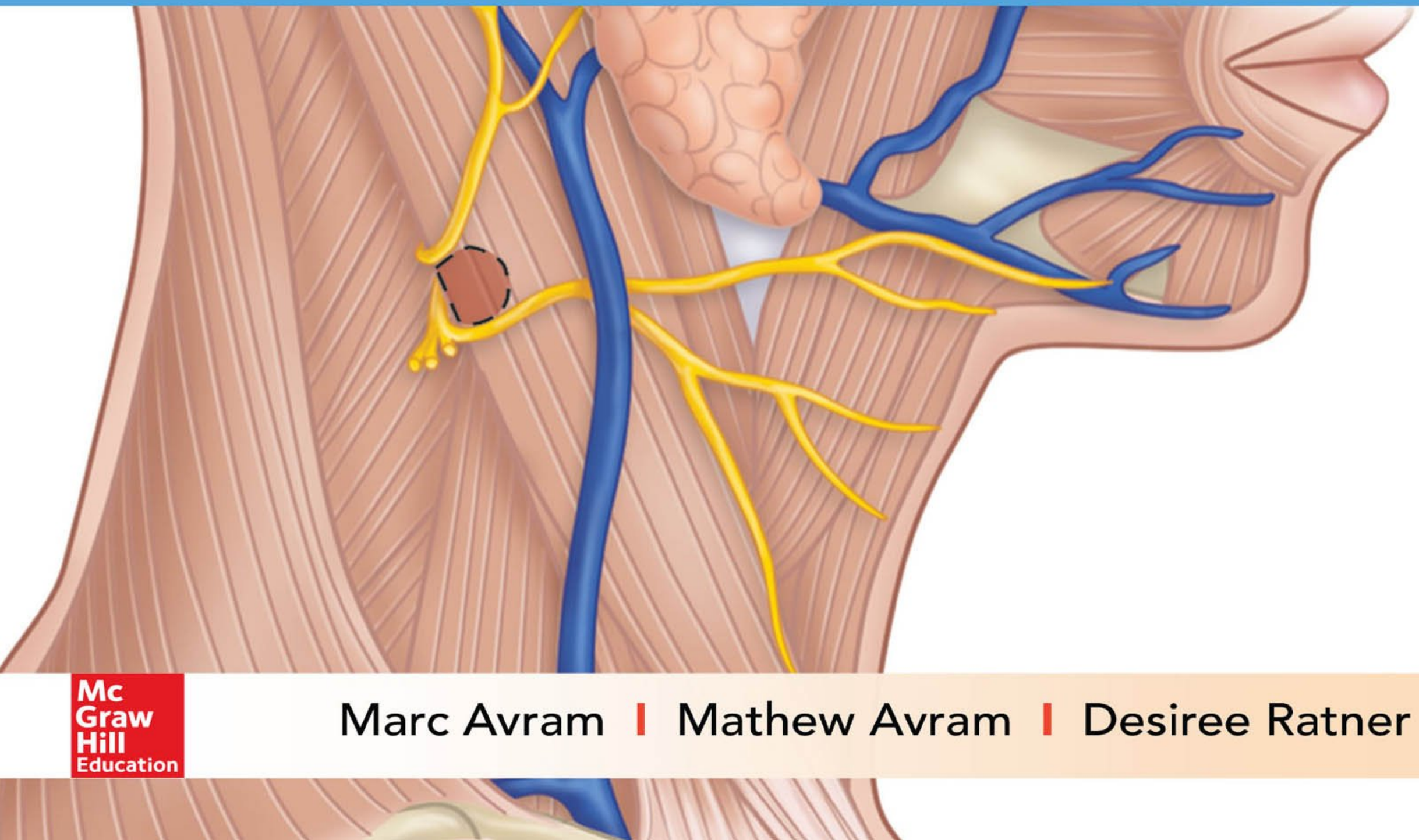
PROCEDURAL DERMATOLOGY



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Procedural Dermatology

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Marc R. Avram, MD

I would like to dedicate this book to my loving parents Morrell and Maria Avram. They have always given me and my family unconditional support and love. Thank you!

To my wife Robin for all of her love and being my best friend. To my sons Robert and Jacob, thank you for always bringing joy, love, and wonder to my life. I am very proud of you both.

Mathew M. Avram, MD, JD

I would like to dedicate this textbook to my parents, Morrell and Maria Avram, who have provided me with their love and support my entire life. To my beautiful wife, Alison. Thank you for your love, understanding and support. To my wonderful children Rachel, Alexander and Noah. Thank you for inspiring me with your love, joy and accomplishments.

Désirée Ratner, MD

To my parents, Marion and Paul Ratner, for always being there, to my children, Xanthe and Evan Thomas, for their love and support, and to Paul Heller, for his kindness, love, and understanding.

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Contents

Section 4 Aesthetic And LASer ProcedureS

Section 5 AeSthetic ProbLemS

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Pr e FACe

Dermatologic surgery is a discipline that has evolved dramatically from the mid-20th century to the present day. Dermabrasion, chemical peels, hair transplantation, Mohs chemosurgery, and excisional surgery were performed principally by a select group of NYU dermatologists in the 1950s, while cutaneous laser surgery was first performed at the University of Cincinnati in the 1960s. Over the years, the number of dermatologists performing these procedures increased significantly. Our knowledge of basic science has also advanced, as has our clinical expertise in wound healing and postoperative care. Dermatologic surgeons have become experts in Mohs micrographic surgery and defect reconstruction, while our cosmetic armamentarium has grown to include botulinum toxins, a variety of soft tissue fillers, sclerotherapy, ambulatory phlebectomy, nonablative and ablative lasers, fractional resurfacing, lasers for the treatment of tattoos, pigmented and vascular lesions, and liposuction. Standards of training, patient safety, informed consent, anesthesia and analgesia, preoperative evaluation, and of ce accreditation are now recognized to be of vital importance, and have become disciplines of their own. Now, decades after a small group of dermatologists founded the American Society for Dermatologic Surgery in 1970, surgical training has become a fundamental and required part of all dermatology residencies, while postgraduate fellowship training in procedural dermatology is an ACGME accredited subspecialty.

Developing knowledge and expertise in any field requires starting with the basics, and building upon them. To that end, we have organized this text into the following sections: surgical principles, surgical skills, skin tumors, aesthetic and laser procedures, and aesthetic problems. We have endeavored to develop a consistent format for each chapter, with high-quality photographic images and graphics to enhance the presentation of each topic. T e reader may choose to progress sequentially through the book, or to focus individually on the topics of interest to them.

Our goal in developing this textbook was to compile the current, state of the art information concerning all aspects of procedural dermatology. T e authors of this text are without exception leaders in their fields with a passion for teaching, and we feel fortunate to have enlisted them to assist us in this endeavor. Our hope is that this book will become a resource for all practitioners of dermatologic surgery, from the novice to the seasoned surgeon looking to refine his or her skills in a particular area. We hope you enjoy reading this book as much as we have enjoyed putting it together.

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2014

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Section 1

Surgical Principles

chapter

Superf cial Head and Neck Anatomy

Hugh T. Greenway, Vineet Mishra, Lee M. Miller, &Salman Alsaad

INTRODUCTION

Mastering surgical anatomy is crucial for the dermatologic surgeon, not only to obtain the best cosmetic outcome but also to avoid complications related to injury of underlying anatomic structures. Since a significant number of dermatologic surgeries are performed on the head and neck, the dermatologic surgeon ought to have full knowledge of the facial nerve and its branches, blood supply, sensory nerves, underlying musculature, and specialized structures such as the parotid duct. With a full understanding of the superficial anatomy of this complex region, the physician will have the confidence to perform a wide range of superficial surgical procedures while keeping possible risks and complications in mind.

NOSE

T e external nose is shaped like a pear or pyramid with several key divisions: dorsum, tip, ala, and lateral sidewalls. T e dorsum of the nose extends from the tip, the lowermost aspect of the nose, to the nasal root. In contrast to the dorsum of the nose where the skin is quite mobile, the skin overlying the tip of the nose is adherent to the underlying fibrous tissue and cartilage. T e nasal bridge is the portion of the nose overlying the nasal bones. T e lateral sides of the nose encompass the area from the nasal dorsum to the cheeks. T e nasofacial sulcus, or margin of the nose, is the transition from the lateral aspects of the nose to the cheeks.[1]

T e ala, a distinct cosmetic subunit, is demarcated from the lateral sidewalls of the nose by the alar crease and separated from the lip by the nasolabial crease. T e root of the nose is the region of attachment of the nose to the glabella. T e nares are the openings into the anterior nasal vestibules. T ey are divided by the nasal septum medially and bounded by the nasal alae laterally. T e columella is the freely movable portion of the nasal septum (Fig. 1-1).[2]

T e skeleton of the nose includes both bone and cartilage. T e two nasal bones lie adjacent to the maxilla laterally and the frontal bone superiorly. T ere are also five major cartilages of the nose which provide the remainder of the nasal framework: two lateral cartilages, two greater alar cartilages, and the single septal cartilage. In the midline, the septal cartilage joins each of the lateral cartilages. T e lateral aspect of the lateral cartilage articulates with the frontal process of the maxilla whereas the superior aspect of the lateral nasal cartilage articulates with the inferior aspect of the nasal bone. T e greater alar cartilages are C-shaped. Each of the medial crura joins at the midline with the septal cartilage to form the nasal septum. T e lateral crura articulate with the inferior aspects of the lateral nasal cartilages.[1]

T ere are several muscles associated with the nose: the nasalis, the depressor septi, the levator labii superioris alaeque nasi, and the procerus muscles. T e transverse

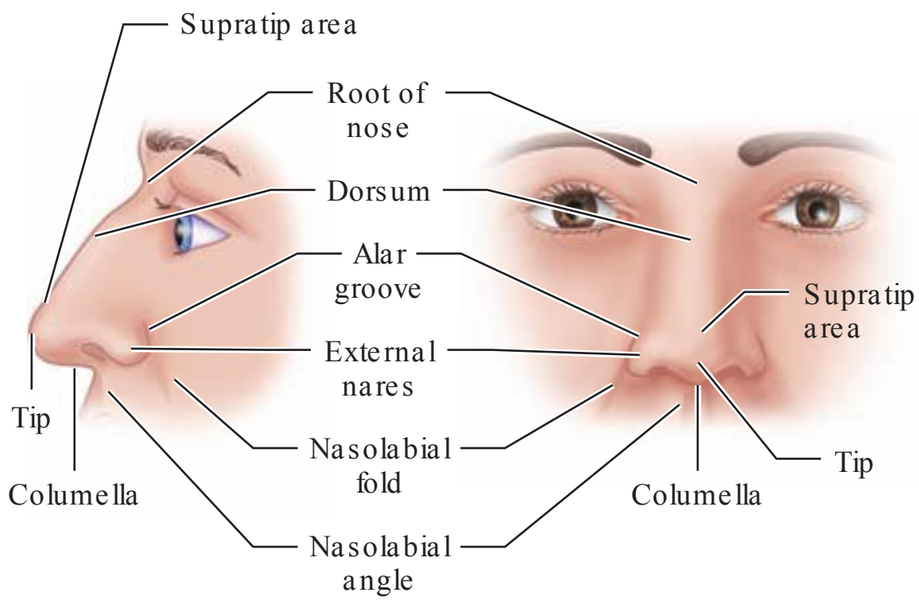

Figure 1-1 Cutaneous anatomy of the nose.

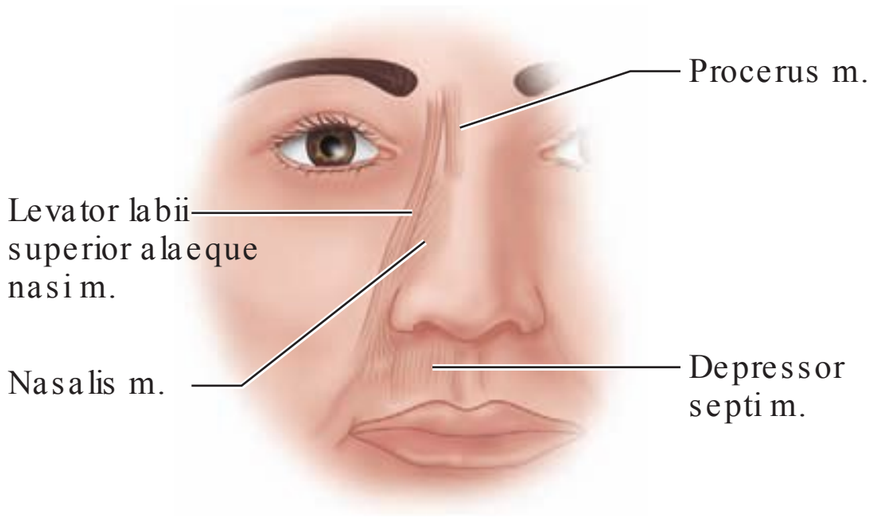

Figure 1-2 Musculature of the nose.

portion of the nasalis muscle originates from the body of the maxilla on each side and inserts into the aponeurosis that overlies the bridge of the nose. A small portion of the nasalis, the alar component, inserts into the lateral crus of the alar cartilage. Contraction of the transverse portion of the nasalis muscle compresses the nares; meanwhile, activation of the alar portion causes dilation of the nares. Innervation of the nasalis muscle occurs via the buccal branch of the facial nerve. The depressor septi muscle functions to narrow the nares. It originates from the incisive fossa of the maxilla and inserts into the columella and nasal septum. The buccal branches of the facial nerve also innervate this muscle.[3] The levator labii superioris alaeque nasi muscle originates from the frontal process of the maxilla and courses inferiorly with a portion of the muscle inserting into the ala before the main body inserts into the skin of the upper lip. The alar portion dilates the nares and is innervated by the buccal branches of the facial nerve. The procerus muscle arises from the nasal bones and inserts into the skin overlying the glabella. Contraction of this muscle leads to downward pulling of the skin while creating horizontal folds over the bridge of the nose. Unlike the other nasal muscles, the procerus is innervated by the temporal branch of the facial nerve (Fig. 1-2).[1]

Sensory innervation to the nose is provided by the terminal branches of the ophthalmic and maxillary divisions of the trigeminal nerve. The supra- and infratrochlear branches innervate the nasal root, bridge, and upper sidewall. The dorsal external nasal branch of the anterior ethmoidal nerve innervates the lower dorsum and nasal tip. The infraorbital nerve provides sensation to the lateral sidewall and ala whereas the depressor septi branch is responsible for the columella.[4]

The vasculature supply to the nose is quite robust. The arterial supply to the external nose primarily arises from the lateral nasal and septal branches of the superior labial artery, which emanates from the facial artery. There are also secondary branches from the infraorbital artery, nasal branches from the infratrochlear artery via the ophthalmic artery, and the dorsal nasal branch of the anterior ethmoidal artery, also from the ophthalmic artery (Fig. 1-3). The veins of the external nose are tributaries of the anterior facial vein. The nose lies in the "danger area" of the face given the open communication between the anterior facial vein and ophthalmic veins, which drain into the cavernous sinus.[1] The lymphatic drainage of the nose flows into the buccal nodes of the facial lymphatic chain and then subsequently into the submandibular nodes.[4]

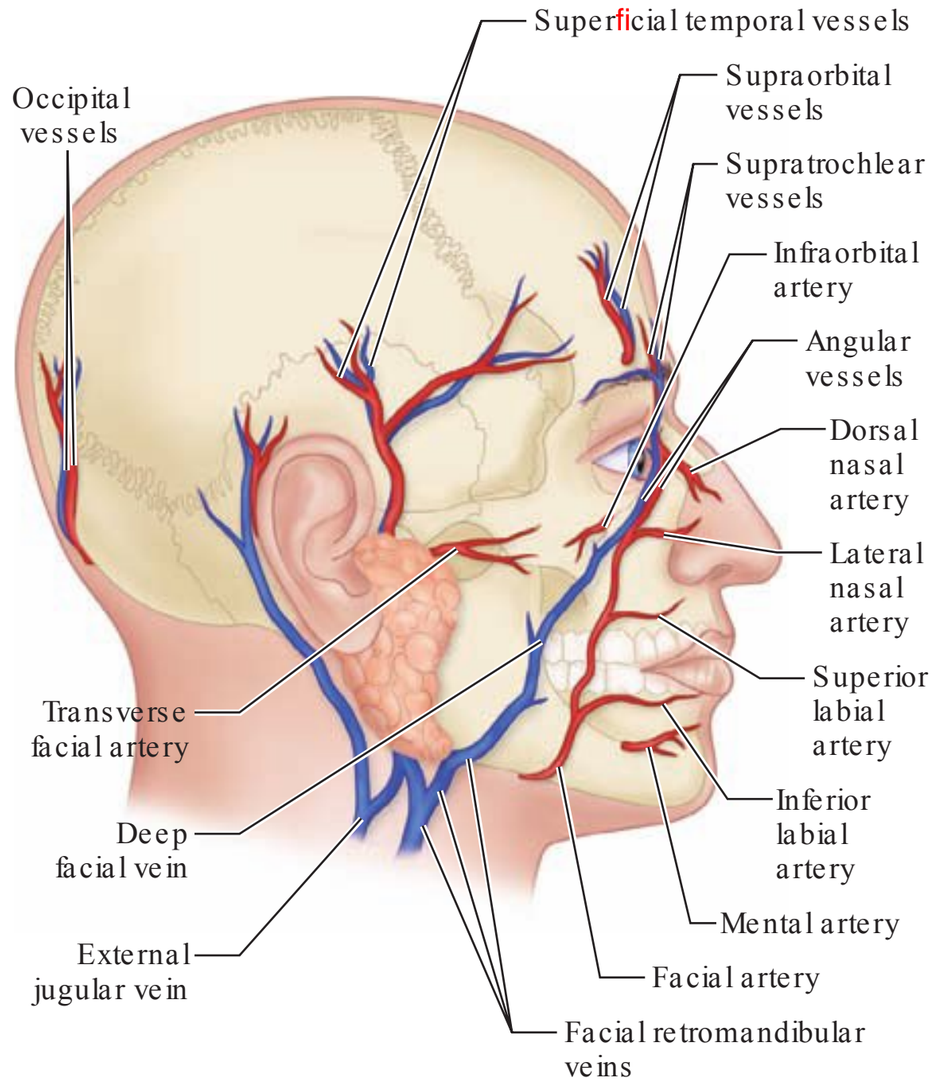

Figure 1-3 Vasculature of the face.

PERIORAL REGION

The labial area of the face is demarcated by the mental region inferiorly, the nose superiorly, and the buccal areas of the cheeks laterally. The lips are two structures defining the entrance to the mouth. The upper and lower lips are joined laterally at the labial commissures. The upper lip is separated from the cheek by the melolabial crease, which extends from the ala of the nose inferiorly and obliquely, and is divided into two halves by the philtrum. The philtrum is a depression in the central upper lip bounded by the two prominences (philtral ridges) laterally, the vermilion border of the upper lip inferiorly, and the nasal septum superiorly. The crossing of the muscle insertions within the superficial portion of the orbicularis oris muscle is hypothesized to lead to the formation of the philtrum.[2] Each lip also has a vermilion border, a region of transition from normal skin to modified epithelium without hair, sweat glands, and sebaceous glands. The junction of the vermilion border with the philtrum is known as "Cupid's bow."[1]

The oral musculature arises from different regions on the face. For purposes of simplification, these can be divided into four major groups: the muscles of the lower lip (depressors), the muscles of the upper lip (elevators), the buccinator, and the orbicularis oris. The muscles of the lower lip are comprised of the depressor anguli oris, the depressor labii inferioris, and the mentalis. The depressor anguli oris depresses the corners of the mouth. It arises from the anterolateral aspect of the body of the mandible and inserts into the skin and mucosa at the labial commissure (Fig. 1-4). Similarly, the depressor labii inferioris originates from the mandible deep to the depressor anguli oris muscle and inserts into the skin and mucosa of the lower lip, thereby depressing the lips. The mentalis originates

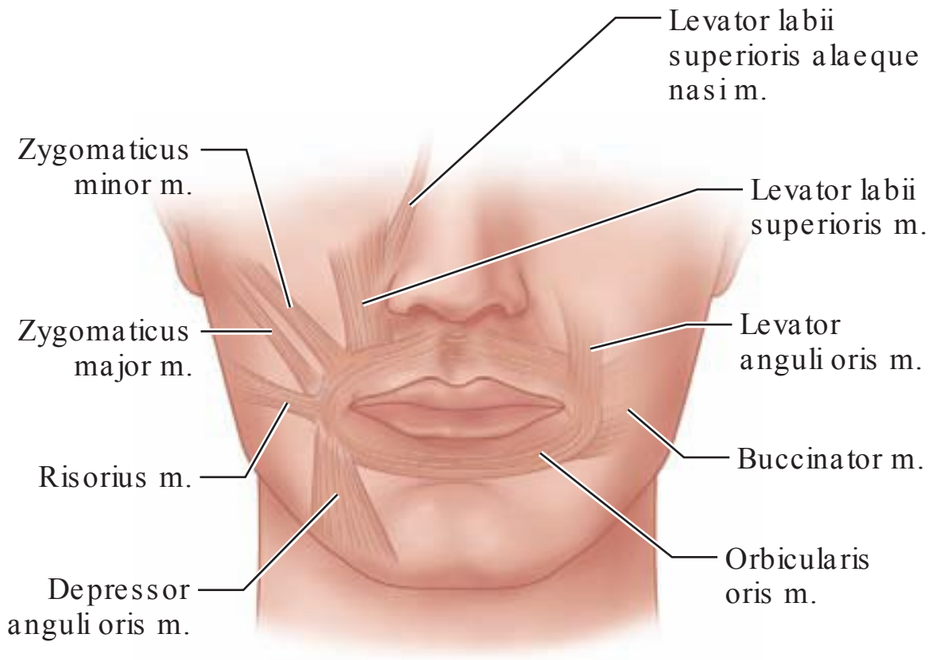

Figure 1-4 Musculature of the perioral area and medial cheek.

from the body of the mandible medial to the depressor labii inferioris and inserts into the skin overlying the tip of the chin. Upon contraction, it leads to protrusion of the lower lip and dimpling of the skin. These muscles are innervated by the marginal mandibular branch of the facial nerve with secondary innervation from the buccal branches of the facial nerve.[5]

The muscles of the upper lip consist of the risorius, zygomaticus major and minor, levator labii superioris, levator labii superioris alaeque nasi, and levator anguli oris muscles (Fig. 1-5). The risorius arises from the parotid fascia and inserts into the skin and mucosa at the corner of the mouth. This leads to pulling of the labial commissure laterally. The zygomaticus major muscle arises from the posterolateral aspect of the zygomatic bone; it inserts into the skin and mucosa of the lateral upper lip and elevates the labial commissure. The zygomaticus minor muscle arises medial to the zygomaticus major and inserts into the skin and mucosa of the upper lip, thus elevating the upper lip. The levator labii superioris originates from the maxilla just above the infraorbital foramen and inserts into the skin and mucosa of the medial upper lip, thereby elevating the upper lip. The levator labii superioris alaeque nasi arises from the medial origin from the orbit, gives of fibers to the nasal ala, and proceeds inferiorly to insert into the skin and mucosa of the medial aspect of the upper lip. This muscle not only elevates the upper lip but also dilates the

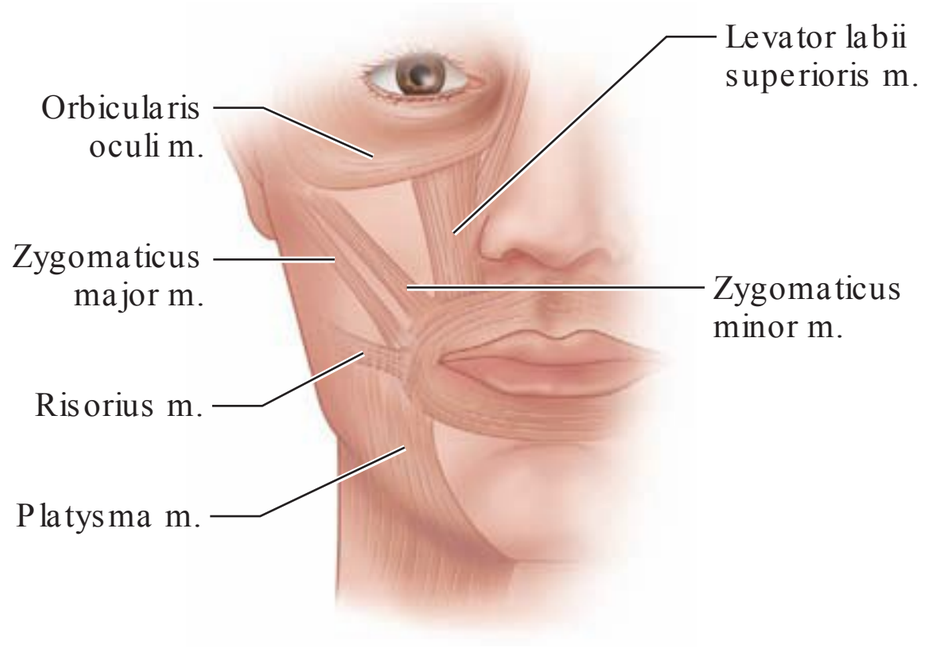

Figure 1-5 Superficial musculature of the perioral area.

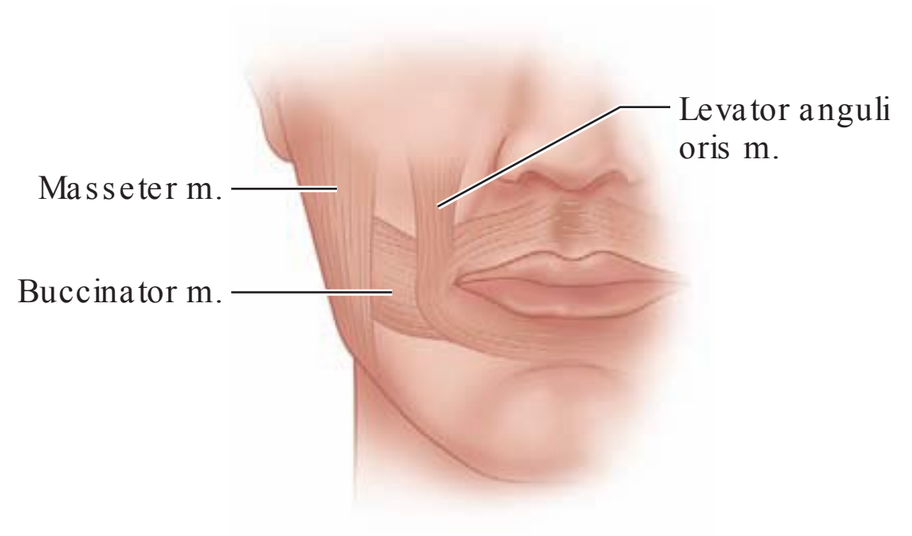

Figure 1-6 Deeper musculature of the perioral area.

nares. The levator anguli oris originates from the maxilla below the infraorbital foramen and inserts into the skin and mucosa at the labial commissure, thus elevating the corners of the mouth. These muscles are innervated by the buccal branches of the facial nerve with secondary innervation from the zygomatic branches of the facial nerve.[2]

The buccinator, also known as the cheek bulk muscle, is the key muscle of the cheek (Fig. 1-6). It originates from the posterolateral aspect of the maxilla, the medial aspect of the mandible near the last molar, and the pterygomandibular raphe. Its insertion is into the skin and mucosa of the labial commissure as well as the upper and lower lips. It serves to press the lips and cheeks against the teeth. Its innervation is from the buccal branches of the facial nerve. Of note, this is the only muscle of facial expression that receives its innervation from the superficial aspect of the facial nerve.[5]

Finally, the orbicularis oris consists of concentrically arranged fibers providing a sphincteric function, which allows for pursing and protrusion of the lips. It is mostly innervated by the buccal branches of the facial nerve.[2]

The sensory supply to the lips is provided by the branches of the trigeminal nerve. The maxillary division of the trigeminal nerve gives of the infraorbital branch, which innervates the upper lip, lower eyelid, infraorbital cheek, parts of the buccal cheek, malar cheek, and lateral aspect of the nose. The mandibular division of the trigeminal nerve branches into the mental nerve, which provides sensation to the lower lip. To identify these nerves, one may visualize an imaginary vertical line dropping inferiorly from the supraorbital notch. The infraorbital foramen and nerve are found approximately 5 mm below the inferior margin of the orbit. The mental foramen and nerve are intersected by this imaginary line and are located midway along the height of the body of the mandible.[5]

The vascular supply to the lips arises from the facial artery via the inferior and superior labial arteries. These tortuous vessels meander through the muscle fibers and anastomose across the midline; therefore, if needed, the facial artery may be ligated at its point of entry into the face without fear of ischemia to the ipsilateral face. The venous drainage from the lips occurs through the superior and inferior labial veins, which eventually join the anterior facial vein. The lymphatic drainage from the lips is multifaceted. The central and ipsilateral regions of the upper lip drain into the ipsilateral submandibular nodes. The lateral regions of the lower lip flow into the ipsilateral submandibular nodes; however, the central portion of the lower

lip flows into both ipsilateral and contralateral submandibular and submental nodes. This is of great importance when there is concern for possible metastasis of tumors in this location.[1]

PERIORBITAL REGION

The orbital rim is composed of the following bones: frontal, zygomatic, maxillary, and lacrimal. Key landmarks include the supraorbital margin, supraorbital notch, and infraorbital margin. The supraorbital notch, from which the supraorbital nerve exits, is found 2 cm from the midline. The lateral orbital tubercle, also known as Whitnall's tubercle, is a small bony prominence located 10 mm below the zygomaticofrontal suture and 4 mm behind the anterolateral orbital rim. This is the site of attachment for the lateral palpebral ligament. In the area of the medial orbital rim is the anterior lacrimal crest, to which the medial palpebral ligament is attached. The lacrimal sac is located posterior to the medial palpebral ligament.[3]

The eyelids form a protective covering for the underlying globe. The upper eyelid is larger than the lower eyelid and provides the bulk of protection to the underlying eyeball. The skin of the eyelids is quite thin and continuous with the conjunctiva at the free margins. The palpebral fissure is the space between the open eyelids. The commissure or canthus is the site where the upper and lower eyelids join. The medial canthus is separated from the surface of the eyeball by the lacrimal caruncle, which appears as an erythematous structure. The space occupied by the lacrimal caruncle is the lacrimal lake. The plica semilunaris is the portion of the folded conjunctiva lateral to the lacrimal caruncle. Along the free margins of the medial eyelids are small elevations at the base of the eyelashes known as lacrimal papillae. Each papilla encircles a punctum, which marks the opening to the lacrimal canalicular system through which the fluid drains into the lacrimal sac. From there, the nasolacrimal duct, which typically runs for approximately 18 mm, empties its contents into the inferior meatus of the nose. The lacrimal gland, located behind the orbital margin of the zygomatic process of the frontal bone, consists of a larger orbital portion and a smaller palpebral portion and provides lubrication for the eye (Fig. 1-7).[4]

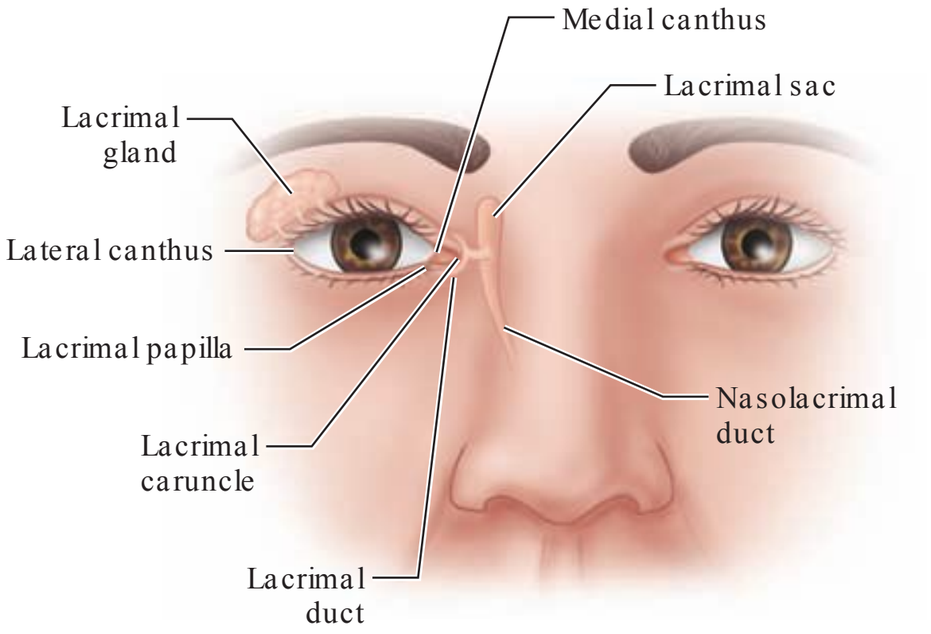

Figure 1-7 Anatomy of the lacrimal system.

The conjunctiva covers the globe and lines the inner surface of the eyelids to form the conjunctival sac. The tarsal plates of the upper and lower lids give form and shape to the eyelids. On average, the height of the tarsal plate in the upper lid varies between 9 and 11 mm whereas that of the lower lid is about 4 to 5 mm. The medial palpebral ligament secures the tarsal plates to the anterior lacrimal crest at the medial orbital rim. The upper and lower tarsal plates are also secured via the lateral palpebral ligament to the lateral orbital tubercle, also known as Whitnall's tubercle.[3]

The key muscles of the eyelid are the orbicularis oculi and levator palpebrae superioris. The orbicularis oculi allows for closure of the eye whereas the levator palpebrae superioris opens the eye. The orbicularis oculi muscle fibers encircle the eyelids, forehead, temple, and cheeks. This is muscle functions as a sphincter and can be divided into palpebral and orbital portions, whose primary and secondary innervation are provided by the zygomatic and temporal branch of the facial nerve, respectively. Upon contraction, the muscle allows for protection of the globe and even spreading of tears across the surface of the eye. Damage to the muscle or its innervation may lead to ectropion of the lower eyelid. The second eyelid muscle of note is the levator palpebrae superioris muscle, which is innervated by the third cranial nerve (the oculomotor nerve).[5] This is muscle originates from the orbital apex and then fans out, molding itself around the globe before inserting onto the anterior surface of the tarsus. It also has fibers above the superior border of the tarsus between the orbicularis oculi and the skin, which forms the supratarsal eyelid crease.

The orbital septum is a fibrous sheath lying beneath the orbicularis oculi muscle. This is structure marks the anterior border of the orbit and encases the orbital fat. The orbital septum originates from the bony margins of the orbit and lies anterior to the tarsal plates. The septum is thinnest at its medial aspect (Fig. 1-8). There are two fat pads posterior to the orbital septum in the upper eyelid and three fat

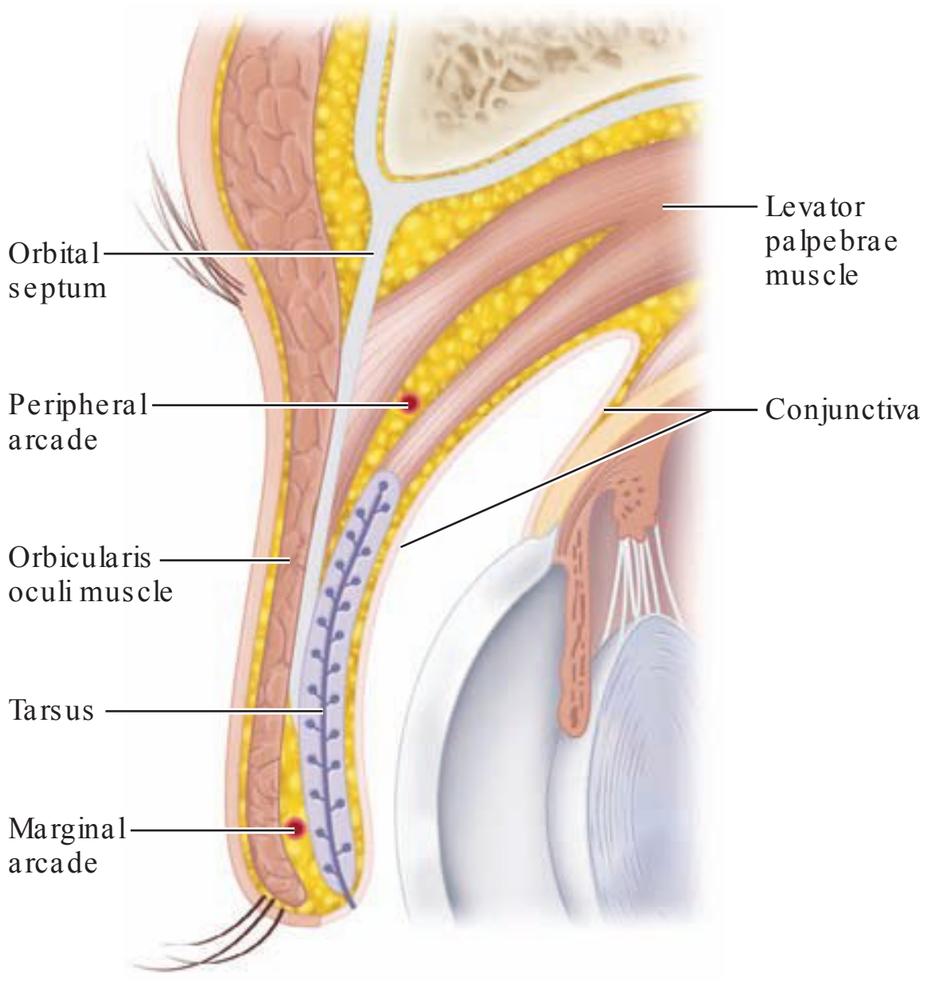

Figure 1-8 Sagittal view of the upper eyelid.

pads posterior to the orbital septum in the lower eyelid. In addition, in the upper eyelid, posterosuperior to the orbital septum, is the lacrimal gland.[1]

The nerve supply to the eyelids consists of both motor and sensory components. The zygomatic and temporal branches of the facial nerve supply motor innervation to the orbicularis oculi. The oculomotor nerve innervates the levator palpebrae superioris. Sensory innervation is provided by the ophthalmic division of the trigeminal nerve. The ophthalmic division of the trigeminal nerve gives rise to the supraorbital, supratrochlear, and lacrimal nerves, which provide sensation to the upper eyelid. The lower eyelid is innervated by the infraorbital nerve, which arises from the maxillary division of the trigeminal nerve. Of note, visual acuity should be documented prior to any surgical procedure in the periorbital region. After surgery, if the eye needs to be patched, extra care should be exercised to ensure that it is patched in the closed position to avoid corneal abrasions and injury to other structures of the eye.[4]

The vascular supply to the eyelids is derived from the rich arterial supply from both the internal and external carotid arteries. The ophthalmic artery arises from the internal carotid artery and gives rise to the supraorbital, supratrochlear, medial palpebral, dorsal nasal, and lacrimal arteries. The medial palpebral arteries include both superior and inferior components that dive through the orbital septum medially and then traverse laterally over the eyelid. Of note, the superior palpebral artery supplies both the peripheral and marginal arcades. The peripheral arcade is located in the upper body of the tarsus whereas the marginal arcade is found in the lower portion of the tarsus just superior to the eyelashes in the pretarsal space. Meanwhile, the external carotid artery gives of the maxillary and superficial temporal arteries. The maxillary artery gives rise to the infraorbital artery, which exits the infraorbital foramen and supplies the lower eyelid. The superficial temporal artery branches into the transverse facial artery, the zygomatico-orbital artery, and the frontal artery. These supply the lateral orbit and forehead. There are two key sites for anastomosis: first in the lateral eyelid, between the superficial temporal artery and the lateral distribution of the infraorbital artery, and second in the medial eyelid, between the inferior medial palpebral artery and the infraorbital artery.[1]

The venous network of the eyelid is also quite extensive, and parallels the arterial system. The veins drain into the ophthalmic venous system, the superficial temporal vein, or the angular vein. Of note, the ophthalmic venous system flows into the dural venous sinus. The lymphatic drainage of the periorbital area is divided into lateral and medial components. The lateral aspects of the upper and lower eyelids drain into the preauricular nodal basin whereas the medial aspects drain to the central facial and submandibular nodes.[3]

SCALP

The scalp consists of the skin and multiple layers of soft tissue overlying bone. The calvarium, which is composed of the frontal, occipital, and two paired parietal bones, serves as the foundation for the scalp. The scalp bones are separated by the coronal, sagittal, lambdoid, and squamosal sutures. The soft tissues above it include five distinct layers which are easily remembered using the following mnemonic:

Skin
Connective tissue (subcutaneous tissue)
Aponeurosis (galea aponeurotica)
Loose areolar connective tissue
Periosteum

On the scalp, the skin extends from the supraorbital ridges of the frontal bone posteriorly to the superior nuchal lines of the occipital bone. The lateral portions cover the parietal bones and temporalis muscle. The forehead may be considered a subsection of the scalp extending from the supraorbital ridges to the highest wrinkle overlying the frontal bone. The flat portion of the forehead is due to the underlying squamous portion of the frontal bone. Posteriorly, the inion is a protuberance which may be palpated at the midline of the occipital bone. Extending laterally from the inion are two ridges forming the superior nuchal lines (Fig. 1-9).[1]

The bones of the calvarium consist of an inner table and an outer table of cortical bone separated by a spongy diploe layer of trabecular bone. These bones receive their blood supply from both the interior and exterior surfaces. On occasion, the dermatologic surgeon may need to sample the outer table to evaluate for invasion of tumor, or may remove portions of the outer table to stimulate the formation of granulation tissue. Care must be taken to avoid penetrating the inner table if sampling is done with the osteotome and mallet. The surgeon should also avoid using an osteotome across suture lines of the calvarium or on a site which has been irradiated previously.[5]

Under the skin of the scalp lies the connective tissue layer (subcutaneous tissue), which contains most of the vascular and nerve supply for the scalp. The scalp vasculature consists of many anastomosing branches which originate from both the internal and external carotid

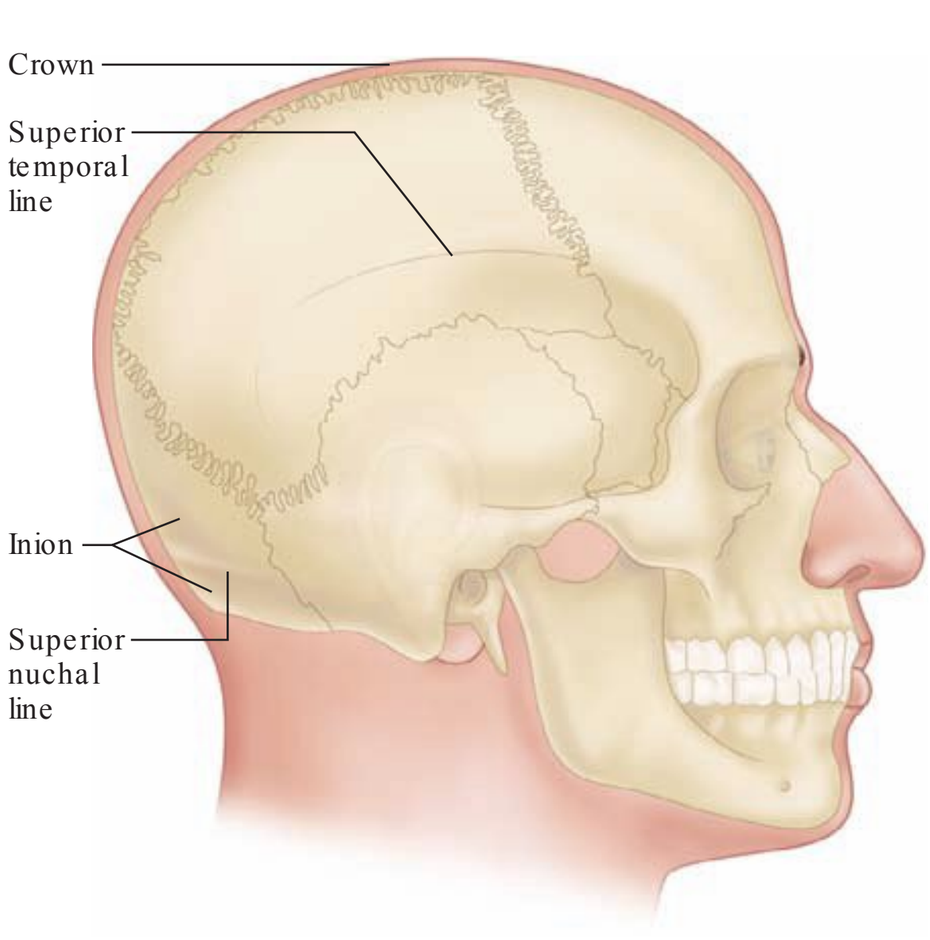

Figure 1-9 Landmarks of the scalp.

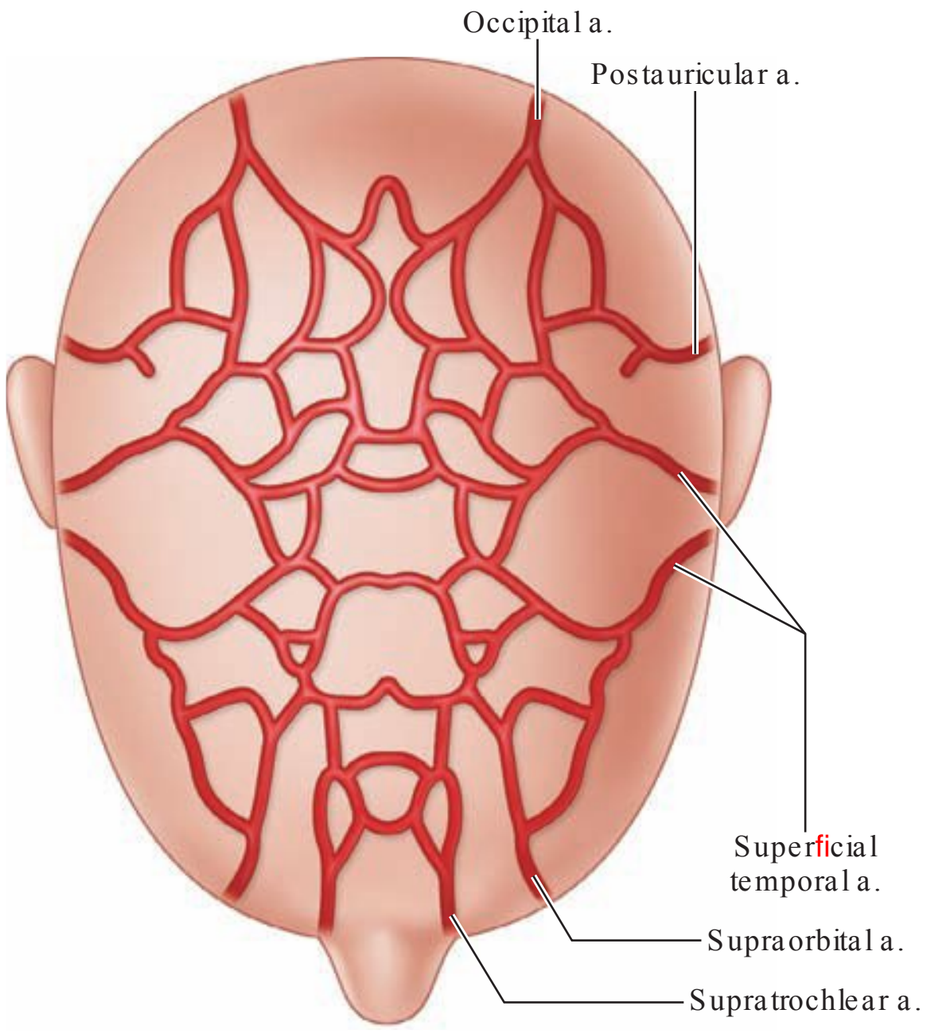

Figure 1-10 Vasculature of the scalp.

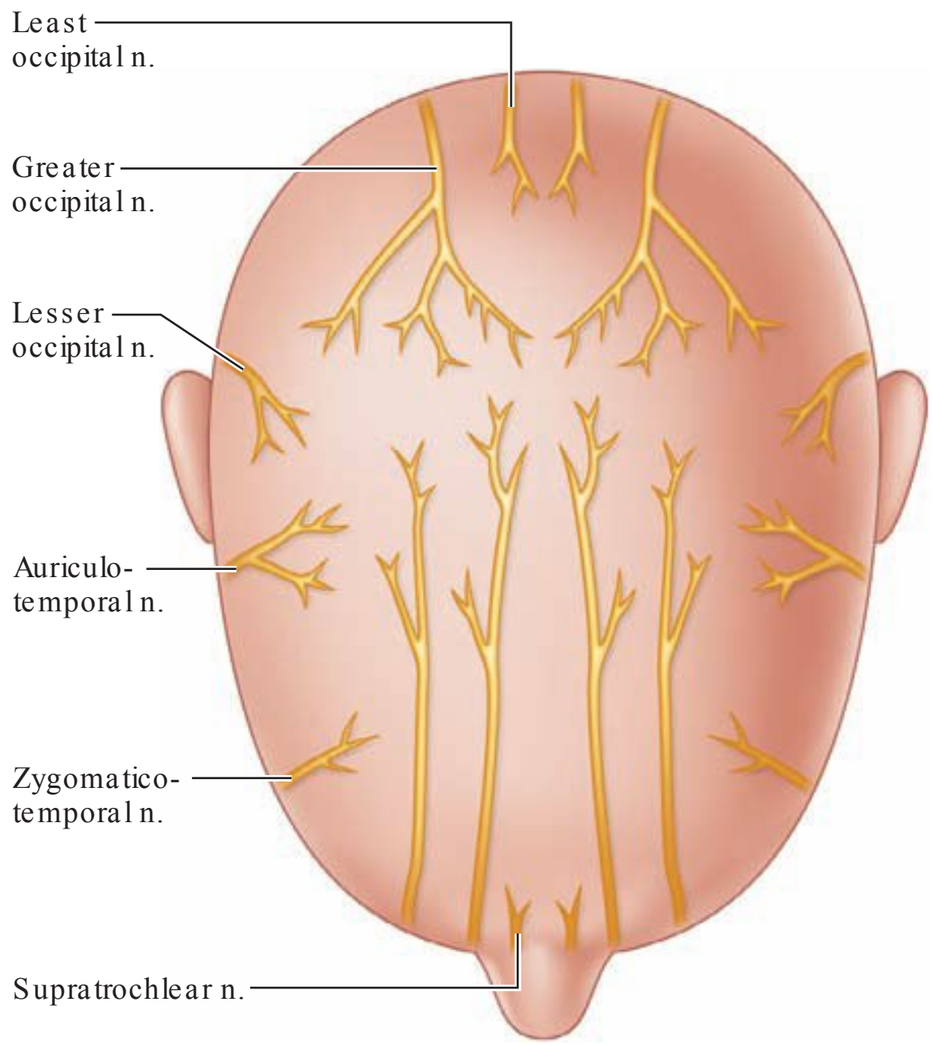

Figure 1-11 Sensory innervation of the scalp.

arteries. The occipital artery and posterior auricular artery are branches from the external carotid which supply the occipital and retroauricular scalp. The external carotid artery eventually branches into the superficial temporal artery, which is palpable at the superior pole of the parotid gland, supplying the temporal scalp. The internal carotid artery, via the ophthalmic artery, divides into the supraorbital, supratrochlear, and lacrimal arterial branches. After traversing the orbit, the supraorbital artery exits the supraorbital foramen as part of a neurovascular bundle approximately 2 to 2.5 cm lateral to the nasion along the superior orbital margin.[4] This foramen is palpable as the supraorbital notch. The supratrochlear artery also traverses the orbit and wraps around the supraorbital rim approximately 1 cm medial to the supraorbital notch to supply the medial forehead. The supraorbital and supratrochlear arteries supply the forehead and anterior scalp. The remainder of the scalp is perfused via anastomosing branches of the occipital, postauricular, superficial temporal, and supraorbital arteries (Fig. 1-10).[2] Of note, the connective tissue of the scalp contains fibrous retinacula which restrain vascular spasm, potentially leading to increased surgical bleeding as vessels are transected.[1] The venous circulation of the scalp generally parallels arterial supply.[6] Veins of the head and neck are devoid of valves and flow is, therefore, determined strictly by pressure gradients. Emissary veins pass through the calvarium, allowing for communication between the extracranial and intracranial vessels. This may serve as a potential route for spread of either infectious or neoplastic processes.

The sensory innervation of the scalp is provided by the terminal branches of cranial nerve V, the cervical plexus, and dorsal rami. The supratrochlear nerve parallels the artery as a neurovascular bundle and innervates the medial forehead. The supraorbital nerve passes through the supraorbital foramen/notch and innervates the lateral forehead. These two nerves course superiorly across the frontal bone and posteriorly along the top of the scalp to innervate the crown.[1] The auriculotemporal nerve originates from the mandibular division of the fifth cranial nerve. It courses posterior to the ramus of the mandible and follows the course of the superficial temporal artery. It supplies sensation for most of the temporal and parietal scalp as well as the anterolateral auricle and anterior half of the external auditory canal.[6] The lesser occipital nerve originates from the cervical plexus and follows the posterior edge of the sternocleidomastoid muscle to supply the skin over the mastoid and lateral occipital region of the scalp. The greater occipital nerve is the dorsal ramus of the second cervical spinal nerve. It penetrates the trapezius muscle to provide sensation to the occipital and parietal regions of the scalp extending anteriorly as far as the vertex (Fig. 1-11).[1]

Deep to the subcutaneous connective tissue of the scalp lies the galea aponeurotica which, along with the occipital and frontalis muscles, forms the most important fascio-muscular unit of the scalp. The small muscular belly of the occipital muscle originates from the posterior mastoid process and the posterior nuchal line of the occipital bone. It inserts into the galea aponeurotica and serves to pull the entire scalp posteriorly. The frontalis muscle originates from the galea aponeurosis and inserts into the skin of the forehead and eyebrows, contiguous with the superficial muscular aponeurotic system (SMAS) consists of fibrous and muscle tissues ensheathing and connecting facial muscles which allow coordinated facial expressions. The occipitalis muscle is innervated by the posterior auricular branch of the facial nerve whereas the frontalis receives its innervation from the temporal branch of the facial nerve. If the temporal branch of the facial nerve is damaged, this may result in drooping of the eyebrow or ptosis of the upper eyelid. If paralysis of the temporalis muscle occurs, the surgeon may try to correct the damage by doing a brow lift, a coronal lift, a blepharoplasty, or a combination of these procedures. The galea is a thick fibrous layer and as

such serves as a barrier against tumor spread.[6] It may also impede tissue movement when considering reconstructive approaches. Galeal resection or relaxing incisions may be necessary to allow adequate motion of flaps in order to optimize reconstruction of scalp defects.

A layer of relatively avascular loose areolar connective tissue lies below the occipitofrontalis muscle and the fibrous galea aponeurotica. The loose and avascular nature of this tissue creates an ideal plane for undermining the overlying skin, connective tissue, and galea, allowing for increased tissue mobility. This area is also sometimes referred to as the "danger zone," due to its potential for accumulation of large volumes of blood or other fluids and the possibility of infection spreading intracranially via emissary veins.[1]

The lymphatic drainage of the scalp closely follows the vascular supply. The occipital scalp drains into scattered occipital nodes along the superior nuchal lines. The postauricular nodes located around the mastoid process receive drainage from the occipital, parietal, and temporal scalp. The forehead and temporal regions drain into the preauricular or superficial parotid nodes.[4,5]

EAR

In general, the ear may be divided into three portions: the inner ear, the middle ear, and the external ear. The external ear consists of the auricle and external auditory meatus, which terminates at the tympanic membrane. The auricle is composed of irregularly shaped elastic cartilage and adipose tissue suspended below in the form of the lobule. This is cartilaginous support is lined with perichondrium and draped with skin. The skin of the anterior auricular surface is tightly adherent to the underlying structure as compared to the posterior surface which is rather loose. There are several anatomic landmarks which prove useful when describing the location of lesions on the ear, including the helix, scaphoid fossa, antihelix, triangular fossa, crus of the helix, crura of the antihelix, concha (conchal bowl), tragus, antitragus, incisure (intertragal notch), and lobule (Fig. 1-12). The entire auricle is fixed to the cranium by auricular ligaments.[1]

The external auditory meatus consists of cartilage and bone lined with skin, which terminates at the tympanic membrane. The anterior and inferior lateral third of the meatus is composed of cartilage, whereas the posterior and superior portions of the lateral third and the entire medial two-thirds are composed of temporal bone. The cartilaginous portion of the lateral third of the meatus is frequently perforated by fissures called the fissures of Santorini. It is important to note that the superior pole of the parotid gland lies immediately anterior and inferior to these fissures, which may, therefore, represent a potential direct route for tumor invasion of the parotid from the external meatus.[4]

The musculature of the ear consists of the anterior, superior, and posterior auricular muscles. They arise from the scalp and insert into the skin and auricle in areas named according to their location. They are innervated by posterior and temporal branches of the facial nerve, but are of minimal clinical significance if their function is lost.[2,5]

The vascular supply of the ear originates from the external carotid artery. The lateral surface receives its supply from the auricular branches of the superficial temporal artery, whereas the medial surface receives its supply from the posterior auricular and occipital arteries. Its venous drainage parallels the arterial supply and its lymphatic drainage flows to the preauricular, postauricular, and superficial cervical nodes.[1] The sensory innervation of the ear includes the auriculotemporal nerve, which originates from the mandibular branch of cranial nerve V. It innervates the anterosuperior quadrant of the anteriomedial half of the auricle, the anterior half of the external auditory meatus, the tympanic membrane, a small portion of the superior auricle on the posterosuperior surface, and most of the temporal scalp.[2,5,6] Cranial nerves VII, IX, and X supply the concha, the posterior half of the external auditory meatus, and a small patch of skin on the posterior auricle. The remainder of the auricle is innervated by the greater auricular nerve and the lesser occipital nerve, which originate from the cervical plexus. The greater auricular nerve appears at the posterior edge of the junction of the middle and upper thirds of the sternocleidomastoid muscle. It ascends across the sternocleidomastoid directly toward the inferior auricle to supply the anterolateral surface of the auricle, as well as most of its posteromedial surface. The lesser occipital nerve parallels the posterior edge of the sternocleidomastoid to supply a small portion of the posteromedial auricle and the skin overlying the mastoid.[1]

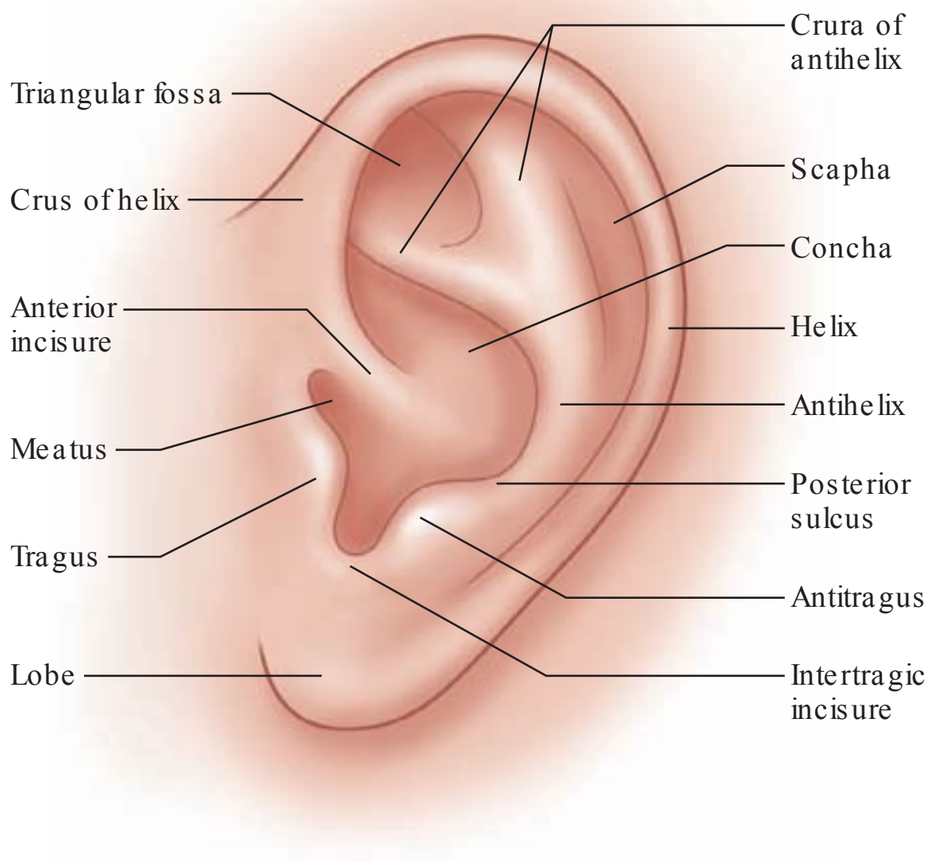

Figure 1-12 Cutaneous anatomy of the ear.

CHEEK

The cheek is a loosely defined area which extends anteriorly from the tragus of the ear to the oral commissure, and from the inferior orbital margin and the zygomatic arch inferiorly to the mandible. The nasolabial fold extends inferolaterally from the alar groove and separates the cheek from the perioral region, extending downward to become the melolabial fold. The cheek can be divided into four subsections: malar, infraorbital, masseteric, and

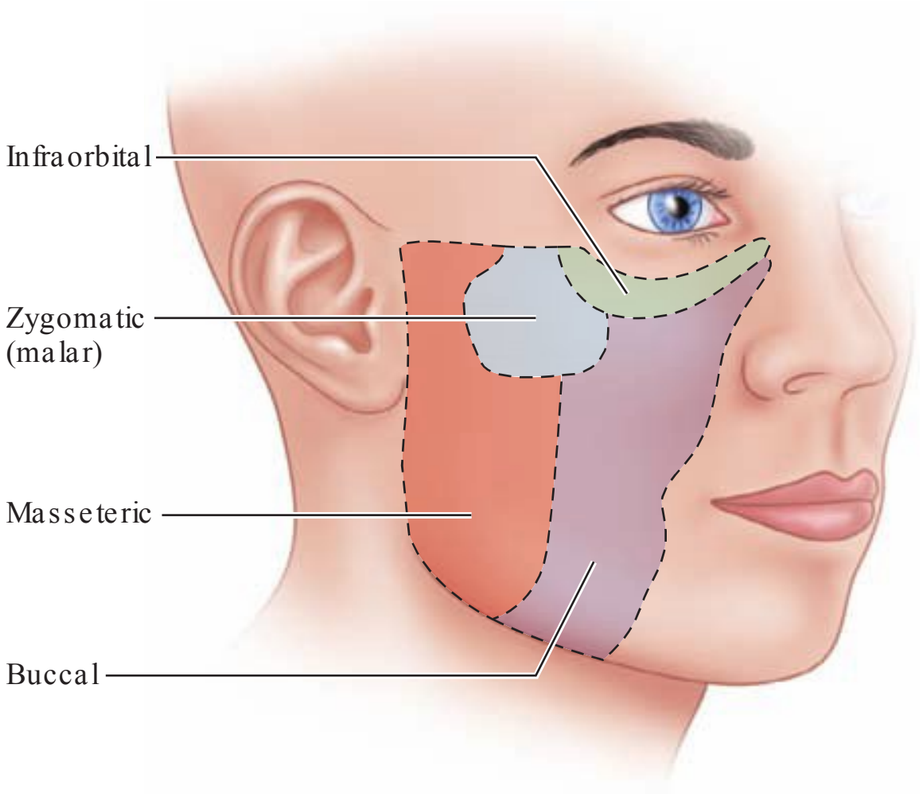

Figure 1-13 Divisions of the cheek.

buccal (Fig. 1-13). The malar portion of the cheek overlies the malar prominence of the zygomatic bone. The infraorbital cheek consists of the small area just below the inferior orbit. The tissue overlying the masseter muscle constitutes the masseteric cheek. The buccal cheek is the largest area and extends from the anterior edge of the masseter muscle to the oral commissure and from the infraorbital area to the mandible.[3]

The underlying bony structure of the cheek includes portions of the maxilla, the zygoma, and the mandible. The temporal process of the zygoma extends posteriorly to join the zygomatic process of the temporal bone, forming the zygomatic arch, and marking the superior aspect of the cheek.[2] While the malar prominence and zygomatic arch of the zygomatic bone contribute to the overall shape of the cheek, the maxilla is the predominant bony support of the cheek and lower face. Deep to the skin of the cheek lies the subcutaneous tissue. The fat of the cheek is thickest in an area of the buccal cheek referred to as the buccal fat pad. It is positioned between the buccinator muscle and the masseter muscle. It continues deep into the face between these two muscles and then superiorly into the temporal and infratemporal fossae. This space may serve as a possible route for deep tumor extension, possibly even as far as the maxilla or maxillary sinus, from the mid cheek.[1]

While not palpable, the parotid gland is an important structure which lies directly over the masseter muscle. The posterior edge of the parotid gland begins at the posterior edge of the mandibular ramus. Its superior pole extends nearly to the zygomatic arch and its inferior margin extends to the angle of the mandible. The anterior margin of the gland extends a variable distance over the masseter. The parotid gland is encapsulated in a thin layer of fascia. The parotid duct, Stensen's duct, is palpable halfway between the zygomatic arch and the oral commissure along the anterior edge of the masseter as the teeth are clenched.[5] This duct pierces the buccinator muscle and empties onto the mucosal cheek opposite the second upper molar. Should the duct be damaged during surgery, immediate repair is indicated.[4–6] In addition to its salivary function, this gland serves to protect the facial nerve as

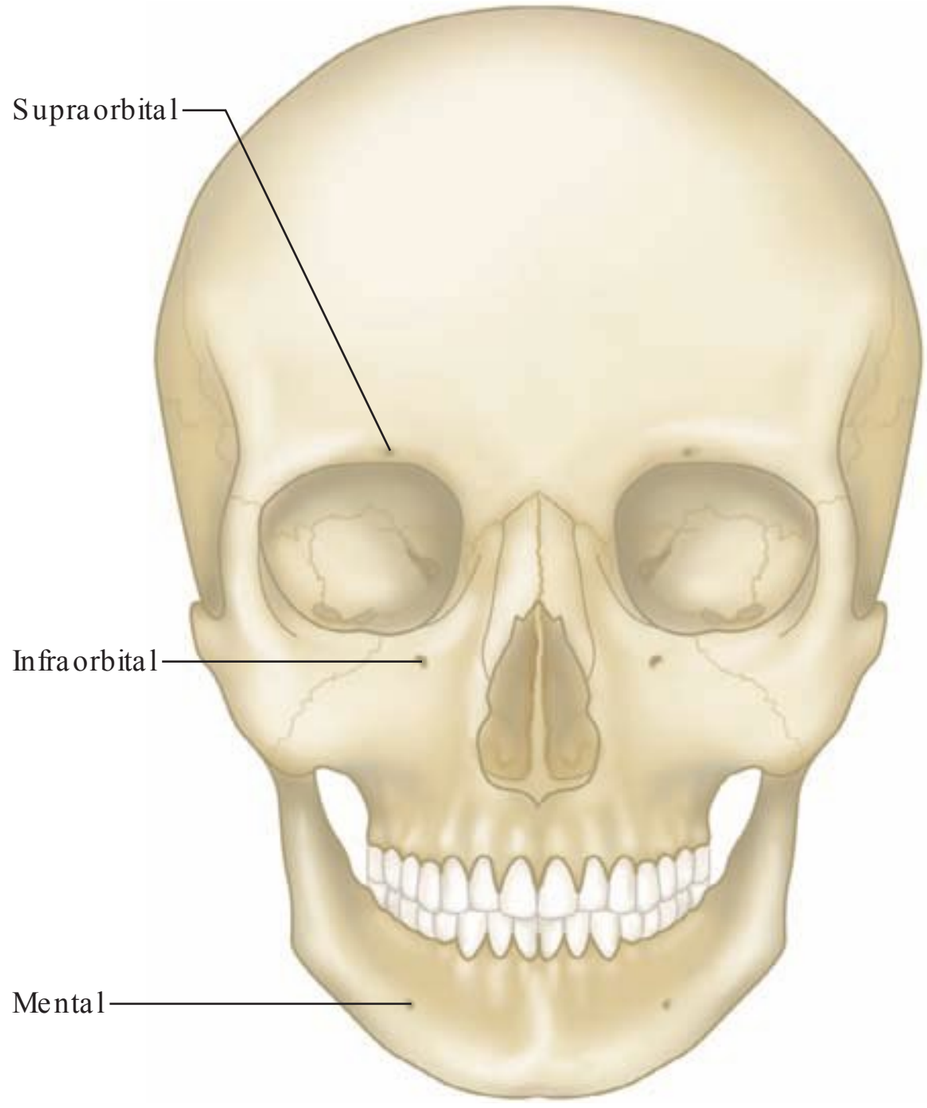

Figure 1-14 Foramina of the face.

it courses through the gland and divides into its various branches.

The sensory innervation for the cheek is derived from multiple nerves, including the infraorbital and zygomaticofacial branches from the maxillary division of cranial nerve V and the auriculotemporal and buccal nerves from the mandibular branch of cranial nerve V. The maxilla contains the infraorbital foramen, through which travels the infraorbital nerve, which supplies the infraorbital, buccal, and portions of the malar cheek, as well as the lower eyelid, lateral nose, and upper lip. If a vertical line is drawn on the face passing through the supraorbital foramen and the pupil, the infraorbital foramen would be found approximately 0.5 to 1.5 cm from the inferior margin of the orbit along that line.[1] The mental foramen is also located approximately 1 cm above the inferior margin of the mandible along this same line (Fig. 1-14). The zygomaticofacial nerve penetrates the zygoma through small foramina in the malar prominence to supply the overlying skin of the malar cheek. As mentioned previously, the auriculotemporal nerve courses superiorly just anterior to the auricle and supplies the masseteric cheek, a portion of the ear, and the temporal scalp. The buccal nerve leaves the infratemporal fossa and descends medial to the mandible into the cheek along the superficial surface of the buccinator muscle. It innervates the overlying skin and buccal mucosa. The greater auricular nerve, which originates from the cervical plexus and ascends across the surface of the sternocleidomastoid muscle, provides the sensory innervation for the lower portion of the masseteric cheek (Fig. 1-15).

The musculature of the cheek can be divided into superficial and deep components. The superficial musculature includes the zygomaticus major and minor, the orbicularis oculi, the risorius, the levator labii superioris, and the platysma. The deeper muscle group includes the

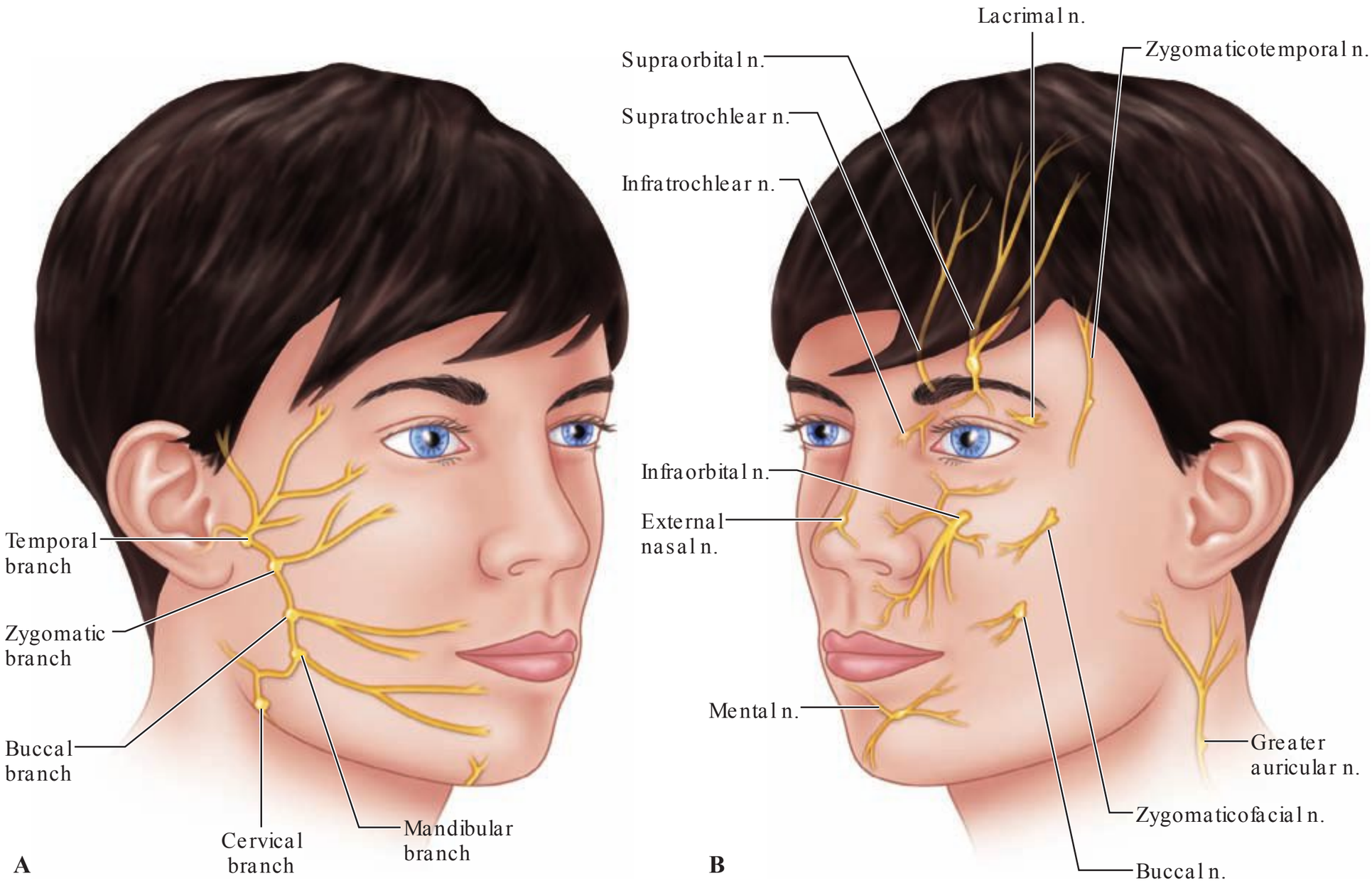

Figure 1-15 Sensory and motor nerves of the face (**A**) motor facial nerve and branches (**B**) sensory nerves.

levator anguli oris and the buccinator muscle. The zygomaticus major and minor originate from the zygoma and insert into the skin of the upper lip, serving to elevate the lip. The orbicularis oculi is a muscle which affects primarily the orbital and periorbital region, but some of its fibers extend onto the infraorbital cheek. Its circumferential course around the orbit serves to close the eye tightly. The risorius arises from the parotid fascia and inserts into the skin at the corner of the mouth, serving to pull the oral commissure laterally. The levator labii superioris originates from the maxilla just above the infraorbital foramen. It inserts into the skin of the upper lip and elevates the lip. The platysma originates from the superficial fascia of the upper thoracic region and the neck. It extends over the mandible and inserts into the skin of the oral commissures and pulls them inferiorly. The levator anguli oris arises from the maxilla below the infraorbital foramen and inserts into the corner of the mouth. It elevates the oral commissures. The buccinator muscle plays a key role in the musculature of the cheek. It originates from the pterygomandibular raphe, the posterolateral aspect of the maxilla, and the medial aspect of the mandible. It inserts into the skin and mucosa of the upper and lower lips, and this muscular wall keeps the cheek pressed against the dentition and assists in mastication.[3]

The muscles of the cheek all receive their motor innervation from the facial nerve with the exception of the platysma, which is innervated by the cervical plexus, and the masseter, which is innervated by the mandibular division of the fifth cranial nerve. The muscles of facial expression all receive their innervation from the deep surface of the muscle, except the buccinator which receives its motor innervation from its superficial surface.[6]

The facial nerve exits the stylomastoid foramen, courses inferiorly, gives of the posterior auricular nerve, and enters the posterior aspect of the parotid gland as a single nerve trunk which arborizes into five branches: temporal, zygomatic, buccal, marginal mandibular, and cervical (Figs. 1-15 and 1-16). These branches exit the parotid along its anterior edge, arborize in either one or two stages, and fan across the face. The temporal branch

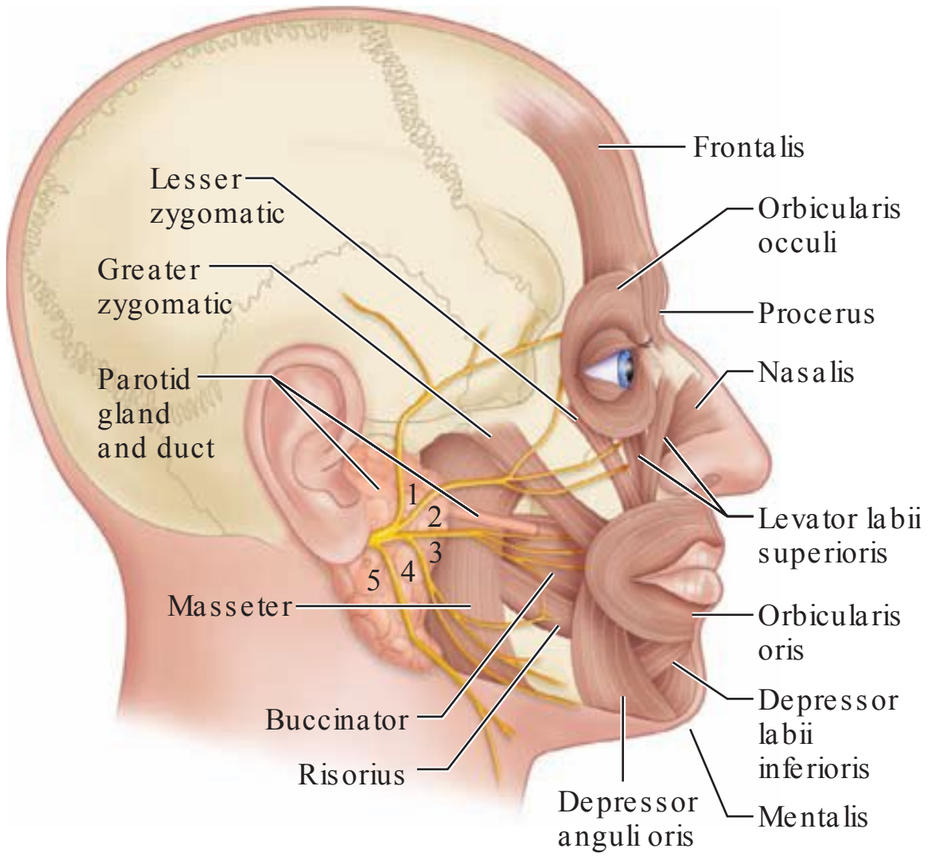

Figure 1-16 Branches of the facial nerve.

innervates the frontalis muscle. The marginal mandibular nerve innervates muscles in the perioral area and the cervical branch innervates the neck. The zygomatic and buccal branches of the facial nerve are, therefore, the only branches which provide innervation to the cheek area. The buccal branch exits the anterior edge of the parotid and parallels the course of the parotid duct. It provides innervation to the zygomaticus major and minor, levator labii superioris, risorius, levator anguli oris, and buccinator muscles. The zygomatic branch provides secondary innervation to the zygomaticus major and minor muscles. While the temporal branch of the facial nerve does not innervate anything in the cheek, it is at risk in the superior malar cheek area.[1] As it exits the superior pole of the parotid, it travels a short distance over the zygomatic arch before it enters the temporal region of the scalp.[2,4] For this short time, it is only protected by skin and a variable amount of adipose tissue and is, therefore, subject to injury. Loss of function of the temporal branch of the facial nerve results in ptosis of the brow and eyelid as well as inability to wrinkle the forehead. The marginal mandibular nerve courses along the inferior edge of the mandible through the parotid in the masseteric cheek. Once it exits, it is also at risk for injury as it too is only covered by skin and a variable amount of subcutaneous tissue. The marginal mandibular branch also remains as a trunk and fails to arborize until it nearly reaches its intended target. This means that surgical injury to this nerve may result in a more complete motor deficit.[1] It is also important to note that in the elderly, the marginal mandibular nerve may descend up to 2 cm below the margin of the mandible. Due to the risk of injury to branches of the facial nerve as they exit the parotid, a schematic "danger zone" has been devised (Fig. 1-17). To create this zone, a line is drawn parallel to and 1 cm above the zygomatic arch from the tragus to Whitnall's tubercle of the lateral margin of the orbit. A vertical line is drawn from the lateral margin of the orbit to the inferior margin of the mandible at the insertion of the masseter muscle. Finally, a line curving 2 cm below the inferior margin of the mandible, coursing posteriorly to the angle of the mandible, is drawn. Within the area delineated by these lines, the branches of the facial nerve are at risk for injury as they have not arborized extensively.[2,4–6] It may, therefore, be wise to avoid vertical incisions overlying the inferior masseter and mandible as well as deep incisions parallel to the zygomatic arch, so as to avoid injury to the marginal mandibular and temporal branches of the facial nerve. The danger zone is an area in which the surgeon may prefer to place incisions parallel to the course of the nerve regardless of the relaxed skin tension lines to minimize the chance of nerve injury. It may also be useful to use larger volumes of local anesthesia or saline for "hydrodissection" to lift the superficial tissues away from these deeper structures.

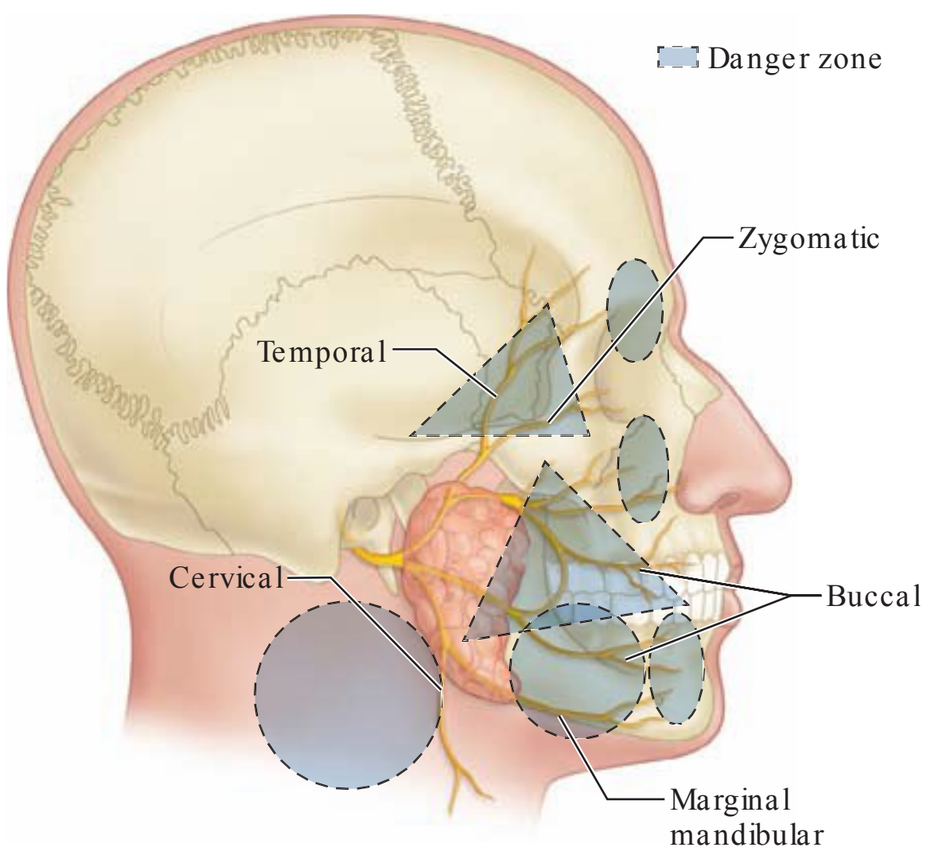

Figure 1-17 Danger zones of the face.

The vascular supply of the cheek originates from the external carotid artery which gives of the facial artery in the neck and crosses the mandible at the anterior edge of the masseter muscle. It courses across the face directly superficial to the buccinator muscle and deep to the platysma, risorius, zygomaticus major and minor, and the levator labii superioris muscles. As it approaches the oral commissure, it gives of the inferior and then the superior labial arteries before continuing along the nasofacial sulcus as the angular artery. The maxillary artery also contributes to the vascular supply of the cheek. It is another branch of the external carotid artery which ultimately branches into the infraorbital and buccal arteries. The infraorbital artery passes through the infraorbital foramen to supply the overlying area of the cheek. The buccal artery originates medial to the mandible, descends inferiorly onto the superficial aspect of the buccinator, and supplies the skin and buccal mucosa. The superficial temporal artery yields the transverse facial artery which passes through the superior pole of the parotid gland and parallels the parotid duct to supply the malar region of the cheek (Fig. 1-18). The venous circulation follows the same pattern as the arterial circulation, including the buccal, infraorbital, superficial temporal, and anterior facial veins. The anterior facial vein is the most important collecting vein for venous drainage of the face. Of note, due to the valveless nature of veins in the head and neck as well as the numerous anastomotic connections between them, it is possible for veins of the face to communicate with the cavernous sinus, most notably via the buccal or infraorbital veins.[1]

Lymphatic drainage from the cheek flows predominantly to the preauricular/parotid nodes. This nodal basin drains the masseteric cheek, the posterior half of the buccal cheek, and the lateral half of the infraorbital cheek. The facial nodes which follow the course of the facial vein drain the remainder of the anterior cheek. From there, the lymph flows into the submandibular and submental nodes (Fig. 1-19).

NECK

The neck can be divided into two triangles: anterior and posterior. The anterior triangle borders are defined by the anterior border of the sternocleidomastoid muscle, the inferior margin of the mandible, and the midline border from the tip of the chin to the jugular notch. The posterior triangle borders are composed of the sternocleidomastoid

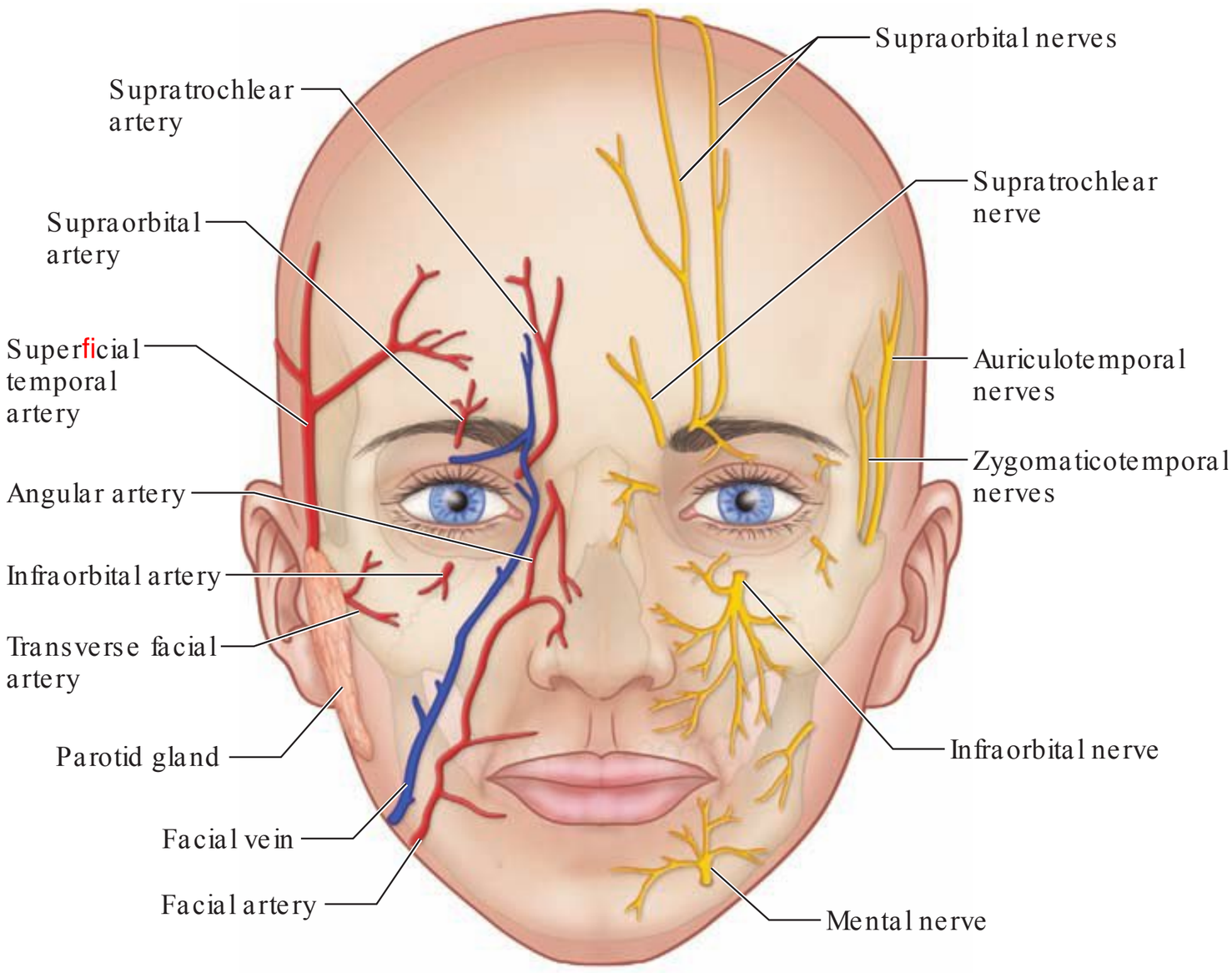

Figure 1-18 Vasculature and sensory innervation of the cheek.

muscle, the clavicle inferiorly, and the anterior edge of the trapezius muscle (Fig. 1-20).

Each triangle can be divided into smaller triangles for more precise localization of skin lesions and better understanding of neck anatomy. The anterior triangle can be subdivided into submental, submandibular, carotid, and muscular triangles. The borders of the submental triangle are the anterior belly of the digastric muscle, the hyoid bone body, and the midline of the neck. The borders of the submandibular triangle are the two bellies of the digastric muscle and the mandible. The borders of the carotid triangle are the sternocleidomastoid muscle, the posterior border of digastric muscle, and the superior belly of the omohyoid muscle. The borders of the muscular triangle are the sternocleidomastoid muscle, the superior belly of the omohyoid muscle, and the midline of the neck.[3]

The posterior triangle of the neck can also be divided into two smaller triangles: the subclavian and the occipital. The subclavian triangle's borders are composed of the clavicle, the sternocleidomastoid muscle and the inferior belly of the omohyoid muscle. The occipital triangle's borders are demarcated by the inferior belly of the omohyoid muscle, the sternocleidomastoid muscle, and the trapezius muscle.[4]

The skin of the neck exhibits various unique properties. First of, it is very delicate and thin. It also contains numerous hair follicles, sweat glands, and some sebaceous glands. The superficial cervical fascia, a thin layer of loose areolar connective tissue, allows for the skin of the neck to be pliable and mobile. Of note, the fascia is continuous with the superficial fascia of the face. The platysma, located within the anterior triangle, is the only superficial muscle in this area. It originates from the fascia of the upper chest and continues superiorly over the mandible to insert into the skin and superficial tissue of the lower face. The platysma muscle tenses the skin of the neck and depresses the mouth. The cervical branch of the facial nerve provides innervation to the platysma. Although the platysma is superficial to the sternocleidomastoid and reaches the most inferior aspect of the occipital triangle, it does not provide coverage or protection to the spinal accessory nerve in the posterior triangle. The sternocleidomastoid muscle arises from the sternum and the clavicle and passes obliquely across the neck to end at the mastoid process.[3]

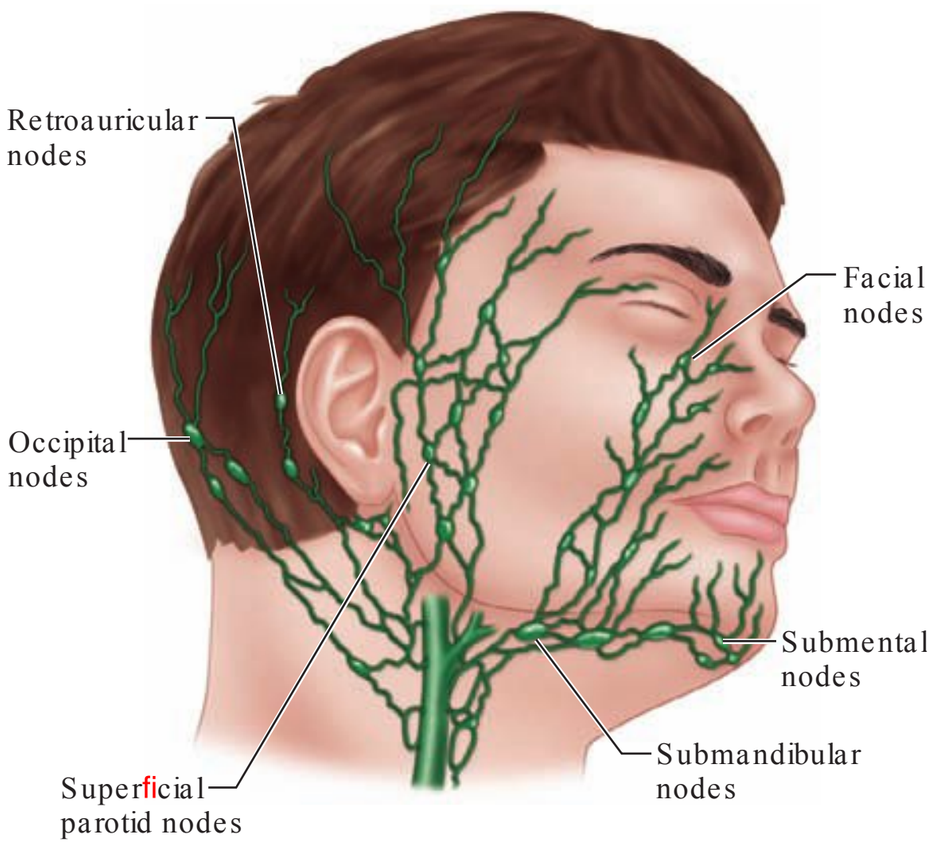

Figure 1-19 Lymphatics of the head and neck.

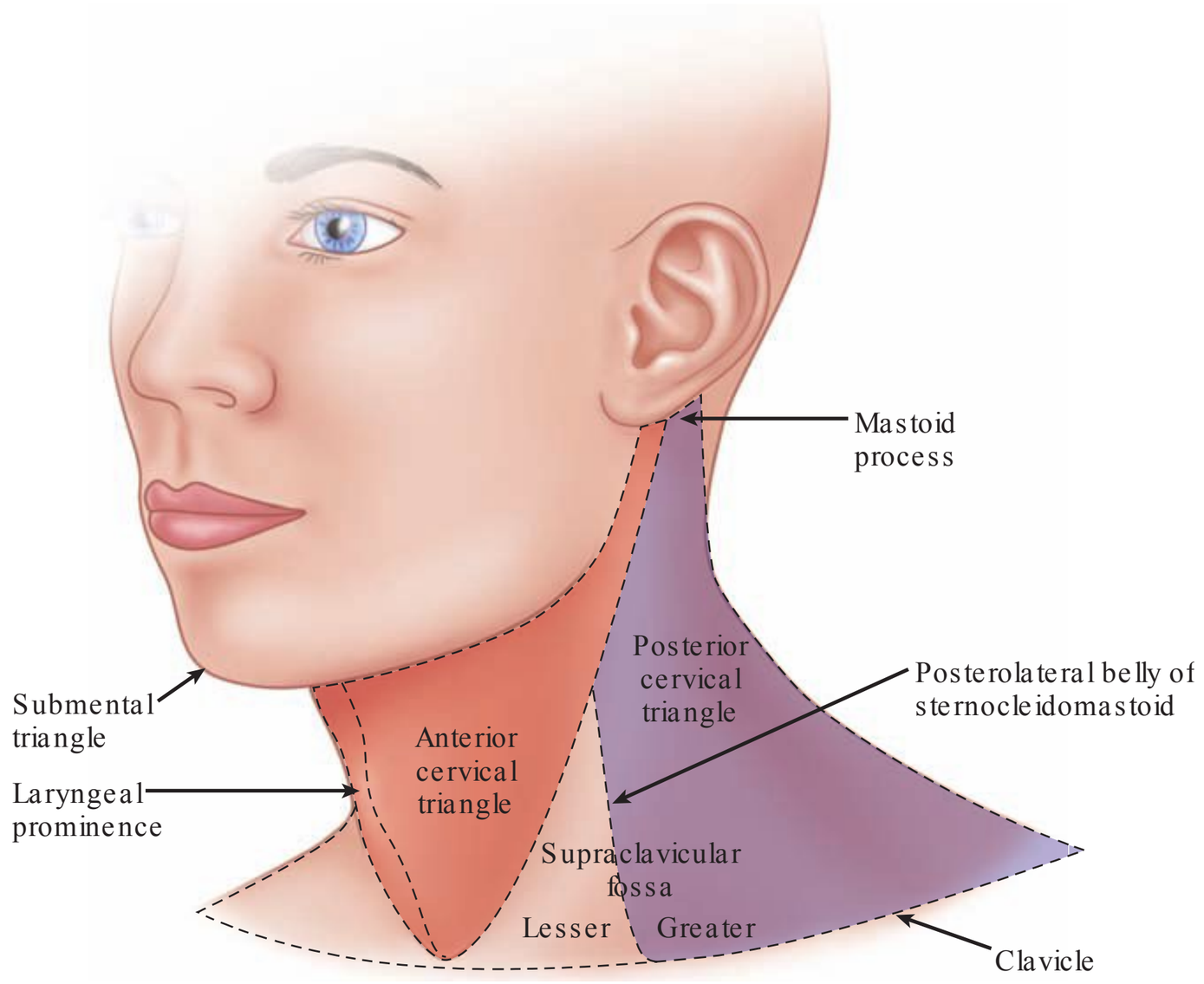

Figure 1-20 Anatomy of the neck. The neck can be further divided into the anterior and posterior cervical triangles.

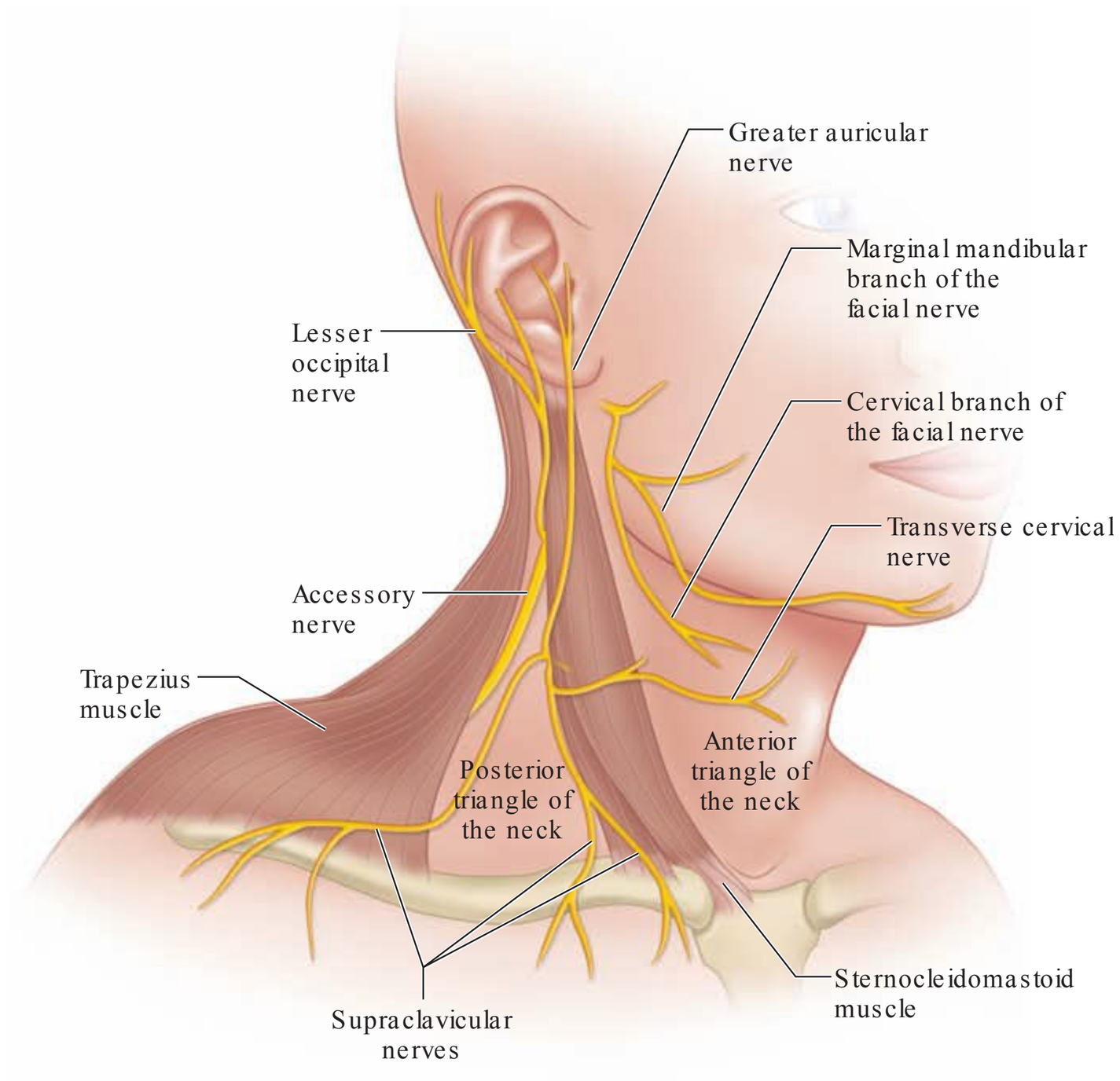

Figure 1-21 Sensory and motor supply of the neck and associated regions.

The hyoid bone is located in the anterior triangle of the neck at the second cervical vertebral level. It can be palpated just inferior to the chin within the midline of the neck. The prominence of the thyroid cartilage is also palpable just below the hyoid bone. The anterior lamina of the cricoid cartilage, along with tracheal rings and the isthmus of the thyroid gland, is located below the thyroid cartilage.[1]

The branches of the external carotid artery and the thyrocervical trunk, a branch of the subclavian artery, provide the arterial supply to the neck skin. The venous drainage of the neck occurs through the anterior superficial venous system deep to the platysma muscle. It then drains into the anterior jugular vein, which may be connected to the common facial vein via a small communicating vein. The superficial veins of the posterior cervical triangle drain into the external jugular vein.[6]

The cervical plexus provides sensory innervation to the skin of the neck. The transverse cervical nerves cross the sternocleidomastoid muscle at the midpoint horizontally to provide sensory innervation to the mid-anterior cervical triangle. A series of supraclavicular nerves pass inferior to the transverse cervical nerves to supply the skin of the anterior and posterior cervical triangles as well as the upper pectoral area. The lesser occipital nerve is found at the posterior border of the sternocleidomastoid muscle near its midpoint. It travels superiorly, paralleling the posterior border of the sternocleidomastoid muscle, to innervate the posterior ear and mastoid area. Similarly, the greater auricular nerve arises from the midpoint of the sternocleidomastoid muscle with a superior trajectory to provide sensory innervation to the lower portion of the ear.

Understanding the location of the nerves in the neck is key for every surgeon so as to avoid complications. The marginal mandibular branch of the facial nerve descends into the anterior triangle of the neck along the mandible before innervating the muscles of the lower lip. The marginal mandibular nerve is at risk during surgery in this area. The spinal accessory nerve, which supplies the sternocleidomastoid and trapezius muscles, is at risk in the posterior triangle during surgery. This is especially true as it exits from the junction of the upper and middle thirds of the sternocleidomastoid muscle to enter the posterior triangle, as it is covered only by skin and superficial fascia at this location (Fig. 1-21). Damage to the spinal accessory nerve can lead to a permanent loss of ability to elevate the shoulder on the ipsilateral side.[1]

CONCLUSION

The head and neck region contains many important anatomic structures that every dermatologic surgeon must be familiar with to provide the highest level of patient care. Preoperative anatomic knowledge and planning, along with intraoperative awareness and precision, are necessary to provide the best cosmetic result with the least possible functional disturbance. With a full understanding of this anatomically complex region, the physician will have the confidence to perform a wide range of superficial surgical procedures and the knowledge required to minimize the likelihood of complications and adverse outcomes.

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Chapter 2

Preoperative Evaluation

Bichchau M. Nguyen & Chrysalyne D. Schmults

Introduction

Preoperative evaluation of patients undergoing cutaneous surgery serves multiple purposes: (1) it provides patients and physicians with the opportunity to discuss pertinent risks, benefits, and alternatives for the planned procedure; (2) to identify significant patient, lesion, or surgical factors that could increase the risk of complications; and (3) to plan care so as to mitigate identified risk factors.

The extent of preoperative evaluation depends in part on the type of procedure, the duration and degree of invasiveness of the procedure, and the use of topical, local, or general anesthesia. The vast majority of dermatologic surgeries are conducted under local anesthesia. A brief questionnaire addressing major risk factors for bleeding, infection, allergies, and implantable cardiac devices that may limit the use of electrocautery devices, will typically suffice for superficial procedures such as biopsies and curettage. Surgical excisions and Mohs micrographic surgery require a more thorough investigation of the patient's past medical history and lesion(s) of interest to identify and appropriately mitigate possible complications. Preoperative evaluation for elective cosmetic procedures such as laser therapies, liposuction, hair transplant, sclerotherapy, injections of fillers, and botulinum toxins varies significantly depending on the procedure and will, therefore, be covered in their corresponding specific chapters.

General Overview

A thorough preoperative evaluation provides a global overview of patient factors that can complicate the planned surgical procedure: past medical and surgical history, medications, allergies, and social history and habits (Table 2-1). Diabetes, cardiovascular heart disease, immunosuppression, blood-borne infections, bleeding disorders, and neuropsychiatric conditions are medical conditions that may influence operative planning. All prior surgeries, surgical complications and outcomes, and the presence of prosthetic joints or implantable cardiac devices should be specifically elicited from the patient.

Obtaining an accurate medication record is crucial but challenging, especially in the elderly population, whose medication lists can be extensive. It can be helpful to confirm the medication list on record with the patient and his or her caregiver, given the varying degrees of compliance and to inquire specifically about over-the-counter medications and herbal supplements as these are often underreported. Information on allergies should contain agent name and type of reaction. Patients may sometime confuse an adverse effect or vasovagal response with a true allergy. Allergic reactions can be anaphylactic (type I) or delayed hypersensitivity (type IV) reactions. Facial edema and respiratory distress are important type I reactions that should be asked about, especially with respect

TABLE 2-1
Preoperative assessment of Surgical Patients

Past medical history	■ Medical: cardiovascular disease, hypertension, diabetes, bleeding disorders, malignancy, immunosuppression, blood-borne infection (HIV, hepatitis C) ■ Neuropsychiatric: claustrophobia, anxiety, panic attacks, dementia
Past surgical history	■ Prosthetic joints, coronary artery stents, implantable cardiac devices ■ Prior surgical complications and outcomes
Medications	■ Prescribed medications: antiplatelet, anticoagulant, antibiotics, analgesics ■ Over-the-counter medications (herbal supplements and vitamins)
Allergies	■ Medications: agent and type of reaction (adverse effects vs. true allergy; delayed hypersensitivity vs. anaphylactic response) ■ Topical preparations: cleansing antiseptics, dressings, adhesives
Social history	■ Alcohol consumption: servings per week ■ Smoking: pack-years ■ Illegal drug use ■ Social situation: caregiver, transportation, type of job, access to medical care

to antibiotic and latex allergies. Aside from medications, patients can also develop allergies (mostly allergic contact dermatitis) to antiseptic agents, topical antibiotics, and surgical dressings.

Social habits such as smoking, consumption of alcohol, and use of illicit drugs should be noted, although data on the true impact of these factors on surgical risks are mixed. Finally, dif cult social situations including lack of social support, caregivers, or stable housing can make postoperative wound care challenging. Long-distance travel, lack of transportation, and limited access to local medical care may delay detection and treatment of postoperative complications. T ese factors should be considered in choosing appropriate surgical repairs and follow-up care.

A focused physical examination should be performed to assess the overall health status of the patient and characteristics of lesion(s) being treated. T e correct procedure, diagnosis, and site should be confirmed with patients (via a mirror if needed) and/or caregivers and verified with available medical records, ideally including photographs. Use of prebiopsy photographs to identify the correct lesion is highly recommended because studies have shown that both patients and physicians are prone to identifying incorrect sites in a significant percentage of surgical cases—16% and 6%, respectively, in a prospective cohort study involving patients undergoing Mohs micrographic surgery.[1] When a lesion cannot be definitively identified using the available resources, frozen biopsy of the suspected site can be performed with continuation to surgery if biopsy is positive for malignancy, or 3 month clinical follow-up if the biopsy is negative.[2]

Once the correct lesion has been identified, the physician can proceed to assess the extent of spread, evidence of local or metastatic spread, proximity to functionally or aesthetically critical structures, feasibility of obtaining adequate surgical margins, and any potential neurologic or vascular complications. Depending on these factors, a nodal examination, imaging studies or consultation with other specialists might be warranted. T e patient should be made aware of potential risks that may pertain to their procedure (such as infection, bleeding, hematoma, seroma, numbness, paralysis, asymmetry, and scarring) prior to surgery. A comprehensive discussion of these risks as well as the benefits of the proposed treatment and possible alternatives is needed in order for the patient to make a fully informed choice to proceed. Expectations regarding the anticipated extent of surgery and the length of recovery should be addressed preoperatively as well.

Medical cOMOrbidities

Patients with certain medical comorbidities deserve additional preoperative consideration due to their suspected or proven impact on the surgical risks of infection, bleeding, or wound healing. T ese special patient populations are discussed in more detail subsequently.

diabeteS

As of 2012, 9.3% of all children and adults in the United States were shown to have diabetes, with 25.9% of patients aged 65 years or older affected.[3] Diabetic patients have an increased risk of infection after general and cardiac surgeries. However, conflicting data exist regarding the risk of postoperative complications in diabetics undergoing dermatologic surgery. In the absence of prophylactic antibiotics, the risk of perioperative infection in these patients ranges from 1.1%[4] to 10%[5] Some studies have found an increased risk of infections in patients with diabetes compared to those without,[6] whereas others have noted no difference between the two groups.[5] Patients with diabetes do not appear to have an increased risk of dehiscence, bleeding, or other noninfectious complications.[6] Little data exist on the effects of insulin dependence or perioperative glycemic control on the risk of dermatologic surgical complications. T e variable prevalence of diabetics in study cohorts, along with different definitions of infection, inconsistent use of cultures, and the observational nature of these studies contribute to the dif culty of translating the available data into management guidelines. One may, therefore, advise patients with diabetes that they may have an increased risk of surgical site infection, but, as addressed later in this chapter, standard use of prophylactic antibiotics in this patient group is not warranted.

HyPer t en SiOn

T e main concern in patients with uncontrolled hypertension is an increased risk of intra- and postoperative bleeding. Studies from the 1970s showed that patients with a blood pressure of 150/100 mm Hg had twice the risk of postoperative hematoma following rhytidectomy compared to normotensive counterparts.[7] More recent meta-analysis has shown that patients with blood pressures as high as 180/100 mm Hg do not have an increased risk of perioperative cardiac complications such as myocardial infarction, arrhythmia, and stroke when undergoing general anesthesia for planned surgeries.[8] Concerns exist that blood pressure lability during cutaneous surgery may be significant, raising the question of whether intraoperative monitoring is warranted for baseline hypertensive patients. However, studies have found little fluctuation in blood pressure before, during, or after cutaneous surgery in both normotensive and hypertensive patients.[9] Cutaneous surgery may be safely conducted in patients with a baseline blood pressure of 180/100 mm Hg or less. For patients with blood pressures >180/100 mm Hg, the procedure, if not urgent or emergent in nature, should be rescheduled so that further evaluation by a primary care physician or hypertension specialist may take place. A component of observed hypertension could be secondary to anxiety as well as the "white coat phenomenon" which can be effectively mitigated with use of low dose anxiolytics and nonmedical interventions such as music and maintaining a calm surgical environment. Patients should also be reminded to continue taking their prescribed antihypertensive medications on the day of surgery.

cardiOvaScular diSeaSe

T ree main considerations in patients with cardiovascular disease undergoing dermatologic surgery are anticoagulation,

implantable cardiac devices, and prophylactic antibiotics for endocarditis. These patients are often prescribed antiplatelet agents, for example, aspirin and clopidogrel, for primary and secondary prevention of coronary artery disease, or to prevent stenosis of coronary artery stents. Patients with atrial fibrillation or prosthetic valves may receive anticoagulation with warfarin to prevent clot formation, thromboembolic events, and valve failure. In general, all of these agents should be continued, since their discontinuation in patients undergoing dermatologic surgery has been associated with catastrophic thrombotic adverse events such as myocardial infarctions and stent restenosis (see Anticoagulants section under medications). Discontinuation of any prescribed anticoagulants should be discussed with and cleared by the patient's primary care physician or cardiologist. Although these agents may increase the risk of bleeding, the reported complications are often localized and not life threatening. Thus, the risks of stopping anticoagulants usually outweigh potential benefits. Rare exceptions may include patients undergoing major reconstructions such as free flaps. In such cases, decisions regarding perioperative anticoagulation can be made with input from the entire physician team. More detailed information on the specific agent and recommended management can be found in the "Anticoagulants" section. Appropriate endocarditis prophylaxis is discussed in the section on prophylactic antibiotics.

Stent S

Dual antiplatelet therapy with aspirin and typically clopidogrel is recommended for patients with recently placed bare metal or drug eluting stents as treatment for coronary arterial disease. Patients with bare metal stents should continue taking aspirin and clopidogrel for at least 1 month after stent placement, while those with drug eluting stents should be on these agents for at least 1 year after the procedure to prevent stent thrombosis.[10] Current published guidelines recommend continuation of dual antiplatelet therapy for all dermatologic procedures, including Mohs micrographic surgery with extensive reconstruction.[11–13] Elective procedures can be delayed until the patient has finished the recommended course of dual antiplatelet therapy. If medically necessary procedures in highly vascularized areas are required, one might consult the managing cardiologist to consider holding clopidogrel therapy for 1 week or ticlopidine for 2 weeks prior to the procedure.

iMPlantable cardiac deviceS

Implantable cardiac devices include pacemakers, defibrillators, and cardiac synchronization therapy devices. Indications for pacemakers include heart blocks, bradycardia, and atrial fibrillation. Defibrillators are indicated for primary prevention of arrhythmias in patients with heart failure, and secondary prevention for ventricular arrhythmias. Cardiac resynchronization therapy devices are designed to improve symptoms in patients with advanced heart failure through the use of ventricular synchronization. These devices contain sensing leads that connect to the patient's cardiac tissue and a pulse generator that discharges electrical signals to treat detected arrhythmias. Electromagnetic interference from electrosurgical devices can either impair the sensing capabilities of the leads or trigger inappropriate electrical discharges from the pulse generator.

The most commonly used electrosurgical device in dermatologic surgery is the hyfrecator, which generates low power, high frequency alternating current through an electrode that can either be monoterminal or biterminal. The hyfrecator itself can transmit current through the tissue through one or two prongs, that is, monopolar or bipolar, respectively. Less electrical current passes through the patient with a bipolar hyfrecator. As a result, recommendations for patients with implantable cardiac devices have included use of handheld bipolar electrocautery, short bursts of current, temporary deactivation of cardiac devices with magnets, intraoperative cardiac monitoring, and postoperative device function testing to reduce the risk of electromagnetic interference.

Recent experimental data, however, suggest that the hyfrecator is safe to use in patients with implantable cardiac devices. Weyer et al. tested the electromagnetic interference of two commonly used hyfrecators on six Medtronic devices (three pacemakers and three defibrillators) using an industry standard saline–collagen gel model that simulates human soft-tissue conduction. Tested settings include monoterminal and biterminal configurations, typical and maximum power (10 and 30 W, respectively), intermittent on/off and continuous on modes, with various distances between the hyfrecators and the cardiac devices. There was no electromagnetic interference at any of the tested settings for the defibrillators and no accidental discharge of electrical currents. Inhibition of ventricular pacing was noted at a distance of 1 cm, and of atrial pacing at 3 cm. The authors prospectively used hyfrecators for hemostasis in 40 patients with cardiac devices and noted no adverse events.[14]

Based on these data, hyfrecators may be safely used within 2 in of cardiac devices without the need for biterminal configuration, intraoperative cardiac monitoring or postoperative testing of device function. Routine dermatologic surgery utilizing only the hyfrecator for hemostasis and electrodesiccation is generally safe for all patients with pacemakers and defibrillators when the surgical site is more than 2 in away from the cardiac devices.

bleedinG diSOrderS

The three most common congenital coagulation disorders are von Willebrand disease, factor VIII deficiency (hemophilia A), and factor IX (hemophilia B).[15] A bleeding diathesis often presents as slow but persistent oozing from multiple operative sites rather than as localized rapid bleeding. Patients with von Willebrand disease have a 10% risk of clinically significant bleeding from a decreased quantity or quality of von Willebrand factor, which provides binding sites for platelets and factor VIII. Treatment with desmopressin, 1-desamino-8-arginine vasopressin (DDAVP), or select factor VIII concentrates is typically indicated for 1 day before and 3 to 5 days after minor surgery and up to 10 days after major surgery. The patient's

bleeding time should be corrected to 100% of normal for the perioperative period. For hemophilia A and B, factor VIII and factor IX concentrates, respectively, can be infused to raise factor levels to 100% of normal preoperatively and 30% to 50% postoperatively during the wound healing phase.[15] Preoperative evaluation should elicit a hemostasis history after prior surgical procedures since laboratory studies such as bleeding time, PTT, and PT cannot reliably predict intra- and postoperative bleeding. Consultation with hematology services is highly recommended for appropriate clinical and laboratory preoperative evaluation, as well as for management of transfusion products during the perioperative period. Meticulous hemostasis, minimizing undermining, and frequent postoperative follow-up are also helpful in reducing the risks of serious bleeding complications in this patient population.

bloOd-bOrne infeCtiOn—HePatitiS and Hiv

With continuing improvements in antiretroviral therapies and supportive care, patients with HIV now have a significantly longer life expectancy and are therefore more commonly encountered in the outpatient and surgical settings. Higher risks of postoperative surgical site infections in patients with HIV are often assumed but there is little available data to confirm this hypothesis. A multicenter prospective cohort study in Italy examining the incidence of surgical site infection in HIV patients undergoing a variety of inpatient surgical procedures revealed a 10% rate of infection overall and a 12.5% rate of infection following plastic/dermatologic surgical procedures. Antibiotic prophylaxis use varied widely among the surgical subspecialties, ranging from 50% of patients undergoing plastic/dermatologic surgical interventions, up to nearly 100% in patients undergoing cesarean sections or thoracic surgeries.[16] However, there were no data on infection rates in non-HIV patients or outpatient surgical procedures in this study, which precludes extrapolation of these data to outpatient dermatologic surgery. A meta-analysis of orthopedic surgery studies showed no statistically significant differences in the incidence of surgical site infection between HIV and non-HIV patients.[17] Given the paucity of data supporting an increased risk of infection in HIV patients, antibiotic prophylaxis for this patient population in the setting of routine outpatient dermatologic surgery is not currently recommended.

Aside from patient safety, one must also consider personnel safety when performing surgical procedures on patients with HIV, hepatitis B or hepatitis C infections. A survey of Mohs micrographic surgeons revealed an astonishingly high 65% rate of needlestick and other exposure injury within the past year, with 5% reporting exposure to patients with HIV or hepatitis. Strategies such as use of double gloves for surgeons, assistants, and laboratory technicians, formal training of laboratory personnel, and warning labels on surgical trays are associated with lower relative risk of exposure injuries. Warning labels on the patient's chart, though used by almost half of surveyed surgeons, did not impact the risk of injuries.[18] Universal precautions are recommended since depending on the clinical setting and patient population, the rate of undiagnosed HIV or hepatitis infection can be as high as 10%.[19]

MediCatiOnS

Polypharmacy, defined as using five or more prescription medications, is especially prevalent in the elderly population, with 29% of patients over the age of 65 taking 10 or more medications in a week.[20] Polypharmacy increases the risks of drug adverse events; one-third of these events relate to the use of insulin, digoxin, or warfarin.[21] Hemorrhagic complications account for nearly one in five adverse events. The remaining adverse effects result from electrolyte, metabolic, and gastrointestinal disturbances.[22] Efforts should be made to obtain an accurate and complete medication list from patients undergoing dermatologic surgery to ensure appropriate medication monitoring. Adjustments to certain medications to reduce the risks of bleeding, blood pressure lability, poor glycemic control, and altered mental status may be indicated. Specific questions regarding the use of anticoagulants and antiplatelet agents, corticosteroids, immunosuppressants, over the counter medications, and herbal supplements should be included in any preoperative questionnaire. Management of these classes of medications is detailed below.

Anticoagulants. Management of anticoagulants in the perioperative period has changed significantly in the past several years. In the past, withholding these agents before and after the procedure was common. Now, consensus guidelines recommend continuation of these agents in the vast majority of patients undergoing dermatologic surgeries, since the risks of catastrophic thrombotic events associated with their discontinuation far outweigh the increased risk of bleeding complications.

The list of medications that affect platelet function and the coagulation cascade continues to grow (Table 2-2). Aspirin and thienopyridines (e.g., clopidogrel, ticlopidine, and prasugrel) inhibit platelet function for the lifespan of the platelets. Heparin, low–molecular-weight heparin (e.g., enoxaparin and dalteparin), direct thrombin inhibitors (e.g., dabigatran, argatroban, and lepirudin), and warfarin all target specific components of the coagulation pathway.[23] Indications for these agents include primary and secondary prevention of cardiac and neurologic thrombotic events, pulmonary embolism, and deep vein thrombosis. The most commonly encountered agents in dermatologic surgery are aspirin, clopidogrel, and warfarin.[24]

Patients on anticoagulants are at increased risk of bleeding complications although the overall absolute risk for these events remains low and none of the reported complications have been life threatening. The risk of severe complications, defined as considerable intra- or postoperative bleeding, acute hematoma, flap or graft necrosis, and dehiscence, was 1.3% for patients taking aspirin, and 5.7% for those taking warfarin based on a meta-analysis of six published studies encompassing approximately 1400 patients.[25] Clopidogrel-containing regimens have a higher risk of severe complications (3%) than aspirin alone (0.5%).[26] A large prospective study on 1900 patients undergoing dermatologic surgeries

TABLE 2-2
anticoagulants encountered in Outpatient Setting

anticoagulant	Mechanism of action	duration of action	risk of Severe complication in cutaneous Surgery	Monitoring
Aspirin	Irreversible inhibition of cyclooxygenase	7–10 d	0.5%–1.3%[25,26]	None
Clopidogrel[a] Ticlopidine Prasugrel	Irreversible inhibition of adenosine diphosphate	7–10 d	3%[a26]	None
Warfarin	Vitamin K antagonist, inhibition of synthesis of factors II, VII, IX, and X	2–5 d	5.7%[25]	PT, INR
LMWH—enoxaparin and dalteparin	Selective inhibition of factor Xa	24 h	N/A	aPTT
Dabigatran, argatroban[a], lepirudin[a]	Selective reversible direct thrombin inhibitor	24 h	N/A	aPTT

[a]Cook-Norris RH, JAAD, 2011—Table 3.

showed that clopidogrel increased bleeding risk by 4.7 fold, warfarin by 10 fold, and both agents by 40 fold.[24] A significantly increased risk of bleeding in patients on two or more anticoagulants compared to those on one or no anticoagulant was confirmed by Shimizu et al.[27] in a large retrospective study involving 1000 patients. No difference in infection rates attributable to use of anticoagulants was noted.

Several new anticoagulants and antiplatelet agents have entered the market in the last 5 years. Three new oral anticoagulants, designed to replace warfarin, have been approved for stroke and systemic embolism prevention in patients with atrial fibrillation: dabigatran, rivaroxaban, and apixaban. No data exist on the effects of these agents on bleeding risk during or after cutaneous surgery. However, Healey et al. compared the periprocedural bleeding rates in 4591 patients on dabigatran (110 mg or 150 mg) or warfarin undergoing at least one invasive procedure, including pacemaker/defibrillator insertion, dental procedures, diagnostic procedures, cataract removal, colonoscopy, and joint replacement. They found no statistically significant differences in bleeding rates among those taking dabigatran 110 mg po daily, 150 mg po daily or warfarin (3.8%, 5.1%, and 4.6%, respectively).[28] Similarly, rivaroxaban appears to have a statistically equivalent risk of major bleeding to warfarin, 3.6% versus 3.4%, in a multicenter randomized controlled trial (RCT) with over 14,000 patients.[29] Apixaban, the latest approved oral anticoagulant, has a slightly lower reported risk of major bleeding compared to warfarin, 2.1% versus 3.09% in an RCT study of over 18,000 patients[30] and a risk of major and clinically relevant nonmajor bleeding events comparable to that of aspirin (4.5% vs. 3.8%) in a randomized study of stroke prevention with 5599 patients.[31] On the basis of these data, management of these three new oral anticoagulants should mirror that of warfarin for cutaneous surgeries, namely, continuation of the agents if they are prescribed as monotherapy, unless patient comorbidities and extent of planned surgery warrant discussion with the prescribing physician regarding consideration of a brief interruption in therapy.

The management of new antiplatelet agents, prasugrel and ticagrelor, is similarly based on comparative data against clopidogrel. Prasugrel functions in a manner similar to that of clopidogrel but has higher potency. In animal models, prasugrel inhibition of platelet aggregation was 10 and 100 times more potent than that of clopidogrel and ticlopidine, respectively.[32] Clinically, this agent has a higher documented risk of fatal bleeding and the need for transfusion in a study of 13,608 patients undergoing percutaneous cardiac intervention for acute coronary syndrome.[33] On the other hand, ticagrelor, a reversible antiplatelet aggregation agent, has demonstrated a risk of severe bleeding equivalent to that of clopidogrel, 2.9% versus 3.2%, in an RCT study of 13,408 patients.[34] Consultation with cardiology regarding the perioperative management of these two agents is prudent, particularly in the case of patients taking prasugrel who may have a higher bleeding risk than those taking clopidogrel.

Heparin and low–molecular-weight heparin preparations, such as enoxaparin and dalteparin, are associated with higher risks of bleeding in patients undergoing cardiac and gynecological surgeries. However, because of their intramuscular and intravenous administration, these agents are relatively rarely seen in the outpatient setting where dermatologic surgeries are conducted.

Although anticoagulants increase the risks of bleeding and severe complications, discontinuation of these agents can result in catastrophic life-threatening thromboembolic events. There have been case reports of stroke, TIA, pulmonary embolism, DVT, MI, clotted prosthetic aortic valve, and retinal artery occlusion occurring shortly after discontinuation of anticoagulants for dermatologic surgery.[35–37] These events are thought to be secondary to a rebound hypercoagulable state due to more rapid depletion of antithrombotic protein C and S relative to clotting factors during the first few days of anticoagulant reinitiation.[38] Given that the risk of thromboembolic events outweighs the risk of bleeding from dermatologic surgery, prescribed monotherapy aspirin, clopidogrel, and warfarin should be continued in the perioperative period. For

patients taking both aspirin and clopidogrel, or aspirin and warfarin, or clopidogrel and warfarin, elective procedures should be delayed until one or both of the agents are no longer medically indicated. For medically necessary procedures, the surgeon should discuss the situation with the prescribing physician to determine whether one of the agents can be safely discontinued. For monitoring of warfarin, older studies suggested that checking INR within 24 hours of surgery and use of a target INR goal of <3.5 could minimize the risks of intra- and postoperative bleeding.[39] However, even with an INR <3.5, patients on warfarin still have an increased risk of bleeding compared to patients not taking anticoagulants.[40] Appropriate surgical techniques should be employed to minimize intra- and postoperative bleeding in these cases.

In summary, all prescribed monotherapy anticoagulants and antiplatelets should be continued for dermatologic surgeries. Aspirin may only be discontinued if it is self-prescribed by the patient or used solely for pain relief. For patients on combination therapy with two or more agents, discussion with the managing physicians is warranted to determine whether any of those agents can be safely discontinued. Appropriate laboratory monitoring, meticulous surgical techniques, and close follow-up will minimize complications in this patient population.

In our practice, nonprescribed aspirin is discontinued 7 days before and 3 days after the procedure, and a preoperative INR is checked within 24 hours with a target of <3.0. Consultation with the managing physician is standard for patients on two or more anticoagulants to determine whether discontinuation of one or more agents is feasible to reduce the risk of bleeding.

Corticosteroids

Chronic corticosteroid use carries two particular concerns relating to dermatologic surgery: (1) hypothalamus–pituitary axis (HPA) suppression requiring supplement stresses dose of steroids and (2) poor wound healing. HPA suppression can occur in patients receiving prednisone 20 mg po or higher dosing daily for more than 3 weeks. These patients should be continued on their normal daily prednisone dose and should receive additional corticosteroid for anticipated surgical stress, the dosing of which depends on the extent of the surgery. Superficial procedures such as skin biopsies and short procedures <1 hour in duration under local anesthesia typically do not require supplementation. For minor procedures or those that last longer than 1 hour, supplemental hydrocortisone 25 mg po once should be considered on the day of the surgery.[41] These recommendations are based on general surgery data since little data exist in the dermatologic surgery literature regarding perioperative steroid management. Chronic glucocorticoid use also interferes with multiple phases of healing, leading to delayed wound healing and an increased risk of infection.[42] Even mid- to high-strength fluorinated topical steroids can delay wound healing through impairment of reepithelialization, collagen formation, and angiogenesis.[43] Patients should be made aware of these potential complications prior to surgery so that nonessential topical steroid use may be discontinued.

Retinoids

Chemoprophylaxis with acitretin does not appear to increase the risks of infection, hypertrophic granulation tissue, scars, or dehiscence in organ transplant recipients undergoing Mohs or surgical excisions for nonmelanoma skin cancers.[44] Therefore, for nonelective surgeries, therefore, patients may continue taking acitretin in the perioperative period. For ablative resurfacing, the standard of care is discontinuation of isotretinoin for 6 months' preprocedure to avoid the risks of delayed healing and excessive granulation tissue based on the manufacturer's recommendations.[45] Acitretin can be withheld for 20 days prior to the procedure if concerns of similar complications exist, although none have been reported in the literature or by the manufacturer. In our practice, patients on low-dose (25 mg daily or less) acitretin for skin cancer prevention continue on this medication in the pre- and postoperative period surrounding Mohs surgery and reconstruction.

Biologic Response Modifiers

Biologic response modifiers or tumor necrosis factor (TNF) antagonists such as infliximab, etanercept, and adalimumab are most commonly encountered in patients with psoriasis, inflammatory bowel disease, or inflammatory arthropathies. These medications may increase the risks of opportunistic bacterial, mycobacterial, and fungal infections. However, available data have shown no significant increase in perioperative infections or dehiscence with TNF antagonists in rheumatoid arthritis and inflammatory bowel disease patients undergoing elective ankle and foot surgeries, joint replacements, and, abdominal surgeries.[46,47] Use of TNF antagonists in patients with inflammatory bowel disease actually improved healing of surgical wounds, fistulas, and mucosal ulcerations. A paucity of data exists on the perioperative risks of psoriatic patients on biologic response modifiers. Perioperative management options include continuing these agents with close postoperative follow-up, or discontinuing them 1 week before and 1 to 2 weeks after the surgery.[41,48] Consultation with the managing physician has been recommended for other immunosuppressive agents such as methotrexate, cyclosporine, tacrolimus, azathioprine, and mycophenolate mofetil since usage, dosing, and perioperative management vary depending on disease under treatment and other medical comorbidities. However, skin cancer is common in organ transplant patients who are often on such medications, and dermatologic surgical procedures are performed routinely in such patients without reports of increased complications. Thus, in our practice, no special perioperative considerations are made for patients taking these drugs (other than discussions with the treating team regarding dose reductions to boost immunity in patients who are developing multiple cancers and may, therefore, be overly immunosuppressed).

Herbal Supplementation

Use of herbal supplements has increased dramatically in the United States, accounting for more than $5 billion

TABLE 2-3
common Herbal Supplements and effects on Operative r isk[50]

Herbal Supplement	Possible ef ects on Surgical r isk
Garlic	■ Increased bleeding due to direct antithrombotic effects and increased effects of warfarin, ASA/NSAIDs ■ Hypotension due to increased effects of antihypertensive drugs
Ginseng	■ Increased bleeding due to direct anticoagulant effects and increased effects of warfarin, ASA/NSAIDs ■ Increased blood pressure and heart rate
Ginkgo	■ Increased bleeding due to direct anticoagulant effects and increased effects of warfarin, ASA/NSAIDs ■ Hypotension due to vasodilator effects
Ma Huang	■ Increased bleeding due to inhibition of complement cascade
Vitamin E	■ Increased bleeding due to inhibition of platelet function and vitamin K-dependent clotting factors
Echinacea	■ Possible poor wound healing in chronic users

in sales every year. T ere are more than 20,000 herbal medicines available on the market, with echinacea, garlic, goldenseal, ginseng, ginkgo, aloe, ma huang, St John's wort, valerian, and cranberry as the top selling products. Up to 85% patients with skin diseases report using complementary and alternative medicines, most commonly vitamins and herbal supplements, yet only a fraction of these patients report their use to physicians.[49,50] T e physiological effects are not fully understood for the majority of the herbal supplements. Studies on the more commonly prescribed agents have shown interference with hepatic cytochrome P450 enzymes, and interactions with many prescribed medications including anticoagulants, antihypertensives and antidepressants, and clinical effects on the cardiovascular, neurologic, and coagulation systems. T ese effects may result in prolonged anesthesia, bleeding, cardiovascular, and neurologic instability in the perioperative period (Table 2-3).[50,51] Given these potential complications, the American Society of Anesthesiologists recommends discontinuation of all herbal medications for 2 weeks prior to surgery under general anesthesia. Although no consensus recommendations exist regarding dermatologic surgery, advising patients to discontinue all herbal supplements 1 to 2 weeks before procedures such as excisions and Mohs micrographic surgery would be reasonable.

SOc ia l Ha bit S: a l c OHOl a n d SMOk in G

Alcohol may increase bleeding tendencies through inhibition of platelet aggregation, decreasing clotting factors, and increasing fibrinolysis and vasodilation.[52] Patients may therefore be instructed to stop or reduce alcohol intake 48 hours before and after surgery.

Smoking has not been definitively shown to increase the risk of surgical complications in patients undergoing routine dermatologic surgery. Large scale meta-analyses comparing complication rates between smokers and nonsmokers undergoing general surgery have found that smokers have an approximately four times higher risk of tissue necrosis, and a two times higher risk of surgical site infection, dehiscence, and healing delay. Former smokers have a significantly lower risk of surgical complications compared to current smokers but still higher than that of nonsmokers.[53] However, prospective 5 year data on surgical excisions of over 7000 skin lesions in 4100 patients revealed no difference in the rates of infection, bleeding, dehiscence, or flap/graft necrosis between smokers and nonsmokers. T e same conclusions held when the two groups were matched for age, gender, geographic location, and outdoor occupational exposure. T e total complication rate was approximately 4% for both groups.[54] Although smoking has deleterious effects on the overall health of the patient, it does not definitively increase complication rates in patients undergoing dermatologic surgery. Perioperative smoking cessation has produced mixed results in terms of reducing complications in smokers undergoing general surgery. Patients who stopped smoking 4 to 8 weeks before and 2 to 4 weeks after the surgical procedure had a reduction in surgical site infections but not in healing complications.[53]

a n t ibiOt ic Pr OPHyl a x iS

Prophylactic antibiotics serve two purposes: (1) to prevent local surgical site infection and (2) to reduce bacteremia resulting in endocarditis and prosthetic joint infections. Recent years have seen a shift away from routine prescription of prophylactic antibiotics, given that the risks of adverse effects from antibiotics outweigh their benefits in most cases based on available data. If a decision is made to provide antibiotic prophylaxis, it must be given within 1 hour of the procedure to ensure biologic availability at the time of incision. Topical antiseptics, surgical field preparation and maintenance, sterile equipment during wound closure, and surgical technique have a greater influence on risk of surgical site infection than the presence or absence of antibiotic prophylaxis.

TABLE 2-4
Dermatologic Procedures with High Risk of Surgical Site Infection[a]

Any procedure breaching oral mucosa
Any procedure involving infected skin or musculoskeletal tissue
High risk of local surgical site infection based on ■ Anatomical site: lower legs, groin ■ Reconstruction: grafts, wedge excision on the ear or lip

[a]Based on published guidelines from American Board of Dermatology and American Society of Dermatologic Surgeons.[57,58]

Prophylaxis Against Local Surgical Site Infection

The risk of surgical site infection in dermatologic surgery is quite low, ranging from 1% to 2% for excisions and Mohs micrographic surgery in multiple studies.[55] Medical comorbidities, smoking status, alcohol consumption, location and extent of tumor, and reconstruction techniques have been examined as potential factors influencing the risk of surgical site infection. Conflicting data on the degree of risk attributable to these factors make establishing consensus guidelines challenging. Diabetic patients and smokers do not necessarily have higher infection risks, as previously discussed. Surgeries on the lower legs or in the groin, wedge excisions of the lip and ear, and reconstruction with grafts carry a > 5% risk of infection based on prospective data from 5000 lesions in 2400 patients from a single practice in Australia.[4] However, prospective cohort data from the United States on 1000 patients undergoing Mohs micrographic surgery showed a culture proven infection rate of only 0.7% overall, and 3.1% for nose flaps.[56] Antibiotic prophylaxis to reduce local surgical site infections may be considered on an individual basis for surgeries on the lower legs and in the groin, wedge excisions, and grafts based on the overall clinical assessment of the patient and procedure (Table 2-4). Universal prophylaxis for all patients under these circumstances is not currently supported by available data.

The choice of antibiotic depends on the organisms that need to be covered, which varies with body location (Table 2-5). Most skin flora, such as Staphylococcus and Streptococcus species, can be treated adequately with cephalexin. Amoxicillin is the preferred agent for procedures that involve the oral mucosa. Patients with penicillin allergy may take levofloxacin for surgeries in the groin or on the lower extremities, and clindamycin or azithromycin for surgeries in other sites.[57] When local surgical site infection is suspected, a clinical evaluation should be performed and a culture should be obtained, followed by empiric treatment with penicillin- or cephalosporin-based antibiotics unless there is a high clinical index of suspicion for infection with methicillin-resistant Staphylococcus aureus (MRSA). The incidence of hospital and community acquired MRSA skin and soft-tissue infections has increased in recent years, especially in elderly, African–American, and male patients, and those with a history of hospitalization or surgeries in the previous year.[59] Rates of MRSA infection also vary between geographic locations and communities. For patients with a history of MRSA infection or colonization, clindamycin or trimethoprim-sulfamethoxazole may be used for empiric coverage for MRSA pending culture sensitivity results.[57]

Prophylaxis Against Endocarditis

Bacteremia from surgical procedures can lead to endocarditis in high-risk patients with specific valvular or cardiac defects. The American Heart Association narrowed its definition of patients at high risk for infective endocarditis in 2007 to include only those with prosthetic heart valves, cardiac transplantation with valvulopathy, cyanotic congenital heart defects that are unrepaired or repaired in the previous 6 months, or history of infective endocarditis (Table 2-6). Patients with mitral valve prolapse, rheumatic heart disease, bicuspid valve disease, calcified aortic stenosis, septal defects, and hypertrophic cardiomyopathy are no longer considered high risk. Furthermore, high-risk patients should only receive infective endocarditis prophylaxis when undergoing high-risk surgical procedures, namely surgeries involving the oral mucosa or clinically infected skin and musculoskeletal tissue.[60] The vast majority of dermatologic surgeries do not require infective endocarditis antibiotic prophylaxis even for high-risk patients. Immunocompetent patients undergoing excisions and Mohs micrographic surgery have a 1% to 3% risk of transient bacteremia,[61] a significantly lower risk than that from routine activities such as brushing teeth and eating.[62] Antibiotic prophylaxis has not been shown to protect patients from endocarditis resulting from these rare transient bacteremias. Moreover, antibiotics have a

TABLE 2-5
Antibiotic Prophylaxis for Prevention of Local Surgical Site Infection

Surgical Site	Antibiotic
Groin and lower legs	Cephalexin 2 g po If penicillin allergic: TMP-SMX-DS 1 tablet po OR Levofoxacin 500 mg po
Wedge excisions of ear or lip, grafts, nose flaps	Cephalexin 2 g po OR Dicloxacillin 2 g po If penicillin allergic: Clindamycin 600 mg po, OR Azithromycin or Clarithromycin 500 mg po

Source: Data from published guidelines from American Board of Dermatology, 2008.

TABLE 2-6
conditions with High r isk of infective endocarditis

Prosthetic cardiac valves
History of infective endocarditis
Cardiac transplant recipients who develop valvulopathy
Cyanotic congenital heart defects that are unrepaired, repaired within last 6 mo, or with residual defects at or near prostheses

Source: Data from American Heart Association consensus guidelines, 2007.[60]

long list of potential side ef ects including gastrointestinal disturbance, drug interactions, cutaneous eruptions, anaphylactic reactions, and drug resistance. Adherence to the published guidelines will reduce the number of patients receiving unnecessary antibiotics without compromising overall protection against infective endocarditis.

Pr OPHyl a x iS a Ga in St HeMat OGen Ou S j Oint in f ec t iOn

Total prosthetic joint infection may result from transient bacteremia during surgery, or more commonly from late onset bacteremia secondary to local surgical site infection.[63] T e occurrence of either scenario may be reduced with preoperative antibiotic prophylaxis. T e same principle guides antibiotic prophylaxis for joint infection as for infective endocarditis: prophylaxis is indicated only in high-risk patients undergoing high-risk procedures. Patients with an increased risk of prosthetic joint infection are generally those with prior joint infections, or a history of insulin-dependent diabetes, malignancy, or immunosuppression. Recognized causes of immunosuppression include inflammatory arthropathies such as systemic lupus erythematosus and rheumatoid arthritis, immunosuppressive medications, radiation, HIV infection, poor nutritional status, and hemophilia (Table 2-7). Patients at high risk of prosthetic joint infection require antibiotic prophylaxis when undergoing surgeries involving oral mucosa, infected skin and tissues, or procedures with high risk of local surgical site infections.[64] Assessment of surgical site infection risk was previously discussed in detail, but mainly takes into account anatomical sites such as legs and groin, and reconstruction with grafts. Routine antibiotic prophylaxis for all patients with prosthetic joints is not recommended for any other procedures including Mohs micrographic surgery. Patients with orthopedic pins, plates, or screws also do not require antibiotic prophylaxis.[57] At postoperative follow-up, both the surgical site and the area with joint prosthesis should be inspected for redness, pain, swelling, and warmth as signs of infection. Suspected local surgical site infection in patients with a prosthetic joint must be aggressively treated with initial empiric therapy designed to cover S. aureus and beta-hemolytic streptococci. T e definitive antibiotic choice should ultimately be guided by sensitivity results from cultures.

TABLE 2-7
Patients at High r isk of Prosthetic joint infection

All patients soon after prosthetic joint surgery
History of prosthetic joint infection
Insulin-dependent diabetes
HIV infection
Malignancy
Inf ammatory arthropathies (lupus, rheumatoid arthritis)
Drug or radiation-induced immunosuppression
Hemophilia
Poor nutritional status

Source: Data from American Association of Orthopedic Surgeons consensus guidelines, 2009.

c On Sen Su S St a t eMen t : Pr OPHyl a c t ic a n t ibiOt ic S

An advisory statement published by the Journal of the American Academy of Dermatology in 2008 recommended prophylactic antibiotics for high-risk patients with surgical sites that are infected or involving oral mucosa. Antibiotics may also be considered in patients with severe inflammatory dermatoses, grafts, flaps on the nose, wedge excisions of the ear or lip, and surgeries on the lower extremities or in the groin, to decrease the risk of surgical site infection.[57]

At our procedural dermatology and Mohs micrographic surgery center in a tertiary care academic institution, prophylactic antibiotics to prevent surgical site infection are routinely used only in patients with infected wounds (although surgery is usually delayed in such cases until infection has resolved), patients with extensive inflammatory skin diseases, or oral flaps/grafts. Saline irrigation is used prior to closure of all wounds. For prevention of infective endocarditis and hematogenous total joint infection, antibiotics are prescribed only for high-risk patients undergoing surgery involving the oral mucosa, groin, lower legs, or infected skin. Cephalexin 2 g po is the preferred antibiotic for cutaneous cases, and amoxicillin 2 g po for mucosal cases. Clindamycin 600 mg po or levofloxacin 500 mg po are alternatives for patients with penicillin allergies (Table 2-8).

Given that the guidelines for antibiotic prophylaxis for infective endocarditis and hematogenous prosthetic joint infection have undergone significant changes in recent years, patients may have come to expect antibiotic prophylaxis even when it is no longer indicated. Consultation with the patient's managing cardiologist or orthopedic surgeon to determine whether extenuating circumstances exist, as well as discussion of the treatment guidelines and patient education will help to clarify the appropriate course of action.

TABLE 2-8
Antibiotic Prophylaxis for Prevention of Infective Endocarditis or Total Prosthetic Joint Infection

Surgical Site	Antibiotic
Skin	Cephalexin 2 g po If penicillin allergic: Azithromycin or Clarithromycin 500 mg po OR Clindamycin 600 mg po
Oral mucosa	Amoxicillin 2 g po If penicillin allergic: Azithromycin or Clarithromycin 500 mg po OR Clindamycin 600 mg po

Source: Data from published guidelines from American Board of Dermatology, 2008.

Conclusion

This chapter has reviewed the major preoperative considerations of which surgeons should be aware prior to performing dermatologic surgical procedures, as well as current data and recommendations regarding antibiotic prophylaxis. Standardized checklists summarizing this information can be helpful in alerting physicians to their patients' medical conditions preoperatively, so that appropriate evaluation and/or action can be taken, and complications can be avoided. Physicians can use this preoperative screening to adjust care as needed in accordance with each individual patient's needs, thereby optimizing both patient safety and outcomes in the dermatologic surgical setting.

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Chapter 3

Informed Consent

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Introduction

A physician's duty has been succinctly described in medicine's famous oath "To Do No Harm." Although this concept may seem straightforward, defining harm can be more complex. A physician is in a unique position, possessing knowledge of the possible harm that may occur as a result of a procedure, which must then be communicated to the patient. There are many types of informed consent, which are generally based on the risk of the procedure to be performed. Traditional informed consent depends on the patient's capacity to comprehend and appreciate the nature and consequences of a decision regarding medical treatment; however, even routine situations can be complicated when they involve patients with individual needs and challenges. Our ultimate goal as physicians is to have open communication with informed and engaged patients. We will address how physicians can do their utmost to appropriately inform every patient in their practice about the risks inherent in the procedures that have been recommended for them, with emphasis on ways to improve the informed consent process.

understanding consent: the Basics

Obtaining proper consent is of paramount importance in dermatologic surgery. By obtaining consent for treatment, a physician invites the patient to take part in medical decision-making. Consent can fall into the categories shown in Figure 3-1.

Implied consent is typically employed in basic office situations, where a patient's conduct can be considered to be an inferred acceptance of a proposed medical intervention. Examples include the following:

- A patient does not withdraw or object when the medical assistant obtains the vital signs.
- A patient removes his or her shirt to show a skin lesion prior to the examination of the area.
- A patient applies sun block when advised to protect the skin from ultraviolet light.

In these situations, the patient's agreement is assumed by his or her conduct in response to the proposed intervention. This type of implied consent, otherwise known as simple consent, is appropriate for low-risk decisions. Accordingly, it is an accepted practice for the dermatologic surgeon to explain the low-risk intervention in simple terms and to accept the patient's implied consent.[1]

As circumstances become more complex, however, it becomes more precarious to rely on implied consent alone. This is because the burden of proof usually lies with the physician to show that the patient consented through his or her conduct.[2] Even in some low-risk situations, it may be wise to obtain express consent. Examples include the following:

- Performing a physical examination of a sensitive area.
- Low-risk procedures that are potentially painful (i.e., cryotherapy, intralesional injection).
- Prescription medications that pose some risk of an undesirable result (i.e., topical chemotherapy).

In the aforementioned examples, it is prudent at minimum to obtain verbal consent as well as verbalization of understanding from the patient.

As circumstances begin to involve a greater degree of risk to the patient, informed consent with shared decision-making comes to the forefront. Examples include the following:

- Using isotretinoin to treat severe acne in a fertile woman.[1]
- Surgical excision versus topical imiquimod for the treatment of a superficial basal-cell carcinoma on the arm.
- The option of a sentinel lymph node biopsy for a Stage II melanoma.

Often, it is best to obtain written consent from the patient. It is important to remember that informed consent is more than a patient's signature on a form; rather, it encompasses a process during which the physician can explore the patient's needs, values, and beliefs and invite patients to participate in their own healthcare decisions. During this process, physicians should discuss the nature

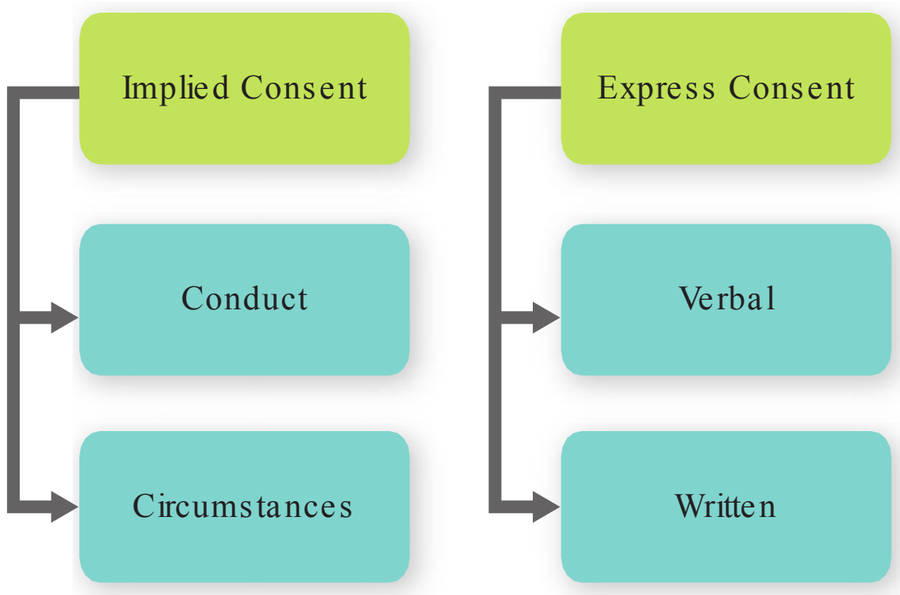

Figure 3-1 Types of consent.

of the medical intervention at length, including the purpose, risks, benefits, and alternatives, after which the patient should explicitly agree to or refuse the proposed intervention.[1]

development of the Informed consent principle: legal Background

Informed consent law derives from the principles of human autonomy and self-determination.[3] These basic human rights are fundamental to medical ethics and provide a basis for informed consent and shared decision-making. In addition, the concept of informed consent derives from the intentional torts of assault and battery. Assault is a volitional act intended to cause another person to fear harmful or offensive contact.[3,4] Battery takes place when there is intentional physical contact to a person against his or her wishes, which a reasonable person would consider harmful or offensive.[3,4] In medical battery cases, the patient (plaintiff) must prove that the unauthorized contact resulted in harm.[3,4] In surgical dermatology, a physician may be subject to allegations of battery under the following circumstances[4,5]:

- The surgeon performs a procedure without a patient's consent.
- The surgeon performs a procedure that is significantly different from the one to which the patient consented.
- The surgeon surpasses the scope of the consent.

As such, obtaining proper consent and acting accordingly are of paramount importance in dermatologic surgery.

The law also recognizes the concept of negligence as a completely separate distinct cause of action from battery law. Medical negligence takes place when a breach of duty occurs that compromises the standard of care and results in unintended harm to the patient. The components required to prove medical negligence are listed in Table 3-1.

Each of these components must be present in order to prove a case of medical negligence. First, physicians have a duty of reasonable care to patients to avoid foreseeable risks of harm. Medical malpractice may take place when a treatment falls below the accepted standard of care (breach of duty), which results in injury to the patient (causation and damages).

Central to the theory of medical negligence is the term standard of care, which refers to the conduct and degree of prudence expected of a similarly situated physician in a particular (similar) circumstance. If a plaintiff (patient) brings a claim that informed consent was absent or defective, it must be shown that the defendant (physician) did not conform to the standard of care when disclosing the pertinent risks, benefits, and alternatives of a procedure.[3,4] In other words, one must determine what a reasonably prudent physician in a similar situation would have disclosed regarding the given procedure.[3,4] But what determines the standard against which a defendant may be measured? Many jurisdictions will consider the community standard to be that prevailing within a physician's local geographic area; however, with advances in communication, technology, and electronic documentation, the scope of the duty has expanded. Thus, a physician's "community" may include a national and even an international level.

In addition to determining what a reasonable physician would disclose to a patient, in most jurisdictions it is important to assess what a reasonably prudent patient would have expected in order to make a decision in a given situation, based on the information presented to that patient. Inadequate information could lead a patient to choose an option that results in an unfavorable outcome. This approach means that in most jurisdictions the dermatologic surgeon should disclose all information that a patient would consider material to his or her decision-making.[6]

What makes a risk material? The answer lies in the risk's frequency of occurrence, as well as its severity.[6] It may not be relevant to focus on risks that are remote or unlikely to arise; however, if such risks could have severe consequences, they could become pertinent to the patient.

It is important to keep in mind that damages must also be incurred in order for an action for medical negligence to be present. In other words, even if informed consent was lacking or defective, the other elements of medical negligence must be satisfied in order for a patient to recover damages. This is means that the actual injury must have arisen as a result of occurrence of the nondisclosed risk.[6]

TABLE 3-1
components of negligence

Duty
breach of Duty
Causation
Damages

adequate Informed consent: elements

Shared decision-making and respect for patient autonomy are the key concepts behind the doctrine of informed consent. The basis for having appropriate informed consent is to promote a patient's self-determination in deciding what can be done to his or her body.[5] In order for consent to be adequate, the following elements must be fulfilled:

- Patient's capacity
- Adequate information
- Freedom to consent (noncoercion)

Capacity refers to the patient's ability to understand his or her medical problem and the need for treatment. The patient should be able to process the information at hand and be able to weigh the risks, benefits, and alternatives of the proposed treatment.[5–7] The patient should have the mental faculty to rationalize the information given and communicate a meaningful acceptance or refusal of that treatment. This is hinges on the requirement that the patient be without cognitive impairment. Specifically, a