Surgery in Thyroid Eye Disease

A Conceptual Approach

Suryasnata Rath
Milind N. Naik
Editors

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Foreword

Thyroid eye disease (TED) is the most common orbital disorder worldwide and can result in distressing changes in appearance, periocular discomfort, or visual disturbance. These complications often disrupt quality of life, ability to perform daily activities, and both social and work lives.

Recent discoveries concerning the pathogenesis of this autoimmune condition have identified more targeted medical therapies, raising hope that someday the more severe manifestations of TED may be avoided. Until then, however, surgery remains a vital component of management, either during the progressive active phase to prevent vision loss from optic nerve compression or corneal exposure, or during the quiescent stable phase to reduce proptosis, straighten eyes, and normalize eyelid position and appearance.

This surgical atlas is an invaluable asset for oculoplastic surgeons, comprehensive ophthalmologists, and their students, who wish to learn the most current surgical techniques to manage complications of TED. While there exist chapters on TED-related surgery in oculoplastic atlases, and there are textbooks devoted to Graves' disease and/or orbitopathy, this manual represents the first surgical atlas solely devoted to surgery for TED.

The book is organized within a conceptual framework that recognizes the natural course of the disease (active versus inactive), the periocular structures that are affected, and the order in which the surgeries are usually performed. High-quality intraoperative photographs and illustrations document key steps for each procedure, while succinct text highlights important tips, preoperative considerations, indications, and possible complications. Newer approaches such as endoscopic orbital decompressions, and smaller incisions for orbital and eyelid surgeries, and avoidance of late slippage in strabismus surgery are reviewed. Contributors include well recognized international experts in ophthalmic anatomy and surgery.

The text is authored and edited by two experienced and dedicated oculoplastic and orbital surgeons at the LV Prasad Eye Institute (LVPEI) in India, Dr. Suryasnata Rath and Dr. Milind N Naik. Both share similar educational backgrounds, a strong interest in managing TED and a passion for teaching nationally and internationally.

Suryasnata completed ophthalmology residency followed by fellowship at LVPEI and subsequently joined as faculty in 2006. From 2011 to 2012, he took leave to pursue further fellowship training with me and my colleagues at The University of British Columbia, Canada, where he developed a keen interest in TED. I remember Surya fondly for his jovial personality that

endeared him to patients and colleagues, his enthusiasm and skill as a clinician, surgeon and teacher, and his love of the outdoors, wildlife, and gardening. I also noticed his considerable leadership skills, so was not surprised that following his return to India, he was soon promoted to head the Mithu Tulsi Chanrai Campus in Bhubaneswar as well as director of the division of oculoplastics and orbit at that campus.

Milind completed his ophthalmology training at Christian Medical College, Vellore and joined the faculty of LVPEI in 2001. He took leave for a fellowship in oculoplastics and orbital surgery with Bob Goldberg and colleagues in 2006. I first met Milind at a conference on TED at Jules Stein Campus, a seminal meeting that subsequently led to the formation of the International Thyroid Eye Disease Society. Milind impressed me with his polite manner, quiet sense of humor, and highly analytic mind. His talents as a surgeon, teacher, and writer have led to his highly respected position in the field of ophthalmic plastic surgery in India and globally. Milind has delivered numerous lectures nationally and internationally, and I always feel lucky to attend these, as they are consistently lucid, carefully crafted, entertaining, and highly informative.

This textbook on surgical management of TED reflects the dedication and experience of the two co-editors and of all the contributing authors. The rich dividend of this combined effort will be better surgeons and enhanced quality of life for their patients suffering from TED.

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Foreword

Thyroid eye disease (TED) can make patients miserable. On the horizon, perhaps, are medical treatments that will treat the underlying medical disease: hopefully, future doctors will look back at our surgical treatment of an autoimmune disease as barbaric. In the meantime, we have to go to the operating room to deal with the sequelae of TED. Fortunately, surgery has evolved substantially so that we can truly help our patients with their disfigurement, vision, and comfort. Drs. Rath and Naik, highly regarded international experts in TED, have assembled an outstanding compendium of cutting-edge surgical techniques, clearly described and illustrated, that will allow physicians to improve and refine their ability to help their patients.

It is worth noting that the authors are Ophthalmologists. An anecdote regarding Henry Baylis, the Founding Chief of Orbital and Ophthalmic Plastic Surgery at UCLA and one of the pioneers of our specialty, might provide an informative perspective on the history of orbital decompression. Dr. Baylis had a vision of a robust discipline of Ophthalmic Plastic Surgery, and was a leader in expanding the field beyond treatment of eyelid disorders. In the 1970s, when orbital decompression was performed almost exclusively by otolaryngologists, Hank took the initiative to learn and refine techniques of transantral orbital decompression. He used to enjoy telling the story that when he first presented this work at the AAO meeting in San Francisco, Crowell Beard, one of his mentors, commented that it was malpractice for an ophthalmologist to do orbital decompression. Henry was dismayed but not deterred. To today's trainees, it seems obvious that orbital decompression is a core Oculofacial procedure, but it was not always that way.

That anecdote reflects a fairly recent history. Now, oculofacial surgeons are at the leading edge of developing new surgical treatments for TED, and surgical rehabilitation has continued to improve. We now have minimally invasive techniques for orbital decompression that can be individualized and graded, and are associated with decreased morbidity. Eye muscle surgery continues to evolve conceptually and technically. Taking advantage of developments in aesthetic surgery, we now approach eyelid repositioning and soft tissue rehabilitation with customized, small incision approaches that allow us to more closely recreate our patients' premorbid comfort and appearance. In the chapters that follow, the fruits of these years of hard work and innovation are on display.

We are preceded by giants. Their passion and generosity in teaching, their dedication to their patients, and their unwavering commitment to advancing

the discipline, is their legacy. Drs. Rath and Naik pay homage to this legacy by creating this textbook. I am sure they will be very gratified when their efforts result in better surgeons who improve the quality of life of the patients they treat.

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Preface

The Making of "Surgery in Thyroid Eye Disease: A Conceptual Approach"

Thyroid eye disease (TED) is a debilitating disease that affects the orbit and adnexal structures. While severe TED can affect visual function, even mild to moderate disease can disrupt the work, family, and social life of individuals. Management of TED usually takes months to years. Surgical interventions in TED are spread over a wide spectrum, and choosing the right surgical option can be daunting.

Several excellent comprehensive textbooks and treatises exist that deal with the pathogenesis and management of TED. However, most are descriptive in nature. An oculoplastic surgeon managing TED is often intimidated by the surgical challenges that lie ahead. These challenges are different in the active phase, as against the quiescent phase.

Some of these challenges include the choice of anesthesia, single-stage vs. multi-stage intervention, surgical approach, and finally, the optimum outcome that is just right for the patient. This surgical manual intends to fill this void by focusing on surgery through pictures, schematics, flowcharts, and complementing text to share nuances and pearls to guide the oculoplastic surgeon in this path and improve their outcome.

Dr Rath's interest with TED grew after managing a young lady more than a decade ago. She presented with bilateral severe proptosis with exposure changes. Her disfigurement was the sequel of burnt-out TED, and she had covered her face when she arrived. She had been blind for several years. Disowned by her parents, she was brought to the institute by a local non-governmental charity.

As a young oculoplastic surgeon, Dr Rath was overwhelmed by achieving euthyroid status (which took months), followed by sequential staged orbital decompression, and eyelid surgeries to correct the exposure and improve her appearance. Finally, vision salvage was attempted with penetrating keratoplasty in the right eye after lengthy discussions and considerable counseling. She improved to 20/50 in the right eye. When she walked into the clinic several months later along with a smile and her parents, her appearance said it all (Fig. 12.11a-b). The journey took six long years, but Dr Rath had managed to restore useful vision and rehabilitate her in society. Lessons learned were for

life—TED can devastate an individual physically and emotionally. Nevertheless, a planned, systematic approach comprising a team of specialists can restore normalcy.

For Dr Naik, his trigger patient was a middle-aged lady presenting in 2002 with inactive TED who was depressed because her grandchildren were scared to come close to her. Dr Naik realized how despite vision being normal, TED could change a person's appearance leading to psychosocial disharmony. She was asked by her ophthalmologist to "hide" her eyes behind dark glasses, as orbital decompressions were not being performed in India back then.

There was much learning that happened over a decade and a half for both Dr Rath and Dr Naik while managing patients with TED. For Dr Rath, an international fellowship with Prof. Peter Dolman at the University of British Columbia, Vancouver, Canada, helped vertical learning. The exposure goaded him to delve into understanding the pathogenesis of TED and the role of disease-modifying agents to flatten the curve. For Dr Naik, his International Fellowship in Orbitofacial Plastic Surgery at UCLA, Los Angeles, with Prof. Robert Goldberg paved the way for a deep liking towards TED and minimally invasive surgical approaches for the same.

As Dr Rath and Dr Naik metamorphosed from novice surgeons to experts in the surgical management of TED, on several occasions, both felt the need for a surgical manual that could guide fellow surgeons in this journey.

The process of building the chapter outline, its flow, and highlighting the nuances of surgery was based on this need and turned out to be a fantastic experience for both. As this was a surgical atlas, there was a perpetual obsession to get the best clinical photographs. On several occasions, the precise surgical photograph in their mind took months before it depicted the intended surgical pearl. Their contributing authors across the globe made their job easier by giving their best of talent and photographs!

Finally, both editors had the difficult task of meeting deadlines and sending reminders. The turning point, however, was a marathon session when both of them disappeared from the surface of the earth for 3 days: dug in a secret cove and focused only on the completion of the book! The rest is history.

While new medical therapies keep emerging, there would be a day when surgeries would not be required for new patients of TED, and we could medically avoid the progression of proptosis, strabismus, and lid retraction. Until then, and for the benefit of those patients who are in quiescent phase already, surgical intervention, would remain relevant for rehabilitation. This manual will prove to be a useful, engaging, and ready-reference guide for the oculoplastic surgeons managing TED, both in the clinic and the operating room.

Bhubaneswar, India — Suryasnata Rath
Hyderabad, India — Milind N. Naik

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This book has been a collaborative project from the start. Being a surgical book, the role of our photographers and artists were pivotal. We thank our lead medical photographers, SBN Chary, Naresh Gattu, Pradeep Moharana, and Nishanth B. Their zeal for perfection in clinical and intraoperative photographs has contributed immensely to the making of this book. We also thank Dr. Virangi Doshi, our Ophthalmology resident and Dr. Varshitha H. Vasanthapuram, clinical fellow for their medical illustrations of the highest quality.

We gratefully acknowledge the thoughtful comments and advice of the many clinical fellows in Ophthalmic plastic surgery, visiting scholars, mentors, and well-wishers, both national and international, who supported us through the making of *Surgery in Thyroid Eye Disease: A Conceptual Approach*.

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Contents

About the Editors

Suryasnata Rath, FRCS Dr. Suryasnata Rath is currently Head of the Mithu Tulsi Chanrai (MTC) campus of LV Prasad Eye Institute, Bhubaneswar, India and of the Ophthalmic Plastics, Orbit and Ocular Oncology Services at MTC. Following his basic medical training in Odisha, he completed long-term fellowships (2001–2003 and 2005–2006) at the LV Prasad Eye Institute, Hyderabad, India before joining as faculty in July 2006.

He became a fellow of the Royal College of Physicians and Surgeons (FRCS), Glasgow, in 2004. Since his training in Oculoplastics with Dr. Peter Dolman at the University of British Columbia, Canada, in 2011–2012, he has developed a keen interest in thyroid eye disease and endonasal lacrimal surgery. Suryasnata is the principal investigator of several ongoing projects, including a multicenter randomized trial on orbital radiotherapy in thyroid eye disease and a Department of Biotechnology (DBT)-funded project to explore the role of infection in ocular adnexal lymphoma. Suryasnata recently stepped into the realm of innovation as a recipient of the Biotechnology Industry Research Assistance Council–Biotechnology ignition grant (BIRAC-BIG) to develop a device for the minimally invasive treatment of chronic dacryocystitis. He has written 47 peer-reviewed, indexed publications, and several chapters on non-endoscopic endonasal dacryocystorhinostomy.

Milind N. Naik, MD Dr Milind N. Naik completed his postgraduate training in Ophthalmology at the Christian Medical College, Vellore, India. In 2001, he then completed a fellowship in Ophthalmic plastic surgery at LV Prasad Eye Institute, Hyderabad, India. He completed his Orbitofacial plastic surgery fellowship at the Stein Eye Institute, University of California, Los Angeles, in 2006–2007. His areas of interest include thyroid eye disease, aesthetic orbitofacial surgery, and minimally invasive ophthalmic plastic surgery.

He received the American Academy of Ophthalmology's Senior Achievement Award in 2015 and is a member of the

American Society of Ophthalmic Plastic and Reconstructive Surgery (ASOPRS). He served as the Vice President of the Asia Pacific Society of Ophthalmic Plastic and Reconstructive Surgery (APSOPRS) from 2010 to 2012.

Dr Naik has published over 200 peer-reviewed articles in international journals and lectures extensively around the globe in the field of Ophthalmic plastic surgery and facial aesthetics. Dr Naik is a senior Consultant, Ophthalmic Plastic Surgery Services at LV Prasad Eye Institute, Hyderabad, India. He also serves as Associate Professor, Department of Ophthalmology and Visual Sciences, University of Rochester, New York, USA.

Part I

Anatomy and Pre-operative Planning

1 Anatomy of the Eyelids Pertaining to Thyroid Eye Disease

Jonathan J. Dutton

1.1 Introduction

The major clinical eyelid findings associated with thyroid eye disease (TED) are varied, but most often involve upper and lower eyelid retraction, contour abnormalities, eyelid edema, prolapsed orbital fat, conjunctival injection and chemosis, and meibomian gland dysfunction. The exact causes of these changes remain a matter of controversy, but numerous hypotheses have been proposed, most with limited experimental support.

Eyelid retraction is probably the most significant manifestation of TED. Not only because of it's obvious aesthetic implications for the patient, but because of its relation to corneal exposure and it's potential for vision loss. Three theories related to the etiology of eyelid retraction have been mentioned in the literature and repeated in numerous publications on TED. The first is a mechanical factor related to globe proptosis. Rajabi et al. [1] evaluated 166 TED eyes and found no correlation between lower lid position and inferior fornix depth as a measure of retractor fibrosis, but they did find a significant correlation between proptosis and degree of lid retraction. The authors concluded that the mechanical draping of the lid over a proptotic globe was the main cause of lower lid retraction. This is partially supported by the observation that retraction usually improves following orbital decompression.

Upper eyelid retraction appears to be more complicated. Although proptosis might play some role, it is considered to be a rather minor factor [2]. A more widely quoted mechanism is Müller sympathetic muscle overaction, ever since it was proposed by Pochin in 1939 [3]. One basis for this is that many of the clinical symptoms in hyperthyroidism, such as palpitations, tachycardia, tremor, sweating, and heat intolerance are related to beta-adrenergic activity [4, 5]. Also, beta-adrenergic blockade is well-known to modify these symptoms in Graves patients [6–8]. Morton et al. [9] demonstrated a lateral extension of Müller smooth muscle fibers accompanying the lateral horn of the levator aponeurosis between the lobes of the lacrimal gland. They proposed that overaction of this portion could contribute to the lateral flare frequently seen with eyelid retraction in TED patients. Although Noh et al. [10] found sympathetic overactivity in intraocular smooth muscles of hyperthyroid patients, there was no difference in this activity in patients with or without eyelid retraction. They concluded that eyelid retraction was not caused by sympathetic overactivity alone, but by other factors in addition.

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Other factors that likely contribute to upper lid retraction are inflammation and fibrosis of Müller muscle, but the histologic data are inconsistent. Lowinger et al. [11] found no significant histologic difference in Müller muscle specimens from inactive TED patients compared with normal controls. However, Cokerham et al. [12] and Shih et al. [13] found increased inflammation, fibrosis, and fat infiltration in the Müller muscles of inactive euthyroid TED patients.

Another mechanism proposed to contribute to upper lid retraction is overaction of the levator palpebrae superioris (LPS) muscle. Small [14] showed CT evidence of enlargement of the levator muscle proximal to Whitnall ligament in TED patients compared with controls, and suggested hypertrophy as the likely cause of eyelid retraction. Wesley et al. [15] also demonstrated LPS enlargement and proposed that inferior rectus muscle restriction caused overaction of the superior rectus and levator muscles from Hering law.

Some degree of levator connective tissue system fibrosis and of the suspensory ligaments of the superior conjunctival fornix may be at least partially responsible for upper lid retraction. It is not uncommon to find persistent retraction during surgery even when the levator aponeurosis and Müller muscle are completely detached up to the level of Whitnall ligament. This view is supported further by the observation of diminished levator excursion associated with increasing levels of lid lag and lagophthalmos in TED patients with lid retraction [16].

One final mechanism for eyelid retraction was suggested by Harrison et al. [16] who noted a significant reduction of myofibers in the preseptal orbicularis muscle of hyperthyroid rabbits compared with controls. The weakened tone in the orbicularis muscle could allow overaction of the less opposed levator muscle.

Eyelid edema is a common manifestation of thyroid eye disease that may be unilateral or bilateral. Histologic evidence has shown dilated lymphatic vessels and perivascular cellular infiltrate, mostly lymphocytic, in the dermis of eyelid skin in hyperthyroid patients, but with no deposition of mucopolysaccharides [17]. The mechanism is not clear, but has been suggested to possibly be from reduced lymphatic drainage associated with decreased eyelid motility associated with lid retraction and globe proptosis, or from periorbital venous stasis related to orbital fat prolapse, proptosis, and inflammation [18].

TED patients typically show ocular surface changes with an unstable tear film and severe symptoms of dry eyes [19]. The etiology is not completely understood, and there are some conflicting studies. Gürdal et al. [20] and Ozkan et al. [21] demonstrated increased squamous metaplasia associated with decreased Schirmer tear test and increased tear breakup time in Graves patients compared with controls. However, Kikkawa [22] reported only a trend toward higher conjunctival inflammation that was not significant when compared with normal controls.

With these clinical manifestations of eyelid features in TED patients in mind, we will review eyelid anatomy.

1.2 Anatomy of the Eyelids

In young adults, the interpalpebral fissure measures 10–11 mm vertically, but with advancing age this decreases to only about 8–10 mm. The horizontal length of the fissure is 30–31 mm, and the upper and lower eyelids meet at an angle of approximately 60° medially and laterally (Fig. 1.1) [2]. In primary position, the upper eyelid margin normally lies at the superior corneal limbus in children and 1.5–2 mm below it in adults. The lower eyelid margin rests at the inferior corneal limbus. A well-defined eyelid crease and supra-crease fold in the upper lid mark the approximate zone of attachment of levator aponeurosis fibers to the orbicularis muscle and skin. The lacrimal puncta are situated medially, 6–8 mm from the canthal angle. In thyroid eye disease, the upper and lower eyelids are often retracted so that white sclera is visible between the cornea and the eyelid margins.

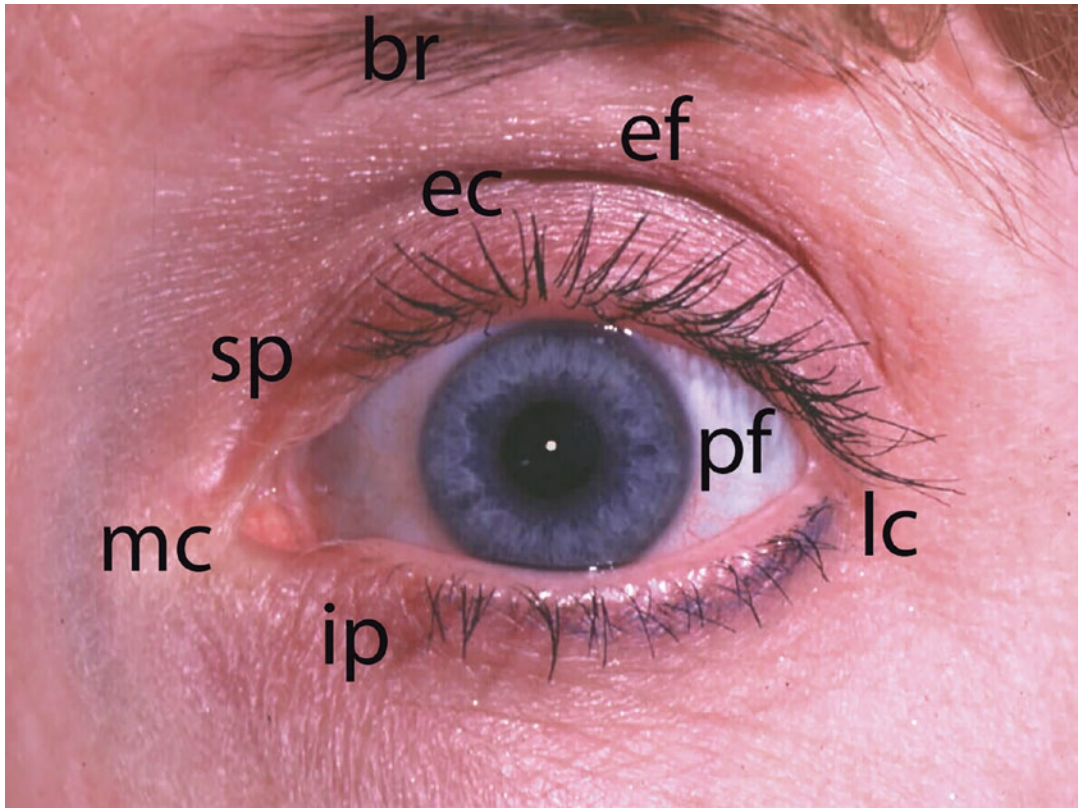

Fig. 1.1 Anatomy of the normal eyelids. This frontal photograph shows the brow (br), the eyelid crease (ec), eyelid fold (ef), inferior punctum (ip), lateral canthus (lc), medial canthus (mc), palpebral fissure (pf), and superior punctum (sp)

1.3 Eyelid Margin

The eyelid margin is about 2-mm thick (Fig. 1.2). It is covered posteriorly by conjunctival epithelium interrupted by the meibomian gland orifices, about 25 in the upper eyelid and 20 in the lower. Anteriorly, the margin is covered with skin from which emerge several rows of eyelashes. Separating these posterior and anterior lamellae, is a faint gray line that represents the marginal projection of specialized horizontal fibers of Riolan muscle that may in part function in meibomian secretion [23]. Tissue lamellae are seen in sagittal section through the lateral upper eyelid in Fig. 1.3.

During eyelid recession procedures for lid retraction, an interpositional graft is usually placed on the posterior lamella to lengthen the capsulopalpebral fascia. A three-dimensional layered dissection through the upper and lower eyelids is demonstrated in Figs. 1.4 and 1.5.

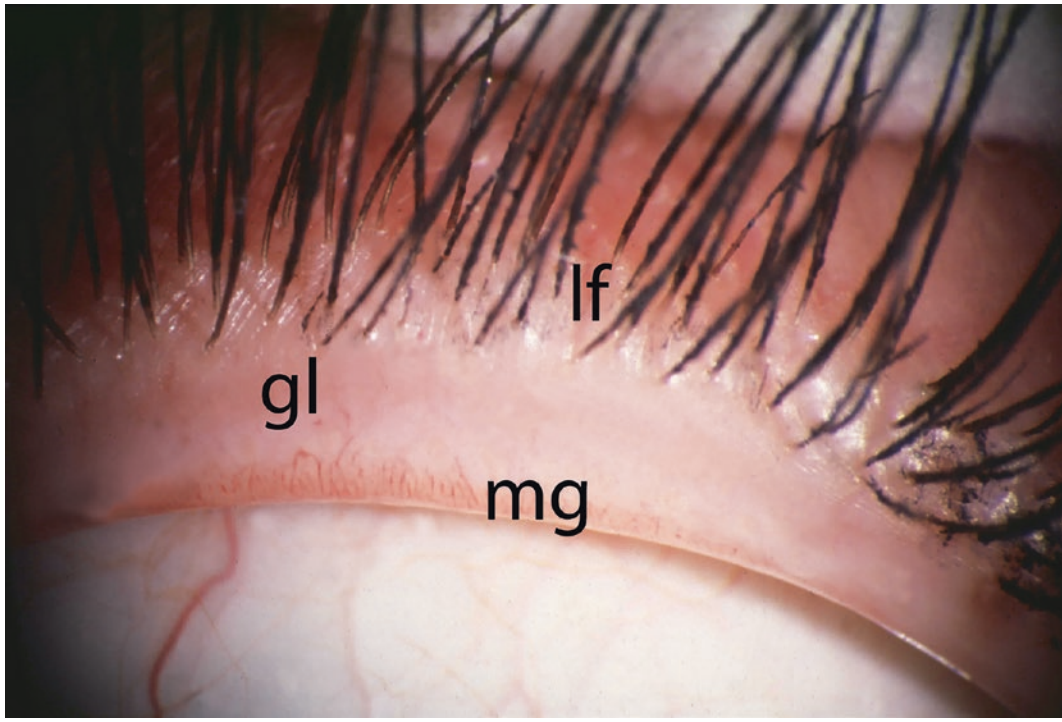

Fig. 1.2 Eyelid margin, showing the meibomial glands (mg), grey line (gl), and lash follicles (If) arranged posterior to anterior

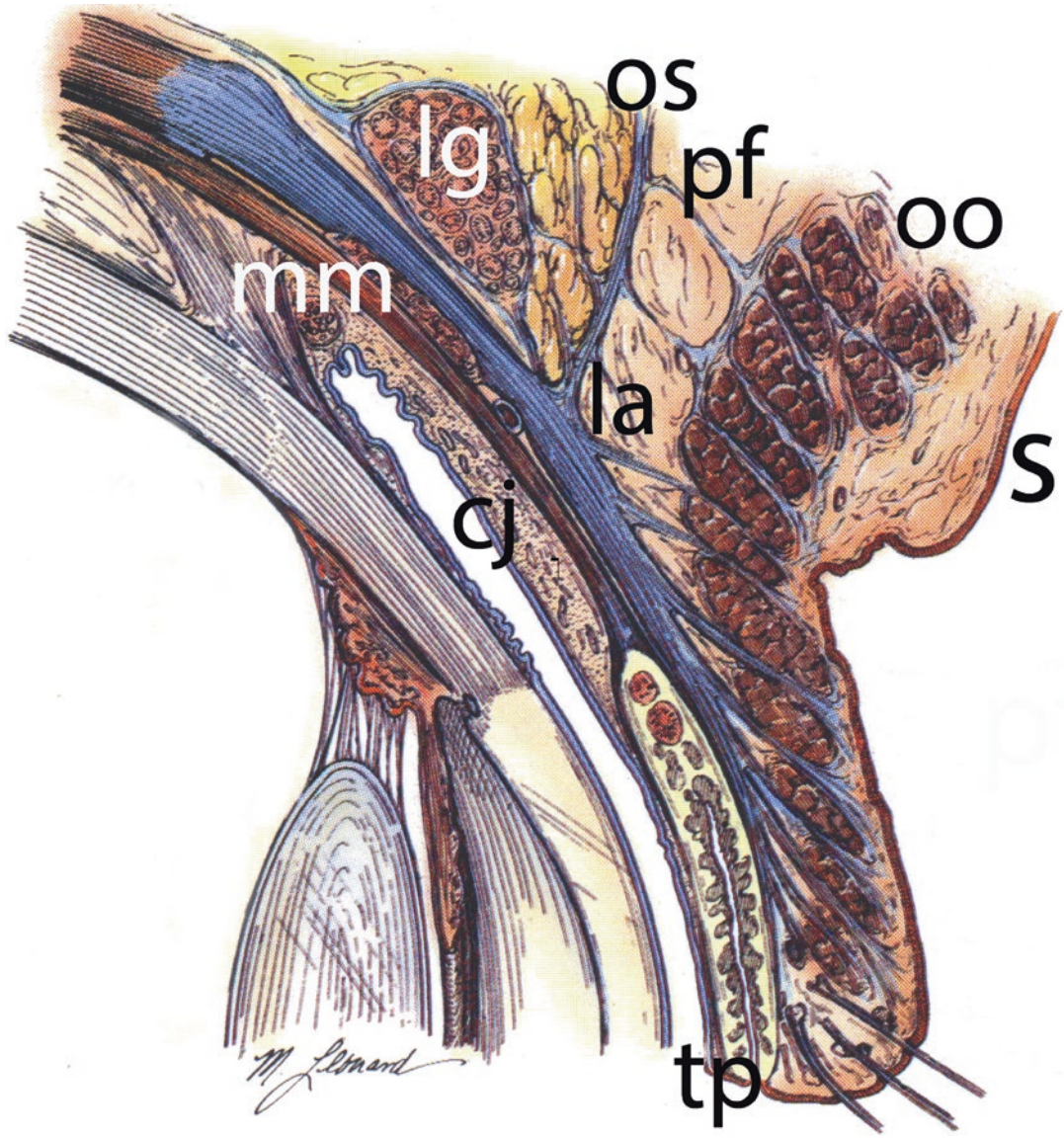

Fig. 1.3 Sagittal section of the upper eyelid showing skin (s), orbicularis oculi muscle (oo), post-orbicular fascial plane (pf), orbital septum (os), levator aponeurosis (la), Muller muscle (mm), lacrimal gland (lg), conjunctiva (cj), and tarsal plate (tp)

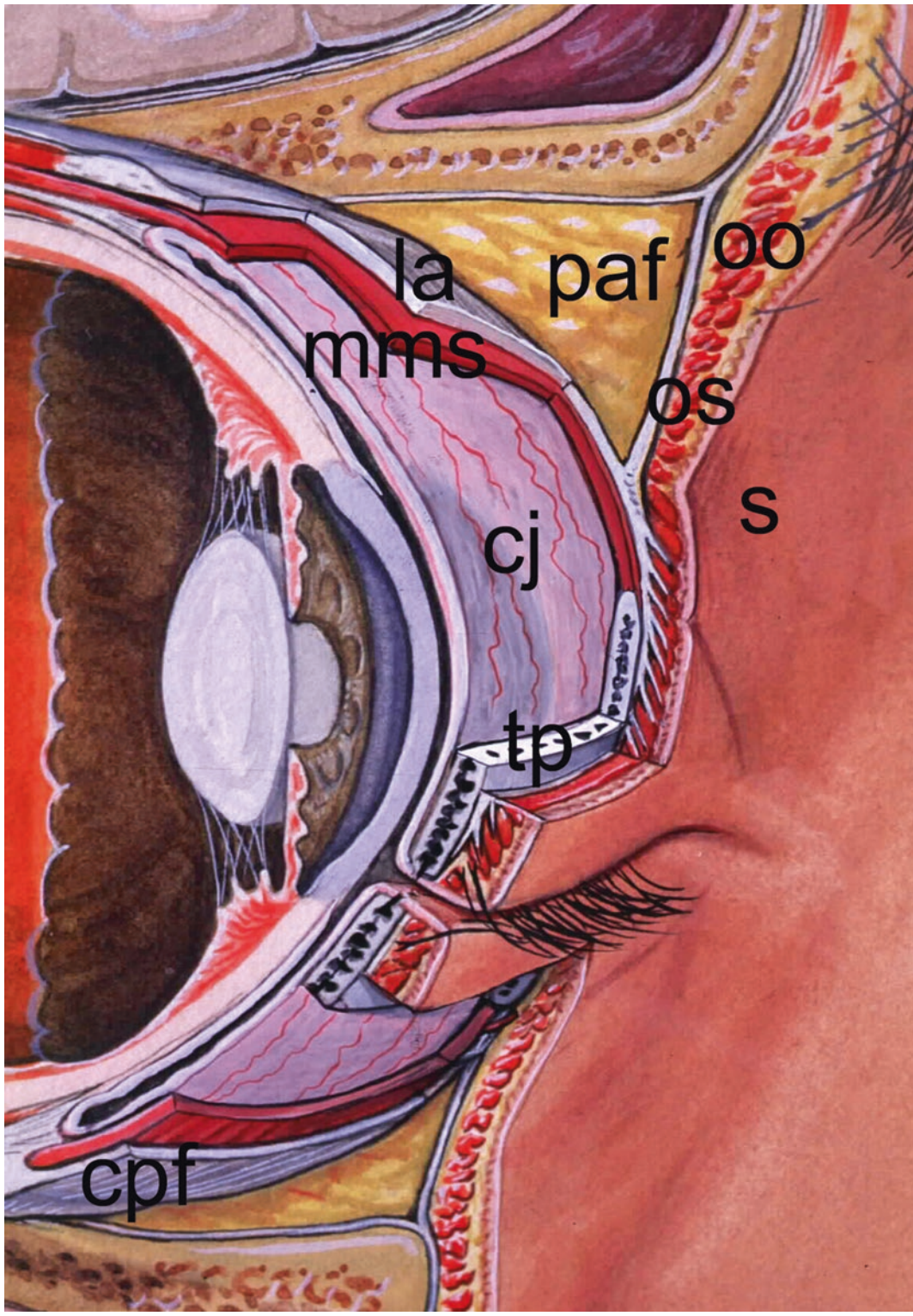

Fig. 1.4 A three-dimensional layered dissection through the upper and lower eyelids showing the skin(s), orbicularis oculi muscle(oo), tarsal plate (tp), conjunctiva (cj), orbital septum (os), preaponeurotic fat (paf); levator aponeurosis (la), superior Muller muscle (mms), and capsuloplalpebral fascia (cpf)

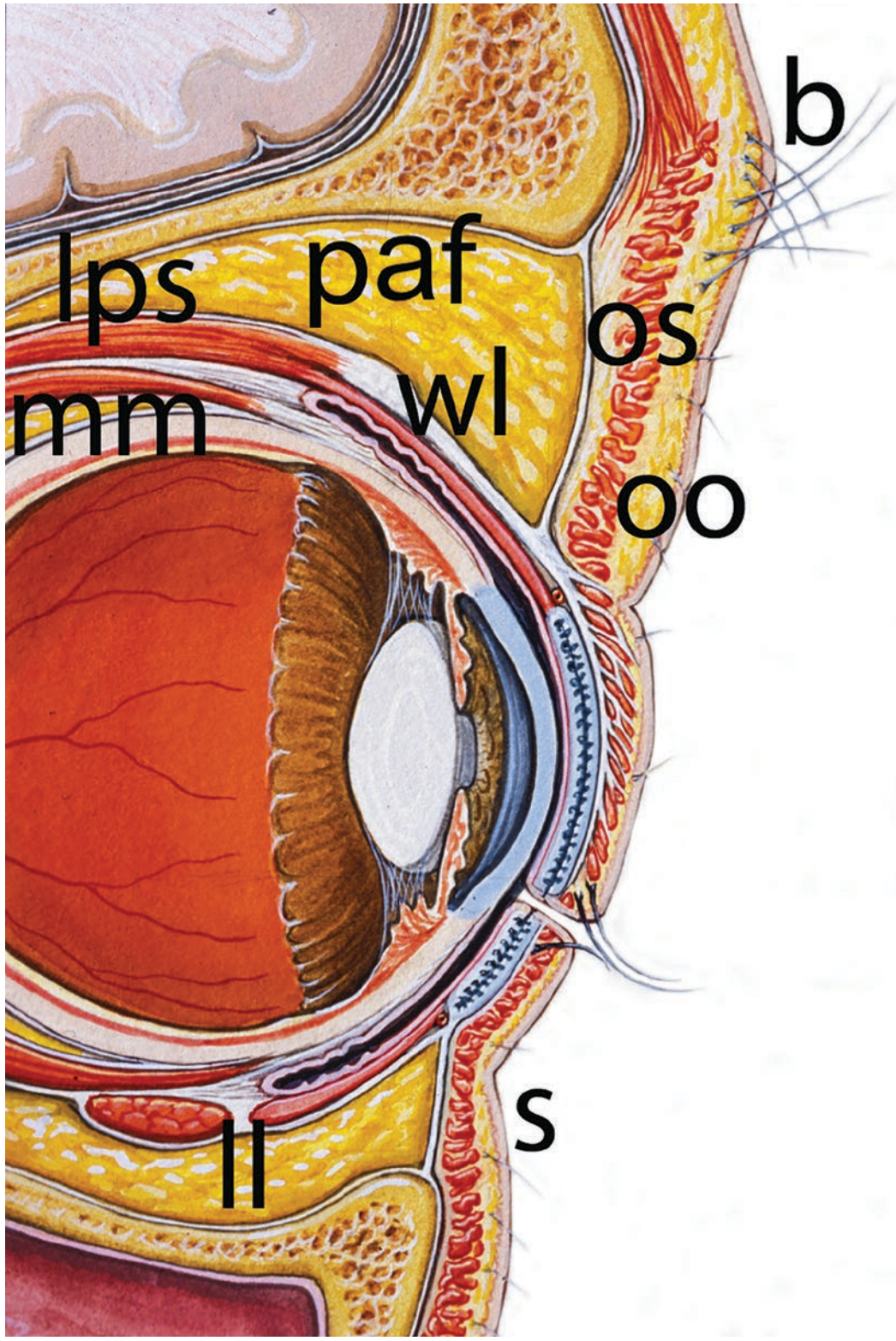

Fig. 1.5 Sagittal section through the globe, upper and lower eyelids showing the brow (b), orbital septum (os), orbicularis oculi muscle (oo), skin (s), Whitnall ligament (wl), Lockwood ligament (ll), levator palpebrae superioris (lps), Muller muscle (mm), and preapnoneurotic fat (paf)

1.4 Orbicularis Oculi

The orbicularis oculi is a complex striated muscle that lies just below the skin (Fig. 1.6). It is divided anatomically into three contiguous parts: orbital, preseptal, and pretarsal [24, 25]. The orbital portion overlies the bony orbital rims. Its fibers pass around the orbital rim to form a continuous ellipse. The palpebral portion overlies the mobile eyelid from the orbital rims to the eyelid margins. It is divided topographically into the preseptal and pretarsal portions (Fig. 1.6). The preseptal portion overlies the orbital septum in both upper and lower eyelids and the pretarsal portion overlies the tarsal plates. Medially, the deep heads of the pretarsal fibers fuse to form Horner muscle that runs behind the posterior limb of the canthal tendon to insert onto the posterior lacrimal crest. Horner muscle helps maintain the posterior position of the canthal angle and may aid in the lacrimal pump mechanism [26].

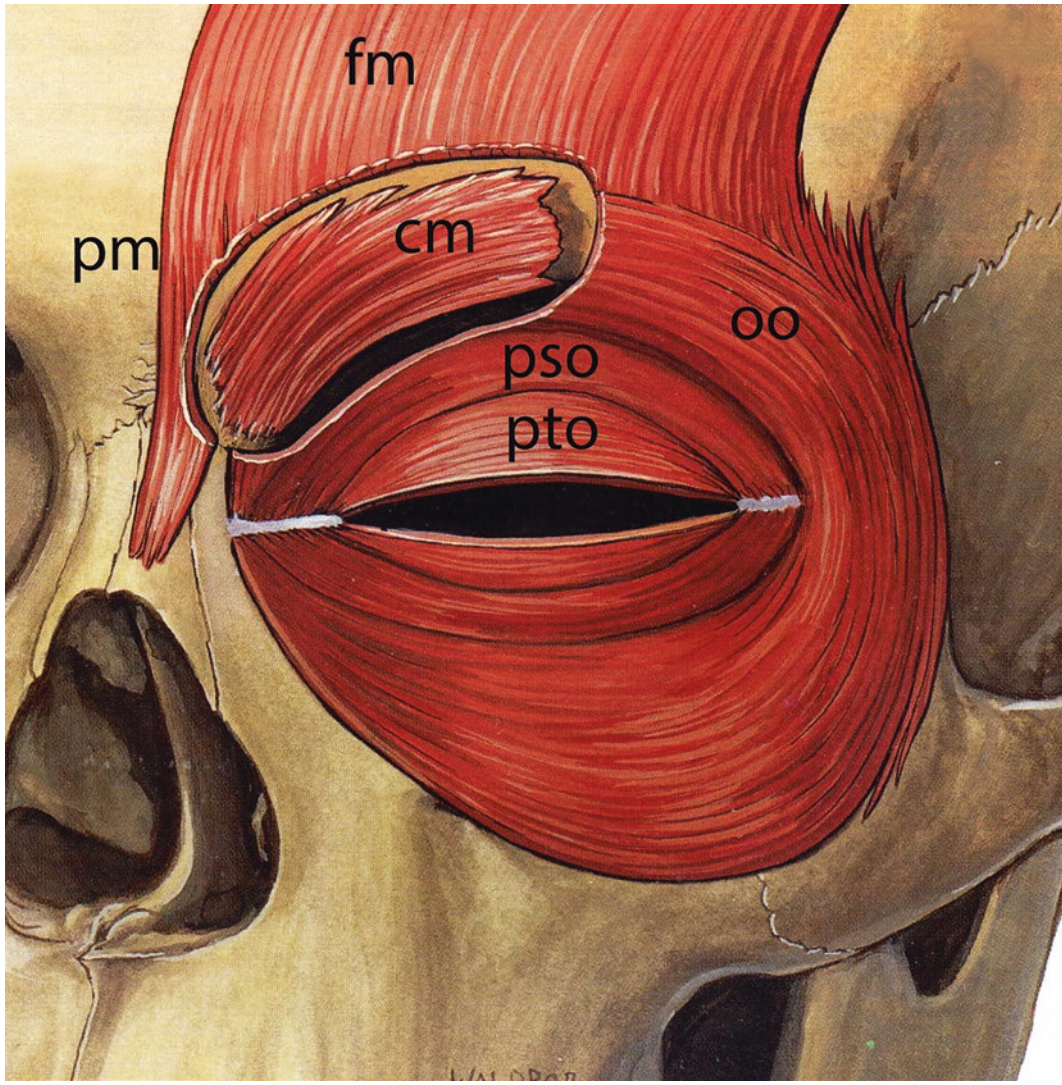

Fig. 1.6 Periorbital musculature. Procerus muscle (pm), frontalis muscle (fm), orbicularis oculi muscle(oo), preseptal orbicularis (pso), pre-tarsal orbicularis (pto), and corrugator muscle (cm)

The preseptal orbicularis muscles are attached to the medial canthal tendon via deep and superficial heads (Fig. 1.7). The superficial heads partially surround the lacrimal canaliculi and contraction during the eyelid blink cycle helps fold these structures to prevent reflux of tears. Along the eyelid margins, Riolan muscle is a specialized portion of the orbicularis with short horizontal fibers that surround the orifices of the Meibomian glands and may help with secretion. The deep heads pass posterior to the canthal tendon where they merge with Riolan muscles to form Horner muscle just posterior to the lacrimal sac.

The muscles of Riolan are distinct bundles of fibers along the eyelid margins (Fig. 1.8). The main portion of the muscle inserts around the lacrimal puncta and ampullae. Deep fibers form short bundles that run in various directions along the lid margin and between tarsus and conjunctiva. Fine bundles surround the eyelash follicles and excretory ducts of the Meibomian glands.

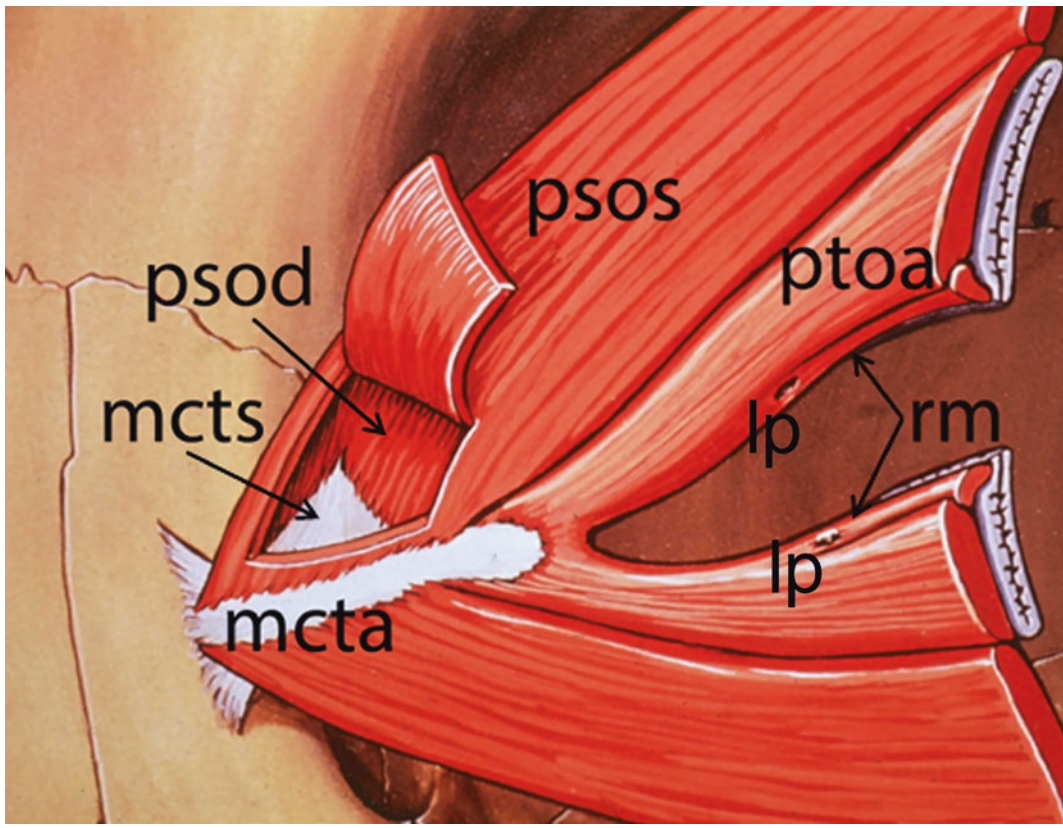

Fig. 1.7 Medial canthal soft tissue anatomy, showing the medial canthal tendon, anterior arm (mcta), medial canthal tendon, superior arm (mcts); Riolan muscle (rm), preseptal orbicularis superficial head (psos), pre-septal orbicularis, deep head (psod), pretarsal orbicularis, anterior (ptoa), and lacrimal puncta (lp)

The *medial canthus* supports structures that maintain alignment and orientation of the medial eyelids [27]. Medially, the tarsal plates pass into fibrous bands that form the crura of the medial canthal tendon. The superior and inferior crura fuse to form a stout common tendon that inserts via three arms (Fig. 1.9). The *anterior arm* inserts onto the orbital process of the maxillary bone in front of and above the anterior lacrimal crest. The *posterior arm* arises from the common tendon and passes between the canaliculi to insert onto the posterior lacrimal crest in front of Horner muscle. The *superior arm* arises as a broad arc of fibers from both the anterior and the posterior limbs. It passes upward to insert onto the orbital process of the frontal bone. It provides vertical support to the canthal angle and appears to play a role in the lacrimal pump mechanism [28].

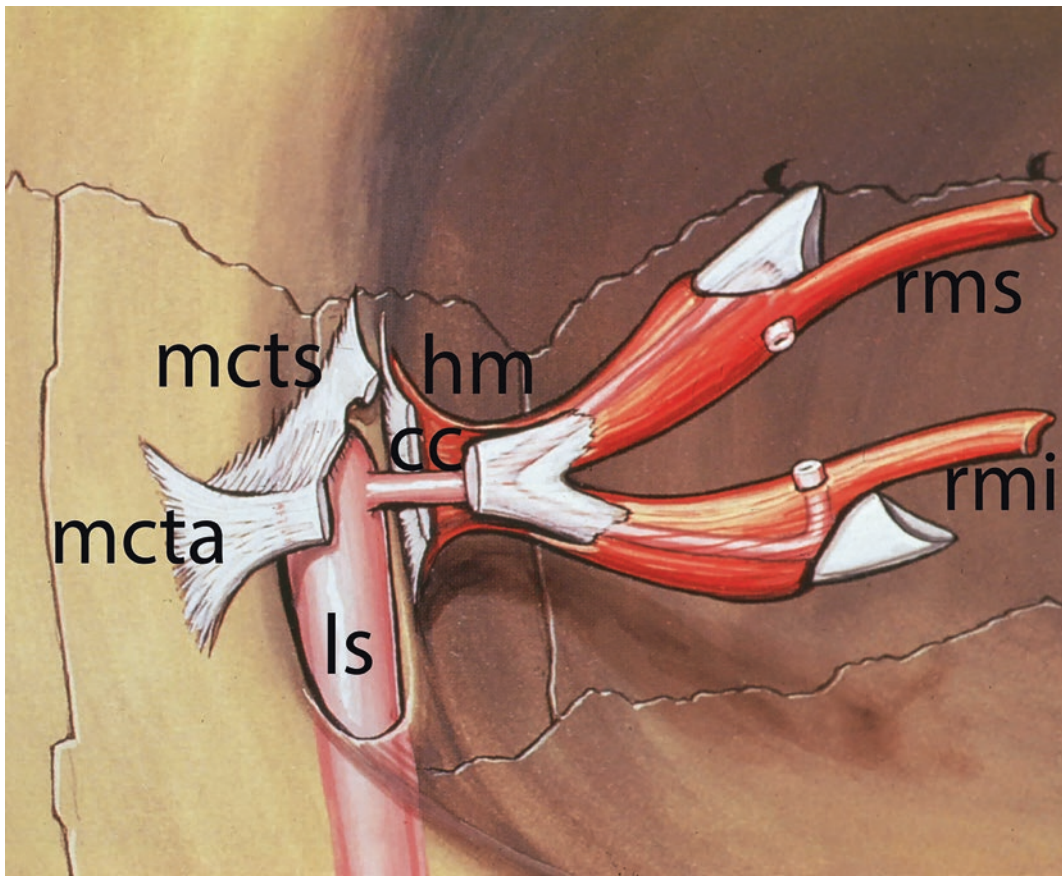

Fig. 1.8 Medial canthal anatomy describing the muscle of Riolan. Lacrimal sac (ls), medial canthal tendon, anterior arm (mcta), medial canthal tendon, superior arm (mcts), superior Riolan muscle (rms), inferior Riolan muscle (rmi), Horner muscle (hm), and common canaliculus (cc)

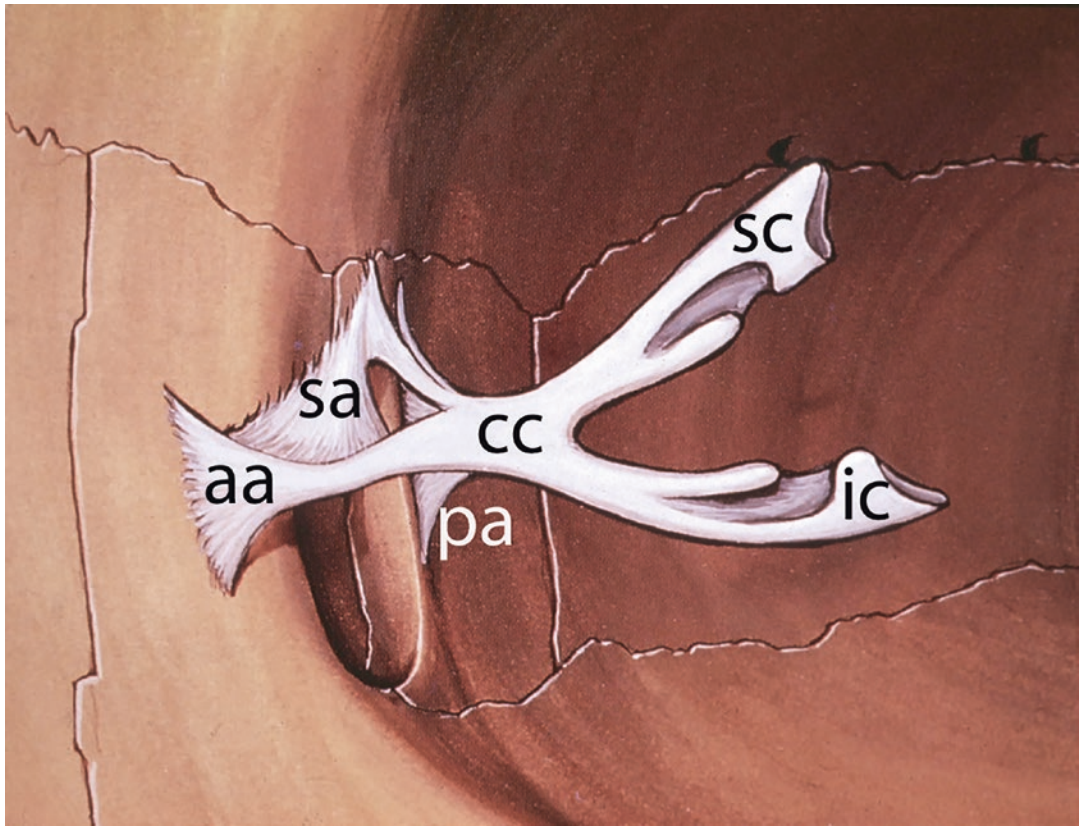

Fig. 1.9 Anatomy of the medial canthal tendon. Medial canthal tendon, anterior arm (aa), medial canthal tendon, superior arm (sa), medial canthal tendon, posterior arm (pa), common canaliculus (cc), superior crus (sc), and inferior crus (ic)

1.5 Orbital Septum

The *orbital septum* is a thin, fibrous, multilayered membrane that begins anatomically at the arcus marginalis along the orbital rims (Fig. 1.10). Distally in the upper eyelid fibers merge into the anterior surface of the levator aponeurosis [29]. The point of insertion usually is about 3–5 mm above the tarsal plate, but it may be as much as 10–15 mm above it [30]. In the lower eyelid, the septum fuses with the capsulopalpebral fascia several millimeters below the tarsus, and the common fascial sheet inserts onto the inferior tarsal edge [31, 32].

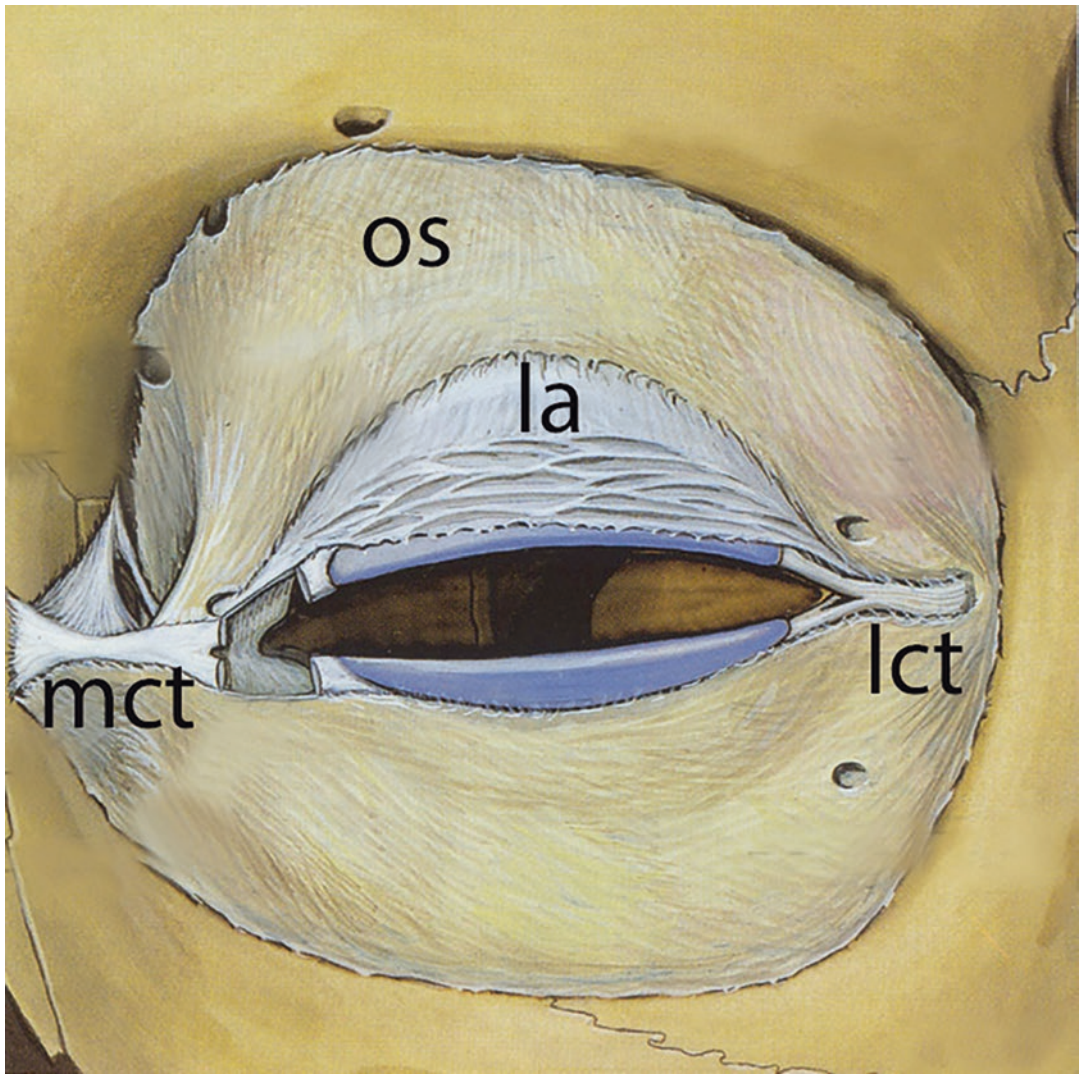

Fig. 1.10 Anatomy of the orbital septum. Medial canthal tendon (mct), lateral canthal tendon (lct), orbital septum (os), and aponeurosis of levator muscle (la)

The preaponeurotic fat pockets in the upper eyelid and the precapsulopalpebral fat pockets in the lower eyelid are anterior extensions of extraconal orbital fat (Fig. 1.11). These are surgically important landmarks and help identify a plane immediately behind the orbital septum and anterior to the major eyelid retractors (levator aponeurosis in the upper lid and capsulopalpebral fascia in the lower lid). In the upper eyelid, two fat pockets are noted: a medial pocket and a central one [33]. Laterally, the lacrimal gland is present but it may be surrounded by a thin layer of fat. In the lower eyelid three fat pockets are noted: medial, central, and lateral [34]. Prolapse of these fat pockets is seen as an aging phenomenon, but also in thyroid eye disease secondary to adipogenesis and proptosis.

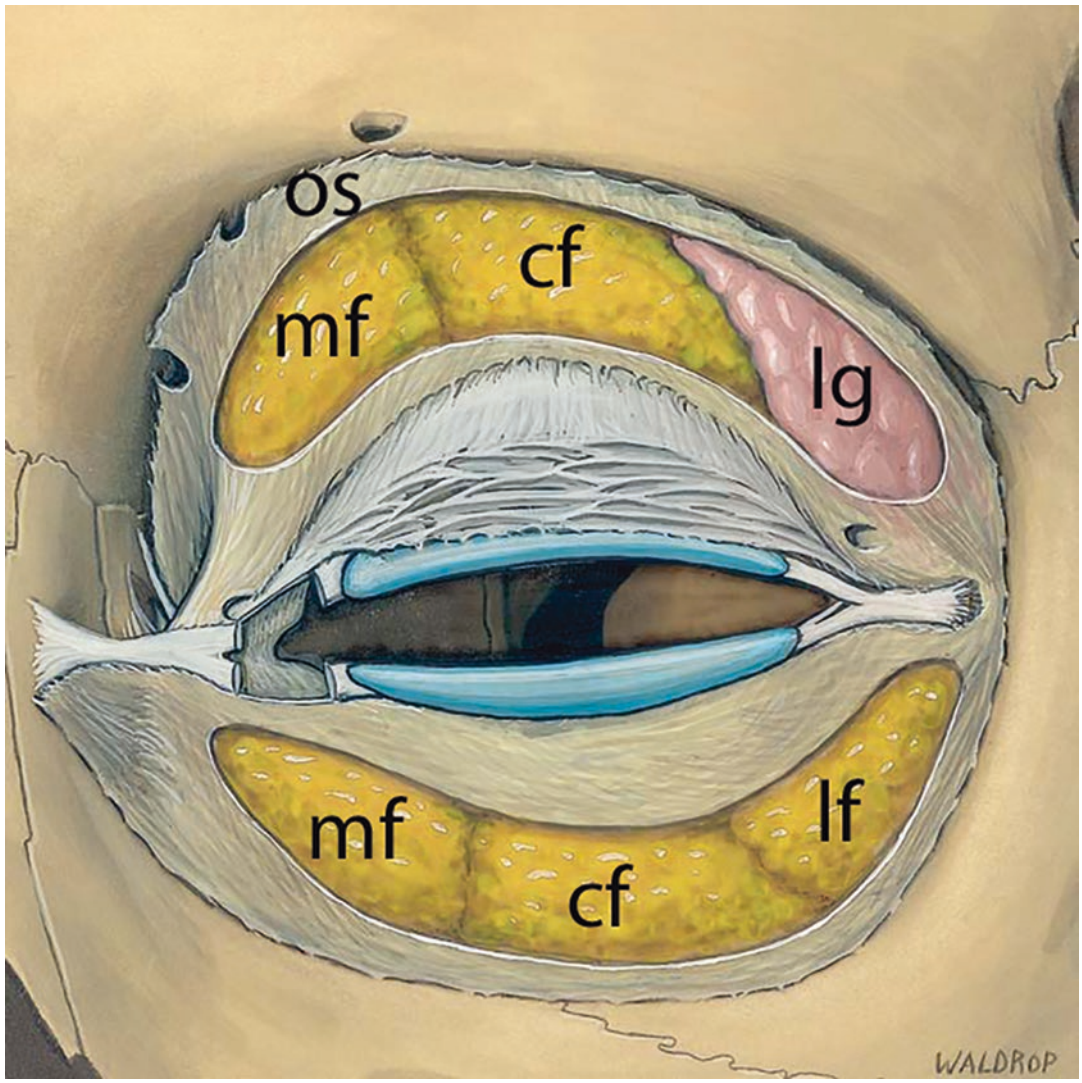

Fig. 1.11 Fat pockets of the upper and lower eyelids. Orbital septum (os), medial fat pocket (mf), central fat pocket (cf); lateral fat pocket (lf), and the lacrimal gland (lg)

1.6 Eyelid Retractors

The retractors of the upper eyelid consist of the levator palpebrae superioris and Müller muscle [35, 36]. The levator palpebrae superioris arises from the lesser sphenoid wing in the orbital apex and runs forward just above the superior rectus muscle. Near the superior orbital rim, a condensation along the muscle sheath attaches medially and laterally to the orbital walls. This is the superior transverse orbital suspensory ligament of Whitnall (Fig. 1.12). The muscle passes into its aponeurosis and continues downward 14–20 mm to its insertion near the marginal tarsal border, about 3–4 mm above the eyelid margin [36, 37]. It also sends delicate slips forward and downward to insert onto the interfascicular septa of the pretarsal orbicularis muscle. These slips maintain the close approximation of the skin, muscle, aponeurosis, and tarsal lamellae, and defines the upper eyelid crease. During eyelid recession procedures, these retractors are recessed upward, usually without a graft. In the lower eyelid, the capsulopalpebral fascia (Fig. 1.12) is a fibrous sheet that arises from Lockwood's ligament and the sheaths around the inferior rectus and inferior oblique muscles [38]. It passes upward and generally fuses with the orbital septum about 4–5 mm below the tarsal plate. From this junction, a common fascial sheet continues upward and inserts onto the lower border of the tarsus.

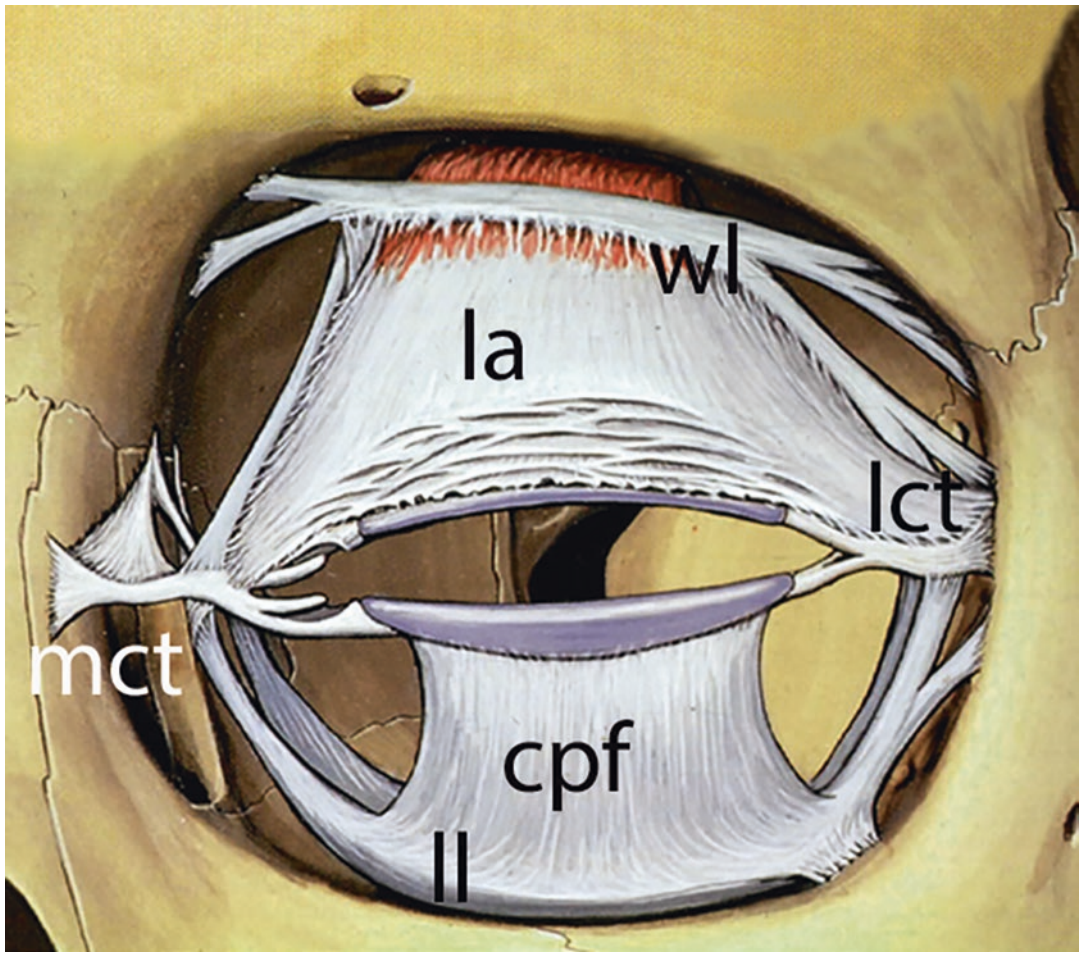

Fig. 1.12 Medial canthal tendon (mct), lateral canthal tendon (lct), levator aponeurosis (la), capsulopalpebral fascia (cpf), Whitnall ligament (wl), and Lockwood ligament (ll)

The eyelids are supported by a complex suspensory system (Fig. 1.13) that supports the canthal angles and redirect vector forces from horizontal in the orbit to vertical in the eyelids [24]. The superior suspensory ligament of Whitnall is a condensation of the levator sheath. It inserts medially and laterally onto the periosteum of the orbital walls. Fine fibers extend from Whitnall ligament to suspend the superior conjunctival fornix. Fibrosis is partially responsible for the eyelid retraction seen in thyroid eye disease. In the lower eyelid, Lockwood ligament forms a fascial condensation involving Tenon capsule, and the conjoined sheaths of the inferior oblique and inferior rectus muscles. The capsulopalpebral fascia extends from this ligament to the inferior tarsus and facilitates retraction of the lower lid in downgaze. In correction of lower lid retraction in thyroid eye disease, this structure is often disinserted or lengthened with an interpositional graft.

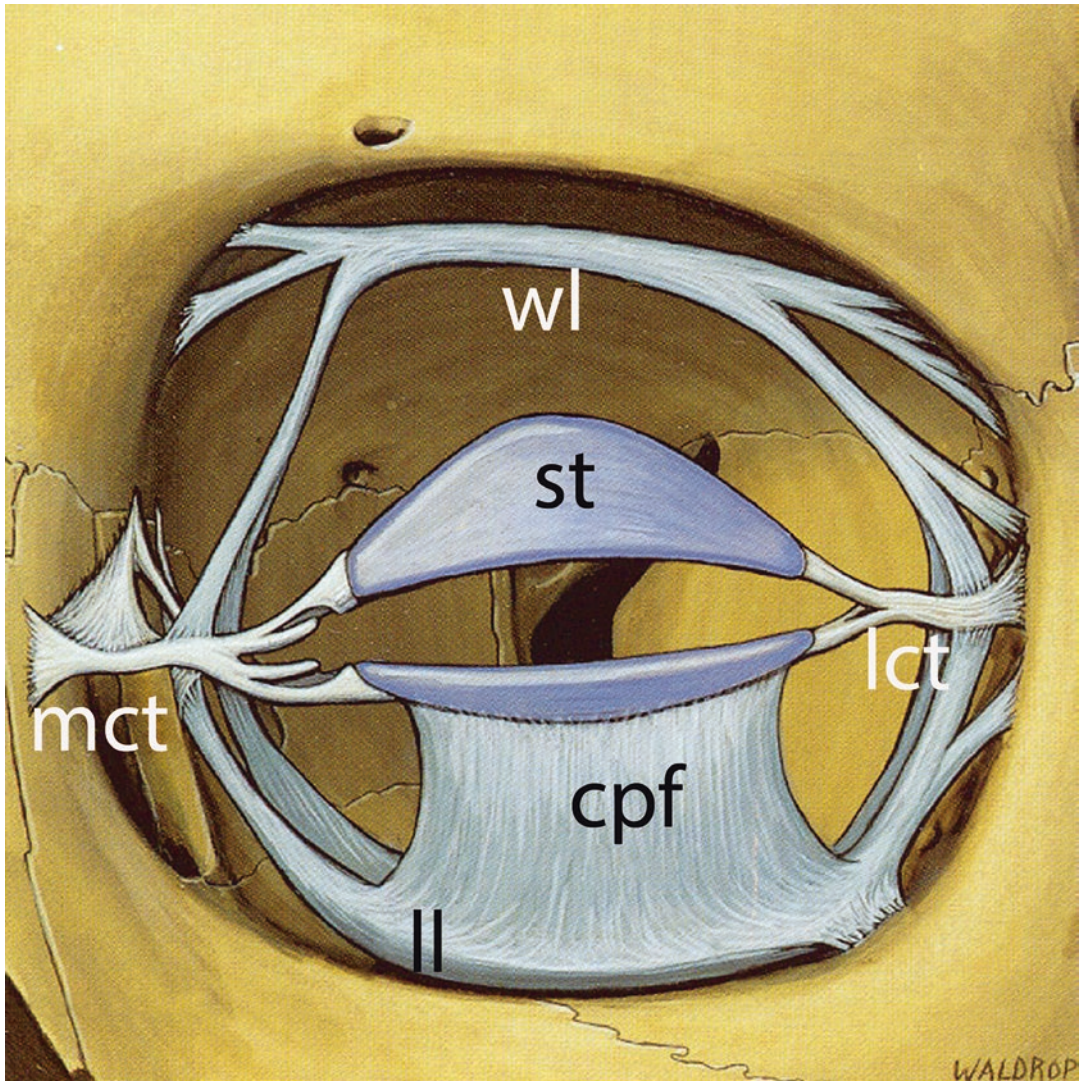

Fig. 1.13 Suspensory system of the eyelids. Whitnall ligament (wl); Lockwood ligament (ll), superior tarsus (st), capsulopalpebral fascia (cpf), medial canthal tendon (mct), and lateral canthal tendon (lct)

1.7 Nerve Supply of the Eyelids

The facial nerve (CN VII) provides motor innervation to the muscles of facial expression. Branches to the periorbital muscles originate in the temporal and zygomatic branches (Fig. 1.14). The temporal branch primarily innervates the frontalis muscle and the upper half of the orbicularis muscle. The zygomatic branch crosses the zygomatic arch and innervates the lower half of the orbicularis muscle. The buccal, mandibular, and cervical branches innervate muscles of the lower face and neck [39].

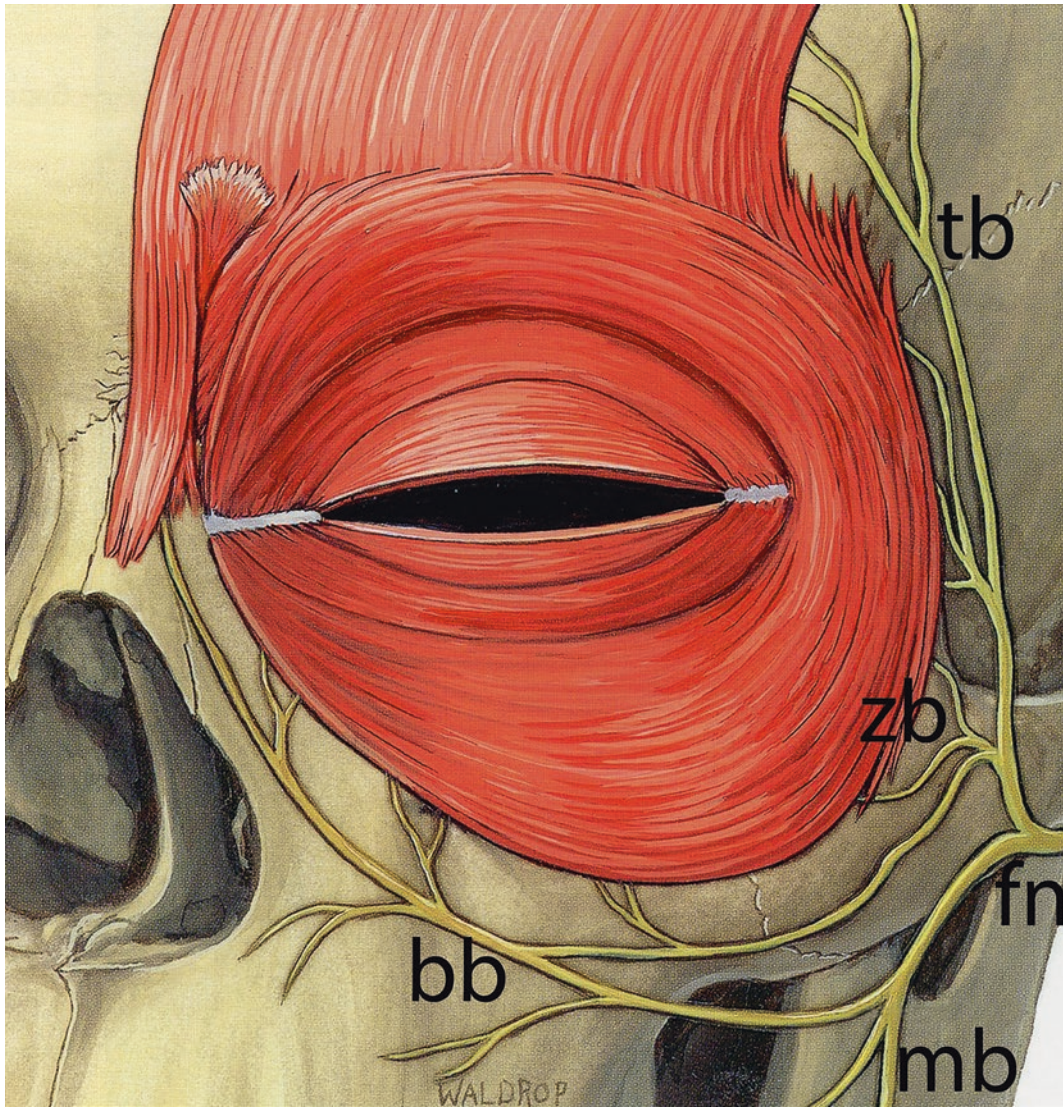

Fig. 1.14 Branches of the Facial nerve. Temporal branch (tb), zygomatic branch (zb), buccal branch (bb), mandibular branch (mb), and facial nerve (fn)

Sensory nerves from the eyelids (Fig. 1.15) derive from the ophthalmic and infraorbital branches of the trigeminal nerve (CN V). These branches pass backward along the orbital walls to the middle cranial fossa. The infraorbital nerve lies within a canal in the orbital floor and is particularly vulnerable to injury during decompression for thyroid eye disease. The zygomaticofacial and zygomaticotemporal nerves pass through the orbital rim and lateral wall where they can be damaged during surgery to advance the rim or decompress the lateral wall.

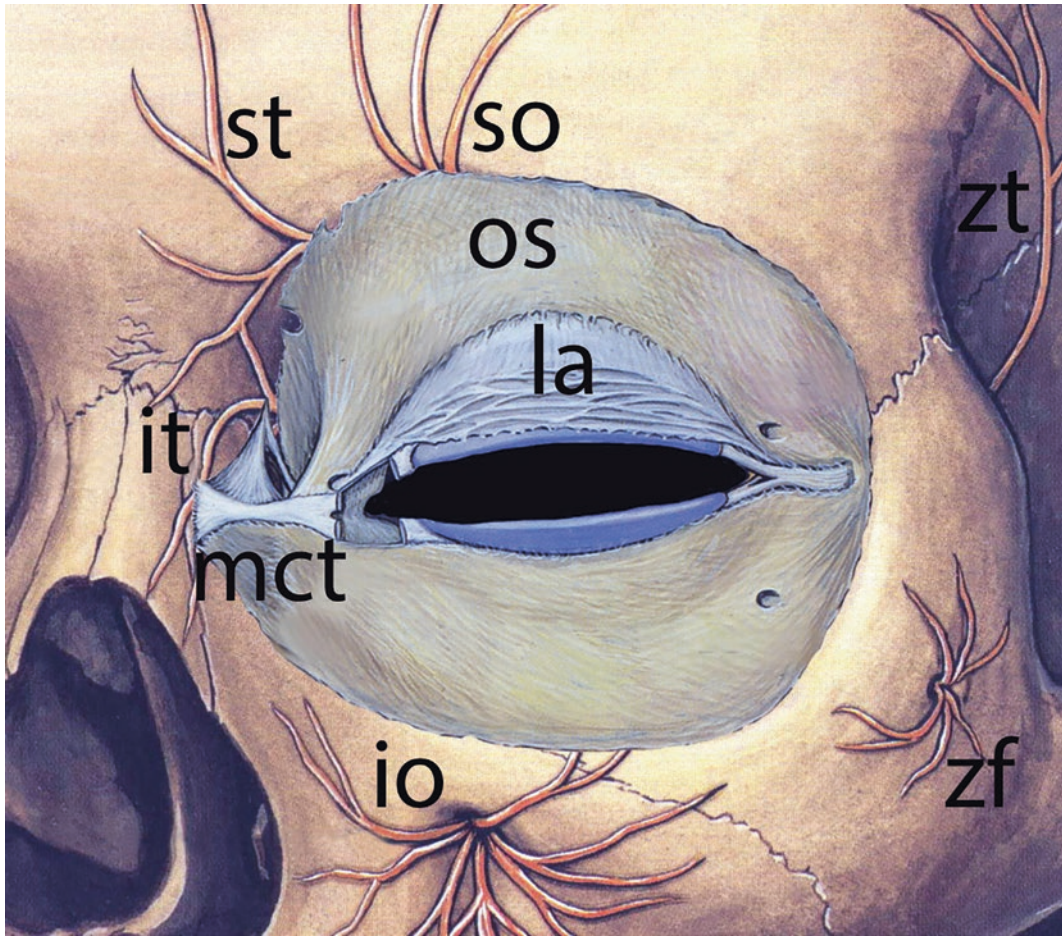

Fig. 1.15 Sensory nerves of the eyelids. Orbital septum (os), supratrochlear nerve (st); *so,* supraorbital nerve (so), levator aponeurosis (la), zygomaticofacial nerve (zf), zygomaticotemporal nerve (zt), infraorbital nerve (io), infratrochlear nerve (it), and medial canthal tendon (mct)

1.8 Vascular Supply of the Eyelids

The posterior eyelid lamellae receive arterial blood through the vascular arcades (Fig. 1.16). In the upper eyelid, a marginal arcade runs about 2 mm from the eyelid margin, and a peripheral arcade extends along the upper border of the tarsus between the levator aponeurosis and Müller muscle. These arcades are supplied medially by the superior medial palpebral vessel from the terminal ophthalmic artery, and laterally by the superior lateral palpebral vessel from the lacrimal artery. They anastomose extensively with the facial arterial system. The lower eyelid arcade receives blood from the medial and lateral inferior palpebral vessels.

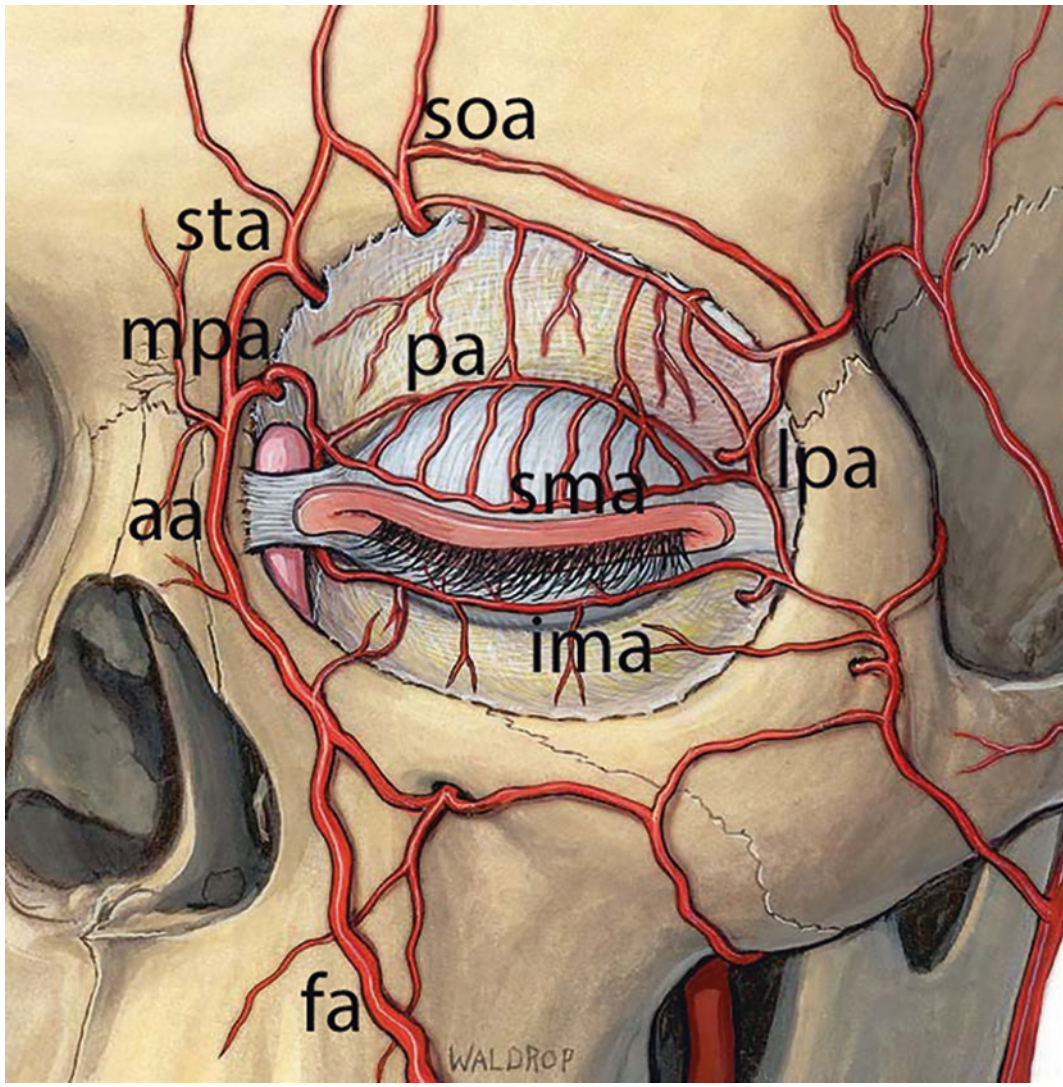

Fig. 1.16 Arterial supply of the Eyelids. Angular artery (aa), medial palpebral artery (mpa), lateral palpebral artery (lpa), supratrochlear artery (sta), supraorbital artery (soa), transverse facial artery (fa), peripheral arcade (pa), superior marginal arcade (sma), and inferior marginal arcade (ima)

The venous drainage system is not as well defined as the arterial system. Drainage is mainly into several large vessels of the facial system and drains both anteriorly into the facial veins, and posteriorly to the cavernous sinus (Fig. 1.17).

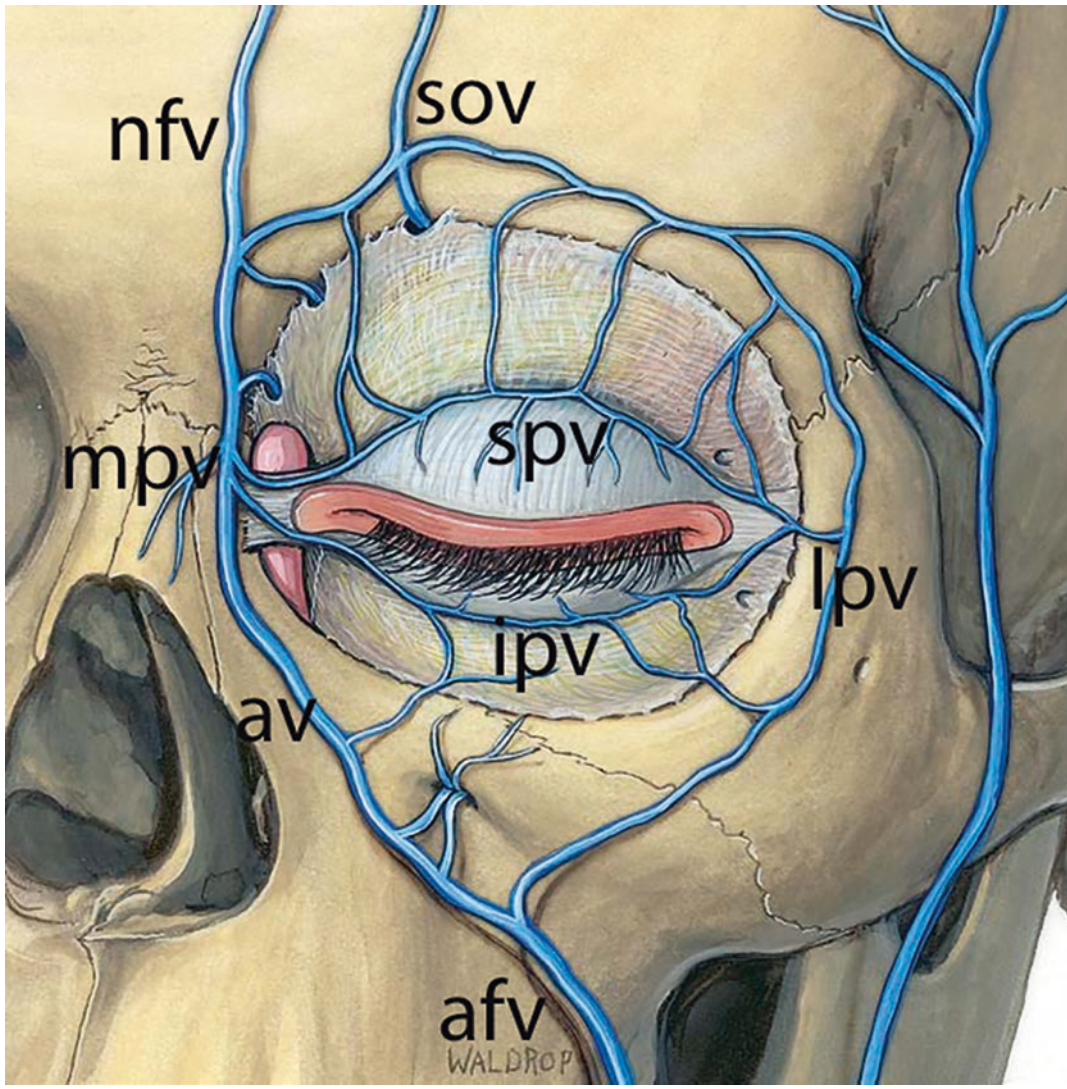

Fig. 1.17 Venous drainage of the Eyelids. Medial palpebral vein (mpv), lateral palpebral vein (lpv), superior palpebral vein (spv), inferior palpebral vein (ipv), nasofrontal vein (nfv), supraorbital vein (sov), angular vein (av), and anterior facial vein (afv)

1.9 Lymphatic Drainage of the Eyelids

The lymphatic drainage from the eyelids is restricted to the region anterior to the orbital septum. Traditional teaching is that lymphatic flow from the lateral two-thirds of the upper eyelid and the lateral one-third of the lower eyelid drain laterally into the deep and superficial parotid nodes, and flow from the medial one-third of the upper eyelid and the medial two-thirds of the lower eyelid drains inferiorly into the submandibular and anterior cervical nodes (Fig. 1.18). However, recent studies have shown a more diffuse drainage from all areas of the eyelids into the parotid nodes [40].

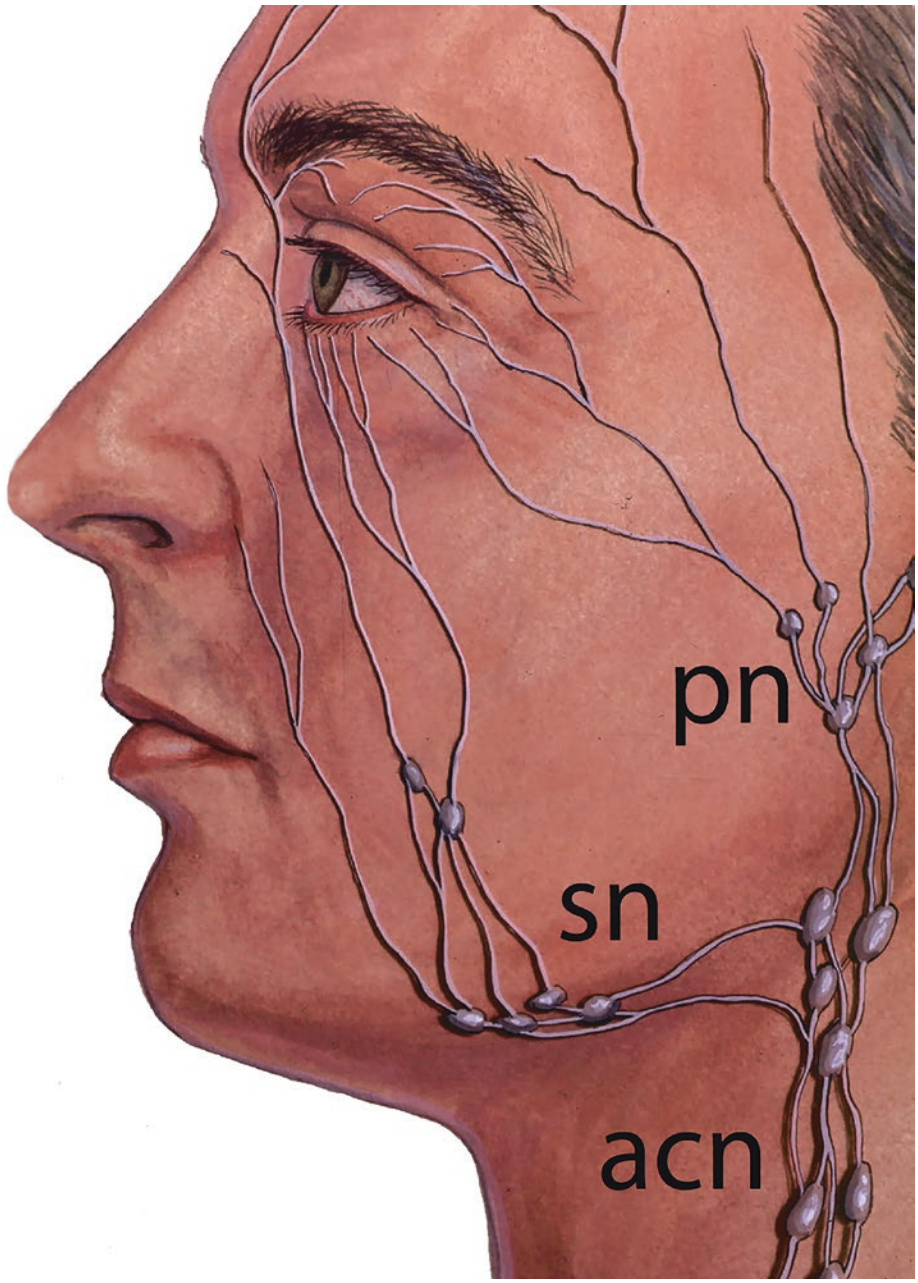

Fig. 1.18 Lymphatic drainage of the Eyelids. Parotid nodes (pn), submandibular nodes (sn), and anterior cervical nodes (acn)

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