. The great auricular nerve is usually not infiltrated by tumor but frequently sacrificed to get a better overview in the operation field. Especially in SND, the great auricular nerve can be spared without limitation of the surgical procedure (Fig. 14.1). This results in a significant better quality of life of the patients, because numbness of the skin is a frequent postoperative complaint by patients.

In nerve-sparing operations, unintended injury of other important nerves than the previously mentioned SAN is unusual. For example, lesions of the marginal branch of the 7th nerve, the 12th nerve, and the sympathetic trunk were 1.26%, 0.42%, and 0.56%, respectively [35].

Once they occur, complications by lesions of the individual nerves are as follows:

- *Marginal branch of the facial nerve:* This palsy is frequently self-limiting and usually not associated with severe problems. Facial impairment may influence speech and eating. Correct placement of retractors and awareness of ptosis of the submandibular gland in older patients may help to minimize this complication.
- *9th cranial nerve:* Lesions of the glossopharyngeal nerve result in difficulty in swallowing and taste, but similar regions are innervated by the vagus nerve. This nerve is rarely identified in cervical surgery.
- *10th cranial nerve:* Loss of one vagus nerve is fully compatible with life due to a high number of anastomoses. Movement of the vocal cords points to the nerve's function. On the other hand, plegia of both vagus nerves is not compatible with life.
- *12th cranial nerve:* Lesions of hypoglossal nerve are characterized by tongue movement towards the side of the lesion and may lead to problems with food intake and speech articulation.
- *Brachial plexus:* Lesions are very rare, but very serious once they occur. Surgeons should be aware that the deep cervical fascia is an excellent cleavage plane.
- *Phrenic nerve:* Monolateral plegia is well tolerated and usually not associated with important clinical consequences. In the spirometry, a

25% decrease of tidal volumes can be observed. Bilateral palsy of the phrenic nerve is very rare and requires permanent assisted ventilation.

– *Bernard-Horner's syndrome:* Occurrence of this syndrome is associated with the dissection of retropharyngeal lymph nodes, RND, and surgery in or around the carotid sheath. The typical ophthalmologic triad of ptosis, miosis, and enophthalmos are the key symptoms of this syndrome. Currently, no specific treatment is available.

References

1. Crile G. III. On the technique of operations upon the head and neck. Ann Surg. 1906;44:842–50.
2. Pagedar NA, Gilbert RW. Selective neck dissection: a review of the evidence. Oral Oncol. 2009;45:416–20.
3. D'Cruz AK, Vaish R, Kapre N, et al. Elective versus therapeutic neck dissection in node-negative oral cancer. N Engl J Med. 2015;373:521–9.
4. Ferlito A, Silver CE, Rinaldo A. Neck dissection: present and future? Eur Arch Otorhinolaryngol. 2008;265:621–6.
5. Ferlito A, Corry J, Silver CE, et al. Planned neck dissection for patients with complete response to chemoradiotherapy: a concept approaching obsolescence. Head Neck. 2010;32:253–61.
6. Mehanna H, Wong WL, Mcconkey CC, et al. PET-CT surveillance versus neck dissection in advanced head and neck cancer. N Engl J Med. 2016;374(15):1444–54.
7. Goguen LA, Chapuy CI, Li Y, et al. Neck dissection after chemoradiotherapy: timing and complications. Archiv Otolaryngol Head Neck Surg. 2010;136:1071–7.
8. Streiff MB. Thrombosis in the setting of cancer. Hematol Am Soc Hematol Educ Program. 2016;2016:196–205.
9. Gueret G, Cosset MF, Mcgee K, et al. Sudden death after neck dissection for cancer. Ann Otol Rhinol Laryngol. 2002;111:115–9.
10. Chang CF, Kuo YL, Pu C, et al. Neck dissection and stroke in patients with oral cavity cancer: a population-based cohort study. Head Neck. 2017;39:63–70.
11. Gorphe P, Tao Y, Blanchard P, et al. Relationship between the time to locoregional recurrence and survival in laryngeal squamous-cell carcinoma. Eur Arch Otorhinolaryngol. 2017;274:2267–71.
12. Agrama MT, Reiter D, Cunnane MF, et al. Nodal yield in neck dissection and the likelihood of metastases. Otolaryngol Head Neck Surg. 2003;128:185–90.
13. Kerawala CJ. Complications of head and neck cancer surgery—prevention and management. Oral Oncol. 2010;46:433–5.
14. Kerawala CJ, Heliotos M. Prevention of complications in neck dissection. Head Neck Oncol. 2009;1:35.
15. Chen YJ, Wang CP, Wang CC, et al. Carotid blowout in patients with head and neck cancer: associated factors and treatment outcomes. Head Neck. 2015;37:265–72.
16. Cleland-Zamudio SS, Wax MK, Smith JD, et al. Ruptured internal jugular vein: a postoperative complication of modified/selected neck dissection. Head Neck. 2003;25:357–60.
17. Smith ME, Riffat F, Jani P. The surgical anatomy and clinical relevance of the neglected right lymphatic duct: review. J Laryngol Otol. 2013;127:128–33.
18. Cernea CR, Hojaij FC, De Carlucci D Jr, et al. Abdominal compression: a new intraoperative maneuver to detect chyle fistulas during left neck dissections that include level IV. Head Neck. 2012;34:1570–3.
19. Ahn D, Sohn JH, Jeong JY. Chyle fistula after neck dissection: an 8-year, single-center, prospective study of incidence, clinical features, and treatment. Ann Surg Oncol. 2015;22(Suppl 3):S1000–6.
20. Prabhu V, Passant C. Left-sided neck dissection and chylothorax: a rare complication and its management. J Laryngol Otol. 2012;126:648–50.
21. Gregor RT. Management of chyle fistulization in association with neck dissection. Otolaryngol Head Neck Surg. 2000;122:434–9.
22. Smoke A, Delegge MH. Chyle leaks: consensus on management? Nutr Clin Pract. 2008;23:529–32.
23. Swanson MS, Hudson RL, Bhandari N, et al. Use of octreotide for the management of chyle fistula following neck dissection. JAMA Otolaryngol Head Neck Surg. 2015;141:723–7.
24. Chen CY, Chen YH, Shiau EL, et al. Therapeutic role of ultrasound-guided intranodal lymphangiography in refractory cervical chylous leakage after neck dissection: report of a case and review of the literature. Head Neck. 2016;38:E54–60.
25. Patel N, Lewandowski RJ, Bove M, et al. Thoracic duct embolization: a new treatment for massive leak after neck dissection. Laryngoscope. 2008;118:680–3.
26. Kierner AC, Zelenka I, Heller S, et al. Surgical anatomy of the spinal accessory nerve and the trapezius branches of the cervical plexus. Arch Surg. 2000;1960(135):1428–31.
27. Salgarelli AC, Landini B, Bellini P, et al. A simple method of identifying the spinal accessory nerve in modified radical neck dissection: anatomic study and clinical implications for resident training. Oral Maxillofac Surg. 2009;13:69–72.
28. Umeda M, Shigeta T, Takahashi H, et al. Shoulder mobility after spinal accessory nerve-sparing modified radical neck dissection in oral cancer patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2010;109:820–4.
29. Cappiello J, Piazza C, Nicolai P. The spinal accessory nerve in head and neck surgery. Curr Opin Otolaryngol Head Neck Surg. 2007;15:107–11.
30. Kierner AC, Zelenka I, Burian M. How do the cervical plexus and the spinal accessory nerve contribute to

the innervation of the trapezius muscle? As seen from within using Sihler's stain. Archiv Otolaryngol Head Neck Surg. 2001;127:1230–2.
31. Cappiello J, Piazza C, Giudice M, et al. Shoulder disability after different selective neck dissections (levels II-IV versus levels II-V): a comparative study. Laryngoscope. 2005;115:259–63.
32. Dijkstra PU, Van Wilgen PC, Buijs RP, et al. Incidence of shoulder pain after neck dissection: a clinical explorative study for risk factors. Head Neck. 2001;23:947–53.
33. Bradley PJ, Ferlito A, Silver CE, et al. Neck treatment and shoulder morbidity: still a challenge. Head Neck. 2011;33:1060–7.
34. Mcgarvey AC, Chiarelli PE, Osmotherly PG, et al. Physiotherapy for accessory nerve shoulder dysfunction following neck dissection surgery: a literature review. Head Neck. 2011;33:274–80.
35. Prim MP, De Diego JI, Verdaguer JM, et al. Neurological complications following functional neck dissection. Eur Arch Otorhinolaryngol. 2006;263:473–6.

Complications in Free Flap Reconstruction

15

Christos Perisanidis, Lorenz Kadletz, and Boban M. Erovic

Contents

15.1 Introduction

Over the past decades, microvascular free-tissue transfer for head and neck reconstruction has become a standardized procedure, particularly following head and neck cancer ablative surgery. Microvascular free flaps offer a tremendously broad variety of reconstructive options for virtually any type of defect in the head and neck area [1]. Free flap reconstruction has shown a success rate of over 90% and high volume centers have been able to reduce flap failure rates below 3% [2, 3]. Nevertheless, complications in microvascular surgery do occur even in the most

C. Perisanidis (✉)
Department of Oral and Maxillofacial Surgery, Dental School, University of Athens, Athens, Greece
e-mail: cperis@dent.uoa.gr; christos.perisanidis@meduniwien.ac.at

L. Kadletz
Department of Otorhinolaryngology, Head and Neck Surgery, Medical University of Vienna, Vienna, Austria
e-mail: lorenz.kadletz@meduniwien.ac.at

B. M. Erovic
Institute of Head and Neck Diseases, Evangelical Hospital Vienna, Vienna, Austria
e-mail: b.erovic@ekhwien.at

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_15

experienced hands and lead to prolonged hospital stay, increased costs, and delays to adjuvant cancer treatment.

15.2 Classification of Complications in Free Flap Surgery

Complications of free flap reconstructive surgery can be classified into three categories: those involving the recipient site, those involving the donor site, and medical complications. Recipient site complications are related to both microvascular anastomosis as well as defect reconstruction and consist of vascular thrombosis, partial or total flap loss, orocutaneous or pharyngocutaneous fistulae, wound dehiscence, surgical site infection, and hematoma and seroma formation. Donor site complications may occur during harvesting of the free flap and include wound dehiscence, surgical site infection, hematoma, seroma, scar formation, and skin graft failure. Medical complications include respiratory, neurological, cardiovascular, renal, and gastrointestinal complications, as well as need for transfusion, multiple organ failure, and death.

15.3 Risk Factors for Complications in Microvascular Surgery

Risk stratification in free flap surgery helps to provide sufficient information to the patient and accordingly determine realistic expectations of clinical outcome. Risk factors for complications in microvascular surgery can be related to either patients' general status or the surgical procedure and technique.

Preoperative evaluation includes assessment of patients' general condition and medical clearance for a prolonged surgical procedure. History and physical examination of donor site is required to exclude previous trauma or surgery to the intended flap or its vascular pedicle. Careful evaluation of recipient site is needed to determine availability and quality of recipient vessels taking into account prior neck dissection or radiation therapy.

Patients requiring microvascular reconstruction of the head and neck often present with a broad variety of risk factors including tobacco smoking, advanced age, and radiation therapy, as well as medical comorbidities including diabetes mellitus, coagulopathy, venous insufficiency, and peripheral artery disease. Tobacco smoking and nicotine have shown a deleterious impact in replantation surgery; however, their negative impact on free flap survival has been discussed controversially in the literature [4]. Ehrl and colleagues retrospectively examined over 900 patients undergoing microvascular surgery and could not find a significant link between smoking and higher risk of flap failure after multivariate analysis [5]. However, smoking has been associated with wound dehiscence and surgical site infection at both the donor and recipient site. In particular, patients with smoking history undergoing reconstruction with an anterolateral thigh flap showed a significantly higher rate of delayed wound healing at the donor site [6]. Moreover, smokers undergoing mandibular reconstruction with a free fibula flap showed a higher rate of local wound infection and plate exposure [7]. Taken together, we recommend that patients should refrain from smoking at least 2 weeks before and 2 weeks after free flap surgery.

Advanced age is neither a contraindication nor a predictor of complications in free flap surgery as long as cardiac and respiratory fitness allows the patient to tolerate the long and extensive surgery. Piazza and colleagues demonstrated that patients over 65 years of age undergoing microvascular surgery did not have a significantly higher rate of major complications, including complications that require intensive care or surgery under general anesthesia; however, those patients have a significantly higher rate of minor complications [8]. Furthermore, restoring adequate function after ablative and reconstructive surgery has been shown to be a more challenging issue in elderly patients compared to younger ones. In particular, it has been found that 25% of patients over 70 years with advanced head and neck cancer did not fully recover from dysphagia

while their swallowing function remained poor after free flap surgery [9].

There is strong evidence suggesting that radiation therapy is associated with an increased risk of free flap failure. Radiotherapy induces inflammation within the connective tissue and vessels eventually leading to thrombosis of small vessels [10]. After radiation, the extracellular matrix becomes impaired due to significant fibrosis resulting in a decrease of cellular regeneration [11]. Herle and colleagues performed a meta-analysis comparing the postoperative outcome in 2842 flaps performed in irradiated fields and 3491 flaps performed in nonirradiated fields [12]. The study showed that patients with previous radiotherapy had a significantly higher rate of free flap complications and flap failure. Moreover, the meta-analysis demonstrated a link between a radiation dosage of over 60 Gy and a higher rate of postoperative complications [12].

Diabetes mellitus has been strongly linked to microvascular disorders and thus patients with diabetes are strongly prone to infections and wound healing disorders. In particular, an experimental model has confirmed that untreated hyperglycemia significantly increases the risk of thrombosis at the venous anastomotic site [13]. Moreover, it has been shown that patients with diabetes mellitus undergoing microvascular reconstruction of the head and neck are more likely to suffer postoperatively from severe bleeding, pneumonia, prolonged ventilation, and myocardial infarction [14]. In the preoperative setting, we recommend testing of blood sugar and HbA1c levels for diagnosis of diabetes mellitus.

History of coagulopathy resulting in a hypercoagulable state (i.e., polycythemia) is a definitive contraindication for free tissue transfer. Relative contraindications include severe obesity, connective tissue disorders, venous insufficiency, and peripheral artery disease. In particular, peripheral artery disease represents a contraindication for microvascular reconstruction of osseous or oseocutaneous defects with a fibula flap. Preoperative assessment of the vascular supply of the lower limb is critical in order to detect potential insufficiencies of the arterial system or the presence of a peroneal magna artery. Ideally, either magnetic resonance angiography or digital subtraction angiography should to be performed prior to fibula harvesting [15].

Complications in free flap surgery may be attributed to numerous surgery-related risk factors: poor anastomosis technique [16], vessel size mismatch, poor-quality of recipient vessels, compression or twisting of anastomosis or pedicle, short pedicle necessitating anastomosis under tension, vasospasm of pedicle, surgical infection and bleeding at recipient-anastomosis site, and mismatch between type and amount of missing tissue and selected free flap. Particularly, selection of the appropriate flap with respect to its composition, size, thickness, color, and function should be based on: (1) location, type, and amount of missing tissue; (2) required pedicle length; and (3) donor site morbidity [17, 18].

15.4 Recipient Site Complications

15.4.1 Vascular Thrombosis

Vascular occlusion following arterial or venous thrombosis at the recipient site remains a major cause for free flap failure in head and neck reconstructive surgery. Beside the traumatic experience of flap failure for both the patient and reconstructive surgeon, necessary adjuvant treatment has to be postponed while the options for sequential reconstruction are getting more limited.

During the acute phase, the elements comprising Virchow's triad, including hypercoagulability, stasis, and endothelial injury, significantly contribute to thrombus formation. Thrombosis of the arterial or venous anastomosis occurs in about 4% of all patients undergoing microvascular surgery [19], while the incidence of venous thrombosis is double compared to arterial thrombosis. The reason for this is that veins are more prone to platelet-complex formation due to the low pressure and subsequent low blood flow in the venous system. After 3 days, the vascular interaction between the flap and adjacent tissue is becoming more and more evident. Thrombosis after this time period is usually the result of infection or

fistulae formation as inflammation induces a prothrombotic state within the tissue and vessels. Another cause of vascular occlusion may be a spasm of arterial vessels induced by manipulation during the anastomosis, placement of microvascular clamps or hypothermia. Technical abilities of the surgeon are key since meticulous preparation of the vessels and suturing of the anastomosis is necessary to keep complication rates at a low level.

15.4.2 Monitoring

Flaps need to be tightly monitored after surgery and a standardized protocol should be used. Arterial occlusion leads to whitening of the skin island (Fig. 15.1) while the temperature of the flap decreases. However, temperature is not a reliable sign in case of intraoral reconstruction due the ambient temperature of the oral cavity. It is also possible to detect a prolongation of the capillary refill time. In addition, no bleeding can be observed or will be delayed when a scratch test is performed. Clinical signs of a partial or total venous compromise are a livid coloration of the flap itself or the skin island (Figs. 15.2 and 15.3). The scratch test will show a very fast discharge of darkened blood. Doppler ultrasound and duplex color investigation can be used to

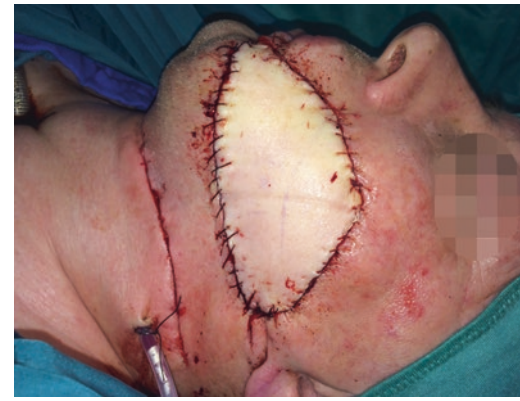


Fig. 15.1 Arterial occlusion due to pedicle compression results in whitening of the skin island

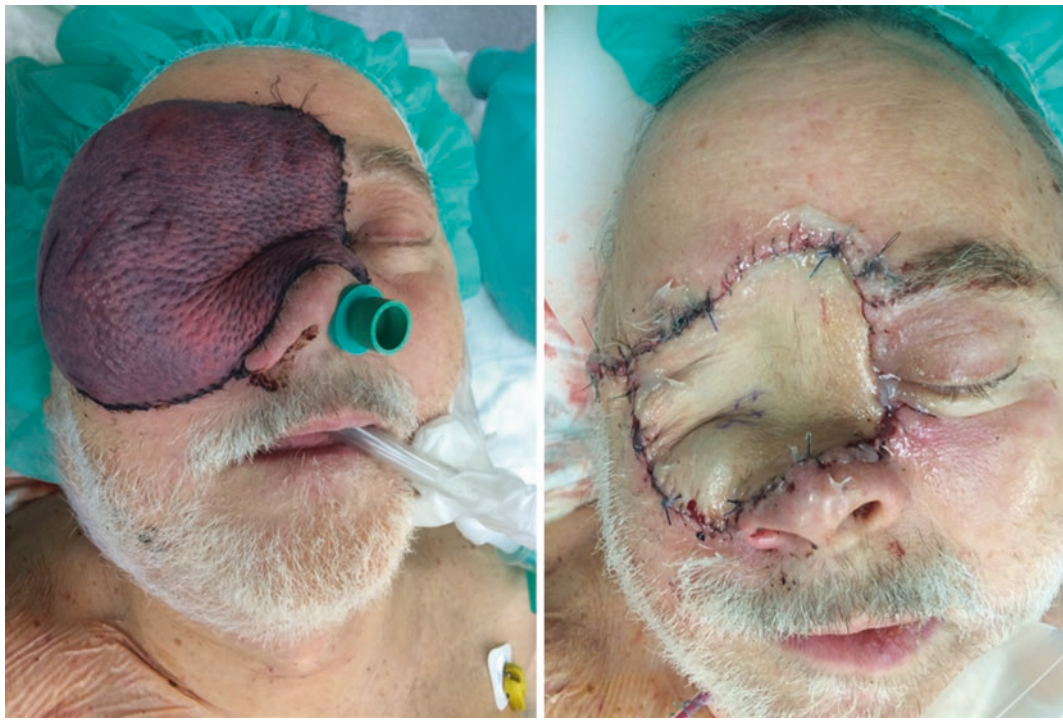


Fig. 15.2 Venous thrombosis of ALT flap (left) followed by flap removal and second microvascular reconstruction with radial forearm flap (right)

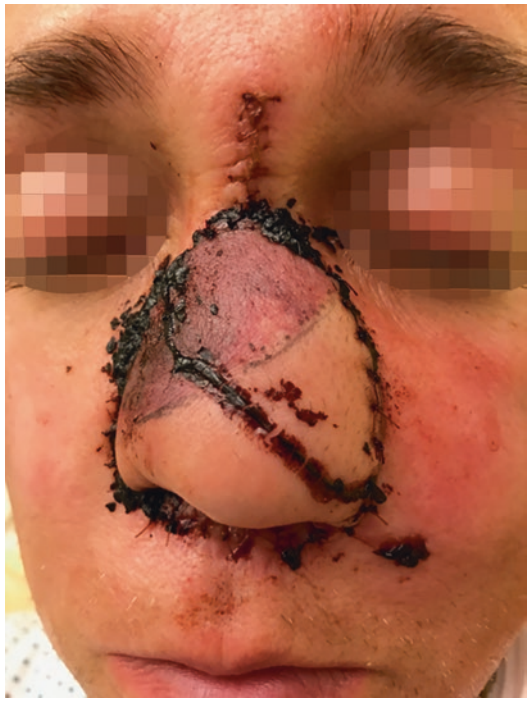


Fig. 15.3 Partial venous compromise of radial forearm flap used for total nose reconstruction

detect arterial and venous occlusion within the pedicle. However, there may be a false positive signal if the anastomosis is too adjacent to the internal jugular vein or the carotid artery. Implantable Doppler systems have been developed over the years to avoid these kinds of false positive signals [20]. Monitoring of capillary glucose and lactate levels in buried flaps has shown promising results in order to diagnose flap failure at an early stage [21]. Tight dressing around the neck with drains close to the vascular pedicle and non-sutured but tied tracheal tube may lead to a significant pressure on the vascular pedicle that subsequently may result in thrombus formation. Hematoma formation should be ruled out by palpation, ultrasound, or careful opening of some stiches.

15.4.3 Reexploration

If there are no obvious causes for vascular compromise of the pedicle, immediate surgical reexploration is indicated. After evaluation of any torsion of the pedicle or misplacement of suction tubes, the vessels are examined for thrombosis. Venous thrombosis occurs more frequently and nearly two-thirds of all flaps can be salvaged as venous congestion can be tolerated longer than arterial occlusion. Reopening of the anastomosis is necessary in case of a missing arterial or venous flow. Vascular spasm may be treated with papaverine rinsing and careful dilation using a Fogarty catheter or dilatator. Excessive flushing with heparinized saline and trimming of the edges should always be performed prior to reanastomosis. In case of increased tension due to a short donor vessel, it is preferable to look for another recipient vessels prior to harvest a vein graft. Vein grafts are prone to re-thrombosis and subsequently this lead to a higher free flap failure rate. In case of venous reanastomosis, we suggest to perform an end-to-side anastomosis to the internal jugular vein as this vessel due its large caliber has the largest back flow. Besides surgical intervention thrombolytic agents such as urokinase, streptokinase, or recombinant tissue plasminogen activator can be used to reestablish vessel perfusion.

15.5 Other Complications at the Recipient Site

Surgical site complications at the recipient site are frequently encountered after reconstruction of the head and neck region with free flaps. In particular, surgical site infection (Fig. 15.4) occurs in about

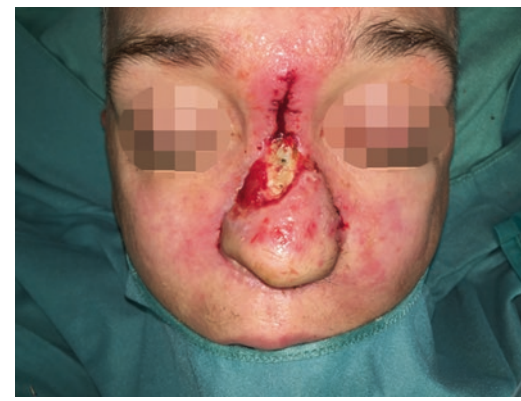


Fig. 15.4 Surgical site infection of radial forearm flap following partial necrosectomy

15% of patients after head and neck reconstruction and may be attributed to the clean-contaminated field and the exposure to the microbiome of the upper aerodigestive tract [22]. When looking at the oral microbiome, *Staphylococcus aureus*, *Enterococcus faecalis*, and Gram-negative bacilli like *Pseudomonas aeruginosa* are most commonly the causes of surgical site infection [23]. There is a strong recommendation for perioperative use of antibiotics. For antibiotic prophylaxis in patients with no history of penicillin allergy, either ampicillin/sulbactam or amoxicillin/clavulanate should be used. Ideally intravenous administration of antibiotics should be done 60 min prior to surgery in order to achieve high serum levels at the beginning of surgery.

The formation of fistulae and wound dehiscence presents a challenging situation after microvascular reconstruction [24]. The incidence of fistulae and dehiscence varies between 8 and 20% [25]. In particular, patients with reconstruction of the pharynx have a higher risk of developing fistulae because the newly created pharynx is highly exposed to the aggressive saliva. Moreover, patients with prior radiotherapy have a significantly increased risked of developing fistulae and wound dehiscence due to the presence of hypovascularized and fibrotic tissue. Small dehiscence and fistulae can be managed conservatively using 3% sodium chloride solution rinsing and the anticholinergic glycopyrrolate (Robinul®). Glycopyrrolate can be used to minimize salivary production and reduce the acidity of the gastric secretions through blocking of muscarinic receptors. When fistulae cannot be closed by conservative approaches, well vascularized tissue needs to be transferred to achieve adequate wound healing. Depending on the vascular status, either another free flap or a pedicled flap (e.g., pectoralis major flap) should be used for coverage [26, 27].

15.6 Donor Site Complications

Complications at the donor site may have severe consequences for the patient. The most frequently observed donor site complications are wound dehiscence and surgical site infection

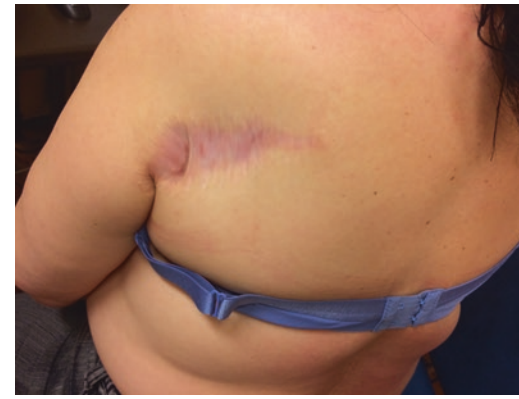


Fig. 15.5 Scar formation following harvesting of parascapular flap

followed by hematoma, seroma, and scar formation (Fig. 15.5).

Donor site complications may happen during harvesting of the free flap. In particular for the radial forearm flap, as the radial artery has to be sacrificed, the vascular supply of the hand depends only on the ulnar artery. The Allen test is an excellent clinical test to preoperatively assess the vascular supply by the ulnar artery alone. Woods and coworkers found no ischemic events in patients with a positive Allen test [28]. However, if during surgery the hand shows signs of arterial undersupply after clamping the radial artery, another donor site has to be chosen. The distal superficial branches of the radial nerve may be injured during flap harvesting [29]. Tendon exposure is the result of failed wound healing and is preventable by suprafascial preparation of the flap. In case of tendon exposure, wound management and physiotherapy have to be initiated as soon as possible [30].

Segmental mandibulectomy or maxillectomy require primary bone reconstruction either with iliac crest, scapula or fibula free flap. Although harvesting of the fibula flap is a standardized and safe procedure, donor-site complications such as bleeding and ischemic complications at the donor leg may occur. Mild bleedings can be managed with dressings whereas persistent or severe bleeding requires immediate hemostasis in the operating room [31]. Arterial undersupply due to relevant stenosis or anatomic abnormalities and development of acute compartment syndrome are

very rare invents [32]. However, in case of clinical signs, immediate surgical reexploration of the compartment is indicated. Li and colleagues report that 57% of their patients suffer from loss of sensation at the calf and toes and due to injury to the peroneal nerve or drop foot and muscle weakness of the affected lower limb due to palsy of the peroneal nerve [33]. Functional problems should be prevented/treated with early physiotherapeutic exercises while partial weight bearing is allowed 4–7 days after surgery.

15.7 Postoperative Management

A key factor for free flap survival is the adequate postoperative management. Patient's poor cardiovascular condition may necessitate intraoperative and/or postoperative use of vasoactive drugs. Vasoconstrictors (e.g., norepinephrine) are commonly used to maintain hemodynamic stability and may lead to vasospasm and vascular thrombosis of the flap pedicle. Vasodilators are used to maintain cardiac output and may decrease blood flow at the anastomosis site. Flap perfusion may be supported with mild hypervolemia and a hematocrit value of approximately 35%. In addition, peri- and postoperative use of heparin has been intensively investigated. Animal studies have shown that low-molecular-weight heparin may improve free flap survival; however, clinical studies could not show any statistical significant benefit for low-molecular-weight heparin or unfractionated heparin [34]. A meta-analysis done by Lee and Mun has demonstrated that there is a lack of evidence for the use of antithrombotics after microvascular reconstruction [35]. Nevertheless, prospective data of randomized controlled trials are missing. Papaverine, a curare derivate that induces vasodilatation, has shown to be effective in preventing vascular spasm [36].

15.8 Conclusion

Microvascular surgery and avoidance of complications start with meticulous preoperative planning. Every patient has to be evaluated regarding potential risk factors for free flap surgery. Early free flap complications are mainly caused by thrombotic events in veins and prompt detection is of utmost importance to increase the chances of flap salvage. The most common late free flap complications are linked to surgical site infections and wound healing deficiencies. Thus, early detection and adequate treatment of complications could prevent free flap loss and prolonged hospital stay and could improve patients' quality of life.

References

1. Erovic BM, Lechner P. Manual of head and neck reconstruction using regional and free flaps. Berlin: Springer; 2016.
2. Celik N, Wei F-C, Chen H-C, et al. Osteoradionecrosis of the mandible after oromandibular cancer surgery. Plast Reconstr Surg. 2002;109:1875–81.
3. Buchbinder D, St Hilaire H. The use of free tissue transfer in advanced osteoradionecrosis of the mandible. J Oral Maxillofac Surg. 2006;64:961–4.
4. Vanadrichem L, Hovius S, Vanstrik R, et al. The acute effect of cigarette-smoking on the microcirculation of a replanted digit. J Hand Surg Am. 1992;17A:230–4.
5. Ehrl D, Heidekrueger PI, Ninkovic M, et al. Effect of preoperative medical status on microsurgical free flap reconstructions: a matched cohort analysis of 969 cases. J Reconstr Microsurg. 2018;34:170–5.
6. Abe Y, Kashiwagi K, Ishida S, et al. Risk factors for delayed healing at the free anterolateral thigh flap donor site. Arch Plast Surg. 2018;45:51–7.
7. Chen Y, Wu J, Gokavarapu S, Shen Q, Ji T. Radiotherapy and smoking history are significant independent predictors for osteosynthesis-associated late complications in vascular free fibula reconstruction of mandible. J Craniofac Surg. 2017;28:1508–13.
8. Piazza C, Grammatica A, Paderno A, et al. Microvascular head and neck reconstruction in the elderly: The University of Brescia experience. Head Neck. 2016;38:E1488–92.
9. Worley ML, Graboyes EM, Blair J, et al. Swallowing outcomes in elderly patients following microvascular reconstruction of the head and neck. Otolaryngol Head Neck Surg. 2018;127:019459981876516.
10. Paderno A, Piazza C, Bresciani L, et al. Microvascular head and neck reconstruction after (chemo)radiation: facts and prejudices. Curr Opin Otolaryngol Head Neck Surg. 2016;24:83–90.
11. Nevens D, Duprez F, Daisne JF, et al. Radiotherapy induced dermatitis is a strong predictor for late fibrosis in head and neck cancer. The development of a predictive model for late fibrosis. Radiother Oncol. 2017;122:212–6.

12. Herle P, Shukla L, Morrison WA, et al. Preoperative radiation and free flap outcomes for head and neck reconstruction: a systematic review and meta-analysis. ANZ J Surg. 2015;85:121–7.
13. Colen LB, Stevenson A, Sidorov V, et al. Microvascular anastomotic thrombosis in experimental diabetes mellitus. Plast Reconstr Surg. 1997;99:156–62.
14. Brady JS, Govindan A, Crippen MM, et al. Impact of diabetes on free flap surgery of the head and neck: a NSQIP analysis. Microsurgery. 2017;37:438.
15. Klein S, Van Lienden KP, Veer MV, et al. Evaluation of the lower limb vasculature before free fibula flap transfer. A prospective blinded comparison between magnetic resonance angiography and digital subtraction angiography. Microsurgery. 2013;33:539–44.
16. Grewal AS, Erovic B, Strumas N, et al. The utility of the microvascular anastomotic coupler in free tissue transfer. Can J Plast Surg. 2012;20:98–102.
17. Haymerle G, Enzenhofer E, Lechner W, et al. The effect of adjuvant radiotherapy on radial forearm free flap volume after soft palate reconstruction in 13 patients. Clin Otolaryngol. 2018;43:742–5.
18. Higgins KM, Erovic BM, Ravi A, et al. Volumetric changes of the anterolateral thigh free flap following adjuvant radiotherapy in total parotidectomy reconstruction. Laryngoscope. 2012;122:767–72.
19. Chiu Y-H, Chang D-H, Perng C-K. Vascular complications and free flap salvage in head and neck reconstructive surgery: analysis of 150 cases of reexploration. Ann Plast Surg. 2017;78:S83–8.
20. Hosein RC, Cornejo A, Wang HT. Postoperative monitoring of free flap reconstruction: a comparison of external Doppler ultrasonography and the implantable Doppler probe. Plast Surg (Oakv). 2016;24:11–9.
21. David G, Vivien M, Sarra C, et al. Monitoring of myocutaneous flaps by intracapillary glucose and lactate measurements: experimental study. Clin Pract. 2017;14:123–32.
22. Khariwala SS, Le B, Vogel RI, et al. Antibiotic use after free tissue reconstruction of head and neck defects: short course vs. long course. Surg Infect (Larchmt). 2016;17:100–5.
23. Cannon RB, Houlton JJ, Mendez E, et al. Methods to reduce postoperative surgical site infections after head and neck oncology surgery. Lancet Oncol. 2017;18:e405–13.
24. Parzefall T, Wolf A, Czeiger S, et al. Effect of postoperative use of diclofenac on pharyngocutaneous fistula development after primary total laryngopharyngectomy: results of a single-center retrospective study. Head Neck. 2016;38(Suppl 1):E1515–20.
25. Bin Do S, Chung CH, Chang YJ, et al. Risk factors of and treatments for pharyngocutaneous fistula occurring after oropharynx and hypopharynx reconstruction. Arch Plast Surg. 2017;44:530–8.
26. Khan MN, Rodriguez LG, Pool CD, et al. The versatility of the serratus anterior free flap in head and neck reconstruction. Laryngoscope. 2017;127:568–73.
27. Higgins KM, Ashford B, Erovic BM, et al. Temporoparietal fascia free flap for pharyngeal coverage after salvage total laryngectomy. Laryngoscope. 2012;122:523–7.
28. Wood JW, Broussard KC, Burkey B. Preoperative testing for radial forearm free flaps to reduce donor site morbidity. JAMA Otolaryngol Head Neck Surg. 2013;139:183–6.
29. Richardson D, Fisher SE, Vaughan ED, et al. Radial forearm flap donor-site complications and morbidity: a prospective study. Plast Reconstr Surg. 1997;99:109–15.
30. David C, Shonka J, Kohli NV, et al. Suprafascial harvest of the radial forearm free flap decreases the risk of postoperative tendon exposure. Ann Otol Rhinol Laryngol. 2017;126:224–8.
31. Sood A, Granick MS, Tomaselli NL. Wound dressings and comparative effectiveness data. Adv Wound Care. 2016;3:511–29.
32. Kerrary S, Schouman T, Cox A, et al. Acute compartment syndrome following fibula flap harvest for mandibular reconstruction. J Craniomaxillofac Surg. 2011;39:206–8.
33. Li P, Fang Q, Qi J, et al. Risk factors for early and late donor-site morbidity after free fibula flap harvest. J Oral Maxillofac Surg. 2015;73:1637–40.
34. Couteau C, Rem K, Guillier D, et al. Improving free-flap survival using intra-operative heparin: ritualistic practice or evidence-base medicine? A systematic review. Ann Chir Plast Esthet. 2017;63(3):e1–5.
35. Lee KT, Mun GH. The efficacy of postoperative antithrombotics in free flap surgery: a systematic review and meta-analysis. Plast Reconstr Surg. 2015;135:1124–39.
36. Gherardini G, Gürlek A, Cromeens D, et al. Drug-induced vasodilation: in vitro and in vivo study on the effects of lidocaine and papaverine on rabbit carotid artery. Microsurgery. 1998;18:90–6.

Complications from Radiotherapy

16

Robert Gassner

Contents

16.1 Introduction

The armamentarium treating oral cancer involves surgery, radiotherapy, chemotherapy, immunotherapy and its combinations. While radiotherapy is one of the powerful options to treat oral cancer successfully the specialty of CMF surgery is challenged when severe complications occur due to radiotherapy.

Known consequences of radiotherapy are explained to patients prior to treatment such as radiogenic mucositis, radiogenic-induced xerostomia, radiogenic-induced trism (Fig. 16.1a, b), radiogenic dysphagia, radiogenic caries (Fig. 16.2) and radiogenic effects on jaw and tooth development (Fig. 16.3) [1].

More severe complications are the loss of jaw and TMJ function such as osteoradionecrosis which may further lead to infected osteoradionecrosis, pathologic fractures and fistulas or radiation-induced stenosis of carotid vessels [2].

R. Gassner (✉)
Department of CMF and Oral Surgery, Medical University of Innsbruck, Innsbruck, Austria
e-mail: robert.gassner@tirol-kliniken.at

Especially long-term cancer survivors may face situations due to complications from successful radiotherapy hampering their quality of life.

Fractured and infected necrotic bones of the mandibular body with fistulas as a complication from radiotherapy in patients who are otherwise healthy are evaluated for treatment options, such as complete surgical resection and microvascular reconstruction. Preferably iliac crest, fibula or scapula is used to restore the defects. Myocutaneous flaps are also necessary to close large fistulas along mandibular body necrosis. Best supportive care is offered to patients when preoperative evaluation reveals additional severe comorbidities inhibiting surgical options.

Case 1

Ten years after radiotherapy, a 73-year-old male patient showed extensive necrotic bone of the mandibular body with pathologic fracture and extraoral buccal fistula (Fig. 16.4a–c). Preoperative evaluation revealed a heart metastasis of almost 8 cm (Fig. 16.4d, e) in diameter, which did not allow to perform surgery with resection and microvascular reconstruction of the

R. Gassner (ed.), *Complications in Cranio-Maxillofacial and Oral Surgery*,
https://doi.org/10.1007/978-3-030-40150-4_16

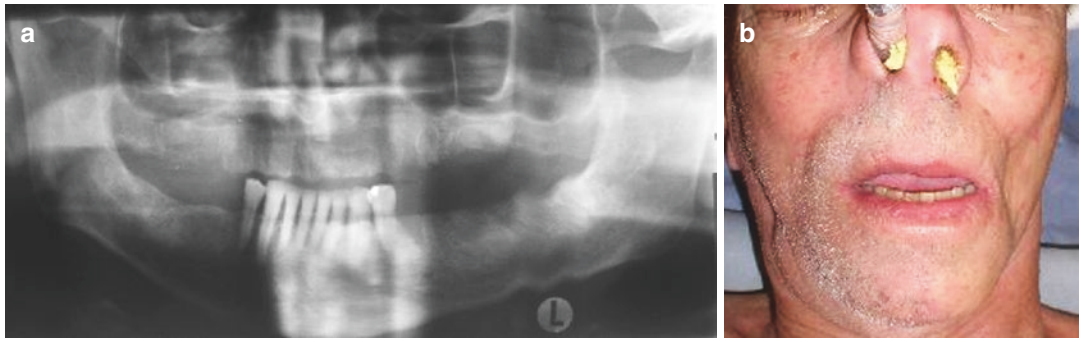


Fig. 16.1 (**a**) Panoramic radiograph with radiogenic-induced trism. (**b**) Same patient with radiogenic-induced trism

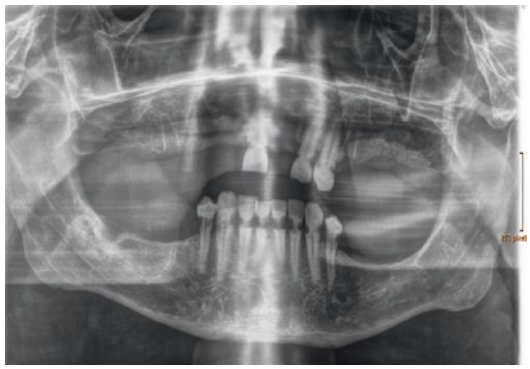


Fig. 16.2 Panoramic radiograph with radiogenic-induced caries

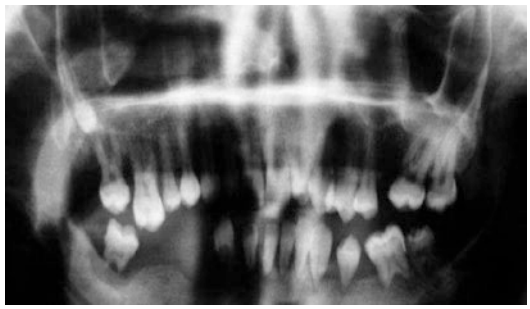


Fig. 16.3 Panoramic radiograph with radiogenic-induced inhibition of jaw and tooth development

defect under general anesthesia, despite his physical fitness due to his daily work out of wood splitting for hours.

Case 2

A 50-year-old female had a sore throat for several months and ENT evaluation with biopsy of the tonsils was negative. After 8 months, carcinoma of the tonsil was finally detected. Extended tonsillectomy and selective neck dissection were performed, followed by radiotherapy. A cascade of complications occurred instead of uneventful healing while surviving her malignancy.

Due to tooth ache on the right side of her mandible 3 months after final radiotherapy, the destroyed molar tooth in the right mandible was successfully removed (Fig. 16.5a–c).

The next complication was limited mouth opening which deteriorated to a level that resection of the muscular process of the lower jaw was performed. This was the onset of osteoradionecrosis of the right mandible. The jaw bone was resected incorporating a reconstruction plate while the infraalveolar nerve was preserved (Fig. 16.5d–g).

Free flap reconstruction ended in loss of the flap due to the severe infection followed by necrosis (Fig. 16.5h, i).

Removal of all hardware slowly allowed recovery (Fig. 16.5j).

A reconstruction plate with condylar head and a pedicled pectoralis major flap was inserted (Fig. 16.5k). Then, a two-piece free fibula bone flap replaced the reconstruction plate (Fig. 16.5l–n).

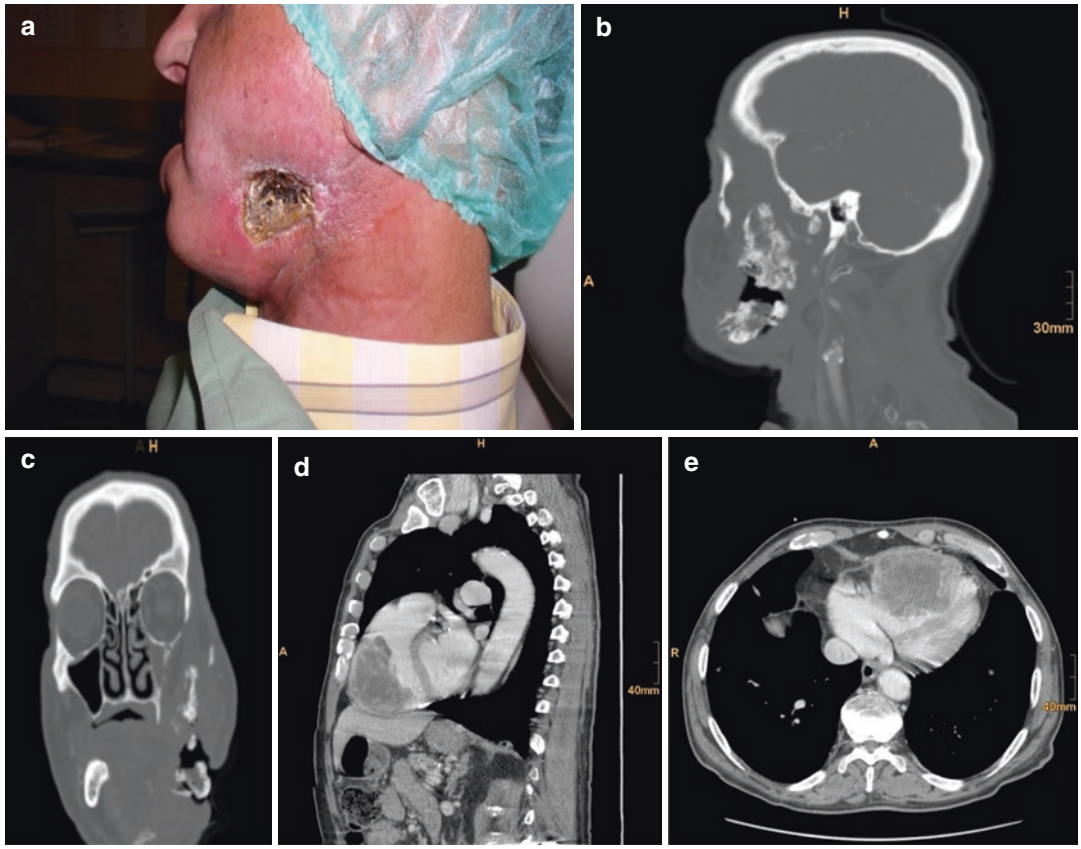


Fig. 16.4 (**a**) Patient with extraoral buccal fistula. (**b**) Same Patient with sagittal CT scan of osteoradionecrosis with pathologic fracture and extraoral buccal fistula. (**c**) Same patient with coronal CT scan of osteoradionecrosis with pathologic fracture and extraoral buccal fistula. (**d**) Same patient with Sagittal CT scan of heart metastasis. (**e**) Same patient with axial CT scan of heart metastasis

Case 3

A 75-year-old female was treated with tonsillectomy, neck dissection, and radiotherapy due to carcinoma of the tonsil. Five years later, infected osteonecrosis of the left and right mandibular body created misery for the patient despite surviving her cancer treatment. She had intra/extraoral non-healing lesions with exposed and infected bone on both sides. Resection of each side of the mandibular body necessitated the incorporation of a long reconstruction plate to stabilize the mandible and covering the defect using a latissimus dorsi flap (Fig. 16.6a, b). While the intraoral closure of the large defect was successful, it was necessary to use the second free flap to cover and close the exposed reconstruction plate (Fig. 16.6c–e).

Case 4

Seven years after radiotherapy, a 65-year-old male patient suffered sudden pain in his left mandibular body during eating. A panoramic radiograph revealed a fractured mandible. The intraoperative picture shows the broken frag-

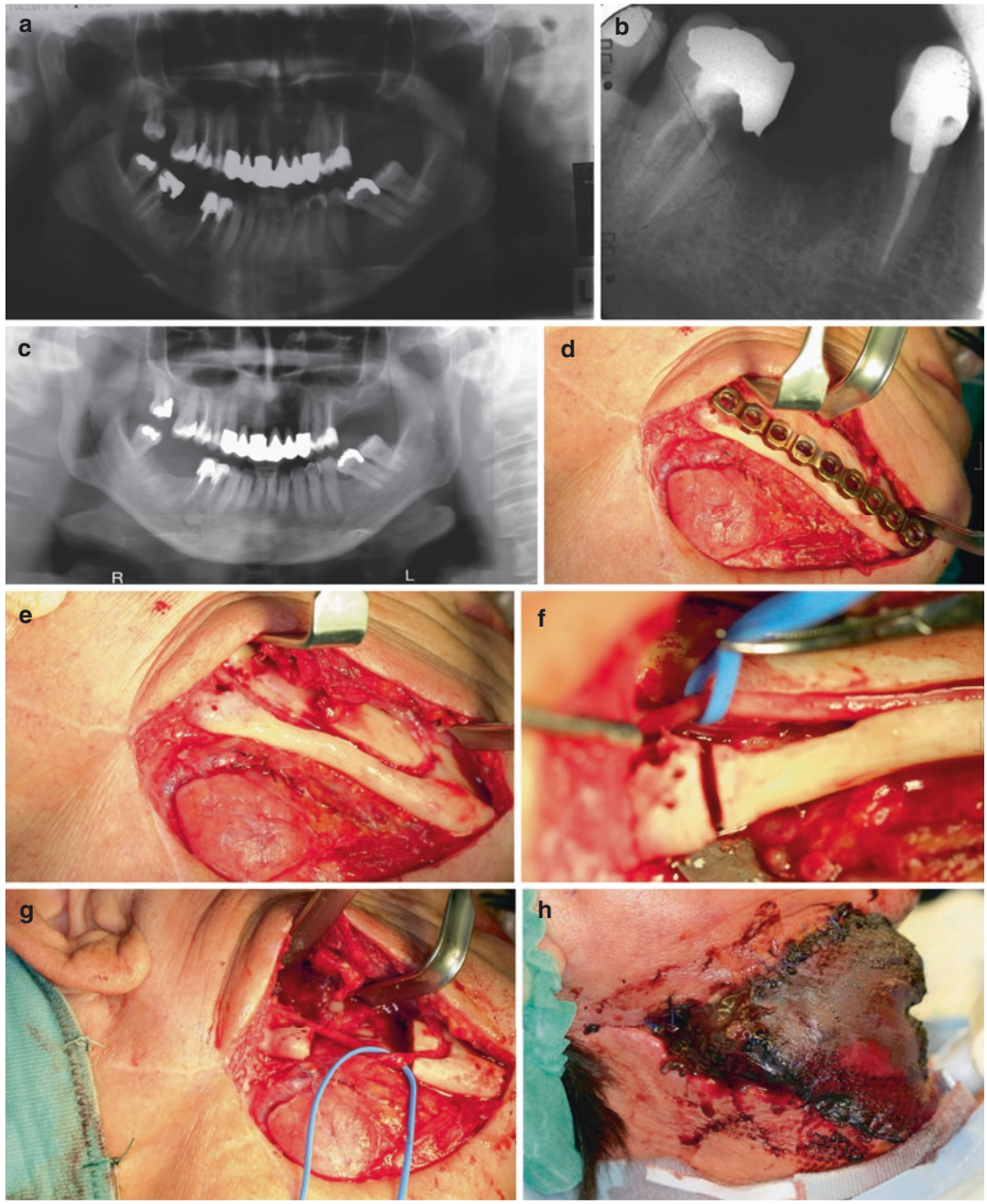


Fig. 16.5 (**a**) Panoramic radiograph following removal of 1st molar after radiotherapy. (**b**) Individual radiograph of tooth decay following removal of molar after radiotherapy. (**c**) Panoramic radiograph following removal of decayed 2nd molar after radiotherapy. (**d**) Preplating of reconstruction plate prior to mandibular body resection due to osteoradionecrosis. (**e**) Preserving the mental nerve. (**f**) Preserved mental nerve after surgical cut of right mandibular body. (**g**) Resected mandibular body and preserved mental nerve. (**h**) Flap necrosis. (**i**) Infected carotid triangle. (**j**) Panoramic radiograph following loss of right mandibular body and condyle. (**k**) Panoramic radiograph with reconstruction plate including artificial condyle. (**l**) Panoramic radiograph with fibula free flap reconstruction and two plates. (**m**) Postoperative mouth opening with deviation of chin. (**n**) Postoperative scaring after reconstruction in irradiated area

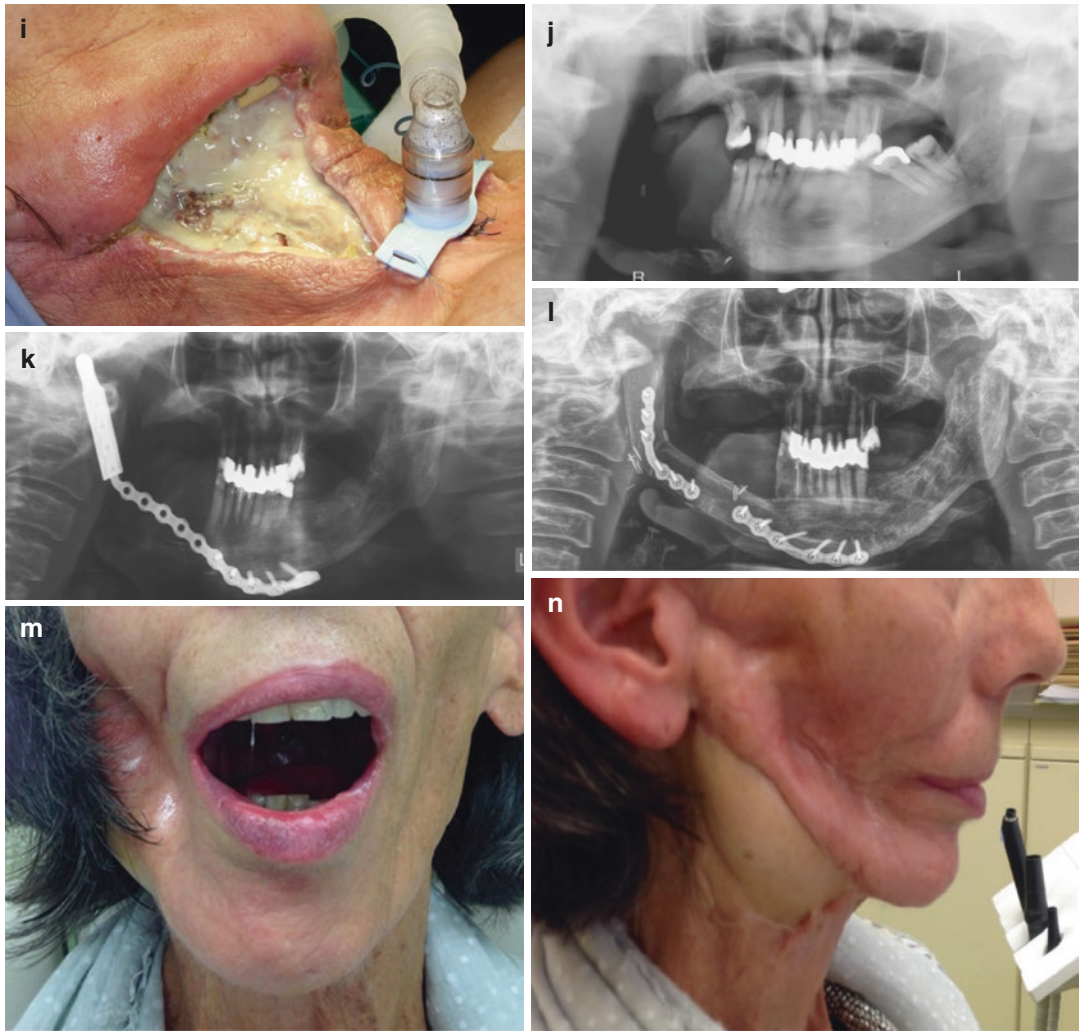


Fig. 16.5 (continued)

ments in the left mandible (Fig. 16.7a). Due to the fracture lines into the condyle, the reconstruction plate included an artificial condylar head. Also shown is the fixation setup for meticulous precise burr hole drilling and screw insertion (Fig. 16.7b). The free tissue transfer is provided using a microvascular pedicle harvested from the iliac crest for mandibular bone reconstruction (Fig. 16.7c). As every step during surgery, especially microsurgery, is crucial to obtain success three steps are depicted during suturing a successful anastomosis of the arteria suprathyreoidea and arteria ilium circumflexa profunda under the microscope (Fig. 16.7d–f). The postoperative panoramic radiograph shows the mandible with reconstructed bone and a reconstruction plate on the left side (Fig. 16.7g).

Although the postoperative 3D image of the mandible shows an acceptable result (Fig. 16.7h), the behavior of radiated tissue in the left lower lip reacted with a prolonged lymphedema (Fig. 16.7i) and the lateral view reveals the exposed reconstruction plate while the bone healed (Fig. 16.7j) and the patient commented, why can't you do a better job?

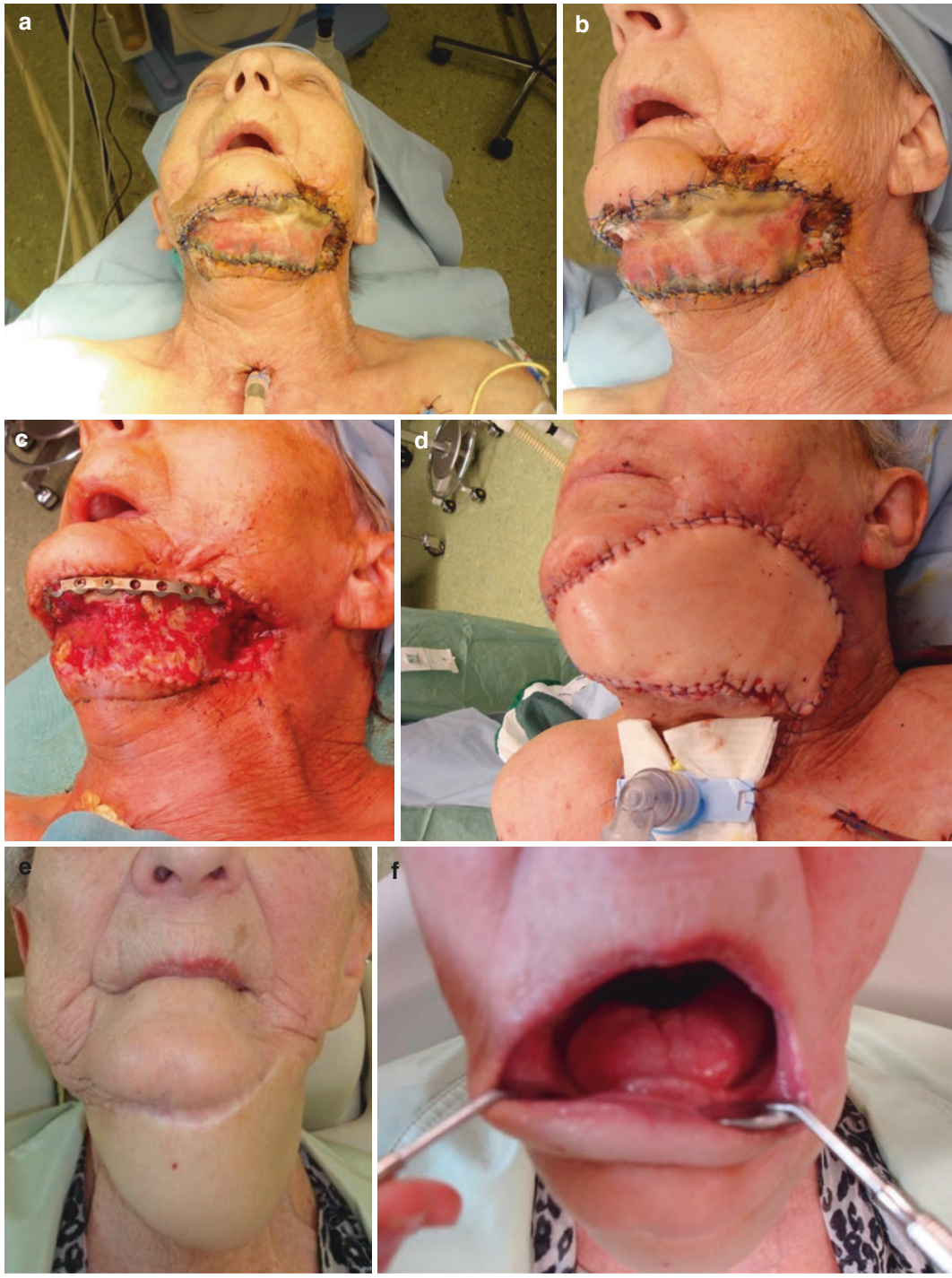


Fig. 16.6 (**a**) Worm's eye view: osteoradionecrosis of mandibular body on both sides after intraoral soft tissue reconstruction with lattissimus dorsi flap. (**b**) Side view: osteoradionecrosis of mandibular body on both sides after intraoral soft tissue reconstruction with lattissimus dorsi flap. (**c**) Extraoral exposure of reconstruction plate after intraoral soft tissue reconstruction with lattissimus dorsi flap. (**d**) Defect closure with 2nd lattissimus dorsi flap. (**e**) Successful treatment of osteoradionecrosis of mandibular body on both sides after intra- and extraoral soft tissue reconstruction with two lattissimus dorsi flaps. (**f**) Intraoral view: successful treatment of osteoradionecrosis of mandibular body on both sides after intra- and extraoral soft tissue reconstruction with two lattissimus dorsi flaps

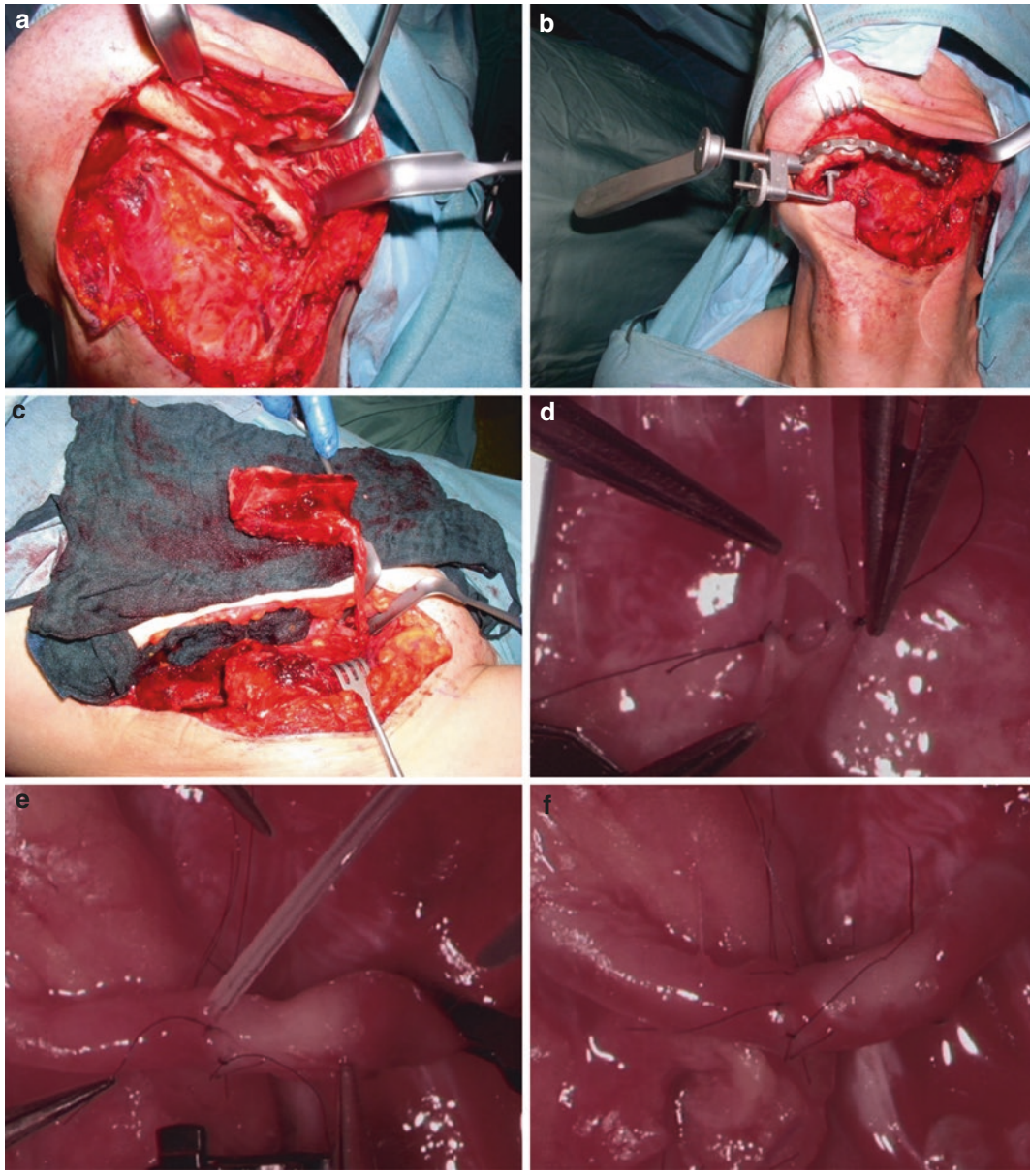


Fig. 16.7 (**a**) Intraoperative view: pathologic mandible fracture following radiation therapy. (**b**) Intraoperative view: reconstruction plate with artificial condyle following mandibular resection. (**c**) Intraoperative view: iliac crest free flap site with vessels circumflexa ilium profunda. (**d**) Microscopic intraoperative view: surgical site of microvascular anastomosis (neck)—suturing the back side first. (**e**) Microscopic intraoperative view: flushing the small vessel before the last stitch. (**f**) Microscopic intraoperative view: blood flow through the anastomosis. (**g**) Panoramic radiograph with iliac crest free flap reconstruction and reconstruction plate including artificial condyle. (**h**) 3D-CT image of iliac crest free flap reconstruction and reconstruction plate including artificial condyle. (**i**) Persisting lip swelling of the patient due to long-term effects of radiation therapy and lymph vessel depletion. (**j**) Extraoral plate exposure due to postoperative scarring including loss of facial nerve function due to scarring as long-term effect of radiation therapy

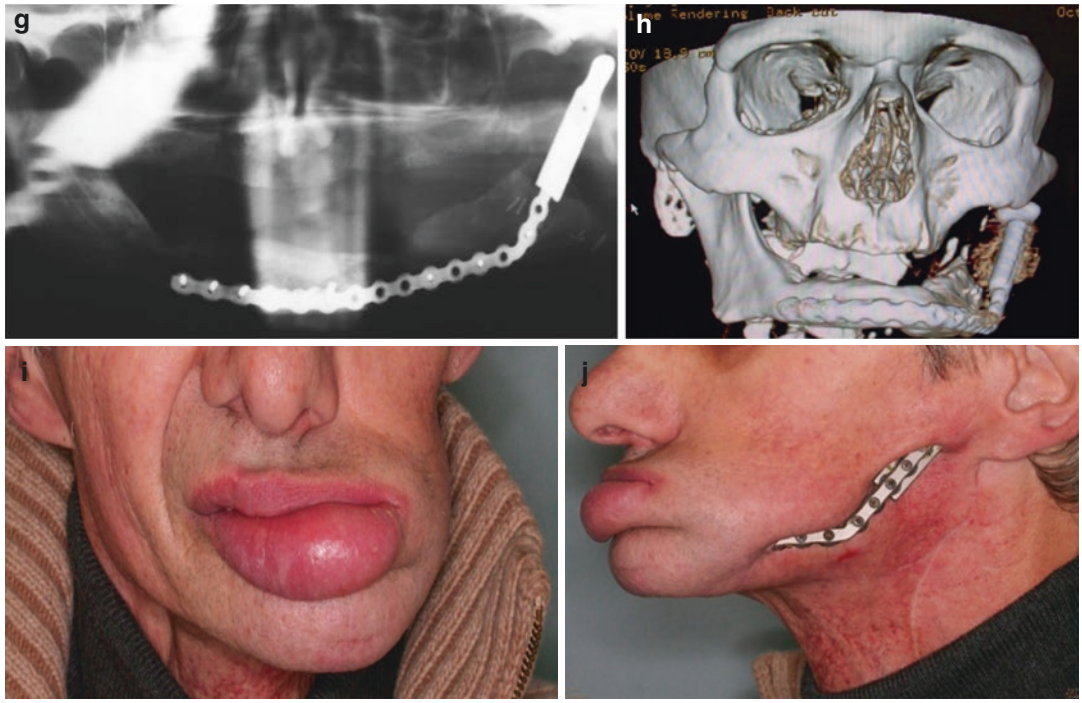


Fig. 16.7 (continued)

After removal of the exposed reconstruction plate, the patient's chin showed a deviation of the chin to the left, especially during mouth opening.

In the past decades efforts in basic and translational science helped to understand how the general and also local conditions for patients can be improved or even avoided to suffer from complications not only due to radiotherapy but also to avoid the onset of malignancies.

First, it has to be mentioned that we provided evidence for the first time in several publications based on funded NIH research that motion has an anti-inflammatory effect in vitro [3–7] and also in vivo [8–13]. Second, we showed in several papers that bone itself is harboring cells especially stem cells regarding the immune response which in turn supports the importance of motion as maintaining health due to changes in blood pressure and its blood flow in general [14–21].

Third, we described in recent publications our translational findings on funded research using nanotechnology [22–25] in improving bone healing in presence of radiotherapy [26–28].

Case 5

An otherwise healthy male complained about a loss of fitness feeling tiredness and fatigue. Blood evaluation was uneventful but ENT evaluation revealed a carcinoma of the tongue base (Fig. 16.8a, b). Tumor board recommendation suggested primary radiotherapy checking treatment response after 30 Gy via CT scan (Fig. 16.8c) and CT documentation at the end of radiotherapy at 70 Gy (Fig. 16.8d).

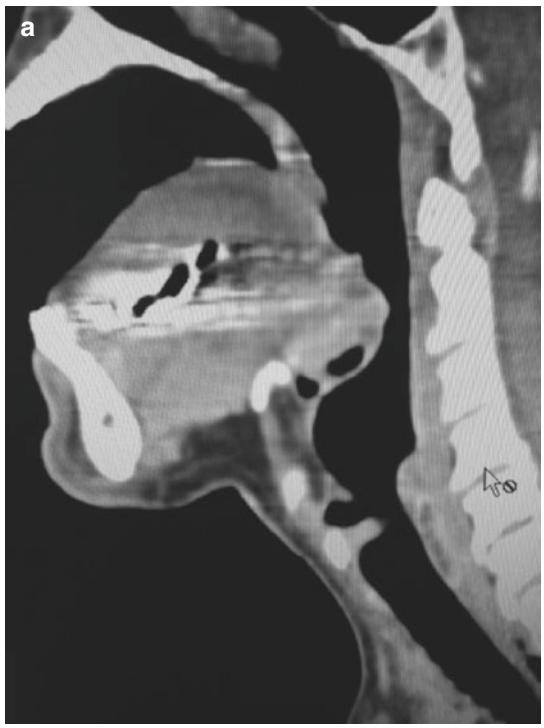
Prior to radiotherapy dental examination showed crowns and bridge work on both sides of his mandibular body. Explaining the possibility of osteoradionecrosis in case of the necessity of tooth removal after radiation he opted for removal of his mandibular premolars and molars (Fig. 16.8e–h) but denied the necessity of bilateral selective neck dissection. Follow-up panoramic X-rays and the clinical situation of the patient showed full recovery from his malignant illness.

He asked for oral rehabilitation to restore mastication which was performed successfully 2 years after radiation treatment (Fig. 16.8j–l).

Four implants on both sides of the mandibular body allowed after osseointegration (Fig. 16.8m–o) oral rehabilitation with a removable partial denture to secure the patient's wish for most efficient control of dental hygiene (Fig. 16.8p–r). So the denture is completely implant supported protecting the radiated gingiva.

Taken together this chapter shows that CMF surgery plays a leading role in case of tissue breakdown when CMF complications occur even years after radiotherapy. It is clear that the high risk of infection in the presence of parodontitis and tooth decay necessitates tooth removal including dental treatment with fluoride in areas of remaining teeth prior to radiotherapy to minimize and avoid the risk of osteoradionecrosis especially in the area of the mandibular body. There is also evidence that motion in general has a beneficial effect on prevention of tissue breakdown and onset of infection [3–21]. Patients overcome and survive their malignant disease, and it is our main goal to avoid the suffering from long-term complications after initial successful therapy.

Prior to radiotherapy, patients have to undergo a dental assessment to rule out potential complications due to untreated dental conditions which trigger osteoradionecrosis of the jaw, especially



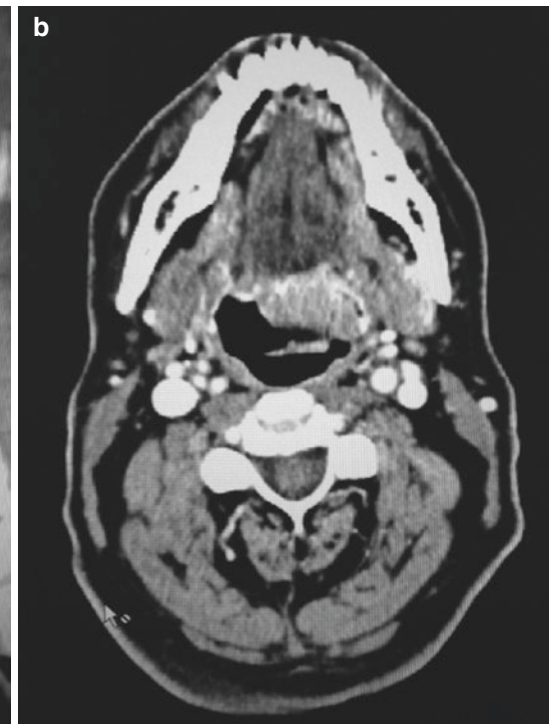


Fig. 16.8 (**a**) Sagittal CT scan of carcinoma of the tongue base. (**b**) Axial CT scan of carcinoma of the tongue base. (**c**) Axial CT scan of the tongue base after radiation with 30 Gy. (**d**) Axial CT scan of the tongue base after radiation with 72 Gy. (**e**) Panoramic radiograph prior to onset of radiation treatment recommending to remove all molars and premolars in the mandible (upper left). (**f**) Panoramic radiograph prior to onset of radiation treatment after removal of all molars and premolars in the mandible (upper right). (**g**) Panoramic radiograph after radiation treatment (lower left). (**h**) Panoramic radiograph 1 year after radiation treatment (lower right). (**i**) Intraoral view of implant placement 2 years after radiation treatment: right side (upper left). (**j**) Intraoral view of implant placement 2 years after radiation treatment: left side (upper right). (**k**) Panoramic radiograph following implant insertion in molar areas of mandible after radiation treatment (lower middle). (**l**) Axial CT scan of inserted implants in the molar areas of the mandible after radiation treatment (left). (**m**) Panoramic radiograph prior to loading of implants in radiated molar areas of mandible. (**n**) Intraoral view with locators (lower right). (**o**) Intraoral view with inserted removable partial prosthesis (left). (**p**) Worm's eye view: removable partial prosthesis (upper right). (**q**) Removable partial prosthesis (lower right)

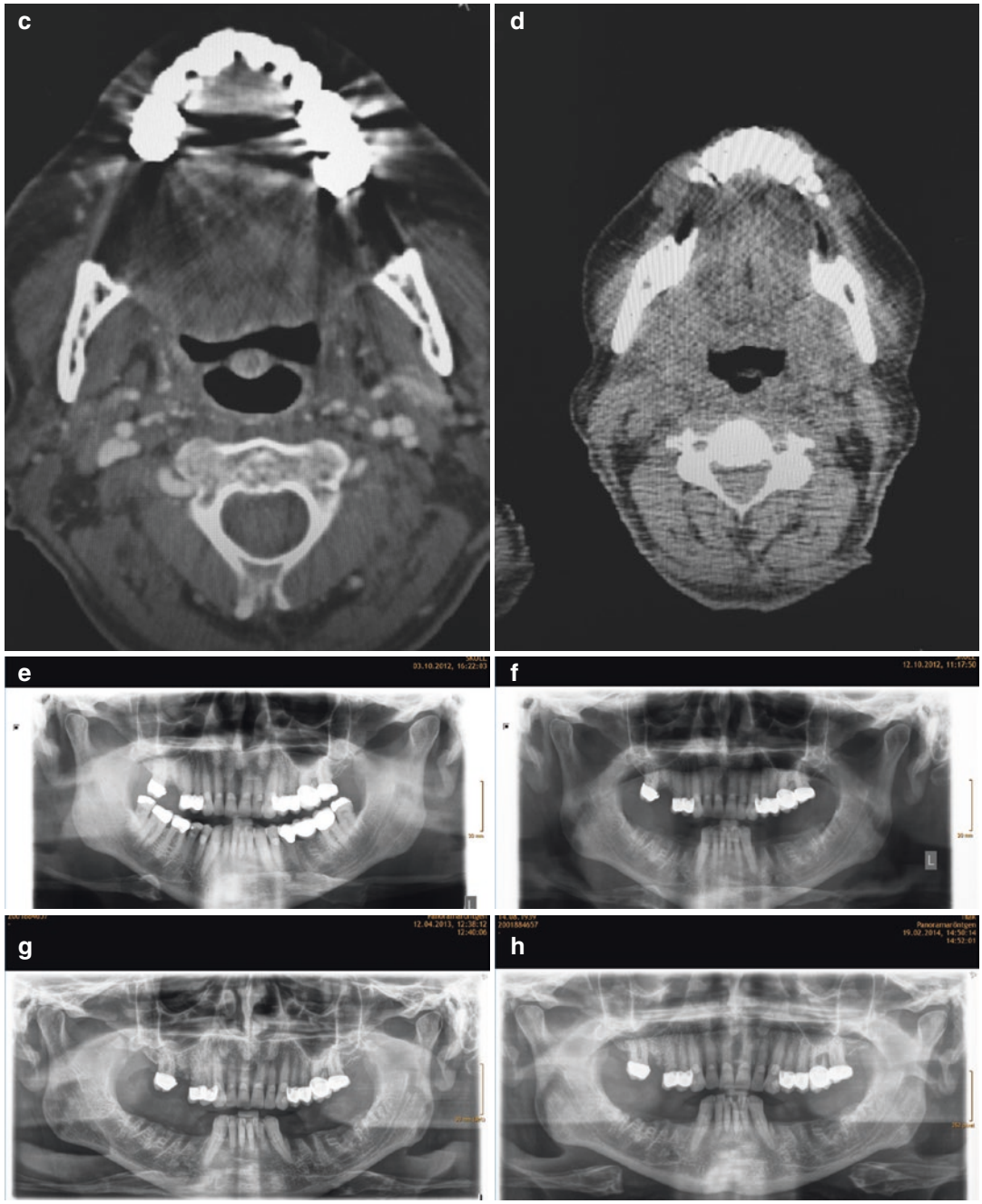


Fig. 16.8 (continued)

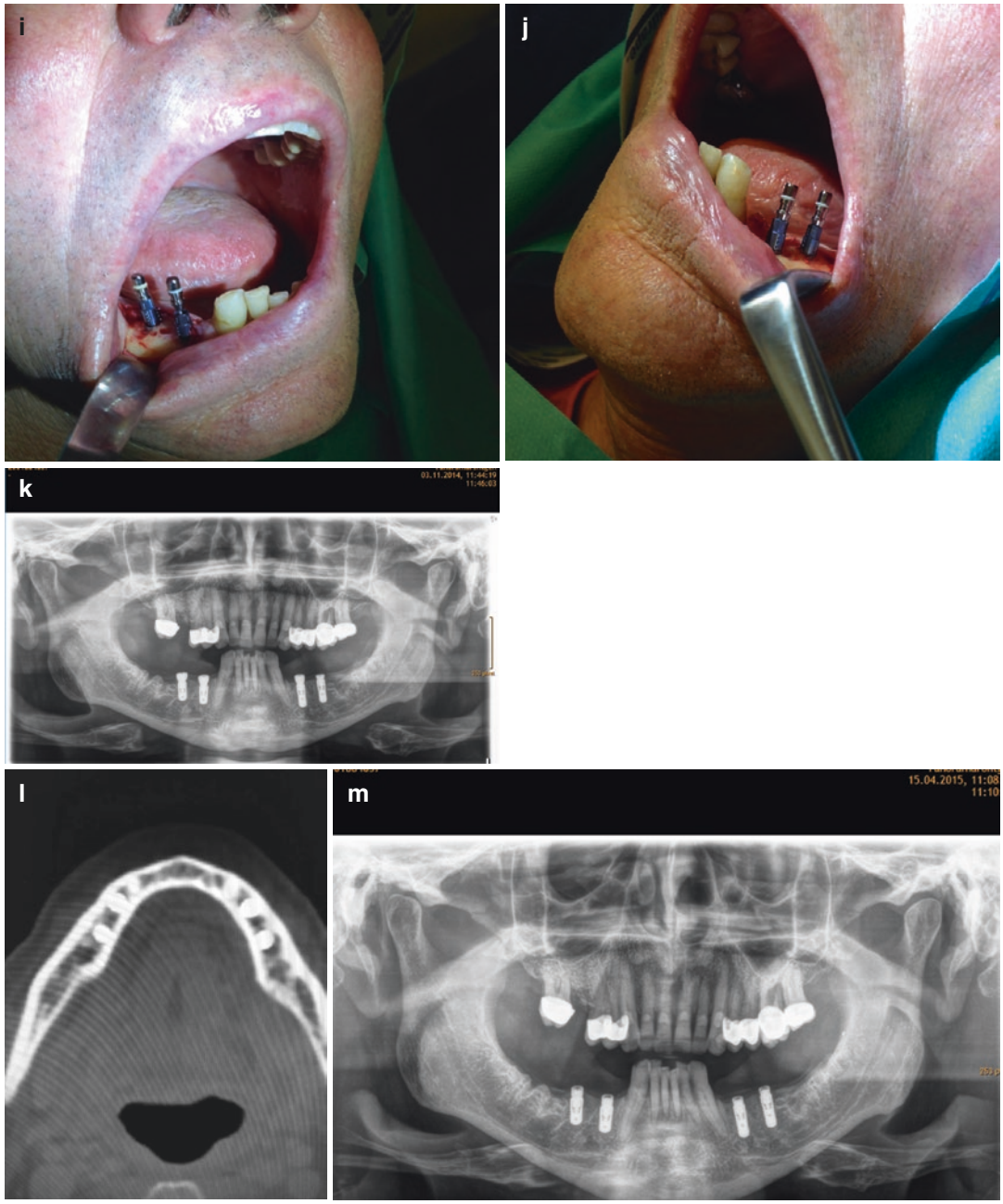


Fig. 16.8 (continued)

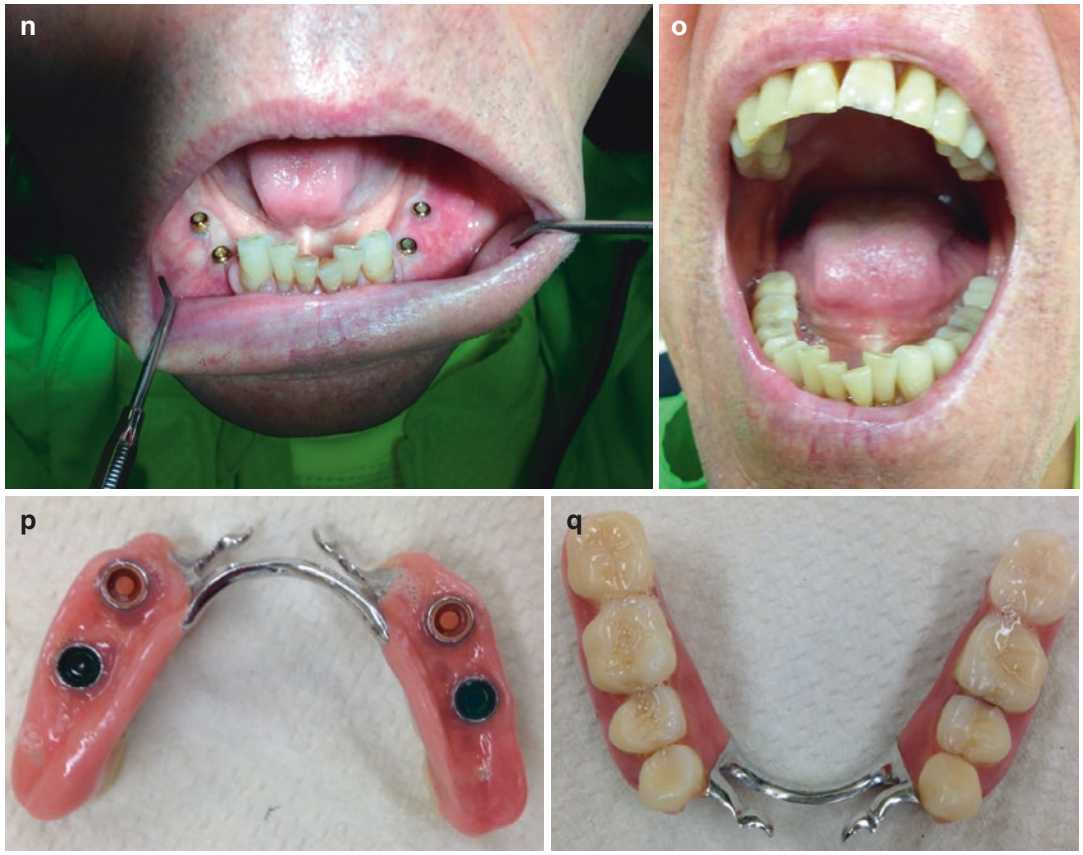


Fig. 16.8 (continued)

in the area of the mandibular body. For patients, it is sometimes difficult to understand that expensive implant, crown and bridge work has to be removed because the necessity of free flap surgery with bone from fibula, iliac crest, and scapula and its donor site morbidity is or sure inferior to maintaining a functioning mandibular body.

But all teeth with caries, periodontitis, root fillings, and cystic lesions need to be removed and soft tissue coverage of the underlying bone is a requirement before starting radiotherapy. In between radiotherapy sessions, wide mouth opening should be exercised to avoid the onset of trismus. Physiologic physical activity is anti-inflammatory itself. Blood turnover including all immunologic cells acts at a higher level and, therefore, provides a protective and preventive effect regarding the onset of osteoradionecrosis. As implants are bioinert in these situations, they can be inserted into otherwise healthy irradiated mandibular bone to provide a higher quality of life.

References

1. Marx RE, Stern D. A rationale for diagnosis and treatment. In: Marx RE, Stern D, editors. Oral and maxillofacial pathology. Chicago: Quintessence; 2003. p. 497.
2. Gassner R, Kloss F, Singh A, et al. Osteoradionecrosis: behavior of human and porcine mesenchymal stem cells following irradiation. J Oral Maxillofac Surg. 2009;67(Suppl 1):50–1.
3. Gassner R, Buckley M, Studer R, et al. Cyclic tensile stress exerts antiinflammatory properties by inhibiting iNOS induction in chondrocytes. J Immunol. 1999;163:2187–92.
4. Agarwal S, Long P, Gassner R, et al. Cyclic tensile strain suppresses catabolic effects of IL-1β in fibrochondrocytes from temporomandibular joint. Arthritis Rheumatol. 2001;44:608–17.

5. Long P, Gassner R, Agarwal S. Tumor necrosis factor-alpha-dependent proinflammatory gene induction is inhibited by cyclic tensile strain. Arthritis Rheumatol. 2001;44:2311–9.
6. Gassner R, Agarwal S. Biological basis for the effectiveness of continuous passive motion-mediated repair in TMJ diseases. J Craniomandibular Pract. 2002;20:152–3.
7. Agarwal S, Long P, Seyedain SA, et al. A central role for the nuclear factor-kappaB pathway in anti-inflammatory and proinflammatory actions of mechanical strain. FASEB J. 2003;17:899–901.
8. Ferretti M, Gassner R, Zheng W, et al. Biomechanical signals suppress proinflammatory responses in cartilage: early events in experimental antigen-induced arthritis. J Immunol. 2006;177:8757–66.
9. Nam J, Perera P, Liu J, et al. Sequential alterations in catabolic and anabolic gene expression parallel pathological changes during progression of monoiodoacetate-induced arthritis. PLoS One. 2011;6:e24320. https://doi.org/10.1371/journal.pone.0024320.
10. Gassner R. Structure and function of the TMJ. In: Fonseca R, Marciani R, Turvey T, editors. Oral and maxillofacial surgery(Ch 42), vol. II. 2nd ed. St Louis: Saunders; 2009. p. 801–15.
11. Gassner R. Pathology of the TMJ. In: Fonseca R, Marciani R, Turvey T, editors. Oral and maxillofacial surgery (Ch 45), vol. II. 2nd ed. St Louis: Saunders; 2009. p. 849–81.
12. Gassner R. Structure and function of the TMJ. In: Fonseca R, editor. Oral and maxillofacial surgery (Ch 35), vol. II. 3rd ed. St Louis: Saunders; 2018. p. 777–90.
13. Gassner R. Pathology of the TMJ. In: Fonseca R, editor. Oral and maxillofacial surgery (Ch 38), vol. II. 3rd ed. St Louis: Saunders; 2018. p. 830–64.
14. Fehrer C, Brunauer R, Laschober G, et al. Reduced oxygen tension attenuates differentiation capacity of human mesenchymal stem cells and prolongs their life span. Aging Cell. 2007;6:745–57.
15. Lepperdinger G, Brunauer R, Gassner R, et al. Changes of the functional capacity of mesenchymal stem cells due to aging or age-associated disease—implications for clinical applications and donor recruitment. Transfus Med Hemother. 2008;35:299–305.
16. Laschober GT, Brunauer R, Jamnig A, et al. Age-specific changes of mesenchymal stem cells are paralleled by upregulation of CD106 expression as a response to an inflammatory environment. Rejuvenation Res. 2011;14:119–31.
17. Herndler-Brandstetter D, Brunner S, Weiskopf D, et al. Post-thymic regulation of CD5 levels in human memory T cells is inversely associated with the strength of responsiveness to IL-15. Hum Immunol. 2011;72:627–31.
18. Herndler-Brandstetter D, Landgraf K, Jenewein B, et al. Human bone marrow hosts polyfunctional memory CD4$^+$ and CD8$^+$ T cells with close contact to IL-15-producing cells. J Immunol. 2011;186:6965–71.
19. Herndler-Brandstetter D, Landgraf K, Tzankov A, et al. The impact of aging on memory T cell phenotype and function in the human bone marrow. J Leukoc Biol. 2011;91(2):197–205. http://www.jleukbio.org/content/early/2011/10/24/jlb.0611299.
20. Theresa P, Katja L-R, Herndler-Brandstetter D, et al. Bone marrow T cells from the femur are similar to iliac crest derived cells in old age and represent a useful tool for studying the aged immune system. Immunity Ageing. 2013;10:17. https://doi.org/10.1186/1742-4933-10-17.
21. Reitinger S, Schimke M, Klepsch S, et al. Systemic impact molds mesenchymal stromal/stem cell aging. Transfus Apher Sci / pii. 2015;S1473-0502(15):72–5.
22. Steinmueller-Nethl D, Kloss F, Najam-Ul-Haq M, et al. Strong binding of bioactive BMP-2 on nanocrystalline diamond by physisorption. Biomaterials. 2006;27:4547–56.
23. Kloss F, Najam-Ul-Haq M, Rainer M, et al. Nanocrystalline diamond—an excellent platform for life science applications. J Nanosci Nanotechnol. 2007;7:4581–7.
24. Kloss FR, Gassner R, Preiner J, et al. The role of oxygen termination of nanocrystalline diamond on immobilisation of BMP-2 and subsequent bone formation. Biomaterials. 2008;29:2433–42.
25. Gassner R. Bioactive BMP-2 on nano-crystalline diamond-coated implants. In: Ewers R, Lambrecht T, editors. Oral implants—bioactivating concepts. Chicago: Quintessence; 2013. p. 26–33.
26. Singh S, Kloss F, Brunauer R, et al. Mesenchymal stem cells show radioresistance in vivo. J Cell Mol Med. 2012;16:877–87.
27. Kloss F, Singh A, Hächl O, et al. BMP-2 immobilized on nanocrystalline diamond-coated titanium screws; demonstration of osteoinductive properties in irradiated bone. Head Neck. 2013;35:235–41.
28. Arnold CR, Kloss F, Singh S, et al. A domestic porcine model for studying the effects of radiation on head and neck cancers. Oral Surg Oral Med Oral Pathol Oral Radiol. 2017;123:536–43.