

Complications in Maxillofacial Cosmetic Surgery

Strategies for Prevention
and Management

Elie M. Ferneini
Charles L. Castiglione
Mohammad Banki
Editors

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Preface

Having practiced the art of cosmetic surgery exclusively for 20 years, the most challenging and stressful aspect of this specialty absolutely revolves around the prevention and management of complications. Lectures and articles that clearly address complications are always popular because of the deficiency of honest and blunt discussions related to this important topic. I commend the authors for taking on the challenge of writing this very thorough book on complications in maxillofacial cosmetic surgery.

The authors clearly delineate the most common complications seen in facial cosmetic surgery in a very thoughtful and easy-to-follow format. One of the best features of this text is the wonderful diversity and expertise of the authors. By using authors from various specialty training, the reader benefits by reviewing a range of treatment modalities not seen in most texts that limit authorship to an isolated specialty. Plastic and reconstructive surgeons, oral and maxillofacial surgeons, otolaryngologists, dermatologists, and any other surgeons performing cosmetic surgery of the face will want to have this book in their library. Frankly, even doctors who limit their cosmetic work to minimally invasive facial cosmetic procedures will benefit tremendously from this book.

Sections in the text are separated into very clear and practical divisions. The first covers wonderful topics often missed in the average complication text. Fantastic chapters dedicated to medicolegal issues involved with the cosmetic surgery patient along with pain management are just a couple that make this an outstanding resource. Also, the text covers anesthesia issues, infection prevention, and treatment standards, along with an excellent discussion involving current therapy for wound treatment and healing.

Detailed management of specific facial cosmetic surgical complications in the book's second section is very up-to-date with the newest and most ideal management for each common procedure. The comprehensive coverage of issues is impressive for a single textbook. The text covers complication treatment for facial implants, facelifts, rhinoplasty, orthognathic surgery, skin resurfacing, and nonsurgical injectables, just to name a few. A wonderful variety of authors cover each procedure and the potential complications openly and succinctly. Even with the large variety and number of authors, the organization and uniformity is superb and demonstrates the dedication and time invested by the editors.

It is extremely challenging to write and edit a textbook covering most every conceivable facet of facial cosmetic surgery complications. This book

achieves an incredible amount of both detail and broad coverage of a vast variety of issues when dealing with problems associated with facial cosmetic surgery. It is a breath of fresh air in its honesty and multispecialty review. I highly recommend this text as a must-have for any surgeon from novice to expert who performs facial cosmetic procedures.

Angelo Cuzalina, M.D., D.D.S.
Tulsa, OK

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Part I

General Topics

History of the Facelift: The First Three Decades

1

Jane A. Petro

Abstract

Innovative surgical procedures may be presented as newly discovered procedures. This may not be true, unless the author has done a thorough search of the prior literature to confirm originality. Many techniques are described, used, and abandoned, only to reappear as a fresh discovery. In reviewing the history of facelift surgery, it is apparent that almost all of the approaches to facial rejuvenation were described in the early decades of the 1900s. Thus, this chapter will provide a summary of the key discoveries during that era. Later approaches reflect refinements and different anatomical understandings and use better materials but are not necessarily as original as claimed. Anyone writing about facelifting today would be well advised to read the contributions of CC Miller, AG Bettman, Suzanne Noël, Jacques Joseph, Julien Bourguet, and the others discussed here.

1.1 Introduction

History is about telling a story using facts that the historian finds relevant along the way. There are contemporaneous accounts to be reviewed such as first person narratives, newspaper reports, and formally presented written books and articles. Doing history once meant access to libraries, finding documents and information wherever possible. Writing history might also require travel to find the sources necessary to conduct historical

inquiry. As we enter the twenty-first century, much of that has changed. Google Books have digitized the archives of many of the most extensive library collections in the world. Online translation services have given access to languages that the historian may not speak. Search engines, like PubMed, Google Scholar, Scopus, etc., have enabled much more detailed searches of the published literature. This includes citations, patents, and government documents across the world in addition to the books, scientific journals, and newspapers.

Scholarship has become both more accessible and more overwhelming. At the same time, respect for history has diminished. Every 5 years, something “new” is presented with little regard to

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any past precedence. Thus, telling the story of the evolution of the facelift requires a careful reckoning of claims to priority, originality, and discovery. It also offers the opportunity to introduce some forgotten and neglected innovators and charlatans. In this chapter, the origins of surgical rejuvenation, including facelifting, skin peeling, fillers, and nerve toxins, will be shown to have arisen well before the “modern” era and that most of these techniques were tried prior to 1930.

1.2 Pre-nineteenth-Century Beauty Surgery

Episodic reports of “cosmetic” surgery before 1900 represent anecdotal reports and simple case presentations that are rarely accurate. In the absence of anesthesia and sterile technique, most such surgery would have been unlikely to be successful or desirable if the only goal was cosmetic. Beauty treatments appear in the Ebers and Smith papyri and continue to appear to the present day but do not involve true surgical approaches. One of the earliest reports of methods to “reverse” aging appears in a treatise by Giovanni Marinello in 1574. This Venetian is recommending a topical herbal infusion.

Surgery for “beauty” does not appear until the eleventh century AD, with reports of procedures by Avicenna (Ibn Sina) in Persia, Albucasis (Abu al-Qasim Khalaf ibn al-Abbas Al-Zahrawi) in Spain, and Averroes (Ibn Roshd) in the twelfth-century Morocco. These are reports of treatment for ectropion, entropion, lagophthalmos, and blepharoptosis, so they do not strictly qualify as cosmetic operations but are often cited as such.

1.3 Early Pioneers and Efforts at Rejuvenation

Fillers as an antiaging strategy begin appearing in 1890s with the introduction by Robert Gersuny, an Austrian surgeon, and J Leonard Corning, an American neurologist, who both recommended the use of paraffin. These treatments grew to include paraffin, Vaseline, olive oil, and various

other “natural” substances. Such treatments became widespread and popular, at least, according to the popular press. Fredrick Strange Kolle’s 1911 textbook *Plastic and Cosmetic Surgery* [1] contains a chapter on hydrocarbon prosthesis that is 129 pages long, as compared to 18 pages on “Principles of Plastic Surgery” or 17 pages on “Blepharoplasty” or 47 pages dedicated to “Cheiloplasty.” His lengthy dissertation on hydrocarbon injections includes several key features:

1. A detailed account of the numerous complications to be anticipated, most notably an anatomic depiction of the veins and arteries of the head and neck, to be avoided during injection (Fig. 1.1)
2. Directions for the preparation of the material to be used
3. Recommendations of the instrumentation needed
4. Descriptions of the many deformities for which it may be used

This textbook, now available in many formats on the web, is of keen historical interest as a review of contemporary techniques for antiseptics, anesthesia, the therapeutic use of electrical current, and, of course, paraffin injection. His chapter on meloplasty (surgery of the cheeks) includes numerous illustrations for flaps that can be used to fill defects but no mention of techniques to eliminate sagging or wrinkles.

1.4 The Original Featural Surgeon

The first textbook to specifically address cosmetic facial cosmetic surgery is credited to Charles Conrad Miller, published in 1907 as *Cosmetic Surgery: The Correction of Featural Imperfections* [2]. This book reflects Miller’s serious attempts to participate in academic practice. He thanks multiple medical journals where he had previously published articles related to the contents of his text, including the *Wisconsin Medical Recorder*, the *American Journal of Dermatology*, the *American Journal of Clinical*

Fig. 1.1 Illustration from Kolle's textbook *Plastic and Cosmetic Surgery* demonstrating the veins and arteries of the head and neck to be avoided with the injection of paraffin or other materials as fillers

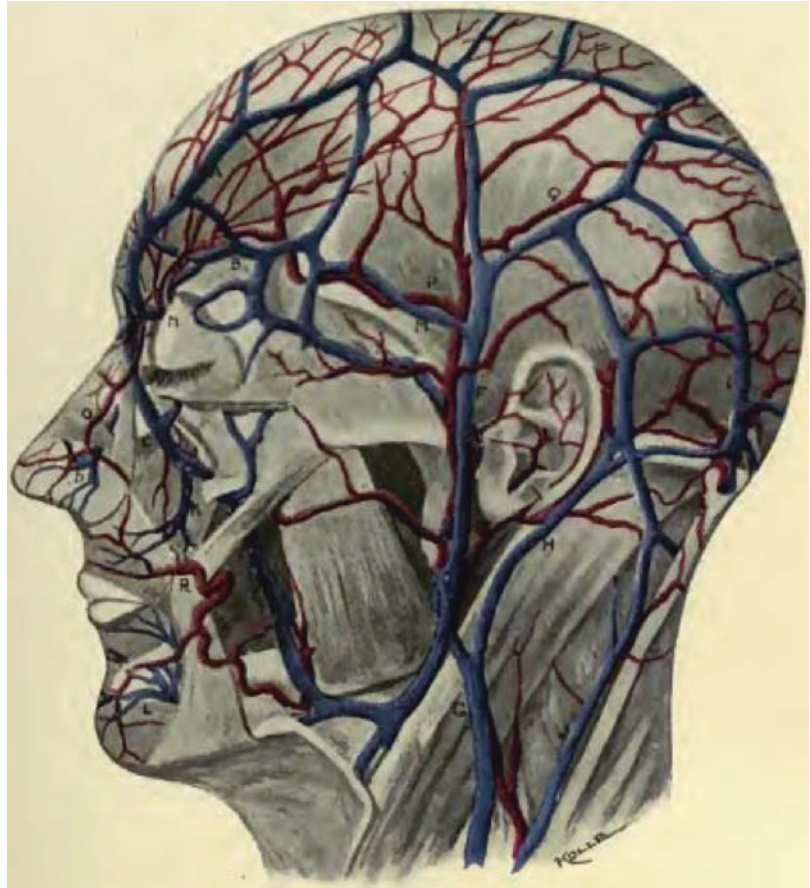


FIG. 283.—CIRCULATION OF THE HEAD

- | | |
|-------------------------|----------------------------------|
| A. Supra-Orbital Vein | J. Post-Ext.-Jugular Vein |
| B. Supra Palpebral Vein | K. Sup. Labial Vein |
| C. Angular Vein | L. Inf. Labial Vein |
| D. Nasal Vein | M. Transverse Facial Vein |
| E. Facial Vein | N. Communicating Br. Ophtal Vein |
| F. Temporal Vein | O. Angular Artery |
| G. Ext.-Jugular Vein | P. Ant. Temporal Artery |
| H. Post-Auricular Vein | Q. Post Temporal Artery |
| I. Occipital Vein | R. Sup. Coronary Artery |

Medicine, etc., providing evidence of his scholarly approach. He appears prescient in many ways with comments made in his introduction:

“A large number of the profession are at the present day apathetic regarding elective surgery for the correction of those featural imperfections which are not actual deformities but such apathy cannot prevent the development of this specialty as the demand for featural surgeons is too great on the part of the public. I feel that this little book is merely the forerunner of works as pretentious as any we have upon special subjects.”

Among the chapters in this book are those specifically on “Folds, bags and wrinkles of the skin about the Eyes,” “The Eradication of the Nasolabial Line,” and “The Double Chin.” In this first edition, he promotes surgical approaches to the face that include blepharoplasty techniques and what we would clearly recognize as cosmetic surgery principles. His description of the lower and upper lid skin excisions could be part of any contemporary guidelines, as he commands that the operator obtain complete hemostasis, placing

the lower lid incision below the lashes with sufficient margin for closure and using fine sutures tied loosely to facilitate removal.

His book attracted considerable attention and was reviewed in the *California State Medical Journal* [3]. The review notes that in facial cosmetic surgery because it is “left largely in the hands of” beauty specialists “and others of that tribe, advances in this field have been limited from want, in part of adequate stimulus on the part of the medical profession.” The review goes on to note that Miller among others “at first looked upon askance have established reputations founded upon honest effort in the uplifting of practice of this kind.” During an incredible period of productivity, Miller published over 30 papers on various procedures related to the correction of featural imperfections.

His second textbook, also titled *Cosmetic Surgery*, was published in 1924. In this edition, Miller shows multiple drawings that demonstrate the same types of incisions still used today in facelifting, such as those illustrated in Fig. 1.2a, b [4]. This new edition is greatly expanded and discusses not only local anesthesia as in the first but also nerve blocks. His chapters on eyelid surgery and facelifting operations are copiously illustrated. These are followed by several chapters on less invasive methods for eradication of lines and wrinkles which he refers to as subcutaneous sectioning that sounds like subcision, which sadly is not well illustrated. It is of note that in this edition, paraffin or Vaseline injections are not recommended or mentioned, unlike those found in his first edition.

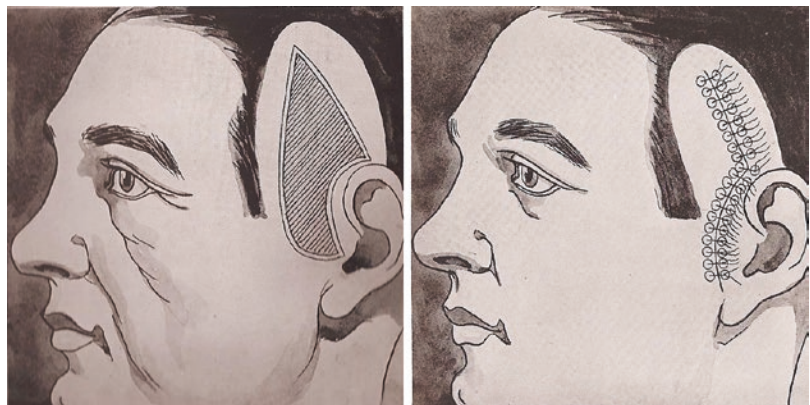
Dr. Miller continued to view his field of cosmetic surgery as an unfairly marginalized practice. In his 1924 edition, he states:

Since 1907 there has been a great deal of cosmetic surgery done by ranking surgeons in all medical centers. But few, indeed, of these surgeons care to write even articles for the medical press upon the subject. This, of course, is due to the desire of these surgeons to escape criticism by their professional brothers, for like all new specialties, cosmetic surgery has been subjected to criticism.

Miller’s career is notable not only for his vigorous defense of cosmetic surgery but for his ability to adopt new ideas quickly. Few if any surgeons were doing the kinds of operations he describes first in reports of 1907 and perfected up to the time of his second book in 1924.

Public interest in cosmetic surgery may be difficult to gauge at that time, but the medical literature, numerous advertisements in newspapers, as well as “beauty column’s” written at the time suggest that such interest was present. Nineteenth-century references to cosmetic surgery are generally negative. For example, in 1870, the *British Medical Journal* (*BMJ*) carried a report of cosmetic surgery used to obliterate the branding iron scar of a convicted soldier. Dismissed as apocryphal, the account contends that such practice would have implications for cosmetic and practical surgery. In 1896 the *BMJ* reports on a Dr. Roderick Maclaren’s discussion of “preventive surgery” which included comments on cosmetic surgery. Maclaren admitted that certain operations, including those measures of so-called cosmetic surgery, are not attended

Fig. 1.2 (a) Illustration from Miller’s *Correction of Featural Imperfections* showing his temporal/preauricular incision to correct the nasolabial fold. (b) Illustration from Miller’s book showing of wound closure and result correcting the nasolabial fold



with exceptional mortality. He goes on to outline certain rules which ought to be applied if preventive surgery is to become more widely applied [5]. These rules are not listed, and the author of the report goes on to state that antiseptic practice and anesthesia cannot justify such preventive surgeries. “A preventive operation should be devoid of risk to life both at the time during the healing stage: but can the prudent surgeon predict this with regard to any cutting operation, however insignificant it may seem to be?” [6]. Ironically, in 1899, the *BMJ* reported a case of sepsis following cosmetic surgery in a young man, part of a discussion among pathologists about the examination of the blood. The young man is described as having a cosmetic operation followed 5 days later by severe influenza-like symptoms, with a low red and white cell count. Diagnosed with sepsis, the surgeon opened the wound and found nothing. Despite antistreptococcus serum injections, the patient died on the ninth postoperative day [7].

CC Miller was not the only practitioner promoting featural surgery in the USA. Arthur Span, self-described as a prominent Viennese physician, advertised in the Pittsburgh and Chicago papers in 1906 and 1907, offering free consultations, a free book. He also guaranteed to provide

correction of crooked noses, blemishes, and baldness or remove a few wrinkles. A full society page endorsement in the July 1, 1906, *Pittsburgh Press* includes photographs of “Dr. Span” performing procedures, as well as before and after photographs. These may be the earliest cosmetic surgery photographs. See Fig. 1.3. His career seems to have been short-lived. In 1908, the Pittsburgh papers reported that he had been fined \$300 for “practicing medicine without going through the formality of registering according to the laws of the commonwealth” [8]. Subsequently at trial, he was found guilty of practicing without obtaining a license but acquitted of practicing without a diploma, when testimony revealed that Dr. Thomas H Wallace actually performed any surgery done in the Span offices [9]. Thereafter his advertisements were small paid promotions for the removal of pimples, red nose, and oiliness and cease completely in 1908.

While Arthur Span is a footnote to facial cosmetic surgery, CC Miller is justly identified as the first truly cosmetic surgeon. A biographical essay by John Milliken describes Miller’s graduation at the top of his class in medical school and his publication a year later of directions for setting up and running an office operating room [10]. The following year, he was a professor of



Fig. 1.3 *Pittsburgh Press* Society Page Promotion of Dr. Arthur Span (The Pittsburgh Press (Pittsburgh, Pennsylvania) Sun, Jul 1, 1906 Page 38)

surgery at the Harvey Medical College in Chicago, a short-lived coeducational medical night school. Miller began publishing a remarkable series of papers, 37 of which are cited by Milliken. Topics include lip reduction, electrolysis, tattoo for scar improvement, dimple creation, and a variety of procedures to reduce wrinkles, bags under the eyes, and rhinoplasty, among others. His early papers recommend mini-excisions for tightening the skin and subcision-type approaches sectioning the facial muscles for wrinkle reduction. He wrote a monograph reporting ten cases of “rupture” (hernia) treated with paraffin injection. He collected his early published “featural” work into his first book, including both illustrations and photographs in 1907. In 1913 he was charged with the sales of narcotics without a prescription, but the negative publicity seems not to have interrupted his practice. His “featural” practice continued through the 1920s. He devised a syringe used to inject fat, harvested from the abdomen, to fill contour deformities [11], and when he felt that this was unsatisfactory, he tried gutta-percha, rubber, ivory, and a number of other substances which he also abandoned as unsatisfactory. Following the economic collapse of 1929, Miller returned to general surgery but continued to publish, including textbooks on thyroid surgery, hemorrhoids, and tonsillectomy.

1.5 The Beginning of the Establishment of Cosmetic Facial Surgery

Other physicians, who trained in surgery, also began practicing featural surgery, as exemplified by Frederick Strange Kelle’s publication of his text *Plastic and Cosmetic Surgery* in 1911 [1]. His principal contribution to facelifting here was the use of paraffin injections to correct glabellar lines and nasolabial folds. Another early pioneer was Friedrich Hollander who published a chapter entitled “Cosmetic Surgery” as part of Max Joseph’s 1912 textbook *Handbuch Der Kosmetik* [12]. In a brief two paragraphs, Hollander describes little incisions in the scalp to correct

aging in the cheek and face and warns against doing larger incisions. In 1932, Hollander claimed to have actually done a more extensive facelift, using a full preauricular incision in 1901. He describes the unsatisfactory results from the small incisions, leading him to develop his more extensive technique. Even when a preauricular strip of 5 cm was excised, he cautioned against undermining. He was the first to describe pulling the skin in an upward and lateral direction. He described excisions of fat for the correction of a double chin and as part of the lifting procedure. He further recommended that the best results were obtained if the patient complained of tension in the incision for the first 3 days following the procedure [13].

Another contemporaneous surgeon, Erich Lexer, claimed in 1921 to have done a facelift in an actress in 1906. In his paper, he describes the elastic external device she used to pull her skin tighter and describes his operation at that time a success [14]. Lexer uses an S-shaped incision, extending from the temporal area, extending in front of and then behind the ear. He is the first to also describe anchoring sutures, securing the skin to the temporal fascial. As with other of his colleagues, he denies successful outcomes from smaller incisions.

Lexer’s story about the actress, and Hollander, who claimed his first facelift was for a vain Polish aristocrat is similar to the story related by the first recognized female cosmetic surgery, Suzanne Noël. Originally interning as an obstetrician gynecologist, Dr. Noël worked alongside her otolaryngology-trained husband, Henri Pertat, during WWI, becoming familiar with minor surgical procedures. She later characterized herself as a dermatologist. Noël related that the great actress, Sarah Bernhardt, visited her in Paris in 1912, showing her the incisions done by a surgeon in Chicago. These incisions had greatly improved her appearance. In their biographical tribute to Dr. Noël, Dr. Kathryn Stephenson, the first woman editor of *Plastic and Reconstructive Surgery*, and Paula Regnault, the Canadian plastic surgeon who worked with Noël, from 1942 to 1950, suggest that that surgeon might have been Charles Conrad Miller [15].

Dr. Noël was intrigued by the results, and after doing experiments on rabbits to see the effects of small excisions on the tightness of the skin, she began to perform cosmetic surgery. She gradually expanded her incisions and refined her techniques for better results but nearly always relied on what she called the “petit” facelift, doing parts in stages. She used local anesthesia, with her office in her home, and prided herself on the patient’s ability to disguise the surgery, going about normal business even on the day of the operation. Dr. Noël’s work using rabbits appears to be the first actual research done in cosmetic surgery.

In 1926, Noël published a textbook *La Chirurgie Esthétique, Son Role Social* [16]. In addition to describing her own particular approach to facelifting, Noël offered a detailed description of the personal and social benefits of cosmetic surgery, the first to do so in such detail using a case report method. Among the surgical texts devoted to facelifting up to this time, Dr. Noël’s is the only one to include numerous pre- and postoperative photographs. Stephenson and Regnault place Noël firmly in the pantheon of early cosmetic surgery. A more recent paper by the feminist scholar Kathy Davis expands the discussion of Noël’s work to its place in the emerging field of feminist body theory and Noël’s life in the context of her additional roles in suffrage and in Parisian society [17]. Noël recognized the negative role that female aging had on the economic life of her patients. As Davis notes, rather than emphasizing the esthetic value of appearance, Noël justified her cosmetic practice by telling the stories of her patient’s desire for surgery and the difference it made in their life circumstances. Thus, she firmly anchors her practice as a social necessity, providing her patients with the means to support themselves, and maintain their professional successes. Her feminist approach was certainly reinforced by the restrictions she faced herself. Noël practiced for most of her career out of her home office, as women were not granted hospital privileges.

Dr. Noël worked within a community of cosmetic surgeons, active following WWI who

exchanged ideas, observing one another and publishing only after considerable time. Noël is known to have worked with Drs. Thierry Martel, Hyppolyte Morestin, and Jacques (or possibly Max?) Joseph as well as surgeons in the USA. Raymond Passot refers to his own masters, Pozzi and Morestin, as well as to Mlle Pertat in his 1919 article on eliminating cheek wrinkles [18]. Mlle Pertat is described as one of only 25 woman physicians in Paris in a memoir about wartime France published in 1917 [19]. Gertrude Atherton, a prolific journalist and author, describes her visit with Madame Pertat who she reports to be one of the most successful doctors in the city. Visiting her shortly following WWI, Pertat tells Atherton that her goal is to focus her practice now on specializing in skin diseases and facial blemishes. Based on Passot’s inclusion of her among his mentors, in 1919, she succeeded. Madame Pertat appears in Atherton’s book and Passot’s paper. Suzanne Noël’s first husband, also a physician, was Henri Pertat, the name that she carried during that time. So this Pertat, who was thanked by Passot, is Suzanne Noël as she became known after her second marriage, in 1919, to Andre Noël.

The end of WWI saw European surgeons trained in facial reconstruction techniques, such as Passot, Morestin, and Julien Bourguet, apply their skills to various surgical procedures to combat wrinkles and aging. Sir Harold Gilles, in England and many American surgeons who had worked with him avoided cosmetic surgery during this time. These cosmetic pioneers worked under local anesthesia and describe detailed presurgical estimates of skin excision, most using various skin pinching to assay the amount to be removed and the ideal location of the incisions, with attention to avoiding visible scars. Passot [18] and Bourguet [20] each published papers in 1919 describing their approach. An American surgeon, Adalbert Bettman, became the first surgeon in the Western USA to publish on his facelifting technique. Following time observing Dr. Staige Davis in Baltimore (who trained with William Halsted) and Dr. Vilray Blair (who worked with Sir Harold Gilles during the

war) in St. Louis, Dr. Bettman became the first US surgeon to describe his technique and provide photographic documentation of the incisions, as well as before and after images in 1920 [21]. This is also the first description of the continuous pre- and postauricular incision that became commonly used for full facelifting. Bettman recommended the use of procaine hydrochloride and epinephrine for his local anesthesia, as did Noël and many others during that time. The first surgeon to hide part of the preauricular incision behind the tragus was Julien Bourguet in 1925 [22]. In this paper, he also expanded on his technique for removing herniated fat in the lower eyelid, the first description of a transconjunctival approach. His advice regarding the facelift for assuring a satisfactory result should apply today:

Make the pull just a little tighter than you expect the fact to appear as there will be some ‘give.’ Above all, make both sides alike. While it is a fact that the two sides of the face are not exactly alike, be very careful not to accentuate it.

The following year, Bourguet published a much more comprehensive discussion [23]. He is the first to describe transferring fat into the nasolabial fold if the furrow cannot be corrected during the facelift. Bourguet also described the relationship of muscular innervations to wrinkles and recommended injecting the frontal branches of the facial nerve to smooth the forehead. He noted that by paralyzing the nerves to the frontal muscles, the lines of the forehead disappeared. He recommended either injecting branches of the facial nerve with 80% alcohol, noting that this lasted about 8 months, or sectioning the nerves directly, a result that was permanent. He treated the resultant droop of the eyebrows by shortening the fibromuscular cutaneous skin within the hairline, in essence a deep brow lift.

All of these surgeons emphasized removal of the skin without undermining prior to 1927. In that year, O H Bames first described the limits of simple skin excision and promoted the value, not only of undermining but also of face peeling as an adjunct to removal of wrinkles. He also advocated suturing the skin to the fascia, describing it

as relatively inelastic compared to the skin sutured solely to the skin [24].

Blair Rogers, in contrasting the progress of cosmetic surgery in Europe to that in the USA, noted that Conrad Miller in 1907, Kolle in 1911, and Bettman in 1920 tended to describe their operations in great detail, while those in Europe, Hollander in 1912, Passot and Bourguet in 1919, and Joseph in 1921 avoided such details in their papers. Yet, the progress of facelifting surgery progressed between the continents in nearly parallel lines [25]. These pioneers fostered cosmetic surgery as an academic discipline, something that could be taught, learned, and improved upon as they shared their ideas and published their papers and texts.

Cosmetic surgery was attractive to a variety of surgeons. Those mentioned are examples of mainstream practitioners who adopted cosmetic surgery and defended it from charges that it was frivolous. By planning their surgery carefully, often using minimally invasive techniques, local anesthesia, and careful planning, they avoided the bad publicity that came with complications. They explored other aspects of medicine discretely. Kolle was a pioneer in the use of X-rays and an instructor in electrotherapeutics as well as an author of several works of fiction. Hollander was an art historian, publishing *Medicine in Classical Paintings* in 1903 before turning to surgery. Lexer was an art student before deciding on a career in medicine and was an accomplished sculptor. Noël was a feminist and activist, working on behalf of women’s right to work and suffrage as well as one of the founders of European soroptimists. Among early pioneers however were many now long forgotten charlatans, like Arthur Span.

One of the early textbooks of plastic surgery in the USA is H Lyons Hunt’s *Plastic Surgery of the Head, Face and Neck* [26]. In it, he describes the facelift, brow lift, and double chin correction. His chapter on Prosthesis is an indictment of the uses of paraffin. He claimed that while he never injected paraffin, he had excised over 100 paraffinomas and lists in detail 16 specific types of complications that can arise from its use. And like many of his

colleagues writing plastic surgery texts, he devotes a very small part of his book to cosmetic issues, with a four-page section on cosmetic operations on the cheek. His drawings show most of the lifting occurring in several iterations of preauricular/temporal incisions. In another brief commentary, three pages long, he recommends a brow lift via a scalping incision and eradication of glabellar lines by vertical excision.

1.6 Cosmetic Surgery and Cosmetic Medicine

The 1920s experienced not only the development of cosmetic surgical operations for the face but also the widespread interest in rejuvenation via hormones. Brown-Séguard, an eminent nineteenth-century medical scientist, made major contributions in physiology and neuropathology. His discoveries included descriptions of the spinal cord tracks controlling motor and sensory function in papers published in 1850 and 1851. In 1856, he was credited with discovering the first hormone, cortisol, making him the founder of endocrinology. But it was his later work, identifying the role of testicular extract, that is important to cosmetic surgery. At the age of 72, Brown-Séguard's self-experimentation suggested that a testicular substance provided a highly beneficial effect [27]. Using extracts from the testes, testicular blood, and seminal fluid taken from dogs and guinea pigs, he characterized the effect as giving him increased mental concentration, stamina, forearm strength, and an improved jet of urination within 3 weeks. The publication of this work resulted in what seems today to be a wildly overzealous pursuit of a youthful appearance. Kahn reviews the history of hormone replacement therapy in aging and notes that over 12,000 physicians were believed to be involved in the administration of "Brown-Séguard Elixir" in the following decades [28]. The elixirs marketed for such rejuvenation were not necessarily what they claimed to be. Other practitioners, rather than relying on medication, offered testicular transplants. H Lyons Hunt became one of those

doctors, largely abandoning his surgical practice after the publication of his textbook. Hunt published a paper describing the effectiveness of testicular or ovarian transplants for men and women in the medical profession [29] and later a review of 500 such cases [30].

The most famous testicular transplant surgeon of that era, John Brinkley, was not a physician, much less a surgeon, but he became one of the wealthiest medical practitioners of the early twentieth century transplanting goat testicles into men. He used direct mail marketing as well as the radio to promote his practice. In the early 1920s, Brinkley's self-promotion caught the attention of the American Medical Association, and Morris Fishbein, an editor of *JAMA*, began his campaign against medical quackery, and John Brinkley specifically, with a series of articles in *JAMA*, followed by the publication of *The Medical Follies* in 1925 [31]. The feud between the AMA and those they deemed charlatans has a long a colorful history and played an important role in the establishment of the FDA.

Thus, the 1920s saw some surgeons, Hunt among them, who were doing facelifting, also begin experimenting with nonsurgical rejuvenation procedures. For the decades following the introduction of cosmetic surgery, this conflation of cosmetic medicine with surgery and the attraction that this type of practice had for charlatans as well as serious practitioners kept cosmetic surgery on the periphery of the medical establishment.

Conclusion

Facial cosmetic surgery was developed simultaneously by surgeons in Europe and the USA, who saw the benefits of rejuvenation for their patients. The challenges of improving appearance while minimizing evidence of the procedure continue to the present. Early founders of the specialty used the "This is how I do it" approach to their clinical presentations. Incremental improvement, as exemplified by the papers of Passot and Hollander, was the standard. They developed both the mini and the maxi facelift, the use of fillers and toxins,

Table 1.1 Table listing the country of origin, practice, and specialty training, if any, of the founders of facelifting in Europe and the USA

| Name | Birth and death dates | Specialty training | Country of origin | Country of practice |
|-----------------------|-----------------------|-------------------------|-------------------|---------------------|
| Charles Conrad Miller | 1881–1950 | None | USA | USA |
| Frederick S Kolle | 1871–1929 | Radiography | Germany | USA |
| Eugen Hollander | 1867–1932 | Surgery | Germany | Germany |
| Erich Lexer | 1867–1937 | Surgery | Germany | Germany |
| Raymond Passot | 1886–1964 | Surgery | | |
| Adalbert G Bettman | 1883–1964 | Surgery | USA | USA |
| Julien Bourget | 1876–1952 | Head and neck surgery | France | France |
| Jacques Joseph | 1865–1934 | Orthopedic surgery | Germany | Germany |
| H Lyons Hunt | 1882–1954 | | USA | USA |
| Suzanne Noël | 1878–1954 | Obstetrics, dermatology | France | France |

subcision, peels, and surgery for the elimination of wrinkles. There are no new ideas, only reiterations of the past (Table 1.1).

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Abstract

The invaluable preoperative visit remains the most important aspect of the proper patient evaluation prior to surgery. The physician's interview should attempt to gather as much information as possible around the patient's medical and surgical history. The physical exam should be focused and thorough. If indicated, appropriate testing and preoperative workup should be obtained. This chapter will discuss the preoperative workup of the maxillofacial cosmetic surgery patient.

2.1 Medical and Anesthetic History

As with any other surgical discipline, a detailed history is essential before planning the adequate anesthetic approach for maxillofacial surgery patients.

Medical history includes current chronic illnesses, allergies, home medications, and recent infections. Social history includes tobacco and alcohol use. NPO status is a very important element of the assessment. Anesthetic history

includes all previous anesthetic experiences, difficult intubations, and perioperative complications (i.e., postoperative nausea and vomiting).

Consulting previous anesthetic records for perioperative management and laryngoscopy remarks is crucial when available. Finally, body weight, history of obstructive sleep apnea, and functional capacity or activity should also be included.

Imaging procedures and laboratory results relevant to the current issue should also be included. Obviously, the relevant family medical and anesthetic history should also be reported. For women, the reporting should include the obstetric history and the possibility of a current undiscovered pregnancy.

This evaluation will allow identifying patients in need of special anesthetic considerations. For example, a patient with a history of difficult intubation will require the preparation

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of advanced laryngoscopy techniques like video laryngoscopes or fiber-optic scopes. A patient with a history of obstructive sleep apnea might require further pulmonary investigation before proceeding with the surgery.

Patients presenting for cosmetic maxillofacial surgery are usually relatively healthy; nonetheless, statements in the medical history might direct the physician to undiagnosed problems possibly compromising the anesthetic management.

Patients are later classified according to ASA physical status classification system to decide if they are fit for surgery. Adequate history taking will lead to a preliminary estimation of the anesthetic risk of the procedure. This will eventually allow the anesthesiologist to educate the patient about his provisional plan and the possible alternative techniques he might adhere to in case of unanticipated events during the procedure.

After the anesthesiologist has a clear picture of the patient's overall medical status and the possible risks, he will be able to request further imaging procedures and lab tests or treatment plans in order to optimize the medical condition before proceeding with the surgery. This will minimize the surgical and anesthetic mortality and morbidity risk.

Furthermore, a well-outlined medical history will lead to reduction in hospital stay, unnecessary tests and consultations, last-minute delays, and additional costs.

2.2 Airway Assessment

A patient presenting for an elective maxillofacial surgery would undergo a full physical examination, covering the cardiovascular, pulmonary, and neurological systems, with a special attention to the airway.

Keeping in mind that some difficult airways cannot be predicted, the physician should always anticipate any difficult situation.

A full assessment starts with a full history and physical exam:

1. History: any congenital or acquired medical, surgical, or anesthetic condition that involves the airway must be noted.
2. Physical exam: Inspection includes:
 - (a) Length of upper incisors
 - (b) Relationship of maxillary and mandibular incisors during normal jaw closure
 - (c) Relationship of maxillary and mandibular incisors during voluntary protrusion of the mandible
 - (d) Inter-incisor distance
 - (e) Visibility of the uvula
 - (f) Shape of the palate
 - (g) Compliance of mandibular space
 - (h) Thyromental distance
 - (i) Length of the neck
 - (j) Thickness of the neck
 - (k) Range of motion of the head and neck
3. Specific tests: Inter-incisor gap, thyromental distance, and Mallampati score.

Several tests of airway assessment have been described, but no single test alone has enough specificity and sensitivity to accurately predict airway difficulty; therefore, one must rely on a combination of available methods.

In particular, the Mallampati score [1] described in 1983 is still widely used, predicting a difficult airway. The soft palate, uvula, and faucial pillars are inspected with the patient sitting upright protruding the tongue and without phonation. Higher Mallampati scores predict problematic laryngoscopy. The initial description only listed three classes; Samsoon and Young added a fourth class later; a class 0 was also described when even the epiglottis is visible upon mouth opening and tongue protrusion. The classification is as follows:

- Class I: Soft palate, fauces, uvula, and pillars visible
- Class II: Soft palate, fauces, and uvula visible
- Class III: Soft palate and base of uvula visible
- Class IV: Soft palate not visible at all

The predictors for difficult face mask ventilation are:

- (a) Obstructive symptoms
- (b) Poor dentition
- (c) Facial hair
- (d) Mallampati III or IV
- (e) BMI > 30 kg/m²
- (f) Limited mandibular protrusion

The predictors for impossible face mask ventilation are:

- (a) Facial hair
- (b) Male
- (c) Obstructive sleep apnea requiring treatment
- (d) Mallampati III or IV
- (e) History of neck radiation

Some cases may be very challenging to manage, as they can have various facial anomalies that could compromise securing the airway. The difficult airway algorithm as published by the American Society of Anesthesiologists [2] encompasses the adequate approach when faced with a difficult intubation/ventilation.

Initially, an assessment of basic management problems should be done. These range from problems in patient consent/cooperation to assessment of difficulties in ventilation, laryngoscopy, and intubation. At this point we should consider possible ways to provide supplemental oxygen during the process itself.

The next step is to consider the available options and their feasibility:

1. Awake intubation vs regular induction and then intubation
2. Direct/video-assisted laryngoscopy vs invasive/surgical airway
3. Using muscle relaxants vs preservation of spontaneous respiration

A backup plan should be planned at all times to secure the airway if initial attempts fail, since a laryngospasm can occur at any time during

direct or awake fiber-optic laryngoscopy, especially in patient with a hyperactive airway.

2.3 Preoperative Cardiac Evaluation and Assessment

The American Society of Anesthesiologists provides guidelines for preoperative cardiac assessment. These guidelines serve to estimate the perioperative cardiac risk for patients undergoing elective and emergency noncardiac surgery. They also help identifying individuals who need further cardiac tests or interventions to optimize their cardiac status preoperatively.

It is estimated that 25–50% of deaths following noncardiac surgery are caused by cardiovascular complications, and patients who have perioperative MI during a cardiac surgery have a mortality rate of 15–25% [3]. Cardiovascular diseases like hypertension and ischemic or valvular disease are frequently diagnosed in the process of preoperative assessment, which may delay a noncardiac surgery for further investigation.

The recommendations for supplemental preoperative evaluation clearly denote which tests are sensitive or specific to draw a bigger picture of the patient's cardiac status. For example, a 60-year-old patient with jugular venous distention presenting for a maxillofacial surgery should be investigated for heart failure. On the other hand, a young patient with no family history of cardiac diseases or risk factors can proceed to surgery without further workup.

When assessing the cardiac risk, the following factors should be considered:

- Patient-related factors: Age, body habitus (obesity), chronic diseases (diabetes, hypertension, etc.), functional status, medical therapy (beta-blockers, anticoagulants, antiarrhythmics, etc.), implantable devices, and previous surgeries
- Surgery-related factors: type of surgery, urgency of operation, duration of operation, possibility of blood loss, and fluid shifts
- Test-related factors: specificity and sensitivity of a test and its effect on the management

Maxillofacial surgery presents an intermediate cardiac risk (1–5%), but cardiac complications can affect any patient when signs and symptoms are ignored. These signs can be as simple as a decrease in the patient’s functional capacity and the capability to do their daily activities like climbing several flights of stairs or getting around the house.

2.3.1 ACC/AHA Recommendations

The ACC/AHA guidelines set the recommendations on how to approach specific cardiovascular conditions diagnosed preoperatively, to prevent complications during noncardiac surgeries. These guidelines are as follows:

- Step 1: Emergency surgery—proceed to surgery with medical risk reduction and perioperative surveillance.
- Step 2: Active cardiac conditions (unstable coronary syndromes, decompensated HF, significant arrhythmias, severe valvular disease)—postpone surgery until stabilized or corrected.
- Step 3: Low-risk surgery—proceed to surgery.
- Step 4: Functional capacity—proceed to surgery if good FC.
- Step 5. Clinical predictors (ischemic heart disease, compensated or prior HF, cerebrovascular disease, diabetes mellitus, renal insufficiency)—proceed to surgery with HR control, or consider noninvasive testing if it will change management (specifically in for intermediate risk surgeries like maxillofacial procedures).

2.4 Preoperative Pulmonary Evaluation and Assessment

As in other system-based evaluations, the preoperative pulmonary assessment focuses on the reduction of postoperative pulmonary adverse events. According to the National Surgical Quality Improvement Program, major pulmonary

complications accounted for the highest cost increase and increase in duration of stay as compared to cardiac, thromboembolic, and infectious complications [4]. It is therefore imperative to attempt to recognize the risk factors routinely during the preoperative visit.

According to the ACP [5], major pulmonary adverse events, which cause an increase in morbidity, include:

Atelectasis
Pneumonia
Exacerbation of chronic lung disease
Respiratory failure

A deep history taking and physical examination are predictably at the heart of the pulmonary assessment, with a goal to detect known risk factors.

2.4.1 Risk Factor Identification

Most sources stratify the risk factors studied for postoperative pulmonary morbidity in two major groups: patient-related or procedure-related risk factors.

1. Patient-related risk factors:
 - (a) Age: Compared with patients below 60, an odds ratio of approx. 2 was found for patients 60–70 years old and 3 for patients above 70 [6]. It should be kept in mind that these findings contrast with the cardiology guidelines, which don’t state age as an independent risk factor. Here however, it addresses the higher risk of an otherwise healthy individual of advanced age.
 - (b) Chronic lung disease: Most commonly studied is chronic obstructive pulmonary disease, which is a well-known risk factor, with an odds ratio of 1.79 according to the ACP. It was found to be independently associated with postoperative pneumonia, reintubation, and failure to wean from ventilator.

- (c) Cigarette use: Mixed data suggest an increase in postoperative adverse events in current smokers. Improved outcomes have been shown with smoking cessation, with the best outcomes tied to the earliest cessation. It is important to address the issue as soon as possible and provide help and support to patients willing to discontinue their intake.
 - (d) Congestive heart failure: The ACP declares that CHF is a strong independent predictor for postoperative pulmonary complication, with an odds ratio of 2.93 [5].
 - (e) Functional dependence: Total dependence (inability to perform any of the activities of daily living) was associated with a 2.51 odds ratio for pulmonary complications, while partial dependence (need for equipment or assistance for some activities) was associated with a 1.65 ratio.
 - (f) ASA classification: While formulated to predict perioperative mortality rates, it was shown to be associated with risks of both pulmonary and cardiovascular events, with increasing odds along with increasing class. An ASA class II or above was found to be associated with a 4.87 odds ratio for pulmonary complications as compared to ASA class I [6].
 - (g) Obesity: Given the physiologic changes associated with obesity, one would expect an increased pulmonary risk for obese patients undergoing surgery. However, the data has shown no significant increase in pulmonary complications for bariatric patients. It is thus not considered a risk factor [7].
 - (h) Asthma: Recent evidence has refuted older reports of postoperative complications in well-controlled asthmatics. If patient is however symptomatic, it should be a priority to optimize medical care 1–2 weeks preoperatively.
 - (i) Obstructive sleep apnea: Data is suggestive of higher rates of hypoxemia, hypercarbia, and reintubation while not yet included in the ACP guidelines [8]. Many patients present with undiagnosed OSA. A targeted interview and assessment are thus warranted to curb postoperative complication rates.
2. Procedure-related risk factors:
 - (a) Surgical site: Relating to respiratory muscle function, it should come as no surprise that surgery to an area close to the diaphragm (i.e., upper abdominal, thoracic surgery) leads to higher rates of pulmonary complications [9]. Furthermore, major vascular surgery, head and neck surgery, and neurosurgery have also been established as causes for postoperative complications [10].
 - (b) Duration of surgery: It is suggested that in high-risk patients, shorter procedures are to be favored, as it has been determined that longer surgical time, ranging from 3 to 4 h, has been independently associated with an increase in pulmonary complications in the recovery phase [11].
 - (c) Anesthetic technique:
 - While still a controversial topic, many studies have suggested a trend toward more pulmonary complications with the use of general anesthesia. The data are more obvious when using general anesthetic techniques in COPD patients [12].
 - The topic of residual neuromuscular blockade and its association with pulmonary complications has now been extensively studied. The recent recommendations show an increased incidence still of postoperative aspiration and pneumonia associated with residual curarization postoperatively with a train-of-four ratio of <0.9 . An effort should be then made to ensure full reversal of neuromuscular blockade prior to extubation [13].
 - (d) Emergency surgery: Shown to be a significant predictor of postoperative pulmonary complications, with an odds ratio of 2.21 (CI, 1.57–3.11) according to the ACP [5].

2.4.2 Preoperative Pulmonary Testing

During the past few years, the trend around perioperative testing has shifted to become more restrictive, as well as tailored to each different patient or disease. As such, routine testing is no longer the norm. The aim behind this approach is the avoidance of unnecessary testing, which may become inefficient and expensive, and may lead to the evaluation of “borderline” or false-positive results, which in turn lead to unnecessary delays, cost, and potential patient risk.

1. Spirometry: According to the ACP [5], spirometry is only helpful before lung resection surgery and in patients with suspected undiagnosed COPD or worsening of baseline symptoms. Positive results will be then helpful to identify patients who might benefit from more aggressive preoperative management.
2. Chest radiography: While frequently ordered for preoperative evaluation, chest x-rays only rarely provide unexpected information that alters preoperative management. Some evidence claims this test to be helpful in patients over 50 years of age with known cardiopulmonary disease, undergoing upper abdominal, thoracic, or AAA surgery.
3. Pulse oxygen saturation: As part of the ARISCAT index, measurement of baseline SpO₂ is a helpful tool for patient risk stratification.
4. Serum albumin: The ACP recommends serum albumin testing for patients with clinical signs that may indicate hypoalbuminemia and the test to be considered in patients with one or more risk factors for pulmonary complications. It has been found that hypoalbuminemia (<35 g/L) is a stronger risk factor than any patient-related risk factor for the development of postoperative pulmonary complications [6].

| | β Regression coefficients | Score* |
|---|---------------------------|--------|
| Age (year) | | |
| ≤50 | 0 | 0 |
| 51–80 | 0.331 | 3 |
| >80 | 1.619 | 16 |
| Preoperative SpO ₂ | | |
| ≥96% | 0 | 0 |
| 91–95% | 0.802 | 8 |
| ≤90% | 2.375 | 24 |
| Respiratory infection in the last month | | |
| No | 0 | 0 |
| Yes | 1.698 | 17 |
| Preoperative anemia (Hb ≤10 g/dL) | | |
| No | 0 | 0 |
| Yes | 1.105 | 11 |
| Surgical incision | | |
| Peripheral | 0 | 0 |
| Upper abdominal | 1.480 | 15 |
| Intrathoracic | 2.431 | 24 |
| Duration of surgery (h) | | |
| <2 | 0 | 0 |
| 2–3 | 1.593 | 16 |
| >3 | 2.268 | 23 |
| Emergency procedure | | |
| No | 0 | 0 |
| Yes | 0.768 | 8 |

*Three levels of risk were indicated by the following cut-offs: <26 points, low risk; 26–44 points, moderate risk; and ≥45 points, high risk

ARISCAT Assess Respiratory Risk in Surgical Patients in Catalonia, *Hb* hemoglobin, *SpO₂* arterial oxyhemoglobin saturation by pulse oximetry

2.4.3 Risk Prediction

With the goal of providing a quantitative risk estimate, four different indices have been developed: the ARISCAT risk index, the two Gupta calculators (one being specific for postoperative respiratory failure, the other for postoperative pneumonia), and the Arozullah respiratory failure index. A risk index can be useful in guiding caretakers to identify patients most likely to benefit from risk-reduction interventions, as well as advising patients during the preoperative visit. Three of the four mentioned indices are not optimized for clinical use or manual calculation, with the remaining one being the most widespread.

| | Multivariate Analysis OR (95% CI) n = 1,624* | β Coefficient | Risk Score† |
|---|---|------------------------|----------------|
| Age, yr | | | |
| ≤50 | 1 | | |
| 51–80 | 1.4 (0.6–3.3) | 0.331 | 3 |
| >80 | 5.1 (1.9–13.3) | 1.619 | 16 |
| Preoperative SpO ₂ , % | | | |
| ≥96 | 1 | | |
| 91–95 | 2.2 (1.2–4.2) | 0.802 | 8 |
| ≤90 | 10.7 (4.1–28.1) | 2.375 | 24 |
| Respiratory infection in the last month | 5.5 (2.6–11.5) | 1.698 | 17 |
| Preoperative anemia (≤10 g/dl) | 3.0 (1.4–6.5) | 1.105 | 11 |
| Surgical incision | | | |
| Peripheral | 1 | | |
| Upper abdominal | 4.4 (2.3–8.5) | 1.480 | 15 |
| Intrathoracic | 11.4 (4.9–26.0) | 2.431 | 24 |
| Duration of surgery, h | | | |
| ≤2 | 1 | | |
| >2 to 3 | 4.9 (2.4–10.1) | 1.593 | 16 |
| >3 | 9.7 (4.7–19.9) | 2.268 | 23 |
| Emergency procedure | 2.2 (1.0–4.5) | 0.768 | 8 |

* Because of a missing value for some variables, three patients were excluded. Logistic regression model constructed with the development subsample, c-index = 0.90; Hosmer-Lemeshow chi-square test = 7.862; $P = 0.447$. † The simplified risk score was the sum of each β logistic regression coefficient multiplied by 10, after rounding off its value.

CI = confidence interval; OR = odds ratio; PPC = postoperative pulmonary complications; SpO₂ = oxyhemoglobin saturation by pulse oximetry breathing air in supine position.

Fig. 2.1 The ARISCAT [14] risk index

The ARISCAT risk index [14] assigns weighted point scores to seven independent risk factors (Fig. 2.1):

1. Advanced age
2. Low preoperative oxygen saturation
3. Respiratory infection within the past month
4. Preoperative anemia
5. Upper abdominal or thoracic surgery
6. Surgery lasting more than 2 h
7. Emergency surgery

This test was proven to be quick and easy to perform at the bedside and allowed practitioners

to stratify patients into low, intermediate, and high risk for postoperative pulmonary complications, which are equivalent to 1.6%, 13.3%, and 42.2%, respectively.

The practitioner should be able to identify the risk factors during the preoperative visit, which would enable him to devise a more tailored operative plan to the individual patient. The patients placed in the higher risk group would be directed toward a more conservative approach, which would allow any modifiable risk factor to be optimized prior to surgery.

2.5 Preoperative Fasting Guidelines

While pulmonary aspiration of gastric contents is a rare condition, it entails significant morbidity and mortality. As such, the American Society of Anesthesiologists (ASA) has developed a series of guidelines to decrease incidence of aspiration (last revised in 2011). The data used to develop these guidelines revolves around decreasing preoperative gastric volume, which is used as a surrogate endpoint for aspiration. As the increase in particulate matter, gastric volume, and gastric acidity may be tied to a worse outcome, the recommendations aim to decrease all three. It is to note that high-powered studies are lacking in this field, and much of the evidence remains equivocal [15].

2.5.1 ASA Recommendations for Preoperative Fasting

The following recommendations apply to healthy patients of all ages undergoing elective surgery. They are to be followed whenever a patient is set to receive general or regional anesthesia, as well as monitored anesthesia care (MAC). The rationale is that any sedative anesthetic may decrease or completely obtund protective airway reflexes, thus increasing risk of regurgitation of gastric contents and subsequent aspiration.

1. Clear fluids:
 - (a) Examples of which are water, coffee or tea without milk, juice without pulp, and carbonated beverages (protein and lipid free).
 - (b) Should not be consumed less than 2 h prior to induction of anesthesia.
 - (c) Clear liquids have been shown to empty rapidly from the stomach, with gastric volumes returning to baseline after 90 min, regardless of volume and whether the beverage contained sugars [16].
2. Breast milk:
 - (a) Recommendation is for otherwise healthy neonates and infants.
 - (b) Should not be consumed within 4 h of induction of anesthesia.
3. Nonhuman milk, infant formula, and light meal:
 - (a) A light meal is illustrated by toast or cereal and a clear liquid drink.
 - (b) Formula and nonhuman milk are considered different, due to the possibility of clump formation, which might then act as a solid. They are also the only case where the ASA recommends the volume intake to be considered.
 - (c) Should not be consumed within 6 h of surgery.
4. Solid food, fatty meals, and meat:
 - (a) Fats and protein take a considerable time before leaving the stomach.
 - (b) The guidelines here are the same across all patient populations.
 - (c) A fasting time of 8 h or more should be observed in these cases.
 - (d) A special consideration for pediatric populations: fasting for over 8 h increases risk of hypoglycemia. Consider replacement or monitoring.
 - (e) Gastric tube feeds are fat and protein rich and should be treated as solids and stopped 8 h preoperatively, especially with patients with uncuffed tracheal tubes or tracheostomies [17].

Concerning the pharmacological treatment to decrease acidity or to speed up gastric emptying,

multiple medications have been brought forward to this end (metoclopramide, sodium citrate, etc.). The ASA however recommends no routine administration of any of those medications and advises reliance on clinical sense to treat patients on a case-per-case basis, as the evidence is still insufficient on this matter.

2.5.2 Special Considerations

A mistake encountered frequently in any hospital would be that of patients omitting important per os medications in an effort to keep up with the preoperative fast. In the case of patients on multiple systematic medications, those should be taken up until the day of surgery, early in the morning, with a few sips of water. Notorious medications, which would cause morbidity, were to be stopped abruptly including beta-blockers [18] and antiepileptic agents, among others.

2.5.3 Special Patient Populations

As mentioned above, the guidelines developed for *nil per os* (NPO) status were built around otherwise healthy adult and pediatric patients. It is still however valuable to mention patients whose digestion and gastric emptying are affected by illness.

1. Diabetic patients with gastroparesis:
 - (a) Gastroparesis is a syndrome found, according to some studies, in up to 50% of patients with long-standing diabetes mellitus of both types I and II.
 - (b) It is defined as delayed gastric emptying in the absence of a distal obstruction [19].
 - (c) Symptoms include early satiety, nausea, vomiting, bloating, and abdominal pain.
 - (d) There are no true recommendations concerning preoperative fasting, but it is to note that these patients may benefit from a prolongation on the current guidelines on healthy patients.
 - (e) As always, it is important to assess each patient clinically and treat accordingly.

2. Pregnant patients:
 - (a) No consensus about the gestational age after which pregnant patients to be considered full stomach.
 - (b) The issue here is twofold: Firstly, the size of the uterus compresses all neighboring intra-abdominal structures, increasing symptoms of reflux as much as urinary frequency, which acts as a mechanical cause for reflux and potential aspiration. Secondly, female sexual hormones may be the cause for an observed decreased tone of the lower esophageal sphincter throughout pregnancy.
 - (c) Gastric emptying however has been shown to be on par with nonpregnant patients [20].
3. Obese patients:
 - (a) No significant increase of risk of aspiration found yet, potentially slower gastric emptying [21].

- If a surgery is considered a life-saving emergency, treatment will have to be conducted in the operating room.
- If a self-limiting disease is present and might affect rates of intraoperative and postoperative complications, the surgery should be delayed until patient improves.

Conclusion

The invaluable preoperative visit remains at the helm of proper patient assessment prior to surgery. The physician's interview should attempt to gather as much information around the patient's medical history, the physical exam should be focused and thorough, and both should guide the physician as to whether further testing should be warranted. The overall trend in the past 50 years has been to gradually tailor our treatment modalities to treat each patient individually, in order to reap the most benefit from our interventions, be it on the economical side or that of risk modification.

2.6 When to Postpone Surgery?

It is important to note at this point in our topic that clinical cases cannot be standardized to an exact outcome. Patients will always react differently to anesthetics and surgical procedures, and the physician's clinical sense will be invaluable as always.

There are no clear-cut indications for surgical cancelation, as too many factors enter the equation: the patient's age, the type of surgery to be performed, whether it's an emergency, the patient's coexisting illnesses.

In an effort to find a common denominator, we should keep in mind that a few points remain valid across the whole spectrum:

- Decision to postpone surgery should be driven by concern for the patient's safety.
- The key to most coexisting disease management lies in medical optimization. The clinician should ask himself whether the patient is receiving the best possible treatment for his condition. Is the illness affecting his daily life? Would he benefit from a more aggressive approach to testing and treatment?

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Abstract

A facial cosmetic surgeon must have an in-depth knowledge of facial anatomy and its associated hard and soft tissues. It is imperative for the surgeon to be able to appreciate and define elements of facial aesthetics as well as the stigmata of an aging face. It is critical to be able to objectively analyze facial features preoperatively in order to correctly identify areas of concern and to arrive at an appropriate treatment plan for each patient. This chapter will review how to properly perform an aesthetic facial evaluation.

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3.1 Evaluating the Patient

Paradigms in cosmetic facial surgery are changing. Facial cosmetic rejuvenation is no longer just filling in wrinkles or lifting ptotic eyelids. The approach to achieving an aesthetically satisfying result for the patient must take into account all the factors that led to the consultation including the patient's personal cosmetic goals, concerns and questions surrounding the surgery, the patient's medical and surgical history, as well as the physical and psychological state of the patient. As Greer stated lucidly in 1984, "understanding the motives, expectations, and desires of a patient seeking cosmetic surgery is at least as important as manual dexterity for achieving consistently satisfactory results" [1]. Hence, during the initial consultation, establishing a strong physician-patient rapport will invariably result in more effective communication on both parties.

Patients are given the opportunity to detail their desired cosmetic look and articulate inquiries of the surgical procedure and postoperative convalescence, while the surgeon convey expectations and limitations of what surgery can achieve for the patient. In addition, it behooves the surgeon to ascertain that patient is fully informed of the nature of the surgery, including surgical benefits, risks, and complications, and nonsurgical alternative therapies [2]. By doing so, the surgeon serves as the patient's advocate to navigate the cosmetic medical landscape in harmony with the patient to achieve better patient satisfaction and, ultimately, excellent surgical outcome.

3.1.1 Introduction

The rich history of facial cosmetic surgery is rooted in antiquity from ancient Egyptian's artistic idealization of aesthetic facial proportions to Greek philosophers' formal study of beauty as a discipline. As canonized by Greek philosophers, Plato and Aristotle; refined by Renaissance artists, da Vinci and Vesalius; and revisited in contemporary times by Farkas, Powell, and Humphreys, "anthropometrics" is the study of human body and its proportions [3]. These aesthetic canons have established the foundation of a paradigm of aesthetic parameters to guide surgeons in the evaluation of human facial proportions and in recognition of deviation from the norm. More than just a set of paradigm, these canonical tenets have withstood the test of time, being as relevant in modern era as it were in antiquity. Despite generational differences in concept of beauty, there remains a certain set of facial symmetry, proportions, and balance of anatomic contours that are universally desired as aesthetically attractive [4].

Perhaps, no generation has paid more exquisite attention to one's physical facial image than in the twenty-first century, where a multitude of social media outlets provide one with direct access to and exhibition of one's physical attractiveness. Such microscopic perusal of one's aesthetic features, while promulgating one's desirable attractive features, can negatively result

in jealously, embarrassment, and decreased self-esteem. In fact, as Neligan and Warren describe, attractive individuals exhibit more positive social attributes, are offered greater opportunities for employment, are more likely to be promoted, and have higher expectations of success [2, 5]. Conversely, less attractive individuals are more likely to be jettisoned for social dates, job offers, or promotions despite similar qualifications and distinction as their more attractive peers. Moreover, pediatric studies demonstrate that craniofacial deformities can adversely influence children's self-image, learning, behavior, and participation in social activities. As the potential benefits of an attractive appearance extend beyond enhanced self-esteem, it is not surprising that there is an increasing societal acceptance of cosmetic surgery as evidenced by the increasingly extensive media coverage that normalizes such procedures [2].

Hence, the current trend toward increasing consumer demand for cosmetic surgeries corroborates the efficacy and success that such procedures can offer patients both enhanced youthful beauty and normalize deviations from norm to correct facial deformities [2, 6, 7]. As such, the study of the aesthetic canons confers to the cosmetic surgeon an indispensable tool to recognize aberrations from the standard proportions and employ surgical and nonsurgical modalities to help patients restore youth, beauty, and harmony. Of course, such appraisal must also include the patient's physical makeup, excess or shortage of skin, intrinsic facial anatomy, and underlying soft tissue as these physical features can be employed to achieve an aesthetically pleasing result [3–5].

3.1.2 Facial Anatomic Landmarks

Facial Analysis Terms

1. **Frankfort horizontal:** imaginary line drawn from superior margin of external auditory meatus through inferior orbital rim (Fig. 3.1).
2. **Glabella:** the smooth prominence of the forehead above and between the eyebrows (Fig. 3.2).

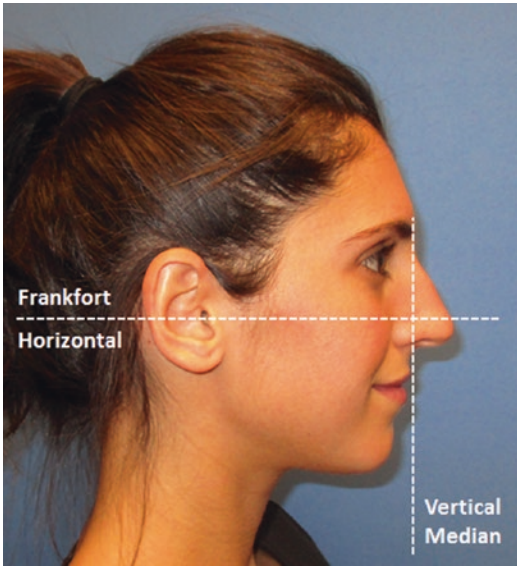


Fig. 3.1 Right profile view: Frankfort horizontal line passes through tragus and bisects the vertical median line drawn tangential to the vermilion border of upper lip

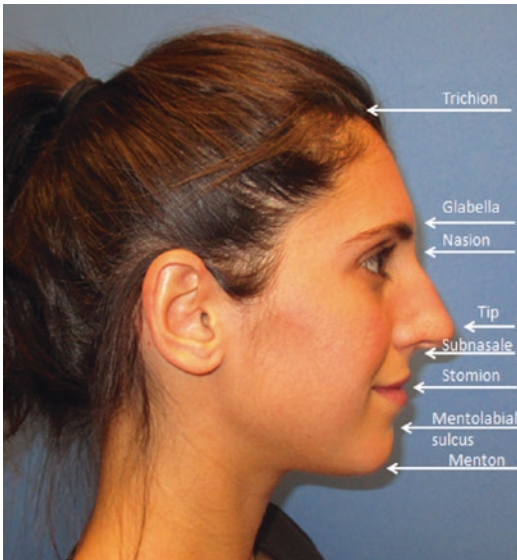


Fig. 3.2 Right profile view detailing anatomic landmarks. Trichion: hairline. Glabella: most prominent forehead flanked by superior orbital rims. Nasion: intersection of frontal and nasal bone. Tip: farthest promontory point from nasal dorsum. Subnasale: point where nasal septum and upper lip meet in midsagittal plane. Stomion: midpoint of oral fissure between upper and lower lips. Mentolabial sulcus: horizontal groove between lower lip vermilion border and the chin. Menton: farthest promontory point of mandible symphysis

3. **Gnathion:** the lowest point on the anterior margin of the lower jaw in the midsagittal plane.
4. **Gonion:** the midpoint at the angle of the mandible.
5. **Labrale superius:** vermilion border of the upper lip.
6. **Menton:** most inferior soft tissue point on chin.
7. **Nasal tip:** the most anterior projection of the nose on profile.
8. **Nasion:** the depression just superior to the bridge of the nose, interting the frontal bone and two nasal bones.
9. **Orbitale:** the most inferior point on the infraorbital rim.
10. **Pogonion:** most forward-projecting point on the anterior surface of the chin.
11. **Radix:** a depression at the root of the nose corresponding to the nasofrontal suture. The position of the radix significantly influences the overall balance of the nasal profile. It impacts nasal contour, length, angulation, and height. The radix marks the origin of the nasal dorsum, directly influencing nasal length. Positioning the radix more cephalically has the effect of lengthening the dorsal line, while caudal displacement shortens the nasal length [8, 9].
12. **Rhinion:** the anterior tip at the end of the suture of the nasal bones.
13. **Sellion:** osseocartilaginous junction of the nasal dorsum.
14. **Stomion:** central portion of interlabial gap, anterior point of contact between upper and lower lip.
15. **Subnasale:** junction of columella and upper lip. The point at which the nasal septum merges in the midsagittal plane with the upper lip.
16. **Trichion:** anterior hairline in the midline of the forehead.

3.1.3 Facial Proportions

The initial patient evaluation starts with the surgeon assessing the face for proportions and symmetry. The Frankfort horizontal plane is a line

which helps define the horizontal plane in the skull so that other facial parameters can be measured more accurately (Fig. 3.1). The Frankfort line is an imaginary line drawn from the opening of the external auditory meatus to the inferior margin of the infraorbital rim and helps standardize the framing of lateral profile photographs such that measurements can be recorded [4, 6, 10]. The human face is three dimensional but can be delineated on a two-dimensional visual platform into aesthetic units. The major units of facial analysis include the forehead, eyebrows, eyes, nose, ears, lips, chin, and neck. These aesthetic units are based on the classical golden ratio of Phi $\Phi = 1.618$, which characterizes the divine proportion [2]. While there is truth to the axioms that “beauty is in the eye of the beholder” and the concept of beauty varies by race, culture, and generation, there is a universal commonality in the perception of physical beauty. As certain facial attributes more closely reflect phi in their proportions, the overall appearance of the facial units appear harmonious and attractive. On the contrary, deviations that detract

from the golden proportion secondary to congenital anomalies, trauma, injury, and aging will distort facial symmetry and result in an aesthetically displeasing facial appearance. Therefore, precise planning of cosmetic surgery requires meticulous analysis of the individual facial subunits that conform to and deviate from established norms such that the surgeon can correct distortions to achieve balance and harmony for an aesthetically pleasing outcome.

On a frontal view, the two-dimensional rendition of the human face can be framed vertically into three successive zones as demarcated by three imaginary horizontal lines at level of the trichion, subnasale, and menton (Fig. 3.3). The first third is measured from the midline of the trichion to the glabella, the second third from the glabella to the subnasale, and the last third from the subnasale to the menton. When examining the lower third zone, it is further divided into thirds: the distance from subnasale to stomion is one third and distance from stomion to gnathion is two thirds (Fig. 3.4). It is critical to mention

Fig. 3.3 (a) Front view: horizontal lines passing through hairline, glabella, subnasale, and menton divide the face into equal thirds. (b) Right profile view: horizontal lines passing through hairline, glabella, subnasale, and menton divide the face into equal thirds

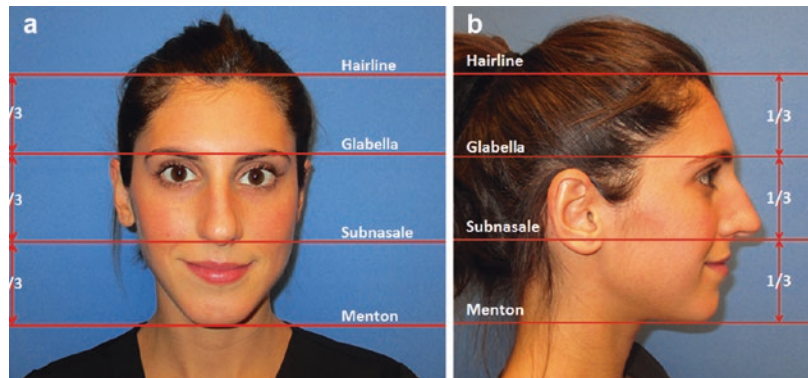
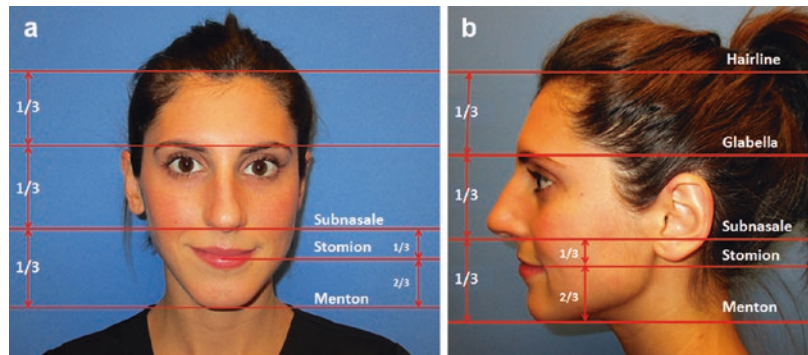


Fig. 3.4 (a) Front view: horizontal lines passing through stomion at union of superior and inferior labia divide lower third into upper one third and lower two thirds. (b) Left profile view: horizontal line through stomion divides lower third zone into upper one third and lower two thirds



two key caveats with these measurements. First, receding hairline with age accentuates the upper third thereby distorting the vertical facial proportions [7, 11, 12]. Second, in women, the upper half of the face is not uncommonly greater than the lower half, whereas men have larger lower half due to more prominent mandible. Furthermore, there are great proportional variations due to individual variation, ethnicity, and hairstyles that create an illusion of a greater or smaller upper third of the face [13]. Alternatively, a second method of assessing facial height excludes the upper third of the face because of common variability regarding hairline positions. Instead, measurements are made from the nasion to the subnasale and from the subnasale to the menton.

Facial width is evaluated by dividing the face into equal fifths (Fig. 3.5). The width of one eye should equal one fifth of the total facial width, as well as the intercanthal distance or nasal base. Vertical line drawn through the medial limbus of each eye should intersect the lateral margin of the oral commissure (Fig. 3.6). The ideal attractiveness of the facial profile stems from the harmony of the symmetrical fifths and can be better appreciated when one considers deviation from this norm [2, 4]. For example, congenital malformations from neuroectodermal crest dysplasia that

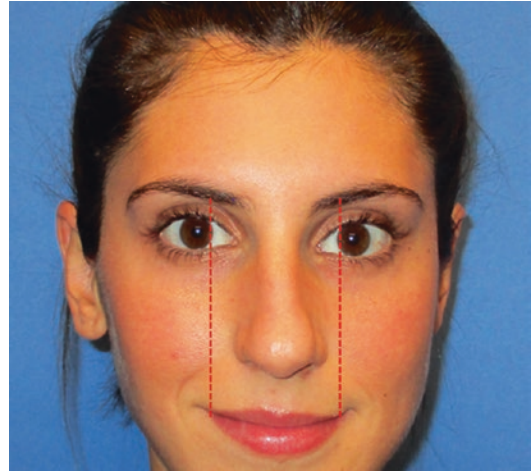


Fig. 3.6 Vertical line drawn through medial limbus should intersect the lateral most edge of oral commissure

results in either hypotelorism or hypertelorism undermine the symmetry and distort the appearance [14, 15]. While widely or narrowly spaced eyes may exude a “cuteness” factor in young children, such distortions in a grown individual create an unnatural look and arouse suspicions of the individuals’ cognition. Corrective surgery to alleviate the distorted widths helps restore symmetry and balance to achieve aesthetically pleasing outcome.



Fig. 3.5 Front view: vertical lines dividing the face into five equal zones, each measuring width of palpebral fissure. Distances are equal from helix to lateral canthus, from lateral canthus to medial canthus, and between the two medial canthi

3.1.4 Forehead

The forehead is a critical landmark of the face as a gentle curvature contour of the forehead creates an aesthetic feature of the face. The boundaries of the forehead are from the trichion to the glabella that comprise the upper third of the face. On a profile view, the nasofrontal angle is formed by a line drawn parallel to the glabella that intersects a line tangent to the nasal dorsum, with an ideal aesthetic value between 115° and 135° (Fig. 3.7). Small changes to the forehead prominence at supraorbital ridges can dramatically affect nasal length and projections and perturb the proportions. As Nahai describes, aging results in receding hairline and subsequent loss of connective tissue matrix, collagen, and elastin that previously serve to confer youth and



Fig. 3.7 Nasofrontal angle is formed by two intersecting lines, one drawn parallel to glabella-nasion plane that bisects second line drawn parallel to nasal dorsum

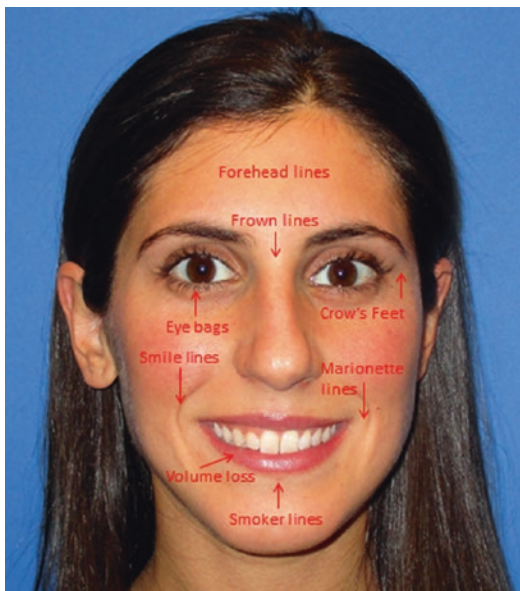


Fig. 3.8 Location of facial rhytids in the aging face

vibrancy to the facial contours [4]. Such rhytids form perpendicular to the long axis of their corresponding muscle fibers. For instance, the vertical muscle fibers of the frontalis muscle are associated with horizontal forehead rhytids, while the circular muscle fibers of the orbicularis oris are associated with vertical-radiating rhytids

or smoker's lines (Fig. 3.8). These phenomena distort the forehead proportions and undermine what was once animated and attractive. To address this issue, rhytidectomy and nonsurgical modality such as botulinum toxin can be used to smooth wrinkles, rejuvenate the skin, and restore youth and beauty.

3.1.5 Eyebrow

There is great variation in what is considered to be a “beautiful brow,” and the facial plastic surgeon must appreciate the nuanced fundamentals of eyebrow configuration that contribute to the overall balance and harmony of the face [16]. Through makeup, aesthetic changes to the eyebrow enhance not only the intrinsic beauty and power of the brow but also that of the eyes and face. The facial plastic surgeon can produce similar nuanced cosmetic changes with surgery. Distinct elevation or depression of the medial, central, and lateral portions of the eyebrow can be achieved with a multitude of advanced brow and forehead lifting techniques. In essence, the ideal shape, length, and position of the eyebrow must be appropriately altered to fit different shapes of faces. Thus, when surgically creating the ideal eyebrow position, the facial plastic surgeon should assess the individual characteristics of the eyebrows and their relationship to the eyes and face to determine the most aesthetic brow appearance for that individual.

The ideal brow tip aesthetic line should follow a smooth and gently curving arc along the superior orbital rim (Fig. 3.9). The brow should begin medially and gradually taper toward its lateral end. An oblique line drawn from the lateral alar margin of the nose through lateral canthus should intersect lateral aspect of the eyebrow (Fig. 3.10). The medial and lateral ends of the brow lie approximately at the same horizontal level for balance and harmony. In women, the eyebrows should lie 2–4 mm above the supraorbital rim with the brow arch apex just superior to the midpoint of lateral limbus and lateral canthus (Fig. 3.11). Important aesthetic quality desired by women is to have the lateral brow raised to

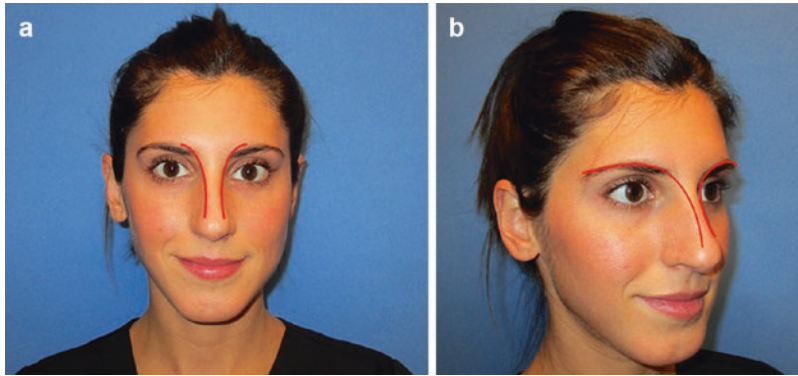


Fig. 3.9 (a) Brow tip aesthetic line formed by medial margin of superior orbital rim as it curves medially and narrows along lateral nasal bone before curving outward along nasal tip. (b) Oblique view: brow tip aesthetic line

should frame the eyebrows and follow a smooth curve trajectory before straightening to form the contours of the nasal dorsum

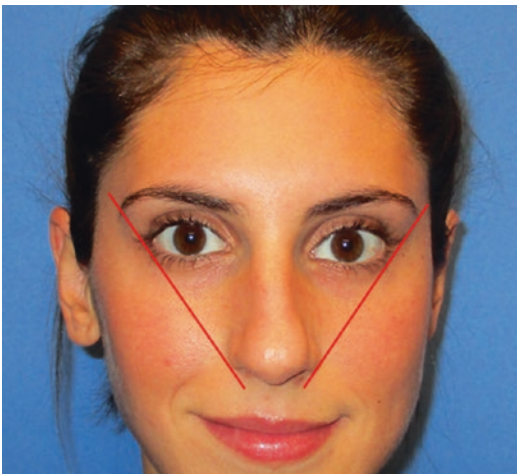


Fig. 3.10 Oblique line drawn from the lateral alar margin of the nose through lateral canthus should cross lateral extent of the eyebrow



Fig. 3.11 Brow arch apex should lie just superior to the midpoint of lateral limb and lateral canthus in women

achieve a sharp elegance [2, 4]. On the contrary, in men, eyebrows are less arched and should lie at level of supraorbital rim to achieve straight, commanding appearance [2, 4]. However, these ideal eyebrow positions in men and women can vary with style trends, and the uppermost point may actually lie anywhere from the lateral limb to the lateral canthus.

Of note, there are common pitfalls in eyebrow aesthetics to consider. As Yalcinkaya advises, surgical over-elevation of the brow and medial placement of the brow apex create an unnatural “surprised” look. This exaggerated brow apex

can feminize men’s appearance, creating an undesirable overarched eyebrow [16]. Moreover, a low medial brow with a high lateral apex creates a frustrated “angry” look. And overdepression of the eyebrow exudes an uncharacteristic “fatigued” look. Similarly, aging can result in eyebrow ptosis, which crowds the supraorbital region and creates an imbalance to the facial third proportions by exaggerating the middle one third. Thus, while it is not feasible to define an ideal eyebrow that is suitable for every face, the facial plastic surgeon must understand the essentials of brow aesthetic elements and be attuned to the

nuances of present-day style, patient preference, gender differences, and periorbital features when performing brow surgery.

3.1.6 Eyes

Perhaps no facial feature makes a more striking first impression than the pair of eyes. The size, shape, color of eyes, eyelashes, eyelids, and periorbital structures all confer important aesthetic features to the overall appearance of the eyes and the face as a whole. The width of the eyes from medial to lateral canthi should be equidistant and one fifth of the width of the face. Additionally, vertical lines drawn through medial canthus, medial limbus, lateral limbus, and lateral canthus should divide the eye into symmetrical thirds (Fig. 3.12). Hypertelorism and hypotelorism create an unbalanced facial proportion and detract from the symmetry. The concept of the canthal tilt is critical as it serves as an important indicator of aging [2, 4]. A positive canthal tilt with lower eyelid lightly tilting upward along lateral canthus exudes youth, health, and exuberance. On the contrary, a depressed tilt projects old age, melancholy, and weariness. Likewise, the periorbital area should appear full and voluminous for a

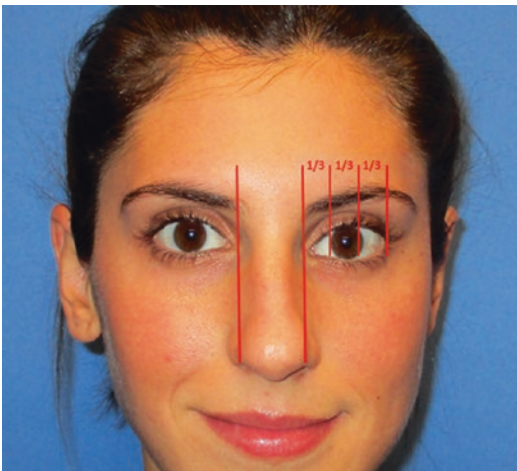


Fig. 3.12 Vertical line drawn through lateral nasal tip passes through medial canthi. Vertical lines drawn through medial canthi, medial limbus, lateral limbus, and lateral canthi divide eye into three equal vertical zones

youthful appearance. Any hollowing can display a sense of sickness or old age.

As noted by Papel, the upper eyelid should have an almond-type shape with an acute upper eyelid angle medially, followed by a gentle angle laterally [7, 17]. The upper lid should cover 1–2 mm of superior limbus, while lower eyelid should be at level of inferior limbus. Eyes with incomplete creases over which eyelid skin drools down give the illusion of a single eyelid. As a result, the eye seems small and swollen over the region of the line. On the other hand, an eye with double eyelid appears to be more attractive and refined because they exude a sense of vibrance through larger eyes and well-matched eyelashes. Not uncommonly, this phenomenon is a common indication for blepharoplasty among Asians, who desire a more prominent upper eyelid to minimize the appearance of eyelid drooping and achieve a more Western look [18–20].

Both sun damage and aging can contribute to weariness appearance of the eyes. Ptotic eyelids secondary to superior tarsal of muller and/or levator palpebrae superioris muscle weakness also contribute to a lethargic appearance. For droopy upper lids, blepharoplasty can remove excess skin to restore a crisper crease in the upper lid, thereby making the eyes look more open, alert, and youthful. Similarly, for lower edematous eyelids which can form tear trough deformity, the use of fat grafting can correct the tear trough deformity by blending the junction of the lower lid and cheek, making it less distinct and creating a smoother more naturally aesthetic appearance [7, 17].

3.1.7 Nose

The nose is the central unit of the face and plays an indispensable role in facial aesthetics. Minimal deviations are immediately recognizable and distort the symmetry and balance of the face. On lateral profile view, the radix is the deepest part of the nose and is aligned vertically with the pupil, separating the nose from the forehead. The radix demarcates the transition point from the glabella to the dorsum of the nose. The dorsum



Fig. 3.13 Nasofrontal angle is formed by two intersecting lines, one drawn parallel to glabella-nasion plane that bisects second line drawn parallel to nasal dorsum

runs from the radix to the nasal tip and is frequently described in colloquial terms as the bridge of the nose as it connects the tip of the nose to the nasal tip (Fig. 3.13). The dorsum has considerable influence over the aesthetics of the nose and the entire face. From frontal view, the dorsum should form graceful aesthetic lines from the medial border of eyebrows to the nasal tip (Fig. 3.9). These lines should track smoothly, and any interruption or asymmetric position of these lines will cause the nose to look uneven and disproportional [2, 4]. Similarly, on a lateral profile view, the dorsum should exhibit a continuous graceful line extending from the glabella to the nasal tip. While there is not an ideal shape of the dorsal aesthetic lines, it is critical that they run parallel through the nasal bridge with a gentle horizontal tapering as they near the nasal tip to achieve a harmonious aesthetic appearance.

A common reason for cosmetic facial evaluation for rhinoplasty is correction of dorsal hump, a deviation caused by large nasal bones, large septum, or frequently both [21–23]. The dorsal hump is routinely caused by underlying enlargement of perpendicular plate of the ethmoid bone, vomer spurs, and quadrangular cartilage. Correction of the dorsal hump involves reducing

the aforementioned bones and cartilages to the desired height to reestablish the graceful dorsal aesthetic lines [24, 25]. Removing too little will not resolve the issue. Likewise, removing too much will create a ski jump or saddle nose deformity. The dorsal hump, as a central entity of the human face, confers important aesthetic qualities unique to each gender, to achieve strength, dominance, authority, demureness, and elegance. For instance, men may prefer a nose with a hint of a dorsal hump, which exudes strength, power, and stability. On the contrary, women may desire a straight dorsum or, not uncommonly, a slightly concave dorsum. The slight concavity produces a certain feminine beauty about the nose, whereas, a straighter contour of the dorsum achieves a more sophisticated look. Of course, other tangible and intangible factors including ethnicity, societal appraisal of the ideal nose, age, facial proportions, and personal preference all factor into the decision-making process to achieve an individually tailored aesthetic result.

On worm's eye view, the columella, or "small column" in Latin, forms the central buttress of the nasal tip [2, 26] (Fig. 3.14). Flanking the columella are bilateral ala, or "wings" in Latin, that

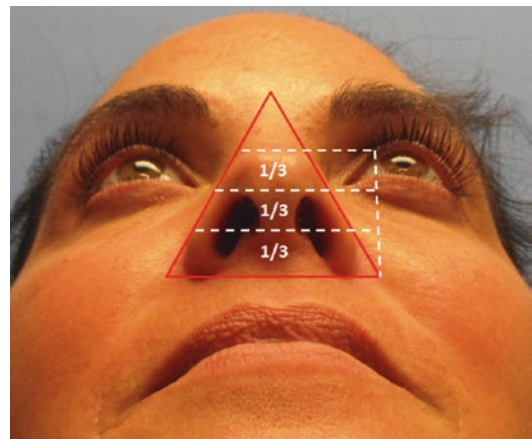


Fig. 3.14 Worm's eye view. Columella is divided into three equal zones: upper lobular, middle, and basal zones. The nasal tip and bilateral alar rim form three equal legs of an equilateral 60–60–60 triangle. Ideal width of nose at base of alar margin should equal the intercanthal distance between two medial canthi. The nostril should comprise two thirds of the length of the columella. Nasal tip occupies the remaining one third

create the rounded curved cartilaginous folds of the nasal base. The undersurface of the columella and ala house the medial and lateral crural cartilages that are frequently manipulated during rhinoplasty and septoplasty to correct nasal tip and septal deviation, respectively. Another common indication for initial consult is widened nasal bridge. When confronted with a wide intercanthal distance, the facial plastic surgeon should be cautious about further widening the nasal bridge. However, wider bridge may benefit for patient with short intercanthal distance by conferring an illusion of greater width and thereby further separates the distance between the two medial canthi.

3.1.8 Ears

Well-proportioned ears create a harmonious balance that nicely adorns the lateral edges of the face. The ideal ears should span from the brow superiorly to the nasal supratip or base of nasal columella inferiorly (Fig. 3.15). The base of the tragus begins approximately a single ear length adjacent to the lateral orbital rim. The width of the ear should be 50–60% of its length (Fig. 3.15). The ear's vertical axis is inclined posteriorly at 15°–30°. The upper third of the helical rim should measure 1–1.2 cm from the scalp; the middle third should measure 1.6–1.8 cm at midpoint of the helix; and the lobule should be located 2–2.2 cm from the mastoid. The normal angle between the helical rim and the mastoid is 25°–35° and that between the concha and scaphoid

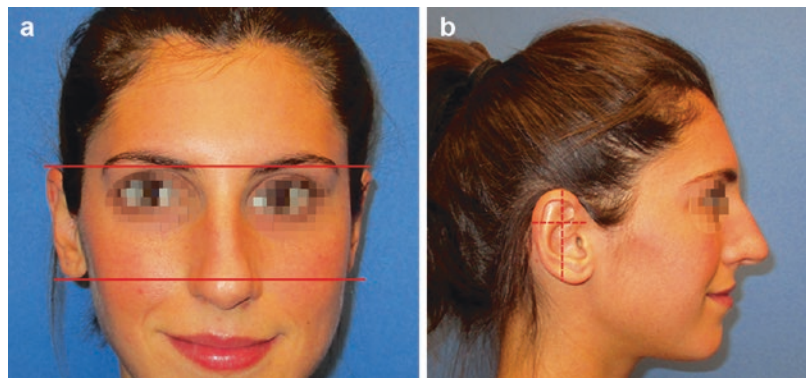
fossa is 80°–90°. Prominent laterally protruding ears are usually the result of either underdeveloped antihelix and/or large conchal bowl and are a frequent indication for corrective surgery.

As Shiffman describes, when assessing for prominent ear deformity, the facial plastic surgeon must be attuned to the following five key features: (1) underdeveloped or absent antihelical fold, (2) increased distance between the helical rim and scalp, (3) increased angle between the mastoid and the helical rim, (4) overdeveloped concha with deep conchal wall, and (5) protruding lobule. By doing so, the facial plastic surgeon can better treat congenital ear defect or injury-induced distorted ears to achieve a more natural shape, thereby restoring balance and proportion to the ears and face. Additionally, surgical correction of even minor ear deformities not only improves the individual's aesthetic appearance but also raises self-esteem. In particular, children with oversized, asymmetric, or malpositioned ears are frequently embarrassed by their physical deformity and are not uncommonly teased at school [27]. With attention to the aesthetic tenets, facial plastic surgeons can reduce the apparent size of large, prominent ears, restore symmetry and balance, and reshape the children's ears to look more natural and aesthetically pleasing.

3.1.9 Lips

Traditionally, full and luscious lips appear more youthful and contribute to facial beauty as well as evoking cardinal human emotions [2, 7]. The

Fig. 3.15 (a) Front view: apex of helix should be at level of eyebrows and glabella, while inferior tip of lobule should be at level of nasal supratip or base of columella. (b) Profile view: width of ear is approximately 50–60% of length



upper lip extends from subnasale to stomion and comprises one third of the total lip height, while the lower lip and chin are measured from stomion to gnathion and comprise the second two thirds. Ideal width of oral commissure should be defined by vertical line drawn from medial limbus (Fig. 3.6). The philtra columns extend parallel from the columella to the Cupid's bow of the upper lip (Fig. 3.16). The lower lip has a gentle inward convex curvature to the gnathion of the chin. In full smile, two third of the crown should show (Fig. 3.16). Certain ethnic differences should be noted. In the Caucasian race, there is a central fullness in the upper vermilion border, while in the Black race, the upper and lower vermilion heights are more balanced with equal proportions [13].

Additionally, soft tissue structures surrounding the lips also contribute to and detract from facial beauty. For instance, aging, sun damage, and scars all undermine underlying tissue elasticity and architecture, which manifest as timeworn prominent nasolabial folds, perioral rhytids, smoker's lines, and marionette lines [28]. With aging, significant volume loss in the lips and peri-

oral tissues leaves a thin deflated vermilion that rolls inward. The upper lip lengthens and sags creating a melancholic effect. With repeated muscle action, the soft tissues at the oral commissures wear down leaving downturned and inverted corners. The progressive volume loss can extend further downward to the mandibular border creating prominent marionette lines with a dejected appearance [2, 7]. Thus, the rejuvenation of lips and the perioral area is of prime concern to many patients. Fortunately, both surgical and nonsurgical modalities such as hyaluronic acid and dermal fillers and non-ablative lasers are highly effective in counteracting these changes to restore youth and beauty. Of note, Sataloff advises that keeping the facial harmony and balance by treating other perioral regions for volume loss rather than simply focusing on augmenting the lips can avoid a poor aesthetic result and achieve a more natural outcome [28].

3.1.10 Chin

The chin has a profound effect on facial beauty and much of the character of the lower face. A strong chin has classically been associated with positive attributes such as strength, confidence, and assertiveness. Conversely, a weak chin is associated with weakness, indecisiveness, and reticence. The chin is defined superiorly by labio-mental sulcus and inferiorly by gnathion. Changes to these vertical and horizontal contours can dramatically impact the appearance of the lower lip, labio-mental sulcus, and contour of the neck. Vertical height of the chin should be twice the distance measured from subnasale to stomion (Fig. 3.4). Chin projection measured by angle between a line through the glabella and subnasale and line from subnasale through pogonion, angle should be 11° . A common indication for chin augmentation is a hypoplastic mentum secondary to aging or genetic hypoplasia of the mandible [2]. Moreover, it behooves the surgeon in the initial evaluation to consider occlusal abnormalities as they pertain to microgenia and micrognathia. While the former can be corrected with chin augmentation, the latter usually requires orthognathic



Fig. 3.16 Philtral columns align the double peaks of Cupid's bow to base of columella or nostril opening

advancement of the mandible. Nonsurgical alteration of the chin can be achieved by botulinum toxin injections and dermal fillers used as implants for enhancement.

3.1.11 Neck

The neck is commonly an overlooked aesthetic component when evaluating the cosmetic patient. Nonetheless, the shape and contour of the neck markedly affect the appearance of the chin and lower face. Powell and Humphreys defined the cervicomental angle as a line of the neck drawn from the glabella to the pogonion intersecting a second line drawn from the menton to the innermost point between the submental area and the neck (Fig. 3.17). The angle created between the two intersecting lines should ideally be between 85° and 95° with acceptable ranges of 90° – 110° . As delineated by Connell [29] and Neligan [2], the hallmark of a youthful face is a well-contoured neck. Neligan further describes the critical measurements of the neck to include a modified ver-

sion of Powell and Humphreys' cervicomental angle of 105° – 120° , a distinct inferior mandibular border, a slightly visible thyroid cartilage, and a visible anterior sternocleidomastoid border [2].

Here, it's noteworthy to discuss the clinical anatomy to better appreciate how aging contributes to neck laxity and how the SMAS facelift technique can tighten the neck muscle and reposition the submental fat to recreate youthful vivacious neck contour. The three important planes in the neck are (1) superficial plane between the skin and the platysma, (2) superficial cervical fascia that envelope the platysma, and (3) deep cervical fascial plane containing the subplatysmal fat, the anterior belly of the digastric muscles, and the submandibular glands [2]. The platysma muscles are thin, bilateral structures that are continuous with the superficial musculoaponeurotic system (SMAS) in the face, extending from the clavicles inferiorly and abutting the lower angle of the mandible. Because the platysma extends into the SMAS superiorly with minimal anchoring to the bone, any laxity in the SMAS is transmitted back to the platysma and neck. Hence, aging directly contributes to neck laxity through causing platysma and surrounding soft tissue disintegration. Similarly, elderly decline in collagen, elastin, and submental fat further worsens the skin laxity and results in more obtuse cervicomental angle, thereby creating a more laden, weighed-down appearance. In patients whose neck-chin contour has been distorted negatively with aging, the neck and face are ideally treated simultaneously with both necklift and facelift to correct the obtuse neck droop, reestablish the harmonious cervicomental angle, and rejuvenate facial beauty.



Fig. 3.17 Cervicomental angle is created by two intersecting lines, one drawn parallel to horizontal plane of submental region from menton to hyoid bone and a second line drawn parallel to vertical plane of neck

Conclusion

Facial analysis must be performed in a systematic manner in order to adequately evaluate different facial subunits. A thorough understanding of factors that impart harmony, balance, as well as youth and beauty is critical in order for the cosmetic surgeon to arrive at an individualized diagnosis and execute the proper procedure for each patient.

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Abstract

Wound healing is the biological response to tissue injury. It is a multifaceted and dynamic process. An overall understanding of this complex mechanism will optimize postoperative wounds as well as their appearance. This chapter will review the phases of wound healing and factors that can affect this process. Agents that can optimize this response will also be discussed.

4.1 Introduction

Wound healing is the biological response to tissue injury. The wound healing process involves a complex interplay of components, with disruptions leading to non-healing wounds or abnormal scarring. Local and systemic factors influence outcomes, as does maintenance of the healing environment. An overall understanding of the

mechanism behind tissue healing remains integral to optimizing postoperative wounds and their appearance.

4.2 Basic Anatomic Concepts

The skin is comprised of two layers, the epidermis and dermis. The epidermis represents the outer barrier to the environment, providing physical protection, temperature regulation, and pigmentation. The epidermis can be subdivided into the stratum basale, stratum spinosum, stratum granulosum, stratum lucidum, and stratum corneum. Close to 90% of the epidermis is composed of keratinocytes, which serve as a barrier to microorganisms and a means to minimize moisture loss [1]. Other cell populations include melanocytes, Langerhans cells, and Merkel cells.

The dermis underlies the epidermis. Composed of thick fibroelastic tissue, the dermis is responsible for the strength and flexibility of the skin.

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A superficial layer is named the papillary dermis, containing fingerlike projections known as papillae, which carry capillary blood to the epidermis. Deep to the papillary dermis is the reticular dermis. Within this thick connective tissue layer are blood vessels and hair follicles, as well as oil and sweat glands.

4.3 Phases of Wound Healing

Wound healing can be divided into four main phases: hemostasis, inflammation, proliferation, and remodeling (Fig. 4.1). When the steps are perturbed, wound healing may delay or arrest, leading to improperly healed wounds or chronic non-healing wounds.

4.3.1 Hemostasis

Hemostasis is the body's initial response to minimize blood loss. Wounding leads to the rapid release of inflammatory cytokines, such as thromboxane and ADP, which promote vasoconstriction of the surrounding vessels [2]. Catecholamines are released into the systemic circulation by the adrenal medulla, further slowing blood loss [3].

The formation of a temporary platelet plug provides primary hemostasis. Upon encountering damaged endothelium, platelets increase the expression of surface receptors that enable binding to the site of injury. Degranulation by platelets releases ADP and thromboxane, which in

turn recruits more platelets [4]. The overall aggregation of platelets forms a temporary plug at the damaged vessel wall.

The creation of a blood clot, known as coagulation, represents secondary hemostasis. The coagulation cascade is comprised of intrinsic and extrinsic pathways, which ultimately lead to the conversion of fibrinogen to fibrin. The intrinsic pathway becomes triggered by elements within the blood vessel, while the extrinsic pathway activates due to blood extravasation [5]. Both pathways lead to the activation of Factor X, which in turn activates the enzyme thrombin. Thrombin plays the key role in converting fibrinogen into fibrin. Fibrin strands strengthen the platelet plug and additionally trap erythrocytes in the meshwork, creating a mature clot [6].

4.3.2 Inflammatory Phase

In the inflammatory phase, cells and cellular elements migrate to the site of healing. In order to facilitate cellular transport, the initial period of vasoconstriction is followed by the release of histamine and subsequent vasodilation. Neutrophils predominate in the first 48 h following tissue injury and serve to clean the wound from microbes and cellular debris [7]. Macrophages play a primary role in wound healing over the following days. In addition to debriding the wound, macrophages secrete growth factors to promote new tissue creation as well as chemokines to recruit fibroblasts and endothelial cells [8].

4.3.3 Proliferative Phase

The proliferative phase is responsible for the generation of granulation tissue. Three to five days after injury, fibroblasts begin depositing collagen in the wound defect. Type III collagen predominates in the early period, with type I collagen eventually becoming the principal form [9]. Collagen adds strength to the healing wound and aids in restoring skin integrity.

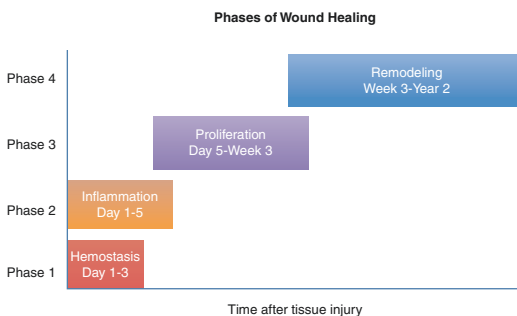


Fig. 4.1 The four phases of wound healing

Angiogenesis at the wound site gives granulation tissue its characteristic bright red color. New vessel formation is facilitated by the angiogenic factors secreted from macrophages in response to local hypoxia [10]. Increased blood flow provides granulation tissue with the necessary oxygen and nutrients to sustain the healing process.

Epithelialization of the wound surface occurs as epithelial cells migrate from the wound edges across granulation tissue. This process results in a protective barrier from the environment. Wound size is made smaller by contraction, in which myofibroblasts at the periphery of the wound function to bring wound edges closer together. Contraction commences soon after tissue injury and continues for 2–3 weeks [11].

4.3.4 Remodeling Phase

Remodeling is the final stage of wound healing, starting several weeks after injury and lasting up to 2 years. During this period, the rates of collagen synthesis and breakdown equalize, and type III collagen is replaced by type I collagen. Collagen fibers become organized and cross-linked, further reinforcing the wound.

The tensile strength of the wound gradually increases over time. At 3 weeks, the wound has achieved 20% of its full strength [12]. A wound's maximum tensile strength peaks at 3 months, where it reaches at 80% of its pre-injury level [13].

4.4 Factors Affecting Wound Healing

Patient characteristics play an important role in the rate and integrity of wound healing. Nutrition, age, diabetes, smoking status, and concomitant medications are among the significant factors impacting tissue repair on a systemic level. Microenvironmental factors, such as oxygenation, infection, necrotic tissue/foreign bodies, and wound tension, are also important considerations.

4.4.1 Systemic Factors

4.4.1.1 Nutrition

Adequate nutrition is central to optimal wound healing. Deficiencies in carbohydrates, proteins, fats, and vitamins have long been identified as inhibitors of tissue regeneration. Protein plays a primary role, as shortages can lead to reduced deposition of collagen in the wound matrix and an increased likelihood of wound dehiscence [14]. Animal studies have also demonstrated decreased tensile strength of wounds in the presence of low protein intake [15].

Vitamins A, C, and E are important cofactors. Vitamin C deficiency bears historical significance owing to its connection to the condition scurvy. Vitamin C is necessary for the hydroxylation of proline and lysine residues in pro-collagen, thereby providing mature collagen fibers with their structural integrity [16]. Vitamin A enhances the post-injury inflammatory response, increasing the number of monocytes and macrophages at the wound site [17]. It also likely has a stimulatory response on collagen accumulation and fibroplasia in healing wounds [18]. Vitamin E is a potent antioxidant and provides a stabilizing effect on cell membranes [19]. Topical application of vitamin E has been widely used to speed wound healing and improve the appearance of scars, based mostly on anecdotal evidence. However, scientific studies remain conflicting, with several clinical trials failing to show improvements in wound healing or scar appearance with topical vitamin E application [20–22].

4.4.1.2 Age

Increasing age is associated with declines in healing capability. Elderly patients (over 60 years old) exhibit temporal delays in wound healing, although the final scar quality may end up equivalent to that of younger patients [23]. Factors contributing to slower wound healing in the elderly include decreases in epithelialization, macrophage migration, angiogenesis, and collagen synthesis [24].

4.4.1.3 Diabetes

Patients with diabetes are particularly susceptible to wound healing complications. Diabetes is associated both with delays in healing of acute wounds and a heightened risk of developing chronic, non-healing wounds [25]. Nearly all phases of wound healing are impaired in the diabetic patient, an effect compounded by the reduced ability to fight infection and the common presence of neuropathy [26, 27].

4.4.1.4 Smoking

Smoking is associated with decreased capillary perfusion and tissue hypoxia [28, 29]. In maxillofacial surgery, smoking has been identified as a risk factor for delayed postoperative healing and heightened risk of infection [30]. Cosmetic outcomes are worse in patients who smoke, as seen with increased complication rates and tissue necrosis rates as well as a higher likelihood of required reoperation [31].

4.4.1.5 Concomitant Medications

Glucocorticoids are used to treat a wide range of inflammatory disorders. Acute corticosteroid use (<10 days) has not been shown to produce a clinically significant effect on wound healing [32]. Chronic use, however, impairs wound healing through several mechanisms. Glucocorticoids cause decreases in collagen synthesis and epithelialization, along with deficiencies in inflammatory cell migration [33]. As a result, patients on chronic steroids experience delayed healing as well as increased rates of surgical site infections and wound dehiscence [34, 35].

Nonsteroidal anti-inflammatory drugs (NSAIDs) are frequently used for pain management as well as several inflammatory conditions. Short-term, low-dose use of NSAIDs does not appear to play a significant role in recovery following soft tissue injury [36]. However, the effects of long-term treatment with NSAIDs have not been fully elucidated. Animal studies indicate that NSAIDs may retard inflammation and reduce fibroblast proliferation, leading to decreased wound strength [37, 38]. Temporary discontinuation of NSAIDs, aspirin, and anticoagulants prior to cutaneous surgery may be advocated by clini-

cians as a means to reduce bleeding, but these measures should be weighed against the individual patient's increased risk of vascular complications [39].

4.4.2 Local Factors

4.4.2.1 Oxygenation

Tissue oxygenation is essential to cellular metabolism and directly impacts healing outcomes. After injury, wounds experience local hypoxia. This state results from reduced vascular supply, an increased oxygen demand of healing tissue, and the depletion of oxygen as inflammatory cells generate reactive oxygen species [40]. Although acute hypoxia stimulates the wound healing process, chronic hypoxia has a detrimental effect, eventually leading to anaerobic metabolism and inadequate ATP production [41]. Importantly, the administration of supplemental oxygen in the perioperative period has been shown to decrease the incidence of surgical wound infections [42].

4.4.2.2 Infection

Wounding causes a disruption of the skin barrier and facilitates the entry of bacteria into the tissues. Infection results from an imbalance between host defenses and bacteria, commonly defined when bacterial growth exceeds 100,000 organisms per gram of tissue [43]. Bacteria release pro-inflammatory endotoxins, such as interleukin 1 and tumor necrosis factor alpha, with continued contamination causing the inflammatory process to be abnormally prolonged [44]. Persistent infection leads to wound healing failure and the creation of a chronic wound.

4.4.2.3 Necrotic Tissue/Foreign Bodies

Necrotic tissue impairs wound healing by promoting bacterial growth and increasing the risk of infection. In addition, nonviable tissue releases toxic products and impedes granulation tissue formation and reepithelialization [45]. Foreign bodies in the wound elicit an acute inflammatory response. When these materials are retained, a chronic inflammatory response develops, which can be accompanied by the destruction of adjacent tissue, delayed wound

healing, and infection [46]. Debridement of nonviable tissue and foreign bodies is critical for the healing process to complete [47].

4.4.2.4 Tension

The tension present at closed wound edges affects the ability for the wound to heal. When wounds are closed under high tension, the strength and vascularization rate of regenerated tissue is impaired [48]. Obese patients are more susceptible to increased tissue pressure from wound closure, which can lead to decreased oxygenation and a heightened risk of dehiscence [49].

4.5 Acute vs. Chronic Wounds

4.5.1 Acute Wounds

Acute wounds occur due to trauma or surgical intervention. The integrity of the skin is breached and varying amounts of tissue loss may be incurred. Acute wounds progress through the phases of wound healing without noticeable disruption and typically heal within several weeks.

4.5.2 Chronic Wounds

Chronic wounds begin as acute wounds but experience perturbations in the wound healing process. Wounds are typically defined as chronic when signs of healing have not begun by 4 weeks and healing has not completed by 3 months [50]. Chronic wounds are sources of significant morbidity and mortality, affecting up to 6.5 million patients in the USA [51].

4.6 Healing by Primary vs. Secondary vs. Tertiary Intention

4.6.1 Primary Intention

Primary closure refers to the process of directly approximating edges of the wound. Sutures, staples, skin glue, or skin tape may all be appropriate

methods of closure. Healing occurs with the aid of fibrous adhesion and minimal formation of granulation tissue.

4.6.2 Secondary Intention

With increased tissue loss or local tissue disruption, wounds may not be directly amenable to primary closure. In such cases, healing by secondary intention occurs, in which the wound bed is allowed to granulate and gradually fill the tissue defect. Healing by secondary intention occurs more slowly than by primary intention and may result in more noticeable scarring [52].

4.6.3 Tertiary Intention

Healing by tertiary intention may also be referred to as delayed primary closure. This method is most commonly employed in contaminated wounds, where immediate primary closure risks the development of infection. The wound is thoroughly cleaned and typically observed for several days until primary closure can be performed.

4.7 Excessive Scarring

4.7.1 Hypertrophic Scarring

Hypertrophic scarring occurs when collagen is overproduced at the wound site. This excess collagen deposition gives rise to a raised scar, which may be pruritic or painful [53]. Factors that impact the risk of hypertrophic scarring include age, infection, high wound tension, ethnicity, and degree of wound trauma [54].

4.7.2 Keloids

Similar to hypertrophic scars, keloids are pathological scars that result from the abnormal proliferation of dermal tissue at the wound. However, in contrast to hypertrophic scars, keloids extend beyond the geometric boundaries

of the wound. Keloids are unlikely to regress and often recur following surgical excision [55]. The etiology of keloid scar formation remains unclear; genetic predisposition appears to play an important role, with an incidence of keloid formation as high as 16% in African-American and Hispanic patients [56].

4.8 Agents that Optimize Wound Healing

4.8.1 Dressings

A wide range of wound dressings are utilized in clinical practice. Gauze-based applications historically served as a mainstay, but advances in synthetic materials have allowed for more tailored approaches to varying types of wounds. Different types of dressings may be appropriate for a single wound as it progresses through the various stages of healing.

4.8.1.1 Hydrocolloids

Hydrocolloid dressings contain carboxymethylcellulose, pectin, gelatin, and elastomers, which form into a gel-like substance over the wound. This material absorbs exudate and helps to create a warm and moist healing environment. Hydrocolloid dressings are occlusive and commonly used for wounds with mild to moderate exudate, including minor burns and pressure sores [57].

4.8.1.2 Alginates

Alginate dressings are derived from the alginic acid of seaweed and contain calcium and sodium salts. Alginates are highly absorptive and ideal for wounds with large amounts of exudate, such as advanced ulcers and full-thickness burns. Due to their dehydrating properties, alginates should not be used on dry wounds or those with hard overlying eschar [58].

4.8.1.3 Hydrogels

Hydrogel dressings are made from networks of hydrophilic polymer chains that hold significant water content. The polymer gel allows wounds to

absorb water from the dressing, providing a hydrating effect. Hydrogels dressings are therefore used for necrotic or dry wound beds and should be avoided when heavy exudate is present [59].

4.8.2 Debriding Agents

Enzymatic debridement is used as a supplement or alternative to surgical debridement in treating wounds with necrotic tissue. These products contain exogenous enzymes that are designed to degrade nonviable tissue while preserving healthy areas of granulation. Enzymatic agents are commonly used for optimizing wound beds in pressure ulcers, leg ulcers, and partial-thickness wounds and may be preferred to sharp debridement in patients with bleeding disorders or those on anticoagulant therapy [60].

Collagenase and papain-urea-based products are the most commonly used enzymatic agents. Collagenase is derived from the bacterium *Clostridium histolyticum* and contains peptidases that digest collagen in the triple helix form. Papain-urea is activated by necrotic tissue, which stimulates it to degrade fibrinous material. Both collagenase and papain-urea formulations have been shown to provide debridement benefits in wounds with high bacterial loads [61].

4.8.3 Topical Antibiotics

Although the use of topical antibiotics in treating cutaneous infections is well established, its prophylactic role in the healing of non-infected wounds remains less clear. Some studies have demonstrated reduced infection rates with antibiotic versus placebo ointments after minor skin trauma and following suturing of lacerations [62–64]. However, evidence also exists that petroleum-based ointments may be equally efficacious to antibiotic preparations in promoting wound healing after minor cutaneous procedures, while also avoiding the potential for contact dermatitis caused by antibiotic ointments [65].

4.8.4 Growth Factors

Growth factors are important mediators in the wound healing process. These cytokines promote inflammatory cell migration, stimulate cell proliferation, and upregulate extracellular matrix deposition [66]. Platelet-derived growth factor BB (PDGF-BB) is a topical growth factor (produced as the drug becaplermin) that has received FDA approval in the treatment of diabetic ulcers. Additionally, PDGA-BB has shown utility in the healing of separated surgical wounds [67].

Conclusion

Wound healing is a multifaceted and dynamic process. Numerous components of the immune system integrate to achieve this objective, which may be affected on a systemic or local level by characteristics specific to each patient. Advances in wound dressings and adjuvant therapies have aided in speeding recovery and reducing chronic wounds. Addressing the wound healing process from the beginning of tissue injury allows the clinician to maximize proper wound healing and patient satisfaction.

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Improving Pain Management in Maxillofacial Cosmetic Surgical Procedures

5

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Abstract

Pain still ranks among the highest patient as well as surgeon concerns for unwanted surgical outcomes. Thus, pain management plays a major role in patient satisfaction and improving our patients' safety. This chapter will review the pathogenesis of pain. Strategies for pain control will be addressed. Additionally, various pain disorders which can affect our patients' postoperative course will be discussed.

5.1 Introduction

Maxillofacial surgeons have become increasingly interested in postoperative pain control. This has occurred in part not only as an attempt to improve the patient experience, but it also has been mandated by the Joint Commission on Accreditation of Health Care Organizations. In 2001, Joint Commission on Accreditation of Health Care Organizations required adequate assessment, monitoring, and treatment of pain as a condition for hospital accreditation [1].

Despite advancements in the understanding of the pathophysiology of pain and pharmacotherapeutics, pain remains poorly treated in both the inpatient and ambulatory setting [2]. It has been reported that 30–80% of patients undergoing outpatient surgery encountered moderate to severe pain postoperatively [1, 3]. Pain still ranks among the highest patient and physician concerns for undesirable surgical outcomes, and if poorly treated, there is a risk for the development of chronic pain and its incumbent morbidity [4].

Onset of pain usually begins about 6–8 h, as the effects of the anesthetics subside [5, 6]. Unless treated, moderate to severe pain usually occurs during the first 24 h, with a peak intensity after recovering from anesthesia. Today, acute postoperative pain is recognized to have two components, an earlier inflammatory component and a later neuropathic component. Just alleviating the inflammatory component in susceptible patients might not be sufficient; addressing the

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neuropathic pain component can be equally important in the prevention of chronic pain [7].

An understanding of the pathogenesis of pain and the two major components that contribute to perioperative pain is essential for adequate management of postoperative pain after maxillofacial surgical procedures.

5.2 Neurophysiology of Postoperative Pain

Acute pain felt at the moment of injury results from activation of the nociceptive sensory endings in the affected tissues. This direct nociceptive activation usually subsides within minutes after withdrawal of the noxious stimulus, but the resulting pain often lasts much longer from hours to days. In surgery, the nociceptive stimulus endures in the presence of extensive chemically sensitized traumatized tissues. Acute pain occurs because inflammatory mediators such as cytokines, bradykinin, and prostaglandins are released from injured and inflammatory cells. Those substances activate the nociceptive sensory endings at the site of tissue damage. Nociceptors demonstrate reversible plasticity in response to inflammatory mediators. The activation threshold of nociceptors is lowered, resulting in enhanced pain sensitivity at the site of tissue injury called peripheral sensitization [8] (Table 5.1).

Potassium and adenosine triphosphate (ATP) are the most common substances involved in the

immediate response to tissue damage and pain. Because potassium channels are responsible for the major resting permeability in all sensory neurons, an increase in potassium will excite those neurons as well as nociceptors. ATP opens non-specific cation channels on sensory neurons, thus causing their excitation upon ATP release during tissue damage [9]. Whenever blood vessels are damaged, the prekallikrein is converted to kallikrein, resulting in the release of bradykinin into the tissues. Bradykinin has a wide range of proinflammatory actions: it is a potent activator of nociceptors, increases vascular permeability, promotes vasodilatation, and induces chemotaxis of leucocytes [10]. Serotonin can be released from activated platelets and from mast cells [11]. Serotonin is able to activate nociceptors and potentiate the effect of other inflammatory substances such as bradykinin [11]. Following tissue damage, the phospholipids of the cell wall are broken down to arachidonic acid mediated by the enzyme phospholipase A2. Arachidonic acid is then converted to prostaglandins, prostacyclins, and thromboxanes by the enzyme cyclooxygenase (COX) and to leukotrienes and hydroperoxy acid derivatives by lipoxygenase [12]. COX-2 is associated mainly with inflammation and pain. PGE2 does not produce pain directly, but it does sensitize receptors on afferent nerve endings to the actions of bradykinin and histamine [13]. Bradykinin in turn stimulates the release of prostaglandins, causing a mutual potentiation [14]. In animal models, the hyperalgesia produced by prostacyclin is immediate and of short duration, with a maximum effect of 2 h [15]. This is in contrast with PGE2 which is characterized by a slow onset and a long duration exceeding 3 h [14].

In addition, tissue injury facilitates the release of a multitude of neuropeptides from the body of the nerve cell of the A delta and C fibers, namely, substance P and CGRP. These neuropeptides will modulate inflammation by altering the release of histamine and prostaglandin PGE2. Tissue pH decreases immediately after incision and is sustained for several days and then recovers by the seventh day in parallel with wound healing [16]; also it has been shown that tissue injury increases also lactate concentrations [17]. The peak occurs

Table 5.1 Effect of inflammatory mediators on nociceptors and pain

| Mediator ^a | Effect on nociceptors | Pain effect |
|-----------------------|-----------------------|-------------|
| Potassium | Activate | ++ |
| ATP | Activate | ++ |
| Serotonin | Activate | ++ |
| Bradykinin | Activate | +++ |
| Histamine | Activate | + |
| Prostaglandins | Sensitize | ± |
| Leukotrienes | Sensitize | ± |
| Substance P | Sensitize | ± |
| CGRP | Sensitize | ± |

^aATP indicates adenosine triphosphate; *CGRP* calcitonin gene-related peptide

on postoperative day four and returns to normal levels between postoperative days 7 and 10. The facilitation of pH responses by lactate is suggested to be a mechanism of ischemic pain [18].

Nonsteroidal anti-inflammatory drugs inhibit cyclooxygenase-2 enzymes and the production of prostaglandin E2 and hence reduce peripheral sensitization and pain [8]. The inflammatory pain, secondary to local stimulation, usually subsides once the source of the mediators disappears, upon tissue healing or when the disease process is controlled [7]. Heightened pain sensitivity can contribute to healing by protecting the damaged body part until repair has occurred [8]. The pain that lasts hours to days after surgery is not a direct result of the initial injury but a delayed reaction of a series of changes in the peripheral tissues and the associated trigeminal nucleus.

In response to painful stimuli, the central nervous system (CNS) also demonstrates plasticity, and pain signal within the spinal cord can be enhanced. With ongoing nociceptive input, the stimulus–response relationship is altered, and an increase in excitability of the CNS may occur, known as central sensitization. Clinically, this might manifest as an increased response to painful stimuli (hyperalgesia) or pain secondary to normally non-painful tactile stimuli (allodynia) [19]. Central sensitization refers to an abnormal amplification of incoming sensory signals in the CNS, particularly in the spinal cord or the trigeminal nucleus. Central sensitization amplifies not only the signal of nociceptors but also the low-threshold A-beta sensory fibers. This is why the A-beta touch input will be felt as pain [20]. Hence, central sensitization is associated with windup, long-term potentiation and secondary hyperalgesia. Windup follows repeated stimulation of C fibers and activation of the glutamate at NMDA (*N*-methyl-D-aspartate) receptors. Normally, a magnesium ion blocks the NMDA receptor. With persistent painful stimuli, the magnesium block dissipates, and the response of second-order neurons to painful stimuli is amplified. The use of NMDA receptor antagonists in pain management, such as ketamine, is useful in attenuating or blocking windup [21]. The response of second-order neurons may outlast the

initial stimulus, and this is known as long-term potentiation, contributing to hyperalgesia. Pain threshold outside the area of inflammation (secondary hyperalgesia) decreases because of increased activation of second-order neurons in the dorsal horn of the spinal cord.

Nerve injury plays a role in chronic postsurgical pain. Following nerve damage, spontaneous ectopic discharges from injured nerves and nearby uninjured nerves lead to spontaneous pain [22]. The increased nociceptive input into the dorsal horn leads to central sensitization [23]. This concept of excess excitatory transmission and loss of inhibitory transmission leads to an unfettered barrage of CNS input from the dorsal horn and facilitation of pain transmission [24].

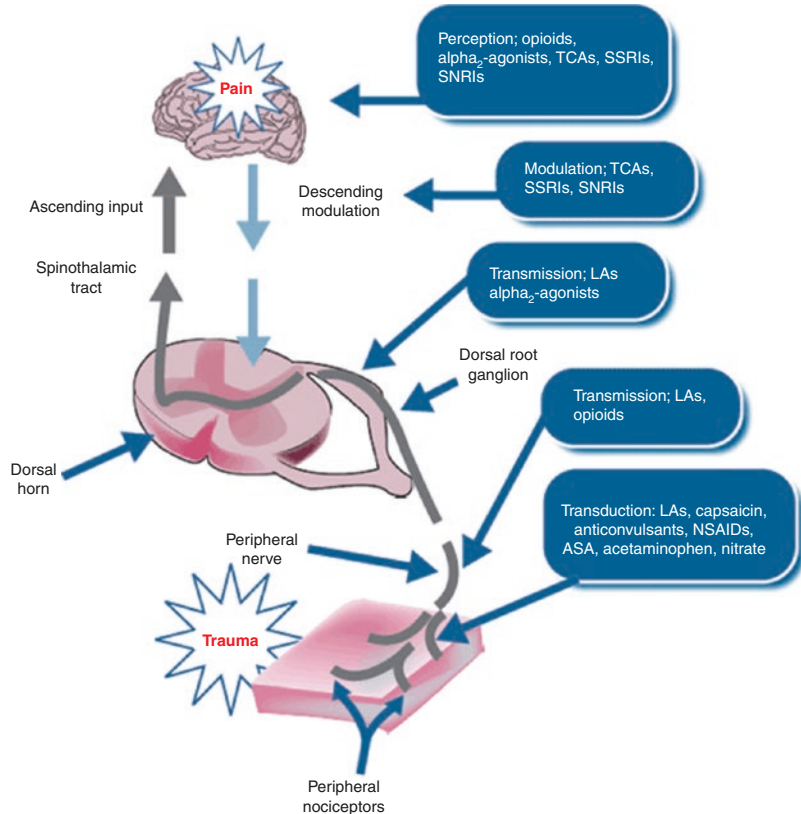
The process of central sensitization is thought to be important for the development of chronic pain [25]. Hence, surgical techniques and pharmacological interventions to minimize central sensitization are of great interest. Moreover, the concept of preemptive analgesia (analgesic intervention before nociception) focuses primarily on the early timing of analgesic therapy, and preventive analgesia (analgesia effect lasts longer than the pain killer half-life) focuses primarily on timing, duration, and efficacy of analgesic therapy [26]. The preventive model of analgesia is the basis of the multimodal technique and has proved some clinical benefit and can potentially prevent induction of central sensitization after injury by blocking the arrival of a nociceptive input to the spinal cord.

5.3 Therapeutic Strategies for Postoperative Pain Management

5.3.1 Acute Postsurgical Pain Management

Opioids still remain the corner stone of perioperative pain management (Fig. 5.1). Their use offers analgesia through central and peripheral mechanisms. However, they are associated with many side effects including an increased incidence of postoperative nausea and vomit-

Fig. 5.1 Multimodal analgesic approach to pain management. ASA aspirin, LAs local anesthetics, NSAIDs nonsteroidal anti-inflammatory drugs, SNRIs serotonin–norepinephrine reuptake inhibitors, SSRIs selective serotonin reuptake inhibitors, TCAs tricyclic antidepressants. Source: Kehlet H, Dahl JB. *Anesth Analg.* 1993;77:1048–1056



ing (PONV), sedation, drowsiness, and pruritus, which delay discharge and add cost to postoperative care [27, 28]. Of interest, animal studies showed an increase in tumor growth and tumor angiogenesis through the μ -opioid receptor [29, 30].

NSAIDs, including COX-2 inhibitors, provide opioid-sparing effect, reducing some opioid-related side effects [31, 32]. On the other hand, when bleeding is a concern for the surgical procedures, the use of nonselective NSAIDs should be avoided [33, 34]. A meta-analysis showed that the selective COX-2 inhibitors have a safety profile in this setting [35]. After the withdrawal of several COX-2 inhibitor products due to their long-term cardiovascular risks, their use for short durations in the acute postoperative period remains beneficial [36]. There is no increased cardiovascular risk in patients receiving short-term selective COX-2 inhibitors after noncardiac surgery [37].

Ketamine, an NMDA receptor antagonist, has been studied extensively, especially in preventing centralization of pain. Usually, small doses ($0.15 \text{ mg kg}^{-1} \text{ i.v.}$) improve recovery after outpatient arthroscopy [38]. Moreover, in a large analysis, ketamine was also opioid sparing with a low incidence of side effects [39]. Early administration of ketamine might prevent the progression to chronic pain.

Dextromethorphan is another *N*-methyl-D-aspartate-type glutamate receptor antagonist that prevents central windup and has other antinociceptive mechanisms of action [40]. Despite a fair number of studies on its use, the results remain conflicting [27].

Clonidine an α_2 agonist has been used for many years perioperatively for its analgesic, sedating, and hypotensive effects [41]. However, dexmedetomidine, another α_2 agonist, has been shown to reduce opioid-related side effects and enhance analgesia and is lacking side effects

when used as part of i.v. patient-controlled analgesia regimen for acute postoperative pain control [42]. When compared in association with morphine to morphine alone for postoperative analgesia and recovery, dexmedetomidine showed an additive effect [43]. Moreover, dexmedetomidine as part of a perioperative analgesic regimen decreases opioid requirements, postoperative nausea and vomiting, and postoperative stay [44].

Nerve block is an important component of a multimodal analgesia regimen. Local anesthetic administration into the wound as part of a multimodal regimen showed some benefits in the immediate postoperative period [45, 46]. Studies from single-shot peripheral nerve block also confirm this effect, with early postoperative pain relief, but a high percentage of patients require adjuvant pain therapy at 24 h and up to 7 days [47]. The prolonged duration of action of liposomal bupivacaine is interesting in the maxillofacial pain management regimen. This is due to the delivery system encapsulating the bupivacaine molecules and not altering their structure. As the body breaks down, the liposomal wall-free bupivacaine is released. The duration of action is up to 72 h [48–50]. Liposomal bupivacaine is packaged in a 20-cm³ vial (266 mg) and can be diluted with saline when large volume is needed.

Nitrous oxide has also been suggested to reduce the incidence of progression to chronic pain; however, further study is needed [51].

Ketorolac (Toradol) is an additional nonnarcotic analgesic with documented efficacy in reducing pain and narcotic use. Ketorolac is available for intravenous and local injection use only.

Non-pharmacological adjuvants can help in reducing postoperative pain. Transcutaneous electrical nerve stimulation (TENS) can be used at sub-noxious frequency over the wound area, and it decreases postoperative analgesic consumption [52]. Postoperative wound cooling significantly decreases postoperative analgesic consumption without an increase in wound infections [53].

5.4 Chronic Postsurgical Pain Management

5.4.1 Trigeminal Neuralgia (TN)

Neuropathic pain is characterized as a persistent and severe pain, associated with hyperalgesia, allodynia, sympathetic hyperactivity, and secondary myofascial pain. It is described as burning, sharp, and stabbing. The prevalence of neuropathic pain as a result of maxillofacial surgery has not been established. It is known that the prevalence of neuropathic pain varies according to the site and type of surgery, age of the patient, and coexisting medical conditions. A 0.51% incidence of neuropathic pain after bilateral sagittal split osteotomy was reported by Politis et al. [54], whereas permanent injury caused by injection of local analgesics is much less frequent at 0.0001–0.01% [55, 56]. Furthermore, maxillofacial surgeon take to provide sufficient information to the patient concerning the risks of chronic pain which is an unexpected complication of surgery. Accurate and comprehensible preoperative information may change the way the patient conceives pain and can reduce the risk for developing postoperative chronic pain.

The most commonly injured trigeminal nerve branches are the inferior alveolar nerve (IAN) and the lingual nerves (LNs), whereby the LN sits loosely in soft tissue compared with the IAN that resides in a bony canal. Peripheral sensory nerve injuries are more likely to be persistent when the injury is severe; if the patient is older, diabetic, and having peripheral nerve disease; if the wound was complicated with infection, hematoma, the presence of foreign materials; if the time elapsed between the cause of the injury and the review of the patient is of longer duration; and when the injury is more proximal to the cell body. Subsequent to iatrogenic trigeminal nerve injury, the patient often complains about a reduced quality of life, psychological discomfort, social disabilities, and being handicap [57]. Many patients can experience daily disability predominantly caused by elicited mechanical or cold allodynia resulting in pain on eating, drinking,

kissing sleeping, and other essential functions. The inferior alveolar nerve (IAN) supplies general sensation to the lower chin, lip, vermilion gums, and teeth unilaterally. The lingual nerve (LN) supplies general sensory to dorsal and ventral aspects of the tongue and lingual gingivae. Pain control is of paramount importance, and rehabilitation needs to be instituted as first-line treatment. Early intervention is important for optimal physiologic and functional recovery [58]. Reparative surgery may be indicated when the patient complains of persistent problems related to the nerve injury; however, there remains a significant deficiency in evidence base to support this practice. Surgery is more successful in inferior alveolar than in lingual nerve injuries, and the presence of a neuroma is a negative prognostic factor [59, 60].

TN is generally hard to treat, and the management is directed at decreasing the pain intensity and frequency. Pharmacological treatment includes carbamazepine which is highly effective in controlling TN and is usually the first line of treatment [61, 62]. It has been reported that 30% of patients may be initially resistant, and up to 50% will become refractory to carbamazepine therapy [63, 64]. Alternatively, oxcarbazepine, a carbamazepine derivative, is also effective in TN with fewer side effects [65]. Baclofen has been successfully used in TN and, because of its low side-effect profile, may be titrated to high doses (80 mg/day) [66]. Moreover, baclofen is suitable for combined therapy as it has a strong synergistic effect with carbamazepine.

Gabapentin has not been thoroughly studied in TN but may be considered in selected patients. Practically, therapy is initiated with carbamazepine, and patients are rapidly shifted to the controlled-release formulation that has fewer side effects. If carbamazepine causes troublesome side effects, the dose is reduced and baclofen is added, or oxcarbazepine is tried. Add-on therapy with lamotrigine or baclofen should be initiated in refractory cases before changing drugs. In those intractable cases, the most promising alternative is gabapentin and pregabalin which are efficient in peripheral neuropathies (postherpetic or diabetic neuropathy) and

may also be good treatment options in traumatic neuropathy. Furthermore, topiramate, or even the older anticonvulsants valproate and phenytoin, may be tried [67]. All patients on anticonvulsants need baseline and follow-up tests of hematologic, electrolyte, and liver function. Even though the treatment with anticonvulsants can control TN pain, exacerbations may occur and require temporary dose adjustment.

In addition, antidepressants and 5% lidocaine patches can be added to the first-line agents. Opioids (including tramadol) are kept as second-line agents. Tricyclic antidepressants (amitriptyline, nortriptyline, dothiepin) as low as 25 mg at bed time showed benefit with approximately every other patient experiencing painful polyneuropathies [68]. With this dose, most of the patients are able to tolerate the treatment with minimal side effects of dry mouth, drowsiness, and weight gain. Moreover, this should improve the sleep pattern supporting pain rehabilitation [69].

Serotonin and norepinephrine reuptake inhibitors (SNRIs) such as duloxetine and venlafaxine are used off label for neuropathic pain. Mexiletine, a nonselective sodium channel blocker used for cardiac arrhythmias, could be useful in theory if a lidocaine infusion showed a positive response. Occasionally with severe breakthrough pain, the patient might be hospitalized under the care of a pain clinic team for IV opioids or NMDA antagonist (ketamine) use and titration of optimal doses [70]. Stellate ganglion blocks with bupivacaine, steroids, or guanethidine can be considered to help break the pain cycle [71].

Peripheral nerve blocks provide temporary but complete pain relief in TN. Reports about trigeminal neurectomy involved small series with short-term follow-up and showed conflicting results with a success rate between 50 and 64% [72]. In any event, pain in TN invariably recurs after neurectomy within a mean period of 2 years [73]. Cryotherapy of peripheral branches usually provides pain relief for 6 months and could be repeated with good results [74]. Alcohol injections may be effective for about 1 year but are painful and are complicated with fibrosis which makes further injections technically difficult

[75]. Other complications include full-thickness skin or mucosal ulceration, cranial nerve palsies, herpes zoster reactivation, and bony necrosis [72]. It has been reported a 60% success rate at 24 months after peripheral glycerol injection; however, others published a pain relapse at 7 months [75]. Moreover, a single reinjection of glycerol has been reported with successful results [76]. Neurectomy, cryotherapy, and alcohol block have all resulted in neuropathic pain, termed *anesthesia dolorosa*. Peripheral procedures damage the nerve and therefore carry the risk of dysesthesias in the territory of the nerve. Those procedures should be kept as last resort or for patients with medical problems who cannot tolerate other treatment modalities [72].

Central percutaneous trigeminal nerve ablation procedures are directed at the trigeminal ganglion and include radiofrequency rhizotomy, glycerol injection, or balloon compression. The three treatment modalities provide approximately the same initial pain relief (around 90%) but are associated with different rates of pain recurrence and complications [77]. Overall, radiofrequency rhizotomy consistently provides the highest rates of sustained pain relief but is associated with high frequencies of facial and corneal numbness.

Psychological treatment is fundamental for many patients with secondary orofacial pain. The incidence of psychiatric disorders in patients with chronic orofacial pain is 72% [78]. The presence of negative thoughts and anxiety increased pain intensity fourfold in patients with acute postoperative orofacial pain [79]. The maintenance of physical and social activities is an important aspect of multimodal therapy.

There is evidence from animal studies on the use of potent antineuropathic peptides which was recently identified. It includes the opioid peptides endomorphin-1 and endomorphin-2 [80]. The fragment of substance P known as SP1–SP7 is pain relieving and comprises seven of the eleven amino acids of the algogenic parent substance P [81]. Furthermore, some fatty acids such as maresin and resolvins have antineuropathic activities [82]. The main advantage of these studied compounds whenever available for human use is

the decrease of side effects and drug interactions compared to the current drugs.

Neuromodulation of the trigeminal nerve is another area of contemporary research for intractable craniofacial pain [83]. Modalities include direct electrical stimulation (spinal cord stimulation) and transcranial magnetic stimulation and other devices to deliver substances and readjust the neurochemistry of the CNS.

Preliminary studies of autologous stem cell therapy showed that this modality is safe and effective for treating neuropathic pain. Results of the human study showed a reduction in pain intensity by 78% which lasted for 6 months from a single administration of stem cells at the trigeminal pain site [84].

5.4.2 Glossopharyngeal Neuralgia (GN)

Glossopharyngeal neuralgia is difficult to diagnose, and adequate treatment is often delayed several years because of its location, clinical features, and rarity (0.7 cases/100,000) [85]. GN is characterized by pain attacks similar to those in trigeminal neuralgia but is located depending on the affected territory of the two sensory branches. The pain in the pharyngeal GN is located mostly in the pharynx, tonsil, soft palate, or posterior tongue base and radiates upward to the inner ear or the angle of the mandible. On the other hand, the pain of the tympanic GN is confined to the ear but may subsequently radiate to the pharynx. Because of its innervation, the GN trigger areas are activated through chewing, swallowing, yawning, coughing, or phonation [86]. Moreover, sneezing, clearing the throat, blowing the nose, touching the gingiva or oral mucosa, or rubbing the ear might trigger the pain [85]. Topical analgesia to trigger areas will eliminate the pain and may help diagnose GN. This neuralgia is reported to induce syncope and bradycardia, probably mediated by functional central connections between visceral afferents of cranial nerves (IX and X) and autonomic medullary nuclei. Management and pharmacotherapy are similar to that for trigeminal neuralgia.

5.4.3 Persistent Idiopathic Facial Pain (PIFP)

Atypical facial pain was first described by Frazier and Russell in 1924 and was renamed persistent idiopathic facial pain.

“Atypical” pain is a diagnosis of exclusion and cannot be attributed to any pathologic process and is characterized by chronic, constant pain in the absence of any apparent cause in the face or brain [87]. Different categories of idiopathic facial pain conditions were labeled PIFP and might include neuropathic pain due to sensory nerve damage, complex regional pain syndrome (CRPS) from sympathetic nerve damage, and atypical facial pain. Phantom tooth pain or atypical odontalgia is a variation of atypical facial pain where intense discomfort is centered on a tooth or group of teeth with no obvious dental or oral disease. Occasionally, the pain may spread to adjacent teeth, especially after extraction of the painful tooth. The relationship with previous surgical intervention concludes that this condition may be a postsurgical neuropathy of the superior alveolar nerves. One of the main issues is to convince the patient, and inform their dentist, that the cause of their pain is not dental, so avoiding unnecessary irreversible invasive dental procedures. In 60 and 70% of these patients, the pain may be due to psychological or psychiatric disease [88]. It is more common in women aged between 30 and 50 years. Although any area of the face can be involved, the most commonly affected area is the maxillary region. Pain is often associated to a surgical or other invasive procedures [89]. PIFP has a variable presentation and is characterized by continuous, daily pain of variable intensity. Classically, the pain is deep and poorly localized; it is described as dull and aching and does not waken the patient from sleep. At the beginning the pain is usually confined to a limited area on one side of the face, but later it may spread to involve the other side in 40% [89]. A careful history and physical examination is essential, along with laboratory and imaging studies to rule out occult pathology. A psychiatric evaluation and a dental consultation might be indicated. The lack of a clear pathophysiologic

basis precludes the establishment of a treatment protocol. Medical treatment of PIFP is usually less satisfactory than other facial pain syndromes [90]. Medications used to treat PIFP include antidepressants, anticonvulsants, topical anesthetics, *N*-methyl-*D*-aspartate (NMDA) antagonists, substance P depletion agents, and opioid medications. The use of low-dose amitriptyline at bedtime and anticonvulsants appears to be the most effective.

5.4.4 Complex Regional Pain Syndrome (CRPS)

Utmost sympathetically mediated pain known as reflex sympathetic dystrophy or causalgia is termed currently complex regional pain syndrome (CRPS). According to the International Association for the Study of Pain, CRPS stands for a variety of painful conditions that usually follow an injury; it is described as spontaneous pain accompanied by allodynia and hyperalgesia that are not limited to dermatomal regions and exceeds in both magnitude and duration of the expected course of the inciting event. CRPS has a distal predominance and is associated with edema, abnormal skin blood flow, or sudomotor activity in the region of the pain at some time during the course of the illness. CRPS is divided into type 1 and the less frequent form type 2. CRPS I includes conditions caused by tissue injury or after minor local trauma, such as sprains or surgery, and was previously referred to as reflex sympathetic dystrophy. This condition results in minor or no identifiable nerve lesions with disproportionate pain. CRPS II is provoked by major nerve injury and was previously referred to as causalgia.

Both syndromes may have clinical evidence of a noradrenergic sympathetic influence on the development of pain. Nevertheless, this finding is not a prerequisite for diagnosing CRPS. During the acute stage, more than 80% have edema and cutaneous vasodilation, with the skin appearing red, increased sweating, and trophic phenomena [91]. In the chronic stages, this may subsequently reverse into vasoconstriction, resulting in cold,

bluish skin, with atrophic changes in the skin, nails, and muscles [92]. This results in weakness, contraction, fibrosis, and tremor of the affected site in advanced cases [91]. Over time, pain is usually present and may spread to involve the adjacent body quadrant or even half of the body, suggesting central involvement. The severity of CRPS pain and the loss of function predispose many patients to anxiety and depression. However, there is no evidence that secondary psychological factors developing early after an injury predispose to CRPS [93].

The historical dependence on sympathetic involvement for diagnosing CRPS has probably prevented the identification and documentation of head and neck cases. Thus, reports have relied on cervical sympathectomy, clonidine, guanethidine, and stellate ganglion blockade to confirm CRPS [94]. Some features, such as trophic changes and skin atrophy, are unreported in the trigeminal region, and motor disturbances are rare. There are no diagnostic tests for CRPS, which is at present a matter of clinical judgment, and not surprisingly, opinions differ in relation to individual patients [93].

Therapy should aim to reduce pain and restore function. Depending on the disease stage and symptomatology, steroids and sympathetic blocks may be indicated. Antidepressants and anticonvulsants may relieve neuropathic pain components, and opioids should be tried if these fail [91].

Conclusion

The pain management regime should start during patient assessment in the preoperative area, associated to a detailed communication with the surgeon about the type of surgery. The more the patient possesses risk factors, the more aggressive the anesthesiologist should be in their preventative pain management. Regional anesthesia, either local infiltration or peripheral nerve block, should always be considered when indicated. Breakthrough pain is well described in the chronic pain literature, and it similarly occurs in the acute postoperative situation. It is best managed with a rapid onset, short-lasting

agent (e.g., fentanyl in the recovery room) or an agent of a different class than those previously administered.

For expectant mild pain from minor surgery, it is recommended to use acetaminophen, Tramal, NSAIDs, or both local anesthetic wound infiltration and intraoperative opioid therapy.

Non-pharmacological therapy (e.g., TENS, cooling packs) should also be used when appropriate after operation.

For expected moderate pain, we suggest two to three agents to be used intraoperatively, including regional anesthesia. A combination of opioids and NSAIDs should also be considered for postoperative pain management.

For expected severe pain, we recommend that regional anesthesia be strongly considered unless contraindicated, with an IV patient control analgesia infusion. Intraoperative management should also consist of aggressive multimodal agent regimen, with prompt attention and treatment of postoperative pain.

In patients with a history of chronic opioid use or where the risk of chronic pain is high, both ketamine and regional anesthesia should be considered both intraoperatively and postoperatively.

Chronic neuropathic pain is a complex disease which varies among patients, even after identical injuries. This variability is probably caused by an interplay of multiple potential factors including surgery, environmental, infection, trauma, psychosocial, and genetic factors. With preemptive analgesia, preoperative treatment is designed to reduce or eliminate the initial sensory barrage and prevent central sensitization.

A patient with orofacial pain must be investigated comprehensively to identify if the type of his pain is neuropathic or nociceptive. Moreover, neuronal connections to the sympathetic ganglia may result in sympathetically maintained pain. Treatment of neuropathic pain must be multidisciplinary to provide appropriate medication with different modes and sites of action which will lead to improved efficacy with reduced side effects.

The first-line systemic antineuropathics include drugs such as amitriptyline, gabapentin, pregabalin, and duloxetine. New developing therapies such as neuromodulation are being investigated. Identification and treatment of the common comorbidities like anxiety and depression is imperative in patients with maxillofacial pain to regain physical and social activity.

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Medical Complications Following Maxillofacial Surgery

6

Henry Ward and Andre Ward

Abstract

Cosmetic maxillofacial surgical procedures are elective ones. Optimizing our patient's medical management will ultimately improve our surgical outcomes. This is usually accomplished by a detailed preoperative evaluation which includes a comprehensive history and physical examination. This chapter will focus on important aspects of the perioperative management of the cosmetic surgery patient. Potential complications will be reviewed. Management of these complications will also be discussed.

6.1 Introduction

A safer surgery and a short hospital stay are obvious goals for the surgeon and the patient. It is best to anticipate and hence avoid and minimize intra- and postoperative complications. This can be accomplished by a thorough history and

physical examination and a meticulous review of the list of medications.

A family history of anesthesia-related complications and thromboembolism should be obtained since this may provide an important clue for potential postoperative complications. The presence of medical comorbidities such as diabetes mellitus, congestive heart failure, arrhythmias, coronary artery disease, or chronic obstructive pulmonary disease and kidney disease should prompt the surgeon to obtain appropriate consultations regarding preoperative and postoperative management.

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6.2 Postoperative Pain

Postoperative pain is common in the initial hours and first 2 days after maxillofacial surgery and is the manifestation of inflammation due to tissue injury. There is usually a correlation between the

extent and duration of surgery and the pain severity. Pain is measured using the standard pain intensity scales.

Pain management in this setting is simple but at times challenging. The ideal postoperative analgesic treatment should have a short onset of action and be effective with a low incidence of side effects or drug interaction [1].

There is no benefit from the preoperative use of ibuprofen and similar drugs in decreasing the intensity of the pain in the postoperative period [2].

Analgesics include mild agents such as paracetamol and nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen and celecoxib, moderate analgesics such as codeine, and stronger opioids such as oxycodone and fentanyl.

6.2.1 Paracetamol/Acetaminophen

It is of course difficult to use the oral route immediately after surgery. The intravenous form (1 g IV) is effective and safe for treatment of postoperative pain [3]. Paracetamol can be used alone or in conjunction with other pain relief drugs such as nonsteroidal anti-inflammatory drugs or morphine. It is effective regardless of age, sex, and weight [1]. It is safe to use in patients with a history of peptic ulcer disease and bronchial asthma and does not interfere with platelet function. The analgesic effect starts within 5 min, peaks at 1 h, and lasts 4–6 h [4]. The dose of IV paracetamol should be reduced in patients with hepatic insufficiency, chronic alcoholism, and chronic malnutrition [1].

6.2.2 Nonsteroidal Anti-inflammatory Drugs

The most commonly used are ibuprofen, naproxen, indomethacin, diclofenac, sulindac, and ketorolac.

Nonsteroidal anti-inflammatory drugs are often used in the management of moderate to severe pain alone or in combination with opioids [5]. Different NSAIDs have varying inhibitory potentials of the two isoforms of cyclooxygenase,

COX-1 and COX-2. Their prolonged use may lead to renal functional impairment and gastrointestinal bleeding. NSAIDs can interact with numerous drugs that include anticoagulants and antiplatelet agents enhancing the potential for gastrointestinal bleeding and other bleeding sites, antihypertensive agents lowering their antihypertensive effect, and diuretics minimizing their natriuretic effect. They may interact with the antiplatelet effect of aspirin so the use of NSAIDs should be avoided, if possible, in patients taking low-dose aspirin for cardiovascular protection. The gastrointestinal side effects such as dyspepsia and gastric ulcerations can be minimized with the use of a proton pump inhibitor. NSAIDs may lead to renal insufficiency related to renal vasoconstriction and/or acute interstitial nephritis. Their use should be monitored carefully in patients who have underlying chronic kidney disease. Naproxen is probably the preferred nonselective NSAID in patients with cardiovascular disease.

6.2.3 Opioids

Opioids are quite effective for severe pain experienced by patients undergoing maxillofacial surgery.

The most commonly used first-line opioids are codeine, morphine sulfate, hydromorphone, fentanyl, and oxycodone. Fentanyl is 80–100 times more potent than morphine sulfate and can be used sublingually. The potency of 10 mg of morphine sulfate equals the potency of 2 mg of Dilaudid. They are often combined with non-opioid analgesics to maximize analgesia and decrease opioid requirements. It is important to remember few concepts when these drugs are being used:

1. Most opioids are renally excreted and the dose needs to be adjusted in chronic kidney disease.
2. Predictors of opioid-related adverse events include old age, obesity, renal failure, hepatic failure, chronic obstructive pulmonary disease, and combined use of sedative agents such as benzodiazepine.

3. A history of chronic opioid use will require the use of higher doses for pain control.
4. In the acute postoperative period, there is no need to be concerned about addiction and tolerance.
5. Naloxone is the reversal agent at a dose of 5–10 µg/kg given as a single dose intravenously.

These drugs may cause adverse events such as drowsiness and respiratory depression, nausea, vomiting, and constipation. Occasionally, hyperalgesia and agitation/delirium can be seen. It is important to emphasize that the use of benzodiazepines increases the risk of respiratory depression.

6.3 Fever After Maxillofacial Surgery

Postoperative fever may be due to both infectious and noninfectious causes. Most of the time, it is a benign event related to an inflammatory response [6]. A complete history and a thorough physical examination are usually sufficient to determine the etiology of postoperative fever.

Infectious complications following maxillofacial surgery have lessened with the use of prophylactic antibiotics but remain significant causes of postoperative morbidity. A superficial wound infection presents as localized pain associated with redness and minimal discharge usually caused by staphylococci. Cellulitis may present within the first week and may spread locally if it's not treated appropriately with antibiotics. The presence of an abscess requires suture removal and antibiotic therapy. A deeper abscess may require surgical re-exploration.

The use of compressed air and an air rotor handpiece in third molar extractions and dental implant can lead to mediastinitis and subcutaneous emphysema associated with fever [7].

In the presence of fever, unexplained tachycardia and tachypnea, and a drop in the blood pressure, sepsis should be suspected. A CBC, blood cultures, and consultation with the infectious disease department should be done immediately. Appropriate antibiotic therapy is to be implemented immediately and surgical drainage of an abscess performed.

Systemic inflammatory response syndrome (SIRS) may at times be seen and may lead to end-organ dysfunction/failure with a high morbidity and mortality. It is defined by the presence of two or more of the following: temperature greater than 38 °C or less than 36 °C, heart rate greater than 90 bpm, respiratory rate greater than 20, and white cell count greater than 12,000 cells/mm³, less than 4000 cells/mm³, and more than 10% immature forms.

6.4 Nausea and Vomiting

Postoperative nausea and vomiting (PONV) is defined as any nausea, retching, or vomiting occurring during the first 24–48 h after surgery [8]. Nausea and vomiting are relatively common and can delay patient discharge and increase healthcare costs. Postoperative nausea and vomiting can be triggered by several stimuli, including opioids, volatile anesthetics, anxiety, and adverse drug reactions. Details of the mechanism of these symptoms are beyond the scope of this review. Multiple neurotransmitter pathways are implicated in the physiology of nausea and vomiting, and multiple antiemetics can be combined to reduce the risk of these symptoms.

A number of risk factors have been associated with an increased incidence of PONV [8].

Well-Established Risk Factors

1. *Female gender*: A female patient is probably three times more likely to suffer from PONV than a male. It is the strongest patient-specific predictor [9].
2. *Nonsmoking status*: A nonsmoking status doubles the patient's risk of PONV.
3. *History of motion sickness*.
4. *Younger age group (less than 50 years of age)*.
5. *The use of volatile anesthetics*: These agents decrease the serum level of anandamide, an endogenous cannabinoid neurotransmitter that suppresses nausea and vomiting.
6. *Intraoperative and postoperative opioid use*.
7. *Long duration of anesthesia (greater than 60 min)*.

Potential Risk Factors

1. *History of migraine headache*
2. *History of anxiety*
3. *Facemask ventilation*
4. *Type of surgery performed*

The patient's baseline risk for PONV should be assessed using a composite score based on four of these predictors. The Apfel risk score considers female sex, history of PONV and/or motion sickness, nonsmoking status, and the use of postoperative opioids [9]. The incidence of PONV with the presence of zero, one, two, three, and four risk factors is approximately 10%, 20%, 40%, 60%, and 80%, respectively [10]. Knowing these risk factors, strategies can be implemented to decrease the incidence of PONV such as avoidance of general anesthesia by the use of regional anesthesia, the use of propofol for induction and maintenance of anesthesia, and avoidance of nitrous oxide and volatile anesthetics, minimizing intraoperative and postoperative opioid use and adequate hydration [9].

The first step in the treatment of postoperative nausea and vomiting is to give antiemetics prophylactically. One can choose from the following agents: 5-hydroxytryptamine, receptor antagonists such as ondansetron, corticosteroids such as dexamethasone, butyrophenones such as haloperidol, antihistamines such as meclizine, and anticholinergics such as transdermal scopolamine. Patients at high risk should receive prophylaxis with combination therapy. Antiemetics with different modes of action have additive effects on the incidence of PONV. For example, 50 mg of oral meclizine plus 4 mg of intravenous ondansetron is more effective than either alone [11].

If nausea and vomiting occur postoperatively, the administration of the medications used for prophylaxis does not confer any benefit [12]. Antiemetic medications from a different pharmacologic class should be used such as 2–4 mg intravenous dexamethasone or 0.625 mg intravenous droperidol. Isopropyl alcohol inhalation may help to reduce the severity of the nausea [13].

One note of caution is to watch for the QT interval on the resting electrocardiogram prior to the administration of ondansetron since this drug may prolong the QT interval and potentially lead to ventricular tachycardia [14].

6.5 Cardiovascular Complications

Cardiovascular complications are the most significant risks for patients undergoing maxillofacial surgery. They include hemodynamic instability, atrial and ventricular dysrhythmias, myocardial ischemia with angina pectoris, and acute myocardial infarction and congestive heart failure. The likelihood of cardiovascular problems is related to the severity of the preexisting cardiovascular comorbidities such as coronary artery disease and congestive heart failure, the severity of the blood loss, and the effects of anesthetic agents [15]. Estimation of the baseline risk for myocardial infarction, congestive heart failure, atrial fibrillation, or other arrhythmias is crucial, and a consultation with a cardiologist is very helpful.

6.5.1 Hypotension

There is no widely accepted definition of perioperative hypotension though some values may help as a guide for treatment: systolic blood pressure below 90 mmHg or mean blood pressure below 65 mmHg or a 20% decrease in baseline systolic blood pressure [16].

When faced with a patient with hypotension, the first step is to ensure the accuracy of the blood pressure measurement. I would encourage the treating physician to recheck the blood pressure using the appropriate blood pressure cuff in both arms.

The second step is to assess the clinical picture associated with the low blood pressure. Is the patient awake and comfortable or drowsy and distressed? Is the skin warm and dry or cold and wet? Assessment of the capillary refill time is

quite helpful, the normal being less than 2 s. If hypotension is confirmed, infuse 250–500 mL of isotonic crystalloid solution, and if necessary administer intravenous phenylephrine in 40–100 µg increments or ephedrine 5–10 mg increments. For severe hypotension, give small boluses of 10–15 µg of diluted intravenous epinephrine, 5–10 µg of norepinephrine, or 1–4 units of vasopressin.

Assessment of the underlying etiology is then required. It is crucial to begin by reviewing the preoperative and intraoperative medications used. Antihypertensive agents given prior to the operation may lead to postoperative hypotension and of course must be discontinued. Residual effects of the anesthetic agents or excessive opioid use may lead to hypotension. In this setting, give 0.2 mg increments of intravenous flumazenil up to 3 mg within 1 h to treat benzodiazepine excess. Give 400 µg of naloxone every 5 min for excessive opioid dosage.

Anaphylactic shock is to be suspected if the hypotension is associated with rash, bronchospasm, and facial edema. In this setting, administer 10–50 µg of diluted intravenous epinephrine, intravenous glucocorticoids, and intravenous crystalloids.

An electrocardiogram should be obtained to rule out acute myocardial ischemia, and appropriate therapy should be implemented with the help of a consulting cardiologist.

Acute pulmonary embolism is to be suspected if the patient has been bedridden, and the systolic blood pressure is associated with dyspnea, hypoxia, and sinus tachycardia. A CT scan of the chest should then be obtained and anticoagulation initiated with the help of a pulmonologist or a cardiologist.

Sepsis may be the cause of hypotension, and the septic state may predate the surgical intervention or may develop intraoperatively. A CBC and blood cultures should be obtained if the patient is demonstrating an elevated temperature and is tachycardic. Appropriate intravenous antibiotics and appropriate fluid administration are warranted in addition to consultation with the infectious disease department.

Rarely, acute adrenal insufficiency may be causing the hypotension, and this should be suspected in a patient on chronic glucocorticoid therapy not given perioperative replacement therapy. A 100–200 mg of intravenous hydrocortisone should be given along with a normal saline infusion.

Patients receiving an ACE inhibitor or an angiotensin receptor blocker preoperatively have a higher and more profound risk of hypotension with a decreased responsiveness to alpha adrenergic agonists [17]. When faced with volume and catecholamine-resistant hypotension in a patient previously treated with angiotensin II blockade, administration of intravenous vasopressin as a bolus or infusion can be helpful to restore quickly normal hemodynamics [18].

6.5.2 Hypertension

It is well established that the most important risk factor for postoperative systemic hypertension is preexisting systemic hypertension. A sustained stage 1 or 2 systemic hypertension prior to the surgical intervention can potentially lead to diastolic left ventricular dysfunction and obstructive, at times asymptomatic, coronary artery disease.

It is important to keep in mind that induction of anesthesia is associated with sympathetic activation and can cause the blood pressure to rise by 20–30 mmHg in normotensive individual [19]. The systolic blood pressure may increase up to 90 mmHg in individuals with untreated systemic hypertension.

Mild arterial hypertension is common in the postoperative period and is usually attributed to stress, pain, and anxiety. It is usually transient and self-limited [20].

A more severe elevation of the arterial pressure should be concerning because of the potential for left ventricular systolic or diastolic dysfunction, atrial and ventricular dysrhythmias, and myocardial ischemia.

Factors contributing to postoperative hypertension include preexisting, undiagnosed, or poorly controlled hypertension, pain-causing

sympathetic stimulation, hypercarbia and hypoxia-causing sympathetic stimulation, intravascular volume expansion with excess fluid administration, and withdrawal of centrally acting sympatholytic drugs such as clonidine and methyldopa, causing rebound systemic hypertension above the pretreatment level, related to rapid return of catecholamine secretion that had been suppressed [21]. Not to be overlooked is bladder distention which causes sympathetic stimulation. Rarely postoperative systemic hypertension can be the first manifestation of pheochromocytoma. In the proper context, alcohol and opioid withdrawal are to be ruled out (Table 6.1).

The management of postoperative hypertension includes correction of the exacerbating factors such as pain, anxiety agitation, bladder distention, and resumption of preoperative antihypertensive therapy. For patients unable to take oral medications, intravenous equivalent medication should be given. If the systolic blood pressure is maintained over 180 mmHg or the diastolic blood pressure is greater than 110 mmHg, 10–20 mg of intravenous labetalol can be given as a bolus every 10 min or metoprolol 5 mg intravenously every 5 min three times or intravenous enalapril 2.5–5 mg every 6 h. Hydralazine 10–20 mg can be given intravenously every 6 h; however, it has an unpredictable hypotensive effect and in general it should be avoided [22]. Oral or transdermal clonidine should be given in case of clonidine withdrawal syndrome associated with hypertension. Transcutaneous nitrates can also be quite effective.

Table 6.1 Factors contributing to postoperative hypertension

| |
|---|
| 1. Preexisting systemic hypertension |
| 2. Pain and anxiety |
| 3. Hypercarbia and hypoxia |
| 4. Hypervolemia |
| 5. Hypothermia |
| 6. Withdrawal of centrally acting sympatholytic drugs |
| 7. Bladder distention |
| Pheochromocytoma |
| Opioid and alcohol withdrawal |

6.6 Postoperative Cardiac Arrhythmias

Postoperative atrial and ventricular arrhythmias are most likely to occur in patients with structural heart disease or ischemic heart disease. The incidence depends on the age of the patient, underlying heart disease, transient hypoxemia or cardiac ischemia, catecholamine excess, electrolyte imbalance, and autonomic activation related to pain or hypotension. The morbidity of the arrhythmic event is related to the type of arrhythmia and its duration, underlying left ventricular structure and function and its hemodynamic impact. The arrhythmias are classified as bradyarrhythmias or tachyarrhythmias.

The bradyarrhythmias are usually secondary to heightened vagal tone caused by pain or laryngoscopy. They consist of sinus bradycardia defined as a heart rate below 60 bpm, sinus pause, sinoatrial block, sinus arrest, and varying degrees of AV blocks. First-degree AV block is defined as a delay in atrioventricular conduction with a prolonged PR interval on the resting surface electrocardiogram (greater than 200 ms). Mobitz 1 AV block is defined as progressive prolongation of the PR interval followed by a nonconducted P wave. Mobitz 2 AV block is defined by the absence of conduction of every other P wave. Complete heart block is defined by total AV dissociation. If the bradycardic event is transient without any hemodynamic compromise, then there is no need for treatment and simple observation is sufficient. If the bradycardic event is sustained and symptomatic, use 0.4–0.5 mg intravenous atropine up to a maximal dose of 2 mg, a beta-agonist, or an external or transvenous pacemaker. For episodes of Mobitz 2 AV block and complete heart block, the patient should be transferred to the intensive care unit with consideration of either a transcutaneous pacemaker or a transvenous pacemaker and potentially permanent pacing.

The tachyarrhythmias are classified as narrow QRS tachycardias or wide QRS tachycardias. The narrow QRS tachycardias are supraventricular tachycardias and include sinus tachycardia, atrial tachycardia, multifocal atrial tachycardia,

AV nodal reentry tachycardia, atrial flutter, atrial fibrillation, and AV junctional tachycardia. Most patients with a supraventricular tachycardia remain hemodynamically stable, and ventricular rate control is the goal of therapy using AV nodal blocking agents such as intravenous beta-blockers, intravenous diltiazem or verapamil, or intravenous adenosine.

Sinus tachycardia is a normal physiologic response to excessive release of catecholamines. Several factors can be responsible for the increase in heart rate such as fever, volume depletion, hypotension, sepsis, anemia, hypoxia, pain and anxiety, left ventricular systolic dysfunction, and chronic obstructive pulmonary disease.

Multifocal atrial tachycardia is an irregular rhythm with three or more different P-wave morphology. It is usually seen in this setting of chronic obstructive pulmonary disease, electrolyte imbalance, and myocardial ischemia. The treatment is usually beta-blocking agents and calcium channel-blocking agents.

AV nodal reentry tachycardia is usually aborted with intravenous adenosine given as a 6 mg bolus. A repeated dose with 12 mg intravenously can be given. If hemodynamic compromise with hypotension is noted, then prompt synchronized cardioversion is advised to prevent the life-threatening complications of hypoperfusion.

Paroxysmal atrial fibrillation is the most common perioperative arrhythmia. Potential reversible causes include hypokalemia and hypomagnesemia, hypoxia, infection, hypotension, alcohol withdrawal, and myocardial ischemia. Control of ventricular response with an AV nodal-blocking agent is the first step in the management of this dysrhythmia. If hemodynamic compromise is noted with chest pain or hypotension, then a synchronized direct current cardioversion is warranted. If atrial fibrillation persists for more than 1 day, then consideration is to be given for the initiation of an antithrombotic therapy to minimize the risk of an embolic stroke. The timing of anticoagulation should be a shared decision between the surgeon and the cardiologist. A transthoracic echocardiographic study should be obtained for further evaluation of left ventricular function and structure [23].

The wide QRS tachycardias are either supraventricular tachycardias aberrantly conducted or supraventricular tachycardias in the setting of preexisting left bundle branch block. They can also be supraventricular tachycardias with antegrade conduction via an accessory pathway, or ventricular tachycardias.

Ventricular tachycardias are classified into sustained and nonsustained. They can be either monomorphic or polymorphic.

Nonsustained ventricular tachycardias are usually slow below 100 bpm and do not lead to hemodynamic instability. They do not typically require any therapy. Close observation is of course crucial. Correction of electrolyte imbalance, control of a high blood pressure, and treatment of potential myocardial ischemia are to be pursued.

The monomorphic variety of sustained ventricular tachycardia is a reentry tachycardia and is treated best with intravenous lidocaine or intravenous amiodarone. Polymorphic ventricular tachycardia associated with a normal QT interval usually occurs in the setting of myocardial ischemia or structural heart disease. It may degenerate into ventricular fibrillation. Treatment with intravenous amiodarone and initiation of anti-ischemic therapy are warranted. Myocardial infarction is to be ruled out in this setting, and beta-blockade should be initiated. Hypokalemia and hypomagnesemia should be corrected. An immediate consultation with a cardiologist is warranted.

The polymorphic variety can be seen in the setting of prolonged QT interval and is labeled torsade de pointes. It requires immediate asynchronous DC countershock and therapy with intravenous magnesium and intravenous lidocaine. A temporary pacemaker insertion may be needed [24].

6.7 Transvenous Pacemakers and Implantable Cardioverter Defibrillators

The major concern in this setting is the risk of electromagnetic interference (EMI) from the electrocautery. EMI can inhibit pacemaker output with potential for increasing the pacing rate

and leading to the implantable cardioverter defibrillator (ICD) firing [24]. It is recommended the ICD be reprogrammed to stop arrhythmia detection and potential shock delivery in cases where electrocautery is used. Interrogation of the ICD after a surgical procedure is recommended to ensure the device integrity.

6.8 Angina Pectoris

Myocardial ischemia leading to chest pain is referred to as angina pectoris. Myocardial ischemia is the result of a mismatch between myocardial oxygen demand and myocardial oxygen supply.

The determinants of myocardial oxygen demand are heart rate, systolic blood pressure, myocardial wall stress, and myocardial contractility. During surgery at least two of these determinants are increased: the heart rate and the systolic blood pressure.

The determinants of myocardial oxygen supply are the coronary artery diameter, the coronary perfusion pressure determined by both the diastolic arterial pressure and left ventricular end-diastolic pressure, the heart rate affecting the duration of diastolic coronary filling time, and the oxygen-carrying capacity of the blood. Surgery may affect myocardial oxygen supply if it is associated with reduced blood pressure, increased heart rate, anemia, and hypoxia.

The increased demand and reduced supply of myocardial oxygen noted during surgery may result in angina pectoris characterized by anterior chest pressure, squeezing, choking, or burning. The pain is not positional in nature, diffuse rather than localized, and associated at times with dyspnea, diaphoresis, or radiation to the left arm and jaw. This should warrant obtaining an immediate electrocardiogram and consultation with a cardiologist. The patient should be given sublingual nitroglycerin and four tablets of chewable aspirin if possible (the alternative would be suppository of aspirin). Initiation of beta-blockade and nitrate therapy should be guided by the consulting cardiologist. The patient should be transferred to the intensive care unit for further observation and therapy.

Coronary arterial spasm is another possibility leading to angina pectoris in the immediate postoperative period, especially if the patient is under a great degree of stress and is hyperventilating. A history of previous cocaine use is a clue for entertaining this diagnosis [25]. In this setting, sublingual nitroglycerin, transcutaneous nitrate, and intravenous diltiazem are the treatment of choice.

6.9 Myocardial Infarction

Myocardial infarction is a rare occurrence in the relatively young patient undergoing maxillofacial surgery. It carries a high risk of morbidity and mortality [26].

The diagnosis should be suspected when the patient starts complaining of a prolonged anterior chest pain associated with shortness of breath and diaphoresis and ventricular ectopy. The symptoms can be muted or atypical due to the administration of anesthetic/analgesic agents during surgery [27].

An immediate electrocardiogram will be diagnostic showing ST segment elevation or new T-wave inversion or a new left bundle branch block. The patient should be given morphine sulfate to alleviate the chest pain and sublingual nitroglycerin and four tablets of chewable aspirin. The patient should be transferred immediately to the coronary care unit, and a stat consultation with a cardiologist should be obtained. Cardiac catheterization should be carried out immediately for stenting of the infarct-related artery.

Preoperative cardiac risk assessment in patients with a previous history of angina pectoris, ventricular ectopy, heart failure, and diabetes mellitus and patients over 60 years of age can certainly help prevent this potentially fatal complication.

6.10 Congestive Heart Failure

In the absence of valvular heart disease, diabetes mellitus, and chronic kidney disease, the occurrence of heart failure in patients undergoing

maxillofacial surgery is rather unusual. Preoperative evaluation of these patients should allow the physician to prevent this complication by optimizing their medical therapy and avoiding fluid overload intra- and postoperatively.

Symptoms and signs of heart failure include exertional dyspnea, orthopnea associated at times with hypoxia, sinus tachycardia, elevated jugular venous pressure, and an apical third heart sound. In the acute setting, ankle edema is absent. The chest x-ray is helpful here demonstrating evidence of vascular congestion. Brain natriuretic peptides are elevated. An electrocardiogram looking for evidence of silent myocardial ischemia should be obtained. Serial cardiac enzyme measurements are included in the assessment of these patients. A transthoracic echocardiogram is to be requested. A consultation with a cardiologist is of course needed.

The differential diagnosis of postoperative dyspnea includes myocardial ischemia, aspiration pneumonitis, pneumonia, bronchial asthma anxiety, pulmonary embolus and early sepsis. A thorough physical examination along with chest x-ray and electrocardiogram should allow the clinician to differentiate among these entities.

Pulmonary edema resulting from postanesthesia laryngospasm has been described. This syndrome is sometimes called negative pressure pulmonary edema [28]. The prognosis of these patients is excellent. Rarely pheochromocytoma can present as postoperative acute pulmonary edema related to a catecholamine storm [29].

The most common precipitating factor for postoperative heart failure is fluid overload especially if it is combined with malnutrition and a low oncotic pressure reflected by a low serum albumin level. Myocardial ischemia is always to be ruled out by serial electrocardiograms and serial cardiac biomarkers.

The standard treatment for postoperative heart failure includes intravenous diuretics and vasodilator therapy using angiotensin II blockade and nitrates. Once the patient is stable, beta-blockade and aldosterone-blocking agents are added if reduced ejection fraction is documented by a transthoracic echocardiographic study. Further diagnostic workup and management will be dictated by the consulting cardiologist.

6.11 Venous Thromboembolism

Patients undergoing maxillofacial surgery represent a low-risk group for the development of postoperative venous thromboembolism. However, old age, anticipated prolonged immobilization, a prior history of venous thromboembolism, and the presence of malignancy should lead to initiation of thrombosis prophylaxis.

A large number of surgical patients are not given thromboprophylaxis [30]. This predisposes them to pulmonary embolism, the most preventable cause of hospital death [31].

Early and frequent ambulation is essential for preventing deep venous thrombosis. Mechanical prophylaxis using intermittent pneumatic compression is sometimes sufficient. Pharmacologic prophylaxis is preferred in moderate- or higher-risk patients.

Low molecular weight heparin is preferred over unfractionated heparin and is the agent of choice for preventing venous thromboembolism in high-risk patients [32]. Warfarin or direct thrombin inhibitors such as dabigatran and anti-factor Xa inhibitors such as rivaroxaban, apixaban, or edoxaban are excellent agents and can be substituted for warfarin or heparin. Extended venous thromboembolism prophylaxis is usually not needed.

6.12 Perioperative Management of Patients with Diabetes Mellitus

Diabetes mellitus and new onset hyperglycemia are important risk factors for perioperative cardiac complications. Surgery leads to a hyperglycemic response mediated by epinephrine, catecholamines, and inflammatory cytokines and represents a hemodynamic stress that worsens the prothrombotic state of these patients.

A few simple goals of therapy are to be followed for the diabetic patient undergoing maxillofacial surgery (modified from UpToDate):

1. Patients should have the surgery early in the morning while being nothing by mouth.

2. Patients should hold their oral hypoglycemic and non-insulin injectable drugs the morning of surgery. If hyperglycemia is noted in these patients, then short- or rapid-acting insulin may be given subcutaneously, typically every 6 h.
3. Patients managed by diet alone may require supplemental short (regular)- or rapid-acting insulin (lispro, aspart, or glulisine).
4. Patients taking insulin should reduce the dose by about 50%.
5. Once the patient is eating well, then the preoperative treatment regimen is to be resumed.

6.13 Neurological Complications

Neurological complications are rather rare in the patients undergoing maxillofacial surgery considering the young age group and the generally good health status of these patients. They include delayed emergence, delirium, cerebrovascular ischemic events such as a transient ischemic attack or a completed stroke, and seizure activities.

Delayed emergence is the failure to return to normal consciousness in a timely fashion following administration of general anesthesia. Urgent evaluation is required if the patient is unresponsive or awakening more slowly than expected (greater than 30–60 min). The first step in the evaluation of these patients is to ensure adequate reversal of neuromuscular blockade and opioid effect [33]. A basic neurologic examination should be performed and a stat blood sugar and arterial blood gases be obtained to rule out hypoglycemia, hypoxemia, and hypercarbia. It would be quite rare to implicate severe hyponatremia in the setting of maxillofacial surgery causing the delayed emergence. An acute neurologic disorder such as acute stroke and hypoxic–ischemic encephalopathy is to be entertained requiring a stat CAT scan or MRI of the head and an immediate neurological consultation.

Postoperative delirium is another neurological complication that may be encountered

mostly in the elderly with preexisting cognitive dysfunction [34]. It may develop within hours to days and is characterized by disorganized thinking, alteration in consciousness, hallucination, lethargy, or agitation. Several factors may precipitate this syndrome such as sepsis, hypoxemia, hypercarbia, allergic drug reactions, H₂ receptor blockers, and hypnotics.

On rare occasions, seizures may be encountered. Hypoxia, hypoglycemia, severe hyponatremia, and alcohol and drug withdrawal should be ruled out.

A preoperative carotid ultrasound imaging should be considered in patients with an audible carotid bruit or a previous transient ischemic attack. In patients with known significant carotid arterial stenosis, special care should be paid to avoid periods of sustained hypotension maintaining a mean arterial pressure between 60 and 70 mmHg. If aspirin or clopidogrel was stopped prior to surgery, it should be restarted as soon as possible—perhaps 12–24 h after the surgical intervention. In patients with atrial fibrillation, oral anticoagulants should also be started as soon as possible.

6.14 Pulmonary Complications

Clinically significant pulmonary complications following maxillofacial surgery include atelectasis, pulmonary aspiration leading to pneumonitis/pneumonia, exacerbation of underlying chronic obstructive pulmonary disease, bronchospasm, upper airway obstruction, and respiratory failure related to cardiogenic and noncardiogenic pulmonary edema.

Continuous monitoring of the respiratory rate and peripheral oxygen saturation along with intermittent check of airway patency are essential for early detection of these complications. The nursing staff should be on the lookout for tachypnea (respiratory rate greater than 30 breaths per minute), bradypnea (respiratory rate less than 8 breaths per minute), hypoxemia (peripheral arterial oxygen saturation less than 93%), cyanosis, use of accessory respiratory muscles, snoring, inspiratory stridor indicative

of airway obstruction, and localized wheezing related to lower airway obstruction with either a mucous plug or foreign body. Diffuse wheezing may be related to bronchospasm in the setting of chronic obstructive pulmonary disease, bronchial asthma, aspiration, or allergic reaction. Obtundation may be related to hypoventilation with hypercarbia, residual opioid effects, or neurological complications. Coarse rhonchi are usually associated with excessive airway secretions or acute pulmonary edema. Keep in mind that symptoms of respiratory insufficiency such as dyspnea may be mild or absent in a sedated patient.

Preoperative strategies to reduce these morbidities include counseling on cigarette smoking cessation, optimizing the bronchodilator and glucocorticoid regimen in patients with chronic obstructive pulmonary disease, including a stress dose of glucocorticoids prior to the induction of anesthesia in patients treated for more than 3 weeks prior to the previous 6 month of surgery. Surgery should be canceled during an exacerbation of chronic obstructive pulmonary disease and the presence of wheezing [35]. Emergency airway equipment and resuscitation drugs must be immediately available.

At the time of initial sedation, hypoxemia may be observed, and administration of supplemental oxygen is necessary. Short-acting neuromuscular-blocking agents are preferred, and complete reversal of these agents at the end of the procedure is essential to avoid residual postoperative hypoventilation.

6.14.1 Atelectasis

Atelectasis is uncommon following maxillofacial surgery. The symptoms are determined by the size of the affected area and the presence or absence of a superimposed infectious process. The patient may complain of pleuritic chest pain and mild dyspnea. Hypoxemia is noted if the size of the atelectasis is significant. The treatment usually consists of encouraging the patient to take frequent deep breath, incentive spirometry, and early ambulation.

6.14.2 Aspiration Pneumonitis and Pneumonia

Aspiration is defined as the inhalation of oral, pharyngeal, or gastric contents into the larynx and lower respiratory tract [36]. The aspiration of bacteria from the oral and pharyngeal area causes aspiration pneumonia. The aspiration of gastric contents leads to aspiration pneumonitis. The symptoms include tachypnea and dyspnea, cough, wheezing, and at that times an elevated temperature and hypoxemia. Decreased breath sounds at one of the lung bases and localized rales are noted on physical examination. The chest x-ray reveals an infiltrate usually in the superior segment of the lower lobes.

Aspiration of gastric contents leads to an intense parenchymal inflammatory reaction, and the patients may become severely dyspneic and cyanotic and at times may develop pulmonary edema and hypotension and severe acute respiratory distress syndrome [37].

Aspiration pneumonia related to the inhalation of colonized oropharyngeal material resembles bacterial pneumonia and is less malignant than acidic pneumonitis [38]. Anaerobic organisms are the predominant pathogens, and antibiotic therapy against these organisms is usually required.

Aspiration of large solid particles may cause them to dislodge in the larynx or trachea causing sudden respiratory distress, aphonia, cyanosis, and even death. Immediate extraction is to be performed.

6.14.3 Upper Airway Obstruction

Upper airway obstruction may occur in the pharynx, larynx, or large airways. The patient will be dyspneic, tachypneic, and tachycardic. Signs of upper airway obstruction include intercostal and suprasternal retraction, snoring if partial obstruction is above the larynx, and inspiratory stridor if the obstruction is paralaryngeal [39]. If the obstruction is complete, aphonia is noted.

Factors to consider in the setting of upper airway obstruction are:

- Residual anesthetic effects or sedative effects and inadequate antagonism of neuromuscular blockade leading to pharyngeal muscular weakness causing obstruction of the supraglottic inlet, laryngospasm
- Bilateral recurrent laryngeal nerve palsies leading to vocal cords apposition at the midline
- Massive tongue swelling
- Anaphylaxis
- Angioedema
- Airway edema related to a prolonged head down position and fluid overload
- Aspiration of foreign body such as dislodged teeth

(This complication is a medical emergency and may necessitate immediate endotracheal intubation and inhaled bronchodilating medications. Helium–oxygen mixture may be beneficial if immediate intubation is not required.)

6.14.4 Bronchospasm

Postoperative bronchospasm is most commonly seen in the setting of a previous history of bronchial asthma or chronic obstructive pulmonary disease. It can be caused by reflex constriction of the bronchial smooth muscles related to tracheal stimulation by secretions on suctioning and endotracheal intubation. Bronchospasm could also be related to aspiration or histamine release induced by medications such as opiates and tubocurarine. Pulmonary vascular congestion may at times present as wheezing. In this setting, intravenous loop diuretic is needed. Short-acting inhaled beta-2 agonists and short-acting inhaled anticholinergic agents are the treatment of choice. At times intravenous glucocorticoids need to be used.

6.14.5 Acute Pulmonary Edema

Postoperative pulmonary edema can be cardiogenic or noncardiogenic. Cardiogenic pulmonary

edema may occur in the setting of known left ventricular systolic dysfunction or diastolic dysfunction related to ischemic and/or hypertensive heart disease. An intraoperative myocardial ischemic event or fluid overload is usually the triggering factor. Patients often complain of significant dyspnea. Sinus tachycardia, tachypnea, and hypoxemia are noted. The physical examination usually reveals an elevated jugular venous pressure, bilateral rales, and a ventricular or atrial gallop sound. A chest x-ray shows pulmonary vascular congestion. An electrocardiogram may reveal evidence of myocardial ischemia. The patient should be given an intravenous loop diuretic, intravenous or subcutaneous morphine sulfate, transcutaneous nitrate and supplemental oxygen.

Noncardiogenic pulmonary edema may occur as a result of airway obstruction due to laryngeal spasm or pharyngeal obstruction. This is referred to as negative pressure pulmonary edema related to upper airway obstruction and inspiratory effort against a closed glottis (reverse Valsalva maneuver) leading to increased left ventricular filling, increased pulmonary hydrostatic pressure, and increased afterload [40]. An intravenous loop diuretic and noninvasive continuous positive airway pressure (CPAP) are the treatment of choice. Some patients may require reintubation.

6.14.6 Postoperative Opioid-Induced Respiratory Depression

Opioid analgesics are the most commonly used drugs to treat postoperative pain. These drugs are usually safe. However, opioid-induced respiratory rate depression is often missed by physicians and is a significant cause of acute respiratory failure, death, and hypoxic brain damage in the postoperative period [41]. Understanding the pharmacokinetics and pharmacodynamics of analgesic opioids and their antagonists is essential [42]. The risk of opioid-induced respiratory depression is higher in patients on chronic opiate therapy, obesity, obstructive sleep apnea, and the elderly. It is important to emphasize that oxygen saturation is a measure of gas exchange in the lung rather than an indicator of ventilatory

efficacy and that carbon dioxide concentration is a direct measurement of ventilation.

Continuous monitoring of the heart rate, respiratory rate, pulse oximetry, and capnography combined with an automated alert system to detect early respiratory deterioration can result in improved outcome.

The opioid antagonist naloxone can be used as a rescue medication. The standard dose is 0.4 mg intravenous bolus. In case of recurrent respiratory depression, continuous or repetitive naloxone infusion can be used at a dose of 4–8 µg/kg/h. Potential side effects related to massive release of catecholamines are cardiac arrest, pulmonary edema, cardiac arrhythmias, and seizures [43]. Supplemental oxygen and resuscitation with positive pressure ventilation are to be implemented in the setting of opioid-induced respiratory depression.

It is always essential to identify the patients at risk for this complication and prevent it by the use of the lowest efficacious opioid dose.

Conclusion

Optimizing medical care will ultimately improve our maxillofacial surgery patient safety and outcomes. The maxillofacial surgeon must anticipate and hence avoid and minimize intra- and postoperative complications. This can be accomplished by a thorough preoperative workup which includes a comprehensive history and physical examination, a meticulous review of the list of medications and appropriate consultation with different specialists.

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Anesthesia Complications: Management and Prevention

7

Elie M. Ferneini and Jeffrey Bennett

Abstract

The volume of cosmetic surgery continues to grow rapidly, so does the demand for skilled anesthesia care. Although anesthesia for facial cosmetic surgical procedures remains remarkably safe, no anesthesia should be considered minor. Complications can occur at any time. Proper preoperative evaluation, patient selection, incorporation of a safety checklist, and close collaboration with the surgeon will decrease adverse events leading to increased patient safety and improved outcomes.

7.1 Introduction

The volume of cosmetic surgery continues to grow rapidly. More than ten million cosmetic procedures (surgical and nonsurgical) were performed in 2014 alone, a tenfold increase from 1997 (one million procedures) [1]. In fact, Americans spent more than 12 billion dollars on cosmetic procedures during each calendar year in 2013 and 2014 [1]. More than 80% of these pro-

cedures were performed as outpatient and 60% in an office facility. These procedures have very low rates of perioperative mortality and complications, not exceeding 0.002% and 0.7%, respectively [2–7]. However, facial cosmetic surgery patients may be at a higher anesthesia risk [8].

7.2 Perioperative and Anesthetic Management

Anesthetic management of patients undergoing facial cosmetic surgery presents a unique challenge to the maxillofacial surgeon and anesthesiologist. The essential anesthesia requirements for facial cosmetic surgery include a quiet operating room, clear and clean surgical field, non-stimulating emergence from anesthesia, a rapid return of consciousness and protective airway reflexes, prevention of postoperative nausea and vomiting (PONV), as well as fast-tracking patients for discharge.

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Maintenance of a clear and clean surgical environment is a critical part of facial cosmetic surgical procedures. This is due to the high vascularity of the head and neck area where even a small amount of bleeding can have a significant impact on intra-operative exposure and visualization. Moderate hypotension, local anesthesia with epinephrine infiltration, adequate hemostasis, and meticulous surgical technique allow for optimal outcomes.

Smooth emergence from anesthesia and prevention of PONV are essential to prevent post-op complications such as hematoma formation, postoperative bleeding, and wound dehiscence [9, 10]. This is critical in rhytidectomy and neck procedures [9].

Like any surgery, preoperative risk assessment is essential. For facial cosmetic surgical procedures, patient's health status and type of procedure performed should be taken into consideration [11]. Patients presenting for maxillofacial cosmetic surgery are classified as ASA I (healthy patient), ASA II (patient with mild, controlled systemic disease), or ASA III (patient with severe systemic disease, definite functional impairment). Patients with ASA III physical status have not been shown to have an increased incidence of perioperative complications. Additionally, many patients presenting for esthetic surgery are older. While older age can increase the risks associated with surgery and anesthesia, older age is not a contraindication to ambulatory surgery. An appropriate and detailed preoperative evaluation is essential to decrease adverse events. Having a clear and detailed anesthetic plan is an important part of working up the cosmetic surgery patient. In fact, a wide variety of facial cosmetic surgical procedures can be safely and efficiently performed under monitored anesthesia care (MAC) sedation or general anesthesia [4, 12]. The choice is often dictated by the surgeon's comfort level and preference as well as the patient's desires.

According to the ASA Closed Claims Project database containing 8954 anesthesia malpractice claims, some of the most common major anesthesia-related complications included death (26%), nerve damage (22%), permanent brain damage (9%), airway injury (7%), and medication errors (7%) [13].

Adverse respiratory and cardiovascular incidents accounted for 17% and 13% of all claims associated with anesthesia-related sentinel events [13]. Among the respiratory incidents, difficult intubation, inadequate oxygenation or ventilation, pulmonary aspiration, and airway fires were the most common. In fact, inadequate oxygenation/ventilation has become a growing problem during MAC cases and administration of anesthesia in the non-OR environment [13]. Moreover, between 1997 and 2007, more than 40% of malpractice claims associated with MAC involved the patient's death or permanent brain damage; and as ambulatory esthetic surgery has increased in popularity, the incidence of airway fire claims has grown to be 17% of all anesthetic closed claims [14].

Cardiovascular incidents are less common. Most cardiovascular events leading to anesthesia claims between 1990 and 2007 were related to hemorrhage/blood replacement, fluid management/electrolyte abnormalities, and stroke [13]. Medication problems are relatively common and represented 7% of anesthesia claims between 1990 and 2007 [13]. These claims were equally distributed between adverse drug reactions and medication errors. Additionally, most medication errors were considered preventable [13].

Most anesthesia-related complications in the outpatient setting are minor. The most common include [15]:

- PONV 4.7%
- Shivering 2.2%
- Eye injury 0.056%
- Dental injury 0.02%
- Ulnar neuropathy 0.47%
- Sore throat 28%

7.2.1 MAC Sedation

Many facial cosmetic surgical procedures can be performed under MAC sedation. This eliminates the need for general anesthesia. MAC avoids invasive airway management, removes emergence phenomena, reduces the incidence of PONV, and allows quicker patient discharge [3].

However, oxygenation and ventilatory monitoring may be more challenging in the patient whose airway is open and unprotected. Additionally, high oxygen concentrations (>30% with or without nitrous oxide) and oxygen trapping must be avoided, especially when using electrocautery and lasers. Experience and comfort with MAC sedation is required to prevent adverse events.

MAC claims in facial cosmetic surgery are relatively common, accounting for more than 25% of all MAC claims between 1997 and 2007 [14]. Respiratory depression is the leading cause of inadequate oxygenation/ventilation and death or permanent brain damage in MAC malpractice claims [14]. This was the result of either absolute or relative overdose of sedative or opioid drugs. Nearly half of these claims were judged as preventable by [14]:

1. Better monitoring (including capnography)
2. Improved vigilance
3. Presence of audible monitoring alarms

Conducting safe and successful MAC requires the surgeon to appreciate pharmacokinetic and pharmacodynamic interactions of the chosen intravenous sedative agents, as well as the role of pharmacologic antagonists for opioids and benzodiazepines [16]. A synergistic effect of the sedating agents should be always kept in mind to avoid rapid onset of respiratory depression and upper airway obstruction. Additionally, the doses of the administered medications should be appropriately reduced [17].

Benzodiazepines and opioids are an appropriate combination for sedation and analgesia. Benzodiazepines provide anxiolysis, sedation, and amnesia. Opioids provide analgesia. However, opioids have a proportionately greater degree of respiratory depressant effects compared with benzodiazepines. Despite the respiratory depressant effects of opioids and benzodiazepines, both drugs have specific antagonists [18–20]. Appropriate and planned anesthetic management should avoid the need for reversal agents. Sedation and analgesia should be titrated slowly. The peak onset of midazolam and fentanyl is 8 and 6 min, respectively, and is

slower in the older and/or medically compromised patient. Maximal anesthetic effect may require several minutes.

7.2.2 General Anesthesia

Compared to MAC sedation, general anesthesia with an advanced airway protects the patient's airway, assures adequate ventilation and oxygenation, and removes patient movement. The deeper anesthetic depth more likely will provide more profound amnesia; however, this is dependent on the anesthetic technique and selection of anesthetic agents.

Detailed preoperative airway assessment should be performed on every cosmetic surgery patient. During this examination, risk factors for difficult mask ventilation, airway management, and tracheal intubation must be assessed [21, 22]. A detailed medical and surgical history must be obtained. For example, previous cosmetic procedures, such as chin implant, may mask preexisting retrognathia, thus leading to unanticipated difficult direct laryngoscopy and intubation [23]. Although tracheal intubation is usually performed for most cosmetic facial surgical procedures, the authors believe that a laryngeal mask airway (LMA) can be used for most of these procedures [12]. In fact, the use of a LMA for most cosmetic maxillofacial surgical procedures compared to an open airway provides airway protection and may provide an improved quality of the surgical field. The use of a LMA compared to endotracheal intubation can provide comparable airway protection, improved hemodynamic stability, reduced use of anesthetic medications, a shortened operative time, a decreased likelihood of airway reflex stimulation, and smoother emergence from anesthesia [24–26].

7.3 Complications

Anesthesia-related morbidity ranges from minor complications that can affect our patient's experience with minimal long-term effects to complications with long-term effects and dire adverse

events. The most common complications are cardiovascular and respiratory in nature. Thorough preoperative assessment can help identify risk factors and stratify patients so that we can optimize care.

7.3.1 Cardiovascular Complications

The most common perioperative cardiac complications include myocardial infarction (MI), thromboembolism, arrhythmias, and cardiac arrest.

7.3.2 Myocardial Infarction

The risk of a myocardial infarction (MI) is low: most recent studies suggest up to 5% of patients undergoing elective noncardiac surgery have MI [27]. Most perioperative ischemic events are usually silent without any clinical signs and symptoms. However, the incidence is reported at 4.4% in the presence of a cardiac risk factor [28].

Perioperative MI is usually hard to predict and prevent. It usually occurs in the first 48 h postoperatively [29]. Most of the cause of a perioperative MI is an oxygen-demand mismatch. Surgery in general confers a physiologic stress to the patient that leads to increased oxygen demand. Patient risk stratification is important in assessing the likelihood of perioperative cardiac complications. The most commonly used risk index is the Revised Cardiac Risk Index (RCRI). This index identifies six independent predictors of major cardiac complications. The risk increases with the presence of each additional risk factor. These include [30]:

- High-risk surgery, e.g., intraperitoneal, intrathoracic
- History of ischemic heart disease: history of MI, history of positive exercise test, current chest pain considered due to MI, use of nitrate therapy, ECG with pathological Q waves
- History of heart failure: pulmonary edema, paroxysmal nocturnal dyspnea
- History of cerebrovascular disease: prior TIA or CVA

- Diabetes requiring insulin
- Chronic renal impairment: preoperative creatinine >2 mg/dL

Depending on each patient's risk factors, measures should be taken to improve cardiac outcomes. β -Blockers have been shown to decrease the incidence of perioperative MI; however there is a potential increase in the incidence of stroke [28, 31]. In fact, perioperative β -blockade started within 1 day or less before noncardiac surgery prevents nonfatal MI but increases risks of stroke, death, hypotension, and bradycardia [32]. Perioperative statins can be beneficial: statin treatment in statin-naive patients reduces atrial fibrillation, myocardial infarction, and duration of hospital stay in patients undergoing cardiac or noncardiac surgery [33]. Clonidine and aspirin could be beneficial in reducing the risk of major cardiovascular events. However, in a recent study, the administration of aspirin before surgery and throughout the early postsurgical period had no significant effect on the rate of a composite of death or nonfatal MI but increased the risk of major bleeding [34].

7.3.3 Thromboembolism

Venous thromboembolism (VTE) is a significant cause of morbidity and mortality in the perioperative period. VTE includes both deep vein thrombosis (DVT) and pulmonary embolism (PE). In patients undergoing plastic surgery, the overall incidence of VTE is 1.69% [35]. The executive committee of the American Society of Plastic Surgeons (ASPS)-approved Venous Thromboembolism Task Force Report identified the best practices for DVT/PE prevention and treatment [36]. Additionally, the ASPS has published articles offering recommendation for DVT prophylaxis based on the risk levels [37, 38]. The highest risk is associated with liposuction and abdominoplasty procedures. For maxillofacial cosmetic surgical procedures, facelift procedures carry a higher risk of VTE. In fact, rhytidectomy and its association with DVT/PE have been documented by Rigg, Reinisch, and Abboushi [39–41].

| | | |
|--|---|---|
| Each Risk Factor = 1 point Age 41-60 years Minor surgery planned Prior major surgery (<1 month) Varicose veins History of inflammatory bowel disease Swollen legs Obesity (BMI>25) Acute myocardial infarction Congestive heart failure (<1 month) Sepsis (<1 month) Serious lung disease (<1 month) Abnormal pulmonary function (e.g. COPD) Medical patient currently at bed rest Other risk factors | Each Risk Factor = 2 points Age 60-74 Arthroscopic surgery Malignancy (present or previous) Major surgery (>45minutes) Laparoscopic surgery (>45minutes) Patient confined to bed (>72 hours) Immobilizing plaster cast (<1 month) Central venous access | Each Risk Factor = 5 points Elective major lower extremity arthroplasty Hip, pelvis or leg fracture (<1 month) Stroke (<1 month) Multiple trauma (<1 month) Acute spinal cord injury (<1 month) |
| | Each Risk Factor = 3 points Age > 75 years History of DVT/PE Family history of thrombosis Positive Factor V Leiden Positive Prothrombin 20210A Elevated serum homocysteine Positive lupus anticoagulant Elevated anticardiolipin antibodies Heparin-induced thrombocytopenia Other congenital/acquired thrombophilia | For Women Each Risk Factor = 1 point Oral contraceptives or hormone replacement therapy Pregnancy or postpartum (<1 month) History of unexplained stillborn infant, recurrent spontaneous abortion (>3), premature birth with toxemia or growth-restricted infant |

Fig. 7.1 Thrombosis risk assessment (Data from Murphy RX, Schmitz D, Rosolowski K. Evidence based practices for thromboembolism prevention: a report from the ASPS

Venous Thromboembolism Task Force. Arlington Heights (IL): American Society of Plastic Surgeons; 2011)

The maxillofacial surgeon must assess and stratify the risk of VTE for each patient. The Caprini risk assessment model is a helpful tool to stratify your patients (Fig. 7.1) [42]. Current recommendations on thromboprophylaxis in surgery patients are based on the calculated risk of VTE and consideration of the risk of bleeding associated with any intervention. These recommendations are summarized in Table 7.1 [43].

7.3.4 Arrhythmias

Arrhythmias are rare in the perioperative period. In fact, less than 1% of all surgery patients experience a bradyarrhythmia or ventricular arrhythmia that is severe enough to require treatment [44]. In the perioperative period, atrial fibrillation is the most common arrhythmia with an incidence of 0.37–20% in noncardiac surgery patients [45]. Preoperative risk factors for the development of atrial fibrillation include increasing age, male gender, preexisting heart disease, ASA III and/or IV, and preoperative electrolyte disturbances [45].

For most new-onset arrhythmias in the perioperative period, no medical management is required. They are usually self-limiting with more than 80% reverting to sinus rhythm before

Table 7.1 Incidence of VTE and recommendations for thromboprophylaxis based on Caprini risk assessment model (RAM) score

| Caprini RAM score | Risk of VTE (%) | Recommended thromboprophylaxis |
|-------------------|-----------------|--|
| 0 | <0.5 | Nil/ambulate early |
| 1–2 | 1–5 | Mechanical prophylaxis (IPC) |
| 3–4 | 3.0 | If not high risk for major bleeding: LMWH or LDUH or mechanical prophylaxis (IPC) If high risk for major bleeding: mechanical prophylaxis (IPC) |
| >5 | 6.0 | LMWH or LDUH and mechanical prophylaxis (IPC) |

Abbreviations: IPC intermittent pneumatic compression, LDUH low-dose unfractionated heparin, LMWH low molecular weight heparin

Data from Gould MK. Prevention of VTE in nonorthopedic surgical patients: antithrombotic therapy and prevention of thrombosis. Chest 2012; 141:e2275–775

discharge [45]. Management includes recognition and initiating rate and rhythm control.

7.3.5 Cardiac Arrest

The risk of anesthesia-related cardiac arrest is 1.86:10,000 [46]. General anesthesia is a risk factor for cardiac arrest. In fact, more than

90% of anesthesia-related cardiac arrests are related to airway management or medication administration.

7.3.6 Respiratory Complications

Respiratory complications are major predictors of morbidity in the perioperative period. Its incidence is at 6.8% with serious complications occurring at 2.6% [47]. Adverse respiratory events were the primary contributing factors resulting in death in out-of-operating room locations, of which 50% were associated with MAC sedation [48]. The complications discussed in this section include hypoxia/hypopnea/apnea, aspiration, bronchospasm, atelectasis, and airway crisis.

7.3.7 Hypoxia/Hypopnea/Apnea

Anesthetic agents depress ventilatory drive and contribute to loss of upper airway patency. The latter results from the loss of pharyngeal dilator muscular support affecting the tensor palatini, genioglossus, and hyoid muscles, which dilate the nasopharynx, oropharynx, and laryngopharynx, respectively, and maintain airway patency. As patients age, these muscles become less efficacious and the patients are more susceptible to becoming obstructed. In order to detect impairment of airway exchange either from respiratory depression or obstruction, the patient must be monitored using both pulse oximetry (providing oxygen saturation) and capnography and auscultation (providing respiratory rate and end-tidal carbon dioxide). While oxygen saturation is obviously important, it provides limited information when registering 100% saturation in the oxygenated patient because it does not provide the actual arterial oxygen content. The limitation is illustrated by the following. The arterial oxygen content in a patient receiving 40% oxygen may approximate 180 mmHg with an oxygen saturation of 100%; the same patient whose airway

and/or ventilation is compromised may have an oxygen saturation of 100 mmHg with an oxygen saturation of 100%. The incorporation of ventilatory monitoring into routine anesthesia care can detect respiratory depression that may be the primary factor contributing to the lower arterial oxygen content. The first ASA closed claims report suggested the lack of ventilatory monitoring as the most significant component contributing to morbidity and mortality.

Capnography/capnometry is the standard of care for monitoring ventilation. It provides a waveform that shows the respiratory pattern, the respiratory rate, and a measurement of the end-tidal carbon dioxide. In the patient with an advanced airway (e.g., intubation), the practitioner can set ventilatory parameters based on end-tidal carbon dioxide. The absolute end-tidal carbon dioxide has limited value in the patient with an open airway. In this situation, the generated waveform and corresponding respiratory rate can monitor ventilation and demonstrate potentially ominous changes before they may be manifested by changes in oxygen saturation. There are several ways in which air sampling can be achieved. A nasal cannula in which oxygen is delivered via one naris and carbon dioxide is sampled via the other naris is noninterfering with most procedures. These authors also recommend the use of an electronic wireless stethoscope that magnifies sounds and is placed in the pre-tracheal region in the open-airway patient. Auscultation can identify respiratory changes not detected by capnography. Furthermore, the sampling line can become displaced or obstructed in the open-airway patient. The viewable capnographic screen and the stethoscope wirelessly connected to a speaker allow the entire team to be observant of real-time ventilatory changes.

7.3.8 Aspiration

Aspiration of gastric contents into the airway is the most common cause of airway-related death during anesthesia [49]. It occurs in 1:4000 patients undergoing general anesthesia. The

highest risk is at intubation as well as extubation [49, 50]. Prevention is an important step in decreasing the incidence of aspiration. It is aimed at identifying patients at risk. These factors are divided into four categories: patient factors,

surgical factors, anesthetic factors, and device factors. Box 7.1 summarizes these risk factors [51]. The surgeon must follow specific strategies to reduce the risk of aspiration. Box 7.2 discusses some of these strategies.

Box 7.1 Risk Factors for Aspiration (Adapted from Asai [6] with Permission from the British Journal of Anaesthesia)

Patient factors

- (a) Full stomach
 - Emergency surgery
 - Inadequate fasting time
 - Gastrointestinal obstruction
- (b) Delayed gastric emptying
 - Systemic diseases, including diabetes mellitus and chronic kidney disease
 - Recent trauma
 - Opioids
 - Raised intracranial pressure
 - Previous gastrointestinal surgery
 - Pregnancy (including active labor)
- (c) Incompetent lower esophageal sphincter
 - Hiatus hernia
 - Recurrent regurgitation

- Dyspepsia
- Previous upper gastrointestinal surgery
- Pregnancy
- (d) Esophageal diseases
 - Previous gastrointestinal surgery
 - Morbid obesity

Surgical factors

- Upper gastrointestinal surgery
- Lithotomy or head down position
- Laparoscopy
- Cholecystectomy

Anesthetic factors

- Light anesthesia
- Supraglottic airways
- Positive pressure ventilation
- Length of surgery >2 h
- Difficult airway

Device factors

- First-generation supraglottic airway devices

Box 7.2 A Summary of the Available Strategies for Reducing Aspiration Risk

| | |
|---------------------------------|---|
| Reducing gastric volume | Preoperative fasting |
| | Nasogastric aspiration |
| | Prokinetic premedication |
| Avoidance of general anesthetic | Regional anesthesia |
| Reducing pH of gastric contents | Antacids |
| | H ₂ histamine antagonists |
| | Proton pump inhibitors |
| Airway protection | Tracheal intubation |
| | Second-generation supraglottic airway devices |
| Prevent regurgitation | Cricoid pressure |
| | Rapid sequence induction |
| Extubation | Awake after return of airway reflexes |
| | Position (lateral, head down, or upright) |

Guidelines to reduce the risk of aspiration include:

- Experienced anesthesia assistance and supervision available at all times.
- Applying appropriate cricoid pressure with all inductions using neuromuscular blocking agents.
- Consider intubation in patients with:
 - Delayed gastric emptying (opioids, diabetes mellitus, renal failure)
 - Increased intra-abdominal pressure (obesity)
- Extubating high-risk cases awake and on their side and extubating all others on their side.
- A LMA does not protect against aspiration and endotracheal intubation should be considered in at-risk patients.

7.3.9 Bronchospasm

Bronchospasm occurs in 0.2% of patients undergoing general anesthesia. It is a lower airway obstruction due to contraction or spasm of bronchial smooth muscle. It can lead to hypoxia, hypotension, or even death [52, 53]. Patients with preexisting airway disease, a recent or active upper respiratory tract infection, and smoking history are at increased risk of developing a bronchospasm. Manifestations of bronchospasm include rapidly increasing peak inspiratory pressure (with plateau pressure unchanged), wheezing, slowly increasing wave on the capnograph, and decreasing exhaled tidal volumes. The patient will actually exhibit dyspnea and wheezing attributed to chest obstruction. The key triggers include:

- Airway instrumentation
- Airway irritation
- Early surgical stimulation without adequate depth of anesthesia
- Some medications (e.g., β -blockers, neostigmine, morphine)
- Anaphylaxis

Prevention of bronchospasm relies on optimizing any underlying airway disease, encouraging smoking cessation, delaying elective cosmetic surgery if recent or acute upper respiratory infec-

Box 7.3 Bronchospasm: Acute Treatment Preoperative

- Supplemental oxygen
- Inhaled β_2 -agonists (e.g., albuterol)
- Intravenous steroids

Intraoperative

1. Deepen anesthetic—increase volatile anesthetic concentration
 - (a) All volatile anesthetic agents are bronchodilators
 - (b) Ketamine
 - (c) Propofol (protection against bronchoconstriction)
2. Consider alternative causes of high airway pressures, e.g., kinked tube, endobronchial intubation, etc.
3. Inhaled β_2 -agonists—delivered to the inspiratory limb of the circuit through a metered dose inhaler or nebulized
4. Epinephrine—subcutaneous (1:1000) versus intravenous (1:10,000). If the severity of bronchospasm prohibits delivery of inhaled beta-agonists, consider infusions of IV agonists such as terbutaline or epinephrine
5. Consider administering intravenous steroids

tion, and avoiding unnecessary airway manipulation. Box 7.3 discusses appropriate management of an acute bronchospasm.

7.3.10 Laryngospasm

Laryngospasm is a serious complication which can lead to airway obstruction and even death. It is a reflex spasm of the striated muscles of the larynx, resulting in partial or complete closure of the glottis and an inability to ventilate the lungs. Any patient under IV sedation or general anesthesia whose airway is not protected by an ET tube may experience laryngospasm. Laryngospasm can be also caused

by airway manipulation (extubation, insertion of a LMA, suctioning), secretions in the pharynx, surgical stimulus due to light sedation, vomiting, and patient movement. Signs include inspiratory stridor, complete airway obstruction, increased inspiratory efforts, paradoxical chest and abdominal movements, desaturation, and bradycardia.

Airway patency may be restored with jaw thrust and positive pressure mask ventilation with 100% oxygen. Deepening anesthesia with a rapidly acting intravenous agent, such as propofol, is frequently helpful. If these measures fail, a muscle relaxant, typically succinylcholine, is administered. Endotracheal intubation may not be necessary because succinylcholine is short acting. Mask ventilation until the return of spontaneous breathing is often sufficient. The patient may require intubation if he or she has difficult mask ventilation or if the laryngospasm doesn't resolve. The complications of laryngospasm include aspiration and negative inspiratory pressure pulmonary edema, and the patient may require ventilatory support in an intensive care setting [54].

7.3.11 Airway Crisis: "Cannot Ventilate, Cannot Intubate"

Patients with difficult airway usually require multiple attempts at intubation during airway crisis. These attempts have been associated with death or permanent brain damage [13]. The office should be capable of performing direct laryngoscopy with both a Macintosh and Miller blade and videolaryngoscopy. Videolaryngoscopy provides a more rapid view of the glottis but is an indirect technique that requires a different hand-eye coordination compared to traditional laryngoscopy. Videolaryngoscopy has become the standard of care in managing the difficult airway. Attempts at laryngoscopy and intubation should be limited. A good rule of thumb is to limit the intubation attempts to three before placement of a LMA. If ventilation with a LMA is not successful, a surgical airway should be secured.

The importance of preoperative airway assessment to identify patients with a difficult airway cannot be overemphasized. A patient with a suspected difficult airway should not

have a procedure in an outpatient facility, unless the cosmetic procedure is performed under minimal sedation.

7.3.12 Postoperative Nausea and Vomiting

The incidence of vomiting is about 30%, and the incidence of nausea is about 50%. In general, PONV is experienced by a high number of our cosmetic surgery patients: 20–30%. It can be as high as 70–80% in high-risk patients [55–57]. Unresolved PONV can result in prolonged postanesthesia care unit (PACU) stay and increased patient dissatisfaction. Additionally, it increases adverse events of maxillofacial cosmetic surgical procedures. The goal of PONV prophylaxis is to decrease the incidence of PONV leading to decreasing patient-related distress and healthcare costs [58–60].

Preoperative assessment of high-risk patients can lead to prophylactic management which leads to decreased incidence. Box 7.4 summarizes risk factors for PONV [55, 61]. Four factors (female sex, nonsmoking status, history of PONV or motion sickness, and opioid use) have been well documented. In fact, the incidence of PONV is estimated at 10%, 20%, 40%, 60%, and 80% depending on the presence of none, 1, 2, 3, or 4 risk factors, respectively [62]. Guidelines have been developed that identify patients at risk for PONV, approaches for

Box 7.4 PONV Risk Factors

- 1 Patient factors
 - Female gender postpuberty
 - Nonsmoking status
 - History of PONV or motion sickness
- 2 Anesthetic factors
 - Use of volatile anesthetics
 - Use of nitrous oxide
 - Use of intraoperative/postoperative opioids
 - Use of large-dose neostigmine
- 3 Surgical factors
 - Duration of surgery >30 min
 - Type of surgery (plastic and maxillofacial)

reducing baseline risks for PONV, effective antiemetic therapy for PONV prophylaxis, and strategies for treatment of PONV. Tables 7.2, 7.3, and 7.4 provide an algorithm for the

management of individuals at increased risk for PONV as well as steps to ensure PONV prevention and treatment are implemented [63, 64]. Some elements to consider in patient management are as follows: decadron is both an anti-inflammatory and antiemetic agent at 4 mg; midazolam 2 mg provides anxiolysis, amnesia, and sedation and a dose as little as 2 mg given a minimum of 30 min prior to the end of the case reduces PONV; several of the antiemetics act on the dopamine receptor and have the potential to produce extrapyramidal effects which if it occurs is treated with administration of diphenhydramine; several of the antiemetics (even the serotonin antagonists, more likely with higher

Table 7.2 Strategies to reduce risk of PONV

Table 7.3 Risk-adapted PONV prevention algorithm (with no prevention in low-risk patients) [63]

| | Estimated risk for PONV, for example, as determined by a risk score | | |
|-------------------------------|--|--|--|
| | Low | Medium | High |
| Interventions for prophylaxis | No prevention (“wait and see”) | Drug A + Drug B or TIVA | Drug A + Drug B + TIVA On a case-by-case decision: further interventions |
| Interventions for treatment | 1. Drug B 2. Drug C (in case of ineffectiveness of treatment in stage 1) (i.e., Drug B) | 1. Drug C 2. Drug D (in case of ineffectiveness of treatment in stage 1) (i.e., Drug C) | 1. Drug C 2. Drug D (in case of ineffectiveness of treatment in stage 1) (i.e., Drug C) |

Example interventions: Drug A, dexamethasone 4 mg in adults/0.15 mg/kg of body weight in children; Drug B, ondansetron 4 mg in adults/0.1 mg/kg of body weight in children; Drug C, droperidol 1 mg in adults/10–15 µg/kg of body weight in children; Drug D, dimenhydrinate 1 mg/kg of body weight in adults/0.5–1.0 mg/kg of body weight in children. Given drug examples are used to illustrate how the algorithm may be actually implemented but may not represent the most favorable approach. The latter may be context sensitive (children, adults, or other issues). In the event of treatment failure, a timely assessment and alternative antiemetics should be used. A multimodal treatment approach may be appropriate to increase the likelihood of success

TIVA total intravenous anesthesia, that is, propofol induction and maintenance, no nitrous oxide

Table 7.4 PONV prevention algorithm in all patients including low-risk patients plus additional interventions for high-risk patients [63]

| | Estimated risk for PONV, for example, as determined by a risk score | | |
|-------------------------------|--|--|--|
| | Low | Medium | High |
| Interventions for prophylaxis | Drug A + (Drug B or TIVA) | Drug A + (Drug B or TIVA) | Drug A + Drug B + TIVA On a case-by-case decision: further interventions |
| Interventions for treatment | 1. Drug C 2. Drug D (in case of ineffectiveness of treatment in stage 1) (i.e., Drug C) | 1. Drug C 2. Drug D (in case of ineffectiveness of treatment in stage 1) (i.e., Drug C) | 1. Drug C 2. Drug D (in case of ineffectiveness of treatment in stage 1) (i.e., Drug C) |

Example interventions: Drug A, dexamethasone 4 mg in adults/0.15 mg/kg of body weight in children; Drug B, ondansetron 4 mg in adults/0.1 mg/kg of body weight in children; Drug C, droperidol 1 mg in adults/10–15 µg/kg of body weight in children; Drug D, dimenhydrinate 1 mg/kg of body weight in adults/0.5–1.0 mg/kg of body weight in children. Given drug examples are used to illustrate how the algorithm may be actually implemented but may not represent the most favorable approach. The latter may be context sensitive (children, adults, or other issues). In the event of treatment failure, a timely assessment and alternative antiemetics should be used. A multimodal treatment approach may be appropriate to increase the likelihood of success

TIVA total intravenous anesthesia, that is, propofol induction and maintenance, no nitrous oxide

doses used with chemotherapy and/or in patients with cardiovascular disease) can cause QT prolongation [65, 66].

7.3.13 Malignant Hyperthermia

Malignant hyperthermia (MH) is a true anesthesia emergency and a potentially fatal condition which results in tachycardia, muscle rigidity, hypercapnia, and hyperthermia. It is an autosomal dominant, pharmacogenetic disorder of the skeletal muscle. When exposed to a trigger agent, patients susceptible to MH have disordered regulation of calcium within the skeletal muscle leading to a hypermetabolic state [67, 68]. Major triggers include all volatile anesthetic agents and depolarizing muscle relaxants (succinylcholine).

The incidence of MH is in the order of 1:10,000 to 1:50,000; however the prevalence of MH is up to 1:3000 patients [67]. The annual number of suspected MH cases per year in the United States is around 700. Larach et al. reported that in 291 MH episodes recorded in the North American Malignant Hyperthermia Registry database between 1987 and 2006, there were eight cardiac arrests and four deaths [69].

MH may occur either in the operating room (OR) or in the early postoperative period. The earliest sign is an increase in end-tidal carbon dioxide. A fulminant reaction then occurs with very high end-tidal carbon dioxide (>100 mmHg), a low pH with a metabolic component, tachycardia and dysrhythmias, rigidity, rapidly increasing temperature, hyperkalemia, myoglobinuria, muscle edema, increased cellular permeability, disseminated intravascular coagulopathy (DIC), and even cardiac and renal failure.

Treatment consists of early recognition, removal of the trigger agent (including the necessity for high flow oxygen from a machine not exposed to a potent inhalational agent), and administration of dantrolene. Dantrolene is available as Dantrium (Revonto) or Ryanodex. Dantrium comes as a vial containing 20 mg dantrolene powder that is reconstituted with 60 mL sterile water. The vial is shaken until the solution is clear. Ryanodex comes as a vial containing

250 mg dantrolene. The powder is reconstituted with 5 mL sterile water and is shaken creating an orange-colored opaque suspension. Preparation time comparing Dantrium to Ryanodex is 860 s compared to 51 s, respectively. The initial dose is 2.5 mg/kg for either agent. The Malignant Hyperthermia Association of the United States (MHAUS) recommends that any office in which an inhalational agent (excluding nitrous oxide) or succinylcholine may be administered must have dantrolene. A patient with a history of MH can be safely anesthetized if triggering agents are avoided. The caffeine halothane contracture test (CHCT) is the standard for establishing the diagnosis of MH. The test is performed on freshly biopsied muscle tissue [70].

The issue with managing a patient with MH pertains to the potential for emergent airway intubation when succinylcholine is absolutely contraindicated. Rocuronium 1.2 mg/kg administered intravenously has an onset of 60–90 s providing satisfactory intubating conditions. However, rocuronium provides a neuromuscular blockade of intermediate duration. The FDA approved use of sugammadex in December 2015 to reverse the neuromuscular blockade induced by rocuronium. Sugammadex 16 mg/kg can be administered 3 min after the administration of an intubating dose of rocuronium providing neuromuscular blockade reversal in approximately 4.4 min from the time of rocuronium administration.

7.3.14 Sore Throat

Sore throat is a common postoperative complaint and affects up to 12.1% of patients at 24 h after surgery [71, 72]. It is directly related to the degree of airway instrumentation. The use of a LMA reduces the incidence to between 17.5% and 34% compared to 50% with endotracheal tube [71, 72]. The incidence of sore throat increases with the diameter of the endotracheal tube, as a result of increased pressure at the tube-mucosal interface [73]. Other factors which increase the incidence of sore throat include high-volume pressure cuffs, uncuffed endotracheal tubes, and lidocaine gel use for lubrication. For most maxillofacial cosmetic surgical procedures, using a LMA

decreases the incidence of sore throat. Some factors that increase this incidence include:

- Larger size LMA
- Multiple attempts at insertion
- Longer surgery time

For most cosmetic surgery patients, a sore throat is a self-limiting complication that does not require specific treatment and responds well to simple analgesia.

7.3.15 Operating Room Fire

Fires in the operating room are unanticipated, devastating events that can lead to significant morbidity, even mortality, for the patient and medical personnel. As maxillofacial surgeons, the topic of fire safety demands attention and preparedness. Approximately 78% of all fires occur during facial, neck, and tonsil surgery [74]. In fact, operating room fires account for nearly a fifth of MAC-related closed claims [75]. The most common ignition source in operating room fires is the electrosurgical unit, which is used in most facial cosmetic surgical procedures. Additionally, supplemental oxygen was also present [76, 77]. Fires can occur in any setting, provided these three elements are in close proximity under the right conditions: (1) heat or an ignition source, (2) fuel, and (3) an oxidizer [78].

In maxillofacial cosmetic surgery, lasers and electrocautery are major causes of fires. Use of a bur on metal (e.g., sectioning a crown to facilitate extraction of a tooth) may also promote a spark which may cause a fire. Oxidizers are always present and can increase the chance of a fire. Oxygen is a primary oxidizer and is typically present in every MAC or general anesthetic. Additionally, nitrous oxide, when heated, decomposes into its constituent elements, thus increasing the ambient oxygen. In fact, in most MAC cases of a fire, supplemental oxygen was used. Ninety-five percent of cases involved the head or neck, while alcohol-containing prep solutions and/or drapes were the most common

fuel sources [75]. Special attention must be directed to patient draping to avoid oxygen from being trapped within the surgical field. The flow rate of supplemental oxygen, such as a nasal cannula, should be set to the lowest possible setting to reduce unnecessary excess oxygen while still maintaining safe blood oxygenation saturation levels [79, 80]. Ideally, if electrocautery or laser is being used, oxygen administration should be replaced with medical grade air. Consideration to using advanced airways (e.g., intubation) removes the potential for an oxygen-enriched surgical field. If an advanced airway, either a specific laser tube or protective measures must be employed to avoid igniting the airway.

The best method of fighting a fire is to avoid one in the first place. Great emphasis should be placed on prevention. Education and training in fire risk reduction strategies for nurses, surgical technicians, anesthetists, and surgeons is essential to promote and maintain a fire-safe environment (Table 7.5).

If a fire occurs, it is the responsibility of all team members to protect the patient. Each individual plays a vital role in fire control. Often, the RACE method is used to guide actions when a fire occurs.

Table 7.5 Training programs should include details on the role-specific actions that are to be taken by the operating room staff to prevent fires

| Examples include |
|--|
| • Keep the electric cautery tip in a holster when it is not being used |
| • Turn off all high-intensity light sources when not in use |
| • Using cuffed endotracheal tubes and appropriate use of oxygen gas |
| • Beware of patient draping so as to not create occlusive “tent” to trap in oxidizing agents |
| • Attempt to use water-soluble preparation solutions and ointments |
| • Consider flame-resistant surgical drapes |
| • Know the location of the fire extinguishers, alarms, and gas valves |
| • Obtain proficiency with fire extinguishers and equipment |
| • Know the location of all fire alarm and fire exits [80] |

The fire-protection mnemonic stands for [80]:

- **R**escue: In the event of a fire, rescue means moving patients out of harm's way. All airway gases should be stopped and the safety of the patient and team members should be ensured.
- **A**larm (or alert): The person who notices the fire should call out and alert others to the situation. The surgical and anesthesia teams should work together. The surgeon must determine the point at which it is safe to stop the cosmetic procedure, while the rest of the team will decide how and where to move the patient.
- **C**onfine (or contain): After moving the patient away from the immediate fire danger, team members should work on containing the fire in order to prevent smoke and fire from entering other surgical rooms.
- **E**xtinguish: Extinguishing the fire is an important step. Three types of portable extinguishers are available: ABC, carbon dioxide, and water mist [80].

7.3.16 Local Anesthetic Toxicity

Local anesthesia is a major adjunct to maxillofacial cosmetic surgery. Local anesthesia can be used by itself or in tumescent solution. In fact, tumescent solution is often used in many procedures such as facelift, neck lift, and brow lift.

Lidocaine with epinephrine is usually added to the tumescent solution. The addition of epinephrine is important for hemostasis, prolonged anesthesia, and lowering the peak lidocaine levels.

7.3.17 Lidocaine Safe Dosing

Potential lidocaine toxicity is an important consideration, especially when using tumescent anesthesia. Traditionally, 7 mg/kg of lidocaine (with epinephrine) is accepted as the maximum dose for dermal infusion [81]. However, when used for tumescence, the ceiling is dramatically increased to 35 mg/kg (others report as high as 55 mg/kg). With this dosage, plasma concentrations of lidocaine remain below toxic levels (<5 mg/L) [82, 83].

7.3.18 Lidocaine Toxicity

Potential lidocaine toxicity can occur with tumescent anesthesia. Maxillofacial surgeons must recognize the signs and symptoms. Some of these symptoms can occur at 6 µg/mL but usually manifest at concentrations exceeding 10 µg/mL. Some of the signs include drowsiness, disorientation, cardiovascular instability, and seizures. Table 7.6 reports the CNS and cardiovascular effects of lidocaine toxicity [84].

Table 7.6 Effects of lidocaine on the CNS and cardiovascular system (CVS) [84]

| Concentration (mg/mL) | CNS effect ^a | CVS effect ^a |
|-----------------------|---|---|
| <5 | Anticonvulsant activity Mild sedation Analgesia Circumoral paresthesia | Antiarrhythmic activity Mild increases in mean blood pressure with similar increases in cardiac output or peripheral vascular resistance |
| 5–10 | Light-headedness, slurred speech, drowsiness, restlessness, euphoria | Cardiovascular instability |
| 10–15 | Disorientation, uncontrollable tremors, respiratory depression, tonic-clonic seizures | |
| 15–20 | Coma Respiratory arrest | |
| >20 | | Profound myocardial depression, vasodilatation, cardiovascular collapse |

^aCNS and CVS effects are listed in approximate order of occurrence with increasing blood concentration

Adapted from Yagiela JA. Local anesthetics. *Anesth Prog* 1991; 38:128–41; and Butterwick KJ. Goldman MP Sripachya-Anunt S. Lidocaine levels during the first 2 hours of infiltration of dilute anesthetic solution for tumescent liposuction rapid versus slow delivery. *Dermatol Surg* 1999; 25(9):681–5

Table 7.7 Checklist for treatment of local anesthetic systemic toxicity [85]

| |
|--|
| 1. Get help |
| 2. Initial focus |
| (a) Airway management |
| (b) Seizure suppression: benzodiazepines are preferred |
| (c) Alert the nearest facility having cardiopulmonary bypass capability |
| 3. Management of cardiac arrhythmias |
| (a) Basic and advanced cardiac life support |
| (b) Avoid vasopressin, calcium channel blockers, beta-blockers, or local anesthetic |
| (c) Reduce individual epinephrine dose to <1 µg/kg |
| 4. Lipid emulsion (20%) therapy |
| (a) Bolus 1.5 mL/kg (lean body mass) |
| (b) Continuous infusion 0.25 mL/kg/min |
| (c) Repeat bolus once or twice for persistent cardiovascular collapse |
| (d) Double the infusion rate to 0.5 mL/kg/min if blood pressure remains low |
| (e) Continuous infusion for at least 10 min after attaining circulatory stability |
| (f) Recommended upper limit: approximately 10 mL/kg lipid emulsion over the first 30 min |

7.3.19 Intravenous Lipid Emulsion Therapy for the Management of Local Anesthetic Toxicity

The administration of intravenous lipid emulsion for the management of local anesthetic systemic toxicity has evolved over the past several years. The American Society of Regional Anesthesia and Pain Management has published recommendations, although no prospective randomized trial has been performed (Table 7.7) [85].

7.4 Cosmetic Procedure-Related Anesthesia Safety Considerations

7.4.1 Rhytidectomy

Rhytidectomy can be performed using endotracheal intubation, LMA, or MAC sedation. If MAC sedation is performed, tumescent local anesthesia is often used. During a facelift procedure, monitoring the facial nerve twitches during local anesthetic

injection in the facial nerve planes is recommended. This reduces the possibility of direct trauma to and/or paralysis of the facial nerve.

If the surgeon prefers tracheal intubation, succinylcholine can be administered. However, in conjunction with the inhalational anesthetic agents, this may trigger MH in susceptible patients. A combination of intravenous remifentanyl 2 mg/kg, propofol 2 mg/kg, and lidocaine 1.5 mg/kg and only half of an intubating dose of rocuronium (0.3 mg/kg) will achieve intubating conditions similar to those from administration of intravenous succinylcholine 1.5 mg/kg [86]. Postoperative hematoma is an undesired adverse event. Aggressive control of postoperative hypertension and PONV is essential in preventing this complication. Additionally, limiting intravenous fluids and bladder distension will also help in reducing postoperative patient discomfort, agitation, and hypertensive responses.

As mentioned above, DVT and PE constitute the greatest cause of morbidity and mortality after ambulatory surgery and are the most feared complications of prolonged facial cosmetic surgery, such as a rhytidectomy [87]. However, their incidence is extremely low: 0.35% for a DVT and 0.14% after a PE [40]. Prophylaxis plays a major role in prevention. Additionally, early ambulation and adequate postoperative pain control are important in reducing the risk of a VTE [40, 88].

7.4.2 Blepharoplasty

Blepharoplasty was among the top 5 cosmetic surgical procedures performed in 2014 [1]. Most cases can be performed under local anesthesia with or without sedation [12]. Some surgeons prefer general anesthesia for this procedure. If performed under MAC sedation, deeper planes of sedation may be required while injecting local anesthesia into the lower eyelids to prevent patient discomfort [89]. In some cases, general anesthesia might be indicated in order to prevent patient's movement, thus decreasing the risk of complications such as an intravascular injection and globe damage [89, 90].

7.4.3 Rhinoplasty

Because of the increased risk of laryngospasm or intraoperative coughing because of increased aspiration of blood and/or secretions, general anesthesia is preferred over MAC sedation.

A rare postoperative complication is Tapia syndrome. It is characterized by concomitant paralysis of the hypoglossal and the recurrent laryngeal branch of the vagus nerve. It is usually associated with endotracheal anesthesia for rhinoplasty, possibly resulting from excessive pressure of the throat pack in the hypopharynx [91].

Other complications include postoperative skull base defects and tension pneumocephalus. Tension pneumocephalus, a medical emergency, is usually a result of a poor surgical technique [92].

Conclusion

As the number of cosmetic surgical procedures performed in the United States continues to grow, so does the demand for skilled anesthesia care. Although anesthesia for facial cosmetic surgical procedures remains remarkably safe, no anesthesia should be considered minor. Complications can occur at any time. Proper preparation, preoperative evaluation, patient selection, incorporation of a safety checklist, and close collaboration and communication with the surgeon will decrease adverse events leading to increased patient safety and patient satisfaction.

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Abstract

Although postoperative infection in facial cosmetic surgery is rare, its consequence is devastating to the patient as well as the surgeon. Prevention is the primary goal. This chapter will discuss postoperative infections associated with different cosmetic maxillofacial surgical procedures. Management as well as prevention of these complications will also be addressed.

8.1 Introduction

Infection is an important consideration in facial cosmetic surgery. Fortunately, several influences contribute to low infection rates in the head and

neck region. These factors include an abundance of blood supply in the anatomical area, a reduced risk of surgical contamination, and several aspects of patient selection.

The extensive vascularity of the head and neck region leads to accelerated healing and a heightened resistance against infection. Abundant vascularity also allows for faster integration of tissue grafts and implants, which can decrease the risk of skin necrosis.

Nearly all facial cosmetic surgeries involve clean or clean-contaminated wounds, which lack gross contamination and are not associated with the alimentary or respiratory tract. Surgical procedures that do involve mucosal incisions or the use of alloplastic implants may have an increased risk of infection, although rates are still relatively low. Furthermore, the majority of facial cosmetic procedures are performed on healthy patients who typically follow skin care regimens; these habits of frequently cleaning and exfoliating the skin can play a positive role in limiting infection risk.

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Patients with significant medical comorbidities, such as diabetes mellitus, hypertension, coronary artery disease, or chronic obstructive pulmonary disease, are not ideal candidates for cosmetic procedures. For example, in hypertensive patients, hematoma formation is more common, which in turn can lead to tissue necrosis and infection. Diabetes mellitus is associated with delayed wound healing and elevated rates of infection.

As facial cosmetic surgery is elective in nature, procedures should be deferred until a patient has been optimized. Obese patients are encouraged to lose weight before surgery in order to lower the risk of infection and tissue necrosis. Smoking remains a major risk factor for postoperative infection, and complete cessation of tobacco is advised. At a minimum, patients should abstain from smoking at least 3 weeks prior to planned surgery, and cessation should continue until adequate healing of the surgical wound has been achieved. Combined, these factors allow facial aesthetic surgeries to have low risks of postoperative infection [1–4].

In addition to careful case selection, meticulous surgical technique is paramount to infection control. The skin should be appropriately washed and prepared, and attention should be paid to sterile procedures. Delicate surgical technique can reduce the incidence of hematoma, wound tension, and skin necrosis, all of which provide a nidus for bacterial growth and ultimately lead to primary or secondary wound infection.

The indications for prophylactic antibiotics in facial cosmetic surgery are controversial. Nevertheless, the use of short-term prophylactic antibiotics remains a popular practice among surgeons, as the consequences of postoperative infection can lead to scar formation or disfigurement. Skin resurfacing generates a large area of deepithelialized wound, which requires careful attention to wound care. Herpetic infections are particularly feared complications following skin resurfacing due to the widespread area of involvement. Therefore, anti-herpetic prophylaxis is often utilized, especially in procedures involving manipulation of the bone or cartilage or with the use of alloplastic implants or packing [1, 5, 6].

It is important to identify postoperative infections early and rule out other possible diagnoses, such as hypersensitivity reactions. When a postoperative infection is encountered, treatment should begin immediately and aggressively using broad-spectrum antibiotics. Culture and sensitivity tests should be utilized to tailor antibiotic therapy to the identified organism. Surgical management, such as incision and drainage, hematoma evacuation, or debridement of necrotic tissue, may be indicated. Once treatment has begun, frequent follow-up appointments are recommended in order to monitor the progress of the infection. Consultation with an infectious disease specialist should be considered when an infection does not respond to routine treatment modalities. Given the rising incidence of methicillin-resistant *Staphylococcus aureus* (MRSA) and atypical mycobacterial infections following facial aesthetic surgery, early recognition and prompt consultation with an infectious disease specialist can be crucial [7–9].

In treating postoperative infections, meticulous clinical evaluation and frequent follow-up appointments are required. Once a complication has been diagnosed, the patient must be honestly informed of the problem and a detailed treatment plan formulated. The possibility of late revision surgery should also be considered and discussed with the patient.

As patients who undergo aesthetic procedures are acutely concerned with their appearance, complications can be associated with severe anxiety. Patients may demand an immediate solution to the problem. However, emotional distress from the patient should not alter the surgeon's clinical judgment or treatment plan. When the clinician maintains integrity and professionalism, patients are more likely to be satisfied and less likely to seek legal action [1].

8.2 Skin Resurfacing

Dermabrasion, chemical peels, and laser therapy are the most frequently utilized techniques for skin resurfacing. Skin resurfacing is designed to generate a large area of deepithelialized surgical

wound in order to induce skin regeneration. As a result, postoperative complications of skin resurfacing are managed in a similar manner, regardless of the technique used. Although antibiotic prophylaxis for a routine procedure on the skin surface has limited support in the literature, the use of perioperative antibiotics is widely practiced among surgeons.

Perioperative infection associated with cutaneous resurfacing may be bacterial, viral, or fungal in origin [10–12]. Bacterial etiologies are most commonly *Staphylococcus aureus*, *Streptococcus spp.*, and *Pseudomonas spp.* (Fig. 8.1). Cephalexin (500 mg four times per day) or ciprofloxacin (500 mg twice daily) are frequently prescribed as antibacterial agents. For skin resurfacing, the typical antibacterial regimen involves dosing for 1 day preoperatively and 7 days postoperatively. In the case of MRSA-positive patients, doxycycline (100 mg twice daily) or trimethoprim/sulfamethoxazole (160 mg/800 mg twice daily) can be used, and oral rifampin or topical mupirocin may be added in conjunction with this regimen. Although rare, an increased incidence of infection has been associated with nontuberculous or atypical mycobacteria after resurfacing, and treatment may involve long-term multidrug therapy [1, 11–16].

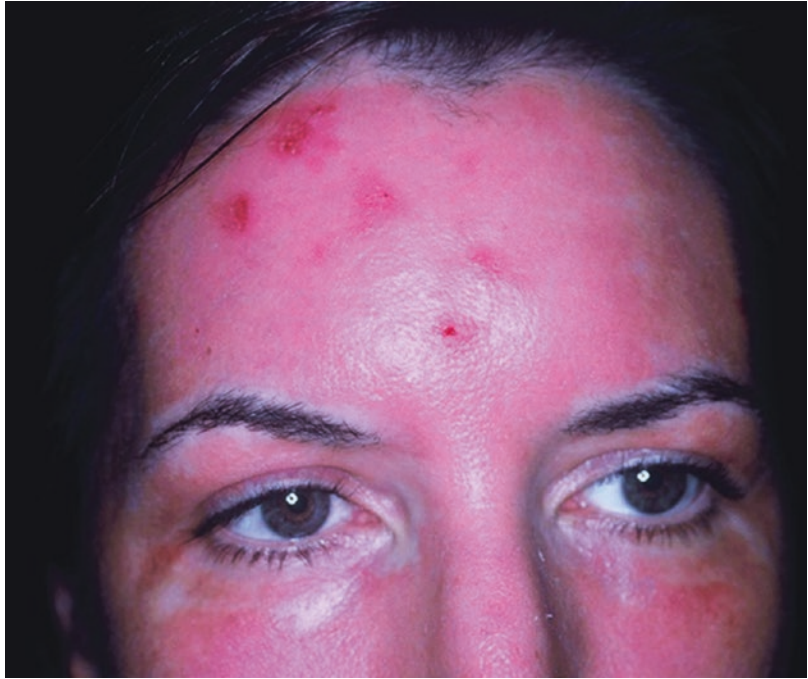
Candida spp. are the most frequent cause of fungal infections after skin resurfacing. Occlusive dressings are commonly used postoperatively to facilitate the healing of deepithelialized skin; however, these dressings may serve as a medium for bacterial and fungal growth. Therefore, when an occlusive dressing is used for more than 48 h, antifungal prophylaxis, such as fluconazole (100 mg daily) for 1 day preoperatively and 7 days postoperatively, is indicated [1, 11, 17].

Among viral infections, herpes simplex virus (HSV) is one of the most concerning complications in post-resurfacing patients, as it can result in significant and permanent scarring. Because of the deepithelialization process in skin resurfacing, the vesicles of herpetic infection may not be seen (Fig. 8.2), and the patient may present with only pain and erythematous erosions. Without prophylaxis, HSV infection following skin resurfacing has been reported in 1.7% of all cases and in 4.6–9.0% of patients with a history of perioral herpetic outbreaks. All patients, especially those with a history of oral-labial HSV infection, should be given an antiviral regimen. Effective anti-herpetic regimens include acyclovir (400 mg five times daily), valacyclovir (1000 mg twice daily), and famciclovir (500 mg twice daily). Dosing begins 1 day preoperatively and is



Fig. 8.1 (a) Culture-proven *Pseudomonas* infection after skin resurfacing. (b) Source of the infection—*Pseudomonas* paronychia (Courtesy of Dr. William M. Ramsdell)

Fig. 8.2 Culture-positive herpes simplex infection in a patient after skin resurfacing. Typical herpetic vesicles may not be seen due to deepithelialization (Courtesy of Dr. William M. Ramsdell)



continued 7–14 days postoperatively, until reepithelialization has been achieved [1, 11, 18, 19].

The presence of facial warts should be identified preoperatively, as infection may be exacerbated through a deepithelialized skin surface. Furthermore, spread of the virus to the operator can occur when laser resurfacing is performed on facial warts. Exacerbated facial warts that occur after skin resurfacing should be treated with topical retinoic acid for 4–6 weeks [20, 21].

Infections in post-resurfacing patients may have atypical presentations due to deepithelialization, and it is often difficult to clinically identify an isolated cause. Therefore, when a post-resurfacing patient presents with signs and symptoms of infection, the patient should be treated aggressively for all bacterial, viral, and fungal origins.

8.3 Soft Tissue Fillers

Infections from soft tissue fillers present as one or more nodules that are tender, erythematous, or fluctuant. All injectable soft tissue fillers are, in essence, implanted foreign bodies. Postinjection

nodules are categorized as either inflammatory or non-inflammatory. Inflammatory nodules can then be further classified by their origins, either infectious or immune mediated. The time of onset and clinical examination are helpful in forming the diagnosis (Fig. 8.3) [1, 22, 23].

Immune-mediated inflammatory nodules are observed with various types of injectable fillers and are likely secondary to trace amounts of proteins in the filler material. These inflammatory nodules are the product of granulomatous inflammation surrounding implanted filler particles, commonly referred to as “sterile abscesses.” Consequently, the nodules appear as a late complication (weeks to several years post-procedure) and frequently present with tenderness and erythema without fluctuance. Treatment centers on oral antibiotics, although some authors advocate the use of high-dose corticosteroids, either systemic (prednisone 60 mg daily) or locally (injection of triamcinolone 40 mg/mL) [22–27].

Infectious inflammatory nodules present as erythematous and tender nodules that may occur anywhere from days to years after injection. The hypothesis behind this variable onset is that a low-grade infection from normal skin

Fig. 8.3 Swelling of the lips immediately after HA injection with needles (Courtesy of Dr. Amit Luthra)



flora, such as *Staphylococcus epidermidis* or *Propionibacterium acnes*, forms a biofilm around the injected filler particles. Therefore, a patient should be started on broad-spectrum antibiotics, such as tetracycline or macrolides. If there is an abscess or no response to antibiotic therapy within 48 h, incision and drainage, culture, and biopsy (2 mm dermal punch) are indicated to tailor the antibiotic treatment. Abscess formation is more common when injectable fillers are used in patients with HIV-associated facial lipoatrophy [22, 23, 26, 28].

Non-inflammatory nodules occur secondary to the localized accumulation of implanted filler material, likely from inappropriate filler placement. These nodules are commonly seen with collagen or hyaluronic acid fillers. Typically, non-inflammatory nodules occur immediately after the implantation of fillers and lack associated pain, erythema, or tenderness. As these nodules are noninfectious, there is no role for surgical or antimicrobial treatment, and conservative management is instead indicated [1, 22, 23].

Fillers represent foreign bodies, and therefore care should be taken to avoid injecting soft tissue fillers in close proximity to infected areas. This caution is especially relevant in a patient

with active perioral herpetic lesions. Because there is no evidence in the literature that fillers trigger herpetic reactivation, prophylactic anti-herpetic medications are not indicated in all patients with a history of perioral herpetic lesions. However, anti-herpetic prophylaxis is beneficial for patients with a prior history of herpetic reactivation following filler injection (Fig. 8.4) [26, 27].

8.4 Rhinoplasty

Infection after rhinoplasty is a rare event. However, the complication requires immediate attention, as the severity of infection may range from localized erythema to bacteremia, with the latter able to cause toxic shock syndrome, cavernous sinus thrombosis, or even brain abscesses. Rhinoplasty is considered a clean-contaminated procedure. Preoperative antibiotic use has been advocated in the following situations: patients with immunosuppression, those at high risk for infectious endocarditis, cases of complex nasal surgery with the presence of hematoma, the use of implants or grafts, or the extended use of nasal packing beyond 24 h [1, 29–32].

Fig. 8.4 Herpetic reactivation after fillers (Courtesy of Dr. Amit Luthra)



Postoperative infection following rhinoplasty is most frequently minor, and the patient typically presents with localized erythema or cellulitis. After ruling out an allergic reaction, antibiotics and intranasal bacitracin should be initiated. In the case of an abscess, which can present up to several years after as a suture abscess, incision and drainage is indicated [30, 32].

Some rhinoplasty candidates with aesthetic complaints may also have functional problems, such as allergic rhinitis or chronic rhinosinusitis. Performing rhinoplasty in these patients can worsen rhinosinusitis due to changes in the ostio-meatal complex. Therefore, it is important to form a comprehensive treatment plan with anti-inflammatory medications, functional endoscopic sinus surgery, and concurrent rhinoplasty [1, 33, 34].

Toxic shock syndrome (TSS), most commonly observed with the use of tampons, is also associated with nasal packing in rhinoplasty. Although extremely rare following rhinoplasty, TSS may present as a life-threatening condition that carries a high mortality rate. TSS occurs when *Staphylococcus aureus* bacteria release toxic shock syndrome toxin 1 into the bloodstream of a susceptible and previously colonized individual.

The complication presents early, during postoperative days 3–5, with fever, nausea, vomiting, diarrhea, hypotension, and erythema, which quickly lead to multi-organ failure. TSS requires aggressive hemodynamic support in an intensive care unit setting, along with immediate removal of the nasal packing/splint, systemic antibiotics, and infectious disease consultation [1, 34, 35].

Manipulation of the nasal septum or median osteotomy during septorhinoplasty can cause a traumatic fracture of the cribriform plate or a dural tear, which may lead to cerebrospinal fluid (CSF) leak and intracranial complications. Although infrequent, life-threatening conditions do result, such as cavernous sinus thrombosis, meningitis, subdural empyema, brain abscess, subarachnoid hemorrhage, or pneumocephalus. When complications are identified, broad-spectrum antibiotic coverage and a multidisciplinary approach with neurosurgery are recommended [36–38].

Reconstruction of nasal structure requires meticulous surgical skills to achieve both adequate airway potency as well as an aesthetically pleasing outcome. Although autologous grafts provide for lower infection rates, alloplastic augmentation of the nasal architecture has gained

popularity due to its simplicity, shorter operative time, and lack of donor-site morbidity. As with any other implant material, nasal alloplastic implants are prone to infection and extrusion.

The most commonly used alloplastic materials are silicone polymers, expanded polytetrafluoroethylene (Gore-Tex®), and porous high-density polyethylene (Medpor®). Silicone rubber, or Silastic®, is a relatively inert material that is nonporous and does not allow ingrowth of tissue or neovascularization. However, chronic inflammation surrounding the silicone implant may cause fibrous capsule formation and bacterial growth. Expanded polytetrafluoroethylene (Gore-Tex®) has pore sizes ranging from 10 to 30 µm and has limited tissue ingrowth due to its small pore size. Early animal studies showed excellent anchoring of the implant, as well as low rates of infection and inflammation. Porous high-density polyethylene (Medpor®) has much larger pore sizes, between 100 and 300 µm, which allows rapid tissue integration and vascularization. This factor, in turn, provides an increased resistance to extrusion and superior host immune response to potential infection. Overall, the rate of infection among different alloplastic implants has been shown to be similar, with the rate depending on factors such as implant location and surgical technique. As discussed earlier, regardless of the type of implant material, all implants are predisposed to infection. Thus, strict sterile technique and minimal handling of the implant should be implemented in order to prevent bacterial inoculation during the procedure. The use of an antibiotic implant-soaking solution (bacitracin, 50,000 units in 1 L of normal saline) can further reduce the risk of implant-related infection [1, 39–41].

8.5 Facelift and Forehead Lift

Facelift surgery, or rhytidectomy, has become one of the standard treatments for the aging face. According to the national database of the American Society for Aesthetic Plastic Surgeons, the number of rhytidectomy procedures increased by 27.7% between 1997 and 2014. Owing to excellent blood supply in the facial region, the

incidence of infection in post-rhytidectomy patients is extremely low, ranging from 0.3% to 1%. Risk factors include male gender, obesity, smoking, hypertension, and diabetes. Although the overall infection rate is low, the consequence can be potentially severe, and appropriate preventative measures should be applied at all levels of patient care. Preoperative scrubbing and surgical site preparation with chlorhexidine gluconate are standard antiseptic measures.

The effectiveness of perioperative antibiotics has conflicting evidence in the literature. Still, the use of antibiotics remains a popular practice among surgeons. A survey from the American Society of Plastic and Reconstructive Surgeons showed that 72% of surgeons use perioperative antibiotics, most commonly less experienced surgeons. When used, it is recommended that preoperative antibiotics be administered within 60 minutes of surgical incision (within 120 minutes for vancomycin and fluoroquinolone) and discontinued within 24 h of procedure completion. Infection typically occurs in the first postoperative week, and therefore a follow-up appointment within this time period is recommended. In addition, patients should be educated to recognize the early signs and symptoms of infection in order to facilitate timely diagnosis. In general, the initial presentation of post-rhytidectomy infection is localized erythema, which is later accompanied by an increase in tenderness. Once infection has been diagnosed, aggressive treatment should be undertaken with empiric antibiotics and incision and drainage of any abscesses. When possible, culture and sensitivity tests should be performed to identify infectious agents and tailor antibiotic therapy accordingly [42–44].

The most common pathogens in post-rhytidectomy infections are *Staphylococcus spp.* and *Streptococcus spp.* Infections caused by these bacteria are easily treated with empiric antibiotic therapy. However, MRSA is now the leading cause of skin and soft tissue infections, including surgical site infections (SSI). Consequently, any SSI in a post-rhytidectomy patient should raise the suspicion for MRSA infection, especially when it is nonresponsive to empiric antibiotic

treatment. Surgical drainage of the MRSA-infected collection is the single most effective treatment modality; however, incision and drainage is occasionally not performed in a timely manner due to the surgeon's fear of a poor cosmetic outcome. Nevertheless, uncontrolled MRSA infections can rapidly spread along the surgical tissue plane, and therefore delaying surgical intervention can lead to even worse cosmetic results, such as skin loss and scarring. Common choices for antibiotic therapy in MRSA infection include vancomycin, linezolid, daptomycin, tigecyclin, trimethoprim-sulfamethoxazole, minocycline, clindamycin, fluoroquinolone, or rifampin. However, considering the diverse resistance profile of MRSA, susceptibility testing is strongly advised in order to direct antibiotic treatment. Atypical mycobacteria infection commonly presents as a chronic, persistent, low-grade granulomatous infection along the incision sites. Atypical mycobacterial infection may lead to scar formation, even after resolution of the infection, and thus early recognition, prompt treatment, and consultation with infectious disease specialists are recommended [7, 42–44].

8.6 Blepharoplasty

Given the robust vascularization of the eyelids, postoperative infection after blepharoplasty is rarely encountered. The incidence has been reported at 0.2% without concurrent laser resurfacing and 0.4% with concurrent laser resurfacing. The majority of post-blepharoplasty infections are transient and easily treatable; however, infection of the eyelid has the potential for serious complications, such as cavernous sinus thrombosis or permanent vision loss. Cellulitis in the periorbital region is divided into two groups: preseptal cellulitis, which is confined to the soft tissue of the eyelid, and postseptal (orbital) cellulitis, where the infection has spread to the retroseptal space and orbit. Preseptal cellulitis presents with erythema, induration, and edema of the eyelid but without

abnormalities in vision, pupillary reaction, or ocular motor function. Preseptal cellulitis is effectively treated with a third-generation cephalosporin or fluoroquinolone and can be managed as an outpatient. However, if no improvement with empiric antibiotic treatment is noted within 48 h, hospitalization for close observation and intravenous antibiotics should be considered.

In contrast to preseptal cellulitis, postseptal cellulitis presents with severe pain, proptosis, chemosis, ophthalmoplegia, an afferent pupillary defect, and a decrease in visual acuity. Postseptal cellulitis is best managed in an inpatient setting due to these possible outcomes. Intravenous antibiotic treatment should begin promptly, along with contrast-enhanced computed tomography to assess the extent of the infection, rule out cavernous sinus thrombosis, and identify any abscesses. An expanding abscess in the closed orbital space may behave similarly to a retrobulbar hemorrhage, in which the expanding abscess causes compression of central retinal artery and optic nerve, subsequently leading to permanent vision loss. Therefore, when worsening visual impairment is noted in the presence of an orbital abscess, surgical evacuation is indicated. In the setting of rapidly worsening visual acuity and increased intraocular pressure, emergent bedside lateral canthotomy or cantholysis is indicated to prevent permanent vision loss. Mannitol, dexamethasone, or timolol ophthalmic solution can be used as adjunctive pharmacological agents to lower intraocular pressure. An ophthalmology consult should follow after surgical intervention to treat abscesses in the orbital space [45, 46].

The most common pathogenic organisms are skin flora, *Staphylococcus spp.*, and *Streptococcus spp.* The incidence of MRSA is rising in post-blepharoplasty infections, and there should be a high index of suspicion when an infection is non-responsive to empiric antibiotic therapy. Furthermore, rare cases of atypical mycobacterial infection and necrotizing fasciitis have been reported, and an infectious disease specialist should be involved in the treatment of these rare cases [45–49].

8.7 Otoplasty

Postoperative infection following otoplasty is generally prevented by the high vascularity in the auricular structures, similar to other structures in the head and neck region. The incidence of infection lies between 2.4 and 5.2%. An infection may arise in the setting of hematoma or wound dehiscence, but improper sterile technique can also contribute. Sterile preparation should include the external ear, the hair surrounding the ear, and the external auditory meatus. The external auditory meatus should be protected using a cotton ball covered with ointment, and antibacterial ointment can be used over the closed surgical incision. Postoperative infection most commonly results from *Staphylococcus spp.* and *Streptococcus spp.* and less frequently from *Escherichia coli* and *Pseudomonas aeruginosa*. When perioperative antibiotic use is desired, antipseudomonal coverage should be included. Infections typically present 3–5 days postoperatively with visible erythema, edema, discharge, and clinically disproportional pain. Failure to aggressively treat infections can lead to perichondritis and permanent deformity of the ear. Therefore, empiric antibiotic therapy should be initiated, and a wound culture and sensitivity test should be performed to guide further treatment. If purulent discharge is noted, the surgical site should be appropriately drained and irrigated, and any necrotic skin or cartilage should be carefully debrided. Skin or cartilage necrosis, which can become a nidus for bacterial growth, is frequently caused by an expanding hematoma or overly tight flap closure [50–52].

Careful patient selection may also reduce the incidence of post-otoplasty infection. Otitis externa should be fully treated prior to otoplasty. Patients with postauricular eczema have an increased risk of infection after otoplasty, as eczematous skin has a higher rate of *Staphylococcus aureus* colonization. Therefore, surgery should be postponed until the eczema is cleared [52].

8.8 Facial Implants

Facial contour is a delicate balance between bony structures, adipose tissue, and facial musculature. Historically, volume defects were inadequately augmented by only addressing the soft tissues. Recent advancements in bony augmentation techniques have revolutionized the field of facial cosmetic surgery and facial rejuvenation. Techniques for facial augmentation range from the correction of large craniofacial defects to minor cosmetic imperfections. Although autologous grafts have several advantages, such as lower rates of infection and inflammation, their use has been declining due to donor site morbidity, longer operative and recovery time, limited volume, and unpredictable resorption. Alloplastic facial implants have become widely popular and are currently used in the augmentation of facial defects in various regions, such as the internal orbit, infraorbital rim, malar region, nasal/paranasal region, mandible, chin, and frontal and temporal areas. However, the use of alloplastic implants raises concern due to the risk of implant infection and subsequent implant extrusion. Implant infection and extrusion rates vary depending upon the type of implant, location of augmentation, and surgical approach. There are conflicting reports regarding the rate of infection among different implant materials, and no implant material has been shown to be significantly superior to others. A recent study reported the overall infection and extrusion rates for all alloplastic facial implants at 0.8% and 0.4%, respectively.

The most common types of implant materials are silicone polymers, expanded polytetrafluoroethylene (Gore-Tex[®]), porous high-density polyethylene (Medpor[®]), and non-resorbable polyester fiber (Mersilene[®]). Infections from porous implants, such as Gore-Tex[®] or Medpor[®], tend to present as early complications; in contrast, infections from smooth surface implants, such as silicone, tend to manifest as late and chronic complications. Porous implant materials allow for vascularized tissue ingrowth, which in

turn provides an avenue for host immunity and resistance to infection. However, porous materials are more prone to biofilm formation, and due to tissue ingrowth, the removal of an infected implant becomes challenging [53–56].

To date, there have been no controlled studies evaluating the efficacy of preventative modalities for alloplastic infection. However, several preventative measures are popularly practiced among surgeons, largely focused on maintaining implant and wound sterility. Intravenous preoperative and/or intraoperative antibiotics, such as cefazolin or clindamycin, are administered at least 30 min prior to surgical incision. Intraoperatively, strict sterile technique and minimal manipulation of the implant material should be followed. In addition, bathing the implant in antibiotic solution (bacitracin 50,000 U/I saline solution) or irrigation of the implant pocket with antibiotic solution (clindamycin 300 mg in 30 mL of sterile water) can be performed. A transoral surgical approach may be avoided to further reduce the risk of infection; when a transoral approach is used, the incision should be closed with water-tight closure. Lastly, a short course of postoperative oral antibiotics can be used to further reduce the risk of infection. In

the case of infection, broad-spectrum antibiotics should be started promptly and later switched to culture-appropriate agents. Treatment should be followed by the removal of infected implants and irrigation of the area. Implant reinsertion should be planned at least 6–8 weeks after the resolution of an infection [55, 56].

8.9 Hair Transplantation

Hair loss affects approximately 1.2 billion people around the world, and hair transplant surgery (HTS) has rapidly gained popularity over the past decade. Due to its excellent vascularization, the scalp is fairly resistant to postoperative infection, with rates reported at less than 1%. Although most infections are minor and localized, more severe cases have been reported, such as osteomyelitis of the calvarium or herpes zoster in head and neck area. *Staphylococcus spp.* are the most common infectious organism; however, gram-negative bacteria, fungal organisms, and even atypical mycobacterium have been reported. Although rare, there is an increased incidence of MRSA infection following HTS (Fig. 8.5) [57–60].

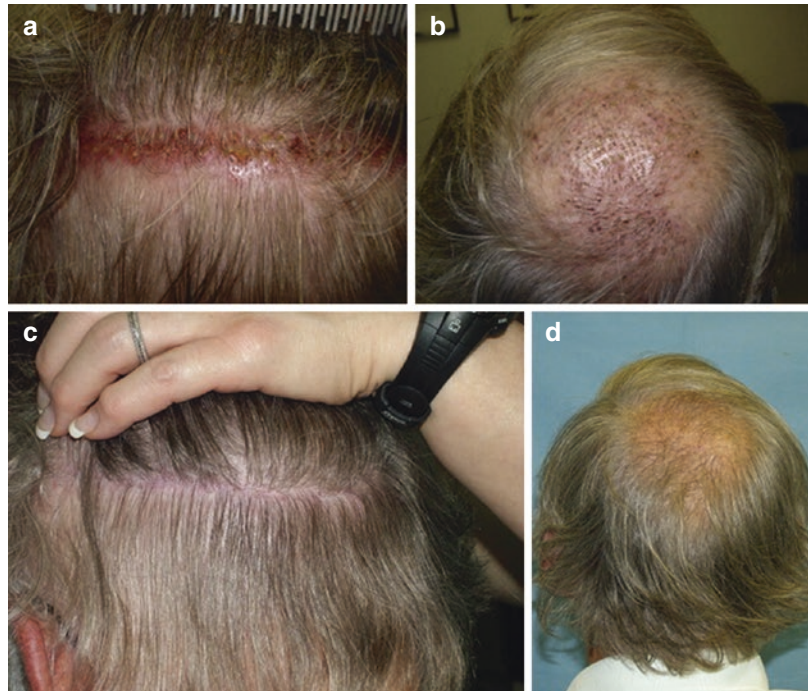


Fig. 8.5 Post-op day 7 from hair transplant surgery. Pustules and redness in the donor area (a) and in the recipient area (b), both culture-positive for methicillin-resistant *Staphylococcus aureus* (MRSA). (c) At 8 months post-op: the donor site (c) and the recipient site (d) healed without complications (Courtesy of Dr. David Perez-Meza)

Fig. 8.6 Post-op day 9 following hair transplant surgery. Post-op infection noted in the right recipient area (a) and left recipient area (b). Twelve months post-op: both areas (c and d) healed without complication (Courtesy of Dr. David Perez-Meza)



Postoperative infection may occur at both donor and recipient sites, often in relation to pre-existing medical comorbidities, poor wound hygiene, or excessive crust formation (Fig. 8.6). Donor-site infection is commonly due to circulatory compromise from high-tension closure, generally manifesting as excessive crust formation and inflammation along the suture or staple line. Recipient-site infection typically presents as localized papulopustules. Minor infections in both sites are easily treated with suture/staple removal, wound hygiene and crust removal, and topical antibiotic ointment application. Occasionally, a patient may present with an abscess and associated erythema/tenderness. The abscess must be appropriately drained and cultured, and broad-spectrum antibiotics instituted until sensitivity results are obtained. Wet-to-dry dressings are applied to the open wound, and

once the wound is completely healed by secondary intention, scar revision can be discussed with the patient as needed [57].

Conclusion

Postoperative infection in facial cosmetic surgery is rarely encountered due to robust vascularization of the head and neck region, low rates of surgical site contamination, and patient-selection factors. Although the risk of infection is relatively low, the consequence can be devastating to the patient, both clinically and emotionally. As with any other type of infection, prevention is the primary goal. It involves the utmost attention to sterile preparation, meticulous surgical technique, and adequate medical optimization of patients with an elevated risk of infection. Although perioperative and prophylactic antibiotic use

has conflicting investigational support, it is popularly practiced among surgeons. Antibiotic coverage is strongly recommended in procedures involving grafts, implants, packing, or manipulation of the bone or cartilage. A surgeon should be able to recognize the early signs of infection and differentiate them from other causes of inflammatory response. Once encountered, the treatment for infection begins promptly with broad-spectrum antibiotic coverage and frequent follow-up visits. When indicated, surgical intervention with debridement, implant/packing removal, incision and drainage, and wound culture should be performed. Atypical, complex, or chronic infections may require a multidisciplinary approach with an infectious disease specialist. Lastly, a surgeon should be mindful of the emotional challenges experienced by the patient and remain truthful and empathetic throughout the course of treatment.

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Managing Medicolegal Issues Surrounding Esthetic Facial Surgery

9

Christy B. Durant and James R. Hupp

Abstract

With the increased pressures in striving for perfection, beauty, and youth in today's society, the use of cosmetic surgery to achieve such goals is on the rise. People are turning to dermal fillers, lasers, and chemical peels, and even undergoing more invasive surgical procedures to better one's appearance and rewind the hands of time with the goal of creating a more youthful-looking face. Although cosmetic maxillofacial surgery may be an area of healthcare gaining increased popularity among both patients and practitioners, it nonetheless, carries with it the same medicolegal issues and considerations that come with any surgical procedures. In fact, it could be said that with patients undergoing cosmetic procedures, the risks for postsurgical legal ramifications could be greater than many other elective or medically necessary surgical interventions in light of the psychological component that leads many patients to seek treatment, as well as the patient's subjective perception of the success of the outcome. For most patients, undergoing esthetic procedures is the overt desire to change their physical appearance or function. However, patients often have unspoken desires, which may include improving self-esteem and overall quality of life (Sykes, *JAMA Facial Plast Surg* 15:81, 2013). Although unstated, these desires to improve overall quality of life clearly affect an individual patient's eventual satisfaction with his or her surgical outcome (Sykes, *JAMA Facial Plast Surg* 15:81, 2013). Given these factors in order to decrease the likelihood of litigation or legal repercussions, it is imperative

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for the operating surgeon to evaluate and consider each case individually in conjunction with the needs and desires of the patient. It is only with a well-documented medical record containing all relevant information, a written informed consent from the patient, and a strong doctor/patient relationship built on trust and open communication that a provider will be able to successfully defend him or herself in the unfortunate situation of a legal event.

9.1 Establishing the Doctor/Patient Relationship

With the upward trend of patients undergoing cosmetic procedures, naturally there is an increase in dissatisfied patients and obliging attorneys targeting doctors [1]. As previously stated, in today's society, cosmetic procedures are sought by patients for a number of reasons, most significantly to better one's appearance and strive for perfection. Whether compelled to have such procedures as a result of their profession or merely for personal satisfaction, cosmetic procedures are deemed "need felt." It is an innate desire to "look good" that drives the patient to seek such esthetic procedures [1]. Be that as it may, surgeons should never feel overly enthusiastic about the performance of cosmetic procedures and need not succumb to the needs of an insistent and highly demanding patient. Just like any other medical procedure, it is always preferable to err on the side of safety rather than being too accommodative. Doctors should continually be cognizant that while patient safety is always of the utmost importance, the doctor's interest should also be safeguarded as the intention is not to harm the patient.

Establishing a healthy doctor/patient relationship is the first step necessary to achieving a successful outcome for both provider and patient. This relationship is a unique one, as it is the foundation upon which all further communications are based. Just as a doctor begins assessing a patient from the moment they enter a consultation room, so too is the patient assessing a potential doctor to determine if they want this individual as a surgeon. From appearance, tone of voice, empathy, and perceived competence in the subject matter, a patient is continuously forming an impression of their potential healthcare provider. A doctor's rap-

port with a patient is an integral element in not only establishing trust and confidence in a patient but also instilling a level of comfort in the patient that can directly influence a patient's willingness and ability to effectively and adequately communicate with their doctor. The more at ease a patient feels in interacting with a clinician, the more open the lines of communication will be between the parties. Maintaining open communication can only serve to benefit the doctor and the patient both from a liability standpoint and a quality of care measure. In fact, research has found that patients who have a good doctor/patient relationship with their provider, the greater the likelihood the patient will make full disclosures regarding prior medical history, including a sense of comfort in discussing their most personal of medical matters, as well as their true motivations and expectations from a procedure. An open discussion of a patient's motivation and expectations allows a doctor to not only ascertain whether or not the patient's goals are realistic but also allows the doctor to fully evaluate a patient's psychological profile and candidacy as a cosmetic surgical patient.

One of the key elements in creating a successful doctor/patient relationship with a patient is to always exercise transparency. Transparency by both a patient and a doctor directly impacts patient safety and provides a doctor with essential information necessary to safely and effectively perform surgery. As a doctor, effective communication is the principal way to establish trust with a patient. This is achieved by formulating and maintaining clear expectations of the procedure as well as the patient's anticipated outcome. It is important for a doctor to remember that, unlike non-cosmetic surgery patients who hope a surgeon will recommend not having surgery, patients seeking cosmetic pro-

cedures desire change and usually request surgery. For this reason, doctors must clearly articulate to the patient a detailed description of the requested procedure in addition to factors that may predispose the patient toward post-procedure dissatisfaction with the cosmetic result [1].

Separate and apart from the numerous reasons why having a strong doctor/patient relationship is integral to a successful outcome, from a legal standpoint, the formation of a doctor/patient relationship is the first element necessary that establishes liability. Disappointments in the doctor/patient relationship can contribute to a negative spiraling of confidence for all involved and have consequences for both the surgeon and the patient. The doctor/patient relationship is a fiduciary one,¹ in which establishing and maintaining trust and confidence of the patient, as well as maintaining fidelity to the patient when confronted with conflicting interests, is absolutely essential. From that point forward, the doctor not only assumes the care and treatment of a patient from a practical standpoint, but it also creates a legal obligation on behalf of the doctor commonly known as a duty of care. More specifically, once a doctor/patient relationship is established by, in this case, the surgeon agreeing to care for the patient, a doctor has the duty to possess the medical skill and knowledge required of a reasonably² competent doctor practicing in the same field or specialty. The doctor further carries a duty to exercise reasonable care, skill, and diligence as doctors and surgeons in a similar field ordinarily exercise. The failure to meet these duties is a setup for malpractice. Thus, it is incumbent upon the surgeon to determine whether they have the needed training and experience to manage the patient's problem. Although

proof of completion of a residency in a specialty that offers training in facial esthetic surgery may be considered sufficient during a lawsuit, the surgeon has an ethical obligation to ensure that they are capable of skillfully executing the planned surgery. In addition, a skilled plaintiff's attorney may probe deeper into the defendant doctor's training when trying to determine if the surgeon was sufficiently prepared to manage the patient's condition. The specific elements of negligence in the unfortunate event of a malpractice action are further discussed later in this chapter.

While there may be many reasons for filing a claim, legal precedent has shown that dissatisfaction with some aspect of communication with their doctor is a strong underlying current to many claims. The most frequent grievances expressed by the patients are that no one involved in their care fully explained the potential for adverse effects, that the doctor misled them, that the doctor did not talk to them or answer questions, and that the doctor would not listen to their concerns [2]. Again, utilizing effective communication during the doctor/patient relationship is an easy way to avoid falling into this trap.

9.1.1 Elective Versus Medically Necessary Surgery

The biggest challenge in winning a cosmetic surgery malpractice case comes down to the fact that the procedure is elective and not usually considered medically necessary [3]. Almost all cosmetic procedures are elective. Therefore, a patient must understand the nature of the treatment/procedure being recommended and the distinction between elective and medically necessary. A detailed discussion about a procedure goes a long way in winning a patient's confidence as well as giving them the comfort of knowing the nature of a procedure beforehand. Elective surgery is a planned, non-emergency surgical procedure. Such procedures may be either medically required (i.e., cataract surgery) or optional (i.e., breast implant) surgery. Just because certain surgeries are considered elective, they may still serve to extend life or improve the quality of life

¹A fiduciary relationship is where one person places complete confidence in another with regard to a specific transaction. The relationship is not necessarily formally established but can be one ethical responsibility, due to the superior knowledge and training of the fiduciary as compared to the other party to the relationship.

²Note that when the words "reasonable" and "reasonably" are used in legal discussions, the eventual determination of what is reasonable will be left to the jury to decide in a jury trial.

physically and/or psychologically. Cosmetic and reconstructive procedures, such as a facelift, tummy tuck, or nose job, may not be medically indicated, but they may benefit the patient in terms of raising self-esteem. Other procedures may improve quality of life even though they are technically an “optional” or elective surgery. There are also those elective surgeries that are necessary to prolong life, such as an angioplasty; however, they are unlike emergency surgeries which must be performed immediately. Required elective procedures can be scheduled at a patient’s and surgeon’s convenience. Each case is different. Regardless of the reason a patient is undergoing a cosmetic procedure, whether medically indicated or optional, the patient must be advised of all aspects of the type of procedure recommended for them. Confusion by a patient as to the difference between those procedures that are purely optional, and usually patient driven, and those procedures that seek to improve functional quality of life can open the door to a liability claim down the road.

During the patient discussion about the type of treatment/procedure recommended, it is important for a doctor to avoid making guarantees regarding an outcome. For obvious reasons, surgeons rarely execute written contracts with patients that purport to guarantee or otherwise ensure certain results from specific medical treatment/procedures [4]. That being said, the use of certain language and even diagrams, drawings, or computer projections can be interpreted from a legal standpoint as an express warranty to a patient. Patients can reasonably rely on such language and illustrations, causing a material departure from the results to potentially give rise to a breach of contract claim by a patient. It goes without saying that in their discussions with patients, surgeons might make verbal representations that minimize certain risks of a procedure or exaggerate the likely benefits; however, particularly in circumstances where there is a significant disparity between the verbal representations of the surgeon and any written disclaimer or consents, judges are inclined to permit the introduction of such discrepancies as evidence in cases of medical negligence to demon-

strate material misrepresentation in the doctor/patient relationship [4]. The critical distinction that courts seek to make is between statements that fall within the category of “therapeutic reassurance” and statements that cross the line into a guarantee for certain results or warranty of a cure. For example, telling a patient “you are in good hands and will be taken care of” is no more than a reassurance to put a patient at ease, even if the outcome is not the one hoped for by with the patient or the doctor. However, telling a patient undergoing a rhinoplasty that they will “never have breathing problems again” can arguably be construed as a warranty of a specific result. There are certainly gray areas in conveying information to patients that fall right on the line of mere reassurance and warranty. Avoiding these gray areas is just another element in practicing effective communication with a patient, particularly regarding the nature, risks, and benefits of a procedure [4]. Thus, the surgeon must be particularly careful when using a computer-generated projection of the surgical results to clearly explain that the patient’s final appearance after surgery may not resemble the computer projection and discuss why.

9.1.2 Medical History

Obtaining an accurate medical history from a patient goes directly to patient safety and obtaining the expected outcome from any procedure. The importance of a good history and thorough clinical examination for every patient cannot be overstressed. It is based on the patient history and examination that the diagnosis is made and the treatment plan is based. An exact, legible record of the original consultation is essential to assess progress following the treatment. Not having all the relevant facts about a patient’s medical history, due to a doctor’s ineffective communication or lack of attention to the importance of certain information, is a direct road to liability. An adequate medical history is the most useful information available to determine whether a patient can safely undergo a planned procedure. It also serves to identify relevant information that may alter the

anticipated outcome of the planned procedure or the anesthetic plans.

Patients should be provided with a concise, clearly written medical history questionnaire for them to complete prior to consultation. This document should be written in a nonscientific language that can be easily understood by the patient. In compliance with the Health Insurance Portability and Accountability Act (HIPAA), the questionnaire should further contain a brief statement informing the patient that all information disclosed on the document will be kept confidential unless the patient specifically authorizes disclosure to a third party. A space should be provided to allow the patient to list any third party to which their confidential information can be disclosed. Lastly, the medical history questionnaire should contain an area for the patient to sign and date, which indicates that they understood the form and acknowledge the accuracy of the answers provided.

In addition to providing a patient medical history questionnaire to be completed by the patient, taking the time to sit with a patient and engage in an open dialogue about the history provided is an integral part of the doctor/patient relationship. The use of effective communication skills and maintaining an open dialogue with a patient serves to illicit additional information from a patient that they perhaps were not even aware would be relevant for the surgeon to know. This includes not only a patient's physical history but also their social history and occupation. During the interview process, the doctor can use information identified on the medical history questionnaire to formulate questions for the patient as a means of acquiring supplementary facts. The initial consultation is the most opportune time to identify potentially problematic patients, both medically and psychologically. This starts with having the patient write their chief reason (chief complaint) that has led them to seek care from the surgeon. Requiring a written statement helps both the patient and doctor begin to ensure that both agree on what concerns the patient. This may help the doctor recognize patients with unfounded views of themselves. Other "red flags" may include a patient giving a past history of

malpractice suits, patients having frequent facial procedures or repeat surgeries on the same part of the face, or patient expressing complaints about prior providers.

Selecting the right patient from the beginning is necessary to obtaining a successful outcome in cosmetic surgery. While surgeons are taught how and when to operate, the skill of knowing when not to operate requires experience and good judgment. It has been said that at the end of their careers, surgeons are rarely disappointed that they chose not to operate on a particular patient; however, operating on the wrong patient (one with a poor medical or psychological profile) often creates anger and frustration for both the patient and the doctor and may result in a claim of negligence by the patient [5]. A valuable communication tool to use with patients, not only in discussing their medical history but really in discussing all aspects of treatment, is reflective listening [5]. This includes repeating or paraphrasing the patient's words back to them. An example of this would be using the phrases "what I hear you saying is..." or "so it sounds like you're saying..." This type of exchange with a patient allows the patient to feel that they are being heard and can alert the doctor to potential misunderstandings. Feeling as if a doctor understands and is listening to what they are saying aids in putting the patient at ease and allows for a more open line of communication, particularly in conveying prior medical history, as well as motive and expectations. The doctor should document in the medical record the content of any discussions had with the patient and questions answered.

9.2 Informed Consent

Giving the patient a thorough explanation of the planned procedure including potential material risks, along with an appropriately written and signed informed consent from a patient, is the cornerstones to a doctor avoiding liability in a medical malpractice action. Providing information about the planned procedure and risks can be supplemented through the use of brochures and/or videos. The word "consent" means to give an

approval, assent or permission, and a voluntary agreement to another proposition [6]. In simple terms, the consent of a patient is an instrument of mutual communication between a doctor and a patient with an expression of authorization/permission by the patient for the doctor to act in a particular way, after achieving an understanding of the relevant medical facts and risks involved. Providing an informed consent to a patient is based upon the principles of autonomy and privacy and has become the requirement at the center of morally valid decision-making in healthcare and research. Patient autonomy means that patients have the right to participate with their doctor in their own healthcare decision-making [6]. Under this ethical principle, they have the freedom to decide what should or should not happen to their own body and to gather information before undergoing a test/procedure/surgery. There are seven (7) general criteria that have been defined for an informed consent to be considered complete: (1) competence of the patient to understand and to decide, (2) voluntary decision-making, (3) disclosure of material information such as risks and alternative forms of treatment, (4) recommendation of the plan, (5) comprehension of terms, (6) decision in favor of the plan, and (7) authorization of the plan [6]. A patient gives informed consent only when all seven (7) criteria are met. In the event that all the criteria are met and a patient rejects the treatment plan, the patient is deemed to have made an informed refusal. Some states may have additional requirements, either by statute or case precedent, including the doctor's obligation to discuss alternative available treatment options with a patient. This is one reason why it is imperative for every practitioner to be familiar with their individual state's requirements. Most states do require, however, that a patient be afforded the opportunity to speak with the doctor about any questions or concerns they may have. It is further necessary that the doctor himself/herself always obtain a patient's valid informed consent prior to a procedure and allocate sufficient time in an adequate environment to discuss the patient's questions and concerns. The presence of a witness is also recommended while taking an informed consent.

9.2.1 Standard for Disclosing Risks, Benefits, and Complications

The rights of patients to be informed about healthcare decisions in clinical practice have periodically come under scrutiny not only in the United States but around the world. The well-ingrained ethical-legal process of informed consent, so fundamental to patient autonomy, has been the subject of many legal cases. Most recently in a 2015 UK Supreme Court case (*Montgomery v Lanarkshire Health Board*), the Supreme Court ruled that the standard for what doctors should inform patients about the risks, benefits, and alternatives of treatment will no longer be determined by what a responsible body of doctors deems important but rather by what a reasonable patient deems important [7]. By virtue of this ruling, the Court embraced a new patient-centered standard. In the United States, more than half of the states have moved to adopt the reasonable-patient standard as well, viewing the informed consent communication process from the patient's perspective. Practicing in a state where this standard has been adopted requires doctors and other healthcare practitioners to disclose all relevant information about the risks, benefits, and alternatives of a proposed treatment that an objective patient would find material in making an intelligent and informed decision about whether to proceed with the proposed treatment.

The mere act of a patient engaging in a consultation with a doctor and expressing his/her medical problem is commonly taken as an implied (or implicit) consent for a general physical examination and routine investigation. However, an intimate physical examination, undergoing invasive tests or undergoing risky procedures, particularly surgery, requires a specific expressed consent [8]. While expressed consent can be oral or written, written consents are always encouraged, especially in cosmetic procedures and surgery, in order to avoid later allegations of denying that consent was given. In order to obtain informed consent from a patient, the doctor must disclose sufficient information to the patient related to the procedure at hand. Claims

based on lack of informed consent are a staple in the medical malpractice arena and can often be avoided. Patients challenge treatment rendered on the grounds that adequate information was not provided to the patient in order to make a proper and knowledgeable decision. Therefore, accurate, adequate, and relevant information must be provided truthfully to a patient in a simply written form using nonscientific terms and language that the average patient can understand. While some providers choose to use a general template informed consent form for all procedures/treatments, an all-encompassing (global) consent is not valid and leads to patient confusion. Doctors are encouraged to use more detailed informed consent forms specific to the type of treatment/procedure being considered so that patients understand the ramifications of the specific treatment they have selected. The doctor should discuss with a patient the advantages, disadvantages, alternatives, possible adverse effects, the generally expected outcomes of the procedure chosen for the patient, and the need to follow instructions before and after the procedure. The use of brochures and/or leaflets providing a patient with comprehensive, objective information for specific procedures is a beneficial way for a doctor to make additional information available to patients that they can take home and further understand the discussed treatment options. It is good practice for the doctor to document in the patient's medical record when such materials are provided to a patient as an added measure to demonstrate that a full and thorough explanation of treatment has been given. This is in addition to the final phase of the informed consent procedure, which is to always document in the patient record when an informed consent was obtained with the written consent form itself included in the chart as well. For offices using electronic record keeping, having patients use an electronic signature pad is another valid means to obtain and document consent.

The advantages of an appropriately signed informed consent are not limited to fulfilling legal obligations. Having a patient who fully understands the nature of their condition and has a practical and sensible expectation of the out-

come is less likely to sue. Furthermore, a well-documented written informed consent is valuable support to defending claims based on misunderstanding or unrealistic expectations of treatment.

9.2.2 HIPAA and HITECH Compliance

The Health Insurance Portability and Accountability Act (HIPAA) was passed by the US Congress in August 1996. It is the first comprehensive federal protection of the privacy of health information. The primary purposes of the act are to improve the efficiency and effectiveness of healthcare delivery by creating a national framework for health privacy protection as well as to protect and enhance the rights of patients by providing them access to their health information, and controlling inappropriate use or disclosure of that information, while helping to improve the quality of healthcare by restoring trust in the healthcare system among all those involved [9]. The core principal of the HIPAA Privacy Rule is the protection, use, and disclosure of protected health information ("PHI"). Protected health information means individually identifiable health information (Fig. 9.1) that is transmitted or maintained by electronic or other media, such as computer storage devices [9]. The privacy rule protects all PHI held or transmitted by a covered entity, which includes healthcare providers, health plans, and healthcare clearinghouses. Use and disclosure are the two fundamental concepts in the HIPAA Privacy Rule. Under HIPAA, "use" limits the sharing of information within a covered entity, while "disclosure" restricts the sharing of information outside the entity holding the information [9]. A written authorization must be obtained from a patient before sharing that information with anyone, unless the disclosure falls into one of the limited exceptions when patient information may be disclosed without authorization (Fig. 9.2). The privacy rule is designed to provide strong privacy protections that do not interfere with patient access to healthcare or the quality of healthcare delivery.

- Name
- Home address
- Phone numbers
- Fax numbers
- Dates (birth, death, admission, discharge, etc.)
- Social Security Number
- E-mail address(es)
- Medical record numbers
- Health plan beneficiary numbers
- Account numbers
- Certificate or license numbers
- Vehicle identifiers and serial numbers, including license plate numbers
- Web Universal Resource Locators (URLs)
- Internet Protocol (IP) address numbers

Fig. 9.1 Individually identifiable health information [9]

- Required by Law
- Public Health Activities
- Victims of Abuse, Neglect or Domestic Violence
- Health Oversight Activities
- Funeral Director/Coroners
- Law enforcement to identify or locate missing person or fugitive; victim of suspected crime; alert of person's death; information is evidence of a crime, or medical emergency
- Correctional Institutions
- Research
- Organ, eye and tissue donation centers
- Serious Threat to Health and Safety
- Essential Government Functions
- Workmen's Compensation
- Judicial/Administrative proceedings at the patient's request or as directed by a subpoena or court order

Fig. 9.2 Permitted disclosures without a written authorization [9]

The first step in informing patients of the HIPAA protections is the communication of patient's rights. All patients must be provided with a written document called the Notice of Privacy Practices which sets forth in plain and simple language how patient's medical information may be used and disclosed as well as how to get access to the information. A copy of the Notice of Privacy Practices should be posted in the doctor's office where it can be easily seen by all patients. An easy way for practitioners to protect themselves and ensure that patients are aware of an office's use and disclosure of protected health information is to have patients sign and acknowledge that they have been informed and provided a copy (or advised where it is available to access) of the Notice of Privacy Practices.

The privacy and protection of patients' identifiable healthcare information have been made a top priority not only by the federal government but by individual states over the last 10 years. It is not only imperative for a healthcare provider to be aware of the HIPAA privacy laws and the ramifications for breach thereof, but administrative and clinical staff must be trained on this subject as well. A doctor is required to take reasonable administrative, physical, and technical safeguards to protect an individual's health information from incidental disclosure to third parties. These safeguards have most recently extended to any and all business associates of a covered entity that may have access to confidential patient information. To reduce a breach of confidentiality within a practice, offices are required to appoint a security officer who is responsible for the doctor's office security and perform a risk analysis to determine information security risks and vulnerabilities. Established policies and procedures must be in place to allow access to protected health information on a need to know basis only. Staff training should be done to ensure awareness about the significance of maintaining patient confidentiality. Finally, an office must have written policies and procedures regarding how to address a HIPAA breach, as well as a disciplinary policy

in the employee manual in the event of an employee's violation of HIPAA.

With the increased use of electronic medical records, doctors must be cognizant of how patient information is stored or delivered by their office. The HIPAA security rule specifies how patient information is protected on computer displays, computer networks, the Internet, disks, and other storage media extranet. The Health Information Technology for Economic and Clinical Health (HITECH) Act, enacted as part of the American Recovery and Reinvestment Act of 2009, was signed into law on February 17, 2009, to promote the adoption and meaningful use of health information technology [10]. Subtitle D of the HITECH Act addresses the privacy and security concerns associated with the electronic transmission of health information, in part, through several provisions that strengthen the civil and criminal enforcement of the HIPAA rules. Offices that utilize electronic medical records should be familiar with requirements set forth in Subtitle D of HITECH which enforces the privacy protections necessary for handling and transmitting electronic medical records.

Recent amendments to the federal privacy laws have also extended liability for HIPAA violations to business associates. Therefore, doctors should ensure that they have an updated business associate agreement with all vendors to their office who may access individual identifiable health information. A provider should also be cognizant of their individual state laws regarding the confidentiality of protected healthcare information. State laws may be more stringent regarding the protection of individual identifiable healthcare information than federal law. When such laws are in conflict, the one which is most strict is the law that must be followed. The importance of showing due attention to the protection of the patient's medical information and applicable confidentiality laws cannot be overstated. A breach of protected healthcare information can result not only in monetary damages to rectify the problem, but in some states patients have been

permitted to file malpractice actions on a HIPAA breach alone. Furthermore, with the Office of Civil Rights under the federal Department of Health and Human Services increasing its audits on healthcare facilities and individual practices, not having the appropriate safeguards, and policies and procedures in place to limit the incidence of a healthcare breach, can result in huge monetary fines.

A patient must have trust in the doctor and his or her practice and know their healthcare information will be kept confidential. Without having these essential components, patients are less likely to have confidence in disclosing important healthcare information to their doctor. Any interruption in the patient's ability to communicate effectively and convey necessary medical information to their doctor can be detrimental to achieving a successful result and can expose a practitioner to liability (Figs. 9.1 and 9.2).

9.3 Medical Record

Medical records are one of the most important aspects on which practically almost every medicolegal battle is won or lost. The key to disposing most medical negligence claims rests with the quality of the medical records. A good medical record serves the interest of the doctor as well as the patient as it is the solitary document that explains all details about the patient's history, clinical findings, diagnostic test results, pre- and post-procedure care, patient's progress, and medications. Medical record keeping has evolved into a science and takes a conscious effort by a doctor to master over time. Record maintenance is often the only way for the doctor to prove that the treatment was carried out properly [11]. By the time a medical negligence claim comes to fruition, the medical record is often the only source of the truth and is likely far more reliable than memory. In the legal system, clear and legible documentation is regarded as an essential element and is given much greater evidentiary strength than the

memories of the parties involved or any witnesses. The failure to document relevant data is in itself considered a significant breach of and deviation from the standard of care. Aside from the protections for legal jeopardy that a well-documented record can provide to a doctor, the patient's record provides the only endearing version of the care as it evolves over time and serves as a reference of significant value in emergency care and quality assurance.

Many practitioners believe that writing more is the solution to having a good medical record; however, simply writing with greater efficiency and being attuned to what is most relevant to the patient's care is of greater value. Practitioners should keep in mind three sovereign principles of documentation which closely resemble the principles of medical decision analysis [12]. The first principle is to record the risk-benefit analysis of important decisions in the clinical care of the patient. This analysis should include even obvious or the given risks and benefits. In reviewing medical records, it is not uncommon to see doctors focus much of their attention on the risk side of a patient undergoing treatment/procedure without giving equal attention to the benefit side of the decision. Conducting this analysis holds true for all decision-making involving a patient. This includes not only documenting the risk benefit of a treatment/procedure itself but also any medications prescribed, as well as post-procedure treatment recommendations. The second essential principle of documentation is the use of clinical judgment at crucial decision points [12]. A common definition attributed to clinical judgment is "an assessment of the clinical situation and a response congruent to that assessment." There are several reasons why documenting this essential element is useful in liability prevention. One of those reasons, and perhaps the most obvious, is the exercise of clinical judgment which is based on both objective and subjective clinician factors that emerge from the actual patient encounter; no one else had direct experience with the patient. To derive a benefit from the immediacy of these observations, it is critical to identify and document the decision-making process that goes into a treatment decision. This is especially

true if the surgeon finds it necessary to deviate from their preoperative plans or the customarily used technique. In the event of a future medical negligence action, an expert witness is charged with the responsibility of reviewing a medical record to determine whether or not the standard of care was met. While the benefit of the doubt is given to the individual who was present at the time of the patient encounter or procedure, this benefit is lost when the clinical judgment is not recorded. The final principle of documentation relates to documenting the patient's capacity to participate in his or her own care. This includes making note of a patient ability to understand all aspects of the treatment/procedure being recommended for them, as well as understanding the purpose of medications prescribed and discharge instructions.

9.3.1 Documentation

The treating doctor should always have a legible and concise medical record. Giving consideration to who may be a possible reader of the medical record is one way for a doctor to achieve sufficient clarity, avoid cryptic communication styles, and achieve the goals of the record in both patient care and liability prevention. Remember that in most of the 50 states, there is a process whereby patients are permitted to view their own records. It further goes without saying that medical records should never be altered from their original form. If a correction or amendment needs to be made to the record, one option for achieving this is to create a new entry with the current date and indicate that it is an addendum to the original entry and then describe the correction. Practitioners should at all times avoid making cross outs and insertions and using arrows to rectify an error. In the event that a strike-through of a word or phrase is made, be certain that the original text is still visible to a reader and initial and date the correction. Making use of a new entry to describe any corrections in the record is the preferred method of rectifying a mistake or inaccuracy and falls in line with the principle of transparency. This is clearly also the case with

electronic records where computer forensics can reveal when and what changes have been made to an electronic record. It further eliminates any perceived notion that a record is misleading or has been fabricated in any way. In the legal world of medical negligence, it is commonly said “if it’s not in the record it didn’t happen.” Put another way, poor records mean a poor defense and no records mean no defense. Regardless of what a doctor recalls of the circumstances involving a patient, in the event of an adverse outcome, it is the medical record that will serve as the primary and most trusted source of information in the view of judges and juries.

Aside from utilizing the medical record to document the medical history, all discussions and interactions with patients, and the conducting of a risk-benefit analysis, the medical record should also contain a descriptive operative report, a complete anesthesia record, and the informed consent signed by the patient.

9.3.2 Anesthesia Record

With the increased attention being given to the administration of anesthesia by cosmetic surgery providers, particularly in an office setting, the importance of a complete and legible anesthesia record cannot be overstressed. In the unfortunate circumstance of an adverse event relating to the administration of anesthesia, the anesthesia record will serve as a key piece of evidence to the defense to demonstrate exactly what occurred during an operative procedure. When it comes to documenting the elements of anesthesia administration to a patient, the more information written, the better. In addition to general patient identifiers (e.g., name, date of birth, height and weight, BMI, and Mallampatti score), the anesthesia record should contain, at a minimum, the name and dosage of all anesthetic medication provided to a patient, the time each medication was administered, and the patient’s vital signs, not only at the start and stop of a procedure but at intervals throughout. The anesthesia record should also document, either by initials or name, everyone that was present during the operative

procedure. It should further be documented the start and stop time of the anesthesia. Finally, the anesthesia record should be signed by the anesthesia provider. Anesthesia record templates are available to use as a guideline from multiple professional associations and societies, including the American Dental Association, as well as the American Association of Oral and Maxillofacial Surgeons. Additionally, with the changeover to electronic medical records, computer software is available that includes custom anesthesia record forms that can be tailored to a doctor’s individual needs. Finally, to the extent that any patient monitors are used during an operative procedure that have the capacity to print a record of the patient’s vital signs, the printed strips should be attached to the anesthesia record and placed in the patient’s medical record. Such strips are particularly important if they document any problematic periods while the patient was under anesthesia.

9.3.3 Discharge Notes and Instructions

Of equal importance to documenting an initial patient consultation, due diligence should be given to making a proper discharge note in the medical record, as well as preparing adequate written discharge instructions for the patient. Post procedure care and patient responsibilities should be communicated clearly to the patient in both verbal and written forms. Ensuring that a patient thoroughly understands post-procedure care instructions is vital to preventing the occurrence of a non-compliant patient due to confusion or lack of information. Documentation of the advice given to a patient is as important as the verbal communication. A doctor can be held negligent if proper instructions are not given to a patient upon discharge regarding medications to be taken, physical care required, the need for urgent reporting if any untoward complication happen, and advised follow-up appointment. A doctor should have strict discharge criteria in place as to the parameters required before a patient may be discharged. In the hospital

environment, the postanesthesia care unit may have discharge criteria in place as standard protocol.

Similar to an informed consent document, discharge instructions should be written in simple, nonscientific terms that are set forth in a concise and legible manner. A doctor should also have the patient sign a copy of the discharge instructions, to be placed in the medical record, to document both understanding of the instructions and receipt of the same. Patients should be apprised of all post-procedure care instructions to be followed, including medication information, as well as any signs and symptoms that should prompt the patient to seek further medical treatment. It is also important to give the patient very specific information on how to reach the surgeon or another doctor covering for them if the patient develops problems or has concerns.

9.4 Unanticipated Outcomes

It goes without saying that unanticipated outcomes are inevitable when performing surgery, especially cosmetic maxillofacial surgery, where the results directly impact the physical appearance of a patient and where success or failure is almost solely determined by the patient's own perception. Whether the unanticipated outcome is a dissatisfied patient, a non-compliant patient, or a clinical error occurred on the part of the doctor, all of these scenarios should be addressed promptly and efficiently by the doctor and his/her staff. Turning a blind eye to an unforeseen outcome, regardless of how minor or complicated, will almost certainly have a negative result for the doctor and frequently is the main trigger of a lawsuit.

9.4.1 Dissatisfied Patient

Even the most experienced and skilled surgeon can have a dissatisfied patient, particularly one undergoing a cosmetic procedure where the motivations for having treatment/surgery are often exceedingly personal. History has shown that

there is a higher incidence of patient dissatisfaction in those with underlying psychological profiles. Fortunately, with the use of an effective and thorough initial patient consultation and medical examination, hopefully a doctor has already identified those individuals with a psychological background that may be more likely to contribute to the feelings of dissatisfaction or frustration with the outcome of a procedure/treatment even if everything went according to the plan. As mentioned earlier in this chapter, there will be patients who have a motivation for cosmetic treatment because of an underlying psychological issue, whether depression, anxiety, or some other mental health condition. These patients tend to have unrealistic expectations as to what can be achieved from cosmetic treatment and therefore are almost impossible to appease. This is yet another reason why selecting the right patient for a procedure/treatment is integral to avoiding a malpractice claim later on by a patient who is dissatisfied.

In the event a doctor encounters a dissatisfied patient, it is imperative for the doctor to listen carefully to the patient's concerns. In most cases where a patient expresses dissatisfaction, the doctor and the patient can reach a mutual understanding on how to rectify the situation and the patient ultimately walks away happy. Having the foundation of a strong and healthy doctor/patient relationship can serve to help remedy unsatisfactory outcomes. A patient is more likely to sue if the patient perceives that his/her doctor is lacking empathy and communication skills, particularly in addressing a patient's unhappiness of a procedure.

9.4.2 Non-compliant Patient

The non-compliant patient can often be described as the one that does not listen. The patient's inability or refusal to comply with post-procedure care instructions is more likely to result in a poor clinical outcome for the patient, as well as increase the risk for post-procedure/treatment complications, including, most significantly, infection. A treating doctor must insure that a

patient's non-compliance is not the result of confusion with the discharge instructions or simply a lack of attention by the doctor to appropriately inform a patient about necessary post-procedure care. A doctor's failure to do so will not bode well in establishing a defense later in the event of a liability action. If a patient is non-compliant due to their own negligence or lack of caring, it is imperative that the doctor continues to reinforce to the patient the importance of following medical instructions and complying with all post-procedure care obligations to avoid complications and carefully document the non-compliant behavior. Ideally, this should be conveyed to the patient both in writing and orally. The doctor should document in the patient's medical record each time these conversations occur, including the instructions that were provided to the patient. A doctor's concerns with the potential of a patient's non-compliance should also be documented. These key steps, although seemingly minor, can serve to protect the doctor should a liability claim be asserted. Unfortunately, even giving extra time and attention to a patient and reinforcing the importance of compliance with instructions from the start to the end of treatment, a doctor will still encounter those patients that simply will not comply. The primary ways to handle these patients are through continuous communication and documentation.

9.5 Medical Negligence and What to Do About It

In 2015 there were 15.9 million surgical and minimally invasive cosmetic procedures performed in the United States, including neuromodulators, facial fillers, laser hair removal, and liposuction [13]. Given this huge number of procedures, it is not unreasonable that bad results such as infections, allergic reactions, uneven skin color, hematomas, tissue necrosis, abnormal scarring, and even death have led to large recoveries by patients through medical negligence actions [14]. Patients file malpractice lawsuits for a variety of reasons, including poor relationships with their doctor that preempt the alleged malpractice, medical

advice to seek a legal remedy, and media advertising. Most surgeons will be involved in a medical malpractice case some time in their career in one of the several capacities, such as the defendant treating surgeon, a fact witness, or an expert witness. Regardless of the circumstances surrounding a lawsuit, it is helpful to be able to put malpractice claims in context and to understand the elements of malpractice. The laws of malpractice, the procedures involved, and the judicial process vary from state to state and from country to country. The most common types of medical malpractice litigation, ranked in order of frequency, are (1) lack of due care, (2) lack of informed consent/battery, (3) vicarious liability/ respondeat superior/negligent supervision, (4) injury to third parties, and (5) abandonment [15].

To understand medical negligence, it is perhaps easier to take things from the point of view of the patient. Generally, a patient approaching a doctor for medical treatment, either medically necessary or elective, expects that the doctor or medical professional will provide the best treatment possible, based on their knowledge and skill, and, further, that this professional will not do anything to harm the patient, either because of their own negligence or carelessness or the carelessness of their staff.

As a claim for lack of due care is the most common stated cause for filing a malpractice lawsuit that is what this chapter will focus on. In any medical malpractice or medical negligence case, an affected patient will need to prove four things to the court in order to prevail: (1) that the doctor owed them a duty of care; (2) that the doctor breached that duty of care, via some type of negligence; (3) that negligence caused the harm from which they claim to be suffering; and finally (4) that the harm is sufficiently severe to merit the awarding of damages (compensation) [16]. To rebut those allegations, the doctor must provide evidence that shows either (1) the doctor owed the patient no such duty (as discussed earlier in this chapter), (2) the doctor was not negligent in rendering medical services, (3) that the harm the patient is claiming to have suffered was not a direct result of any of the doctor's actions, or (4) that the damages are of an insignificant magni-

tude to merit a monetary award. Simply put, the person making the claim for medical negligence or malpractice must establish that it was more likely than not that the negligence occurred by the doctor and further that negligence was the direct cause of their injuries and if serious enough to deserve compensation.

As previously stated, it is the creation of the doctor/patient relationship that creates the fiduciary duty on the part of the doctor to treat the patient and to treat him or her properly. The patient brings their medical issue to a doctor expecting relief, and should the doctor accept this patient, a doctor/patient relationship is formed [17]. As described in more detail earlier in this chapter, this relationship takes the form of a contract³ (as a result of such things as informed consent and payment of a fee for a specific service), and any breach of that contract by the doctor will result in certain consequences. However, it is important to understand that, as contractual as these relationships may seem, they are also seamlessly intertwined with elements of tort, as the breach of these contractual duties owed to a patient is often due to negligence; thus, courts do not typically use contract law to determine the outcome. In various professional vocations, a “duty” is often alluded to as a moral code of which an individual should aspire to and uphold when carrying out various job responsibilities. This concept is especially prevalent in the medical community, as the goal is mainly about preservation of life. However, despite the existence of this moral code, medical professionals have a *legal* duty to the patients under their care.

A majority of claims for medical negligence do not succeed because the plaintiff (patient, in this scenario) cannot establish that harm has occurred as a direct result of a negligent act *or* a failure to act, as the case may be [18]. Remember, negligence *cannot* merely be inferred from the existence of a bad result. Adverse results are a risk of any procedure. Unless the patient can prove that their bad result was directly linked to a doctor’s negligence, that is, the duty to treat the

patient with the skill and judgment that would be exercised by other surgeons under similar circumstances, the case will be dismissed as a matter of law. In certain circumstances, a plaintiff may however prevail under a theory of negligence called “*res ipsa loquitur*.” In such a case, evidence presented may reflect that the doctor in question was not the direct cause of the injury in question; however, it may be determined that the injury could not have just resulted “on its own” and would have had to have happened due to some sort of negligence in the procedure. In this case, the doctor may still be liable for the plaintiff’s injuries. See, generally, *Shepard v. United States*, 811 F. Supp. 98 (1993).

But how can an individual doctor know what the standard of care may be and whether or not they have truly breached it? In the United States, doctors are generally held to a national standard of care. This means that any particular doctor’s actions are held to what a reasonable doctor in like or similar circumstances would do. Historically, this was not the case; a strict locality rule was imposed, meaning that a doctor in a particular area was held to what another doctor in that same area would have done in the same or similar circumstances. However, this gave way to the national standard of care with the sophistication of modern medicine and technology, as well as the fact that, procedurally, it was difficult to entice doctors within the same communities to testify against each other as experts in court.

Based on this information, we can infer that a breach of this duty would be a diversion from the national standard of care or, to put it in like terms, a diversion from what a reasonable doctor would do in the same or similar circumstances. Though a doctor may not always be in a position to follow proper protocol, he/she is still expected to use his/her special knowledge and skill in an appropriate manner while also keeping in mind the interests of the patient. To avoid negligence, and a dereliction and breach of his/her duties, as described earlier in this chapter, the doctor must seek informed consent from the patient before any procedure. It cannot be overstated that the process of obtaining informed consent must not only include procuring the patient’s permission

³In this case, a type of implied contract that is still considered legally binding

prior to performing the procedure but also informing the patient as to the potential risks, benefits, and alternatives of the specific procedure they are seeking to undergo. Asking the patient questions about their personal and familial medical history, their day-to-day routines and diet, and about their overall well-being can serve to provide some of the most helpful information needed by a doctor prior to rendering treatment. If all of these steps to maintaining open communication are met, it is less likely that the doctor will face a lawsuit due to medical negligence.

9.6 Ten Tips to Practice By

Consider the case of two doctors who have both just been named in a medical malpractice suit. One of the doctors is much older and more senior to the other who is just newly out of residency. While the nervousness is evident on the faces of both practitioners for having been accused of medical negligence, the young doctor is far more distraught and continuously expresses his utter dismay at having a patient make allegations of wrongdoing against him. It is at that time that the senior doctor turns to the other doctor and says “welcome to the practice of medicine, you are not truly a surgeon until you’ve had a claim filed against you.” The reality of the matter is that we live in a very litigious society where doctors, particularly surgeons, are vulnerable to claims of liability. The unfortunate circumstance of being involved in a medical malpractice action is never anticipated nor an easy process for anyone involved. It is also important to remember that the mere filing of a lawsuit does not mean there was negligence. However, in the event that it happens, if you can look at the way that you practice medicine on a daily basis and say with confidence that you have made every effort to provide the safest quality of care to your patients within your capacity, by exercising open communication, maintaining adequately documented medical records, establishing strong doctor/patient relationships, and providing an overall safe and secure environment for patient care, then you are one step ahead in the game. The rule of thumb is

not to wait until you find yourself in the midst of a malpractice action to recognize that you have to make changes to the way you practice medicine. Following the below tips are just a few ways to help avoid liability and prepare your defense in the event it happens:

1. Be transparent. Not only with patients but staff as well. Transparency and effective communication will contribute to establishing a strong doctor/patient relationship and create a healthy and safe work environment.
2. Give of your time. Give each individual patient and each case the attention it deserves. Regardless of how busy your schedule and how full the waiting room, recognize that each patient is seeking your professional advice and experience on what is oftentimes a very personal issue that goes beyond medicine and delves into the psyche. A patient needs time to fully understand the cosmetic maxillofacial procedure they are considering and ask all questions they may have. Adequate time with a patient also affords the doctor the ability to ascertain a complete medical history and fully evaluate a patient’s motivations and expectations. Rushing a consultation, appointment, or even the procedure itself doesn’t benefit anyone in the end.
3. Document. Adequately document in the medical record all relevant aspects of a surgical procedure from patient evaluation all the way through post-procedure assessments. The medical record is your diary for this patient and should identify both risks and benefits of a procedure, a patient’s candidacy for treatment, alternatives to treatment, as well as the anticipated outcome and complications encountered. Remember “*if it’s not in the record, it didn’t happen.*”
4. Be honest. Don’t be afraid to tell a patient “no.” A doctor should never lose sight of their ethical obligations to a patient and providing safe, quality care in lieu of financial gain.
5. Use sound judgment. Trust your instinct. If there is any factor in evaluating a patient for treatment, whether surgical or not, that gives

you pause, causes unease, or raises a potential concern, do not proceed with the treatment. Your training, education, and experience all play an essential role in establishing clinical judgment. A doctor should never underestimate the importance of clinical judgment in their decision-making. It is always better to pass up a potential surgery than second guess your decision down the road, especially in the face of liability.

6. Record carefully. Don't underestimate the importance of a complete and legible anesthesia record. With the increased attention in this country being given to anesthesia administration, particularly office-based anesthesia, the need for heightened awareness by healthcare providers to patient safety and to the necessary evaluation and monitoring of patients undergoing anesthesia, has never been greater.
7. Be humble. Be cognizant of your limitations and never be afraid to ask for help. A continuous theme in medical malpractice cases includes practitioners acting beyond the scope of their experience and expertise and failing to seek a consultation or second opinion. There is no shame in asking for a second opinion or requesting a consultation.
8. Prepare. Be prepared for a patient emergency in the office setting before it happens. This includes the following: have an adequate written policy/plan in place that sets forth protocol and procedures that can be followed by all staff in the event of a surgical emergency; orient staff to the policy/plan so they are familiar with everyone's role; periodically run emergency drills and subsequent debriefing with office staff including assignment of both live and post-event documentation in the medical record (particularly adverse anesthesia events); and, finally, actually follow the policy/plan. Remember, it is better to have no policy at all than for an office to have written policies that are not followed.
9. Know the law. Be familiar with your individual state laws, as well as the rules and regulations of state and federal regulating

agencies regarding any and all necessary licensing, permitting, and certifications that are applicable to your office practice and the types of cosmetic procedures performed.

10. Stay Updated. Have a set schedule for the year establishing designated time periods for reviewing, revising, and updating office policies/procedures, patient forms, and any informational brochures or leaflets. Add to this schedule specific dates for reviewing and inspecting office equipment, including any and all patient monitors, radiographic equipment, as well as emergency equipment.

Conclusion

Although cosmetic maxillofacial surgery is safe, it still carries with it the same medicolegal issues that come with any surgical procedure. In order to decrease the likelihood of litigation or legal actions, it is important that the surgeon evaluates and considers each case individually emphasizing the importance of a well-documented and organized medical record, informed consent, and a strong doctor/patient relationship.

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Part II

Complications of Specific Procedures

Complications of Facial Resurfacing

10

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Abstract

Facial resurfacing includes minimally invasive procedures that alter the texture as well as the appearance of the skin. These procedures have a high satisfaction rate with minimal complications. However, if complications occur, this can lead to patient dissatisfaction. This chapter will review the various complications and ways to prevent them. Additionally, proper management of these complications will be discussed.

10.1 Introduction

Facial resurfacing describes procedures designed to alter the texture and appearance of the skin of the face. The most common reasons people pursue facial resurfacing procedures are to reverse the effects of photoaging and acne scarring [1]. High complication rates or frequent undesired

outcomes are not well tolerated for cosmetic procedures such as these. Prevention is of the utmost importance when considering facial resurfacing. The vast majority of complications can be avoided with proper patient selection, appropriate pre- and postoperative care, and guidance of expectations. Identifying and properly treating patients in a timely manner in the event of a complication is also crucial to ensuring the long-term success of the procedure.

As with most procedures, there are some well-known factors that make one a poor candidate for facial resurfacing. Any history of hypertrophic scarring, keloids, or general poor healing, as well as severe active rosacea or acne are relative contraindications to facial resurfacing. Other systemic conditions with skin involvement such as history of herpetic infection, vitiligo, lichen planus, psoriasis, and verrucae can be induced or recruited to the surgical area (Koebner's phenomenon) and thus also may prohibit skin resurfacing. Lastly, any therapy that directly affects the

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pilosebaceous subunit can have a negative influence on healing. Isotretinoin (Accutane), for example, suppresses activity of the pilosebaceous subunit and interferes with proper healing after facial resurfacing.

Despite proper preoperative consultation and surgical preparation, some complications are unavoidable. In general, complications associated with facial resurfacing can be categorized as either pigmentary, cicatricial, infectious, inflammatory, or systemic [2].

10.2 Pigmentary Complications

10.2.1 Transient Hyperpigmentation

Dyschromia is the most common complication associated with facial resurfacing and can occur at significant rates despite proper preoperative preparation and accurate history taking. The risk and severity of pigmentary change are directly related to the type and depth of treatment. For example, dyschromia that results from superficial wounding with peeling agents such as Jessner's solution or trichloroacetic acid (TCA) below 20%, for instance, is almost universally reversible [3].

Transient hyperpigmentation occurs with significant frequency, experienced by as many as 36–37% of patients with Fitzpatrick III and VI skin types [4, 5]. The problem is usually identified in the first month and commonly resolves without intervention over the next several months. Pathogenesis of post-inflammatory hyperpigmentation (PIH) is hypothesized to be the normal response of melanocytes and keratinocytes to the induced thermal, chemical, or mechanical damage that occurs during resurfacing. Following cutaneous inflammation, there is an overproduction of melanin and an abnormal distribution of pigment [6]. In order to expedite the resolution, patients with PIH can be started on a hydroquinone-containing preparation or either topical retinoic, azelaic, or glycolic acid compounds after the first month. These preparations may be irritating to the skin and should be combined with a mild topical corticosteroid [7].

Sun exposure, which can worsen the situation, is avoided or handled with proper protection. With appropriate treatment, the hyperpigmentation often resolves within a few months.

Topical agents such as tretinoin, hydroquinone, or glycolic acid used in the preoperative period have failed to lower the rate of PIH [8]. In contrast, at least one study found that using topical steroids briefly in the immediate postoperative period reduced the incidence of PIH from 75% down to 40% following ablative fractional resurfacing [9]. In this study, clobetasol propionate 0.05% ointment was applied for 2 days postoperatively, followed by petrolatum jelly for 5 additional days. Clobetasol propionate has also been demonstrated to have the most potent suppression of UVB-induced erythema and pigmentation [10].

10.2.2 Hypopigmentation

Delayed-onset hypopigmentation is a much more serious form of dyschromia that can occur following resurfacing procedures. This complication does not become apparent until the residual erythema and hyperpigmentation have resolved, which can last for 6–12 months. Prior to use of fractional laser techniques, delayed-onset hypopigmentation occurred in up to 19% of cases, although less than half of which were found to be true hypopigmentation [11]. Currently, with the use of fractional CO₂ laser resurfacing, some studies demonstrate 0% incidence of long-term hypopigmentation [12]. Despite the safety profile of newer fractionated systems, if used inappropriately, they are still capable of doing irreversible damage and disrupting normal melanogenesis.

It is important to stress that resurfacing should include cosmetic subunits. In the case of delayed-onset hypopigmentation, feathering a chemical peel (glycolic acid 30–40% or light trichloroacetic acid (15%)) into the untreated area or low-energy treatment of untreated areas can camouflage the otherwise discrete and unsightly transitions from one area to another [7]. Makeup can also be used to the same effect.

10.3 Infectious Complications

The integumentary system is an important barrier in preventing infections. When this barrier is interrupted, as it is by most of the commonly used facial resurfacing techniques, one becomes susceptible to bacterial, viral, and even fungal infections. The most common infection (or reactivation) associated with facial resurfacing is herpes simplex virus (HSV) infection [5]. The incidence of HSV infection is roughly 2–7% [5, 7] with conventional laser surgery. This relatively high incidence of infection is in spite of the widespread use of antiviral prophylaxis. Some authorities call into question the need for antiviral prophylaxis due to low incidences of infection with some of the newer methods for resurfacing [12, 13]. Today, HSV infection rates of only 0.3–2% are observed with fractional laser skin resurfacing [14, 15]. Common regimens for HSV prophylaxis prior to resurfacing procedures include acyclovir 400 mg three times per day, valacyclovir 500 mg twice per day, or famciclovir 250 mg daily [16]. Treatment starts 1 day prior to the procedure and is continued for 5–7 days following the procedure.

Bacterial infections are uncommon but do occur in such a frequency as to not be ignored. Administration of broad spectrum antibiotics for prophylactic reasons is not a substitute for proper surgical technique, but even with impeccable antiseptic practices, bacterial infections occur at clinically significant rates. One study demonstrated that postoperative bacterial infections following CO₂ laser resurfacing occur at a frequency of 8% [17]. This incidence was reduced to approximately 4% when systemic antibiotics (ciprofloxacin) were started preoperatively [17]. Further reductions in the incidence of bacterial infections are seen with fractional photothermolysis, with incidences as low as 0.1% being commonly achieved [14, 15]. Although differing antibiotic regimens have been described in the literature, duration of use is most commonly until reepithelialization has occurred [13]. Bacterial infections, when they do arise, occur early in the postoperative course. *Staphylococcus*, *Streptococcus*, and *Pseudomonas* are the most commonly encountered organisms [2].

Fungal infections following resurfacing remain rare, at roughly 1% [5, 17]. With such low rates of occurrence, the prophylactic use of antifungal medication is unwarranted. Fungal infections, when they occur, are most frequently seen late in the postoperative period, at postoperative day 10, or later.

Regardless of the time of onset or the causative microorganism, prompt and appropriate treatment is essential for preventing adverse sequelae. Culture, followed by gentle and frequent cleansing, is essential. Daily monitoring for improvement is also important in successful management of postoperative infections associated with facial resurfacing.

10.4 Cicatricial Complications

The most feared complication of facial resurfacing is scarring. A thorough knowledge of wound healing is essential in understanding and preventing cicatricial complications. It is well known that reepithelialization occurs from the base of the pilosebaceous subunit. Therefore, resurfacing procedures that penetrate to a depth below the pilosebaceous subunits and within the reticular dermis can result in extensive scarring. Overtreatment therefore should be avoided, and the novice provider should take special care when treating areas such as the perioral region, mandibular borders, and lower eyelid that have a higher incidence of scarring [18]. Additionally, areas of lower density of pilosebaceous units may be more prone to scarring or more noticeable scarring. For instance, the neck has many fewer pilosebaceous units than the face (face has 30 times more pilosebaceous units [18]) and therefore should be treated less aggressively.

As mentioned earlier, patients with a history of poor wound healing, hypertrophic scar formation, or keloids are poor candidates for aggressive or deep resurfacing procedures. Infection, also noted elsewhere, is closely associated with scarring. In one series, a 3.8% incidence of scarring was documented following facial resurfacing, all of which were attributed to postoperative infection [11]. Prompt diagnosis and treatment can

limit and even prevent scar formation after infection.

Another factor alluded to earlier that warrants special mention is prior isotretinoin treatment [19]. Preoperative [19] and even postoperative [20] isotretinoin therapy are noted to increase risk of postoperative complications. Additionally, electrolysis, which can result in significant injury to sebaceous glands, may predispose these patients to scarring following facial resurfacing.

Cicatricial complications may not present until late in the healing phase. Intense, prolonged erythema may portend the presence of scar tissue at a surgical site [2]. Most areas following resurfacing should be well healed by 2–3 weeks. Areas that take longer to heal are likely to result in some amount of scar tissue formation. The skin that heals with hypertrophic scarring can usually be managed with topical and intralesional steroid injections, silicone sheeting, and pulsed-dye laser therapy [18]. Areas of scarring that appear to be resistant to conventional management should be referred promptly to a wound care specialist.



Fig. 10.1 Facial erythema following facial resurfacing (Courtesy of Dr. Nuveen)

10.5 Inflammatory Complications

Some degree of erythema is expected following facial resurfacing and is often an indicator of successful treatment (Fig. 10.1). As with cicatricial complications, postoperative erythema directly correlates with the depth of treatment. Most commonly the posttreatment erythema resulting from resurfacing procedures resolves over the next several days. Prolonged erythema is that which lasts longer than 1 month when considering ablative therapies [21]. Incidences of prolonged erythema higher than 12.5% have been reported, with most of the cases resolving over the next 2 months [21]. Some attempts at limiting the frequency at which this complication is encountered for laser treatment have been successful, including light-emitting diode (LED) photomodulation and vitamin C preparations [18]. Although unpleasant, patients should be informed that some postoperative erythema is expected and that it may be protracted in some instances. Even with aggressive treatment,

reepithelialization is usually complete by 2 weeks, at which time makeup can be used to mask this temporary complication.

10.6 Systemic Complications

Systemic complications, which in some instances can be life threatening, are fortunately quite rare. Phenol, which is the only peeling medium that can reproduce results similar to laser resurfacing for moderate photoaging and scarring [22], is cardiotoxic. In humans, the incidence of cardiac dysrhythmias has been recorded as high as 23% in some studies [23]. Fortunately, the majority of the dysrhythmias encountered during chemical peeling have been mild, including tachycardia and premature ventricular beats. More worrisome rhythms, however, such as bigeminy and atrial and ventricular tachycardia have also been recorded [24]. In one retrospective study, in which preventative measures such as hydration and preoperative propranolol administration

were performed, dysrhythmias were limited to an incidence of 6.6% [23]. Phenols by in large are usually excreted by the kidney or detoxified by the liver. The remainder of the agent is metabolized into CO₂ and H₂O and subsequently exhaled. Blood levels of phenol following application of 3 mL of a 50% solution have been previously quantified (0.68 mg/dL) [23]. This level is significantly lower than that found in studies in which patients were exposed to large amounts of phenol and treated for significant systemic complications.

There is still controversy as to whether the observed cardiac dysrhythmias seen during a phenol chemical peel procedure are directly related to the peeling agent. The number of dysrhythmias identified in asymptomatic individuals in the general population is equivalent to, or exceeds, the incidence of dysrhythmias identified in many studies of chemical resurfacing with phenol. This however does not preclude the need to have cardiopulmonary monitoring in use during all deep chemical peeling procedures in which phenol is used.

Another systemic complication related specifically to phenol is laryngeal edema. Laryngeal edema in combination with other respiratory findings such as stridor, hoarseness, and tachypnea following a phenol peel was identified in 1.2% of patients in one study. Of note, all the patients in which these findings developed were heavy smokers [3]. Fortunately, the symptoms resolved within 48 h of starting warm mist therapy.

Toxic shock syndrome, another systemic complication of facial resurfacing, is exceedingly rare. Toxic shock syndrome has indeed been documented following facial resurfacing procedures and thus warrants specific mention. Although it was once thought to occur more frequently with occlusive dressings, it has been shown that this is not the case [14]. Toxic shock syndrome tends to present early in the postoperative course, most commonly 2–4 days after the procedure. The provider should be alerted to this diagnosis by early fever in combination with syncopal hypotension. This is then followed by a scarlatiniform rash and desquamation [3].

Treatment includes hospitalization, supportive care, and prompt administration of broad spectrum antibiotics with antistaphylococcal coverage.

10.7 Acne and Milia

Milia are keratin retention cysts, most often caused by blocked hair follicles. They are quite common with both traditional laser resurfacing and fractionated techniques, 14% and 19%, respectively [21]. They are most commonly observed with overuse of obstructive ointments. The affected areas tend to resolve with the help of normal postoperative cleansing procedures. In rare instances needle-assisted enucleation can be used for persistent lesions [14].

Acneiform eruptions, another mild postoperative complication of facial resurfacing procedures, are common (5.3% [25], 3.5% [12]). The interruption of follicular subunits and abnormal reepithelialization contribute to acne exacerbations. In mild cases, topical clindamycin can be applied to aid in resolution of the breakout [18]. In moderate to severe acne exacerbations, a course of tetracycline-based oral antibiotics may be indicated. Patients that have a history of worsening acne following resurfacing may be started on antibiotics prophylactically to limit the duration or severity of the event [26].

10.8 Patient Satisfaction

As noted earlier, guidance of expectations and verbalization of discrete goals are imperative in ensuring a successful provider-patient relationship. Photodocumentation cannot be overemphasized when contemplating a cosmetic procedure. Standardization of the photographs in the pre- and postoperative phases will be extremely illustrative in demonstrating the accomplishment of pre-stated goals.

Successful facial resurfacing, like all surgical procedures, necessitates patient compliance and direct patient involvement. Peeling, for instance, is most successful when combined with pre-peel

conditioning. Those patients that cannot tolerate “priming” may not be great candidates for this type of procedure and can expect higher rates of postpeel problems [27]. Explicit postoperative instructions that have been verbally reviewed and given to the patient are standard practice, and it must be stressed that they be followed.

Conclusion

Finally, facial resurfacing is a safe and successful procedure in treating the damaging effects of sun exposure and acne. Recent advances, specifically to the units used in photothermolysis, have helped to reduce complication rates even further. In spite of these improvements, extreme caution is advised for the novice user. The goal is to avoid any unwanted adverse event, but if one does occur, proper and timely management is advised.

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Abstract

Laser treatment has become a vital part of any maxillofacial cosmetic surgery practice. This treatment includes hair removal, photofacial rejuvenation, laser ablative skin resurfacing, and nonsurgical skin tightening procedures. This chapter will review the history and principles of laser treatment. Potential complications will be discussed. Appropriate management of these complications will also be addressed.

11.1 Introduction

Laser treatment: Options for facial rejuvenation have increased dramatically in the past 30 years.

With the technological advancement of laser equipment, patient demand for nonsurgical and minimally invasive surgical facial rejuvenation procedures, combined with the media's fascination with laser treatments, the number and scope of (facial rejuvenation) procedures have increased dramatically. According to ASAPS (American Society of Aesthetic Plastic Surgery) 2015, cos-

metic procedure statistics, hair removal laser, photofacial rejuvenation, laser ablative skin resurfacing, and nonsurgical skin tightening procedures have gained popularity. As the number of procedures has increased, so has the number of complications and undesirable outcomes. Therefore, it is important to understand the source of these complications for prevention and minimization of patient dissatisfaction [1].

11.2 History

Use of light energy to treat skin disorders dates as far back as 1899, when lupus vulgaris was treated with Finsen lamp, and artificial UV light sources were used to treat wounds and rickets in 1901 [2]. The birth of modern lasers (light amplification by stimulated emission of radiation) can be attributed to Albert Einstein. In his 1917 paper, The Quantum Theory of Radiation, Einstein postulated that excited atoms in isolation can return to

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a lower energy state by emitting photons, a process called spontaneous emission. His work was furthered by Charles Townes, Nikolay Basov, and Alexander Prokhorov, who were awarded the Nobel Prize in physics for their creation of MASER (microwave amplification by stimulated emission of radiation). This work was advanced to operate in the optical and infrared region by the discovery of a solid-state MASER by G. Makov, C. Kikuchi, J. Lambe, and R.W. Terhune. Finally, in 1960 the theory became reality when Maiman developed the first laser at Bell Laboratories [3]. The first clinical laser developed was a ruby laser, which was used for retinal tears, detachments, and tumors. Leon Goldman first published the use of laser energy to treat skin lesions, describing selective destruction of pigmented structures of the skin including hair follicles [2]. The first three continuous wave lasers were all developed in 1964 and included the CO₂ laser, Nd:YAG laser, and argon laser [3]. Further innovation led to pulsed laser (Shutter and Q-switched) technology, decreasing complications and undesirable side effects of laser treatment.

In 1983 Anderson and Parrish revolutionized the laser industry with the development of the concept of selective photothermolysis. The concept states that by using a laser with a preferentially absorbed wavelength, sufficient fluence, and the appropriate pulse width, it is possible to selectively target tissues with sparing of healthy surrounding tissue [4]. Further refinements of laser delivery technology with the advent of “fractionation” of the beam further reduced risk of hyperpigmentation, hypopigmentation, blistering, and scarring. The recent introduction of nano- and picosecond laser technology will further improve safety profile of the laser treatments.

11.3 Principles (Basic Science) of Treatment

LASER (light amplification by stimulated emission of radiation) energy is monochromatic, coherent, and collimated. These properties allow

the use of specific laser energy to target selective chromophores: melanin, hemoglobin, water, and tattoo ink, sparing the surrounding tissue collateral damage during treatment [5]. During laser treatments, a portion of energy is reflected, transmitted, or absorbed by the skin. The absorbed energy is responsible for clinical changes of facial skin.

In order to understand the uses of lasers in the clinical setting, a brief understanding of the components and variables of the laser is needed in order to select the most appropriate one. Three variables in addition to the wavelength must be considered in determining the correct laser:

1. Fluence/power density
2. Pulse width
3. Mode of delivery

The fluence or power density describes the rate of energy delivered per unit area (joules/cm²). The fluence is also dependent on the amount of chromophore concentration; therefore, the ideal fluence to achieve a clinical goal is dependent on the skin type of the patient. Patients with darker skin types have more melanin and potentially can have increased adverse reactions such as hypertrophic scarring secondary to collateral damage.

The pulse width is the duration of action on the tissue. The pulse width must be approximately equal to or less than the thermal relaxation time, which is the amount of time it takes to transfer two-thirds of the resultant heat to the surrounding tissues. The thermal relaxation time is directly correlated to the size of the target, i.e., small tattoo pigments use pulse width of nanoseconds or even picoseconds, whereas lasers that target hair follicles may use pulse durations of several milliseconds [4].

Wavelengths of each laser determine the target chromophore, which is responsible for absorbing the light and resulting production of the heat through a transfer of energy. The three main chromophores in the skin are melanin, hemoglobin or oxyhemoglobin, and water.

11.4 Chromophores in the Head and Neck [5]

| Chromophore | Target | Devices used |
|-----------------------------|---|---|
| Water | Textural changes; wrinkles, correction of skin laxity | <ol style="list-style-type: none"> 1. Fractional and nonfractional CO₂ laser 2. Fractional and nonfractional erbium laser 3. IPL devices |
| Melanin | Pigment lesions | <ol style="list-style-type: none"> 1. KTP 2. Q-switch Nd:YAG 3. Pico 4. Ruby 5. Alexandrite 6. IPL 7. LED lasers |
| Hemoglobin or oxyhemoglobin | Vascular lesions | <ol style="list-style-type: none"> 1. KTP 2. Q-switch 3. Pico 4. IPL 5. Pulsed dye 6. Long-pulsed wavelength |
| Fat | Excess fat, e.g. neck | Wavelengths between 900 nm and 1500 nm |

The mode of delivery can either be continuous or pulsed, which was determined with early use of lasers as the complications were decreased with pulsed vs. continuous lasers. Delivery methods that use pulsed laser energy result in higher peak intensities for a shorter duration of time compared to continuous laser energy. Between pulsed laser energy, the target tissue and the surrounding healthy tissue are allowed a period of cooling, with reduced collateral damage in surrounding tissue. The three main parameters and wavelengths allow the concept of selective photothermolysis to target desired tissue with reduced unintentional damage to surrounding healthy tissue.

11.5 Fractional Photothermolysis

In 2004, a newer class of therapy was developed called fractional photothermolysis. The concept is that rather than uniformly treating the entire epidermis, the stratum corneum is left largely

intact and thousands of areas (is this the correct word? Is there a unit to describe the area? Microns I think) of thermal damage occur in the epidermis and dermis. These microtreatment areas of the skin, which are known as microthermal treatment zones (MTZs), are unaffected. The major effects of fractional photothermolysis are the epidermal coagulation for resurfacing and the dermal heating leading to collagen remodeling. The parameters used for fractional lasers in addition to fluence and pulse width are pitch and energy. Pitch is defined as the amount of MTZs in the treatment area, i.e., the amount of space in between each MTZ.

Fractional lasers can further be categorized in either ablative or non-ablative delivery. Ablative lasers remove the entire epidermis in a narrow column with surrounding tissues in a zone of thermal injury. The combination of the ablative ability and the zone of thermal injury to the tissue contributes to the efficacy of ablative lasers. In comparison, the non-ablative lasers do not involve the epidermis, and only subepidermal columns are the thermal injury zones [6].

11.6 Complications of Cosmetic Laser Procedures of the Head and Neck

Cosmetic laser procedures of the face and neck skin predominantly deal with unwanted hair removal, lentigines, vascular lesions, facial erythema, acne scars, wrinkles, tattoo removal, and fat reduction. The laser system chosen to treat these conditions differs on the basis of the particular chromophore present in the lesion to reduce complications and increase efficacy of laser treatments for optimal results. Possible side effects and complications of the laser procedure and anesthesia should be discussed with the patient for informed consent [1].

Ablative lasers and fat removal lasers have a higher risk profile. Newer, non-ablative, and fractional laser systems are relatively safe but need multiple treatments to achieve the goal. Patient expectations and how much downtime the patient

will require also determine the type of laser system chosen for the treatment. Although intense pulse light (IPL) is not a true laser and the light energy includes numerous wavelengths (500–1200 nm.), it is used for the treatment of pigmented and vascular lesions, hair removal, as well as photorejuvenation of the face. As with any light-based therapy, hyperpigmentation, hypopigmentation, erythema, blistering, and scarring are associated with IPL treatment [1].

11.6.1 Laser Safety

All surgical lasers are classified as Class IV devices—high-power lasers that are hazardous to view under any condition (directly or diffused/scattered) and which are also a potential fire and skin hazard. The patient and all personnel present in the room should wear wavelength-specific protective safety goggles. When enriched O₂ is present in the treatment field, precautions should be taken to prevent flash burns or inhalational burn injuries with use of the lowest possible fraction of inspired oxygen (FIO₂). When using lasers for periorbital rejuvenation, corneal eye shields are useful to prevent eye injury [7]. Lasers also can create significant laser plumes with facial skin resurfacing and hair removal. The use of special laser masks and a plume evacuator can reduce inhalation of plume particles [7].

11.6.2 Absolute Contraindications to Laser Treatment

11.6.2.1 Infection

Elective aesthetic procedures should not be performed when bacterial, viral, or fungal infections are present in the treatment area of the face and neck [8].

11.6.2.2 Appendageal Abnormality

Patient with abnormalities of hair follicles and sebaceous glands may have problems with wound healing. Concurrent or recent oral retinoid use is generally considered an absolute contraindication for resurfacing procedures for 6 months–2

years after cessation of oral retinoid. Skin grafts and extensive electrolysis may also be considered as contraindications for full field resurfacing, but fractional and superficial full field resurfacing may be safe [8].

11.6.3 Relative Contraindications to Laser Treatment

Relative contraindications to laser treatment procedures of the face and neck include unrealistic expectations, keloid or hypertrophic scarring history, darker-skinned individuals, previous deep (phenol) chemical peels, and history of cold sores [8].

11.6.3.1 Classifications of Complications

General complications of laser skin treatment procedures can be classified depending on the location of target chromophore as:

1. Epidermal complications
2. Dermal complications [1]

11.6.3.2 Epidermal Complications

Primary epidermal complications include hyperpigmentation, hypopigmentation, postoperative blistering and postoperative crusting, and milia formation. Hyperpigmentation can be observed with any type of laser and intense pulse light treatments. It is more common with darker skin colors. Patients with fresh tans are more likely to develop hyperpigmentation. Hyperpigmentation is more common with ablative procedures, i.e., CO₂ resurfacing of facial skin. Risk of hyperpigmentation with laser-assisted hair removal is related to seasonal variations, presence of a tan, and a patient's skin type [1] (Fig. 11.1).

Idiosyncratic hyperpigmentation may also occur with laser treatment. Excessive cooling of epidermal cells with a cryogen spray itself can cause epidermal damage leading to hyperpigmentation [9]. If the patient has a tendency for hyperpigmentation, perioperative conditioning of facial skin with topical retinoids and skin bleaching agents helps to minimize the incidence.

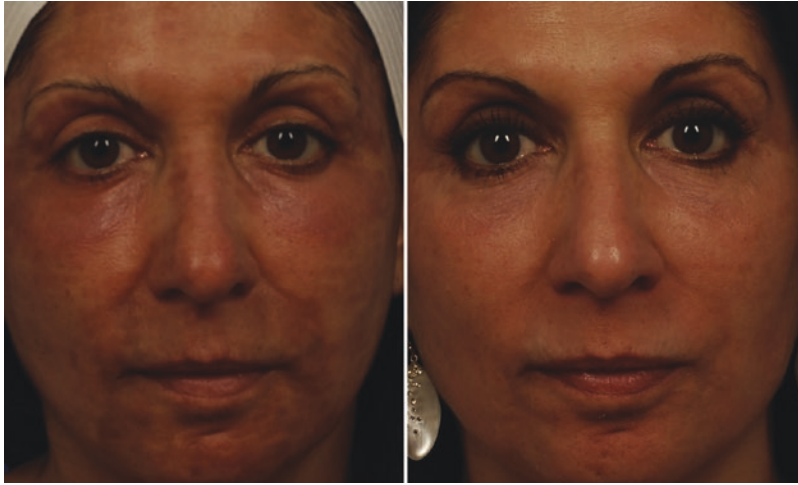


Fig. 11.1 Hyperpigmentation after laser resurfacing and correction with skin care (With permission from Pozner et al: *Laser Resurfacing: Full Field and Fractional*, Clin Plastic Surg 43:515–525, 2016)

11.6.3.3 Hypopigmentation

Hypopigmentation is more common with lasers that target melanin as chromophore or pigment-specific laser irradiation such as hair removal, pigmented lesions, and tattoo removal with Q-switched ruby, alexandrite, and Nd:YAG lasers [1]. Hypopigmentation is more common in patients with darker skin color and those who undergo multiple treatments. Hypopigmentation can occur up to 10% of patients treated for hair removal with long-pulsed ruby and alexandrite laser (Nanni & Alster) [10]. Just like hyperpigmentation, hypopigmentation is often temporary. Delayed permanent hypopigmentation has been recognized as complications after ablative laser resurfacing with CO₂ laser.

11.6.3.4 Blistering

Postoperative blister formation following laser treatment which is usually due to thermal damage of the epidermis is seen with all types of lasers and IPL treatments. Proper intraoperative skin cooling protocol with direct contact cooling with a chilled tip, cryogen spray, or cold air flow can minimize the incidence and improve patient comfort [11].

11.6.3.5 Postoperative Crusting

Crusting after laser treatments is common with Q-switched lasers for tattoo removal, and other laser treatment crusting is inevitable after ablative

laser resurfacing procedures and can be minimized with appropriate postoperative care [1].

11.6.3.6 Milia

Commonly known as whiteheads, milia can occur after facial skin resurfacing procedure during healing period. They can be treated with manual extractions and topical tretinoin and/or glycolic peels [1].

11.7 Primary Dermal Complications

11.7.1 Purpura

Purpura is often associated with vascular lesion ablation with pulse dye laser treatments. With longer pulse duration, pulse dye lasers have decreased the incidence of purpura. Purpura is usually transient, lasting 7–14 days. Proper selection of fluence for treatment can minimize the incidence of bruising [1].

11.7.2 Scarring

Scarring is the most feared complication of cosmetic laser treatment. Any time there is ablation of tissue, scarring is possible. Scarring is more com-

mon with continuous wave lasers. With the advent of pulsed, Q-switched, and fractional lasers, the risk of scarring has decreased. Scarring is always due to excess damage to the collagen within the dermis, secondary to laser-induced thermal damage, or due to postoperative infection.

Risk of scarring is low with pigment-specific lasers, pulsed vascular lasers, non-ablative lasers, and pulsed hair removal lasers. Facial skin resurfacing (CO₂, erbium, and YSGG) procedures have a higher risk of scarring because of the need for dermal destruction for intended facial skin improvement. The risk of scarring may be dependent on the laser system used and factors such as the number of pulses delivered. This dreaded complication may occur even at the hands of experienced surgeon and may occur in only a portion of treated area [1].

11.8 Other Complications

11.8.1 Delayed Wound Healing

Delayed healing is sometimes associated with ablative laser treatments such as CO₂, erbium, and YSGG laser resurfacing. It is important to avoid these procedures in patients with history of poor healing (e.g., lupus erythematosus and connective tissue disorders), active infections, or the potential for infections. Low-grade postoperative infection is another source of poor or delayed healing. Once these causes of delayed wound healing are ruled out, wounds are best managed with conservative wound management techniques.

11.8.2 Wound Infection

Wound infection is most common with laser skin resurfacing procedures. Superficial viral, bacterial, and fungal infections are possible. Anti-hepatic prophylaxis is recommended for all perioral, full facial laser resurfacing, and hair removal procedures. Bacterial infections are typically caused by *Staphylococcus aureus* and *Pseudomonas* organisms. Candidal infections may occur after facial resurfacing procedures. Prolonged occlusive dressing may contribute to increased incidence of wound infection.

11.9 Darkening of Cosmetic Tattoos

Darkening of flesh-colored cosmetic tattoos was first reported with the use of Q-switched ruby laser. The change in the color of tattoo ink is attributed to laser-induced conversion of ferric oxide to ferrous oxide in cosmetic tattoo ink pigments, producing an insoluble black pigmentation within the skin. This undesirable side effect was also reported with the use of Q-switched Nd:YAG, Q-switched alexandrite, and 510-nm pulsed dye lasers. A careful history of cosmetic facial tattoos and avoidance of area of facial tattoos with laser treatment are prudent, since the changes are permanent in nature [12].

11.10 Chrysiasis After Laser Treatment

Persistent hyperpigmentation is noted with Q-switched laser treatments, in patients who received gold therapy for other dermatologic conditions. The hyperpigmentation is thought to be related to alteration of gold particles, already present in the skin [13].

11.11 Allergic Reactions After Laser Treatment Of Tattoos

Allergic reactions, including anaphylaxis, have been reported with Q-switched laser treatment of tattoos and are thought to be secondary to alteration of altered antigenicity of the tattoo pigment by the laser irradiation [14].

11.12 Postoperative Erythema

Some degree of postprocedure redness is present and transient in nature with all laser procedures. Prolonged erythema is present after all ablative laser facial skin resurfacing procedures. Duration of redness depends on the depth and degree of dermal wounding. Erbium and Er:YSGG laser resurfacing typically produce less redness compared to CO₂ laser treatment. This may be a func-



Fig. 11.2 Area of transient redness after laser hair removal (Courtesy of Dr. Elie M. Ferneini)

tion of more superficial nature of the treatment and also attributed to lower degree of residual thermal necrosis of dermis with erbium laser treatment. It is important to rule out infection as a contributing factor for prolonged erythema [1] (Fig. 11.2).

11.13 Rhabdomyolysis

Rhabdomyolysis has been reported after laser-assisted liposuction by Shin and Chang [1, 15].

11.14 Strategies to Prevent Complications Following Specific Cosmetic Laser Procedures

Discussion of techniques of various laser treatments and protocols is beyond the scope of this chapter. However, strategies to prevent complications following specific facial cosmetic laser procedure are discussed below. Selecting the appropriate laser technology platform for the chromophore that is being targeted is the first step in preventing complications. The use of appropriate settings and use of test patch treatments to choose appropriate fluence, pulse duration for an individual patient is important, especially when treating patients with darker skin color (skin types IV–VI). Perioperative skin care program with 4% hydroquinone and topical retinoid may decrease undesirable pigimentary changes of skin. Patients with fresh tans should not be treated when melanin is the target chromophore.

11.14.1 Hair Removal Laser

The most commonly used hair removal platforms include long-pulse alexandrite (755 nm), the diode laser (810 nm), and long-pulse Nd:YAG laser (1064 nm). Alexandrite and diode lasers can be safely used in skin types III and IV. Nd:YAG laser is the treatment of choice for skin types IV–VI. Multiple treatment sessions will be necessary to effectively reduce hair growth and to maintain hair reduction. Treatment sessions should be scheduled 4–6 weeks apart to accommodate hair reentry in to anagen phase. There are some reports of increased hair growth after laser hair removal treatment. Treatment should be discontinued if patient reports increased hair growth.

In darker skin-type patients, there is significant amount of melanin present in the epidermal layer. Long-pulse Nd:YAG laser and epidermal cooling devices help to protect melanin for safe and effective laser hair removal treatment [16].

11.14.2 Vascular Lesions and Facial Erythema

Facial erythema and telangiectasia of facial skin remain one of the most common complaints of cosmetic patients. Variable and long pulse, pulse dye lasers (585–595 nm), alexandrite (755 nm), diode lasers (810 nm), long-pulse Nd:YAG lasers (1064 nm), and IPL treatments are all useful to target oxyhemoglobin to treat vascular lesions of the face and neck. Selection of appropriate fluence and pulse width, with test spot treatment, can minimize complications [16].

11.14.3 Pigment and Tattoo Removal

Pigmented lesions need to be carefully evaluated, prior to laser treatment, and a biopsy should be obtained of any suspicious lesions. They can be safely treated with Q-switched lasers, which deliver high-intensity energy in a very short-pulse duration, targeting individual melanocytes. Recently introduced ultrashort-pulse lasers with picosecond pulse duration are effective to treat

tattoos. Despite increased safety, multiple treatments [1, 8–10, 13] are needed for satisfactory removal of pigment. Adverse effects include scarring and dyspigmentation, particularly in darker skin-type patients. Despite all the precautions and treatments, tattoos may never be removed completely, and the patient should be aware of this fact as part of informed consent [16].

11.14.4 Laser Facial Skin Resurfacing

Since introduction of concept of fractional photothermolysis for cutaneous remodeling, by Manstein D, Herron GS, Sink RK et al., fractional facial skin resurfacing has become an essential component of facial aesthetic treatments [17]. Fractional skin resurfacing can be classified into non-ablative and ablative treatments. Current ablative fractional laser treatment systems are based on CO₂, Er:YAG, YSGG, and Nd:YAG laser platforms [18]. Indications for fractional facial resurfacing include photoaging, acne and facial scarring, and pigmentary disorders.

A careful history of prior laser procedures and responses, any history of delayed healing and hypertrophic scar formation, and history of herpes simplex viral infections, immune-compromised status, and level of anxiety are useful in customizing the treatment.

Patients with history of connective tissue diseases, diabetes, and drug, alcohol, and tobacco abuse should be treated cautiously. Patients with a personal and family history of vitiligo should not be treated with facial resurfacing procedures. Patients who have used isotretinoin in the past 6 months–1 year are at increased risk for impaired healing. Multiple non-ablative treatments are necessary to achieve the results of ablative facial resurfacing procedure. However, the ablative procedure has higher wound healing risk profile and significant downtime for wound healing [18].

All patients should receive HSV prophylaxis with acyclovir 400 mg, three times a day or valacyclovir 500 mg, twice a day for 7–10 days, starting on the day of procedure. Perioperative antibiotic coverage is debatable, given the low incidence of bacterial and fungal infections [18]. Darker skin-

type patients and patients with history of melasma can be pretreated with 4% hydroquinone and perioperative sunscreen, and sun protection is useful in avoiding pigment-related complications [18].

11.14.4.1 Complications

Complications of ablative fractional resurfacing are lower than traditional ablative facial resurfacing. Complications include infections, acneform eruptions, prolonged erythema, change in pigmentation, and scarring.

11.14.4.2 Infections

As discussed previously, the most common infections following fractional ablative resurfacing of the face are herpes simplex viral eruptions, with an incidence ranging from 0.3% to 2% (compared to 2–7% with traditional ablative resurfacing) [10, 19]. The incidence of bacterial infections with fractional resurfacing procedures is extremely low (0.1%) in all treated cases. Primary bacteria isolated include *Staphylococcus aureus* and *Pseudomonas aeruginosa*. A case of *Mycobacterium chelonae* infection has been reported. Given the prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA), special attention with wound culture and treatment is warranted if the wound fails to improve as expected [19] (Fig. 11.3).

11.14.4.3 Acneform Eruptions

The incidence of acneform eruptions is lower with fractional resurfacing (2–10%). The incidence of milia has been reported in up to 19% of



Fig. 11.3 Herpes infection after laser resurfacing (With permission from Pozner et al: Laser Resurfacing: Full Field and Fractional, Clin Plastic Surg 43:515–525, 2016)

treated cases. Occlusive dressings and moisturizers can exacerbate acne eruptions. A short course of oral tetracycline antibiotic is helpful for treatment and to prevent future breakouts [20].

11.14.4.4 Prolonged Erythema

Prolonged erythema is defined as posttreatment redness which persists longer than 4 days after non-ablative resurfacing and beyond 1 month, with ablative treatment. The incidence of prolonged erythema is less than 1% with non-ablative procedures and more than 12.5% with ablative procedures. The redness is associated with heat shock protein persistence indicating new collagen formation and remodeling [19].

11.14.4.5 Change in Pigmentation

Patients with darker skin types (Fitzpatrick III–VI) have higher incidence of post-inflammatory hyperpigmentation following skin resurfacing procedure. However, the incidence is lower with fractional resurfacing compared to traditional resurfacing procedures. High-risk patients should avoid sun exposure for 2 weeks

before and after the procedure and apply 4% topical hydroquinone early in the recovery period [21].

11.14.4.6 Scarring

Hypertrophic scarring is an adverse effect of traditional and fractional laser skin resurfacing of face. The use of excessively high-energy densities, technical problem with repeated stamping or scanning in the same area, and treatment of the skin with fewer adnexal structures, such as neck and chest, can lead to hypertrophic scarring. Periorbital, perioral, and mandibular skin are considered scar prone areas and need conservative treatment protocols. If these areas become infected, scarring may occur.

Early treatment of hypertrophic scarring includes use of topical corticosteroids, silicone gel products, intralesional corticosteroid injections, and PDL (pulse dye laser) therapy [22]. Ablative and non-ablative fractional laser treatments can also be used to improve established scars at low-energy and low-density parameters (Fig. 11.4).



Fig. 11.4 Hypertrophic scar after laser resurfacing and after correction with fractional erbium, IPL, and intralesional steroids (With permission from Pozner et al: *Laser*

Resurfacing: Full Field and Fractional, Clin Plastic Surg 43:515–525, 2016)

11.14.4.7 Ectropion

Incidence of ectropion (sclera show of 1 mm or more), following lower eyelid skin laser resurfacing, is reported as 0.3%, with liberal use of lateral canthal suspension to tighten a lax lower lid and use of transconjunctival approach for fat removal [23].

Conclusion

With the increased demand of minimally invasive facial cosmetic procedures, the number and scope of laser treatment has increased. Although rare, complications can occur following the different treatment modalities. Minimizing these complications is important for achieving predictable outcomes. The surgeon must be familiar with strategies to prevent complications following specific facial cosmetic laser procedures. If these complications occur, the surgeon must be able to diagnose and appropriately manage these complications in a timely manner.

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Complications in the Cosmetic Use of Botulinum Toxin Type A: Prevention and Management

12

Whitney Florin and Jacob Haiavy

Abstract

Botulinum toxin type A (BTX-A) has become the most frequently requested nonsurgical procedure performed around the world. The use of BTX-A in cosmetics has expanded greatly since its approval by the Food and Drug Administration for reduction of glabellar rhytids. Experienced clinicians have been using BTX-A in other areas off-label to improve the appearance of rhytids and facial harmony. Adverse effects associated with BTX-A are rare and tend to be mild and temporary, such as bruising, swelling, and pain at injection sites. More serious complications, such as brow ptosis or eyelid ptosis, can occur. These complications can be minimized by a thorough understanding of the anatomy of the face and careful injection technique, which will be outlined in this article.

12.1 Introduction

Photoaging, volume loss, and rhytids all contribute to the stigmata of the aging face. Nonsurgical treatment of the aging face is most effective when it addresses all contributing factors. Hyperdynamic rhytids, most notable in the upper third of the face, contribute to the appearance of the aging face. Over time, contraction of the muscles of facial expression can contribute to dermal atrophy and lead to static rhytids [1–3].

Botulinum toxin type A (BTX-A) has become the most frequently requested nonsurgical procedure performed around the world [4–6]. In 2015, there were 6.7 million treatments with BTX-A performed, which is up 1% from 2014, and up 759% from 2000 [5]. BTX-A was initially

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approved by the US Food and Drug Administration (FDA) in 1989 for the treatment of strabismus and blepharospasm associated with dystonia [4, 6, 7]. In 1992, Carruthers and Carruthers published their findings that patients injected with BTX-A for blepharospasm also had improvement in the appearance of glabellar rhytids [8]. In 2002, BTX-A was approved for the temporary improvement in the appearance of moderate to severe glabellar lines in adult patients [4, 6, 7]. Since then, BTX-A has been widely used for the treatment of glabellar rhytids as well as other off-label locations in the head and neck. Currently, BTX-A is FDA approved for a wide variety of conditions, including glabellar rhytids, migraine headaches, hyperhidrosis, strabismus, hemifacial spasm, blepharospasm, cervical dystonia, and upper limb spasticity [4, 6, 7, 9].

There are three formulations of BTX-A that are FDA approved for cosmetic use and available commercially: onabotulinum-toxin A (Botox; Allergan Inc), abobotulinum-toxin A (Dysport; Galderma Laboratories), and incobotulinum-toxin A (Xeomin; Merz Pharmaceuticals, LLC). These formulations are FDA approved for the temporary improvement in the appearance of glabellar rhytids in patients aged 18–65 [9–11]. Onabotulinum-toxin A (Botox) is the only product that is also FDA approved for the treatment of lateral canthal lines [9]. It should be noted that all other uses of these formulations for cosmetic purposes are considered off-label. The effects of BTX-A generally last 3–6 months [9–11].

There are variations between these three products in their potency, onset time, and duration [12]. The unit dosing for all three formulations are not interchangeable, with the most notable difference between Dysport and the other two formulations (Botox and Xeomin) [9–11]. Botox and Xeomin are dosed in comparable unit values, whereas Dysport requires 2.5–3 times of its own unit value (Speywood units) to achieve the same result [9–11]. It should be noted that the recommended doses of BTX-A listed in this article will apply to units of Botox and Xeomin. The clinician should make the appropriate adjustment in the number of units used for Dysport.

12.2 Patient Selection and Education

Patient selection and patient education are important factors in avoiding complications with neurotoxin use. There are few contraindications to the use of BTX-A. Patients with neuromuscular disorders, such as myasthenia gravis, amyotrophic lateral sclerosis, Eaton-Lambert syndrome, and motor neuron diseases can be worsened by administration of BTX-A [9–11]. Certain medications (i.e., some antibiotics such as aminoglycosides, calcium channel blockers, neuromuscular-blocking agents, anticholinesterases) can also interfere with neuromuscular transmission and should be avoided as they can enhance the paralytic effect of BTX-A [13, 14].

Relative contraindications include anticoagulant use. If possible, patients are asked to discontinue aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs) 7–10 days prior to treatment to avoid excessive bruising [1]. BTX-A is a category C medication for pregnancy and lactation and should be avoided in patients who are pregnant or actively breast-feeding [9–11].

It is important to educate patients how neurotoxins work. Many patients do not understand how neurotoxins work, how they differ from fillers, and what are the limitations of these modalities. In our practice, patients are asked to point out in a mirror which lines bother them. The practitioner can evaluate which of these areas can be improved with neurotoxin use, specifically dynamic versus resting lines. Patients should be educated that neuromodulators will improve dynamic lines in the forehead, glabella, and lateral canthal region mainly. However, static lines, which are made worse by the actions of muscles of facial expression, will not be treated entirely by neurotoxin use. These lines should also be addressed with skin resurfacing or volume replacement (i.e., fillers or fat transfer) [1, 3, 4].

Any asymmetries should be pointed out to the patient and documented in the medical record. New patients should be photographed at rest and in animation prior to treatment. In addition, they should be encouraged to return to the clinic at 2 weeks posttreatment for an evaluation for the

need for touch-up, as well as for photographs. This allows the injector to determine the number of units and location of injections that work best for that particular patient next time. There is a range of units of BTX-A recommended; however, this must be tailored for each patient to improve outcomes and patient satisfaction. This 2-week follow-up can be particularly useful for the novice injector.

12.3 Safety

While patients must be informed about potential adverse effects of BTX-A, patients should be reassured of the long history of safety [1, 2, 4, 15]. Adverse effects associated with BTX-A are rare and tend to be mild and temporary. Common side effects include bruising, swelling, pain around the injection site, headache, and flu-like symptoms [1, 2, 4, 15]. More serious complications, such as brow ptosis or eyelid ptosis, can occur and can be quite disconcerting to both the patient and the practitioner [1, 2, 4]. These complications can be minimized by a thorough understanding of the anatomy of the face and careful injection technique, which will be outlined in this article [1, 2, 4].

The likelihood of bruising can be decreased by advising patients to avoid anticoagulants 7–10 days prior to treatment, if possible [1, 2]. Prominent veins should be noted and avoided to minimize excessive bruising. Vein illumination devices can be helpful in identifying smaller veins, especially in patients with dark complexion.

12.4 Injection Technique for the Upper Face

12.4.1 Glabella

The glabellar complex is responsible for vertical frown lines, which is the most common site for BTX-A injections. Contraction of corrugators, depressor supercillii, and medial orbicularis oculi produces the vertical dynamic lines between the

eyebrows, known as “11’s.” The procerus and depressor supercillii produce the horizontal lines over the bridge of the nose. Together, these frown lines can cause a person to look angry or worried and can project advanced age [1, 2]. The product inserts for Botox injections recommend five injection points (one in the procerus and two in the corrugators bilaterally) (Fig. 12.1) [9]. Some injectors prefer more injection sites along the length of the corrugator supercillii (Fig. 12.2). The recommended dose for the glabellar complex is approximately 20 units. However, most clinicians will tailor the injection technique depending on the gender of the patient, the strength of the muscles, the pattern of rhytids, and the symmetry. Women typically need a lower dose of neurotoxin than men [2]. It is also important to understand if a patient wants to soften the movement of these muscles or eliminate movement of the muscles. Most patients are moving away from the “frozen” look and do want to be able to use these muscles for expression [1, 2].

Care must be taken to inject at least 1 cm from the superior orbital rim and to inject perpendicular to the belly of the muscle. Diffusion of the neurotoxin can cause paralysis of the levator palpebrae superioris, which can lead to upper eyelid ptosis [2, 13, 14, 16]. The abobotulinum-toxin A



Fig. 12.1 Typical sites for five injection points to glabella



Fig. 12.2 Typical sites for seven injection points to glabella

(Dysport; Galderma Laboratories) has been shown to have a wider dispersion radius than the other BTX-A formulations. It is therefore this author's opinion that the clinician should be even more cautious with abobotulinum-toxin A in the lateral brow region especially in older individuals with existing brow ptosis and aging [17]. Ptosis can be managed with apraclonidine 0.5% eye drops (three times per day) until symptoms resolve [13, 14, 16]. Apraclonidine is an alpha-2-adrenergic agonist, which stimulates Mueller's muscle and can lead to a 2 mm elevation of the ptotic eyelid [1].

12.4.2 Forehead

The frontalis muscle elevates the brow and leads to horizontal forehead rhytids. The frontalis is a large, vertically oriented muscle with significant variation between individuals. While it is usually depicted as two fan-shaped bands, there may be overlap of midline fibers. Those that have a central tendinous attachment may not need midline injections. The forehead shape varies in both the horizontal and vertical direction [1, 2]. The rhytid pattern also varies, with some patients having

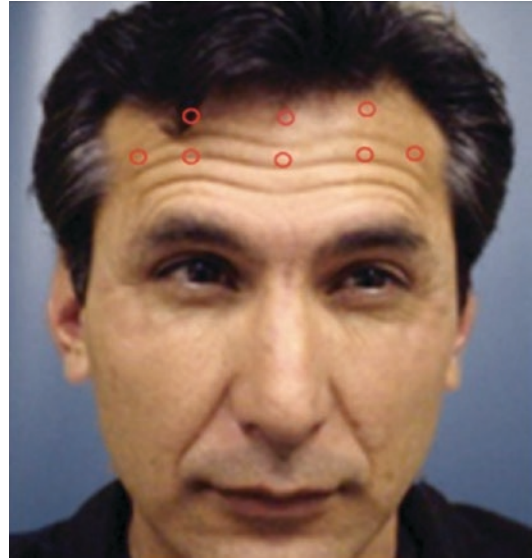


Fig. 12.3 Typical sites for injections to the frontalis muscle. Injections should be 2.5–3 cm above the orbital rim. A total of 10–25 units can be divided into 4–10 injection points based on the pattern of rhytids

numerous fine lines whereas others have one or two deep lines. These anatomic variations, as well as the amount of movement the patient desires, affect the injection pattern and dose of neurotoxin used.

In general, the goal of treatment should be to soften the forehead lines while maintaining expressiveness and avoiding brow ptosis. Some recommend injecting above the midline of the forehead to avoid brow ptosis. Others site a safe zone 2.5–3 cm above the orbital rim. Typically the glabella is injected along with the forehead for a harmonious effect. Typical dosing for the forehead is between 10 and 25 units, which can be divided into 4–10 injection points depending on the rhytid pattern (Fig. 12.3) [1, 2, 6].

Patients may return complaining of asymmetry or peaking of the lateral brow after BTX-A injections to the forehead (Fig. 12.4). A thorough preoperative evaluation of eyebrow position and movement may help avoid asymmetries following neurotoxin use. Placing injection sites lateral and high enough on the forehead can minimize peaking of the lateral brow and at the same time avoid brow ptosis [1, 2]. These situations can be easily managed at the follow-up visit by injecting



Fig. 12.4 Peaked lateral brows can occur after neurotoxin injection to the frontalis. This woman presented with peaked lateral brows 2 weeks after injection of 15 units to the frontalis muscle

small amounts of neurotoxin in the area with too much movement. For lateral peaking, 2–3 units of BTX-A can be injected in the lateral forehead. It is advisable to wait 2 weeks after the initial injection before doing a touch-up, as there may be variation in how quickly muscles respond to the neurotoxin [2, 9]. It is important to document all touch-ups so that the following treatment can be tailored accordingly. It is a good idea to mention to patients prior to injecting them that small corrections may be needed at the follow-up visit.

12.4.3 Brow Elevation

The resting position of the brow is dependent upon the balance between the depressors of the brow and the elevator of the brow (the frontalis). Injection of neurotoxin in the glabellar complex can produce up to a 2 mm medial brow elevation. Injecting the superolateral orbicularis oculi immediately below the desired level of elevation can lead to lateral brow elevation. This injection should be superficial. Injecting too high in this area can lead to spread of neurotoxin to the frontalis which may lead to paradoxical brow ptosis.



Fig. 12.5 Typical sites for injection to orbicularis oculi (crow's feet). A total dose of 10–30 units can be used, divided into 2–5 points per side. Injection points should remain 1–1.5 cm from the orbital rim and 1.5 cm from the lateral canthus

Injecting too close to the orbital rim may lead to unwanted spread of neurotoxin which can lead to upper eyelid ptosis [1, 2]. Typically women will prefer greater brow elevation than men [2].

12.4.4 Crow's Feet

Lateral orbital rhytids (“crow’s feet”) result from contraction of the orbicularis oculi as well as photoaging. The orbicularis oculi is a sphincter-like muscle, which encircles the eye. Smiling or squinting causes contraction of the muscle and over time, can lead to rhytids which extend radially from the lateral canthus. The injection technique in this region should match the pattern of wrinkles and should remain superficial, producing a characteristic wheal. A total dose of 10–30 units can be used, divided into 2–5 points per side (Fig. 12.5) [1, 2]. Typically men will tolerate more smile lines than women [2].

Injection points should remain 1–1.5 cm from the orbital rim and 1.5 cm from the lateral canthus to avoid unwanted spread of neurotoxin [1, 2, 18]. Spread into the orbit can weaken the lateral rectus or inferior rectus leading to diplopia



Fig. 12.6 Significant bruising after BTX-A injection to the crow's feet

[18]. Practitioners must be careful not to inject too far inferiorly. Inadvertent injection of the zygomaticus major muscle can affect upper lip tone and lead to lip asymmetry [13, 14, 16].

Lower-eyelid lid laxity should be evaluated with a snap test prior to injection. Those with laxity have increased risk of ectropion; therefore, injections should not be placed too medial or too inferior [2]. In general, injections should not be placed medial to the mid-pupillary line as there is increased risk of ectropion and epiphora [19]. There tends to be small, tortuous veins lateral to the eye, so bruising may occur with these injections (Fig. 12.6). A vein illuminator device may be helpful in avoiding veins. Ice before and after injections may also help to minimize bruising.

12.5 Bunny Lines

“Bunny lines” occur from animation of the periocular region and wrinkling of the nose. Contraction of the transverse portion of the nasalis muscle leads to wrinkling on the lateral nasal sidewall [1, 2]. The nasalis muscle originates from the maxilla and runs diagonally across the bridge of the nose [2]. It is important to differentiate bunny lines from transverse lines resulting from contraction of the procerus muscle as the injection technique is different [2].



Fig. 12.7 Typical appearance of bunny lines, caused by contraction of the nasalis. One to four units is injected superficially per side

A low dose (1–4 units) can be injected per side to target the transverse portion of the nasalis (Fig. 12.7). Injections should be superficial as this is a vascular area [2]. Care must be taken to palpate and avoid the angular artery, which tracks superiorly along the lateral nose [1]. Injection sites should avoid the levator labii alaeque nasi and levator labii superioris to prevent drooping of the upper lip. Injection sites should not be vigorously massaged in a downward direction as this may also cause unwanted spread of the neurotoxin [2].

12.6 Injection Technique for the Perioral Region

Sun damage, smoking, aging, and expression can lead to excessive vertical perioral rhytids. These unesthetic fine lines are best addressed with a combination of neurotoxin, fillers, and skin resurfacing [1–3]. The benefits and limita-

tions of each of these modalities should be thoroughly discussed with the patient. Neurotoxin use in the perioral region should be reserved for experienced practitioners as there is a complex interaction of muscles, specifically the orbicularis oris, depressor anguli oris, and mentalis [2].

12.6.1 Orbicularis Oris

The orbicularis oris is a sphincter-like muscle, which encircles the lips and contributes to the formation of perioral rhytids. Injection sites should be superficial and symmetric. Injection sites are typically along the upper and lower lip, with at least one site per quadrant of the mouth. The midline should be avoided as it may lead to flattening of the lip. The total dose for both lips is approximately 5–6 units with each injection point containing 0.5–2 units (Fig. 12.8). Injections can be placed just above the vermilion border or a few millimeters above the border. Injections that are too high can cause unwanted effects on the upper lip, such as eversion, inversion, or ptosis. Ice and/or topical numbing cream can be used for pain control [2].

Overtreatment of the perioral region can lead to serious dysfunction, such as difficulty with speech (“b” and “p” sounds), eating, drinking through a straw, pursing the lips, brushing the teeth, as well as decreased proprioception. Patient selection and education is paramount. Individuals who rely on their lips for their profession (i.e., musicians, singers, public speakers) are not good candidates for



Fig. 12.8 Typical injection sites for the orbicularis oris. The total dose is approximately 5–6 units with each injection point containing 0.5–2 units

perioral neurotoxin injections. Treatment should be conservative and cautious [2].

12.6.2 Depressor Anguli Oris

The depressor anguli oris (DAO) originates from the mandible and inserts into the angle of the mouth. The DAO pulls the corner of the mouth down and back, which contributes to a frowning appearance and the “marionette lines.”

The DAO should be injected with low doses (2–5 units per side), typically with two injection sites per side (Fig. 12.9). If injection of the DAO is too close to the mouth, it can lead to unwanted spread of neurotoxin leading to oral incompetence, an asymmetric smile, and drooling (Fig. 12.10) [2].

12.6.3 Mentalis Muscle

The mentalis muscle is a paired muscle in the midline of the chin. It originates from the man-



Fig. 12.9 Typical injection sites for the depressor anguli oris. A total of 2–5 units is injected per side



Fig. 12.10 An asymmetric smile is a potential complication that can occur if injections of the depressor anguli oris are too close to the mouth. This woman presented 2 weeks after 5 units were injected to mentalis and 2.5 units to each depressor anguli oris

dible, covers the chin, and inserts into the skin below the lower lip. Contraction of the mentalis raises the level of chin and everts the lower lip. Contraction of the muscle, along with loss of collagen and subcutaneous fat, causes the *peau d'orange* (dimpling) of the chin. While the mentalis is a paired muscle, only one central injection with neurotoxin is needed. The injection should be in the midline of the mentalis, angled upward. The muscle should be massaged laterally. The typical starting dose of BTX-A is 4–6 units but can be as much as 10–12 units [2].

Care should be taken to avoid injecting too high on the chin as this can affect the orbicularis oris, leading to lip incompetence and drooling. Avoid injecting the depressor labii as this can cause the lower lip to droop. Some patients with a dimpled chin may have a hypertrophic mentalis muscle in the setting of retrogenia and oral incompetence. These individuals should not be treated with neurotoxin as it may worsen the oral incompetence [2].

Some patients are unaware of chin dimpling, which typically appears during speech and with

animation. This can be demonstrated with a hand mirror. Women are treated more often than men as their chins are more likely to develop dimpling. Treatment of this region can be beneficial in those undergoing a chin implant [2].

12.7 Injection Technique for the Neck

12.7.1 Platysma Bands

Platysma bands in the neck may become prominent with age or after rhytidectomy. BTX-A can be a useful way to soften the bands in patients with adequate skin elasticity and minimal submental fat. The platysma depresses the mandible, everts the lower lip, and pulls the corners of the mouth down and back. Injection sites are placed approximately 1 cm apart along the band, with 3–5 sites per band (Fig. 12.11). The total dose ranges from 20 to 30 units depending on how many bands are treated and the number of injection sites per band. The patient should be asked to contract and the practitioner should grasp the band with the nondominant hand while injecting neurotoxin directly into the belly of the muscle. This technique avoids unwanted diffusion of neurotoxin into strap muscles which



Fig. 12.11 Typical sites for injections to platysma bands. Injection sites are placed approximately 1 cm apart along the band, with 3–5 sites per band, for a total of 20–30 units

can lead to neck weakness, dysphagia, and dysphonia [2, 19].

Patient selection is important as this works best in younger patients with good skin elasticity. Patient's expectations should be reasonable as platysma band injection is not a substitute for surgery and will not improve skin elasticity. Platysma band injection can be useful approximately 2 weeks before liposuction of the submental region or before skin resurfacing. Platysma band injection can be combined with injection of the DAO and fillers to restore volume in the perioral region [2, 19].

Conclusion

While not true complications, there are unwanted sequelae of neurotoxin use, such as swelling, injection site pain, needle marks, mild erythema, bruising, and rarely hematoma [1, 9–11, 13, 18]. Earlier onset of action in one anatomic site occurs commonly and is not a true complication [1]. This may manifest as an asymmetry in brow movement and patients should be warned that this might happen. It is best to wait 2 weeks for neurotoxin to take full effect before considering a touch-up, as minor discrepancies may correct themselves as the neurotoxin takes effect [9, 10].

Pain at injection sites can be minimized by using smaller-gauged needles. Ice or topical numbing cream can be used. Bleeding and bruising can be minimized by holding anticoagulation 7–10 days prior to the procedure, icing immediately before injection, avoiding vessels, and injecting superficially. Bleeding can be managed by holding pressure and applying ice.

The most disconcerting complications are unwanted spread of neurotoxin and neuromuscular blockade of unintended muscles. For example, injection too low on the forehead may lead to brow ptosis. Injection of the glabella, which is too close to the orbital rim, may lead to upper eyelid ptosis. Injection of the crow's feet, which is too close to the eye, may result in diplopia. Injection of the crow's feet too low on the cheek can lead to upper lip asymmetry. Injection in the perioral region can lead to oral incompetence, difficulty with

speech and eating, lip asymmetry, and drooling. Injection of the platysma bands can lead to dysphonia, dysphagia, and neck weakness [1, 2, 13, 14, 16, 19]. In general, such complications are rare and can be avoided by respecting the anatomy of the face and neck and injecting with care.

The use of botulinum toxin type A in cosmetics has expanded greatly since its approval by the Food and Drug Administration (FDA) for reduction of glabellar rhytids. Botox is also approved for lateral canthal lines [9]. Experienced clinicians have been using BTX-A in other areas off-label to improve the appearance of rhytids and facial harmony.

The recommendations and diagrams presented in this article should serve only as general guidelines. Treatment planning must be tailored to individual variation. Thorough preoperative evaluation of the muscles of facial expression and rhytid pattern as well as careful injection technique can avoid most complications associated with neurotoxin use. Understanding the patient's goals of treatment and pointing out any limitations will improve outcomes and patient satisfaction.

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Soft Tissue Fillers and an Overview of Their Potential Complications

13

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Abstract

Minor facial cosmetic surgical procedures, such as soft tissue fillers, are becoming more popular in maxillofacial cosmetic surgery. Their ease of use and relative safety profile make them a valuable cosmetic treatment option. Side effects, although uncommon, present a challenge for management. This chapter will discuss the range of complications associated with facial soft tissue fillers. Diagnosis, management, and prevention of these adverse events will also be discussed.

13.1 Introduction

In recent years, cosmetic facial surgery has emerged to meet the demand for nonsurgical and surgical procedures designed to overcome the

effects of aging, scars, and skin defects. Often marketed as low-cost, low-risk procedures, facial soft tissue augmentation through the use of injectable dermal fillers provides a predictable and safe solution. These procedures are popular and safe compared to more invasive surgical treatments. The American Society for Aesthetic Plastic Surgery reports a 22% increase in the number of nonsurgical procedures from 2014 to 2015 with a 44% increase since 2011 [1]. Given the anticipated growth of these procedures, the number of complications will also likely increase. The majority of complications related to filler injections are transient and resolve without further treatment. Serious complication rates are relatively low; however, the competent surgeon should understand facial anatomy, dermal filler properties, injection techniques, and side effects. This chapter will identify potential complications of soft tissue fillers as a guide to diagnosis and management.

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13.2 Filler Types

Many complications of soft tissue fillers are a result of their physical and chemical properties. Knowledge of these properties can help avoid unintended consequences. The section which follows discusses the most common soft tissue fillers—both temporary and permanent.

13.2.1 Temporary (Biodegradable) Fillers

13.2.1.1 Hyaluronic Acid

Due to its efficacy, safety, biocompatibility, and reversibility, hyaluronic acid (HA) remains the hallmark for soft tissue filler augmentation. A polysaccharide of the human extracellular matrix, HA forms a scaffold for collagen and elastin. Its chemical and physical properties make it ideal for soft tissue augmentation. Known for its moisturizing characteristics, HA is a hydrophilic molecule composed of linear polymeric dimers organized in long unbranched chains. A higher degree of cross-linking increases both viscosity and elasticity. More cross-linking increases the resistance of HA to degradation, thereby extending its longevity [2]. HA fillers typically last from 6 to 8 months depending on the extent of cross-linking, concentration, uniformity, and size.

13.2.1.2 Collagen Fillers, Autologous Fat, and Calcium Hydroxylapatite

Collagen, autologous fat, and calcium hydroxylapatite are also naturally produced substances that can be manipulated to augment soft tissue. Collagen of bovine, human, or porcine origin can correct fine lines but has less predictable results than HA. Similarly autologous fat has unpredictable results and requires an operating room setting and a donor site. Calcium hydroxylapatite works by producing an inflammatory response which stimulates the deposition of collagen [2].

13.2.2 Permanent (Nonbiodegradable) Fillers

Permanent (or nonbiodegradable fillers) harness the bodies' immunologic defense system to produce desired results. Examples include polymethyl-methacrylate (PMMA) microspheres, polyacrylamide hydrogel, and silicone oil. These fillers work by producing a foreign body reaction. The fibroblasts of the host cell deposit collagen and fibrin around the nonabsorbable microspheres [3]. For example, over the course of 1–3 months, the collagen of PMMA (80% bovine collagen, 20% PMMA microspheres) dissolves leaving only the microspheres surrounded by fibrin [3]. Silicone functions in a similar way with collagen surrounding silicone particles. Although effective as dermal fillers, the complications of these fillers tend to be permanent, longer lasting, or appear years later.

13.3 Mild Complications

As with any surgical procedure, there are risks and benefits. Complications with soft tissue fillers range from mild and transient to long lasting and permanent. These complications can leave patients with both physical and emotional scars.

13.3.1 Hypersensitivity Reactions: Ecchymosis, Erythema, and Edema

Ecchymosis—Ecchymosis or bruising is the result of local blood vessel trauma during injection. Bruising is often observed following injection in the dermal and immediate subdermal planes and is far less common in the preperiosteal levels [2, 3]. The best way to avoid this skin reaction is a thorough medical history with attention to substances affecting platelet aggregation (warfarin, aspirin, clopidogrel, NSAIDs, herbal supplements) [4]. Technique and surgeon's experience can also affect the clinical outcomes. The use of small gauge needles, slow injection techniques and volumes, and blunt cannulas can

ameliorate bruising. Ecchymosis may be unavoidable in areas of thin tissue such as the eyelids or lips [5]. Recommended treatment includes cold compresses, ointments with heparinoid or vitamin K, and in rare instances pulsed dye light or potassium titanyl phosphate lasers [2].

Erythema—Post-procedural erythema represents either a mild, transient reaction or conversely a potentially more serious complication. Redness often signals injections which are too superficial and marks a minor inflammatory response [6]. Persistence of erythema can indicate a serious hypersensitivity reaction, while significant erythema could represent necrosis or infection. Erythema from an allergic reaction—although unlikely—is normally accompanied by significant swelling, pruritus, and pain [5].

Edema—Edema is an expected consequence of filler injection. Short-term posttraumatic edema itself is a product of the hydrophilic nature of many types of filler. The response normally resolves within the first 36 to 48 h. Surgeons and patients can manage the edema with cold compresses, pressure, and observation. Long-term (delayed) edema is likely a result of one of two biological responses: antibody-mediated edema or non-antibody-mediated edema. Angioedema (a type of antibody-mediated edema) develops as

immunoglobulin E (Ig-E) and mediates a hypersensitivity reaction [2]. In contrast, a non-antibody-mediated edema is a product of T lymphocytes and occurs within 24 h after injection. The treatment of both reactions depends on the types of filler used as well as the magnitude and severity of reaction.

Although relatively uncommon, virtually any non-autogenous filler material may cause a hypersensitivity reaction. As described above, these most commonly present as ecchymosis, erythema, edema, and/or induration at or near the site of filler injection [7]. In one published case report, a patient developed symptoms of an allergic reaction following injection of Restylane-L, a common HA filler, into the patient's nasolabial folds. Induration at the site of injection along with periorbital edema and erythema developed several days after injection (Figs. 13.1 and 13.2) [8].

Treatment consisted of intravenous dexamethasone and 6-day oral methylprednisolone dose-pack at postinjection day 5. The patient was again seen on postinjection day 7 with continued periorbital edema and erythema. Induration also developed at the site of filler injection. At this visit, the patient received hyaluronidase injection for reversal.

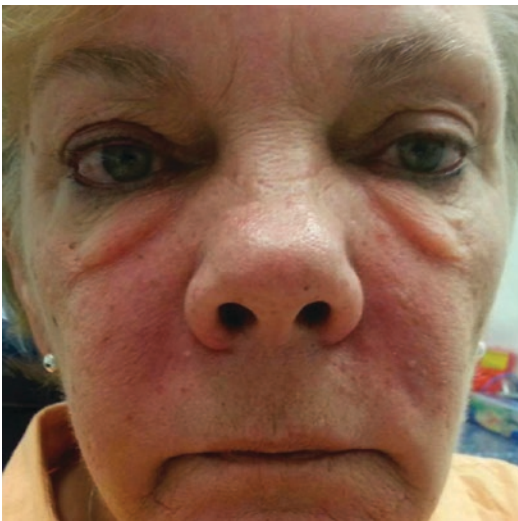


Fig. 13.1 Postinjection day 4 [8]



Fig. 13.2 Postinjection day 7 [8]

More reversal agent was given at postinjection days 9 and 10. The patient was observed over the next several weeks with significant symptom improvement (Figs. 13.3 and 13.4) [8].

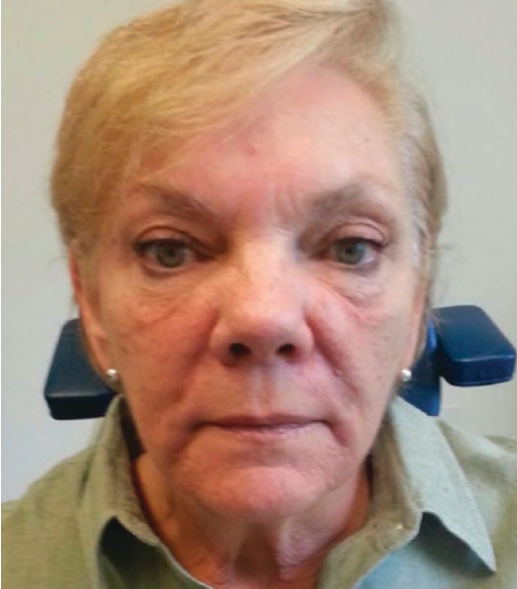


Fig. 13.3 2 weeks after injection with hyaluronidase [8]

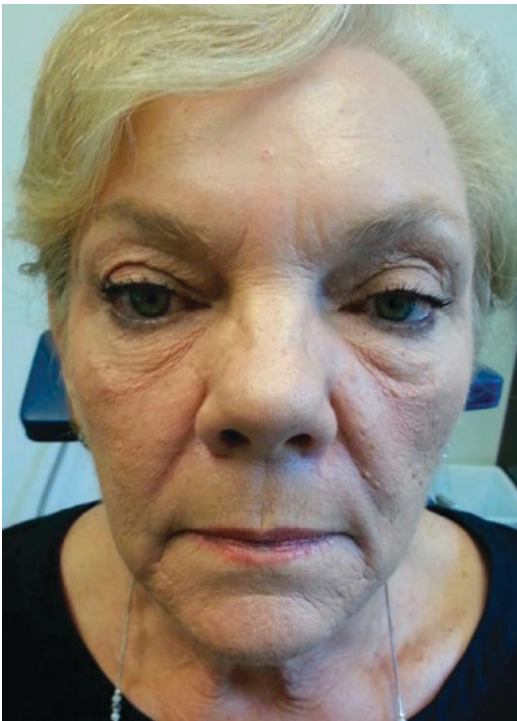


Fig. 13.4 4 weeks after injection with hyaluronidase [8]

13.3.2 Esthetic Concerns: Clumping, Uneven Product Distribution, and Tyndall Effect

Esthetic complications such as clumping, uneven product distribution, and the Tyndall effect influence patient satisfaction. All of these complications relate to the surgeon's technique and experience. Clumping may indicate a problem with filler consistency, while uneven product distribution may demonstrate improper use of serial puncture, linear threading, fanning, or cross-hatching techniques [9]. The Tyndall effect, an observable bluish hue, results from superficial placement of material in the upper dermis or above.

13.3.3 Infectious Reactions: Bacterial and Viral Causes

Every surgical procedure carries a risk of postoperative infection. While the risk is minor, injectable fillers also share this risk. It is believed that these infections are a result of contamination at the time of injection. This includes improper sterile technique, injecting through glands containing bacteria, fluctuations in the skin's normal immune response, and transient bacteremia at the time of injection. [10] Viral infections and complications related to fillers have also been documented in the literature.

13.3.3.1 Bacterial Infections

Early signs of infection such as erythema, edema, pain, acne formation, or nodule formation may begin as soon as several days after the procedure. Organisms such as *P. acnes* and *S. epidermidis* are the most common bacteria causing low-grade infection due to their colonization of the skin. Abscess formation caused by methicillin-resistant *Staphylococcus aureus* and *Streptococcus* species is less common after filler injection. They typically occur in patients treated for HIV-related lipoatrophy. Often presenting as an erythematous nodule, biofilms present a diagnostic challenge to surgeons. A biofilm is a colony of microorganisms surrounded by a protective matrix. This slowly metabolizing strongly adhesive matrix forms a barrier to the

Table 13.1 Spectrum of activity for oral antibiotics against gram-positive organisms [10]

| | MSSA | MRSA | CoNS | β-strep | P. acnes |
|-----------------------------|------|------|------|---------|----------|
| Penicillin VK | – | – | – | + | + |
| Amoxicillin | – | – | – | + | + |
| Amoxicillin/clavulanic acid | + | – | – | + | + |
| Dicloxacillin | + | – | +/- | +/- | – |
| Cephalexin | + | – | – | + | + |
| Cefpodoxime | +/- | – | – | + | + |
| Moxifloxacin | +/- | – | – | + | – |
| Ciprofloxacin | – | – | – | – | – |
| Azithromycin | – | – | – | +/- | +/- |
| Linezolid | + | + | + | + | + |
| TMP-SMX | + | + | – | – | – |
| Clindamycin | +/- | +/- | +/- | + | + |
| Doxycycline | +/- | +/- | +/- | +/- | +/- |
| Metronidazole | – | – | – | – | – |
| Rifampin ^a | + | + | + | + | +/- |

^aRifampin should always be used as part of combination therapy. MSSA methicillin-sensitive *Staphylococcus aureus*, MRSA methicillin-resistant *Staphylococcus aureus*, CoNS coagulase-negative *Staphylococcus* [10]

immune system and usually cultures negative [11]. Often appearing as tender nodules, biofilms may show up weeks to months after injections. These biofilms are 100 times more resistant to antibiotics and require the use of either hyaluronidase or prolonged use of antibiotics to resolve [3, 11]. The best treatment for bacterial infections related to filler injection is through careful prevention. Proper aseptic technique is critical at all points of the procedure. Therefore, it is essential to disinfect the injection site with a topical disinfectant, such as 70% isopropyl rubbing alcohol, Betadine, or a chlorhexidine-containing solution. Some surgeons will prescribe prophylactic antibiotics, but there is no evidence to support this practice [10]. Below is a list of the common organisms causing injectable filler-related infections and susceptibility to various antibiotics [10] (Table 13.1).

13.3.3.2 Viral Infections

Reactivation of herpes simplex I virus can occur following filler injection in the perioral regions. According to some studies, this virus affects 50% of high socioeconomic patients and can reactivate as a result of procedural massage, stress, or swelling [5, 12]. The characteristic lesions normally appear in the nasal mucosa, mucosa of the hard palate, and perioral area [2].

Pretreatment with acyclovir in patients with a known history is the safest way to avoid this complication.

13.4 Moderate/Severe Complications

13.4.1 Skin Discoloration: Hyperpigmentation, Dyspigmentation, and Hypertrophic Scar

Although considered mild complications by some, due to the psychological effects of skin discoloration, the authors of this chapter consider hyperpigmentation, dyspigmentation, and hypertrophic scars as moderate complications. Hyperpigmentation is observed in all patients regardless of skin type or color and is a result of localized infection or inflammation. As previously discussed, dyspigmentation (or the Tyndall effect) results from fillers placed too superficially and may require a small incision and expression of material for resolution. Hypertrophic scarring, a result of injection or complication, is a rare but serious complication. Its etiology is often difficult to determine and sometimes impossible to avoid.

13.4.2 Foreign Body Granuloma

Inflammatory granulomas are visible immune responses by a host to a foreign body. A product of a type IV hypersensitivity reaction, granulomas are a form of localized nodular inflammation with an incidence rate of 0.1–1% [13]. They may appear months to years after injection as soft and dark purple or red nodules [5]. Perhaps the most effective treatment available at this point is intralesional steroids.

13.4.3 Embolization and Arterial Occlusion

13.4.3.1 Tissue Necrosis

Arterial occlusion resulting in tissue necrosis is most often an immediate consequence of injection into an artery or by arterial compression

related to the mass of nearby filler. Estimated to occur in 0.001% of all filler procedures, this is a rare but potentially very serious complication that can follow injection of fillers, especially in the maxillofacial region. Injecting a filler into an artery can produce an embolism with the potential for either anterograde or retrograde tissue necrosis. The areas of susceptibility are those with no—or little—collateral circulation, depending on a single arterial branch for its blood supply. Individuals with comorbidities contributing to poor circulation are at an elevated risk.

Areas in the maxillofacial region that were found to be at an elevated risk for tissue necrosis are the nose, nasolabial folds, and glabellar region [11]. Caution should be used when injecting near major vessels and foramen such as the angular artery, supratrochlear artery, supraorbital notch and foramen, and infraorbital foramen (Fig. 13.5) [11, 14].

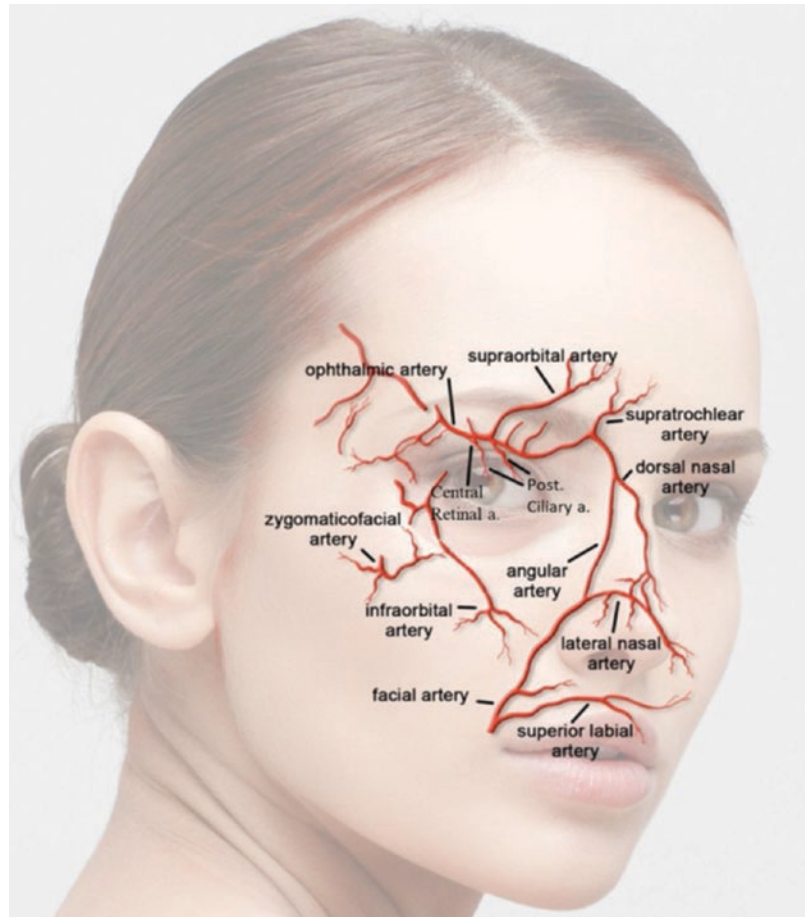


Fig. 13.5 Arterial blood supply. *a* artery; *post* posterior [14]

Clinical warning signs include severe pain either immediately or within hours after injection, skin blanching, and white or violet skin discoloration. Treatment options vary for suspicion of tissue necrosis. Injection of filler should be stopped immediately followed by aspiration if possible. Noninvasive measures such as massage and warm compresses can be tried. Nitroglycerine topical paste and hyaluronidase, if applicable, have also been shown to improve outcomes [14].

13.4.3.2 Retinal Artery Occlusion

Perhaps the most severe complication is retinal artery occlusion with its potential to cause blindness. Occlusion of the retinal artery is often the result of retrograde embolism from an injection into the supraorbital or supratrochlear artery. The best way to avoid this complication is a thorough knowledge of facial anatomy and to inject at the preperiosteal level. Aspiration before injection should always be performed. If the surgeon is concerned about a vascular injection, pressure can be applied to occlude the proximal vessels to the injection site. Symptoms include acute vision loss, severe pain, headache, ptosis, or ophthalmoplegia [14]. Immediate ophthalmology consultation is necessary. Treatment modalities include methods to lower intraocular pressure, induce vasodilation, and ocular massage to propagate the material to a more distal arterial branch in hope of reducing the field of visual loss.

Conclusion

As our patients' acceptance of dermal fillers increases, the number of procedures and subsequent complications will also rise. Although the incidence of these complications varies, it is important to inform our cosmetic patients of the risks and benefits of treatment as well as the severity and magnitude of these risks. Prevention is key to avoiding these consequences. When complications occur, it is

imperative that the surgeon is trained to recognize and be comfortable with managing them.

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Prevention of Complications of Orthognathic Surgery

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Abstract

Orthognathic surgery is a complex operation requiring a sophisticated understanding of facial anatomy as well as an advanced knowledge of the interplay between the associated soft and hard tissues. A thorough analysis of the patient's preoperative form and function along with the mastery of different techniques involved could circumvent many potential complications. Additionally, a thorough and detailed discussion of these complications with the patients and their families could prevent future disappointments or unreasonable expectations. This chapter will review surgical complications involving maxillary and mandibular osteotomies and will discuss different ways to recognize and correct them.

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14.1 Introduction

Orthognathic surgery has a prominent history in maxillofacial surgery with highly favorable outcomes. As with all procedures that are performed, complications can arise, and this chapter will discuss the most common complications that can occur. The surgeon must be vigilant in surgery and expect to encounter the unexpected at times. There are many factors that can contribute to surgical complications including surgical site, surgical approach, duration of surgery, wound contamination, psychology of patient, postoperative care, and the surgeon's skills and ability to recognize and treat complications appropriately [1]. It is always in the surgeon's favor to have a thorough discussion with the patient regarding the most common potential unwanted occurrences during and after surgery. Then these "complications" may be seen as potential sequela

and understood up front by the patient and their family members. If this topic is not discussed beforehand, the patient may potentially see any deviation from what is expected as an error on the surgeon's part. The contents will be divided into maxillary and mandibular procedures with rare complications being discussed for reader familiarity.

14.2 Preoperative Phase

Working as a team with the general dentist and orthodontist is paramount to help prevent miscommunication and operative error. Once the initial orthodontic and surgical analysis has taken place and the surgery(s) indicated are agreed upon, it is important to work with the orthodontist throughout the entire process from start to finish. The patient, as well as a guardian, significant other, and close family members, should be involved and understand the sequenced timing and surgery to be performed to try and prevent patient dissatisfaction. It is imperative the surgeon listen to the patient to understand their concern and treat what the patient sees as an imbalance. If the patient and surgeon cannot agree upon the concern, or have varied opinions, the process should not proceed until each party is satisfied and comfortable with the plan. Once the surgeon, orthodontist, general dentist, and patient agree upon the overall treatment plan, the patient must understand they are committing to a long-term plan that has significant implication if they do not follow all the way through until completion. The surgeon should request from the orthodontist the following to initiate the dental setup for surgery. The orthodontist will need to make sure the teeth are leveled and aligned, and any root divergence or step occlusion is set up for planned segmental osteotomies. All dental compensations should be resolved. It must be determined if an absolute or relative transverse discrepancy is present and how it will be treated. Depending on the extent of the transverse discrepancy, the surgeon can decide if the maxilla will be widened initially with a surgically assisted maxillary expansion and then followed by a maxillary Le Fort osteotomy or if it will be treated in

one surgery involving segmentation with transverse widening. Attention must be given to make sure that there is not overaggressive orthodontic treatment to widen the arch which can lead to excessive tipping, periodontal deficiencies, and defects leading to buccal bone deficiency. Soft tissue recession/thinning, shortening of roots, as well as relapse can occur if the teeth are not set up properly within alveolar bone. Once the patient is set up and ready for surgery, accurate records and models are paramount for success in the operating room. The surgical planning by traditional cephalometric analysis and model surgery versus virtual planned surgery through computer aid is per the surgeons' choice. Virtual planned surgery will allow analysis in three dimensions and is quickly becoming the routine for orthognathic surgery. Regardless, errors that occur in the preoperative planning phase will transfer to the operating room. The surgeon needs to make sure the surgical splints are fabricated with appropriate design and fit and should be evaluated on the final models. Discussion and placement of the type of orthodontic brackets and wire for intraoperative strength and postoperative stability are confirmed. The final wire should be in a passive position, so no changes occur after the surgeons' final models are produced. With all this said, it shows how important the preoperative planning is to the success of the intraoperative surgery and final outcome.

14.3 Mandibular Surgery

14.3.1 BSSRO (Bilateral Sagittal Split Ramus Osteotomy)

14.3.1.1 Intraoperative Complications

BSSRO potential complications may include unfavorable osteotomy split, injury to the inferior alveolar nerve, TMJ dysfunction, bleeding, infection, malpositioning, and condylar resorption.

14.3.1.2 Unfavorable Osteotomy Split

Terminology such as "irregular osteotomy pattern and/or bad split" [2] has all been used when

discussing an osteotomy that does not go as planned. The incidence has been cited between 0.2 and 14.6% [3, 4]. When a bad split arises, the buccal plate of the proximal segment (52.7%) and lingual fractures of the posterior aspect of the distal segment (49.9%) are the most common anatomical locations. Condyle and coronoid fractures can also occur [2, 3, 5]. Buccal plate fractures distal to the second molar are common secondary to thinning of the plate and insufficient osteotomy(s) cuts especially at the inferior border. Small fracture segments can be removed. Large fractures should be visualized, approximated without stripping the periosteum completely, if possible, and plated. There are conflicting reports in the literature regarding the relationship of impacted third molars being removed at the time of orthognathic surgery and unfavorable fractures [5, 6]. The consensus appears that extractions of full bony impactions at the time of orthognathic surgery are acceptable without a significant increase in the risk of a bad split. If impacted wisdom teeth are not removed at the time of BSSO, then they should be removed at least 6–9 months prior to surgery. With condyle fractures, the condyle needs to be placed into the glenoid fossa for passive natural seating. Unfavorable fracture involving the condylar neck or a high ramus fracture is best treated with closed reduction. Fractures low along the ramus are generally treated with plates and screws. Lingual segment fractures can be difficult to fixate. This fracture is most common at the third molar region where cortical bone is thin if a third molar is present [3]. Proximal and distal osteotomy segments can be stabilized with monocortical screws and plates along the buccal aspect and the lingual fracture stabilized with bicortical screws.

14.3.2 Nerve Injury

While sagittal osteotomy of the mandibular ramus for advancement is the preferred approach to minimize injury to the inferior alveolar nerve, the risk of injury to this nerve remains a significant consideration. As Turvey and colleagues reported, transection of the inferior alveolar nerve occurred in 3.5% of BSSRO, and when

combined with an osseous genioplasty, nearly 70% of patients exhibited some degree of neurosensory deficit at 1 year [7]. The deficit is perceived as paresthesia or as reduced sensitivity over an anatomic dermatome innervated by the inferior alveolar nerve, most commonly the lower lip. Iannetti and colleagues reported in their retrospective study on 3236 orthognathic surgical cases including BSSRO that the neurosensory complication was due to a surgical mistake and occurred most frequently during the sagittal osteotomy cut (97.6% cases) [8]. Additionally, Seo and colleagues demonstrated that the alveolar nerve damage is the result of excessive traction forces applied during the osteotomy cuts and mechanical shear forces during the osteotome manipulation [9]. Nonetheless, such sensory disturbances represent a temporary event generally followed by a complete recovery between 6 and 12 months after surgery.

14.3.3 Temporomandibular Joint (TMJ) Dysfunctions

As described in the literature, improper seating of the mandibular condyles following BSSRO can result in rotation of the proximal segment and TMJ symptoms including pain, speech limitations, condylar resorption, and skeletal relapse. Valladares-Neto and colleagues' appraisal of the conflicting literature demonstrates that while the mandible is advanced and fixed, surrounding soft tissues including muscles and supporting ligaments are stretched and tend to displace the distal segment back to its original position [10]. Additionally, the amount of mandibular advancement either anteriorly or posteriorly to correct for retrognathism and prognathism, respectively, and increased joint loading secondary to greater rigidity of mandibular fixation contribute to poor TMJ position, disc instability, displacement, and condylar resorption [8, 11]. The authors propose that use of more rigid fixation techniques (positional screws) should be used for patients with greater bite force and larger advancements >7 mm, while a less rigid fixation (mini plates) is a better choice for patients who require smaller

advancements to minimize temporomandibular joint dysfunction. The proximal segment should be positioned to allow for passive anatomical seating of the mandibular condyle prior to fixation.

14.3.3.1 Bleeding

The risk of significant life-threatening bleeding for mandibular surgery is less than that associated with maxillary surgery and rarely occurs [12]. With advancements in surgical technique, hypotensive anesthesia, instrumentation, and hospital blood loss protocols, the need for blood transfusion is rare. Potential sources of bleeding are the maxillary, facial, and inferior alveolar arteries and the retromandibular vein. With appropriate instrument positioning subperiosteally and limiting depth of instrument penetration, bleeding can be minimized. Use of hypotensive anesthesia, mechanical pressure, and topical agents usually make bleeding controllable. In rare instances, ligation of the external carotid artery or angiography with embolization is indicated. Angiography is applicable in active bleeding and may diagnose anomalies, such as a vascular malformation.

14.3.3.2 Malpositioning of Mobilized Segments

Malpositioning of the mobilized segment can lead to open bite or lateral mandibular shift. Midline lateral shift can occur from inadequate advancement of the distal segment of one side or not seating the condyle properly at the time of fixation. Open bites can result from inadequate fixation followed by counter clockwise rotation of the proximal segment.

14.4 IVRO (Intraoral Vertical Ramus Osteotomy)

14.4.1 Indications

Intraoral vertical ramus osteotomy (IVRO) is a popular surgical procedure to correct mandibular asymmetry, mandibular prognathism, and symptomatic temporomandibular joint (TMJ)

dysfunction. The goal of IVRO is to make a full-thickness vertical osteotomy through the ramus of the mandible posterior to the mandibular foramen. This cut creates a proximal segment consisting of the condyle and posterior ramus and a distal segment containing the anterior ramus, coronoid process, inferior alveolar nerve, and mandibular teeth.

The benefits of IVRO may include improved mandibular excess, mandibular disc and condyle stability, enhanced aesthetic outcome, and increased efficacy when combined with sagittal split ramus osteotomy on the contralateral side to correct mandibular asymmetry [13]. In addition, IVRO has several advantages over SSRO. IVRO does not require internal fixation, reduces the surgical time and intraoperative bleeding, and is less likely to cause facial or inferior alveolar nerve injury [14]. However, IVRO has certain disadvantages compared with SSRO. The most common complication is condylar displacement or luxation from the glenoid fossa leading to unfavorable osteotomy split. Other major complications include bleeding, infection, and malpositioning of mobilized segments.

14.4.2 Condylar Luxation Secondary to Unfavorable Osteotomy Split

Angling the vertical ramus osteotomy posteriorly as the cut is made inferiorly will decrease muscle attachment and can lead to condylar sag. Condylar luxation is a major complication of IVRO. Hall and colleagues report an incidence of up to 0.2% of anterior condylar dislocation after modified condylotomy [15]. The extent of medial pterygoid muscle extirpation from its bony insertion site is considered to be a causal factor of this complication. Condylar luxation is also considered to be related to condylar sag which occurs as anteroinferior displacement of the proximal segment following the vertical osteotomy cut. Anterior force exerted from the medial pterygoid muscle on the inferior mandibular border also contributes to condylar luxation. More recently, Kawase-Koga and colleagues report the critical time for condylar luxation is during extubation and awakening

from anesthesia as gagging and coughing produce a forward vector force that increases the chance for anterior translocation of the condyle [13]. The shape of osteotomy line also influenced the proclivity of the condyle to luxation. Comparing the three osteotomy types (vertical, C-shaped, or oblique), Kawase-Koga's study showed that the oblique method, contrary to expectation, had the fewest complications, while the vertical type osteotomy had the most [13]. These results suggest that the oblique-type osteotomy with little medial pterygoid attachment is less subject to the ramifications of medial pterygoid stripping and subsequent condylar luxation and sag. Additionally, unopposed activity of the lateral pterygoid muscle on the proximal segment can contribute to condylar subluxation [16]. Thus, correct placement of the osteotomy to preserve adequate proximal segment of medial pterygoid attachment is of paramount importance in preventing condylar sag.

14.4.3 Nerve Injury

Inferior alveolar nerve injury is uncommon in IVRO compared with BSSRO [17, 18]. As Westmarker and colleagues describe, inferior alveolar nerve injury, associated with medial displacement of the proximal segment, occurs up to approximately 8% of cases [18]. The most common etiology involves anterior rotation of the proximal segment of the mandible that can compress the nerve as it enters the mandibular foramen. Frequently, this mechanical injury results in sensory deficits in the form of mandibular gingival and teeth paresthesia as well as lower lip and chin numbness as these dermatomes are innervated by the mental nerve, which is a continuation of the inferior alveolar nerve. Thus, a full-thickness vertical osteotomy is made through the mandibular ramus posterior to the mandibular foramen, which minimizes injury to the inferior alveolar nerve. Prior to making the initial cut, it behooves the surgeon to first identify the anti-lingula, an important anatomic landmark along the lateral aspect of the ramus for localizing the mandibular foramen. Appropriate preoperative panorex and or virtual reality surgical planning

software are utilized to map the distance from the posterior border of the ramus to the inferior alveolar canal. Any postoperative neurosensory alteration must prompt early clinical assessment and, if warranted, immediate repositioning of the rotated anterior segment to alleviate compression on the inferior alveolar nerve to minimize risk of permanent neurapraxia and sensory loss.

Recently, McKenna and colleagues describe that the lingual nerve can also be injured when a forked stripper is used to remove temporalis muscle tendon attachment from the anterior border of the ramus and caution against the use of this instrument [19]. Likewise, marginal mandibular branch of the facial nerve can be injured in the inferior half of the vertical osteotomy, and this risk can be minimized with sufficient elevation of the mandibular periosteum at the inferior border to allow adequate clearance of the oscillating saw from surrounding soft tissue [19].

14.4.4 Bleeding

The greatest potential for profuse bleeding comes from injury to the masseteric artery and branches of the maxillary artery near the coronoid notch [20]. Because of these vessels' close proximity to the medial side of the ramus, they are prone to injury in the vertical osteotomy. Vascular insult can be prevented or minimized with the use of a properly positioned Bauer retractor in the sigmoid notch to protect soft tissue and maintain subperiosteal plane [19]. If the superior cut is made last and significant bleeding encountered, it is usually easier to finish the osteotomy, mobilize the segment, and tamponade the bleeding with packing and firm pressure. As Hara and colleagues demonstrate, variations in the anatomic relationship between the maxillary artery and the sigmoid notch predispose to higher risk of vascular injury in select populations [21].

14.4.5 Malpositioning of Mobilized Segments

Preoperative planning is indispensable to ensuring adequate positioning of mobilized segments.

Failure to do so will result in gaps that precluding adequate contact between proximal and distal segments and contribute to nonphysiologic joint loading. McKenna and colleagues advocate for virtual surgical planning as an instrumental preoperative planning tool to make initial measurements to plan the maximum extent of mandibular setback or advancement [19]. By doing so, the surgeon can assess the amount of proximal-distal segment overlap preoperatively, use the lingula prominence to localize the mandibular foramen, and plan the vertical osteotomy accordingly. Prior to final occlusion and MMF, the surgeon must verify passive close apposition of the proximal and distal segments and perform any additional proximal segment trimming as necessary for proper positioning. Any excess proximal segment tip projection can be trimmed with a Kerrison rongeur. Horizontal excess of the mandible can be addressed using IVRO with posterior positioning of the distal segment. For large mandibular setback, angling the inferior osteotomy anteriorly will allow better overlap and prevent the formation of a narrow and short proximal segment with inadequate medial pterygoid attachment. Overzealous proximal segment trimming can lead to improper setback and malpositioning of mobilized segment. This can be avoided when trimming is performed conservatively as proximal segment is positioned lateral to distal segment for visualization of intersegmental gap width. Thus, with proper surgical care and correct positioning of mobilized segment, IVRO is a useful osteotomy for mandibular setback, advancement, and rotation to restore mandibular form, function, and aesthetics.

14.5 Genioplasty

Complications of genioplasty are rare but can result in untoward effects of minor soft tissue bruising and overstretching of the skin. More serious complications include profuse bleeding, mental nerve and inferior alveolar nerve injury, weakened masticatory muscles, and damage to teeth.

14.5.1 Alloplastic Implants Complication

Alloplastic chin implant with either autologous iliac crest bone or synthetic bone graft is an alternative to an osseous genioplasty for augmenting the chin. Alloplastic chin implants are not technically demanding and have a low complication rate for mild to moderate microgenia and a shallow labiomental fold. Furthermore, these implants may be performed in the outpatient setting with local anesthesia, thereby minimizing the risks associated with general anesthesia. However, a chin implant should only be used for subtle advancements of 5 mm or less. If it is used for larger chin augmentations, compression on the mandibular symphysis can contribute to bony erosion and degeneration. Additionally, a chin implant involves an incision in the skin under the chin, which could potentially leave an unsightly scar. As synthetic implants are foreign, they are prone to capsular formation and contracture, infection, and immune rejection. Implants can be inserted into a potential space without attachment to surrounding structures; they may move as a result of gravity and mentalis muscle movement, producing unwanted animation deformity and foreign body sensation.

14.6 Sliding Osseous Genioplasty Complication

14.6.1 Nerve Injury

In sliding horizontal osteotomy of the mentum, the inferior alveolar nerve (IAN) and mental nerve are vulnerable to an injury. Lindquist and colleagues reported an incidence of inferior alveolar nerve injury of up to 10% during genioplasty [22]. Nishioka and colleagues recorded paresthesia in the mental nerve dermatome in 3.4 and 12% of patients after genioplasty [23]. Ritter and Ousterhout advised that the genioplasty vertical osteotomy line should be made at least 6 mm below the mental foramen [24, 25]. More

recently, Gianni and colleagues reported that the combination of genioplasty and sagittal split osteotomy seems to be more detrimental for the lip sensibility than genioplasty or sagittal split alone. In particular, mental nerve distribution thermal sensation is less affected than tactile sensation, location, and two-point discrimination tests [26]. Hence, paresthesia in the mental nerve distribution has been described to occur in some degree in all patients, lasting for only a few weeks after sliding genioplasty. Nonetheless, generally, 1 year after the operation, normal lip and chin sensitivity were regained in up to 85% of patients. Because the inferior alveolar canal curves superiorly as it approaches the mental foramen at level of the first bicuspid, it behooves the surgeon to position the horizontal osteotomy 2–3 mm below the inferior edge of the mental foramen to prevent injury to this neurovascular bundle.

14.6.2 Bleeding

Bleeding complications in sliding genioplasty are rare and are usually attributed to laceration to geniohyoid or mylohyoid muscles. However, sublingual hematoma in the closed space of the floor of the oral cavity can displace the tongue posteriorly against the pharyngeal wall, leading to life-threatening airway obstruction, mandating urgent respiratory intervention. Lanigan and colleagues advise that a nasopharyngeal airway or tracheostomy performed to protect the airway should such respiratory compromise occur [20]. Generally, genioplasty is frequently combined with Le Fort osteotomy and or sagittal split ramus osteotomy, with similar risk of hemorrhage from injury to the retromandibular vein despite using the Hunsuck modification. This vein often lies just behind the posterior border of the mandible on top of the periosteum. Lanigan and colleagues presented 21 case reports to show that life-threatening hemorrhage associated with mandibular osteotomy is primarily an intraoperative problem, and such incidence is not as great as that following maxillary osteotomies [20]. The principle sources of hemorrhage were reported to

be injury to branches of the maxillary artery and facial artery and retromandibular vein. Such hemorrhage can be controlled with firm pressure packing with gauze, and blood transfusions are rarely necessary.

14.6.3 Chin Ptosis

Chin ptosis can occur secondary to mentalis muscle transection and improper repositioning of the mentalis muscle, resulting in “degloving” injury of the chin. This condition can create soft tissue ptosis with loss of lip support, which manifests clinically as flattening of the mentolabial sulcus, overexposure of the mandibular incisors, and lower lip incompetence. As Zide and McCarthy described, the mentalis muscle is a critical component of proper chin position and lower lip central mobility; and loss of mentalis support can contribute to excess drooling, chin dimpling, denture instability, and lower lip dysfunction [27]. The authors advised that in the event of excess mentalis muscle stretch injury and/or displacement, functional rehabilitation should be employed to help restore normal anatomy to allow the lips to close more effectively and elevate the position of the ptotic chin [27]. Ptosis is best prevented by reapproximating the severed ends of the mentalis muscle when closing the wound. If ptosis develops, surgical intervention entails using Prolene sutures to anchor the lower-oblique mentalis muscle fibers to the buccal and lingual mucosa or anterior mandible.

14.6.4 Mandible Fracture

As Goracy and van Butsele described, fracture of the mandible is a rare complication of genioplasty that can occur if the osteotomy of the two cortical bones is not performed completely before attempting to mobilize segments [28, 29]. If it occurs, the fracture line may extend into the mandibular body and ascending ramus, which require an open reduction.

14.7 Mandibular Surgery: Postoperative Complications

14.7.1 Excessive Swelling

Swelling is a common and expected outcome following orthognathic surgery. On average, 50% of initial swelling may resolve by the third postoperative week. Patients with higher body mass indexes have been shown to have a greater amount of initial swelling with a faster rate of resolution. As expected, patients who undergo double jaw surgery show significantly more postoperative swelling than those who undergo single jaw surgery, regardless of which jaw was operated on [5]. General postoperative guidelines to aid in swelling limitation include keeping the head of bed elevated, applying pressure dressings, providing a postoperative steroid regimen, and proper management of postoperative fluid balance.

14.7.2 Hemorrhage/Hematoma Formation/Thrombosis

Bleeding of varying amounts is common following orthognathic surgery. It is the surgeon's discretion to decide what is acceptable in the postoperative phase. The identification of any medical conditions predisposing the patient to coagulation abnormalities must be performed during the preoperative interview and workup. A lack of proper surgical technique and knowledge of anatomy may be the culprit of this potential complication for the neophyte surgeon.

Sources of significant bleeding following mandibular surgery may include the inferior alveolar, buccal, masseteric, and facial arteries, as well as the retromandibular vein [1, 3, 12]. Very rarely the maxillary artery may be a source of bleeding due to inappropriate dissection of the mandibular ramus [12]. All of the aforementioned vessels may be encountered at some point during surgery, possibly requiring hemostatic control. The incidence of significant bleeding during or after BSSRO surgery ranges between 0.38 and 1.2% [4, 7].

The inappropriate management of intraoperative bleeding will result in postoperative complications. Subperiosteal dissection is critical to limit the amount of bleeding from muscle, as well as from various named vessels. There have been numerous anecdotal accounts of the facial artery being pierced by a transbuccal trocar device during fixation of the mandibular segments at the conclusion of a BSSRO. Some surgeons have advocated using Jackson-Pratt suction drains or pressure dressings immediately following surgery to limit the possibility of hematoma formation. If hematoma formation is apparent, then warm compresses and continued pressure are recommended with close monitoring of the patient's airway. If acute expansion is noted despite these efforts, then the patient must be returned to the operating room for exploration, evacuation, and achievement of hemostasis [3].

Rare postoperative complications involving direct vessel injury have been discussed in the literature. Internal carotid thrombosis following bilateral sagittal split osteotomy caused by blunt trauma and patient positioning has been well described [1, 30, 31]. In addition, Pappa et al. [32] reported a false aneurysm of the facial artery resulting in severe bleeding 1 week after a bilateral sagittal split osteotomy.

14.7.3 Acute Infection

Infection following orthognathic surgery is relatively uncommon. However, surgical site of entry through mucosa during initial mucoves-tibular incision, osteotomy cuts, and screw placements are prone to bacterial colonization. Saliva, residual food, and blood clots might accumulate over these surgical wound sites, thereby creating optimal breeding conditions for infection. The pathophysiology entails translocation of inoculum into subcutaneous tissue and later lymphatic system and vascular system with subsequent clearance by reticuloendothelial system. Infection rates vary according to the study referenced, with some estimates ranging from 0.5 to 2.4% [33] and 1 to 33% [6]. The literature regarding the prevalence of infection

and the role of intraoperative versus postoperative antibiotic use is controversial. Various factors including patient age, length of surgery, type of orthognathic procedure performed, and use of antibiotics may all play a role in the development of infection [6]. Tucker and Ochs [33] reported a 2.4% infection rate in mandibular procedures, compared to 0.5% in maxillary procedures [12, 34]. In contrast, Chow et al. [6] reported a 7.4% overall infection rate, of which 51% involved the maxilla and 49% involved the mandible. Only 21.6% of patients who developed infection did so within the first week following surgery. Multiple reports describe infected hematomas or serous exudate accumulation secondary to hemorrhage as being common sources of infection following bilateral sagittal split osteotomy. Other contributing factors may include the presence of debris within the surgical site [35].

Double jaw surgery has been shown to have a significantly higher rate of postoperative infection when compared to single jaw surgery. Although controversial, some studies have demonstrated significantly less incidence of infection with the administration of a postoperative prophylactic antibiotic course (of varying duration), when compared to an intraoperative dose alone [6]. This finding was supported by Bentley et al. [36] but contradicted by Ruggles and Hann [37]. Chow et al. [6] found that there was a higher rate of infection in segmental versus non-segmental Le Fort osteotomies. They also noted that their rate of postoperative sinusitis following maxillary surgery was 0.86%, consistent with finding by Nustad et al. [38].

Despite adequate postoperative antibiotic regimen and dressing protocol following surgery, infectious events have been reported in literature and associated with poor oral hygiene and/or cigarette smoking, both of which predispose to contaminated wound beds and compromised vascularization leading to poor wound healing [30]. As Iannetti and colleagues describe, for the transbuccal approach to BSSRO, the microbial culprits include viridans streptococci (*S. mutans*, *S. sanguinis*) and anaerobes, which respond well to a course of 7–10 days of oral antibiotics [8]. Most

of these infections clear up by the end of first week, and only 0.2% of cases necessitate percutaneous or intraoral surgical drainage [8].

14.7.4 Chronic Infection

Late postoperative infection has been defined by occurring greater than 6 weeks postoperatively by many authors. Infections following orthognathic surgery have been described as occurring as early as the third day following surgery and as late as 9 years postoperatively. The incidence of infection seems to decrease with time, with 1–2% manifesting at 4 years or more [6]. The majority of chronic infectious complications may be dealt with a combination of oral antibiotic therapy and/or plate and screw removal within 1–8 years following surgery [6, 8]. Rare cases of systemic infection were summarized in a review by Steel and Cope [31]. Although uncommon, these cases included brain abscess following double jaw orthognathic surgery, cervicofacial actinomyces infection, and iliac abscess following bone harvest for grafting in the area of a maxillary advancement [29–41].

14.7.5 Neurosensory Deficit

Neurosensory deficit is perhaps the most predictable side effect in the immediate postoperative phase of orthognathic surgery. Nerve injuries encountered following a bilateral sagittal split osteotomy may involve sensory or motor nerves [3]. Intraoperatively, the inferior alveolar nerve is encountered along the path of the sagittal osteotomy. Although visualization of the nerve is common, transection of the nerve is not [7]. The nerve has been noted to be most vulnerable at the most anterior aspect of the split, although it can be injured anywhere from the pterygomandibular space to the anterior vertical osteotomy [3]. Chau et al. [42] reported a case of a traumatic neuroma of the inferior alveolar nerve due to direct trauma following mandibular osteotomies. Numerous figures can be found in the literature regarding immediate postoperative inferior alveolar nerve

hypoesthesia. Phillips et al. [43] reported a sensory dysfunction in 80–100% of patients in the immediate postoperative phase, while Kim and Park [12] reported inferior alveolar nerve hypoesthesia in 73.3% of patients following a BSSO. A systematic review completed by Colella et al. [44] reported objective neurosensory impairment of the inferior alveolar nerve as 63.3% at 7 days postoperatively and 49.2% after 14 days. In the acute phase, action is not often required. Many authors only advocate surgical intervention if dysesthesias are present following BSSRO. One case of allodynia in the distribution of the inferior alveolar nerve following BSSRO was reported by Vaughan and Cronin [45]. Computed tomography may be helpful in identifying possible nerve compression at the area of mandibular fixation. Potential treatment options may include nerve decompression with or without grafting resulting in variable rates of success [3].

Chronic inferior alveolar nerve hypoesthesia can be troublesome for any patient postoperatively. Colella et al. [44] found 42.5% of patients had neurologic impairment of the inferior alveolar nerve at 1 month following surgery, 33% at 6 months, and 12.8% after 1 year. The percentages for long-term hypoesthesia may vary between 12 and 85% [2, 36]. Multiple contributing factors increase the likelihood of prolonged postoperative neurosensory dysfunction including age of patient greater than 40 years old, increased intraoperative time, and a combination of concurrent genioplasty being performed at the time of mandibular surgery [12]. Hypoesthesia in the areas innervated by the mylohyoid nerve following genioplasty has been well-described secondary to trauma during osteotomy creation. Gradual recovery has been expected within 6 months following surgery [46]. Sensory nerve disturbance with a vertical ramus osteotomy is considerably less than with the bilateral sagittal split osteotomy [47]. Nevertheless, significant injury and even transection may occur [3]. Long-term inferior alveolar nerve impairment from this procedure ranges between 2.3 and 14% [2].

Neurologic complications other than those involving the inferior alveolar nerve from mandibular orthognathic surgery are less often

described. Steel and Cope [31] reported subjective lingual nerve dysfunction ranging from 9.3 to 19.4% following sagittal split osteotomy, although the overall objectively measurable incidence is thought to be much lower. One mechanism of injury to the lingual nerve involves the placement of inappropriately long screws during the fixation phase of the two mandibular segments. Guerissi and Stoyanoff [48] described a case of Frey's syndrome following orthognathic surgery. Aggressive dissection of the posterior boarder of the mandibular ramus was thought to be the etiology of the aberrant nerve regeneration, anastomosis, and dysfunction.

Cranial nerve VII injury has also been well-described following a bilateral sagittal split osteotomy. The vast majority of BSSRO procedures implicated in facial nerve injury involved mandibular setbacks, as opposed to advancements. Although this finding has been supported by numerous studies in the literature, cases of facial nerve injury following mandibular advancement have also been documented [12, 31]. De Vries et al. [49] reported facial nerve palsy in 0.26% of their patient sample. Sakashita et al. [50] reported the incidence of facial nerve palsy following orthognathic surgery as 0.17–0.75%. The mechanism of facial nerve injury has multiple potential factors. These may include compression from the posterior boarder of the mandibular ramus, unfavorable fracture of the mandible, aggressive pressure packing posterior to the mandible, injection of local anesthesia in the peri-mandibular region, hematoma formation, direct injury via errant placement of surgical instruments posterior or inferior to the mandibular ramus, compression of the mastoid process by the proximal segment of the mandible, or direct injury during transcutaneous placement of instruments [51, 52].

Although facial nerve palsy is a rare phenomenon following mandibular orthognathic surgery, communication of its risk and prognosis is important to discuss with patients. Patients with cleft and craniofacial abnormalities have been found to be at a higher risk of facial nerve injury [51]. In the authors' experience, anatomical variation, especially in patients with conditions such as

hemifacial microsomia, adds a higher degree of potential for postoperative facial nerve palsy. Multiple authors have postulated that early treatment with steroids, vitamins, or electrical stimulation may be linked to positive outcomes [53]. Complete recovery has been observed in most cases within a time period ranging from multiple weeks up to a year following injury [51, 54].

14.7.6 Malocclusion/Nonunion

Malocclusion in the early postoperative phase following orthognathic surgery is not a common outcome but can be readily apparent in the early postoperative phase. Early relapse or malocclusion may be due to inadequate mobilization of the jaws, the influence of bony interferences with instability of the internal fixation, and improper passive seating of the condyles into the glenoid fossa during mandibular fixation. Small occlusal discrepancies noted in the immediate postoperative phase may be managed by class 2 or 3 dental elastics or orthopedic appliances. Large, significant discrepancies in occlusion may necessitate prompt return to the operating room [2]. Immediate anterior open bite may result from the inadequate removal of posterior interferences while displacing the condyles from the fossa during fixation. The development of a late-onset anterior open bite may be due to multiple factors including the collapse of transverse expansion, orthodontic relapse, condylar resorption, or increased ramus growth [55].

Mandibular nonunion and fixation failure are both common reasons a surgeon may encounter postoperative malocclusion. Fixation failure can be especially problematic in the mandible resulting in nonunion and potentially proximal segment rotation and resorption. Clinical signs of either fixation failure or nonunion may include mobile mandibular segments, persistent infection, open bite with premature contact on the affected side, Class 3 occlusion on the affected side, and midline shift toward the unaffected side [55]. Robl et al. [55] recommended either a conservative option of limited function and mobility via maxillomandibular fixation or more

aggressive therapy with reoperation and reinforcement of the fixation.

14.7.7 Temporomandibular Joint Dysfunction

Orthognathic surgery may potentially benefit patients with TMJ dysfunction by providing a balanced and stable occlusion. However, outcomes are unpredictable, and this must be accurately communicated to patients [55]. White and Dolwick found that 89.1% of patients with preexisting temporomandibular joint dysfunction showed improvement following orthognathic surgery. Approximately 2.7% had unchanged TMJ function and 8.1% had a worsening in TMJ symptoms. Dysfunction was more prevalent in skeletal class 2 patients than skeletal class 3; however, both groups showed improvement in symptoms postoperatively [56]. Potential contributing factors to postoperative TMJ dysfunction include torqueing of the condylar head during mandibular fixation, high mandibular plane angle, and previous history of condylar changes demonstrated radiographically. Turvey et al. [7] noted in their retrospective review that less than 1% of their patients with postoperative TMJ dysfunction had symptoms persist longer than 6 months. However, a minimal number of patients did require eventual treatment with conservative therapy and arthroscopy.

14.7.8 Condylar Resorption/Aseptic Necrosis

Condylar resorption is a dreaded late complication of orthognathic surgery. Although the exact cause is unknown, there is a correlation with this condition and multiple risk factors. These factors include being a young female with preexisting mandibular retrognathia (often with an increased mandibular plane angle) and having had previous TMJ dysfunction prior to undergoing a mandibular surgery in which there was posterior displacement of the mandibular condyles [57, 58]. Moreover, compared to a vertical oblique

osteotomy, the BSSRO approach ensures a more robust blood supply to the condyles, minimizing the risk of avascular necrosis. The time frame for the manifestation of this condition is thought to be 7–27 months following surgery. Resorption of the condyle is a progressive and gradual phenomenon that often results in a decrease in posterior facial height with an increase anterior open bite [59]. Posnick's review of the topic describes a process that steadily progresses and is followed by a period of stabilization with no further loss of mandibular height [60]. Currently, there is no effective and established treatment available for ongoing condylar resorption. It is generally recommended that no treatment be initiated to correct the malformation until 6 months to 1 year after the resorption has ceased [59, 60]. Although subject to inaccuracies, Tc99 MDP quantitative condylar bone scintigraphy may be a useful adjunct to evaluate whether active resorption is present [60, 61]. A CT scan may be useful to elucidate the extent of condylar resorption.

According to Schnell-Has [62], two potential mechanisms for this condition are a remodeling of the condyle-fossa complex due to a change in loading forces or a compromise of temporomandibular joint vasculature resulting in avascular necrosis. Other theories maintain that sex hormones interact with the temporomandibular joint causing synovial inflammation and condylar resorption [60]. Although not much data regarding the incidence of condylar resorption following orthognathic surgery has been published, one study reported an incidence of 4% at 2 years postoperatively [31, 63]. Appropriate methods of definitive treatment for this condition are controversial. Treatment modalities may include non-surgical therapy such as occlusal splinting and orthodontics or various surgical options involving the TMJ. Some surgeons strongly propose aggressive surgical intervention such as total joint replacement and orthognathic surgery [64].

Aseptic necrosis of the mandible is an extremely infrequent outcome following orthognathic surgery. Lanigan and West [65] published two case reports describing this phenomenon following BSSRO and BSSRO with genioplasty. The cause of necrosis of a segment of the

mandible in the BSSRO patient was thought to be due to aggressive dissection and stripping of the pterygomasseteric sling. Treatment with antibiotics and hyperbaric oxygen therapy was thought to limit the amount of eventual bony sequestration. The second case of aseptic necrosis resulted following a 14 mm advancement of a genioplasty segment. This may have been the result of aggressive dissection of the musculature on the lingual surface of the segment. The authors concluded that this complication should be avoidable with judicious dissection of the mucoperiosteum and muscle attachments to the mandible during the time of osteotomy [65].

14.7.9 Unfavorable Aesthetic Outcomes

An intimate knowledge of the effects that skeletal changes have on the overlying soft tissue is fundamental for the orthognathic surgeon. Surgical repositioning of the mandible and the chin can have dramatic consequences on the contour of lower third of the face. Mandibular setback procedures can unfavorably change the neck profile, possibly necessitating a secondary surgery for aesthetic benefit. Chin ptosis may develop postoperatively from improper resuspension of the mentalis muscle [2]. Furthermore, poor preoperative planning may result in an unfavorable final location of the mandible or chin, causing either an excess or insufficiency of projection. The degree and complexity of the surgical revision required is dependent on the degree of postoperative deformation that is seen.

14.8 Maxillary Surgery

14.8.1 Intraoperative Complications

14.8.1.1 Airway

Nasal intubation by the anesthesia team is typically requested by the surgeon. A preoperative evaluation of the nasal anatomy will help plan on which nares passage may be easier to intubate. Intraoperative management of the tube by securing

with tape and monitoring its position is crucial to help prevent inadvertent extubation. The most frequent time the tube could be at risk is with the septal-vomer separation and the lateral nasal wall osteotomy on the side of the tube [44, 49, 66]. To help minimize a tube laceration, guarded chisels and placement of a malleable to protect the tube is helpful. Nasal turbinates may need to be reduced if maxillary impaction surgery is being performed.

14.8.1.2 Incision Design and Closure

When designing the incision, leaving an adequate cuff of tissue to maintain a blood supply and allow for tissue retraction should be at least 5 mm from above the mucogingival line [66, 67]; delicate and non-overzealous stripping is key to tissue health and healing. If there is history of any previous maxillary surgery, incision design should be more judicious in leaving adequate tissue.

14.8.1.3 Unfavorable Osteotomy/ Split

It is rare to have an unfavorable fracture in the maxilla that alters the course of treatment. The downfracture should be completed with digital pressure only with avoidance of forced pressure. If there is difficulty with the downfracture, thin osteotomes should be utilized to score the sight of resistance, which is most commonly along the posterior maxillary wall or along the posterior lateral nasal wall [66, 68]. The most common altered fracture pattern occurs at the pterygoid plates. There have been postoperative computed tomography studies performed that show a variety of fracture patterns; in the region of the second molar, horizontal pterygoid plate fractures and vertical fractures traveling superiorly to the skull base have been observed [66, 69]. A horizontal osteotomy designed too low in the maxilla can compromise vitality of the dentition or cause direct trauma. Keeping the osteotomy at least 5 mm above the root apices is recommended.

14.8.1.4 Hemorrhage

As with any surgical procedure, bleeding can be expected to some extent. Significant bleeding with orthognathic surgery is rare, but when it does occur, it is more common with maxillary

procedures [33, 68–71]. With that said though, most studies have put severe maxillary bleeding at around 1% [70]. The maxillary artery and terminal branches, the sphenopalatine and descending palatine arteries, the pterygoid venous plexus, and the posterior superior alveolar are the most common sources of bleeding. Precise and attentive surgery, staying in a subperiosteal plane, and being cognizant of local potential bleeding sources will help decrease untoward bleeding. While performing the lateral nasal osteotomies, the osteotome should not be inserted greater than 20 mm blindly. While performing the maxillary nasal septal separation, a blunt spatula will help reduce mucosal tears. When performing the pterygoid maxillary separation, if a chisel is utilized, it should be 15 mm or less and directed in an inferior and anterior direction while placing a finger on the palate for direct tactile feel. Alternatives for pterygoid osteotomies may be an oscillating saw or placing the osteotomy through the tuberosity if the bone is dense and difficulty in separation occurs [69]. Bleeding may occur from direct vascular injury or indirectly usually occurring from fragmented bone pieces. Adjunctive procedure to help reduce bleeding is hypotensive anesthesia (MAP 60 mmHG) and possibly elevating the head in reverse Trendelenburg. When brisk bleeding occurs, the surgeon should complete the downfracture and try to identify with direct visualization the bleeding source. Treatment can be with direct pressure, hemostatic clips, electrocautery, and local anesthesia with vasoconstrictor. Topical hemostatic adjuncts are helpful when direct visualization does not occur and may include Avitene, Gelfoam, and Surgicel. If the previously mentioned attempts fail, ultimately the patient may need an arteriogram and embolization versus direct external carotid ligation. If embolization is required, there are multiple products the radiologist may use including Gelfoam, polyvinyl acetate, Surgicel, Avitene, onyx, and Embosphere to control bleeding [2, 3, 8].

14.8.1.5 Improper Maxillary Repositioning

The appropriate preoperative planning will help reduce intraoperative malpositioning. Accurate

model surgery or VSP can be referenced intraoperatively to confirm desired movements. Stable and accurate reference points for measuring are important and can be external, internal, or a combination depending on the surgeons' preference. Insuring passive reposition of the osteotomies without interferences is paramount to successful surgery. If over-impaction occurs, secondary surgery may be required to gain more favorable results.

14.8.1.6 Perforation of Oral-Nasal Mucosa

This is a rare complication, especially with non-segmental maxillary surgery. Once identified as occurring intraoperatively, the size will determine treatment. If a small tear occurs, these tend to close spontaneously with local wound care. If larger, 6–8 mm, single-layer palatal closure or attempt of layered closure is indicated. Additional measures of a membrane/PRP/tissue glue can be utilized as well as postoperative sinus precautions. If a palatal perforation occurs and an oral-nasal fistula develops, then after the maxilla has healed from initial surgery (6–8 weeks later), an obturator can be fabricated if indicated, until definitive closure with a local flap (anterior, posterior island, tongue) can be accomplished.

14.8.1.7 Nasolacrimal Violation

Injury to the nasolacrimal duct/canal can occur, especially if osteotomy design is higher along the lateral nasal wall. If epiphora or hemolacria occurs (bleeding from the lacrimal puncta), this will usually resolve once the edema subsides. If excessive tearing persistent, dacryocystorhinostomy may be indicated.

14.8.1.8 Segmental Maxillary Surgery

When performing a multipiece surgery, it may be helpful to try to preserve the descending palatine arteries to promote as much blood supply as possible. Controlled use of instruments, judicious irrigation, and slowly stretching especially with multipiece surgery are beneficial maneuvers to limit risks. Not keeping the patient hypotensive for too long of time frame and keeping the patient

warm may help in promoting adequate blood flow.

14.8.1.9 Unfavorable Interdental Osteotomy

The surgeon should have a discussion with the orthodontist to gain at least 5 mm of space between the roots. Divergence is preferred to help prevent inadvertent direct root trauma with the osteotomy and periodontal defects from occurring. A safe saw can be used to help prevent root trauma as well as palatal tissue injury. If dental trauma occurs, this should be discussed with the patient and once healed from surgery the tooth/teeth should be evaluated for endodontic consideration. Minor soft tissue injuries can be treated with local measures (topical rinses) and hygiene education. Ultimately a soft and/or hard tissue regeneration procedure may be indicated. If a tooth is compromised significantly, an extraction may be indicated and discussion of restorative options for replacement.

14.8.1.10 SARPE

When performing surgically assisted rapid palatal expansion, an asymmetric expansion and pain with expansion are usually secondary to an incomplete osteotomy at one or more of the major sites of resistance (mid-palatine suture, zygomaticomaxillary buttress, pterygoid junction and piriform aperture) [72]. Making sure the orthodontic expander expands freely intraoperatively and equally should be confirmed.

14.9 Postoperative Phase

14.9.1 Maxillary Surgery: Postoperative Complications

14.9.1.1 Periodontal Defects/ Devitalized Teeth

Proper preoperative preparation and model surgery are critical to avoiding injury to the dentition and the surrounding periodontium. Careful surgical technique and proper flap design will aid in avoiding this preventable complication. Presurgical orthodontics should provide adequate

spacing between tooth roots for interdental osteotomies in segmental maxillary surgery [2, 3, 55]. While performing osteotomies around teeth, the surgeon must identify the root apices. The osteotomies must be performed at least 5 mm from the root apices, and there must be enough bone between roots as previously discussed. Thin instrumentation with judicious fluid hydration while drilling can help prevent bone injury from occurring. Decrease in blood supply can lead to minor soft tissue alterations to minor and more significant periodontal issues and in rare cases loss of teeth and adjacent bone. Although complete loss of vascularity to a tooth is rare, long-term loss to pulpal stimulation may occur. Bruising of teeth and initial discoloration following maxillary orthognathic surgery has been reported with eventual spontaneous recovery. Long-term evaluation of the involved teeth is necessary by the patient's general dentist or orthodontist. If the teeth do not spontaneously recover, they develop periapical pathology on radiographs, or the apices were obviously violated intraoperatively, and then endodontic therapy is warranted [3, 55]. Soft and hard tissue regeneration as well as dental implants may be indicated to replace unsavable teeth.

14.9.1.2 Hemorrhage/ Pseudoaneurysm/ Arteriovenous Fistulae

As discussed in the previous section on hemorrhage, multiple mechanisms of trauma during orthognathic surgery can result in significant bleeding and vessel injury. Bleeding from the nose should be evaluated with good lighting and with instrumentation that allow appropriate visualization. The source should be identified as venous versus arterial. Positioning the head so it is elevated and keeping the patient calm may be all that is indicated. Nasal tamponade devices (gauze, nasal tampons) can be placed but should not stay for greater than 3 days. More aggressive bleeding may require either an anterior and or posterior packing devices. If bleeding persists, further workup for any hematologic problems may be warranted as this may be the first and only surgical procedure the patient has undergone,

and hematologic deficiency may have not been diagnosed. Pseudoaneurysms, thrombosis, and arteriovenous fistulae have been reported [73, 74]. Although significant postoperative bleeding is one of the most feared complications of elective surgery, the incidence has been found to be very low in orthognathic surgery patients. The vast majority of significant intraoperative bleeding has been related to Le Fort I osteotomies and resultant injury to the descending palatine and sphenopalatine arteries. Most reports of postoperative bleeding occur within 10 weeks of surgery, with the vast majority happening within the first 2 weeks [75]. Severe epistaxis following maxillary orthognathic surgery is an ominous event postoperatively [76]. If bleeding from the nose occurs with facial asymmetry greater than 2 weeks after surgery, pseudoaneurysms should be considered. Although the incidence is reported as less than 1%, life-threatening hemorrhage due to a pseudoaneurysm of the sphenopalatine or internal maxillary artery may initially present in this manner [31, 76, 77]. The inciting etiology of the false aneurysm is direct trauma to the vessel intraoperatively with partial occlusion, vessel extravasation, and finally hematoma formation that undergoes endothelialization. Cases of arteriovenous fistulae of the maxillary artery resulting from unfavorable fractures that extend to the pterygomandibular fossa following pterygomaxillary disjunction have also been reported. Embolization of these vascular anomalies is required [78]. Exceedingly rare anomalies such as cavernous sinus thrombosis and deep vein thrombosis following maxillary orthognathic surgery have also been noted in the literature [79, 80].

14.9.1.3 Ophthalmic Complications

The most dreaded ophthalmic complication following maxillary orthognathic surgery is blindness. Steel and Cope [31] reviewed ten published cases of full or partial blindness following orthognathic surgery. The authors concluded that two cases resulted from propagation of the pterygomaxillary disjunction fracture through the skull base, one due to decreased blood flow to the optic nerve, five of unknown cause, and one

resulting from an arterial aneurysm. Of the nine cases, three showed no recovery; five were able to appreciate light perception, hand motion, or finger count; and one showed gradual improvement to baseline over time. It was also observed that several of these cases included patients with a history of cleft lip and palate or patients who had undergone repeat osteotomies and were thought to have “thickened bone” at pterygomaxillary junction. Most cases were initially managed with high-dose steroid administration and diagnostic imaging which included computed tomography, magnetic resonance, or angiography.

Although the incidence of immediate postoperative retrobulbar hematoma is very low, many surgeons advocate routinely checking the condition of the orbits after the lowering of the drapes following orthognathic surgery. Maxillary surgery case reports have been published describing the expected presentation that would be seen in the case of a retrobulbar hematoma, including proptosis and tense eyelids, which were treated with variable success utilizing a lateral canthotomy [81, 82].

Numerous cases in the literature describe ocular neurologic complications following maxillary orthognathic surgery. Palsy of the abducens nerve and/or the oculomotor nerve has been reported. Most of these reported cases presented with neurologic dysfunction within the first 5 days following surgery [83]. Etiology of the nerve injury included pterygoid plate fracture resulting in hematoma during attempted disjunction, cavernous sinus thrombosis, and propagated fracture involving the sphenoid sinuses [81, 83, 84]. The majority of these nerve injuries recovered spontaneously within 10 weeks following surgery. The vidian nerve and greater petrosal nerve have also been reported as being potential subjects of neurologic injury. Patients who have presented with a lack of tearing following Le Fort orthognathic surgery have been thought to have sustained injury to these nerves [31]. These result in an interruption in parasympathetic innervation to the lacrimal gland [81]. Hemolacria and excessive tearing have also been reported as the result of damage to the nasolacrimal duct during the Le

Fort procedure. Conservative treatment and eventual resolution within 8 months have been observed [81, 85, 86].

14.9.1.4 Neurologic Dysfunction

As previously discussed in the section on neurosensory deficit and ophthalmic complications, neurologic dysfunction has been thoroughly reported following orthognathic surgery. Although expected temporary nerve paresthesia of branches of the trigeminal nerve results from maxillary vestibular incisions and dissection, more obscure cases of nerve dysfunction following maxillary corrective jaw surgery have been reported. Anticipation of the nasopalatine and superior alveolar nerves transections with the Le Fort osteotomy is inevitable with little long-term sequela. CN V2, namely, the infraorbital nerve, is the most common neurosensory disturbance with maxillary surgery. It typically returns over a shorter amount of time. Long-term sensory loss is cited at about 1.5–2% [9, 47]. Case reports of patients sustaining dysfunction of cranial nerves X, XI, and XII have been described by multiple authors following significant intraoperative bleeding with Le Fort maxillary downfracture. The proposed etiology of injury was thought to include manipulation during the maxillary downfracture and/or aggressive pressure packing to control intraoperative bleeding [87, 88]. Cases of abducens (VI) nerve and oculomotor palsy have been reported and typically resolve over weeks. Other documented cases of cranial nerve II, III, and VI have been reported. These rarely seen nerve injuries are most likely to form unfavorable fractures ascending into the cranial base [9, 47].

Catastrophic events such as cerebrovascular accident and hemiparesis following orthognathic surgery have been described in the literature. Newhouse et al. [88] reported a case of significant intraoperative bleeding following maxillary down fracture and mobilization. After initial management of the hemorrhage, angiography demonstrated a traumatic arteriovenous fistula. It was postulated that during the pterygomaxillary disjunction, a sharp piece of bone was displaced posteriorly causing direct vessel injury. Ultimately, the patient remained with hemiparesis

postoperatively. Another case of hemiparesis following double jaw orthognathic surgery was observed due to internal carotid thrombosis resulting from a neck flexion-induced intimal tear [89].

Other rare reported neurologic complications following Le Fort I osteotomies include cranial nerve III dysfunction, sphenopalatine ganglion dysfunction, and hearing impairment. Bendor-Samuel et al. [84] described a Le Fort I osteotomy that resulted in a subarachnoid hemorrhage with a carotid-cavernous fistula and an internal carotid artery aneurysm, initially presenting with cranial nerve III palsy on postoperative day 2. Possible skull fracture during downfracture of the maxilla was a postulated mechanism of injury. Interestingly, as with other rare complications, the patient involved had a history of cleft lip and palate. One report of postoperative secretomotor rhinopathy was described as resulting from a hematoma in the area of the sphenopalatine ganglion following a Le Fort I procedure. This patient was successfully treated with anticholinergic medication [90]. Gotzfried and Thumfart [91] published a review of middle ear function in cleft patients who underwent Le Fort 1 surgery. They found that temporary hearing dysfunction was observed due to edema of the palatal tissues and the Eustachian tubes. At 6 months postoperatively, the majority of these patients achieved significant improvements.

Benign paroxysmal positional vertigo (BPPV) has also been thought to potentially result from orthognathic surgery. Beshkar et al. [92] reported a case of BPPV developed following a double jaw orthognathic surgery. The proposed mechanism has been described in other patients undergoing maxillary surgery. Indirect trauma to the labyrinth of the internal ear from force being applied to the maxilla during the creation of osteotomies can propagate through the temporal bone, dislodging otoliths. BPPV is self-limiting, and symptoms may be relieved by employing a variety of therapies, including the Epley maneuver. Klemm et al. [93] reported a rare case of Eustachian tube dysfunction and tinnitus following Le Fort I osteotomy. The mechanism of injury

was also thought to be force transmission to the inner ear.

14.9.1.5 Skeletal Complications

The ability of the maxilla to maintain its blood supply following a maxillary osteotomy during orthognathic surgery has been a topic of intrigue since the landmark articles by Bell et al. [94–96]. It is important to note that these early studies did not take into account the effect of a large anterior-posterior movement, a large transverse change within the maxilla, or a significant superior movement of the maxilla causing superimposition of the bony segments. Lanigan et al. [97] published a review of 51 cases of various degrees of aseptic necrosis of the maxilla. Bell et al. [98] hypothesized that factors including flap design, osseous repositioning, multi-segment osteotomies of the maxilla, stretching of the palatal vascular pedicle, hypotension, and transection of the descending palatal vessels could potentially contribute to vascular necrosis [31]. Lanigan's review observed that this complication may be more prevalent in patients with a history of cleft palate [97]. With surgical access, osteotomies, and repositioning of the bony segments the blood supply to these sights can be compromised which can affect the hard and soft tissues. Fortunately the vast blood supply makes this a rare occurrence. If vascular compromise does occur, the surgeon should make sure there are not any eliciting local factors such as smoking, poor wound care, and impingement of the surgical stent on the tissue. Treatment should consist of watchful observation and meticulous oral hygiene, as this is usually transient [34]. Topical and systemic antibiotics can be utilized as well as a topical moist dressing applied directly to the sight to prevent secondary infection. If the tissue is significantly compromised, dusky in appearance, consideration of taking the patient back to the OR and removing the rigid fixation and allowing the segments "relax" to see if reperfusion occurs and implementing HBO can be considered. The hyperbaric oxygen therapy is thought to hasten the delineation of necrotic segments, allowing proper debridement of these areas. This therapy has not been shown to prevent or reverse aseptic

necrosis once it has begun, but it may limit its extent [97]. Care should be taken with patients who have had previous maxillary surgery as they will have compromised scar tissue. Alternative incision design, adequate pedicles without overzealous stretching, and possible staging of procedures can limit this sequela. If multipiece surgery is being performed, one should make sure the palatal coverage strut of the splint is not creating any pressure. Conservative but meticulous hygiene is indicated, and the use of topical and systemic antibacterial should be considered. Gentle debridement of tissue and exposed necrotic bone should be performed with timely patient reevaluations. Evaluation of the tissue may reveal initially a white appearance that turns to a graying/dusking. Initial white blanched tissue ischemia may be able to be reversed with alleviating the offending cause (tissue stretching, pinching off of blood supply, etc.). But the difficulty with once avascular necrosis is identified, it has already taken place. Hyperbaric oxygen therapy can be considered but should be implemented in a timely fashion and will not help the current necrotic bone.

Nonunion of the maxilla is another complication that the orthognathic surgeon may see. Smoking, poor nutrition, or other medical conditions that preclude to poor wound healing should have been identified and discussed preoperatively. From a pure surgical standpoint, adequate fixation, especially with large advancements and downward and forward movements, is the most susceptible to nonunion inherent to their instability. Sufficient bone contact is important in attempting to prevent this complication. Movements greater than 5–7 mm should consider bone grafting and possible secondary fixation (IMF or skeletal). If the patient is in IMF (intermaxillary fixation) and the maxilla is mobile, it may be prudent to release the IMF, so the mandible is disengaged. Postoperative elastics in an isolated maxillary surgery patient should be watched closely as the intact mandible has the potential to create significant force that translates directly to the maxilla [71]. Patients with a history of clenching should be identified preoperatively as this habit can cause detriment if not

controlled. Strict postoperative diet control must also be enforced. Chewing of any foods of hard consistency should be eliminated if the nonunion persists; reoperation, removal of fibrous tissue, and grafting with adequate fixation should be performed.

One of many feared complications for the neophyte surgeon is an accidental avulsion of the maxilla during mobilization following a maxillary downfracture. Although this is an exceedingly rare complication, Bendor-Samuel et al. [84] described a case of avulsion of an entire segment of the maxilla in a bilateral cleft palate patient undergoing orthognathic surgery. It was believed that overaggressive soft tissue dissection played a major role in this occurrence. The segment was replaced in its initial position and ultimately survived.

Another rare outcome of maxillary surgery is a cerebrospinal fluid leak. Gruber et al. [99] reported a case which manifested as a clear rhinorrhea from the nostril on postoperative day 3 following a Le Fort I impaction and BSSO. The etiology of the leak was thought to be either the result of a propagation of a fracture to the skull base during the pterygomaxillary disjunction or from aggressive bone removal during the maxillary impaction. This case resolved with lumbar drain placement.

Postoperative malocclusion is another complication that can take place. Malocclusion may be secondary to swelling and neurosensory deficiency early postoperatively and should be addressed with guiding elastics. If the malocclusion does not improve with elastics or is significant, postoperative X-rays should be taken to evaluate hardware and osteotomies. If no hardware failure is confirmed, the malocclusion may be from a lack of seating the condyle properly during mandibular surgery, interferences that were not noticed or a lack of mobilization of the osteotomy(s). Confirmation of correct planned surgical maneuvers should be reevaluated. In segmented Le Fort osteotomies, inadequate intraoperative or postoperative means of maintaining expansion can result in a transverse relapse with malocclusion. This same result can be seen with both fixation failure, as well as nonunion of the

maxilla. A mobile maxilla, an open bite tendency, and a premature contact on the side of malocclusion are clinical signs of maxillary nonunion or fixation failure [55]. Robl et al. [55] recommended both conservative and aggressive options for a mobile maxilla following orthognathic surgery. Conservative management includes prescribing a non-chew diet, decreased or absent traction from elastics, modified splint placement, and infection management. In the case of moderate to severe malocclusion, repeat surgery is indicated. Surgical options include recreation of the maxillary osteotomy, removal of all fibrous tissue, rigid fixation, and potential bone grafting if warranted. In the case of moderate to severe malocclusion, repeat surgery is indicated.

14.9.1.6 Velopharyngeal Insufficiency

A well-known potential consequence of orthognathic surgery in the cleft palate patient is the propagation of velopharyngeal insufficiency (VPI) with maxillary advancement. Although these patients may have previously undergone satisfactory repair of their palate, the velopharyngeal complex may not function adequately in order to create a sealed separation of the nasopharynx and the oropharynx during speech. This dysfunction in the complex results in air being able to be expelled nasally while speaking, causing hypernasal speech along with various articulation abnormalities. It is best to screen these patients before surgery and have the appropriate diagnoses documented. Working with a speech pathologist throughout the course of care can be beneficial and reassuring to all involved. Maxillary advancement in the non-cleft patient does not create this same issue, due to their appropriate ability to compensate for the new position of the soft palate [100].

Effects on the velopharyngeal complex following a Le Fort maxillary advancement have been extensively researched. Watzke et al. [101] found no correlation between the amount of maxillary advancement and velopharyngeal function in cleft patients. However, velopharyngeal deterioration may be expected in a patient with preexisting boarder line function prior to maxillary

advancement [102, 103]. The possibility of worsening hypernasal speech following surgery must be appropriately communicated to the patient prior to surgery. McComb and colleagues [104] demonstrated that cephalometric analysis of palatal length and pharyngeal depth preoperatively may aid in predicting which cleft patients would suffer from post-orthognathic VPI, requiring further surgery for its correction. Costello et al. [100] noted that for the vast majority of these patients, gradual resolution of their VPI should be expected over the first 6 months following surgery. For this reason, the authors advocate waiting at least 6 months prior to undertaking any surgical correction of the velopharyngeal complex. For patients with persistent VPI longer than 6 months to a year despite speech therapy, further diagnostic imaging with naso-endoscopy or videofluoroscopy may be undertaken. If the VPI is extensive and does not improve, surgical option that may be appropriate is a pharyngeal flap or sphincter pharyngoplasty [103].

14.9.1.7 Unfavorable Aesthetic Outcomes

The aesthetic outcomes of orthognathic surgery are a result of the dental, skeletal, and soft tissue changes that have been surgically manipulated. These types of results are best avoided with appropriate diagnosis, treatment planning, and patient education. The treatment of aesthetic shortcomings ranges from skeletal correction to soft tissue revision, depending on the type and degree of imperfection [2, 105]. Orthognathic surgeons must have an intimate knowledge of the effects that movement of the bony skeleton will have on the overlying soft tissue. Significant lip and nose changes result from maxillary repositioning and are extremely noticeable to the patient. Alar width, lip length, and tip projection are influenced by the final position of the upper jaw. Over-retruded and over-impacted maxillae are the two most commonly seen unfavorable maxillary aesthetic orthognathic result [105]. If this result occurs, performing revision surgery is most likely indicated. To reestablish nasal base width, an alar base suture should be performed. Performing a V-Y closure of the lip incision will help keep the lip length

appropriate. Nasal-tip rotational changes can occur and should be anticipated if the maxillary movement predispose to tip changes. Minor tip changes may result in favorable results, but larger changes may need to be predated and addressed with a rhinoplasty at the time of maxillary surgery. In older patients who undergo setback type procedures, there is a risk of “aged” appearance as soft tissue may relax from not having underlying support. Changes of the soft tissue in older patients are less reflective of the bony surgery and harder to predict final outcome [105].

With maxillary surgery there can be a multitude of nasal anatomy changes in the postoperative phase; this includes septal deviation, alar base widening, tip rotation, and dorsal deformities [55]. To prevent septal bowing or deviation, the cephalic septum should be trimmed appropriately and or a trough in the maxillary crest. The septum can be secured to the nasal spine to help maintain its position. If septal deviation is noted in the operating room at the conclusion of surgery, immediate closed reduction of the septum should be attempted and is typically successful, especially if the septum was inadvertently manipulated with extubation. Otherwise, reduction with local anesthesia or sedation may be attempted in the office. If septal deviation is a result of inadequate removal of the caudal septum, then a septoplasty can be performed after initial healing. Although numerous minor issues may be able to be dealt with in an office setting postoperatively, a formal septorhinoplasty may be needed in the future to correct any unwanted outcomes. Also with septal deviation, there can be obstruction of the nasal valve, altering airflow. The alar base should be maintained to its preoperative width, and a cinch suture can help reestablish the anatomy. Depending on the maxillary movement, changes to the nasolabial angle can occur and should be anticipated with preoperative planning.

Conclusion

Orthognathic surgery is the most effective operation to dramatically improve a patient’s facial skeletal and occlusal disharmonies. While this is a powerful technique for addressing dentofacial anomalies, orthognathic surgery can be

fraught with many serious complications. Years of disciplined and dedicated study of the regional anatomy, as well as mastery of the surgical techniques involved are the only true means of avoiding such problems. Furthermore, the surgeon must remain vigilant at all times while paying particular attention to every detail of the operation. A firm knowledge of all potential pitfalls is critical in order to avoid these complications. When complications are encountered, it is imperative for them to be recognized as soon as possible. The surgeon must then have a working knowledge as to the different strategies to utilize in order to optimize the outcome of the operation.

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Abstract

Blepharoplasty is a common procedure to restore the youthful appearance to the upper and lower eyelids. Although this cosmetic procedure is safe, adverse events can still occur. Appropriate knowledge of periorbital anatomy, a systematic preoperative assessment as well as a meticulous surgical technique will minimize the risk of complications. However, if complications occur, timely management is critical. This chapter will review the ocular anatomy, patient assessment, and the different surgical approaches. Additionally, management of a comprehensive spectrum of blepharoplasty complications will be discussed.

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15.1 Introduction

Blepharoplasty is commonly performed to restore youthful appearance to the upper and lower eyelids or to reconstruct the eyelids after skin cancer excision. Although it is a relatively low-risk procedure which can have highly gratifying results, blepharoplasty is not without surgical risks and complications. These may range from temporary benign problems including mild dry eyes and suture granulomas to more serious complications including severe infection, cosmetic deformity, and retrobulbar hemorrhage. The anatomy of the periorbital region is complex, and careful attention to the various functional and cosmetic aspects of the eyelid is integral to maintaining protection of the eye and good aesthetic result. Various surgical techniques and procedures are vital to minimizing risks and

preventing complications. These include patient selection, education, and preoperative assessment; attention to anatomic difference and careful measurements; careful surgical technique; and appropriate postoperative follow-up care.

15.2 Ocular Anatomy

Foremost, a thorough understanding of the periocular anatomy is key when creating a surgical plan for patients undergoing blepharoplasty. In the sagittal plane, the layers are compartmentalized into anterior, middle, and posterior lamella. From superficial to deep, the upper lid anterior lamella comprises skin, subcutaneous tissue, and orbicularis muscle. The eyelid skin epidermis comprises approximately 5–7 layers of keratinized epithelium. The dermis contains lymphovascular network, hair follicles, and sebaceous and sweat glands. The subcutaneous tissue is made of loose areolar adipocytes. The subcutaneous fat is notably sparse in preseptal and preorbital skin and is absent over the medial and lateral palpebral ligaments, where the skin adheres to the underlying fibrous tissue. Common cosmetic issues of the skin and subcutaneous tissue include dermatochalasis and blepharochalasis. Dermatochalasis is redundant and lax eyelid skin caused by gravity, sun exposure, and age-related loss of elastic tissue leading to weakening of the supporting structures of the eyelid. In addition to actinic changes from aging and sun exposure, other contributing factors to upper eyelid dermatochalasis include weakening of the orbital septum that predisposes to preaponeurotic fat pad herniation and weakening of levator aponeurosis causing associated involutional ptosis. These findings are more common in the upper eyelids but can be seen in the lower eyelids as well. Dermatochalasis can be a functional or cosmetic problem for the patients. Functionally, dermatochalasis obstructs the superior visual field and contributes to entropion of the upper eyelid, ectropion of the lower eyelid, blepharitis, and dermatitis. Cosmetically, patients frequently complain of upper eyelid “fullness” or “heaviness” and lower eyelid “bags” and lateral canthi wrinkles. Blepharochalasis, on the other hand, is caused by

recurrent bouts of painless eyelid inflammation that leads to excessive stretching, thinning, and atrophy of eyelid tissue with formation of redundant skin folds over lid margins and protrusion of orbital fat. While blepharochalasis can be treated with blepharoplasty and/or lateral canthoplasty, it is relatively rare and occurs more commonly in younger than older patients.

Orbicularis oris is a muscle of facial expression that is invested by the superficial musculoaponeurotic system (SMAS) and is further subdivided into the pretarsal, preseptal, and orbital layers. The orbital layer consists of circular bands of the skeletal muscle that extends circumferentially and interdigitates with fibers of the frontalis and corrugator supercilii superiorly and lip elevators inferiorly. Its main role is to serve as a powerful eyelid contractor to allow for tight closure of the eyelids. The pretarsal and preseptal orbicularis play a vital role in blinking. Thus, it behooves the surgeon to be meticulous in the preseptal submuscular plane to avoid damage to these orbicularis muscle fibers to minimize risk of incomplete lid closure and subsequent keratoconjunctivitis sicca. Deep to the orbicularis lies the fibrous avascular orbital septum, extending from the orbital periosteum to the superior tarsus. The membranous fibrous septum provides the framework through which levator aponeurosis, vessels, and nerves traverse to provide structural integrity and functional capacity to the eyelid.

The eyelids derive structure from the tarsi, which are two plates of dense connective tissue about 2.5 cm in length, located within the upper and lower eyelid, directly inside the lid margins. The orbicularis retaining ligament attaches circumferentially around the orbital rim, spanning the medial and lateral canthi. It serves to protect the globe and anchor the orbicularis muscle superiorly and inferiorly. The upper eyelid contains two fat pads, the nasal and central fat pads, which lie just below the upper orbital septum, and flanked laterally by the lacrimal gland. The nasal fat pad more commonly herniates with age and may require surgical debulking, whereas the central fat pad may only require debulking in select cases. Extensive removal of the upper lid fat compartments was historically more common but noted to result in greater superior sulcal hollowing, so surgeons shifted toward

preservation of fat particularly in the central compartment with more youthful results. The two upper eyelid elevator muscles, levator palpebrae superioris muscle and superior tarsal muscle of Müller, lie deep to the fat compartment. The levator originates from the lesser wing of the sphenoid bone, courses along the superior orbital rim, and fuses to form levator aponeurosis as it attaches to the upper eyelid and tarsal plate to form the superior eyelid crease. The tarsal muscle originates from the levator and also inserts on the tarsal plate. Together, these two muscles control lid movement and are separated by a vascular plexus. Patients who have dehiscence of the levator from the pretarsal lid may exhibit ptosis and absence of the lid crease. With aging, the upper eyelid exhibits classic changes including subcutaneous brow fat loss, increased skin laxity, enlargement or atrophy of the central fat compartment, and enlargement of the nasal fat compartment. It is critical to evaluate the extent of each of the aforementioned changes to determine the optimal customized surgical approach.

Like the upper lid, the lower lid also has a thin layer of skin overlying the orbicularis muscle. Deep to the muscle lies the lower lid tarsus which is about the same thickness as the upper lid tarsus but only extends about half the height (4 mm as opposed to 8 mm). Like the upper lid retractors, the lower lid retractors attach to the inferior tarsal plate but are less defined anatomically. The lower lid counterparts to the upper lid levator and Müller muscles are the inferior tarsal muscle and the lower lid retractor. Because the lower eyelid buttress receives contribution from malar fat pad and soft tissue of the cheek, lower blepharoplasty consultation also warrants evaluation of midface deformity and age-related skin atrophy. Thus, the surgical goals in eyelid rejuvenation require maintaining the fatty volume and strengthening the eyelid's extrinsic support by fat repositioning, lateral canthopexy, and orbicularis and midface suspension.

15.3 Patient Examination

Prior to any cosmetic eyelid procedure, surgeons must perform a focused medical and comprehensive ophthalmic history and examination.

Blepharoplasty should not be recommended in patients with active or severe dry eye symptoms or recent corneal refractive surgery such as laser-assisted in situ keratomileusis within the past 6 months, as they are at increased risk for exacerbation of dry eye and development of keratopathy. A thorough examination of the upper eyelid is integral to preventing complications including lagophthalmos, ptosis, eyelid malposition, and injury to surrounding structures including the lacrimal gland. It is important to record the position of the upper eyelid crease and fullness, which may indicate a prolapsed lacrimal gland, and explain these findings to the patient. In addition to upper eyelid examination, lower eyelid evaluation is key to preventing chemosis, ectropion, dry eye syndrome, and lid malposition. Lid laxity is traditionally evaluated with the snap test, in which an index finger gently retracts the lid downward and abruptly releases it. Return of the eyelid to the globe represents normal eyelid tone. The distraction test is another technique which involves pulling the lower lid anteriorly. A distance of 6 mm or more is consistent with lower lid laxity. Other anatomic assessments include the position of the medial and lateral canthi, lower eyelid and cheek junction, and the presence of a negative vector eyelid. The canthal tilt, or the angle between the medial and lateral canthi, is a key feature which changes with aging. A negative canthal tilt is observed, and then the lateral canthus dips below the medial canthus and results from excess eyelid skin or increased laxity. A negative vector eyelid is observed when the globe extends anterior to the cheekbone and has increased risk for eyelid malposition following blepharoplasty.

Dermatochalasis must be distinguished from blepharoptosis, in which true ptosis occurs. Elevating the excess skin can allow the examiner to assess the position of the upper lid margin in relation to the upper boundary of the iris. In blepharoptosis, raising the excess skin fold will not rectify the downward displaced upper lid margin. The eyelid elevators can be dysfunctional or damaged from metabolic conditions, autoimmune disorders, trauma, iatrogenic injuries, and medical side effects. Most commonly, age-related changes are associated with laxity of the levator aponeurosis to the tarsal plate contributing to upper lid margin to

be displaced inferiorly creating the fatigued appearance. Other less common causes include myasthenia gravis, cranial nerve III palsy, Horner's syndrome, and facial palsy, in which the eyelid elevators go unbalanced and affect incomplete eyelid closure with paralyzed orbicularis.

15.4 Transconjunctival Blepharoplasty

Current upper and lower eyelid blepharoplasty approaches have favored the transconjunctival incision over the transcutaneous incision for improvement in eyelid retraction and lid malpositioning. The transconjunctival incision can be made either through preseptal or retroseptal plane and extended from the puncta to the lateral canthus about 4–6 mm below the inferior tarsal plate separating the lower lid retractors from the inferior tarsus. The preseptal approach is preferred as it achieves dissection through suborbicularis preseptal plane to allow for direct visualization of the septum and surgical manipulation of each fat pad separately. In cases involving fat repositioning, both medial and central fat pads are usually repositioned followed by debulking of the lateral fat pad to the orbital rim. Autologous fat grafting and/or synthetic subdermal fillers are preferred to redraping techniques for cosmetic rejuvenation of the lateral infraorbital rim [1]. After a sufficient amount of fat has been removed for repositioning, a subperiosteal or suprapariosteal approach is used to dissect along the orbital rim. Various techniques have been described for repositioning, including using buried resorbable sutures [2–4] and externalizing a percutaneous suture securing fat pedicles subperiosteally with 5-0 polypropylene suture on both sides of the infraorbital nerve [5]. Nonresorbable sutures tied over bolsters should be removed a week after surgery.

15.5 Skin-Muscle Flap Blepharoplasty

A variety of approaches to skin-muscle flap blepharoplasty have been described, including a

comprehensive description by Codner et al. [6]. In brief, the surgeon performs a subciliary incision to elevate the skin-muscle flap while maintaining about 4 mm of underlying pretarsal orbicularis muscle. The skin-muscle flap includes the preseptal portion of the orbicularis muscle and is dissected inferiorly in the direction of the infraorbital rim. If aesthetic improvement of the lid-cheek junction is desired, the orbicularis retaining ligament may be released suprapariosteally. Septal incisions enable direct visualization of herniated orbital fat, which may be debulked and/or repositioned subperiosteally along the infraorbital rim. A septal reset procedure may be optimal in some cases. The surgeon then performs a lateral canthopexy as previously described [6]. The skin-muscle flap is lifted suprotemporally, and excess tissue is excised if necessary. The orbicularis is resuspended at the lateral orbital rim, and any excess skin is carefully trimmed before the incision is closed.

15.6 Complications

15.6.1 Eye

15.6.1.1 Infection

Postoperative infection can range from mild to severe but is fortunately a rare complication of eyelid surgery likely due to significant vascularization of the periorbital area. The infection rate has been reported around 0.2–0.4% [7]. Perioperative prophylactic antibiotics are unnecessary as postoperative topical antibiotics are often effective. The types of infection include preseptal cellulitis, orbital cellulitis, necrotizing fasciitis, and dacryocystitis.

Preseptal cellulitis, which usually manifests as mild erythema, swelling, drainage, and pain, is often mild and amenable to outpatient treatment by oral fluoroquinolones or third-generation cephalosporins. In contrast, severe orbital cellulitis should be suspected when the patient presents with severe eye pain, lid erythema and swelling, decreased visual acuity, abnormal pupillary light reflexes, and extraocular muscle dysfunction and pain. It is important to perform

computed tomography with contrast to assess for postseptal infection, abscess, and cavernous sinus thrombosis. Orbital cellulitis most frequently arises from *Streptococcus*, *Staphylococcus* (including methicillin-resistant *Staphylococcus aureus*), and *Mycobacterium* infections and requires hospitalization and intravenous broad-spectrum antibiotic treatment in addition to abscess drainage as needed. Cultures of exposed wounds, drain sites, and peripheral blood are warranted to refine antibiotic regimen and monitor treatment response, which may not become apparent until 24–48 h later. Atypical mycobacterial infections may not become apparent until 1 to several months after surgery and may include multiple tender nodules with abscesses requiring repeated debridement. Confirmatory cultures may take weeks to grow. A several-month course of macrolide antibiotics such as oral clarithromycin has been reported as successful treatment for nontuberculous mycobacterial infections.

Necrotizing fasciitis may result from *Staphylococcus aureus* or group A beta-hemolytic *Streptococcus* infection and may present similarly to preseptal inflammation. The rapid progression of necrotizing fasciitis within avascular fascial layers is a key clinical feature which distinguishes from benign causes of inflammation. Other key clinical manifestations include severe edema, loss of sensation, severe pain, fluid-filled violaceous bullae, black necrotic tissue, and clear delineation between the infected area and surrounding tissue. For severe infection, early intravenous empiric broad-spectrum antibiotics such as piperacillin-tazobactam are critical. Careful debridement of necrotic tissue may be necessary, but extensive removal of tissue should be avoided as it can have adverse consequences. There is insufficient evidence to support the role of hyperbaric oxygen therapy in necrotizing fasciitis [8, 9].

15.6.1.2 Chemosis

Chemosis, or transudative edema of the conjunctiva, may arise following blepharoplasty, sinusitis, or allergies. Postoperative chemosis may result from canthal disruption, exposure of the conjunctiva, periorbital edema, and lymphatic

injury. The incidence of chemosis may be as high as 11.5% in lower lid surgery and usually manifest within the first postoperative week as epiphora, corneal dryness, blurry vision, and foreign body sensation [10]. Intraoperatively, careful surgical technique is critical to prevent chemosis. Postoperative prevention of chemosis includes administration of preservative-free artificial tears every few hours throughout the day and ophthalmic ointments overnight. Conservative management entails application of warm compresses and gentle massage over the eyelids. Chemosis which does not resolve after 1 week of conservative treatment may respond to steroid eye drops and decongestants. Systemic steroids may be helpful in certain cases. Other strategies include using an eye patch comprising transparent occlusive dressing with petrolatum-based ointment on the exposed conjunctiva to maintain pressure and lubrication. When conservative treatment options have failed, surgical management including temporary medial or lateral suture tarsorrhaphy, refixation of the fornix, or a modified conjunctivoplasty with careful cauterization may be necessary. Although most cases are mild and resolve spontaneously, some cases may require up to 6 months to improve.

15.6.1.3 Dry Eye

Keratoconjunctivitis sicca or dry eyes may occur in the early, intermediate, and late postoperative period and are a common complication of blepharoplasty. The incidence of dry eye syndrome after blepharoplasty ranges from 8 to 26% [11–13]. Early dry eye symptoms may be transient and self-resolving, while later presentation of “true” dry eye syndrome may occur due to widening of the palpebral fissure after blepharoplasty particularly in patients with risk factors for dry eye. Associated risk factors include male gender, concomitant upper and lower lid surgeries, preoperative lid laxity, hormone therapy, transcutaneous lower blepharoplasty, lagophthalmos, proptosis, and maxillary hypoplasia. Common presenting symptoms include foreign body sensation, deep eye discomfort, stinging, blurry vision, photophobia, and eye redness. The conjunctiva may appear red and inflamed.

Preoperative assessment of dry eye syndrome commonly includes the Schirmer test, in which a Schirmer strip is placed on the lower lid and the eyes are closed for 5 min. The strip is then removed, and the amount of wetting is noted, with normal tear production ranging from 10 to 15 mm and dry eye syndrome defined as less than 5 mm of Schirmer strip wetting. Diagnosis of dry eye syndrome preoperatively is the best way to prevent postoperative dry eyes. If symptoms present postoperatively, initial conservative treatment includes frequent use of preservative-free artificial tears throughout the day and ophthalmic ointment overnight. If symptoms do not improve, nighttime eye patching, taping the eyelid shut, and/or using a bandage contact lens may help retain moisture. Lacrimal punctal plugs made of silicone or hydrogel may be effective in select patients with persistent symptoms.

15.6.1.4 Epiphora

In the early postoperative period following lower blepharoplasty, epiphora is common. Frequently it is due to exposure keratopathy, chemosis, lower lid ectropion, or impaired lacrimal pump [14]. Epiphora due to corneal irritation or chemosis tends to resolve on its own within several days. Patients with persistent epiphora should be evaluated for mispositioned lacrimal puncta or damage to the canaliculi. Surgical correction of anatomic abnormalities may be achieved through a medial or lateral canthopexy, horizontal tightening procedure, or medial spindle procedure.

15.6.1.5 Corneal Abrasion

Eyelid surgery may result in damage to the corneal epithelium, usually noted immediately postoperatively. Symptoms include severe ocular pain, tearing, foreign body sensation, and photophobia. The diagnosis should be suspected when more serious complications such as globe perforation and retrobulbar hematoma have been ruled out. Diagnosis of corneal abrasion is made using fluorescein applied to the ocular surface and examination for epithelial disruption under cobalt blue light. The etiology of corneal abrasion may be due to ocular surface desiccation during prolonged exposure intraoperatively or direct trauma

to the corneal epithelium. Corneal abrasion can be prevented by using globe shields with ophthalmic lubricant in both operated and nonoperated eyes preoperatively. Alternatively, a 6-0 silk traction suture can lift the conjunctiva superiorly while everting lower eyelid margin to confer cornea protection and direct visualization of sub-biculis preseptal plane for fat pad manipulation.

Treatment of corneal abrasions includes antibiotic ophthalmic drops four times daily, lubricating eye drops, a bandage contact lens, and/or patching and usually improves in a day. If immediate improvement does not occur, daily ophthalmic exams are needed to prevent progression of the abrasion into a corneal ulcer.

15.6.1.6 Retrobulbar Hemorrhage

Retrobulbar hemorrhage (RBH) is one of the most feared complications of blepharoplasty; although it is a rare complication, with an incidence of 1 per 2000 (0.05%), RBH is the most common cause of visual loss following blepharoplasty and can lead to permanent visual loss in 1 per 10,000 (0.0045%) patients [15]. Intraorbital hemorrhage may result from traction on or resection of orbital fat or persistent wound hemorrhage extending posteriorly in patients with poorly controlled systemic hypertension. Visual loss is thought to result from increased intraorbital pressure leading to microvascular compression and ischemic injury to the optic nerve. Alternatively, an increase in orbital pressure exceeding the mean arterial pressure of the ophthalmic or central retinal artery may lead to a central retinal artery occlusion. Prudent risk management and prompt treatment can prevent devastating visual loss.

Thorough preoperative evaluation is integral to managing risk for RBH. Risk factors include hypertension, history of vascular disease or bleeding diathesis, and use of antiplatelet and anticoagulant medications. Prudent management includes adequate control of hypertension and discontinuation of medications such as warfarin, aspirin, and NSAIDs for 2 weeks prior to surgery and 1 week postoperatively [16]. A careful preoperative visual acuity exam is warranted to

detect any preexisting visual impairment and obtain a baseline measurement upon which to gauge any postoperative changes attributable to the procedure.

Intraoperatively, several measures can be undertaken to prevent RBH. Upon administration of local anesthetic with epinephrine, the surgeon should allow 10–15 min to allow for maximal vasoconstriction. Meticulous attention to perioperative mean arterial blood pressure and timely hemostasis is critical to minimize bleeding without compromising tissue perfusion. As bleeding tends to occur from the orbicularis or orbital fat pad vessels, particular attention to achieving adequate hemostasis before closure and minimizing traction during fat excision are imperative. During dissection, the use of insulated fine needle electrocautery, radiosurgery, and carbon dioxide lasers may assist with hemostasis of small vessels and avoid excessive dissection and traction relative to conventional clamping techniques.

Postoperatively, the patient should be counseled to avoid activities that could lead to sudden increases in blood pressure including excessive straining (e.g., Valsalva, heavy lifting) or bending over during the first postoperative week. The patient may be prescribed antitussive and antiemetic medications as needed to prevent straining with vomiting and coughing. Stool softeners such as Colace or Senna are recommended to alleviate constipation that may contribute to increased intra-abdominal pressure that translates to increases in systolic blood pressure. Head elevation, maintenance of the supine position while sleeping, and frequent application of cool compresses during the first 4 days postoperatively may further limit the risk of hemorrhage. Patients should also be given instructions to test their visual acuity by covering each eye and reading small print appropriate to their preoperative level of acuity; concurrently they should be advised that some minor blurring is expected secondary to surgical ointment in the tear film and/or diminished postoperative blink reflex; however, any sudden sustained change in vision postoperatively merits immediate notification of the surgeon.

Prompt recognition of RBH is critical for early intervention and prevention of complications (Fig. 15.1). The majority of patients (82–96%) with RBH develop signs or symptoms within 24 h of the procedure. The two most frequent times of presentation occur intraoperatively or within 1 h (25%) and between 6 and 12 h postoperatively (36%); however, bleeding has been reported as late as 9 days postoperatively [16]. Eye pain and pressure are the most common symptoms. Postoperative bandages and patches should be avoided to allow for early detection of the clinical signs of RBH; commonly, the patient will have significant periorbital hematoma with continued incisional bleeding and/or increased intraocular pressure manifesting as a proptotic globe that resists retro-pulsion (proptosis may be minimal given limitation of anterior displacement of the globe by the medial and lateral canthal tendons). Additional signs include subconjunctival hemorrhage, decreased extraocular motility, superior orbital



Fig. 15.1 Retrobulbar hematoma 12 h after blepharoplasty. Reprinted with permission from Elsevier (Whipple KM, Korn BS, Kikkawa DO. Recognizing and managing complications in blepharoplasty. *Facial Plast Surg Clin North Am.* 2013 Nov;21(4):625–37)

fissure syndrome, decreased visual acuity, and an afferent pupillary defect. Recovery room staff should be educated regarding signs of RBH and instructed to test visual acuity with finger counting postoperatively, as a sudden loss of light perception within the first hour can signify an acute bleed.

RBH is a medical and surgical emergency, as a delay in treatment can lead to irreversible visual loss or impairment in ocular motility. When suspected, the surgeon should immediately open the surgical incision at the bedside to release the orbital compartment syndrome. With the wound open, the surgeon can then evacuate the hematoma and cauterize any active bleeding sites. Often, bleeding has subsided due to tamponade from the hematoma. If the intraorbital pressure remains elevated or the hematoma cannot be completely evacuated, the surgeon should proceed with lateral canthotomy and cantholysis, initially with lysis of the inferior crus and, if needed, lysis of the superior crus of the lateral canthal tendon. If all measures fail, an emergent computed tomographic scan without contrast should be performed. If a retrobulbar hematoma is detected, bony decompression with a lower eyelid and transconjunctival approach to expose the orbital floor and allow creation of a medial floor osteotomy may be necessary to ameliorate compression of the orbital apex. As adjunctive therapy for elevated intraocular pressure (>35–40 mmHg), topical ocular hypotensives (e.g., timolol) may be given [17]. Persistently elevated intraocular pressure may be managed with systemic agents including acetazolamide (500 mg), mannitol (1–2 mg/kg), and corticosteroids in consult with an ophthalmologist. Timely, aggressive treatment in the first 48 h is warranted as it is possible to achieve restoration of vision even in patients with “no light perception” within the first 24 h postoperatively.

15.6.1.7 Globe Perforation

Accidental perforation of the globe is a rare but severe complication of periocular surgeries. The highest risk is during injection of local anesthetic, especially in older patients with delicate upper eyelids. Protective corneal shields can help

prevent injury to the globe. Careful injection technique is also critical. The tip of the needle should be aimed away from the globe at all times, and traction should be placed on the brow and upper eyelid to create enough distance from the globe. Upon entry into the subcutaneous space, injection should be performed slowly to gradually create a fluid wave separating the tissues and protecting delicate structures from the advancing needle tip. Penetration of the globe may result in a range of ocular damage including corneal perforation, traumatic cataract, intraocular hypotension or hypertension, retinal tears or detachment, intraocular hemorrhage, and globe rupture. Any violation of the globe integrity is an ophthalmic emergency and requires urgent evaluation by an ophthalmologist. Treatment includes the use of a Fox shield over the entire eye and broad-spectrum intravenous antibiotics.

15.7 Skin (Eyelids)

15.7.1 Under-resection

Careful avoidance of over-resection may inadvertently lead to under-resection of eyelid tissue as the consequences of the former are more challenging to correct. Reoperation to remove residual skin following blepharoplasty is necessary in approximately 5–10% of patients. Typically, residual skin is noted most commonly on the lateral eyelid and arises from an underestimation of how much dermatochalasis was present preoperatively. Good preoperative counseling is important to set appropriate patient expectations, and thorough preoperative evaluation before the initial and any subsequent blepharoplasties is critical.

Revision should be considered only after healing is complete, typically 3–6 months following the initial blepharoplasty. Evaluation for concurrent brow ptosis should be performed on any patient considering reoperation as any further removal of eyelid skin will lead to exaggeration of brow ptosis. Brow ptosis should be addressed in such patients prior to blepharoplasty revision. Once brow ptosis has been ruled out, a careful

evaluation should be done to ensure adequate skin remains for proper eyelid closure after further resection. The standard distance is 20 mm measured at the center of the eyelid from the lash line to the base of the patient's natural brow. Patients requesting further skin removal when there is inadequate residual skin present should be counseled on the importance of proper eyelid function and the risks of over-resection and lagophthalmos.

15.7.2 Over-resection and Lagophthalmos

Postoperative lagophthalmos, or inability to close the eyelids, has many causes and usually resolves in a few days to weeks following surgery. Causes of transient lagophthalmos include myotoxicity of the pretarsal orbicularis oculi muscle from anesthetics and incomplete lid closure secondary to edema or pain. Causes of lasting lagophthalmos include excessive skin resection (Fig. 15.2), injury to the facial nerve innervating the orbicularis, or inclusion of the orbital septum in skin closure causing lid retraction. Prolonged incomplete closure of the eyelids has serious complications including exposure keratopathy, microbial keratitis, stromal thinning or scarring, and permanent vision loss. Lagophthalmos due to over-resection can be prevented with careful

preoperative examination. Patients who have received prior LASIK surgery are at increased risk for blepharoplasty lagophthalmos [18]. Whereas patients with an intact Bell's reflex may be asymptomatic with up to a 2 mm deficit in eyelid closure, those with existing dry eye disease are at increased risk for exacerbation of symptoms and exposure keratopathy. Careful preoperative markings using pinch test can help prevent over-resection of tissue. If there is concomitant brow ptosis, the ptotic brow is gently elevated in its original anatomic position while performing the pinch test using smooth forceps to pinch the redundant eyelid skin. Subtle movement of the eyelid margin rather than overt lagophthalmos is observed when the appropriate amount of excess skin is pinched. In all cases of lagophthalmos, frequent use of ophthalmic lubricants is essential. This includes the use of preservative-free artificial tears every 30 min to 2 h and an ophthalmic ointment applied at bedtime. Additional treatments include absorbable punctal plugs for short-term (2 months) relief or silicone punctal plugs for long-term punctal occlusion and overnight patching overlying ophthalmic ointment to maintain a moisture chamber. In cases refractory to conservative treatment, surgical elevation of the lower eyelid, skin grafting, or release of orbital-septal adhesions should be considered. In cases of anterior lamellar deficiency, a supraciliary approach to

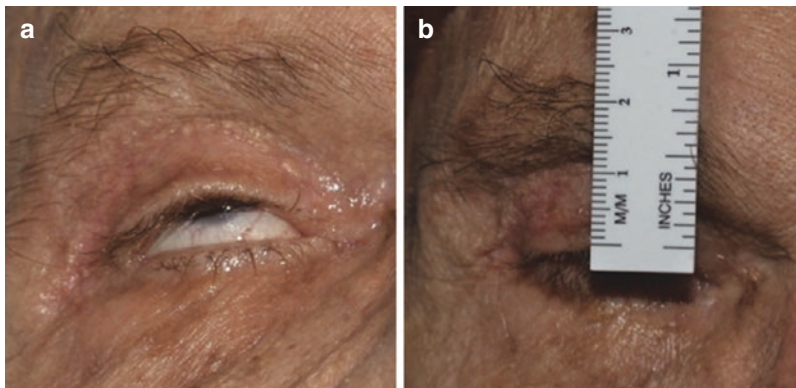


Fig. 15.2 Lagophthalmos following blepharoplasty with skin over-resection (a). Only 12 mm of skin remains between lash line and inferior brow border (b). Reprinted with permission from Elsevier (Whipple KM, Korn BS,

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skin grafting has been described [19]. The same technique for full-thickness blepharotomy to address ophthalmopathy of hyperthyroidism has also successfully addressed post-blepharoplasty lagophthalmos [20].

15.7.3 Scarring

Cicatricial webbing of the tissue at the medial or lateral canthus following blepharoplasty may result in aesthetic and functional consequences. Cicatricial canthal webs can obscure underlying tissues similar to an epicanthal fold and result in a visual field cut on lateral gaze. Risk factors include Asian race, lower-set eyelid crease, poor physiologic wound healing, autoimmune skin disease, preexisting tarsal fold, and brow ptosis. Medial scarring commonly results from incision too close to the lid margin or extension of the incision too far medially or inferiorly. Lateral scarring can result from upper and lower incisions joining to create an inferior angle or over-resection of the skin. Ensuring proper preoperative marking and maintaining a 5 mm distance between the upper and lower incisions may reduce the risk of lateral webbing.

Mild webbing may improve with gentle post-operative massage. Surgical revision should be offered 3–6 months after the initial surgery if moderate to severe webbing is still present. Severe webbing is a considerable challenge as the revision surgery may require complex tissue rearrangement with microflaps. Z-plasty or W-plasty transposition flaps or Y-V advancement procedures are possible approaches to repair [21].

15.7.4 Eyelid Hematoma

The signs of an eyelid hematoma include ecchymosis and mild swelling of the lid and surrounding structures (Fig. 15.3). The first step is to ensure that retrobulbar hemorrhage has been properly ruled out. An eyelid hematoma should lack classic features of retrobulbar hemorrhage including proptosis, severe pain, vision changes,

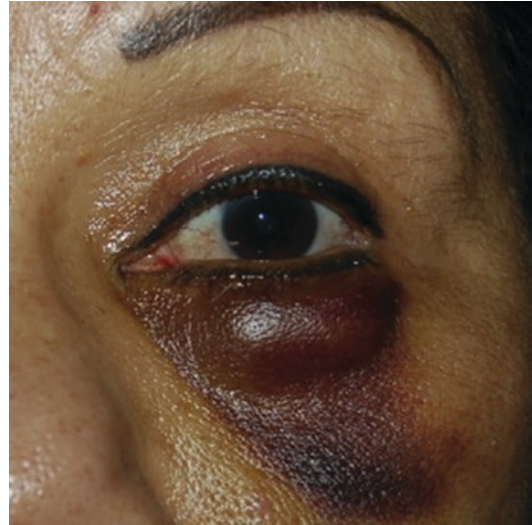


Fig. 15.3 Preseptal hematoma following left lower eyelid blepharoplasty. Reprinted with permission from Elsevier (Whipple KM, Korn BS, Kikkawa DO. *Recognizing and managing complications in blepharoplasty. Facial Plast Surg Clin North Am.* 2013 Nov;21(4):625–37)

and disturbance in extraocular movements. The source of bleeding in most eyelid hematomas is the orbicularis muscle or orbital fat compartment vessels. Intraoperative prevention is achieved with careful hemostasis. Treatment of superficial hematomas includes cold compresses and elevation of the head of the bed. Stable large hematomas may be clinically monitored for 7–10 days for resolution. Severe cases may cause fibrosis and scarring. Actively expanding hematomas necessitate urgent surgical exploration for evacuation and hemostasis.

15.7.5 Eyelid Crease Asymmetry

Following blepharoplasty, patients may note asymmetry between the left and right eyelid creases (Fig. 15.4). The asymmetry may or may not be a result of the surgery. Often patients are not aware of the natural asymmetry between their eyes, so high-quality preoperative photographs documenting asymmetry can be helpful in counseling patients. However, some cases of asymmetry can be attributed to errors in preoperative



Fig. 15.4 Asymmetric eyelid creases following upper eyelid blepharoplasty. Reprinted with permission from Elsevier (Whipple KM, Korn BS, Kikkawa DO. *Recognizing and managing complications in blepharoplasty. Facial Plast Surg Clin North Am.* 2013 Nov;21(4):625–37)

markings and assessment. A complete preoperative examination includes assessment for eyelid ptosis, globe proptosis, and thyroid eye disease. The surgeon should aim to maintain the anatomic location of the upper eyelid, if it is in the correct location prior to surgery. In cases with absent or abnormally located upper eyelid creases, the standard practice is to form the crease 7–8 mm superior to the lash line in Caucasian men and 8–10 mm above the lash line in Caucasian women. In traditional Asian blepharoplasty, the formation of the upper eyelid crease is set lower at 5–6 mm superior to the lash line in men and 6–7 mm above the lash line in women. Postoperative asymmetry in upper eyelid creases should be reassessed after initial swelling has resolved and adequate healing has occurred, usually 3–6 months after the surgery. A crease that is too low may be revised by setting the crease superiorly with an incision above the eyelid crease and subsequent fixation of the orbicularis to a higher point on the levator aponeurosis. A crease that is too high is more difficult to fix. Some advocate increasing the height of the other eye, whereas others recommend an inferior incision with advancement of preaponeurotic fat or placement of free fat pearls to prevent readhesion superiorly.

15.7.6 Blepharoptosis

Ptosis is frequently noted in the early postoperative period, but is not necessarily a consequence of the surgery. Often, preexisting ptosis is not

noticed preoperatively because severe dermatochalasis may confound the true marginal reflex distance-1 (MRD-1), which is defined as the distance between the center of the pupillary light reflex and the upper eyelid margin with the eye in primary gaze. Ptosis is defined as a MRD-1 of 2.5 mm or less, and even a change of 0.5 mm can be noticeable to patients. A thorough preoperative examination for ptosis includes measuring the MRD and palpebral fissure height and testing levator muscle action. In patients with ptosis noted preoperatively or intraoperatively with levator dehiscence, the surgeon should correct the ptosis concomitantly with blepharoplasty. Postoperative ptosis is usually temporary and has many possible causes including eyelid edema, local anesthetic side effect, ecchymosis, or hematoma. Rare cases of permanent ptosis may result from muscle or aponeurotic changes due to prolonged lymphedema or hematoma or accidental disinsertion of the levator muscle during resection of the pretarsal orbicularis or preaponeurotic fat. To prevent injury to the levator aponeurosis, conservative dissection beyond the preaponeurotic fat compartments is advised. Corrective surgery should be delayed for at least 3 months given the possibility for spontaneous improvement within 3 months. Persistent ptosis beyond 3 months may require surgical correction with levator advancement.

15.7.7 Lid Malposition (Entropion, Ectropion)

Lid malposition following blepharoplasty is a relatively common complication. Malposition encompasses retraction (lower eyelid margin malposition without eversion), ectropion (malposition with lid margin eversion away from the globe), and entropion (lid margin inversion). Retraction and ectropion can result from excessive skin resection, postoperative scarring, edema, or hematoma leading to increased downward force on the lid, imbrication of the orbital septum, or orbicularis paralysis; entropion can result from posterior lamellar deficiency. Complications of ectropion include exposure

keratopathy, pain, dryness, and irritation from conjunctival exposure.

Risk factors include preoperative lower lid laxity, globe proptosis, hypoplasia of the malar eminence, high myopia, and thyroid eye disease. Preoperative assessment should include evaluation of existing horizontal lid laxity (e.g., with a positive snapback test demonstrating delayed lid return to position upon release after being pulled away from the globe). An appropriate tightening technique (e.g., lateral canthopexy) should be performed at the time of blepharoplasty if lower lid laxity is detected.

Artificial tears, ophthalmic lubricant, topical steroid ointment, and local massage constitute initial management of lower lid retraction. If excessive skin has been retracted, suture removal in the first 2–3 postoperative days can be done to allow wound granulation of a portion of the eyelid. Though not aesthetically ideal, allowing for granulation can prevent severe bowing or ectropion subsequently requiring skin graft.

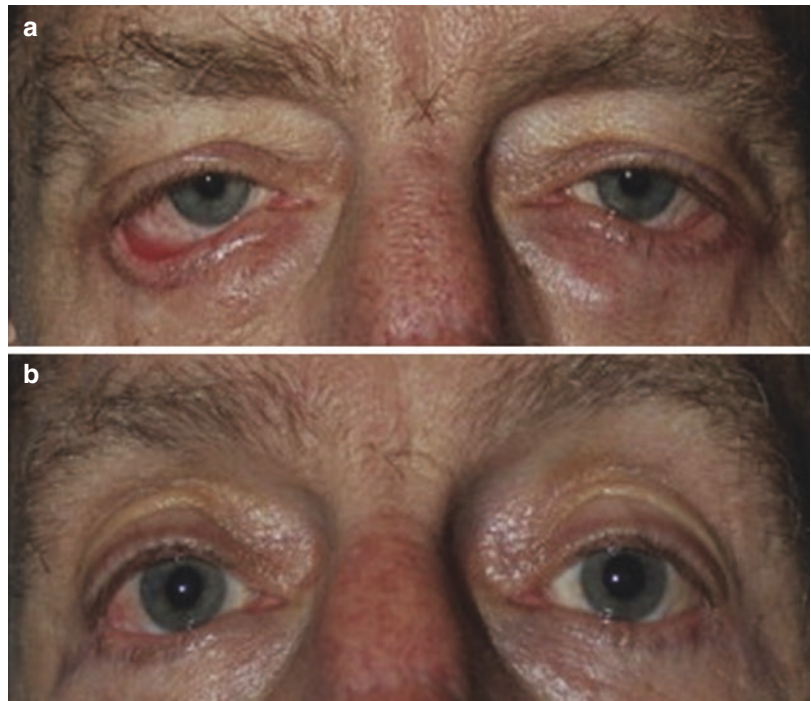
Management of severe lower lid malposition is a surgical challenge; successful reconstruction

relies on careful anatomical evaluation. Standard repair entails anchoring the lateral canthus through canthopexy, canthoplasty, canthal plication, and tarsal strip. In cases of severe lower lid retraction, a transconjunctival approach to the orbital septum followed by release of scar tissue and use of a posterior lamellar graft should be performed as well (Fig. 15.5). A full-thickness skin graft may be required for ectropion repair; autologous tissue option includes hard palate, dermis fat, and acellular dermis. Entropion arising from posterior lamellar deficiency can be repaired by addition of posterior lamella; successful repair has been reported with use of hard palate or alloplastic graft material.

15.7.8 Wound Dehiscence

There are many possible causes of wound dehiscence including accidental trauma, infection, wound healing, high suture tension, and premature suture removal. Patients should be counseled to avoid heaving lifting, bending over, vigorous

Fig. 15.5 Eyelid retraction following lower eyelid blepharoplasty (a). Repair of lower eyelid retraction with cross-linked porcine dermal collagen implants in bilateral lower eyelids (b). Reprinted with permission from Elsevier (Whipple KM, Korn BS, Kikkawa DO. *Recognizing and managing complications in blepharoplasty. Facial Plast Surg Clin North Am.* 2013 Nov;21(4):625–37)



exercise, and excessive facial muscle contraction including eyebrow raising. A nighttime eye shield may help prevent inadvertent rubbing during sleep. Lateral dehiscences are more common in both upper and lower blepharoplasty. Prevention of wound dehiscence includes use of postoperative Steri-Strips and use of nonabsorbable sutures as opposed to absorbable sutures. Sutures should be removed 5–7 days after surgery. Minor dehiscence smaller than 1 cm may be observed for healing via secondary intention. Larger dehiscences or persistent small dehiscences should be repaired using interrupted nonabsorbable sutures in the next largest caliber from the original sutures. Reapproximation of the orbicularis muscle using buried absorbable sutures may help to reduce tension on closure. Wounds which have been open for over 24 h should have their edges carefully deepithelialized before resuturing.

15.7.9 Hyperpigmentation

Some patients may note hyperpigmentation after eyelid surgery, which may be transient or longer lasting. Transient hyperpigmentation may result from hemosiderin deposition from slowly resolving ecchymosis or hematomas and may be prevented by meticulous intraoperative hemostasis and evacuation of persistent postoperative hematomas. Longer-lasting causes of hyperpigmentation include postinflammatory pigmentary changes, which promote melanin deposition in the epidermis. Sun exposure may worsen hyperpigmentation, and ultraviolet light-blocking sunglasses may be protective. Treatment of mild to moderate postinflammatory hyperpigmentation includes topical hydroquinone 4% or hydrocortisone 2.5%.

15.7.10 Suture Granuloma

Inflammation around a suture may present a few weeks postoperative as a nodular erythematous encapsulation, often called a suture granuloma. They can be painful or painless and are more likely to occur near the suture knots. Suture

granulomas are commonly self-resolving, but topical corticosteroids may be helpful in some cases to reduce inflammation. If conservative management fails, the surgeon may remove the suture and drain the inflammatory contents.

15.8 Muscle

15.8.1 Strabismus and Diplopia

Diplopia is an uncommon but serious complication following blepharoplasty. Transient diplopia may be caused by conjunctival edema, hematoma, transient paresis of extraocular muscles or muscle contracture, wound-related inflammation, corneal abrasion, or disruption of tear film due to ophthalmic ointment leading to monocular diplopia. Such cases are self-limited, intermittent, and may clear with blinking. Persistent binocular diplopia has been reported to occur in 0.2% of cases and may result from extraocular muscle injury leading to iatrogenic strabismus. Most commonly, injury involves the inferior oblique muscle, whose position between the nasal and central fat pockets renders it vulnerable to injury during dissection and cautery of orbital fat pads. Injuries to the superior oblique tendon and muscle, medial rectus, and inferior rectus have also been reported. An iatrogenic Brown syndrome may also occur from injury to the trochlea (“pulley” of the superior oblique), which is located just posterior to the orbital rim. Additional causes of persistent diplopia include over-resection of fat tissue, aggressive use of cautery, and persistent hemorrhage leading to neuromuscular compression and ischemia (e.g., superior orbital fissure syndrome).

Intraoperatively, the inferior oblique should be identified and strictly protected from injury during fat excision and dissection. Careful isolation of fat pads prior to removal is critical; retraction of any fat pad should not result in globe movement, which may represent muscle impingement.

Initial assessment of diplopia should differentiate monocular from binocular diplopia. Disruption of the tear film, corneal injury, or

ointment may result in monocular diplopia; symptoms improve with blinking, and the diplopia usually resolves within a few days after surgery. Significant diplopia should be closely observed with monthly orthoptic measurements in all fields of gaze to monitor improvements in ocular motility. Diplopia that persists after 2 months warrants referral a strabismus specialist for surgical exploration and strabismus surgery if necessary. Disabling diplopia in primary gaze can be temporarily managed with prismatic spectacles.

15.9 Fat

15.9.1 Excessive Removal and Hollowing

Overaggressive resection of upper eyelid fat can lead to a hollowed superior sulcus with an aging effect on the face. Thus, upper and lower blepharoplasty cosmetic techniques have trended toward fat conservation. The medial and central fat pads along with the eyebrow fat pad comprise the volume of the upper eyelid. A youthful upper eyelid is full (especially in the lateral aspect); over time, the central fat pad atrophies and the medial fat pad becomes more prominent. Due to variations in carotenoid composition, the medial fat pad is lighter in color. Repositioning or removal of the medial fat pad may be warranted if a patient has a nasal teardrop-shaped fat accumulation. Prior to surgery, examination of the medial and lateral eyelid contour to note the anatomy of the medial and central fat pads is critical to ensure appropriate amount and location of fat removal during blepharoplasty.

Conclusion

Blepharoplasty, like every other surgical procedure, is subject to complications even when performed under the most experienced hands. Both functional and cosmetic complications can arise, and optimal results demand meticulous attention to preoperative, intraoperative, and postoperative measures to decrease risk and promptly recognize and manage compli-

cations when appropriate. Expert knowledge of eyelid anatomy and thorough awareness of potential complications of blepharoplasty will minimize complications and more consistently yield optimal aesthetic and functional outcomes.

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Complications Associated with Rhytidectomy (Facelift Surgery): Avoidance and Correction

Daniella Vega, Sami Tarabishy, Jacob Wood, and Charles L. Castiglione

Abstract

Rhytidectomy (facelift surgery) is an aesthetic rejuvenation surgery of the face which can remove excess sagging skin, minimize the appearance of creases and marionette lines, and correct facial fat depletion or double chin. It is the seventh most commonly requested aesthetic procedure (126,713 procedures performed in 2014) and the most commonly performed aesthetic surgery in patients greater than 65 years old (<http://www.surgery.org/media/statistics>; Gupta et al., *Aesthet Surg J* 36:1–13, 2015). Rhytidectomy generally has a very high satisfaction rate provided patients are properly selected and complications are minimized. This chapter will review important aspects of the preoperative assessment and intraoperative procedure focused on the identification and minimization of complications related to rhytidectomy surgery.

16.1 Introduction

Rhytidectomy (facelift surgery) is an aesthetic rejuvenation surgery of the face which can remove excess sagging skin, minimize the appearance of creases and marionette lines, and correct facial fat depletion or double chin. It is the seventh most commonly requested aesthetic procedure (126,713 procedures performed in 2014) and the most commonly performed aesthetic surgery in patients greater than 65 years old [1, 2]. Rhytidectomy generally has a very high satisfaction rate provided patients are properly selected and complications are minimized. This chapter will review important aspects of the preoperative assessment and intraoperative procedure focused

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on the identification and minimization of complications related to rhytidectomy surgery.

Rhytidectomy has a history dating to the early twentieth century. Procedures were initially limited to simple cutaneous incision and closure without manipulation of subcutaneous or deeper tissue. Although these surgeries were simple and easy to perform, they lacked deep tissue control and longevity. It wasn't until the early 1970s when more elaborate techniques involving dissection and lifting technique of the SMAS (superficial musculoaponeurotic system) layer were described by Skoog [3]. Since its description, SMAS surgery has been modified several times including SMAS plication, minimal access cranial suspension lifts, lateral SMASectomy, extended SMAS lift, composite facelifts, and a subperiosteal facelift [4]. Procedures have advanced with the goal of better results through minimizing scarring, restoring subcutaneous volume, providing long-lasting effects, and preventing complications.

16.2 Preoperative Assessment

Similar to any surgical procedure, rhytidectomy carries a risk of complications, the most common being hematoma [2]. Other complications include skin flap necrosis, nerve injury, scarring, and rarely infection [5]. Complications and unsatisfactory results can be minimized by careful preoperative assessment/surgical planning, specific intraoperative surgical maneuvers, and complete postoperative management.

Preoperative assessment and surgical planning are perhaps the most important steps in assuring proper patient selection and minimization of complications. The main indication for surgery is the potential for facial rejuvenation in a selected patient. Absolute contraindications include bleeding diatheses and ASA classes IV and V. Relative contraindications include diseases which promote poor wound healing such as diabetes, a history of smoking, or patients on long-term steroids [6].

A good preoperative assessment elucidates indications/contraindications for rhytidectomy and should include evaluation of the chief complaint, a thorough history and physical exam, and clear patient expectations.

The chief complaint establishes an indication for surgery and also clarifies patient expectations early. Patients may present with complaints of sagging skin in the midface/jawline, nasolabial fold creases, marionette lines, facial fat loss/displacement, jowls, a double chin, or creasing/sagging in the neck area. All of these complaints would be indications for a rhytidectomy [6]. Other surgeries may be indicated if a patient is complaining of other facial issues such as brow ptosis or excess eyelid skin, in which case brow lift and/or blepharoplasty should be considered.

A good history can also ascertain contraindications for surgery and should ask about the following:

- Prior complications with anesthesia/surgery
- History of easy bruising or bleeding
- Current use of anticoagulants
- Smoking
- Herbal supplements
- Steroids/chemotherapy
- Diabetes mellitus

These important questions help evaluate for contraindications which may predispose to bleeding, anesthetic, or wound healing complications. For example, smoking increases the risk of post-procedure skin necrosis by 12.5× when compared to nonsmokers [2]. This is thought to be due to an acute vasoconstrictive effect, which leads to relative tissue hypoxia and subsequent delayed wound healing.

Based on the history given, physicians should work up potential coagulopathies and evaluate sources of complications in prior surgeries to determine if the patient is suitable for surgery. Aspirin and NSAIDs should be discontinued at least 2 weeks prior to surgery, smoking at least 4 weeks prior to surgery, herbal supplements at least 2 weeks prior to surgery, and steroids/chemotherapy at least several weeks before surgery [7].

Furthermore, patients should be screened for psychological illness such as body dysmorphic disorder and personality disorders, as psychological health is as important as physical health in cosmetic surgical outcomes [8].

Next, a thorough physical exam to assess for signs of facial aging and suitability for rhytidectomy

should be performed and include evaluation of the following:

- Bone structure (malar, jaw, chin)
- Facial asymmetry
- Facial nerve function
- Hair and hairline characteristics
- Ear/earlobe position
- Submandibular glands
- Platysma bands
- Skin tone/laxity/atrophy
- Facial rhytids
- Previous scars
- Dyschromia/telangiectasias
- Facial fat (malar fat pad, midface, submental)
- Nasolabial folds
- Deep neck lines

Lastly, in your preoperative assessment, you must establish patient expectations and goals of surgery. As in all aesthetic procedures, it is important to see if patients have realistic expectations for outcomes and recovery, as well as awareness of the possible need for revisions. Physicians should first listen to patient expectations and then mutually create realistic outcome goals with them. It is imperative that this dialogue is honest and open and that it occurs prior to surgery in order to build rapport with the patient and avoid perceived sub-optimal outcomes postoperatively [7].

16.3 Intraoperative Considerations

Every portion of your procedure is important in avoiding intraoperative complications from induction of anesthesia to a smooth extubation. After induction of anesthesia, the endotracheal tube can be secured to the central incisors in order to avoid tape to the face, which can distort structures. Careful patient positioning helps with proper visibility and sterile technique. Local anesthesia should be injected with enough time to allow the epinephrine to take effect before incisions are made. The patient should be marked carefully, and incision should be made with precision. Incisions are typically made with a #15 scalpel, flaps elevated with tenotomy scissors for the first few cen-

timeters and then facelift scissors, taking care to avoid trauma to the flap and keeping the flap at an appropriate thickness. One can transilluminate the flap in order to ensure the proper plane of dissection. There should be some subcutaneous fat, which the light will shine through. If the flap is too thick, the light will dim, and the surgeon will need to adjust his dissection. Dissection that is too deep may result in injury to the parotid gland [7, 9].

Other critical structures that can be injured during flap dissection include the various nerves and vessels. The temporal branch of the facial nerve is most vulnerable if dissection is too deep anterior to the temporal hairline. The marginal mandibular and cervical branches of the facial nerve are vulnerable to injury if dissection distal to the mandible extends beneath the platysma. Posterior neck flap dissection should be done very superficially to avoid injury to the great auricular nerve and external jugular vein. Even more posteriorly, the spinal accessory nerve is also susceptible to injury [7, 10].

There are additional surgical maneuvers that can be done in order to improve outcomes. Submental or jowl liposuction can be used alone or with lipotransfer to improve contour and symmetry. Plication of SMAS flap is done to create a more youthful cheek and blunt the nasolabial fold but must be done carefully in order to avoid damage to underlying structures [7]. Plication or transection of the platysma can also be done to restore a youthful appearance to the neck and, again, must be done with attention to detail as to avoid injury to deeper structures mentioned before.

16.4 Postoperative Complications

16.4.1 Hematoma

Hematoma is the most common complication following rhytidectomy. Expanding hematoma generally occurs in the first 24 h after surgery and should be promptly addressed to avoid flap necrosis secondary to tissue ischemia and edema [11]. Considerations in avoidance of hematoma include the use of compressive dressings (though not too restrictive as this may lead to tissue ischemia), +/- drains, smooth extubation, avoidance

of anticoagulant/antiplatelet medications, and adequate control of blood pressure. Treatment consists of prompt surgical exploration, hematoma evacuation, and control of bleeding. Bedside evacuation for smaller unilateral hematomas has also been described with success in patients with well-managed blood pressure [12].

It is extremely important to avoid hypertension in the postoperative period in order to try to prevent hematoma formation. Patients should be instructed to take their antihypertensive medications the morning of surgery, and the patient's blood pressure should be kept under control during and after the case, with the use of beta-blockers, calcium channel blockers, or alpha agonists. Incidence of hematoma is about 3% in patients without a history of hypertension but increases to approximately 8% in those with a history of hypertension and in males [13]. In addition, factors that may increase blood pressure should also be adequately controlled, including pain, anxiety, nausea, and vomiting [12].

The incidence of hematoma is increased in patients taking anticoagulant, antiplatelet, or NSAID medications. Intravenous desmopressin can be given intraoperatively if a patient is suspected of having impaired platelet function [14]. Additionally, some supplements that may have antiplatelet or anticoagulant properties include garlic, ginger, vitamin E, fish oil, glucosamine, and green tea. All of the abovementioned agents should be avoided and discontinued 2–3 weeks prior to surgery [15]. Additionally, concurrent open anterior platysmaplasty with rhytidectomy significantly increases the risk of postoperative hematoma [16].

16.4.2 Infection

Infection is a very rare complication of rhytidectomy. Antibiotics are often given perioperatively that target skin pathogens, typically a cephalosporin or vancomycin, though there is little evidence to support their usefulness [7]. Preauricular infections can occur from *Pseudomonas aeruginosa* that can colonize the otic canal and typically responds to oral ciprofloxacin [16]. Infection may also arise from a stitch abscess, which

should respond to stitch removal and local wound care. Significant erythema of the auricular cartilage should be treated with oral antibiotics that cover gram-positive skin flora as well as *Pseudomonas* [7].

16.4.3 Nerve injury

Nerve injury with rhytidectomy has been reported at rates of 0.7–2.5% [9]. If identified intraoperatively, it is best to repair the nerve that has been injured. Nerves amenable to repair include the facial nerve, great auricular nerve, and spinal accessory nerve. The distal end of the nerve injured can be identified via nerve stimulation if paralytic agents are not used during anesthesia. Once it is identified, the nerve can be repaired using a 6-0 nylon suture or similar [7]. A rare complication after nerve injury/repair is a painful neuroma [11].

Mild transient facial paresis is common for up to 12 h after surgery secondary to local anesthetic effects [17]. Prolonged nerve injury can be identified days later and is usually secondary to traction, cautery, sutures, or surgical division. These generally will spontaneously recover in 3–4 months [11, 17]. Permanent nerve injury is likely if function does not return within 2 years and can be seen in both sensory and motor nerves. The most common sensory nerve injured is the great auricular nerve. If identified and repaired during surgery, return of sensation is common but may take 12–18 months for full recovery. The temporal branch of the facial nerve is the most commonly noticeable injured motor nerve, resulting in permanent damage in 0.1% of cases, though may resolve within 18–24 months after injury [7]. Likely the buccal branch of branch of the facial nerve is injured more often than the temporal but is not noticed secondary to arborization and rich collateral innervation [3, 9]. Paralyzing agents such as botulinum toxin can be used to recover facial symmetry while motor nerves recover or as a permanent solution if recovery does not occur [10]. Additionally, if the asymmetry caused by motor nerve injury is more severe, additional surgery such as brow lift may be an effective solution for excessive brow ptosis on

the affected side of an injury to the temporal nerve [7]. Other nerves that can be injured include the marginal mandibular, zygomatic, or cervical branches of the facial nerve and the spinal accessory nerve, with permanent damage typically occurring in less than 1% of cases [18].

16.4.4 Flap Necrosis

Skin flap necrosis is a rare complication following rhytidectomy. Preoperative patient factors such as smoking and diabetes, intraoperative issues such as flaps under excessive tension and thinly dissected flaps, and postoperative issues such as hematoma and tight postoperative dressings may all increase the incidence of flap ischemia [19]. Drains can be left postoperatively in order to decrease the amount of serum fluid collection beneath the flap, although this has not been shown to have an effect on significant hematoma development or evacuation [10]. Decreased intake of salt and water can also decrease the amount of tissue edema that occurs postoperatively [20].

Tissue ischemia often occurs in the periauricular region and can be seen as distinct area of ecchymosis. Treatment for flap necrosis and skin slough should generally be conservative local wound care [10]. Sometimes adjuncts such as nitropaste can be used to reduce the chances of full-thickness skin necrosis. Superficial epidermolysis often heals uneventfully. Areas of necrosis should be allowed to demarcate fully before any tissue debridement occurs. Additionally, the debrided area should be protected with an antimicrobial ointment until secondary healing has occurred [7]. The temptation to perform early wound/scar revision must be resisted, as the tissues must mature and soften before any further manipulation occurs.

16.4.5 Poor Scar Positioning and Other Deformities

Care must be taken to be cognizant of and address common complications related to poor scar posi-

tioning, ear deformities, hypertrophic or keloid scarring, contour deformity, and alopecia/hair-line distortion.

Pixie earlobe is a classic deformity related to overcutting the skin cutback at the base of the earlobe when delivering the earlobe from under the skin flap as the excess is excised. When this happens, the lobe is sutured more inferiorly to the cheek skin and results in an elongation of the lobe along the cheek, leaving an elfin or “pixie” appearance that negatively impacts the aesthetics of the facelift [21].

Hypertrophic or keloid scars can be treated with kenalog injection. Widened, irregular, or very obvious scars can be treated with scar revision once the scar has matured and relaxed, at least 6 months or more after rhytidectomy [10].

Contour deformities can be common in the immediate postoperative period but generally subside as edema resolves and healing ensues. They can be seen for several months, and gentle local digital massage can be used to speed resolution. If contour deformities persist, dermal fillers and fat injection can be used to improve the defects and create better facial symmetry [7].

Alopecia and hairline asymmetry can result from poor incision placement, excessive tension on skin flap, damage to hair follicles from electrocautery or failure to bevel the incision, and elevation or elimination of the temporal hair tuft and temporal hairline. Treatment includes the use of topical minoxidil to shorten temporary loss. Permanent alopecia is addressed with excision of the area or definitive hair replacement surgery, which consists of the insertion of single hair follicular units into the affected areas. Definitive treatment should be delayed until it is certain that the loss is permanent, usually about 12 months [7].

16.4.6 Parotid Fistula

Iatrogenic injury to the parotid gland is an uncommon complication of rhytidectomy but can result in postoperative complications of parotid sialoceles and fistulae. Presenting symptoms include edema, serous drainage through incisions or wounds, erythema, and pain. Treatment includes early drainage and observation, as well

as botulinum toxin administration into the parotid gland if symptoms persist [9].

16.4.7 “Unfavorable Result”

It is important to remember that patient selection is crucial in obtaining satisfactory results. No matter the quality of the surgery and outcome, there are some patients who will never be satisfied with their result. This once again drives home the point that patient goals and expectations must be adequately established and explored prior to surgery.

Unrecognized unilateral hematoma 2 days after facelift, requiring emergency evacuation



Close-up view of same hematoma with ischemia and impending skin necrosis



Conclusion

Rhytidectomy or facelift is the gold standard procedure for surgical facial rejuvenation. Generally, the satisfaction rate is high but only if the surgeon performs a thorough preoperative assessment, plans properly, performs meticulous surgical technique, and provides detailed postoperative management. The goal is to avoid any complication, but if one does occur, manage it properly. As with any aesthetic surgery, managing patient expectations is paramount, whether or not a complication does occur.

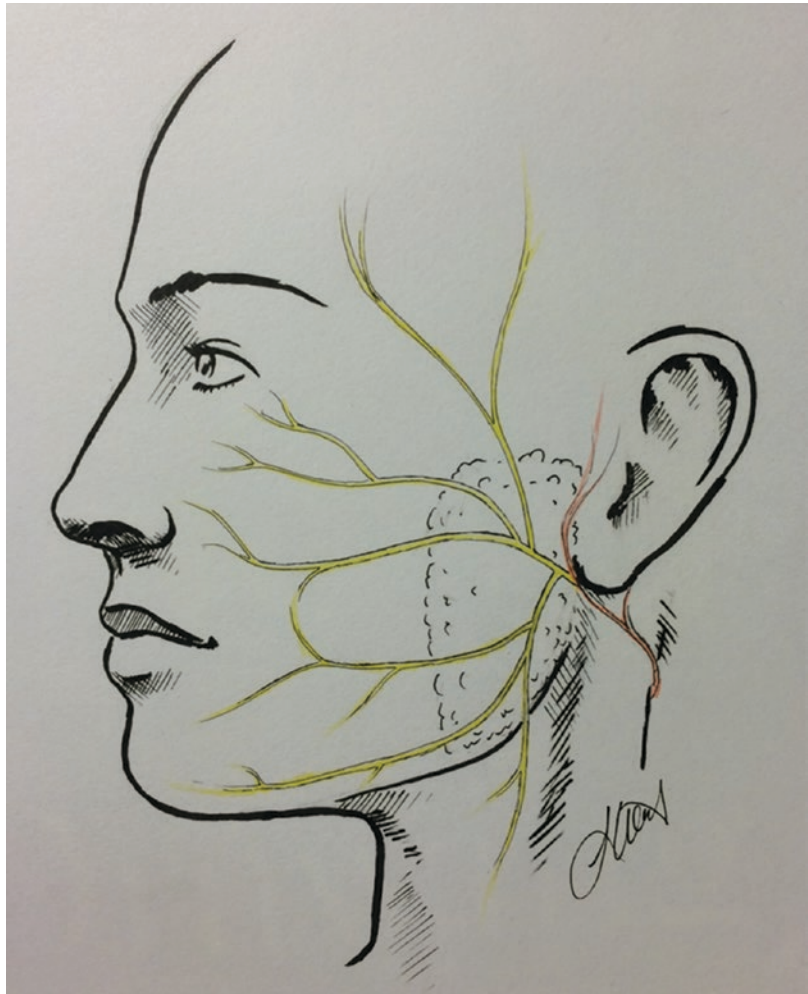
Same patient with unsightly scar after conservative management of skin necrosis



Unsightly scar from skin necrosis caused by tight head dressing



Critical Structures:
Parotid gland and facial
nerve (Illustrated by
Jacob Wood, MD)



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Abstract

Neck lift or cervicoplasty is a common surgical procedure that improves the soft tissue contour of the neck and the jawline to create a youthful and aesthetically pleasing cervicomental angle. A wide range of complications, while rare, can and do occur following neck lift surgery including acute hematomas requiring emergent surgical evacuation to mild self-resolving contour irregularities. It is critical to understand the various etiologies of neck rejuvenation complications in order to devise appropriate prophylactic strategies as well as timely interventions to minimize operative risks. Moreover, appropriate patient selection, mastery of head and neck anatomy, meticulous attention to surgical technique, and a comprehensive postoperative care are all critical factors in minimizing and preventing neck lift surgery complications.

17.1 Introduction

The cervical region is quite possibly the most common reason for patients between the ages of 40 and 75 years to seek consultation for maxillofacial cosmetic surgery. The maxillofacial anatomy

inclusive of the mandible, the position of the airway and its supporting structures and musculature, the subcutaneous fat and the skin vary widely in the presenting patient population. The skin is like a canvas, draped over the deeper structures. Careful preoperative evaluation is paramount, as

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the diagnosis followed by proper recommendations for treatment is necessary to achieve consistent patient satisfaction.

Options for correction or improvement of the cosmetic appearance of the cervical region include nonsurgical and surgical procedures. Nonsurgical treatment includes cosmeceuticals, chemical peels, laser skin resurfacing, ultrasonic therapy, and dermabrasion. Surgical interventions include submental lipoplasty, chin augmentation, isolated anterior only neck lift (platysmaplasty), and complete neck lift.

The cosmetic goals of cosmetic intervention of the neck are to improve:

1. The cervicomental angle
2. Laxity of skin of the jawline to clavicles
3. Jawline definition
4. Cervical banding
5. Submental as well as supra- and/or submental adiposity
6. Support of the submandibular gland

Evaluation of the maxillofacial cosmetic patient for neck modification should begin with obtaining a photographic record. Frontal, right and left lateral, and right and left oblique photos should be standardized with ideal and identical lighting with the same camera and lens at the identical distance from the patient. The face must be held parallel to the floor. The camera must also be held at a consistent Frankfort horizontal. Ideally, one individual should be responsible for all photos and any necessary cropping. No retouching, alteration, or modification of the digital images should ever be performed in the medical documentation without a specific notation in the medical record and the justification for modification. All patients must agree to photographic documentation, and use and restrictions of these images must be detailed and consented for or against.

An objective and reproducible analysis of the cervical region should then be performed with these images. Ellenbogen and Karlin created a list of five visual criteria upon which they felt the neck should be evaluated [1]. Most notably were the clarification of the mandibular border and a cervicomental angle of 105°–120°. Zweig [2] contributed the only

specific text devoted to the analysis of the cervicofacial region in 2000 in the *Atlas of Oral and Maxillofacial Surgery*. Menton is the most inferior point of the mental region curvature noted on lateral images. The cervical point is located where a line tangent to the menton intersects another tangent drawn along the anterior border of the neck. The zero meridian line is drawn from nasion to subnasale. The lower face is then compared to the zero meridian line with a line drawn from the subnasale to pogonion, to establish whether the chin is protruding or retruding. The shape of the chin and position of the chin must be assessed relative to the support it provides for the neck region and the submental distance from the chin to the neck [3]. Dedo is credited with a classification system of the neck ranging from I–IV, ranging from normal to skin laxity, followed by fat accumulation, platysmal banding, retrognathia, and a low hyoid bone [4]. Ideally, the hyoid bone should be a minimum of 20 mm below the inferior border of the mandible and not lower, as well as no farther anterior than the midbody of the mandible [5].

Age is a relative factor in assessment and treatment planning the most ideal treatment for the cervical region. The incidence of complications and satisfaction with results of surgical intervention has been linked to advancing age. This may be a result of increased rates of loss of elasticity, progressive hormonal incompetence, maxillofacial patterns of resorption of supportive bone [6, 7], or general medical complications that increase with age such as hypertension. Specific discussion regarding longevity of the results and increasing complications should be detailed and documented. Skin elasticity should be assessed and documented. Reduced synthesis of fibrillar collagen types I and III is a characteristic of chronologically aged skin and is enhanced in photodamage. This has been well-described using histological and ultrastructural approaches in the past, and metalloproteinases (MMPs) are upregulated in the skin by exposure to chronic UV radiation. This is likely responsible for producing collagen fragmentation in sun-damaged skin. During natural or chronological aging of the skin, the same MMPs that are upregulated acutely in response to UV radiation are gradually

increased [8]. The end result is clinical thinning of the dermis, crepe paper appearance along with loss of jawline definition, laxity of the retaining ligaments, and deposition of fat in either the subcutaneous tissue or subplatysmal planes [9]. Neck banding is a common source of patient concern and may or may not be associated with the platysmal muscle. Identification of ptotic submandibular glands is critical to patient preoperative discussion and the surgical options for management and the inherent risks and complications associated with their treatment.

17.2 Submental and Cervical Lipectomy

The act of tumescent infiltration with Klein's solution is followed with 10 min to achieve hemostasis and anesthesia prior to commencing with surgery. Infralobular stab incisions and a single submental incision are placed 3.0 mm posterior to the submental crease. A 1.5–2.5 mm aspiration cannula is used to suction at 19.0 mmHg. A layer of subcutaneous fat is left 3.0–5.0 mm in thickness on the overlying skin in order to minimize dermal fibrosis and attachment to superficial fascia. This minimizes the risk of cosmetic deformity and the artificial immobility of the cervical skin postoperatively. "What is left behind is more important than what is removed" is often espoused by seasoned surgeons who employ lipoaspiration. Fat aspiration is often minimal, and generally, it is believed that the passing of the cannula causes sufficient trauma to the fatty layer and dermis to achieve the desired result and patient satisfaction. The use of the open liposuction or directional cannula is often employed. This cannula should be directionally controlled so as to aspirate on the internal or deep side rather than turned to the superficial or dermal side. In addition, care must be utilized in approximating the marginal mandibular nerve anterior to the facial artery and submandibular gland region by limiting aspiration at or above the mandibular border. Transient paralysis of the marginal mandibular branch (MMB) of the facial nerve is widely reported to occur 0.003–1% of cervical region surgery [10]. The postop-

erative compressive dressing is thought by many to be of significant importance. About 1/4"–1/2" closed cell foam padding is placed in contact with the skin under a four-tailed Barton bandage using CoFlex or similar product. Care must be emphasized as to limit the pressure at the level of the hyoid as the skin in total thickness of this tissue is often extremely thin and can be subject to pressure necrosis from dressing pressure. We routinely remove this dressing at 6 h after the procedure and follow this with 4 days of elastic and velcro facial garment wear, as tolerated for 24 h a day.

The patients who receive lipoaspiration of the submental area are often improved but minimally. This technique does not address the platysma, the subplatysmal fat, or the submandibular gland ptosis or microgenia. This procedure is ideal for patients with an ideal hyoid position, excellent chin position and plentiful subcutaneous fat. The most frequent error in physician management of the submental region is error in judgment in recommending this procedure when the patient would ostensibly gain higher satisfaction from a more definitive and often aggressive procedure.

17.3 Chin Augmentation

Cephalometric evaluation of the bony chin and the overlying soft tissue is necessary but not mandatory. Clinical examination of the lateral photos with the Frankfort plane horizontal to the floor is routinely sufficient. A photo with the lips in repose and closed may indicate mentalis strain. The soft tissue response to bony or solid silastic implant is a ratio of 1:1; for each 1 mm of chin augmentation, the soft tissue menton protrudes 1 mm. Utilizing a 3.5 cm incision approximately 1.0 mm posterior to the submental crease, access is directly to the inferior border of the mandibular symphysis. A subperiosteal elevation is achieved and lateral tunnels completed to a depth of 4.0 cm lateral to midline immediately superior to the inferior border of the mandible. The skin is re-prepped with Povidone-iodine solution, and the chin implant is immersed in the same solution. The distal end of the implant is inserted into the subperiosteal tunnel and the

midpoint is grabbed by the assistant with a large toothed forceps to prevent it from herniating out the hole, while the surgeon grasps the opposite end with a fine forceps and tucks the proximate end into the subperiosteal tunnel. This midline is fixated with 3-0 Vicryl suture to ensure proper positioning of healing. This entire process takes an average of 7.0 min when performed efficiently and frequently.

17.4 The Isolated Anterior Neck Lift

All of the above lipoaspiration is often performed prior to placement of a 3.5 cm incision approximately 2–3.0 mm posterior to the submental crease. The subcutaneous plane is completely dissected leaving once again 3.0–5.0 mm of fat on the skin flap. This creates an optical cavity for complete management of the superficial muscles and fat of the submental region. Electrocautery is used judiciously to minimize thermal injury and thus reduces postoperative pain [11]. The midline decussation and submental fat is directly excised with electrocautery. The medial borders of the platysma muscle are identified and extensive subplatysmal dissection is employed as necessary to treat the submandibular glands and advance the laterally based flaps to the midline without tension. At the level of the cervical crease, a cut back procedure is utilized to completely transect the entire width of the platysma muscle. At the most inferior aspect of the medial border of the cephalad platysmal flap, a 4-0 PDS suture is buried and fixated to the investing fascia of the hyoid bone. The corset suture is run to the menton and back again to the origin at the cervical point and tied. If the submandibular glands are found to be ptotic, the clinical choice must be made to either resect the superficial lobe of the gland with its inherent risks and complications or to support the gland with the medial corset platysmaplasty [12]. A plethora of options and variations in technique have been reported with similar results [13]. The most obtuse cervicomental angle often benefits from complete platysmal resection, with anterior digastric resection and submandibular gland suspension or resection.

17.5 Complete Neck Lift

The above anterior procedure can be supplemented with an inferior lobular to retroauricular skin incision in order to manage skin redundancy beyond the capability of fibrosis and elasticity to contract. This retroauricular access also allows the surgeon to add a posterior vector of traction to the posterior border of the platysma and fixate it to either the mastoidal superficial fascia or the periosteum for added dramatic and long-lasting effect. Despite clinical research results of patient satisfaction and long-lasting results [14], we find the Giampapa suture technique to be unsatisfactory with patient complaints of “strangulation” or the artificial appearance it creates at the cervical point.

There are primarily two different types of complications of neck procedures: immediate postoperative and delayed

Immediate postoperative complications would include:

1. Hematoma
2. Seroma
3. Infections
4. Postoperative nausea
5. Psychological distress from the compressive head wrap
6. Ischemic necrosis of skin
7. Dehiscence of the wound
8. Sensory loss
9. Motor nerve dysfunction
10. Asymmetry
11. Poor postoperative pain management
12. Sialocele
13. Skin perforations

Delayed-onset complications include:

1. Fibrosis
2. Cobra neck deformity
3. Recurrent banding
4. Facial asymmetry
5. Poor scarring
6. Lack of longevity
7. Salivary gland ptosis
8. Bony restoration under chin implant
9. Venous thromboembolism

17.6 Strategies for Prevention

Some relative contraindications for neck procedures include (1) smoking and/or alcohol abuse; (2) collagen vascular disorders; (3) poor nutritional status; (4) anticoagulation bleeding disorder; (5) use of Accutane, high-dose steroids, or immunosuppressants; and (6) poor medical condition (e.g., uncontrolled hypertension, poorly controlled diabetes, significant chronic airway disease [CAD], significant chronic obstructive pulmonary disease [COPD]). All patients should be medically optimized prior to surgery.

17.7 Hematoma

Hematoma is the most common complication of neck procedures (Fig. 17.1). Frequency is reported from 1.0 to 9.0%. Rates in males are

more than seven times more frequently than female patients, thought to be due to the effects of testosterone and relative blood pressure volatility [15]. Any blood thinning medications or antiplatelet medications should be discontinued per the primary physician recommendations. Optimal blood pressure should be maintained intraoperatively and postoperatively [16]. Clonidine (a centrally acting alpha 2 agonist), 0.25 mg p.o. given at least 30 min prior to surgery, has been shown to reduce average intraoperative blood pressure, with the peak effects in 2–4 h. Beneficial properties include mild sedative properties, and its action is synergistic to other sedative agents, with postoperative effects lasting 12–16 h. Adverse side effects, including (postural) hypotension, bradycardia, lethargy, weakness, and somnolence, are dose dependent [17]. It stimulates alpha adrenoreceptors in the brain, which results in reduced sympathetic outflow



Fig. 17.1 39 year old female underwent upper blepharoplasty, open septorhinoplasty and anterior approach platysmaplasty. Presented on 17 hour post operatively with submental hematoma. This was managed with direct open aspiration and manual massage followed by handheld external ultrasound application of 1.0Hz, amplitude of 2.5 for 6 weeks

from the central nervous system. This reduction causes a decrease in peripheral vascular resistance, renal vascular resistance, blood pressure, and heart rate. Use of true tumescent fluid infiltration such as the Klein solution is now a standard of care in nearly all modern maxillofacial cosmetic procedures. Electrocautery for meticulous hemostasis is mandatory in open procedures. Postoperative compressive garments theoretically limit dead space and provide support for lax tissue immediately after surgery.

The majority of post-operative neck hematomas occur within 12 h after surgery. A minor hematoma with volume less than 3.0 cc requires observation and repeat examination as liquefaction occurs most commonly at 10–14 days after surgery. The clot formation should be amenable to an 18 g needle aspiration once liquefied in order to speed resorption and recovery and to limit fibrosis in response to the local irritation from the blood collection. A major hematoma may require a return to the operating room with either local, intravenous, or general anesthesia in order to completely evacuate the volume and directly control active bleeding with electrocautery or suture ligation. Placement of drains never prevents a hematoma from forming. Psychologically, drains reassure a patient that everything was done in order to prevent such an occurrence, yet hematomas do occur.

17.8 Infection

Bacterial infection after neck surgery is extremely rare. The need for the use of antibiotics after facial cosmetic surgery has been brought under question in recent years. Currently, if the case is determined to be clean, antibiotics are generally not recommended. If intravenous antibiotics are chosen, these should be infused within 60 min prior to incision in order to have the maximum therapeutic dose present at the most common introduction of bacteria. Skin flora most commonly includes:

- *Staphylococcus epidermidis*
- *Staphylococcus aureus*
- *Streptococcus mitis*
- *Streptococcus pyogenes*
- *Propionibacterium acnes*
- *Corynebacterium*
- *Acinetobacter*
- *Pseudomonas aeruginosa*

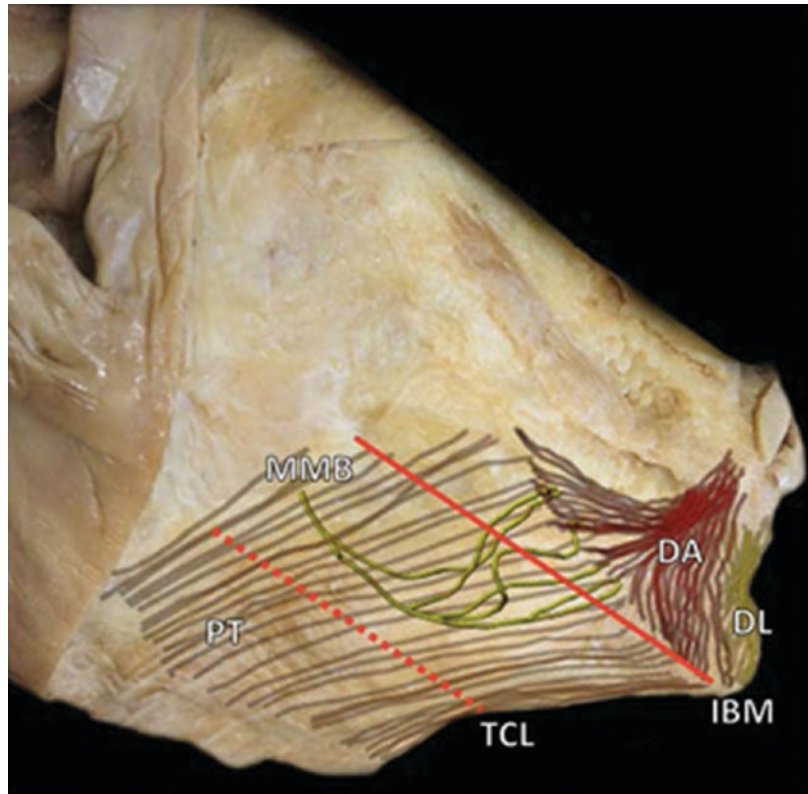
The predominant organisms causing surgical site infections (SSIs) after clean procedures are skin flora, including streptococcal species, *Staphylococcus aureus*, and coagulase-negative staphylococci [18]. The currently recommended presurgical antibiotic is a first-generation cephalosporin. If a patient has a history of allergy, the alternative recommended is clindamycin. Patients unable to take first-generation cephalosporins or clindamycin would be candidates for vancomycin and/or gentamycin. All patients that have had their incisions closed with resorbable sutures should keep them moist with either petroleum jelly or antibiotic ointment as a topical agent in order to properly resorb. Mupirocin ointment is ideal for use in patients with proven colonization with MRSA or culture results indicative of MRSA.

Any loculation in the cervical region must be addressed with basic surgical principles: incision and drainage along with institution of a broad-spectrum or empiric antibiotic while awaiting gram-stain results followed by further adjustment to a more narrow and appropriate choice of antibiotics, if need be, based on the culture and sensitivity results. Suspicion should be high for abnormal flora and previously unknown medical conditions as infection in these patients is extremely rare.

17.9 Facial Nerve Injury

The most common branch of the facial nerve that is injured during neck surgery is the marginal mandibular branch (MMB). The location of the

Fig. 17.2 Anatomical location and course of Marginal Mandibular Branch of facial nerve (MMB)



branch is well documented and remarkably consistent (Fig. 17.2) [19–25].

In order to avoid injury to the MMB with the resultant paralysis of the innervated muscles, it is most important to maintain the ideal plane of dissection throughout the procedure. This is most important at the region of the facial artery and vein as the nerve courses cephalad, crossing the inferior border of the mandible [26]. Temporary paresis of the muscles of facial expression in the region of innervation is routine and most commonly result from infiltrated tumescent solution. During the postoperative follow-up examination at 16–24 h, minor transient paralysis may be noted. Thoughtful and informative conversation about the anticipated resolution may avoid intervention. If the patient desires intervention, neurotoxins may be used to create symmetry by partially paralyzing the opposite side selectively.

The duration of paralysis can be modified based upon the concentration of units utilized.

17.10 Seroma

Serous accumulation of fluid is rare in the primary neck procedure but may require needle aspiration of a closed space for more rapid resolution (Fig. 17.3). Secondary procedures are much more commonly associated with seroma formation, likely due to injury to the lymphatics associated with the superficial fascia and continuous with the platysma. Consideration of drain use is reasonable in the reoperated neck. Either a Blake or Jackson-Pratt drain may be exited in the infralobular or postauricular region and should be only removed when fluid output is 5.0 cc or less in a 24 h period.

Fig. 17.3 64 year old male underwent cervicofacial rhytidectomy and developed submental hematoma as seen in one week photos. This was aspirated and lavaged with saline, followed by 6-8 weeks of manual massage



17.11 Epidermolysis

Epidermolysis is a condition of skin loss as a result of death of some element of the skin, epidermis, or dermis. This can result from hydrodissection, thermal injury, pressure from a head dressing, or excessive tension at the cervical crease most commonly. As with all wounds, verification that the patient has not intentionally caused the injury is important. Patients seeking complications for litigious reasons may inflict damage with curling irons, fingernails, or ice packs. The area is often of diminished sensation for a few days and accidental heat or ice in excess can result in this type of injury. Combined skin resurfacing procedures may also lead to devascularization injury and skin loss. Management is always best with a conservative and consistent approach to wound care. Daily cleansing with antibacterial soap and water is followed by maintaining ideal hydration with either occlusive dressing or nonocclusive hydrogel ointment. The goal is to maintain a clean, non-contaminated wound and an ideal environment for re-epithelialization. It is common to require progression from petroleum to hydrogel or silver ion impregnated hydrogel until re-epithelialization is complete.

17.12 Skin Contour Deformity

Skin has variable ability to contract as a result of insult from surgery or skin resurfacing (Fig. 17.4). If the overlying skin flap is thinner than 3.0 mm of subcutaneous fat on the dermal side, it may be more prone to adherence to the superficial fascia or the platysma muscle. Secondary neck procedures are more prone to seroma formation and, as a result, can develop severe fibrosis and adhesions. Management is primarily by watchful waiting and reassurance of resolution with time alone.

17.13 Sialocele

The superficial musculo-aponeurotic system (SMAS) is contiguous with the investing fascia of the platysma muscle. In performing a neck lift procedure, dissection frequently is both above and below the platysma muscle, in close proximity to the parotidomasseteric fascia or directly in the submandibular gland for gland resection. Sialocele formation is uncommon (Fig. 17.5) but manageable with needle aspiration, compression, and the use of neurotoxin to reduce production of saliva. The presence of amylase in the aspirated fluid confirms the diagnosis of a sialocele.

Fig. 17.4 34 year old female underwent platymaplasty and superficial lobe of submandibular gland resection. He post operative course was complicated with 6 weeks of aspiration of mixed serous and salivary fluid, compression and botox injections



Fig. 17.5 Intermittent fibrosis often results for 3-6 months after chronic seroma or sialocele. All have resolved with tincture of time, manual massage and application of external ultrasound

Conclusion

Neck rejuvenation procedures can dramatically improve the youthful appearance of the cervicomental angle. A thorough preoperative assessment, careful intraoperative technique, and appropriate postoperative care are vital to ensuring not only the success of the operation but more importantly patient safety and surgical outcome satisfaction. The most urgent complications include expanding hematomas, which demand immediate evacuation and close follow-up. Induration may result and may require revisional surgery if skin contour irregularities persist. The great auricular nerve is the most frequently injured sensory nerve, while the marginal mandibular branch of the facial nerve is the most commonly injured motor nerve. Infection as a result of surgery is uncommon, however, surgeons should be wary of the ever increasing prevalence of methicillin-resistant *Staphylococcus aureus* in complications of infectious etiology. Finally, any nonresolving platysmal bands following the primary surgery may be addressed with botox injections, or via corset platysmaplasty or submentoplasty. In general, neck rejuvenation is a relatively safe procedure with few complications and gratifying results.

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Abstract

Rhinoplasty is a challenging aesthetic surgical procedure due to the complex balance between function and aesthetics. A thorough knowledge of nasal anatomy is important in achieving predictable outcomes. This chapter will review potential complications associated with a rhinoplasty procedure. Management and prevention of these complications will also be discussed.

18.1 Introduction

Rhinoplasty is a common procedure that is typically performed by surgeons specifically trained in facial plastic surgery. It is considered a technically difficult procedure because access to the important structures is usually limited and requires 3-D manipulation of structures [1, 2].

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The surgeon must also consider both function and aesthetics while performing the operation. Complications of rhinoplasty can occur intraoperative, short term, and long term [1]. Short-term complications are those that occur 1 week post-operatively, while long-term complications are those that occur greater than 1 week from surgery [1]. This chapter will discuss hemorrhagic, infectious, intraoperative traumatic, functional, and aesthetic complications. Aesthetic complications can be broken down by location of the deformity for simplicity. The three areas of focus for this chapter are the bony pyramid, middle third, and tip and ala [1–3].

18.2 Hemorrhagic Complications

18.2.1 Excessive Bleeding

Inappropriate bleeding is one of the most common complications of nasal surgeries, which can

lead to poor visibility and surgeon's frustration. Additionally, excessive bleeding can cause significantly more edema, ecchymosis, and increased anesthesia time [1].

Although it is not possible to prevent all unexpected bleeding, certain preventive measures should be taken to reduce its likelihood. All patients should be evaluated for possible underlying bleeding disorders [3]. A careful and detailed history and physical examination should be performed on all surgical candidates with special attention to excessive bleeding after previous surgery or trauma and family history of bleeding disorders. All surgical patients should be questioned about taking anticoagulants or any drug that might interfere with hemostasis. Aspirin is one of the most commonly taken medication that significantly inhibits platelet aggregation. Patients should also be specifically asked about herbal medicine such as ginkgo and ginseng, which have shown to increase coagulation time. Aspirin, as well as other anticoagulants and all herbal medications, should be discontinued 14 days prior to procedure after appropriate consultation with patient's primary care physician [4]. In addition, appropriate preoperative injection using local anesthetic mixed with epinephrine into the subcutaneous tissue and topical Afrin (oxymetazoline) on the mucosa, with adequate time given for the onset of these drugs can substantially reduce intraoperative bleeding [5].

Hypertension is another common reason for intraoperative bleeding and can be caused by inadequate depth of anesthesia after initial painful stimulus and at the time of emergence [2]. Patients undergoing rhinoplasty should generally be normotensive pre- and postoperatively in order to minimize bleeding. It is important to remember that local anesthetic with epinephrine, and topical Afrin(oxymetazoline), can raise the blood pressure.

Early postoperative epistaxis can be managed with topical oxymetazoline, or with ice, digital pressure, and head elevation. In case of persistent bleeding despite conservative interventions, the bleeding source should be identified by removing all clots and direct

examination of surgical sites. Epinephrine soaked cotton rolls can minimize the bleeding to help with a thorough and direct examination. Anterior or posterior packing, depending on the location of bleeding, is the next step of intervention. Packing should be coated with antibacterial ointment and should be kept in place for 24–48 h, and sometimes longer. The patient should be instructed to keep the packing moist with saline drops broad-spectrum oral antibiotics should be prescribed, while patient has packing in place to prevent the development of toxic shock syndrome. Nasal packing should be removed slowly to prevent rebleeding.

In the rare situation of uncontrollable bleeding when the maxillary artery or posterior ethmoid artery are thought to be the source, interventional radiologist consultation should be obtained for embolization. A return trip to the operating should also be considered in the case of persistent profuse bleeding which is refractory to all mentioned interventions [2].

18.2.2 Septal Hematoma

A septal hematoma is a potentially serious complication of rhinoplasty, especially when a concomitant septoplasty is performed. The septal hematoma is the result of blood accumulation within any dead space between elevated mucoperichondrial flaps. Several clinical pearls such as transeptal whip sutures, inferiorly based drainage incisions, and use of soft Silastic removable intranasal splints will help minimize hematoma formation [2, 5–7].

Patients may present with symptoms such as nasal obstruction, pain, rhinorrhea, and fever. Diagnosis is confirmed by an ecchymotic nasal septal mass evident on physical examination. An untreated septal hematoma can have serious implications, such as infection and septal necrosis leading to a saddle nose deformity. Proper management involves early recognition with prompt evacuation of the hematoma, either via needle aspiration or incision and drainage [3, 4].

18.3 Infectious Complications

Postoperative infection following rhinoplasty is generally much less common than might be expected. However, it requires an emergent evaluation due to possible life-threatening illnesses such as toxic shock syndrome and cavernous sinus thrombosis.

Local wound infections, such as cellulitis, can be treated with oral antibiotics and intranasal bacitracin with close observation. Untreated septal hematoma can give rise to septal abscess that requires prompt surgical drainage in addition to antibiotic therapy [4, 5].

Toxic shock syndrome has been described after rhinoplasty with the use of both nasal packing and intranasal splints. Toxic shock syndrome is usually caused by the release of an exotoxin, toxic shock syndrome toxin-1, created by *Staphylococcus aureus*. The patient presents with symptoms such as nausea or vomiting, rash, fever, tachycardia, and hypotension. The patient must be admitted to the intensive care unit for supportive care and intravenous antibiotics, and if any packing has been placed, it must immediately be removed [8, 9].

18.4 Intraoperative Traumatic Complications

18.4.1 L-Strut Fracture

Intraoperative fracture of the dorsal portion of the septum “L strut” can be caused by over resection of the septum, excessive septal manipulation, or misguided medial osteotomies. Preserving a 1-cm-wide septal L-strut that remains attached to the perpendicular plate of the ethmoid and the nasal spine–maxillary crest area is recommended by most authors. Failure to repair L-strut fractures can lead to significant dorsal support and saddle nose deformity, since the cartilaginous septal segment tends to rock posteriorly.

L-strut fractures can be treated through several methods: (1) suture fixation of the septal parts, using the mucoperiosteum as support, (2) direct suture fixation of spreader grafts to the

dorsal strut, (3) direct suture fixation to the dorsal osseocartilaginous junction, and (4) percutaneous Kirschner wire (K-wire) fixation of the nasal bones to the L-strut, or any combination of sutures, grafts, and K-wires needed to maintain central structural stability. The Kirschner wires are left in place for 3–4 weeks, when they are removed in the office setting with a wire twister. Location of the fracture and surgeon’s experience can dictate the specific method selected to repair the L-strut fracture [3, 5, 10].

18.4.2 Cerebrospinal Fluid Leak

There are a few case reports in the literature describing extremely rare incidents of cerebrospinal fluid leak after rhinoplasty. Cribriform plate fracture by surgical instruments can result in a cerebrospinal fluid leak, anosmia, and potential intracranial injury. Signs and symptoms of a cerebrospinal fluid leak include clear rhinorrhea and positional headache. The diagnosis should be confirmed by testing the fluid for the presence of beta-2-transferrin, a protein highly specific for cerebrospinal fluid. These patients should be hospitalized with strict bed rest. Most cases will resolve without any surgical interventions; however, chronic leakage might warrant surgical repair via an endoscopic technique. Patient with signs of meningitis should be evaluated by the neurosurgical team for potential placement of a lumbar drain [11].

18.5 Functional Complications

18.5.1 Septal Perforation

The vast majority of nasal septal perforations are result of iatrogenic trauma during septoplasty. Perforation can be caused by opposing tears in the elevated septal mucoperichondrial flaps with no intervening septal cartilage. Other sources of septal perforation include septal abscess and septal necrosis. Small septal perforations that are recognized during surgery are managed with prolonged splinting. Fortunately,

only small percentages of septal perforation are symptomatic. The incidence of troublesome symptoms depends on the size and location of the perforation. Large and anterior septal perforations cause more problems. Symptoms of a septal perforation include crusting, epistaxis, whistling, and nasal airway obstruction. Patients with large perforations lack support of the septum, leading to saddle nose deformity and nasal valve distortion. Severity of symptoms should determine the need for treatment. Conservative treatment with moisturizing ointment and nasal saline irrigation can help with crusting and epistaxis. Office-based prosthetic closure of the septal perforation using prefabricated obturators can also improve some of the symptoms and should be considered in patients who are poor operative candidates. Surgical treatment should be reserved only for large defects and patient who have failed conservative treatments. Mucoperichondrial-periosteal flaps from the septum, floor of the nose, and lateral nasal wall with interposed autogenous free grafts are the foundation of surgical treatment [3, 4].

18.5.2 Internal Nasal Valve Collapse

The internal valve is located in the area of transition between the skin and respiratory epithelium and is formed by the caudal margin of the upper lateral cartilage, nasal septum, floor of the nose, and occasionally the large inferior turbinate. The internal nasal valve is the main site of airway resistance during inspiration and any collapse of this valve can significantly impair nasal breathing. Internal nasal valve collapse is particularly common after excessive removal of the nasal roof including the upper lateral cartilages or excessive resection of the cephalic portion of the lower lateral cartilages. In addition, a poorly executed lateral nasal osteotomy can move the nasal bones too far medially and cause this compromise. This mistake can be prevented by using caution to preserve Webster's triangle (the inferior/posterior portion of the pyriform rim).

Correcting internal nasal valve collapse is usually directed at repositioning the upper lateral cartilages or applying structural grafts to support the lateral nasal wall. Spreader grafts can lateralize the upper lateral cartilages while increasing the width of the middle nasal vault [12, 13].

18.6 Aesthetic Complications

18.6.1 Bony Pyramid

Asymmetries can develop in the bony pyramid for a number of reasons, including differences in osteotomies between sides, asymmetric dorsal reduction, and incomplete osteotomies leading to greenstick fractures, which relapse postoperatively. Following dorsal hump removal or reduction, an open roof deformity usually occurs (space between the lateral nasal bones) [1, 2, 14]. If this space is not reduced, the cross section of the nose will have a trapezoidal shape as opposed to the aesthetically pleasing triangular form [1, 2, 14]. Intraoperatively, this can be masked by edema of the soft tissue envelope and missed by the surgeon if proper palpation is not performed during the operation. This defect can be simply corrected by medial and lateral osteotomies and manual palpation to reduce the space. In certain cases, autogenous graft is necessary if excessive dorsum was removed and osteotomies alone cannot reduce the open space [1, 3]. If the lateral nasal osteotomy is carried too far superiorly onto the frontal bone, a condition known as "Rocker Deformity" can occur (Figs. 18.1 and 18.2) [1–3]. In this situation, when medial digital pressure is applied to the lateral nasal bones, the superior portion of the osteotomy will move laterally causing asymmetry. If the surgeon notices that the osteotomy was performed too far superiorly, a percutaneous approach with a small osteotome can back fracture the superior portion to avoid this complication.

Sweeping osteotomies are performed with an osteotome in a continuous fashion. There is a typical "high-low-high" pattern when performing the lateral nasal bone osteotomy [3, 14].



Fig. 18.1 Open roof deformity after prior rhinoplasty from failure to close with osteotomies



Fig. 18.2 Rocker deformity with osteotomies continuing into frontal bone. Note superior aspect “rocking” laterally when bony base is medialized (From Toriumi DM, Hecht DA. Skeletal modifications in rhinoplasty. *Facial Plast Surg Clin North Am* 2000;8(4):424; with permission)

The initial osteotomy is high along the pyriform aperture, then low along the ascending maxilla and then high along the nasal bones superiorly. If the placement of the lateral osteotomy is anterior to the ascending process of the maxilla, there will be a palpable step off that can also be visible [1, 14]. These deformities are called “stair-step deformities” and can be difficult to repair, and therefore lateral osteotomies must be carefully palpated by the surgeon to ensure proper placement throughout the procedure [1, 3].

18.6.2 Middle Third

Supratip fullness occurs when the lower third of the dorsum projects more than the tip, giving a convex shape to the nasal dorsum [2, 14]. This deformity is referred to as “pollybeak” and occurs by one of four mechanisms: inadequate resection/under resection of the cartilage in middle third and anterior septal angle, over resection of bony pyramid, over resection of supratip structures (dorsal septal cartilage) leading to dead space with subsequent scar/fibrous tissue formation, and postoperative loss of tip projection leading to tip ptosis giving supratip appearance of fullness and relative pollybeak (Fig. 18.3) [1, 2, 14]. The treatment of pollybeak depends on the cause. If the cause is inadequate resection of cartilage at the anterior septal angle or cartilaginous hump removal, then by simply reducing this area in a subsequent surgery will likely repair this deformity. If the deformity is due to over resection of the dorsal hump, then augmentation is required using a variety of grafts. If fibrous tissue and scarring is the problem, then careful injections with triamcinolone can be used along with skin taping in the early postoperative period [1–3, 14].

A “saddle nose” deformity can result when there is over reduction of the cartilaginous vault and the quadrangular cartilage without enough dorsal strut remaining. Typically, an “L”-shaped strut of 1 cm is necessary in the dorsal and caudal



Fig. 18.3 Over-resected nasal bones after prior rhinoplasty. Also note prominent pollybeak



Fig. 18.4 Inverted V deformity is noted with prominence of the bony base and narrowing of the middle third

portion in order to prevent a saddle nose. Saddling can also occur if there is a disarticulation of the keystone area [1, 14]. The keystone area is made up of bones and cartilage in the upper and middle thirds of the nose and consists of nasal bones, upper lateral cartilage, quadrangular cartilage, and perpendicular plate of Ethmoid. If excessive cartilage is removed and is noticed intraoperatively, then the excised cartilage can be placed as an onlay graft. However, if not noticed and revision is necessary, then autogenous materials can be used.

An “inverted V” deformity can occur after dorsal hump reduction if there is inadequate support for the upper lateral cartilage which typically articulates and relies on cephalic portion of nasal bones for support (Fig. 18.4). This will cause the upper lateral cartilages to collapse and thereby cause an accentuated appearance of the caudal margin of nasal bones [1–3]. This deformity will require another procedure in order to camouflage the deformity and typi-

cally requires approximation of the upper lateral cartilage to the nasal dorsum and spreader graft [3, 14].

18.6.3 Nasal Tip/Ala

In order to understand deformities of the nasal tip and ala, it is prudent to discuss the major and minor tip support systems. Major tip support mechanisms include strength of lower lateral cartilage, attachment of the medial crura to septum, and attachment of upper lateral cartilage to lower lateral cartilage [2, 3, 14]. Minor tip support mechanisms include the dorsal septum, interdomal ligament, membranous septum, nasal spine, adjacent skin and soft tissue, and sidewalls of ala.

Over reduction or overly aggressive cephalic resection of lateral crura can result in alar retraction or a pinched tip appearance (Fig. 18.5). This retraction may also lead to alar and columellar disharmony with excessive columellar

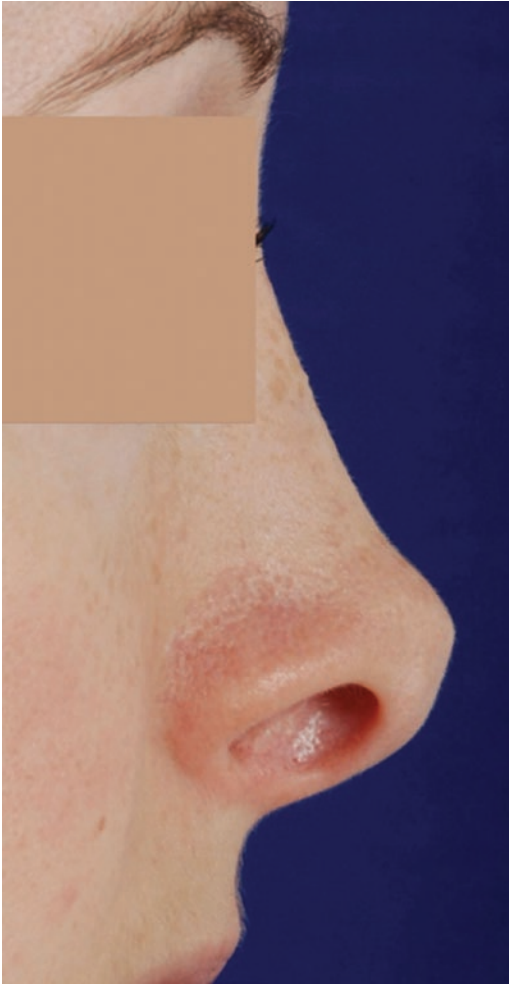


Fig. 18.5 Patient with both alar retraction and hanging columella after rhinoplasty

show [2, 3, 14]. On frontal view, this causes excessive nostril show, which can be aesthetically displeasing. Ideally on lateral view, there should be approximately 2–4 mm of columellar show, any increase in this number suggests alar retraction or that the columella is hanging [2, 3, 14]. In order to prevent over resection of the lower lateral cartilage, some surgeons leave a 7–8 mm rim of cartilage during cephalic trimming. Correction of this deformity requires an additional procedure that involves strut grafts

and lateral crura repositioning. Columellar retraction can also occur from over resection of caudal septum, medial crura and excessive setback of medial crura during tongue-in-groove technique. Tongue-in-groove technique consists of placing the medial crura cephaloposteriorly onto the caudal septum into a surgically created space. This technique can also be used to treat a hanging columella [1–3, 14]. A hanging columella typically results from placement of large strut graft, septal extension graft, or tip graft [1–3, 14–16].

A persistent wide and bulbous tip will occur if there has been a decrease in nasal tip definition, specifically wide interdomal distance, inadequate lower lateral cartilage resection, and thick and inelastic skin [1–3, 14]. In order to correct this deformity, an additional procedure is indicated and can include further cartilage resection and multiple suturing techniques such as transdomal suture, medial crura suture, and interdomal suture. On the contrary to a bulbous tip, a pinched nasal tip is typically caused by collapsed or weak lower lateral crura. This can be corrected using spreader grafts of septal or auricular cartilage that is inserted deep and between the remaining lower lateral cartilage to increase the width and projection [1–3, 14], and/or alar wing grafts to replace over-resected lower lateral cartilages.

Conclusion

Rhinoplasty continues to be a challenge due to the complex balance required between function and aesthetics. A thorough understanding of nasal anatomy, along with consideration of how each maneuver can affect both function as well as aesthetics, is imperative when performing rhinoplasty. Patient selection is most important, and managing patient expectations is critical. Any proposed procedure should be thoroughly discussed with the patient prior to surgery, so that realistic goals are developed, and all possible complications are presented.

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Abstract

Genioplasty is a safe and predictable procedure. It is an aesthetic surgical procedure that creates a balanced facial profile and alters the lower one third of the facial skeleton. It has minimal complications. This chapter will review important aspects of the preoperative evaluation, the surgical procedure, as well as potential complications. Management and prevention of these complications will also be addressed.

19.1 Introduction

Chin augmentation through genioplasty is an excellent and relatively safe procedure used to achieve symmetry and proportionality of the lower third of the face in regard to the upper and middle thirds [1, 2]. Not only can osseous genioplasty be aesthetically pleasing, it also increases

the upper airway space by advancing the genial tubercles, thus advancing the genioglossus and geniohyoid muscle anteriorly. Genioplasty was first described by Aufrecht in 1934 in combination with rhinoplasty in which graft material was harvested. Hofer, in 1942, utilized an extraoral approach, but it wasn't until 1957 that Trauner and Obwegesser described an intraoral approach to the anterior mandible [1, 2]. Through an intraoral incision, osseous genioplasty can be performed as well as implant placement with porous polyethylene, methyl methacrylate, silicone, and Teflon (Fig. 19.1) [3–5].

A systematic physical exam and medical history must be obtained prior to treatment planning, and an in-depth surgical workup must be performed in order to prevent complications. Thorough evaluation of the patients' occlusion should also be conducted to rule out mandibular hypoplasia/hyperplasia that could be the cause of retrogenia or prognathism, respectively, (which could require mandibular/maxillary

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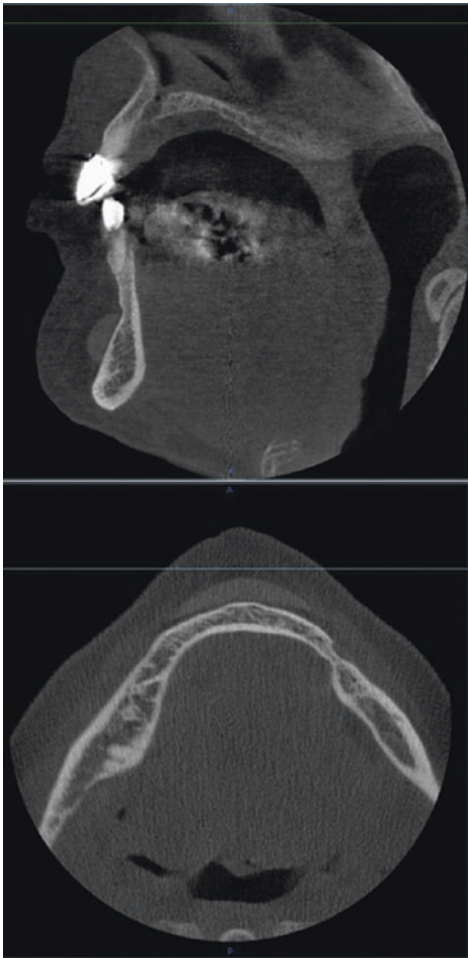


Fig. 19.1 Cone beam imaging of a porous polyethylene implant (courtesy of Dr. Aditya Tadinada, University of Connecticut)

osteotomies as opposed to solely a chin augmentation) [1, 6, 7]. In order to assess antero-posterior projection of the maxilla and mandible, it is prudent for the surgeons to obtain a lateral cephalogram and evaluate SNA and SNB points in relation to each other [6, 7]. There are several methods to evaluate chin projection, two of which include Riedel's plane (a straight line in the lateral view that connects the most prominent parts of the upper and lower lip that should ideally touch the soft tissue pogonion) and Ricketts E-line (a line formed by joining the tip of the nose to soft tissue pogonion) [1, 6, 7]. As

with any surgical procedure, there are surgical complications associated with genioplasty that must be taken into account when treatment planning. These include hematoma, infection, neurosensory disturbance, chin ptosis, relapse, damage to the anterior mandibular teeth, bony resorption, over/under augmentation, and overall patient dissatisfaction [1, 2]. These potential complications, however rare, must be discussed with the patient and accepted prior to surgery. This chapter will discuss in detail the relevant complications of both osseous and implant genioplasty.

19.2 Hematoma

During genioplasty, hematomas, though rare, can occur. Due to location (floor of mouth), these hematomas can be true life-threatening emergencies. Bleeding can occur from one of several places: soft tissue/mentalis muscle, sinusoids and cancellous marrow from osteotomy site in the mandible, and, although rare, blood vessels in the floor of the mouth, such as the lingual artery and deep and dorsal lingual veins [1, 8, 9]. In order to prevent bleeding from soft tissue, electrocautery should be utilized during dissection with coagulation of any bleeding areas as well as a clean subperiosteal dissection. Prior to closure of the incision, copious irrigation should be performed, and inspection for any areas of bleeding should be identified. Bleeding that occurs from the sinusoids and cancellous marrow of the mandible is unavoidable but can possibly be minimized by controlling the patient's blood pressure during osteotomy and upon emergence from anesthesia [1, 2]. Disruption of blood vessels in the floor of the mouth is an uncommon complication but can occur during osseous genioplasty if one significantly penetrates the floor of the mouth with the reciprocating saw during osteotomy. If venous bleeding occurs, one can attempt to ligate the vessel or hold pressure until bleeding ceases. However, if the noted bleeding is arterial in nature, the artery should be identified and ligated. If the vessel cannot be identified and bleeding does not stop

with pressure, one should seek interventional radiology for embolization. Floor of mouth hematomas are typically small and do not require any intervention. If intervention *is* needed, simple needle aspiration is usually sufficient. For quickly expanding floor of mouth hematomas, the patient's airway must be secured via endotracheal intubation as the tongue will elevate and obstruct the upper airway. Once the airway has been secured, the source of bleeding should be investigated, and serial hemoglobin and hematocrit should be drawn [1, 2, 8].

19.3 Infection

Infection is another uncommon but possible occurrence with both osseous genioplasty and implant chin augmentation without osteotomy. Reports have stated infection rates of 5–7% for chin implants with no specification as to the type of implant used [1]. Infections that occur from osseous genioplasty can be a result of bone debris that was not cleared prior to closure. Infections usually occur from gram-positive organisms, such as *Staphylococcus* and *Streptococcus* species. If an infection is of concern, the surgeon should perform a thorough physical exam, obtain a complete blood count with differential, as well as a CT scan or ultrasound to rule out an abscess formation in the

submental region or deep to the implant [3, 10]. If no collection is identified, antibiotics should be initiated to cover for usual skin/oral flora which could consist of a first-generation cephalosporin or clindamycin, both effective against gram-positive organisms, with clindamycin also covering anaerobic organisms. If a collection is identified, then the patient will require incision and drainage with broad-spectrum antibiotic coverage, typically intravenously. Until sensitivities are obtained, broad-spectrum antibiotic coverage can consist of a third- or fourth-generation cephalosporin along with metronidazole, piperacillin/tazobactam, or clindamycin and vancomycin. If a collection is present, the implant will likely also need removal (due to eventual hardware failure) and will require irrigation of the remaining pocket [3, 10, 11]. Fluid culture should be obtained and sent for sensitivity in order for antibiotics to be culture driven. The decision then needs to be made whether to attempt reimplantation once the infection has cleared or to proceed with an osseous genioplasty. During incision and drainage, the surgeon should assess the quality of the underlying bone to rule out bony compromise or osteomyelitis (extremely rare) [3, 8, 10, 11]. The patient should be monitored in a hospital setting for signs of worsening infection or edema after incision and drainage, as elevation of the floor of the mouth can lead to upper airway compro-



Fig. 19.2 Fistula formation after implant placement

mise and need for endotracheal intubation (Fig. 19.2).

19.4 Neurosensory Loss

The mental nerve is a terminal branch of the inferior alveolar nerve (IAN) in which it exits the mandible through the mental foramen to innervate the skin of the chin, mucosa of lower lip, and gingiva of mandibular teeth [12]. The mental foramen typically is found apical to the second mandibular premolar or between the apices of both premolars; however, its course can vary from canine to first molar. The IAN, after giving off the mental nerve, continues within the mandible as the mandibular incisive nerve, to innervate the mandibular canines and incisors [12–15]. The IAN usually courses 4–5 mm below the mental foramen, at an average of 4.5 mm with maximum distance of 8.4 mm, and passes 3–5 mm anteriorly before looping posteriorly to exit the mental foramen (anterior loop). During exposure of the mandibular symphysis for osseous genioplasty, an incision is made from canine to contralateral canine approximately 5–7 mm below the attached mucosa in order to leave a cuff of unattached mucosa for closure. Some surgeons may decide to place an initial incision 1.5 cm anterior to the depth of the vestibule [12–16]. This incision is then carried obliquely through the mentalis muscle down to the periosteum. A subperiosteal dissection is performed to expose the anterior mandible and then carried posteriorly to expose the mental foramen/nerve bilaterally. The osteotomy should be placed 5–8 mm below the apices of the canines, and due to the inferior extent of the IAN, approximately 4–6 mm below the mental foramen to avoid damage to the mental nerve [12–14].

Due to edema, stretching from retraction and, rarely, transection, paresthesia of the lower lip is common in osseous genioplasty. This occurs in

approximately 40–70% of patients; however, it is temporary, resolving within 12 months. Guyuron and Raszewski reported that 56% of their patients reported temporary neurosensory loss that resolved at 12 months [15–17]. Permanent sensory loss has been reported to occur in 0–12% of patients with an average of 3.5% over 12 months. Due to the osteotomy site, sensation will be lost to the anterior incisors as a result of likely transection of the mandibular incisive nerve which cannot be avoided and should be discussed with the patient [15–17]. Paresthesia can also occur without an osteotomy and placement of an implant due to retraction and potential fixation in close proximity of the mental nerve. Paresthesia due to compression or stretch should resolve on its own; however, if symptoms have not resolved in 2–3 weeks, the surgeon must consider investigating implant placement and potential removal versus adjustment [12–16].

19.5 Chin Ptosis

The mentalis muscle fibers arise from the incisive fossa of the mandible and course vertically and inferiorly to insert on the soft tissue of the chin. The mentalis muscle is innervated by the marginal mandibular branch of cranial nerve seven and functions to raise the lower lip, push the lower lip outward, and pout the lower lip [7, 18, 19]. During dissection for both osseous genioplasty and implant placement, the mentalis muscle is incised and reflected in order to approach the anterior mandible [7, 18–20]. It is prudent during closure to re-approximate the mentalis muscle in order to avoid shortening of the muscle which can lead to chin ptosis, lip ptosis, drooling, and skin dimpling [9, 21, 22]. This complication is aesthetically exceptionally displeasing and difficult to correct. There are several techniques documented for the correction of chin ptosis that include botulinum toxin to the unaffected side, chin pad-lifting

Fig. 19.3 This 55-year-old woman presented after osseous genioplasty, having developed facial spasms months after the procedure. These spasms improved with botulinum toxin injections. The patient is shown on frontal (a) and lateral (b) views

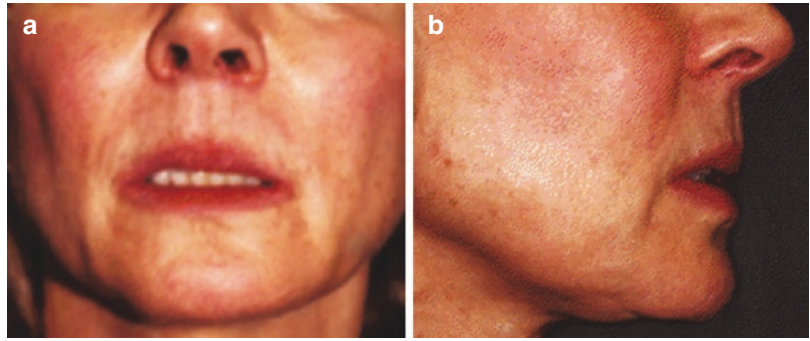


Fig. 19.4 After removal of a large implant with failure to reattach the mentalis muscle, this patient's chin exhibits ptosis with skin dimpling and bunching



Fig. 19.5 Figure demonstrating failure of hardware for sliding genioplasty

techniques, lip mobilization and repositioning with screws, and use of submental soft tissue, all with questionable outcomes (Figs. 19.3 and 19.4) [1, 2, 8].

19.6 Bony Complications

The main bony complications that can occur after both osseous genioplasty and implant placement include fracture, relapse, and resorption of the underlying bone. Fractures to the distal or proximal segment following genioplasty are rare and

likely secondary to early loading or activity leading to trauma [1, 2]. Fractures can occur due to hardware failure, osteotomy design, or stress from overlying musculature (Fig. 19.5). If a fracture does occur, standard technique for mandibular repair can be employed such as closed reduction with arch bar placement or open reduction with internal fixation using plates and screws [1, 2]. Resorption is more common in implant placement than genioplasty and likely due to the pressure applied from the implant on the underlying bone, loss of blood supply after stripping the

periosteum, or micromotion of the implant. A study by Robinson and Shuken showed resorption at a rate of .1 mm per month when using plastic chin implants. Other studies have showed that resorption occurs in more than 50% of patients; however, the soft tissue changes do not lead to noticeable aesthetic changes. Moenning and Wolford performed a comparative study between Proplast implants versus porous block hydroxyapatite (PBHA) over 19 months, which demonstrated 0–3.3 mm and no bony resorption, respectively [4, 5, 23, 24]. It is theorized that the porous nature of the PBHA allows tissue ingrowth as opposed to surrounding fibrous tissue capsule formation, and this decreases underlying micromotion, thereby decreasing bony resorption. Other complications which are far less common but can still occur include tooth damage (from osteotomy or hardware placement), implant malposition, and under/over augmentation of the genial region. These latter complications can be attributed to technical error [4, 5, 23, 24].

Conclusion

Finally, genioplasty is a safe and effective way to create a balanced facial profile and alter the lower one third of the facial skeleton. Although rare, complications such as hematoma, infection, neurosensory disturbance, chin ptosis, relapse, damage to anterior mandibular teeth, bony resorption, over/under augmentation, and overall patient dissatisfaction can be avoided with proper surgical planning, technique, patient selection, and management of patient expectations.

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Abstract

Alloplastic facial implants are indicated for congenital and developmental asymmetry, iatrogenic defects after orthognathic surgery, long-face high-angle orthognathic deformities, and persons seeking beautification of particular facial area. Usually porous polyethylene or hydroxyapatite granulate implants are artistically shaped for each individual's aesthetics. The use of three-dimensional design software and titanium three-dimensional prints to more accurately conform to anatomical and esthetical requirements is described, with emphasis on avoidance of complications. Dehiscence and dissatisfaction with the achieved volume are the main complications.

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20.1 Introduction

Facial implants are widely used for surgical correction of cosmetic, traumatic, and congenital asymmetries. The obvious goal of treatment is an aesthetic result accepted by the patient. Common sites where facial implants are placed include the nasal dorsum, chin, and malar eminence. Even though autologous tissues are the gold standard, they carry the risk of donor-site morbidity, resorption, and restricted ability to mold to the desired shape. Thus, alloplastic materials are frequently implanted for facial augmentation. The alloplastic material must be biocompatible and chemically inert so that it will not cause a significant foreign body reaction and be stable enough to withstand the physiologic load of masticatory and facial mimetic forces. Additionally, the

alloplastic material should be able to be easily shaped so that ideal contour can be achieved [1]. Surgical factors that affect the outcome include appropriate placement by the surgeon and a favorable recipient site with healthy tissues. A deficiency in any of these conditions can lead to complications. Thus, an understanding of these qualities is needed to prevent and treat complications when they occur.

20.2 Complications

Postoperative complications associated with implants are generally minor and temporary. With any surgical procedure, prevention is the best cure. Every step of the surgical process is critical, including patient selection, treatment planning, and surgical protocol.

Early complications can usually be managed with anti-inflammatory agents to reduce swelling and analgesics for pain control. Some patients may experience numbness, which should subside over the first postoperative week. Most patients can be seen at a 1–2 week postoperative visit to evaluate for these early postoperative complications.

20.3 Chronic Pain

Chronic pain can be a complication of implant placement. Patients may experience postoperative pain; however, it should start to subside in 2–3 days. Chronic pain, which is pain that persists for many weeks postoperatively, may be an indication for removal of the prosthesis. The workup should include clinical examination and radiographs, although the radiographs may show nondiagnostic findings. Although rare, causative factors identified on clinical exam may be implant loosening and poor fit leading to the impingement of soft tissues. Prevention of chronic pain includes proper screw tightening and retraction of soft tissues during implant placement. Unsuccessful osseointegration of the screws may cause persistent dull pain. Disproportionate stress on the implant may also cause chronic pain. Analysis of the fit and function of the prosthesis are crucial [2–4].

20.4 Altered Sensation/Nerve Injury

Typical with mandibular implants, compression or trauma to sensory nerves during implant placement may cause regionally altered sensation or complete loss of sensation. Anesthesia post-implant placement probably indicates the implant is resting on the nerve. Intraoperative causes include nerve trauma from traction or thermal injury during instrumentation, local anesthesia injection, implant impingement on the nerve, improper implant size selection, implant migration, or direct nerve transection [5, 6].

The range of altered nerve sensation may be quite varied, ranging from slight hypesthesia to complete anesthesia. Often, the sensory disturbance most bothersome to patients is dysesthesia or hyperesthesia, altered touch, and temperature sensation. In the postoperative care for a patient with altered sensation, immediate clinical and radiographic examinations are indicated. If there is obvious contact with the nerve on radiographic imaging, the implant should be repositioned or replaced. Mapping of the region of altered sensation will allow for comparison with future examinations. Many of the injuries to nerves during implant placement are generally transient, and patients often recover well. However, if on subsequent examinations, the patient does not experience improvement in symptoms or if the symptoms become intolerable to the patient at anytime during the postoperative course, referral to a specialist is indicated [7].

Motor nerve impingement is a potential complication, particularly for malar implants. The malar implants may experience neuropraxia with a slightly higher ratio of motor nerve injuries than sensory. Instrumentation around the facial nerve branches results in weakness of the muscles of facial expression innervated by the temporo-frontal branch of the facial nerve, including zygomaticus, orbicularis oculi, and the frontalis muscles. Altered lip movements may be mimicked by improperly placed or sized malar implants affecting the muscles of facial expression. Also, in placement of chin implants, the marginal mandibular branch of facial nerve is

potentially at risk. Subperiosteal dissection and implant placement are the best method to avoid motor disturbances [8].

Prevention of nerve impingement is best undertaken with preoperative CT scans and possible 3D imaging to evaluate the position and location of the nerve and canals. Being mindful of drill length and screw placement during surgical procedure can help mitigate the most common causes of postoperative altered nerve function.

20.5 Persistent Edema

Edema is an expected postoperative sequela from implant placement; however, persistent or significant edema can cause postoperative complications. The anticipated postoperative course is for edema to decrease over 48 h and to be nearly resolved by 7–10 days. However, edema can persist for 6 months up to 1 year [9]. If edema persists beyond this point, it can lead to wound dehiscence and implant exposure. In extreme cases, this may require implant replacement or repositioning. Most edema is likely related to improper implant fixation with excessive continuing movement causing soft tissue inflammation and poor contour.

Additional causes of persistent edema maybe related to a foreign body reaction to the implant material, use of cement, or infection. These types of inflammatory edematous changes may be prevented with meticulous surgical procedure, copious irrigation, and atraumatic technique.

20.6 Hematoma/Seroma

Although generally a procedure with minimal blood loss, the risk for hematoma or profuse bleeding should be discussed. During the procedure, damage to muscles and soft tissue or perforation of the cortical bone with damage to an underlying blood vessel may cause significant bleeding. Also, an abnormal fluid collection, a seroma, can be the result of inadequate hemostasis, improper dissection, or increased dead space around the implant. Fluid collection can result in

excessive fibrosis or pressure on the surgical site producing soft tissue defects or necrosis [8].

Small hematomas may be monitored and will likely resolve in 1–2 weeks. Danger could ensue if the swelling is large and near the airway in the submental or submandibular spaces. If the source of the bleeding is visible, direct control is advisable with pressure and local measures. Ensuring a secure airway is of first priority, followed by draining or aspirating the fluid collection and close monitoring. In some cases, artery ligation or angiography and embolization of the offending vessel may be indicated.

Prevention of bleeding complications includes obtaining a CT scan with 3D reformatting for identification of regional anatomy as well as careful instrumentation with the surgical drills during the procedure. Intraoperative blood pressure control will help decrease excessive bleeding. Maintaining a subperiosteal dissection plane, minimizing the dead space around the implant, and placement of a drain may help alleviate postoperative hematoma or seroma formation.

20.7 Infection and Inflammation

Peri-implant inflammation, known as peri-implantitis, is a general term referring to inflammation of the soft tissue and bone. While more common in intraoral endosseous dental implants, peri-implant infection, and inflammation may occur in facial implants as well. Infection with the alloplastic material may be due to the synthetic material's ability to promote and harbor biofilm formation in the setting of a hypovascular implant bed. Although it would seem that porous implant materials would have a higher infection rate than smooth implants, these claims are speculative, and there is little objective evidence for proof of these theories. Pore sizes less than 1 μm are too small for bacteria to penetrate; however, a pore size of over 50 μm is required for macrophages to infiltrate the material and deliver immune mediators to prevent and resolve infections. Furthermore, pore sizes over 100 μm enable soft tissue ingrowth and fibrosis [8].

The various implant materials have well studied inherent risks. The biocompatibility of the material is the basis for its suitability for implantation. If the implant material is not accepted by the host patient, a foreign body reaction may ensue. While a mild foreign body reaction typically results in fibrous encapsulation that aids in fixation of the implant, complete healing is determined by the implant material, surface characteristics, ability to withstand mechanical forces, and biostability [8].

In general, porous implants (like polytetrafluoroethylene or high molecular weight polyethylene) and meshes allow for fibrous tissue ingrowth aiding in fixation of the implant at the surgical site. Smooth implants (like silicone and polymethyl methacrylate) are nonporous and become encapsulated by a fibrous connective tissue capsule [8].

The rate of infection with silicone implants is around 3.9% [10]. If the implant pocket is too large, the mobile implant with dead space can cause a seroma or peri-implantitis, both of which can cause the position of the implants to shift or possibly extrude [11].

Porous polyethylene material (MedPor) in theory may be prone to higher rates of infection due to the large particle size. The infection rate seen with MedPor use varies from 0.9 to 12.5% [12]. As discussed, due to the large pore size, potential for large soft tissue ingrowth exists, making a tough fibrous capsule over the implant. This thick capsule may limit the blood flow to the implant and thus decrease ability to fight off infection at the local level.

GoreTex is a polytetrafluoroethylene material with medium pore size. It too has a risk of infection, around 2.2% [10]. GoreTex was shown to be less biocompatible than other implant materials, possibly leading to increased risk of infection [13]. Woven polyester fiber materials, such as Mersilene mesh, have large pore sizes that allow for abundant ingrowth of soft tissue. These materials generally have good outcomes with low infection and resorption rates around 2.3% [11].

There are no formal controlled trials on the use of antibiotics for prevention of infection dur-

ing facial implant placement. No consensus exists on which to develop evidence-based guidelines regarding antibiotic use [14]. While intraoperative antibiotics are routinely given in the head and neck surgical procedures, prevention is best undertaken by dutiful attention to intraoperative sterile technique and implant sterilization procedures. Implants can be submerged in an antibiotic solution prior to placement, in addition to irrigating the implant pocket with antibiotic solution [15]. Some surgeons advocate a short (5–7 days) prophylactic antibiotic course following facial implant surgery. Others also suggest a short course of intraoperative and postoperative intravenous and oral corticosteroids to minimize the body's inflammatory response to the implant. Intraoral versus extraoral surgical access for implant placement does not appear to have a significant effect on infection rates, despite intraoral route exposing the implant to contamination with saliva. Nonetheless, wounds should be well closed with a watertight seal [14].

If the surgeon wishes to save the implant despite existing infection, the implant should be removed and sterilized to decrease bacterial load. Also, the surgical site should be debrided and irrigated prior to implant replacement, and postoperative antibiotics are recommended. If a purulent infection persists after a trial course of antibiotics, drainage of the site and removal of the implant are necessary [8].

20.8 Malposition and Cosmetic Failure

The overall displacement rate is approximately 2.3% for various types of alloplastic implants. Implant shape and implant fixation are integral to preventing migration. Migration is usually the result of over-dissection, selection of the wrong-sized implant, or lack of adequate fixation. If a patient is able to palpate the implant, it is likely due to improper size or contour, poor positioning, inadequate fixation, or contracture of the peri-implant soft tissue [10]. Preoperative or intraoperative shaping of the implant may help

prevent complications such as migration or patient palpation. Techniques described involve cutting grooves and tapering implant edges to help gain immobilization and guide in proper positioning [8, 14].

A major cause of patient dissatisfaction, asymmetry occurs for many reasons, but it is usually caused by surgical malposition or by dissecting too large of a surgical pocket. Asymmetry is more likely to be noticed in malar implants because of the obvious comparison with the contralateral side [16].

Prevention of postoperative cosmetic failure starts with appropriate treatment planning. Preoperative examination is crucial in each individual patient to determine the ideal position of the soft and hard tissue as well as expected surgical outcome. Preoperative treatment planning should address whether the surgical plan will involve repositioning of the soft tissue along with implant augmentation. It is important to point out preexisting asymmetry to the patient before treatment selection. Patients should be well informed of realistic surgical outcomes to help mitigate postoperative dissatisfaction with improper size, shape, contour, or asymmetry. Also, the patient's occlusion and facial proportions must be thoroughly evaluated. It is important for both the surgeon and the patient to realize that when treating inadequacies in skeletal jaw relationships, the discrepancies in facial proportions are merely disguised with facial implants and not comprehensively corrected [14]. Surgical approach may play a role in implant positioning, as subperiosteal implants have been shown to move less than suprapariosteally placed implants [17]. Minor asymmetries may correct themselves over a 6-month postoperative period as edema resolves and the tissue around the implant relaxes and softens [8].

Appropriate implant selection is also important because selecting too large an implant in too small of a surgical place will place undue tension on the wound. The result is decreased tissue perfusion with risk of wound dehiscence, implant exposure, and extrusion. Factors such as prior surgery and history of radiation will decrease the

local vascular supply and result in fibrosis. Prevention is best achieved by tension-free closure, subperiosteal placement, and adequate layers of soft tissue coverage over implant [8]. The surgeon should make proper assessment of tissue quality, with emphasis on perfusion and soft tissue coverage to allow for proper fixation and healing around the implant [17].

20.9 Bone Resorption

Peri-implant bone loss under alloplastic implants has occurred. Early explanations included a local foreign body giant cell reaction between the implant and the bone or increased local pressure from over-sized implants onto underlying bone. It is an expected postoperative sequel that malar and chin implants cause some bony resorption. The incidence of bone resorption approaches 60% and can appear radiographically in as soon as 2 months. Note that this is an anticipated outcome, and it rarely causes clinical significance [8, 18].

20.10 Patient Assessment

Improper placement of the implant is the most common complication followed by improper patient selection. Appropriate patient selection is a very important part of the preoperative workup and evaluation before reconstruction with facial implants. The surgeon must identify and select for ideal candidates for allogenic implantation. Medical comorbidities such as diabetes, immunosuppression, a history of radiation therapy, or osteonecrosis would put the patient at a high risk for postoperative complications [14]. Additional factors, such as psychological problems, drug abuse, or smoking should be taken into account. To mount any response to the implanted material, the host must be healthy, well nourished, and possess a functional immune system. Thus, chronic disease, malnourishment, steroid use, or poor tissue perfusion may lead to postoperative complications [8].

20.11 Site Specific Complications

20.11.1 Malar

Malar or submalar implants are very popular for enhancing patients that may have midface atrophy or an aged appearance. However, as cheeks are bilateral in nature, harmonious correction is difficult, and inadequate correction or overcorrection causes the most common problem of minor asymmetry. In addition to the previously mentioned general complications of malposition, implant migration, infection, extrusion, and neuropraxia, depending on the surgical approach used to place the malar implant, the patient may be at risk for facial nerve injury [8, 15]. For malar implants, the average infection rate is 2.4%, and displacement rate is 2.3% [10]. Malar and submalar implants can be placed through intraoral, subciliary, transconjunctival approach, or concurrent with a facelift through a rhytidectomy approach. The intraoral approach exposes the implant to oral contaminants during placement and, however, offers the cosmetic result of avoiding an extraoral scar. Possible drawbacks of the intraoral incisions include potential incisional weakness in the muscle floor, which could predispose the implant to malpositioning unless fixation is used [19]. While the access via an extraoral approach avoids salivary contamination, this approach is complicated by the risk of external scarring and injury to the facial and infraorbital nerves [8].

20.11.2 Nasal

Nasal implants may be used as primary means of augmenting an underprojected or misshapen nose or may be used in combination with revision rhinoplasty. The most important determinant of the long-term success of a nasal implant is the thickness, vascularity, and amount of the overlying skin coverage [8]. This becomes crucial particularly in the lower aspect of the nose, where the soft tissue coverage is very thin and is susceptible to excess pressure placed by the implant [20]. These complications may be due to implant size

or placement position. The most common reason for implant failure and subsequent removal was displacement or overprominence [21]. While malpositioning was found to be a common problem with silicone implants, possibly as a result of capsular contracture, implant malpositioning may be influenced by surgical approach when implants are placed in the soft tissue compared to a subperiosteal or subperichondrial plane [8]. Other complications of alloplastic nasal implants include extrusion and, due to increased projection from the rest of the face, increased risk of successive nasal trauma [14]. When used in combination with a revision rhinoplasty, the complication rate of nasal implants was 4.6% compared to the slightly lower rate of only 1.9% of primary rhinoplasty [22].

20.11.3 Mandibular

Chin implants are used to treat recessive and deficient hard and soft tissues causing diminished lower face height, including recessive chin, deficient lower lip projection, and prominent prejowl sulcus [8]. Mandibular implant complications are fairly similar to those of midface implants and include inadequate correction or overcorrection, asymmetry, malpositioning, infection, and wound dehiscence. Additional potential complications include soft tissue ptosis or distortion of the overlying soft tissue if the soft tissues are not adequately reapproximated [8, 14]. The strong action of the mentalis muscle overlying a chin implant can place continuous pressure on the prosthesis and theoretically cause increased bony resorption compared to other implant sites. However, while this may happen, the amount of resorption does not appear to be clinically significant [14]. Neuropraxia is also of concern when placing chin implants. In fact, the most common complications were altered sensation due to manipulation of the mental nerves, seen in up to 20% of the patients. A sensory nerve deficit was more commonly found than a motor nerve impairment [11]. Chin implants can be placed through an intraoral or extraoral approach. The use of the intraoral approach is analogous to midface implants. While

avoiding a facial scar, it unfortunately exposes the implant to the intraoral contaminants. Also, during intraoral placement of a chin implant, the mentalis muscle attachment is stripped from the bone of the chin [8]. Failure to resuspend the mentalis properly can lead to chin ptosis, also known as a “witches chin deformity” [8, 23]. Care must be taken with an intraoral approach to not create too large of a dissection for insertion of the prosthesis, as this could increase the chance for malposition. Another unique feature of chin implants is that complications were found to occur very late in the postoperative period, up to almost 50 years after implant placement [10].

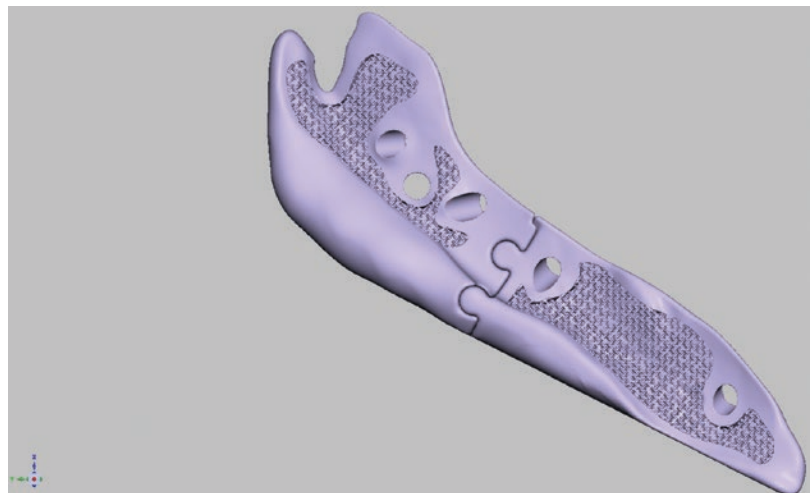
20.11.4 Technique

Mandibular angle implants, even customized titanium implants, are used for jaw angle contouring in cases of congenital and developmental asymmetry, iatrogenic defects after orthognathic surgery, long-face high-angle orthognathic deformities, and persons seeking beautification of particular facial area. Computerized tomography and cone beam computerized tomography data are obtained with specific scanning protocols and provide resolution and slice thinness that are usually superior to those obtained using other diagnostic protocols. The digital imaging and communications in medicine (DICOM)-formatted

datasets are imported into specific software that allows segmentation of the data set into the bone, the soft tissue, and air components (e.g., Proplan®, Materialise, Leuven, Belgium). Segmentation can be semiautomated or performed manually. The quality of segmentation is crucial for the quality of the bone-implant interface. Design software (e.g., Geomagic Freeform Plus 3D Systems, Rock Hill, USA, or 3-matic®, Materialise) is used with a mirroring technique to delineate the shape of a contralateral implant when only one side requires reconstruction. When both mandibular borders require correction, freehand artistic design, sometimes with the help of construct guidelines [24], may be quite challenging. Expectations can be discussed with the patient using Photoshop [25]. The output is a surface tessellation language (STL) file. A scaffold STL is created to overlap with the solid STL in case porosities are desired at the bone-implant interface (Fig. 20.1). Export of the components in global space coordinates, and axis markers are required if part of the implant requires finishing by CNC tooling at less than 50 μm accuracy. The implants may be printed in one or more segments to reduce the extent of the access incision and dissection or reduce the stretch of susceptible nerves (Fig. 20.1).

We have used Titanium alloy Grade 23 ELI (extra low interstitial) exclusively, which is additively manufactured by three-dimensional (3D) printing. We biofunctionalized all implants by

Fig. 20.1 A lattice-structured (scaffold) surface is created where the implant touches the boney surface. The osteoinductive environment leads to integration and fixation of the implant. The implant is segmented to facilitate introduction without jeopardizing the mental nerve



using alumina 590 μm micro-shot peening, and for our last six patients, we also used acid etching and plasma activation.

The implants are designed for a drop-in fit, yet complications may arise during implantation, such as difficulty obtaining the fit, unsuccessful intraoral screw insertion, and difficulty closing the mucosal incision line. These may lead to postoperative complications, such as aberrant morphology, wound dehiscence, and wound bed infection.

There are some ways to reduce intraoperative complications. Reference to the side of a molar tooth helps with guiding the implant into position. This may be accomplished by using a flange (tab) on the border of the distal segment (Fig. 20.2). Providing the implants with at least two screw holes is helpful to control intraoperative rotation while inserting and connecting the second segment (Fig. 20.3). Providing one or two extra holes with a direction suitable for percutaneous fixation is also useful (Fig. 20.4). Wound dehiscence is avoided by placing the incision relatively high on the buccal side of the oral vestibule and by undercorrecting at the level of the alveolus (to cover only the basal bone, which will also remain later after tooth loss) (Fig. 20.4). This reduces stress on the suture line. Double-layer closure is mandatory: a horizontal mattress suture, covered by a running suture. In patients with hemifacial microsomia, when not only the bony but also the soft tissue deficiency may be corrected by the implant, it is wise to design two or even three parts (Fig. 20.5). One part would be

used to reconstruct the bony defect at the base of the mandible, one part to correct the soft tissue deficiency, and one part to correct the bony deficiency at a higher level. As such, the surgeon can decide to insert one, two, or three parts, depending on the difficulty of wound closure in a fibrotic field.

Potential postoperative complications are similar to other facial implants including dislocation, wound dehiscence, wound bed infection, dissatisfaction with the achieved volume, irregular transition at the lower border, and facial nerve paresis. Of the 24 implantations we performed to date, dislocation of a segment occurred in one patient, and dehiscence occurred in two patients (due to early wound bed infection in one patient and late wound bed infection in the other). Two

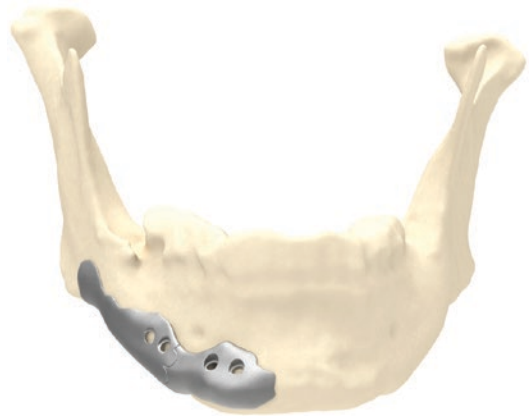


Fig. 20.3 Two screw holes per segment ensure a uniform and stable position during and after implantation

Fig. 20.2 In this patient, flanges aligned with the distal side of the last molar were useful to establish the position of the implant

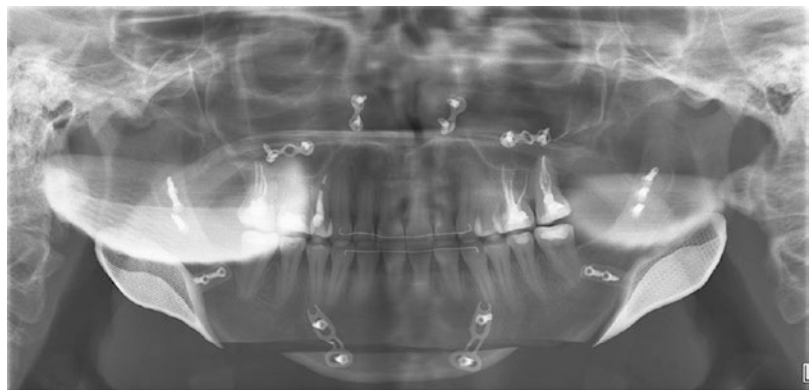


Fig. 20.4 The distal segment contains an extra hole for transbuccal fixation, to be used if transoral fixation cannot be properly performed. Lateral augmentation is achieved on the basal bone, as the alveolus may undergo resorption at an older age

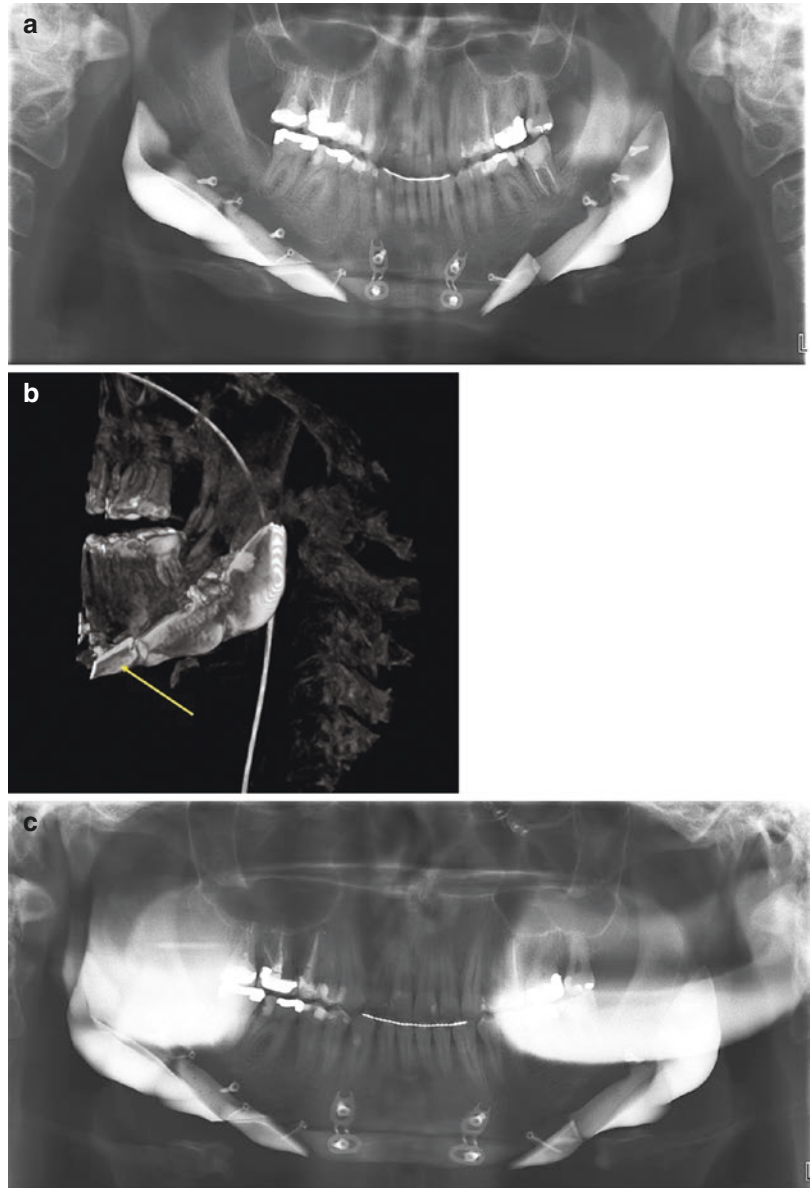


Fig. 20.5 Jaw angle implant for hemifacial microsomia, containing three segments. During surgery, we decided that the cranial segment would not be inserted. However, wound dehiscence occurred, and the implants required removal 5 months after the initial surgery. They were subsequently reinserted 7 months later, using an incision higher in the buccal mucosa

patients insisted on having the implants larger than originally designed and one patient with hemifacial microsomia suffered foreseen temporary weakness of the facial nerve.

For prevention of future complications, guidelines were developed after every complication. The dislocation occurred in a patient in which the augmentation was performed with a tripartite implant. The postoperative dislocation may have occurred because the segments were not connected with a 3D-puzzle design or because the small anterior segment was fixed with only one screw (Fig. 20.6a). The transition between the posterior segments was also palpable. Rotation of the anterior segment was corrected 2 days postoperatively as a minor procedure requiring only local anesthesia. Dehiscence occurred in one patient who had previously undergone two revision operations for a bilateral sagittal split osteotomy, had received two hydroxyapatite onlays to augment the jaw angle [26], and had chosen exaggerated 3D-printed implants. The implants were large (Fig. 20.7a), and their upper edge was close to the scarred vestibule. Dehiscence and chronic infection occurred. The implants were subsequently removed and replaced by smaller ones (Fig. 20.7b), and the incision was placed 1.5 cm higher on the buccal surface than the original incision. The postoperative course after this procedure was uneventful. The patient with hemifacial microsomia who developed facial

Fig. 20.6 (a) Rotation of a small segment, which was fixed with only one screw, as visible on OPG. (b) Intraoperative cone beam computerized tomography showed otherwise good positioning (*yellow arrow*). Note, too, that the connection between the implants was not a 3D-puzzle design, so the connection was relatively imprecise. (c) OPG taken after revision using local anesthesia only



nerve paresis (Fig. 20.8a–c) received an implant that extended 2 cm posteriorly (Fig. 20.9). Dissection of the pocket was performed using gauze packing. As the course of the facial nerve in hemifacial microsomia is unpredictable, sharp dissection was specifically avoided. Full recovery of nerve function occurred within 3 months.

There is discussion about the material to use for customized jaw angle reconstruction. Some surgeons prefer polyether ether ketone (PEEK) that is CNC milled because it can be easily removed. Indeed, implants made of this material are encapsulated, not integrated into bone. However, the untextured surface may increase the likelihood of seromas and late infectious

Fig. 20.7 (a) View of the initial overcontoured design. (b) View of the design of the replacement implants

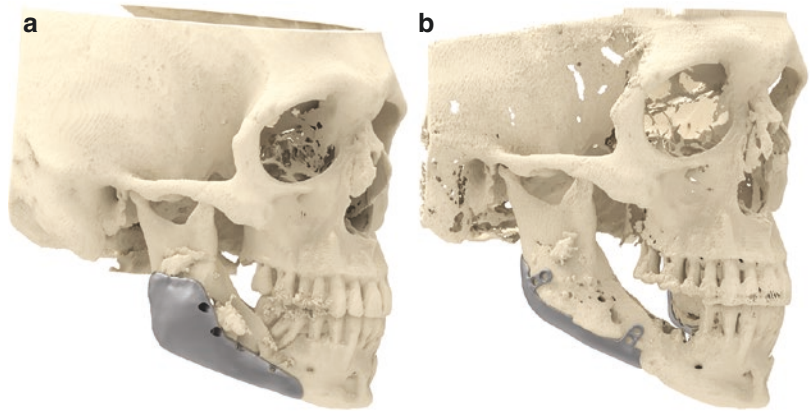


Fig. 20.8 Patient with hemifacial microsomia at end-stage reconstruction. (a) At the age of 4 years, 3 years plagiocephaly correction, prior to total temporomandibular joint reconstruction at the age of 5 years and prior to facial rotation by orthognathic surgery at the age of

15 years. (b) The jaw angle implant extended posteriorly for 2 cm, and blunt dissection stretched the facial nerve, resulting in paresis. (c) Near full recovery of facial nerve function by 3 months postoperatively

complications, with other encapsulated implants. Sandblasted titanium may produce superior periosteal attachment, periosteal regeneration, and muscle reattachment. The porous bone contact interface is osteoinductive. Nevertheless, late removal may be difficult. Early removal because of site infection was not a problem in this small series. Shining through of the gray titanium was not observed. Time will tell which material produces the best outcomes. A registry, such as that created for temporomandibular joint prostheses, may be

necessary to determine the optimal material (Tables 20.1 and 20.2).



Fig. 20.9 Lateral and medial views of the implant used for the patient described in Fig. 20.8

Table 20.1 Complications seen with different areas of reconstruction

| Location | Most common complications |
|------------------------|---|
| Midface | Inadequate correction or overcorrection |
| | Malpositioning |
| | Migration |
| | Infection |
| | Extrusion |
| | Hypesthesia/hypoesthesia |
| | Facial nerve injury |
| Orbit | Diplopia |
| | Enophthalmos |
| | Infection |
| | Extrusion |
| | Migration |
| | Lacrimal duct obstruction |
| | Hematoma formation |
| | Erosion into maxillary sinus |
| Lower eyelid deformity | |
| Nose | Aesthetic failure |
| | Migration |
| | Infection |
| | Extrusion |
| Mandible/chin | Inadequate correction or overcorrection |
| | Asymmetry |
| | Malpositioning |
| | Bone resorption |
| | Infection |
| | Extrusion |
| | Hypesthesia/hypoesthesia |

Conclusion

Facial implants have become increasingly popular and widely used in facial reconstruction. There are many factors that determine the success of the surgical attempt at alloplastic facial augmentation including the patient's health status, the properties of the implant material, and the surgeon's experience. These materials have the advantage of being readily available; they lack the donor-site morbidity and result in decreased operative time. The most common complications include infection, extrusion, implant migration, malpositioning, nerve disturbance, palpable implant, fluid accumulation, and bone resorption. It is still controversial as to whether smooth or porous implants have a

Table 20.2 Complications seen with different implant materials

| Material | Common complications |
|----------------|--|
| Silicone | Late-onset infection |
| | Extrusion |
| | Migration |
| | Seroma formation |
| | Chronic peri-implantitis |
| MedPor | Early-onset infection |
| | Extrusion |
| | Fistula formation |
| | Hypoesthesia |
| | Palpable or visible implant |
| GoreTex | Difficult removal |
| | Infection |
| | Extrusion |
| | Hardening of edges on contact with blood |
| | Palpable or visible implant |
| Mersilene mesh | Over augmentation |
| | Seroma |
| | Increased operative time (secondary to formation of implant in OR) |
| | Decreased solidity |
| | Smaller augmentation |
| | Migration |
| | Difficult removal |

higher overall infection risk. The use of aseptic technique, antibiotic administration, soaking/irrigating the implant in antibiotic solution, careful patient selection, and proper intraoperative techniques should help decrease complications. The surgeon should use the best clinical acumen and systematic preoperative facial analysis to evaluate patients and help manage patient expectations.

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