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Bilateral simultaneous orbital decompression for Graves' orbitopathy with a combined endoscopic and Caldwell-Luc approach

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Graves' orbitopathy can lead to cosmetic deformity, orbital pain, and visual impairment. Surgical intervention can improve proptosis, cosmetic appearance of the eyelids, vision, and orbital pain with minimal morbidity. Ten patients with dysthyroid orbitopathy underwent concurrent bilateral orbital decompressions. Of these, 9 underwent simultaneous bilateral endoscopic and transantral decompressions, and 1 had only bilateral endoscopic decompressions. Medical management of keratopathy was attempted before surgery. All patients were previously treated with radioiodine and high-dose corticosteroids, and 2 patients had prior low-dose orbital irradiation. Preoperative and postoperative visual acuity, color vision testing, and measurement of proptosis were recorded for all patients. In addition, photographs and CT scans of the orbit and sinuses were done. After surgery, visual acuity improved in 8 patients and remained unchanged in 2 patients. Diplopia in the primary and downward gaze improved in 1 patient and remained unchanged in the 3 others who had it before surgery. Color vision deficits in the blue/yellow range were present in 8 patients before surgery and all reversed within 2 weeks after surgery. Proptosis decreased by an average of 4.83 mm (range 4-7 mm) in patients undergoing the combined decompression and decreased by 4 mm in the 1 patient who had the endoscopic decompression only. Ethmoid sinusitis developed in 1 patient but resolved with oral antibiotics, and another patient had an asymmetric result requiring additional unilateral surgery, which corrected the asymmetry. Overall, this approach avoids external incisions and allows excellent visualization in the regions of the orbital apex and ethmoid roof, facilitating maximal decompression without the increased risk of bleeding or visual disturbances. (Otolaryngol Head Neck Surg 2000;122:216-21.)

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Graves' ophthalmopathy is the most common cause of unilateral or bilateral proptosis in adults. Proptosis, measured by Hertel exophthalmometry, is defined as abnormal displacement of the eye beyond a normal range of 14 to 21 mm with a 2-mm disparity. Dysthyroid orbitopathy may result in retrobulbar pain, optic nerve compression, exposure keratopathy, diplopia, and even blindness.[1] This entity is most often seen in patients with diffuse toxic goiter but may also be present in euthyroid or hypothyroid patients and may not correlate with thyroid function.[2]

Although the exact cause of Graves' orbitopathy is unknown, an autoimmune mechanism is implicated. Decreased circulating suppressor T cells and increased B-cell activity produce antibodies that react with antigens on extraocular muscles.[3] Histologically, the extraocular muscles exhibit an inflammatory process consisting of lymphocytes, plasma cells, and mast cells. In addition, endomysial fibroblasts contribute to this inflammatory process by producing glycosaminoglycans, which are deposited in orbital soft tissue and extraocular muscles. Accumulation of inflammatory cells and glycosaminoglycans expands orbital volume and results in restricted ocular mobility, diplopia, orbital apex crowding, and proptosis.[4] The inferior rectus muscle is most commonly involved, followed by the medial rectus, superior rectus-levator complex, and lateral rectus.[5]

Patients may report dissatisfaction with the cosmetic appearance of their eyes, retrobulbar pain, tearing, burning, photophobia, diplopia, and eventually visual problems. As the eye continues to expand, visual loss usually starts with loss of color perception.[5] Physical findings may include lid edema, retraction, chemosis, restricted extraocular mobility, and proptosis.[6] Werner[7] described 6 classes of eye changes in Graves' disease, with class 0 representing no physical signs or symptoms and class VI representing visual loss with optic nerve involvement. Intermediate stages are graded based on soft tissue involvement, severity of proptosis, and extraocular muscle and corneal involvement.[7] CT scans can demonstrate enlargement of the extraocular muscles, crowding at the orbital apex, and optic nerve swelling[8] (Fig 1).

Various therapeutic alternatives for the treatment of Graves' orbitopathy include corticosteroids, immunotherapy, orbital irradiation, surgical decompression of the orbital walls, or a combination of any or all of the above. Corticosteroids are initially useful in decreasing soft tissue inflammatory signs and symptoms in nearly all patients and may improve ocular mobility in one third of patients.[9] However, corticosteroids are not the best choice for long-term therapy because of their deleterious side effects and the recurrence of symptoms after their cessation. Radiation treatment may decrease orbital congestion and relieve symptoms caused by acute visual loss from orbital apex crowding.[10] However, proptosis and ocular mobility are less responsive to radiotherapy or corticosteroids than to surgical decompression. Surgical decompression is still the primary therapy for sight-threatening Graves' disease and is most effective for compressive optic neuropathy at the level of the orbital apex. By expanding the orbital confines, it reduces intraorbital pressure, corneal exposure, proptosis, and optic nerve compression.[11]

Decompression surgery should be done in the least traumatic manner by avoiding external incisions and allowing optimal visualization of all important landmarks. Historically, in 1911 Dollinger[12] performed the first lateral orbitotomy for exophthalmos, modeled after Kronlein's procedure, but the decompression was inadequate. In 1931 Naffziger described the orbital roof approach, which achieved better decompression but involved a craniotomy with its associated risks of meningitis and cerebrospinal fluid leaks.[13] Subsequently, Sewall[14] popularized the medial wall removal through an external ethmoidectomy approach, and Hirsch[15] followed with an inferior orbitotomy approach. In 1957 Walsh and Ogura[11] described a transantral Caldwell-Luc approach to decompress the medial and inferior orbital walls, thereby avoiding external incisions. Although they were successful in 8 patients, their review clearly discusses the limits of exposure with this approach. The endoscopic approach allows for complete medial orbital wall decompression with excellent visualization of the key landmarks.[16] In addition, this technique decreases eye trauma by avoiding violation of the inflamed orbital contents.

METHODS AND MATERIAL

There were 10 patients with Graves' orbitopathy in this study, which included 3 men and 7 women aged 32 to 75 years (average 54 years). Informed consent was obtained from all patients. This study was reviewed and approved by the local institutional review board. All patients had been previously treated with radioiodine and corticosteroids, and 2 had a history of prior orbital irradiation. The dose of radiation was 2000 rads per treatment for 10 treatments during a 2-week period. The mean time between radiation and surgery was 12 months. The mean time between radioiodine treatment and surgery was 11 months (range 6-18 months). All patients had been using topical ophthalmic lubricants for months before surgical intervention. The indications for surgery were exposure keratopathy and/or optic neuropathy. All patients had exposure keratopathy, and 8 had optic neuropathy.

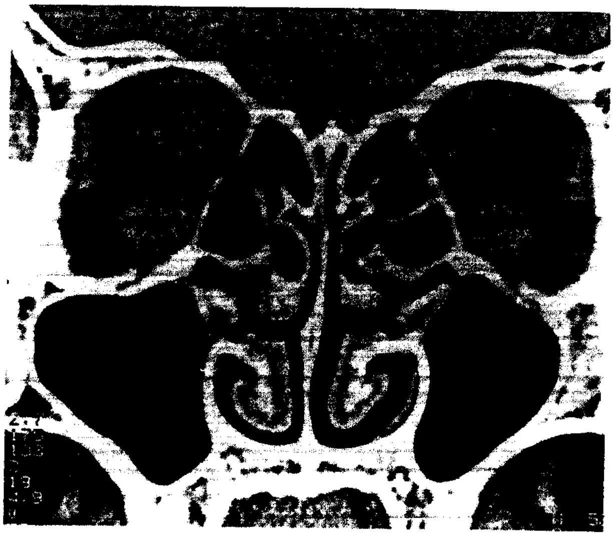

Fig 1. Coronal CT scan of the orbit and sinuses in a patient with Graves' disease showing gross enlargement of the extraocular muscles and crowding at the orbital apex.

Ten patients with Graves' orbitopathy underwent bilateral simultaneous endoscopic orbital decompressions, and 9 of the 10 also had a concurrent transantral Caldwell-Luc decompression. One patient (patient 10; Tables 1 and 2) only had bilateral simultaneous medial wall endoscopic decompression. Two of 10 patients also had a submucous resection of the nasal septum before the decompression. All procedures were performed with patients under general anesthesia.

Ophthalmology and otolaryngology evaluated all patients before and after surgery. Before surgery, patients had a complete ophthalmologic evaluation that included visual acuity, color vision, visual field testing, and Hertel exophthalmometry. Color vision was evaluated with AOHRR American Optical plates. Otolaryngology examination included nasal endoscopy to identify anatomic abnormalities in need of surgical correction at the time of the decompression. Data sheets were completed on each patient; data included preoperative and postoperative visual acuity, Hertel measurements, and presence or absence of diplopia. Preoperative and postopera-

Table 1. Bilateral orbital decompression data: Right eye

		Preoperative			Postoperative		
Patient	Age (y)/sex	Acuity	Diplopia	Hertels	Acuity	Diplopia	Hertels
1	49/F	20/50	+	23	20/20	–	18
2	61/M	20/70	+	28	20/50	+	23
3	33/F	20/25	–	22	20/25	–	17
4	59/F	20/40	–	24	20/20	–	19
5	71/F	20/80	+	23	20/50	+	19
6	65/F	20/30	+	20	20/25	+	16
7	75/M	20/100	–	23	20/20	–	18
8	42/F	20/30	–	25	20/25	–	19
9	32/M	20/20	–	22	20/20	–	17
10	50/F	20/30	–	21	20/20	–	17

Table 2. Bilateral orbital decompression data: Left eye

		Preoperative			Postoperative		
Patient	Age (y)/sex	Acuity	Diplopia	Hertels	Acuity	Diplopia	Hertels
1	49/F	20/50	+	24	20/20	–	19
2	61/M	20/80	+	29	20/30	+	24
3	33/F	20/25	–	23	20/25	–	18
4	59/F	20/80	–	23	20/20	–	19
5	71/F	20/80	+	22	20/50	+	18
6	65/F	20/30	+	21	20/25	+	17
7	75/M	20/80	–	24	20/20	–	17
8	42/F	20/25	–	24	20/25	–	19
9	32/M	20/20	–	23	20/20	–	19
10	50/F	20/30	–	21	20/20	–	17

tive photographs as well as noncontrast CT scans of the orbit and paranasal sinuses in the axial and coronal planes were also obtained.

SURGICAL TECHNIQUE

The patient was placed in a supine position with the head slightly elevated, after the induction of general anesthesia. Cottonoid pledgets soaked with 4% cocaine solution were placed in the nasal cavity for vasoconstriction. The gingivobuccal sulcus and the nose were injected with 1% lidocaine with 1:100,000 epinephrine solution. Bacitracin ophthalmic ointment was placed in the eyes for corneal protection during the procedure.

A submucous resection of the nasal septum was performed in the usual manner in 2 patients with significant deviations for better access into the ethmoid sinuses and lamina papyracea. The endoscopic decompression was begun with an uncinectomy and a complete intranasal ethmoidectomy as described by Kennedy et al.[16] The skull base and the lamina papyracea were visualized, and the frontal recess was avoided to prevent nasofrontal stenosis. The natural maxillary ostium was enlarged to 2 times its anatomic size. The lamina papyracea was gently removed with a freer elevator, superiorly to the ethmoid roof and posteriorly to the face of the sphenoid. As the dissection proceeded posteriorly, approximately 2 mm anterior to the sphenoid face, thicker bone was encountered. This area represented the annulus of Zinn from which the extraocular muscles originate and through which the optic nerve passes. Because the muscle tendons are not involved in Graves' orbitopathy, satisfactory decompression is accomplished without removal of this bone and therefore represented the posterior limit of our dissection.[17] Thick bone was also encountered at the junction of the medial and inferior orbital walls. This could limit adequate inferior wall decompression endoscopically.[16] The periorbita was now exposed and was incised from posterior to anterior, and from inferior to superior, with a sickle knife (Fig 2). Precautions were taken not to injure the medial rectus muscle. The orbital fat was carefully allowed to herniate and teased into the ethmoidal cavity by blunt dissection in the direction of the medial rectus fibers (Fig 3).

Attention was turned toward the gingivobuccal sulcus. A standard Caldwell-Luc approach was used. The periosteum

Fig 2. Intraoperative endoscopic view of the periorbita incised after removal of the lamina papyracea.

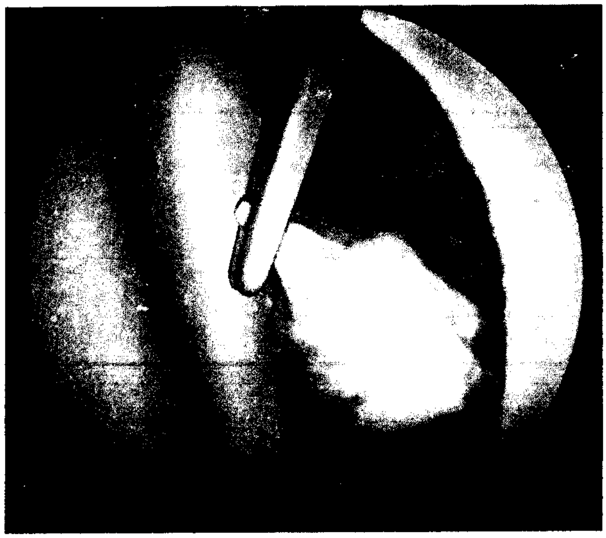

Fig 3. Intraoperative endoscopic view of the orbital fat prolapsing into the nasal cavity.

was elevated to the level of the infraorbital nerve. After identification of the infraorbital nerve, a freer elevator was used to remove the medial portion of the orbital floor from anterior to posterior, preserving the orbital rim (Fig 4). The hard bony connection between the medial and inferior orbital walls was removed, connecting the medial wall to the inferior wall decompressions. A sickle knife was used to incise the periorbita, and the orbital fat was teased into the maxillary sinus. Nasal packing was not used. The gingivobuccal incision was closed with buried 4-0 chromic suture. The patient was observed overnight and was discharged the next morning on oral antibiotics for 1 week.

RESULTS

All patients were proptotic with a range of 21 to 29 mm (Tables 1 and 2) and had exposure keratopathy. Eight of 10 patients had optic nerve compression and color visual deficits in the blue/yellow range. Proptosis was reduced by an average of 4.75 mm (range 4–7 mm) for the 10 patients. However, it was reduced by an average of 4.83 mm for the patients who underwent the combined endoscopic and transantral approaches (n = 9). The decrease in Hertels after decompression was compared for each eye in the same patient (Fig 5). Patient 10, who had medial endoscopic decompression alone, achieved a 4-mm recession (21 > 17), which was considerably less than that achieved with the combined approach. Visual acuity was impaired in 9 patients before surgery, and 8 improved after decompression. There were no cases of worsening vision after decompression. Color vision deficits reversed completely within 2 weeks of decompression in all 8 patients who had preoperative deficits. Diplopia in the primary and

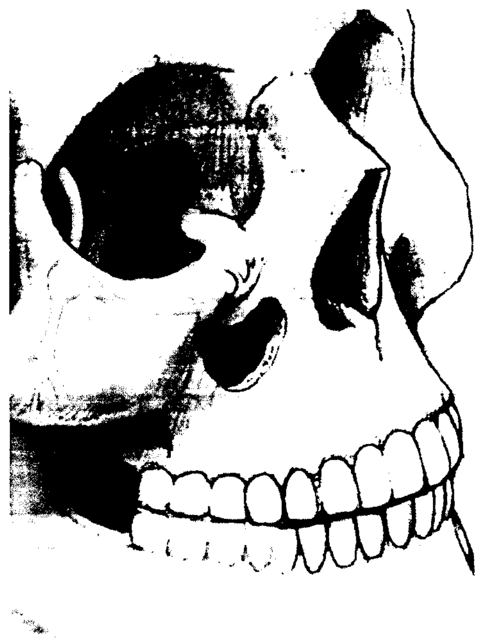

Fig 4. Diagrammatic view of the Caldwell-Luc component to achieving decompression of the inferior orbital wall medial to the infraorbital nerve.

vertical gaze was present in 4 patients before surgery and interfered with the patients' lifestyles. Diplopia improved in 1 patient after decompression but remained unchanged in the other 3 patients. There were no symptoms of new-onset diplopia after decompression. All patients had a dramatic immediate relief of their soft tis-

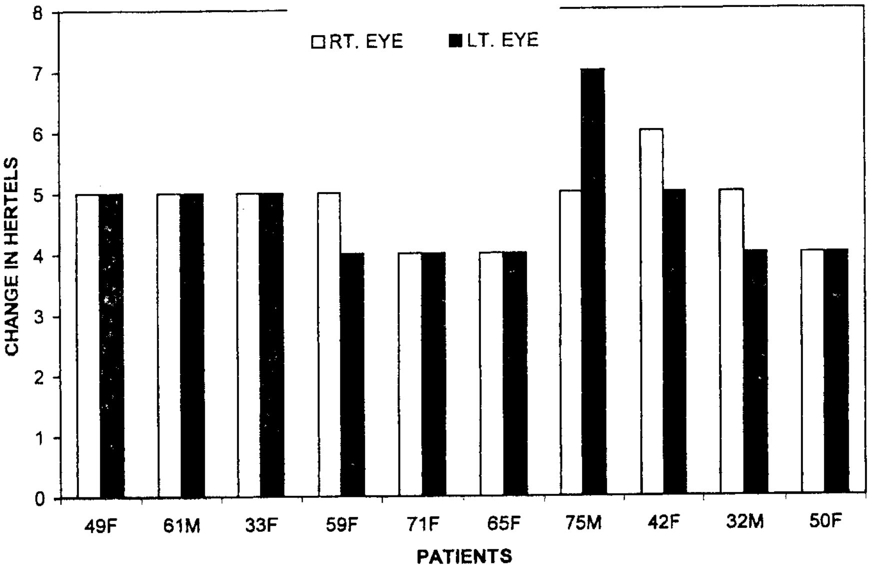

Fig 5. Reduction in proptosis after decompression measured in millimeters in each patient's right as compared with left eye.

sue symptoms including alleviation of the retrobulbar pain.

The average follow-up was 18 months (range 6-38 months). One patient had ethmoid sinusitis 6 months after the decompression, which resolved with oral antibiotics. Another patient had an asymmetrical result and required additional unilateral surgery. This patient was taken back to the operating room 4 months after the first decompression and underwent more extensive endoscopic bony removal on the proptotic side. Infraorbital paresthesias were not encountered in any patient. Three patients with persistent diplopia subsequently had muscle surgery. There were no complications such as hemorrhage, blindness, cerebrospinal fluid leakage, or meningitis as a result of these decompressions.

DISCUSSION

Dysthyroid optic neuropathy occurs in 8.6% of patients with Graves' disease.[18] This condition is refractory to nonsurgical management and can lead to blindness if untreated. Although cosmetic disfigurement has been cited as a surgical indication alone,[19] we performed decompression primarily for the preservation of vision. General anesthesia was used in all patients because of the extent of surgery and the discomfort involved in manipulating the inflamed periorbita. The use of local anesthesia has been reported by Metson et al[17] when using the transnasal technique.

Endoscopic decompression combined with the Caldwell-Luc approach achieved an average decompression of 4.83 mm (range 4–7 mm). This compares favorably with findings of other series, including those of Kennedy et al,[16] who reported an average of 4.7 mm of recession with the intranasal approach alone and 5.7 mm of recession when the transnasal was combined with a lateral orbitotomy approach.

There was a dramatic disappearance of retrobulbar pain and an immediate improvement in the soft tissue inflammatory changes of the eye accompanying orbital decompression. This approach provided excellent visualization of the medial orbital wall posteriorly to the apex. The inferior wall decompression was more safely and completely performed through the Caldwell-Luc approach, which can also provide access to the lateral orbital floor if a more extensive decompression is necessary. By avoiding excessive manipulation of the already inflamed orbital contents, the endonasal and Caldwell-Luc approaches decrease further orbital trauma.

The excellent visualization with the endoscopic technique allows simultaneous decompressions to be performed safely and completely with 1 surgical procedure. An additional benefit of simultaneous decompression is the ability to achieve more symmetry and improve cosmesis. In our series, only 1 patient required further unilateral surgery for an asymmetric result. Kennedy et al,[16] in a total of 13 patients, performed 5 bilateral simultaneous orbital decompressions without untoward results.

Primary and vertical gaze diplopia improved in 1 of 4 patients but did not develop de novo in any patient as a result of the surgery. Other series have reported an increase in diplopia requiring subsequent muscle surgery.[19] In fact, preexisting diplopia often may not improve after decompression, particularly because the increased orbital capacity may allow free movement of the fibrosed extraocular muscles.[16,17] It has been theorized that diplopia may sometimes increase because of the release of orbital pressure unmasking latent muscle dysfunction and intraoperative muscular trauma.[16,19] It has been our experience and that of others[20] that it is important to leave an anterior strut of the inferior orbital wall to prevent vertical gaze diplopia after surgery. Those patients (Nos. 2, 5, and 6; Tables 1 and 2) who had persistent diplopia after surgery underwent additional muscle surgery 3 or more months after the decompression surgery.

Orbital decompression with the herniation of the orbital contents into the nasal and sinus cavities theoretically poses an increased risk of sinusitis and possibly orbital infection. In our series, postoperative ethmoid sinusitis developed in 1 patient, which resolved with oral antibiotics. Walsh and Ogura[11] stated that neither sinusitis nor orbital infections developed in any of their patients and that this fear was "unwarranted." They stressed the need for a large inferior meatus antral window and wide removal of the medial and inferior orbital walls to ensure against orbital complications caused by infection. Hoffer and Kennedy,[21] however, reported on 3 cases of sinus mucoceles following the Walsh-Ogura technique, and all were managed endoscopically. They described the occurrence of sinus infections after decompression to be rare, presumably caused by the rapid formation of a dense fibrous pseudocapsule around the exposed fat.[21]

CONCLUSIONS

In our experience with 10 patients who had bilateral endoscopic simultaneous orbital decompressions, there was precise visualization of the landmarks and excellent hemostatic control, which enabled us to safely perform simultaneous orbital decompressions. Cosmetically, the results in reduction of proptosis compared favorably with those of other studies. There may also be an added benefit in the ability to accomplish more symmetry during a single surgical procedure. Functionally, visual acuity improved in all patients who were impaired and none of the patients had a new onset of diplopia after surgery.

The Caldwell-Luc approach was combined with the endoscopic transnasal technique in 9 patients and was necessary for extensive decompression of the inferior orbital floor to relieve optic nerve compression or severe proptosis. Limited handling of the globe and avoidance of the conjunctiva resulted in the immediate improvement of patients' soft tissue inflammatory symptoms. Finally, we strongly believe that the assistance of an ophthalmologist is important not only before and after surgery but also during surgery.

REFERENCES

1. Tucker SM, Tucker NA, Linberg JV. Thyroid eye disease. In: Tasman W, Jaeger EA, editors. Duane's clinical ophthalmology. Vol 2. Philadelphia: Lippincott, Williams & Wilkins; 1998.
2. Tami H, Nakagawa N, Oshako N, et al. Changes in thyroid function in patients with euthyroid Graves' disease. J Clin Endocrinol Metab 1980;50:108-12.
3. Wall JR. Autoimmunity in Graves' ophthalmology. In: Gorman CA, Waller RR, Dyer JA, editors. The eye and orbit in thyroid disease. New York: Raven Press; 1984.
4. Jakobiec FA, Font RL. Orbit. In: Spencer W, editor. Ophthalmic pathology. Philadelphia: WB Saunders; 1986. p. 2765-77.
5. Sacks EH, Anand VK, Lisman RD. Orbital decompression: endoscopic perspective. In: Anand VK, Panje W, editors. Practical endoscopic sinus surgery. New York: McGraw Hill; 1993. p. 138-59.
6. Trobe JD, Glaser JS, Laflamme P. Dysthyroid optic neuropathy. Arch Ophthalmol 1978;96:1199-209.
7. Werner SC. Modification of the classification of the eye changes of Graves' disease. Am J Ophthalmol 1977;83:725-7.
8. Trokel SI, Hilal SK. Recognition and differential diagnosis of enlarged extraocular muscles in computed tomography. Am J Ophthalmol 1979;87:503-12.
9. Werner SC. Prednisone in emergency treatment of malignant exophthalmos. Lancet 1966;May:1004.
10. Brennan MW, Leone CR, Janaki L. Radiation therapy for Graves' disease. Am J Ophthalmol 1983;96:195.
11. Walsh TE, Ogura JH. Transantral orbital decompression for malignant exophthalmos. Laryngoscope 1957;67:544-69.
12. Dollinger J. Die Druckenlastung der augenhohle durch enfernung der ausseren orbitalwand bei hochgradigem exophthalmus and konsekutiver hornhauterkrankung. Dtsch Med Wochenschr 1911;37:1888-90.
13. Naffziger HC. Progressive exophthalmus following thyroidectomy: its pathology and treatment. Ann Surg 1931;94:582-6.
14. Sewell EC. Operative control of progressive exophthalmus. Arch Otolaryngol 1936;24:621-4.
15. Hirsch O. Surgical decompression of malignant exophthalmos. Arch Otolaryngol 1950;51:325-34.
16. Kennedy DW, Goodstein ML, Miller NR, et al. Endoscopic transnasal orbital decompression. Arch Otolaryngol 1990;116:275-82.
17. Metson R, Dallow RL, Shore JW. Endoscopic orbital decompression. Laryngoscope 1994;104:950-7.
18. Riley FC. Orbital pathology in Graves' disease. Mayo Clin Proc 1972;47:975-9.
19. Baylis HI, Call NB, Shibata CS. The transantral orbital decompression as performed by the ophthalmologist. Ophthalmology 1980;87:1005-12.
20. Leone CR, Piest KL, Newman RJ. Medial and lateral wall decompression for thyroid ophthalmopathy. Am J Ophthalmol 1989;108:160-6.
21. Hoffer ME, Kennedy DW. The endoscopic management of sinus mucoceles following orbital decompression. Am J Rhinol 1994;8:61-5.