

Endocrine Ophthalmopathy

Surgical Treatment

Transpalpebral Decompression by Removal
of Intraorbital Fat

by Neven Olivari

with contributions by
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KADEN Verlag Heidelberg

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Language Editing: Brian D. G. Morgan, MB FRCS, London

Die Deutsche Bibliothek – CIP-Einheitsaufnahme

Olivari, Neven :

Endocrine ophthalmopathy : surgical treatment : transpalpebral decompression by removal of intraorbital fat /
by Neven Olivari. With contrib. by E. Eder... – Heidelberg : Kaden, 2001

ISBN 3-922777-35-x

© 2001 Kaden Verlag, Heidelberg, Germany
Typesetting: Ch. Molter, Kaden Verlag, Heidelberg, Germany
Printing: Druckhaus Darmstadt, Darmstadt, Germany
Binding: Buchbinderei Schaumann, Darmstadt, Germany

ISBN 3-922777-35-x

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*This book is dedicated to
my wife Anna-Maria
and my sons Alexander and Nicolas*

Foreword

Endocrine ophthalmopathy, or Graves disease, is a condition which, over the years, has repeatedly taxed the ingenuity of ophthalmic surgeons and a small group of plastic surgeons interested in this condition. Initially the operations were technically relatively minor but with the advent of craniofacial surgery more major procedures were developed and performed. Unfortunately, these led to only a minimal improvement in results and sometimes the complications, such as diplopia, were significant and frequent. The real problem in terms of treatment was the lack of an adequate analysis of the problem. This has now been greatly helped by more sophisticated imaging techniques such as MRI scanning together with the aid of three-dimensional CT scanning techniques. It has now become obvious that it is the increase in conal fat which seems to be the most significant factor in the causation of this distressing deformity.

The method described by Neven Olivari, and so beautifully presented in this book, is logical. Unfortunately, when it is presented or demonstrated by Neven Olivari, it seems simple to execute. However, at that point we are watching or listening to a master surgeon with a huge amount of experience in all aspects of plastic surgery but particularly in the orbit, demon-

strating a technique which he has pioneered and developed over many years. To many, the method and its execution has been something of a mystery. All has now been revealed in this very clear and well produced treatise. This is undoubtedly a giant step forward in the treatment of this condition which affects so many of our patients. The book must, however, be read extremely carefully and even after having done this I would advise for those who are inexperienced to practice on a cadaver, if possible, using the textbook like a dissection manual. Those who are experienced in orbital surgery, of course, will not require this but they must follow the step-by-step instructions provided by the author. In this way good results will be obtained and complications avoided.

We must sincerely thank Neven Olivari for producing this publication. He has done a great service to orbital surgery and to the unfortunate patients who suffer from the problem of endocrine ophthalmopathy. I am sure that this will be instrumental in many patients retaining their sight who otherwise would not have done so. This may be the greatest complement that can be applied to this account of his lifelong study of this distressing problem.

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Foreword

Endocrine orbitopathy represents one of the main challenges in the therapy of Graves' disease, even today. Although medicational and definitive treatment of Graves' hyperthyreosis by means of radioactive iodine or surgery is effective and can be carried out with a calculable risk, the benefit for the manifestation and long-term development of endocrine orbitopathy has remained uncalculable to a great extent. Improved diagnosis using ophthalmological examination, determination of the activity score, and also imaging procedures has brought progress in the estimation of illness during the last few years: the severe forms of endocrine orbitopathy, with their considerable physical and psychological impairments, remain a serious challenge nonetheless. The progress in surgical treatment of endocrine orbitopathy represent a milestone here. A particularly special aspect is the transpalpebral decompression by means of resection of the fatty body. Improved and optimized short- and long-term post-operative care in prospectively structured investiga-

tions have decisively aided many patients. The cosmetic results, so important for women in their social surroundings, are surprisingly favourable. Professor Olivari here summarizes his many years' experience in particular of surgical treatment of endocrine orbitopathy, and in so doing has created a standard work for all those interested in Graves' disease. The superb didactic structure and the clear illustrations explain many connections much more quickly than ever so many words would do.

I would like to congratulate the author and the publisher on the appearance of this extensive work, which can be recommended without reserve, especially to those colleagues working in this area.

Prof. Dr. med. Klaus Mann

Director of the Endocrinological Department
University Clinic Essen

Preface

The fate of patients suffering from Endocrine Ophthalmopathy (EO) is not an easy one. The basic illness, that is immune hyperthyroidism, can be healed in 95% of cases by means of either surgery (thyroidectomy), thyreostatic medication, or radioactive iodine therapy. The success of therapy for EO is modest, however. Conservative therapy in the acute stages improves the changes in the soft tissue area of the lids, but has hardly any influence upon the protrusion of the eyeball, visual acuity, and optic motility. According to optimistic estimates, exophthalmos reduces spontaneously in about 10% of cases. The rate of remission is probably around 5%, but only in the first 6–12 months. After two years at the latest, the protrusion is stable and cannot be influenced by medication.

EO is a severe blow for many of the patients. They are affected not only by reduced visual acuity, double vision, and other typical symptoms of EO, but also the protruding eyeballs, often seems to change the facial expression quite dramatically. The eyes are the centre of the face. Exophthalmos changes the entire face, indeed, frequently the entire personality. In a later phase, patients show a certain helplessness. Once attractive, outgoing persons are shy and unsure of themselves. Such a patient develops inferiority complexes and depressions; social contacts are drastically reduced. The patient visits his or her endocrinologist, ophthalmologist, and nuclear medicine specialist regularly and is constantly asking whether an operation would help.

The answer of the physicians is objective. Bony operative decompression is indicated only in cases of threatening loss of vision, but not in cases of protrusion without visual problems, or diplopia and certainly not in cases of aesthetic problems. The operation is complex and there are many complications. As only about 3–5% of patients have problems with reduced visual acuity, more than 95% of patients are doomed to live with exophthalmos.

There are few non-malignant illnesses with a lower rate of successful treatment than EO. Probably some 160,000 patients live with this problem in Germany alone.

In the summer of 1985, a friend who is an internist and endocrinologist, Dr. Mies from Cologne, asked me whether I could help his wife, who was suffering from an endocrine ophthalmopathy. Although at that time I already had some 20 years' experience in lid surgery, I replied that I had never carried out a bony decom-

pression, and I recommended a clinic to him in which such operations were performed. Dr. Mies was of the opinion that he had done his homework, and refused an ossary decompression for his wife. However, he requested that I treat his wife from the aesthetic aspects, as she suffered greatly with her appearance.

It was clear to me that the enormous swelling of the eyelids could be corrected by removing fatty tissue. I performed a classic plastic surgical blepharoplasty. Upon opening the orbital septum, a large quantity of fat prolapsed outward under pressure. I removed as much as possible from the upper and lower lids, but also from the deeper regions of the orbits.

To my surprise, not only did the patient's aesthetic situation improve, but the Hertel values were reduced.

The well-known nuclear medicine specialist Dr. Mödder, also from Cologne, heard of this operation and asked me to operate on some of his many patients suffering from EO, who had undergone conservative treatment for years without benefit. He himself has written numerous books and many papers on thyroid illnesses.

I let myself be introduced to the complexities of EO by him, in the course of long, friendly discussions. After studying the literature concerning EO, anatomy, and the operative procedures hitherto applied, I decided to perform individual operations.

During the years 1985–87, a period of three years, only 8 patients and 15 orbits were operated on. All of these patients had undergone conservative treatment for a period of years with no success. All 15 operations were successful.

From the beginning, the following examinations were essential prior to advising an operation:

1. Metabolic condition, the patient must be euthyroid
2. Ophthalmologic examination
3. CT or MRI of the orbits to determine the degree of involvement of the extraocular muscles.

From the second patient onward the volume of fat removed was measured exactly and all data were stored. The patients were examined postoperatively by us as well as by ophthalmologists and endocrinologists or nuclear medicine specialists.

Orbits were operated on in several cadavers, in order to attain more accuracy in operating.

I would like to thank Dr. Mies and Dr. Mödder for moral support. I would also like to express my gratitude to Dr. Neubauer, the recognized ophthalmo-

logic surgeon from Cologne, for his support. I presented my results to him at the start of 1991 and he found the method to be worthy of mention in the book “Surgical Ophthalmology”, of which he was co-author.

I performed the first one hundred orbital operations by myself; thereafter I delegated the operation more and more to my colleagues.

The first short publication was 1988 [106]. The results following 147 operations were published in *Plastic and Reconstructive Surgery* [107].

For this publication I received the “Award for the Best Clinical Paper 1991” from the American Association of Maxillofacial Surgeons.

I would like particularly to thank my photographer of many years’ collaboration, Mrs. Inge Goldberg, for the excellent photographs of operations and patients, as well as my colleagues E. Eder, D. Richter, G. Noever, B. Stark, and G. Deutsch for their clinical and statistical support during the work on this book.

My wife Anna-Maria patiently kept writing new, corrected versions on the computer.

I was able to find the necessary peace and quiet needed for the final version of the book on the balcony of the Club Coronado, belonging to the Schütte family on the island of Fuerteventura.

I would like to express my sincere gratitude to Dr. Brian Morgan, who reviewed the text with care and friendship to make this a well-edited book.

Prof. Dr. med. Neven Olivari

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1. The History of Endocrine Ophthalmopathy

In the English-speaking world (USA, UK, etc.) immune hyperthyroidism is called “Graves’ Disease”, whilst in parts of continental Europe it is called “Basedow’s Disease” (in German: “Basedow’sche Erkrankung”).

Robert James Graves was one of the most ambiguous personalities in Irish medical history. He was born in 1796, the seventh of ten children. His father, as also his maternal grandfather, was a professor of theology at Dublin University. He enjoyed a good school education and attended the University at which his father taught. After completing his medical studies, Graves travelled throughout Europe, increasing his medical knowledge at London, Göttingen, Berlin, Vienna, Copenhagen, and Edinburgh. Thereafter he returned to Dublin and began his career as a physician. Owing to his numerous foreign university visits, he possessed an excellent education and had personal contact with several well-known European physicians. In Dublin he rapidly acquired a reputation as a lecturer and clinician, but also as a writer. An inspired speaker, for years he held lectures at Meath Hospital, which were regarded virtually as social events.

In one of these lectures, held in 1834, Graves described 3 patients, who all showed similar symptoms:

1. tachycardia
2. enlargement of the thyroid gland.

His colleague William Stoke mentioned a fourth patient with exophthalmos at the conclusion of the lecture.

Graves published this report in 1835 in the *London Medical and Surgical Journal* under the title “Lecture N° 12 of the Clinical Lectures” [43]. The journal was not well known and the title unfortunate. Even in Great Britain, this report was forgotten after only a few years [Fig. 1.1].

In 1843 Graves published his medical textbook “A System of Clinical Medicine”, which enjoyed great success, being translated into German, French, and Italian.

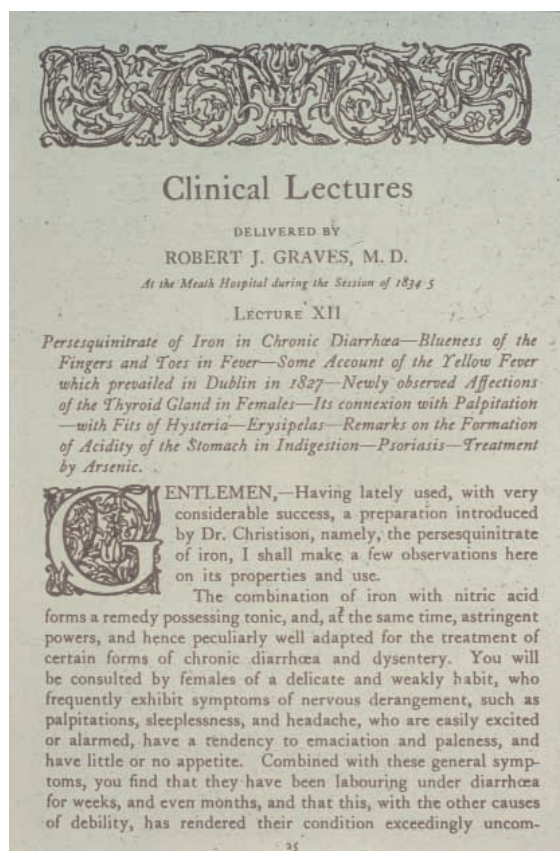


Fig. 1.1 Original publication by Robert J. Graves, 1835. He discovered the connection between tachycardia, goitre, and exophthalmos. This work was forgotten for years.

Nobody overlooked the publication by the German physician Karl von Basedow from Merseburg an der Saale. Basedow, three years younger than Graves, was born in 1799 as the son of the governmental head of Dessau. He studied in Paris and Halle, where he obtained his doctorate of medicine and surgery in 1821.

In the year 1840 he published a report on four cases of exophthalmos, goitre, and tachycardia (Merseburger Triad) [12]. Basedow had made a special study of the symptoms of exophthalmos. He recognized that the eyes themselves are not swollen, but rather the tissue behind them. He applied iodine and leeches by way of therapy [Fig. 1.2].

Basedow's description of the illness became popular in Europe, and, as it was published three years before Graves' textbook appeared, the name "Basedow's Disease" became established there.

Only the French scientist and admirer of Graves, Armand Trousseau, knew that Graves had already recognized the connexion between the symptoms and had described this. Trousseau reported this fact to the Medical Academy in Paris in 1862; this was ignored in

continental Europe, however.

A further fact was not known to Trousseau. In 1825 the son of the physician Chaleb Parry (1755–1822) published the reports of his father posthumously. Chaleb Parry was a practising physician in the elegant English town of Bath. He had described the connexion of tachycardia and goitre, with occasional exophthalmos, as an independent illness. The publication was, however, ignored for the most part.

Neither Graves nor Basedow knew of the causes of the disease described by them. 150 years later, we are not very much further.

It is noteworthy that Bartisch from Königsbrück had published a lengthy book in 1583 on ophthalmology [9]. One illustration clearly shows a patient suffering from exophthalmos. Naturally this was not correctly interpreted as being connected with the thyroid (Figs. 1.3, 1.4).

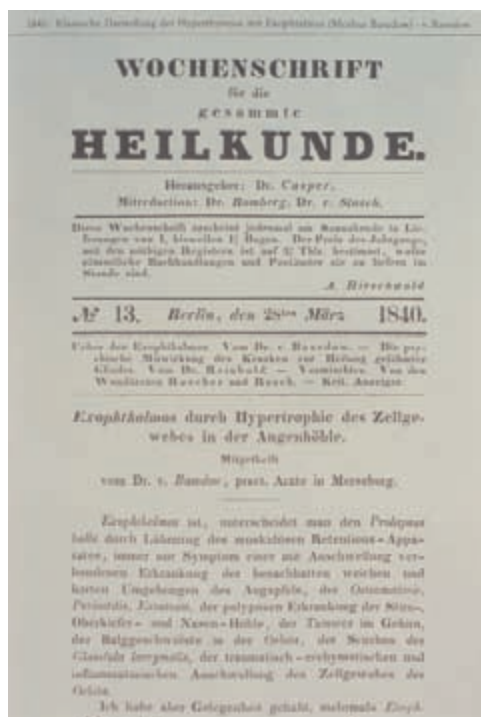


Fig. 1.2 In 1840 Basedow, without knowing about Graves' discovery, published the so-called Merseburger Triad: tachycardia, goitre, and exophthalmos. He established that the globe was not enlarged, but rather the tissue behind the eye.



Fig. 1.3 Bartisch from Königsbrück published an extensive book on ophthalmology in 1583.



Fig. 1.4 In Bartisch's book a patient is shown with exophthalmos. Naturally the eye condition was not associated with the thyroid function.

2. Anatomy

The eye lies in the orbit, which is formed by the skull. The bulbus oculi (eyeball) is covered in the frontal area by the eyelids, and is otherwise embedded in the soft orbital fatty tissue. The eye muscles end with their tendons at the bulbus oculi. Their fibres converge in the rear part of the orbits, thus forming a funnel, called the anulus tendineus communis (Zinn's ring, tendinous ring), into which the optic nerve, originating from the eyeball, leads.

Nerves and vessels enter the orbit to supply the structures located there – the globe, the optic nerve, the exterior eye muscles, and the lacrimal gland, which lies on the globe in the lateral part of the orbital roof (Figs. 2.2, 2.6).

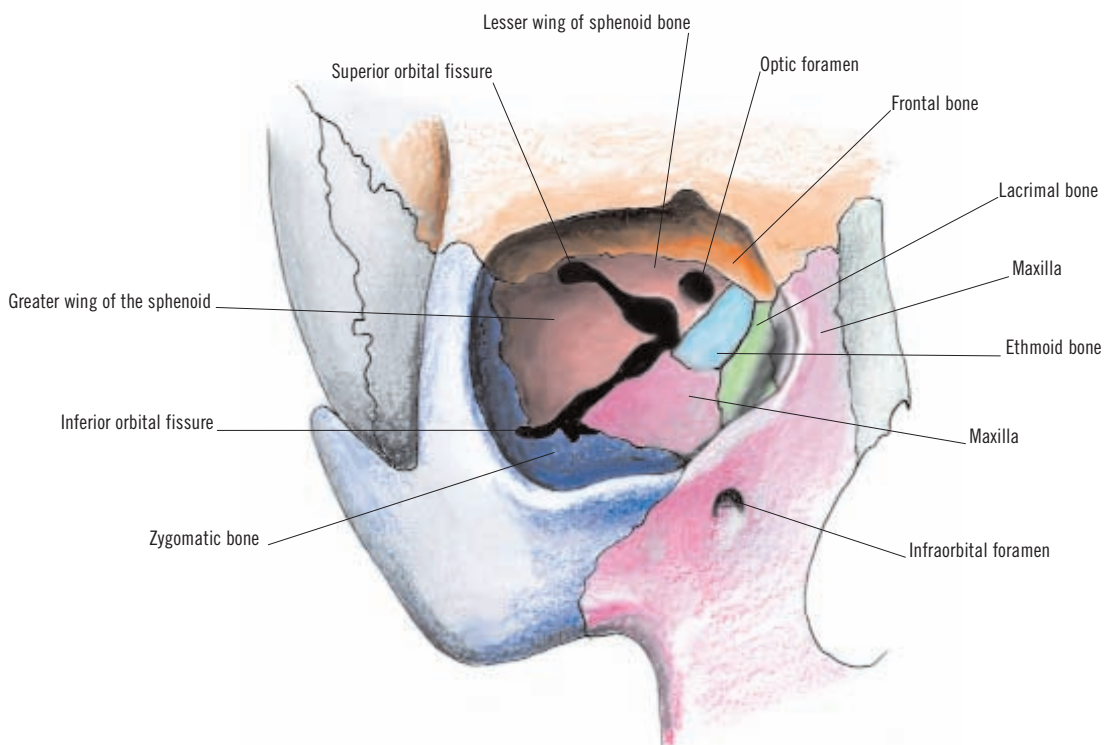


Fig. 2.1 The bony orbit

2.1 The Structure of the Bony Orbit

The bony orbit has the form of a funnel, open towards the front. It is limited nasally by the orbital plate of the ethmoid bone, the frontal process of the maxilla, the lacrimal bone, and the orbital process of the palatine bone. The orbital floor is formed by the maxilla, the zygomatic bone, and the orbital process of the palatine bone. The orbital roof consists of the orbital part of the frontal bone and the lesser wing of the sphenoid. Laterally the orbit is limited by the zygomatic bone, the lesser wing of the sphenoid, and the zygomatic process of the frontal bone (Fig. 2.1).

2.2 The Extraocular Muscles

The extraocular muscles have their common origin in the depths of the orbits, in front of the optic foramen, entering from the tendinous ring (Zinn's ring). They enclose the optic nerve. The four straight eye muscles (Superior rectus, inferior rectus, medial rectus, and lateral rectus) run along the upper, lower, medial, and lateral walls of the orbit to the bulbus oculi, and radiate into the sclera. The two obliquely positioned eye muscles, the superior oblique and the inferior oblique, run from the front medially to the back laterally along the globe, and attach to this temporarily behind the coronal equator of the eye (Fig. 2.2, 2.3).

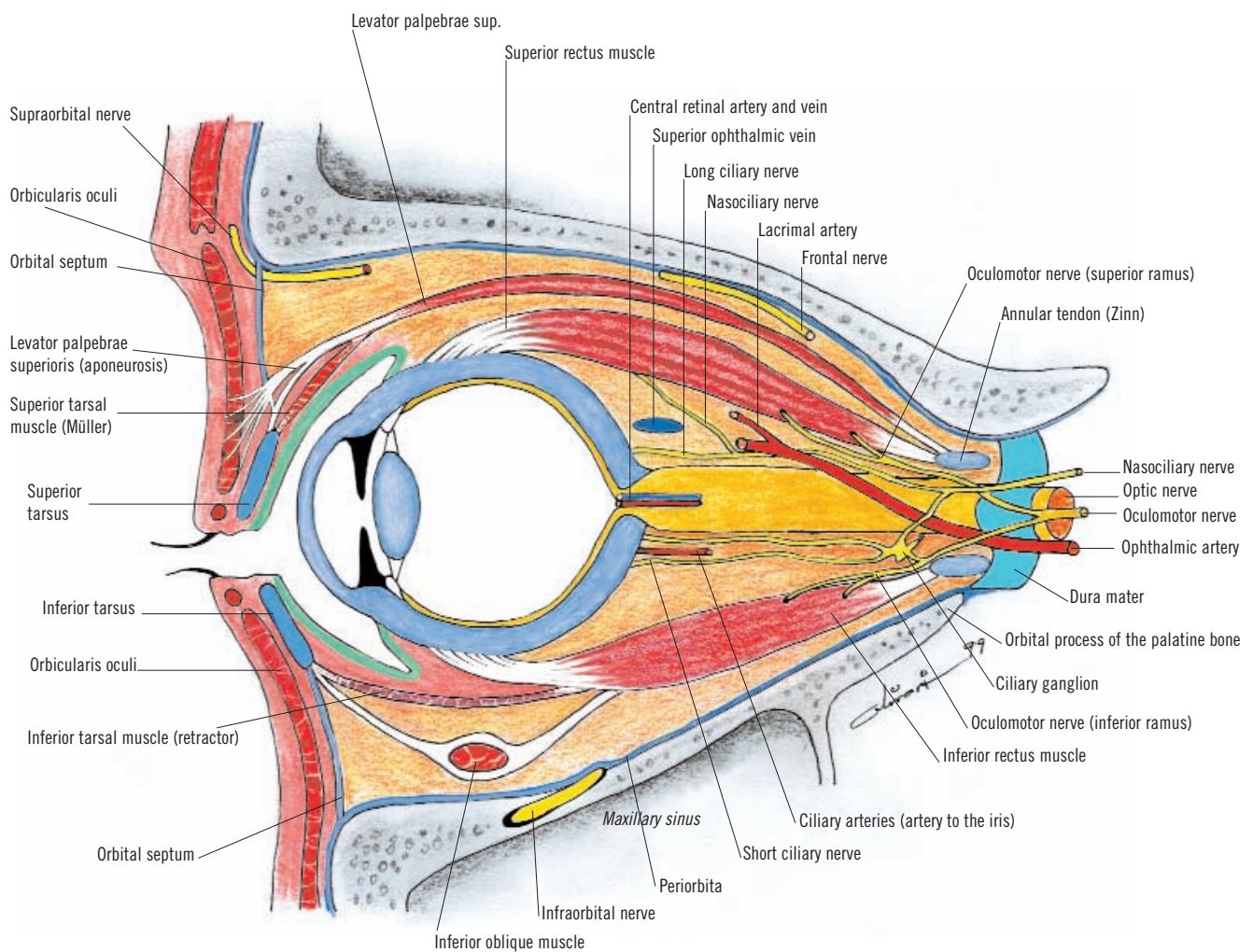


Fig. 2.2 Sagittal section through the orbit.

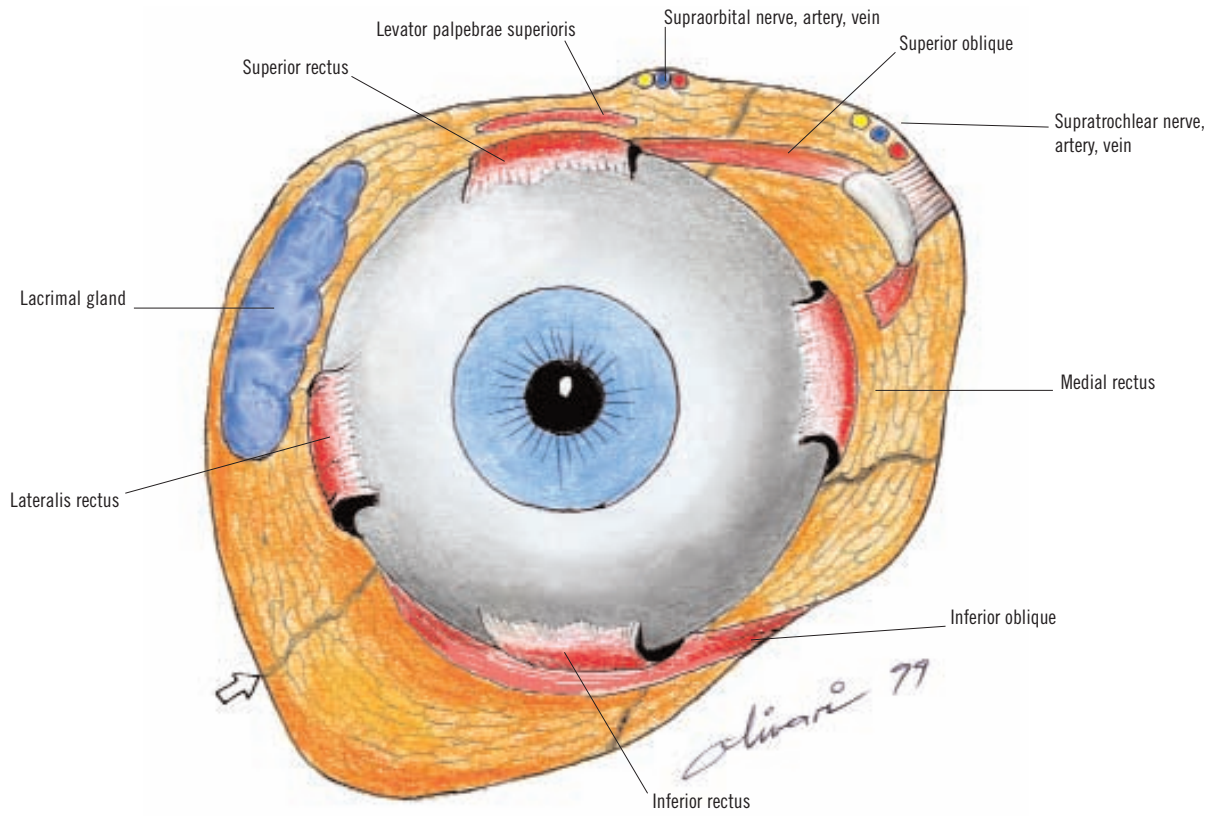


Fig. 2.3 Anatomy of the anterior part of the orbit (coronal section). The fat compartments and the septi between the fatty portions are clearly visible.

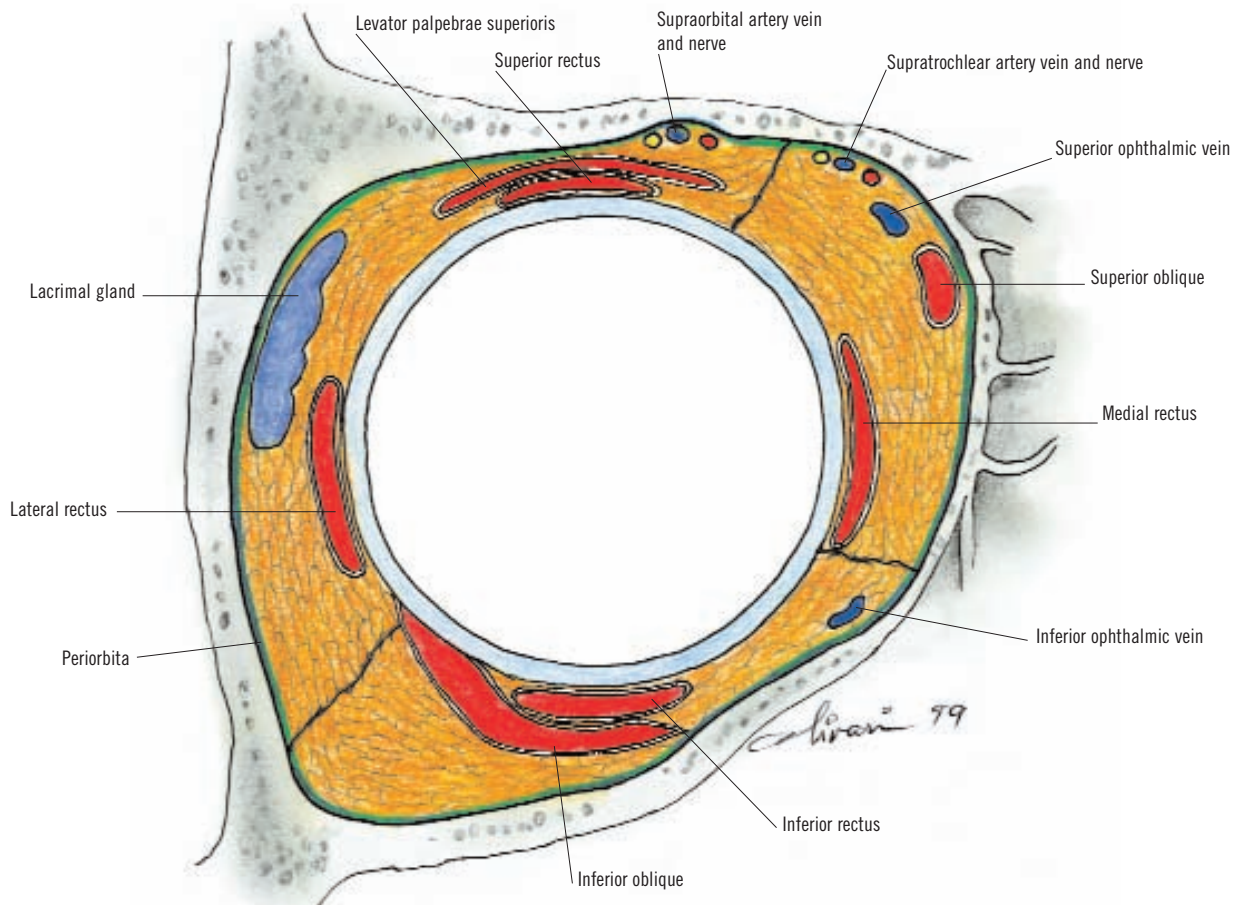


Fig. 2.4 Coronal section in the middle of the globe.

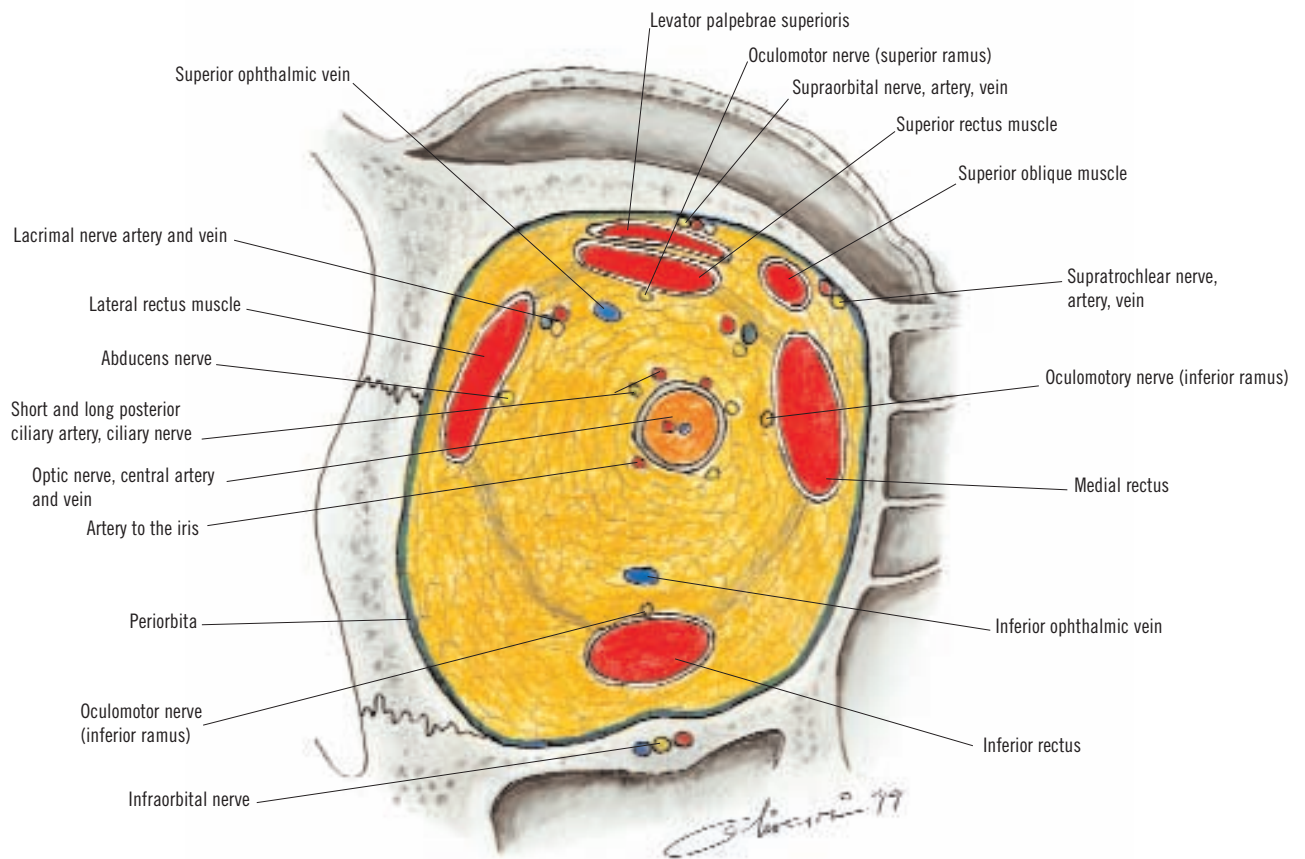


Fig. 2.5 Anatomy of the retrobulbar space. Coronal section 0.5 cm dorsal from the globe.

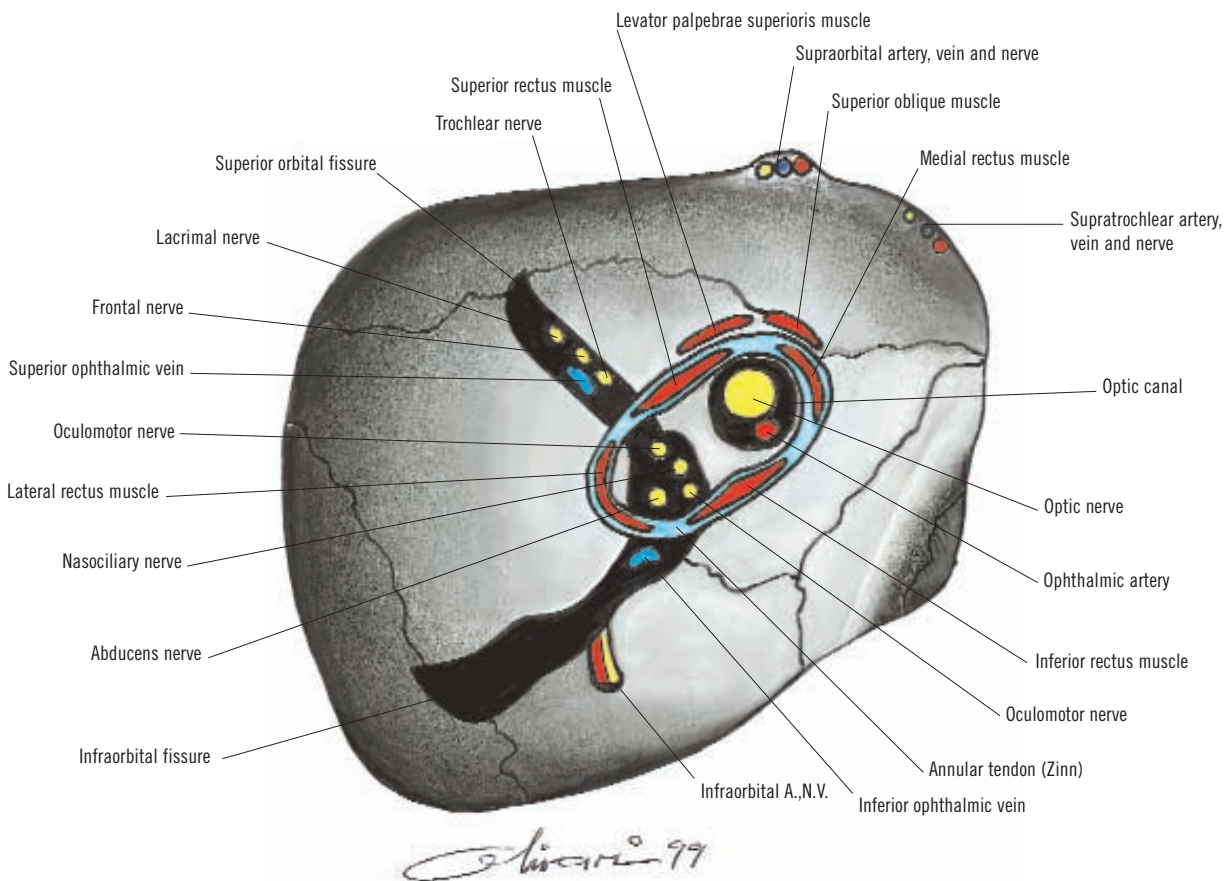


Fig. 2.6 Topographical view of the apex of the orbit (coronal section). The optic nerve, ophthalmic artery, oculomotor nerves, abducens nerve, and the nasociliary nerve lie within the annular tendon (Zinn's ring). Other important nerves and vessels enter through the superior and inferior orbital fissures.

2.3 The Innervation of the Extraocular Muscles

The extraocular muscles are innervated by three cranial nerves, the oculomotor nerve, the abducent nerve, and the trochlear nerve.

The oculomotor nerve (IIIrd cranial nerve) contains somatic and autonomic fibres. It innervates the medial rectus muscle, the inferior rectus, the superior rectus, the inferior oblique and the levator palpebrae superioris. The abducent nerve (VIth cranial nerve) is a somatic nerve, which supplies the lateral muscle. The trochlear nerve (IVth cranial nerve) is also a purely somatic nerve, innervating the superior oblique muscle.

All of the above-mentioned nerves enter the orbit via the superior orbital fissure. There the oculomotor nerve divides into two branches; the inferior branch (Inferior ramus) supplies the inferior rectus, the medial rectus, and the inferior oblique; the superior branch supplies the superior rectus and the levator palpebrae superioris. The oculomotor nerve innervates the interior eye muscles with its autonomic portion.

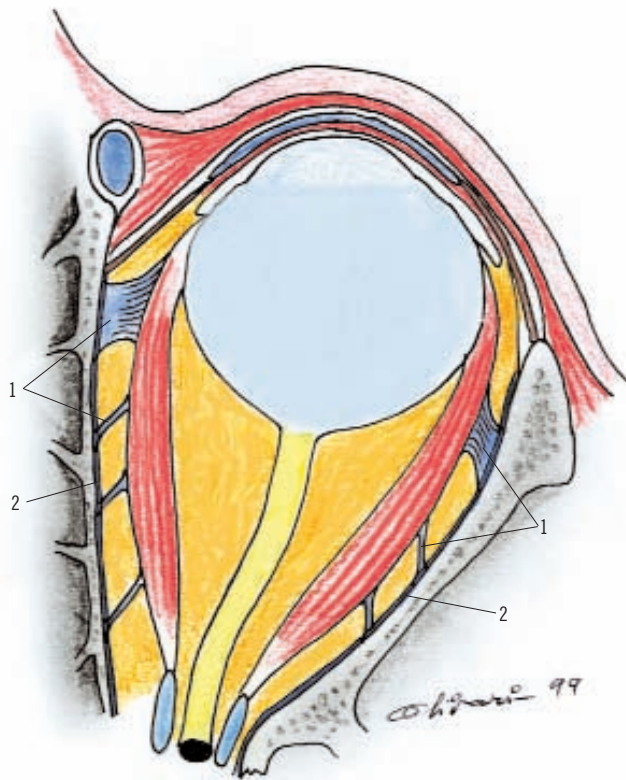


Fig. 2.7 Horizontal section through the middle of the right orbit. The medial rectus and the lateral rectus are attached by fine thread ligaments (1) to the periorbita. In transpalpebral decompression with fat removal the muscles remain in their natural position.

The branches of the IIIrd, IVth, and VIth cranial nerves are inserted into the muscles on the inner side of the cone. This has a practical consequence: when removing fat, there is hardly any danger of injury.

2.4 Vascular Supply of the Orbits

The most important artery of the orbit is the ophthalmic artery, which originates from the internal carotid artery (A. carotis interna). It runs beneath the optic nerve through the optic canal. Within the orbit, it runs within the tendinous ring and describes a helix around the optic nerve. It turns lateral to and then above and finally medial to the optic nerve. In this area the lacrimal artery branches off, running along the upper edge of the lateral rectus to the lacrimal gland and the lateral corner of the eye. In the further course of the ophthalmic artery the ciliary artery and the central retinal artery branch off, which are important for the supply of the globe (Fig. 2.2).

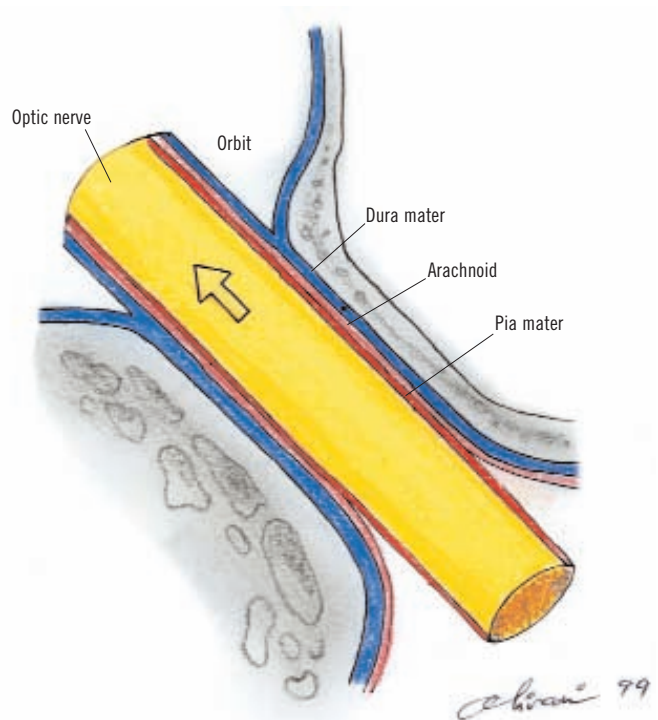


Fig. 2.8 Anatomy of the optic canal. The optic nerve is surrounded by the dura mater. Every pull on the optic nerve and thus on the dura mater in a ventral direction in exophthalmos causes retrobulbar pressure ('burning') and headaches.



Fig. 2.9 a,b View of the orbit from above following removal of the orbital ceiling.

F = frontal nerve

SO = supraorbital nerve

ST = supratrochlear nerve

L = lacrimal nerve

IV = Branches of the IVth cranial nerve to the muscles. Plentiful distribution of fat is visible.

b View under the above structures. LEV = levator palpebrae superioris, SR = superior rectus muscle. Here, too, there is plentiful distribution of fat (more medially than laterally).

2.5 The Lacrimal Gland

The lacrimal gland lies in the upper temporal portion of the orbit, in the lacrimal gland fossa of the frontal bone. It is divided by the tendon of the M. levator palpebrae into an orbital part, lying on the bone, and a palpebral part, lying in the eyelid. Approximately ten small ducts secrete tears above the lateral corner of the eye into the superior conjunctival fornix. The gland is supplied by the lacrimal artery and the lacrimal nerve.

2.6 Intraorbital Fat and Connective Tissue

The space in the eye socket not taken up by muscles, vessels, and nerves is filled by a fatty tissue interspersed with connective tissue. A pyramidal cone exists within the four straight eye muscles. The connective tissue in the fatty tissue thickens towards the muscles, but in particular towards the globe, forming a more solid layer, which offers the eye ball a bed.

The periosteum of the orbit, the periorbit, lines the bony eye socket. It fades into the dura mater through the optic canal and the superior orbital fissure. The orbital septum limits the orbital content in front. It is a ring-shaped, nearly vertical plate of connective tissue it runs from the orbit edge to the superior and inferior tarsus. It is penetrated by several nerves and vessels (Figs. 2.3–5).

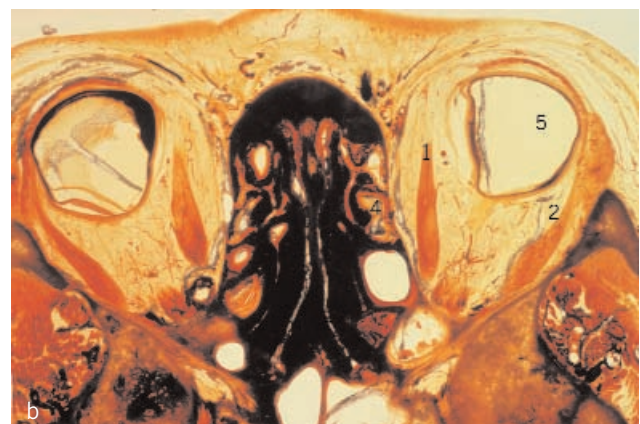
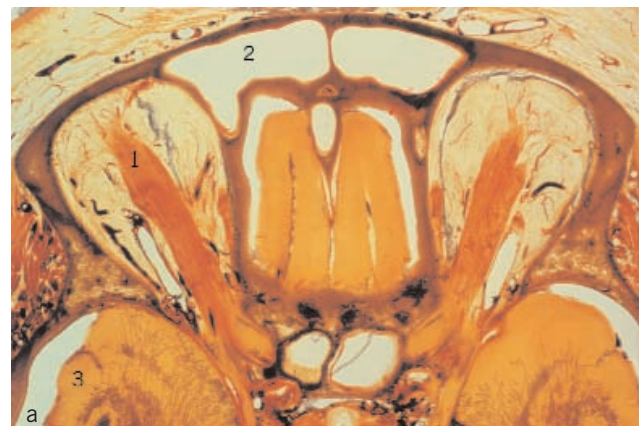


Fig. 2.10 a,b Horizontal section in the upper part of the orbit. 1. Levator palpebrae, 2. Frontal sinus, 3. Frontal lobe of the brain. b Horizontal section at the height of the middle of the orbit. 1. Medial rectus, 2. Lateral rectus, 3. Optic nerve – apical part, 4. Ethmoid sinus, 5. Globe

[We would like to thank Prof. Dr. Koebeke of the Anatomical Institute of the University of Cologne for this illustrative material].

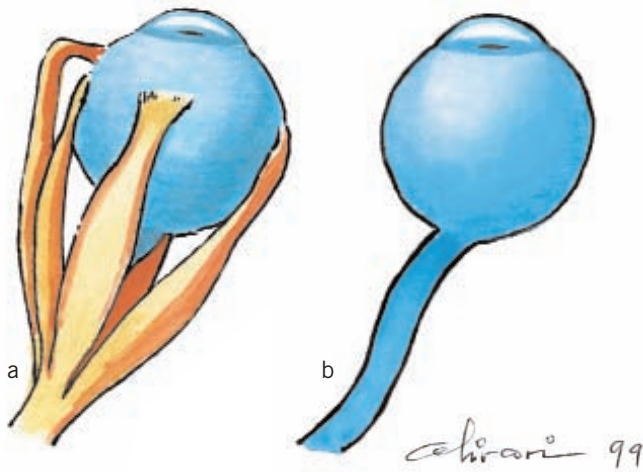


Fig. 2.11 a,b The extraocular muscles in the normal orbit. The drawings follow the 3D-CT display; viewed from beneath. a All muscles in a relatively relaxed condition, b Optic nerve relaxed, with many curves.

2.7 Optic Nerve

The optic nerve runs from the globe dorsally through the tendinous ring and the optic canal, leads to the chiasma. The optic nerve is relaxed in the primary position of the eyeball, curving slightly. This permits corresponding mobility during the movements of the globe. The optic nerve is surrounded by the dura mater, and this in its turn is fixed to the orbital periosteum. A traction of the optic nerve on the dura mater (in cases of exophthalmos) causes subjective complaints, such as retrobulbar “burning” and headaches (Fig. 2.8).