OCULAR MUSCLE SURGERY IN GRAVES' DISEASE*

BY *John A. Dyer,* MD

GRAVES[1] AND VON BASEDOW[2] ARE CREDITED WITH THE first publications describing the association between thyroid disease and exophthalmos (1835 and 1840). As Wybar[3] has stated, there is still no true understanding of the basic nature of the exophthalmic process. More advanced tests and accurate clinical techniques have been developed to confirm the diagnosis of Graves' disease, but the underlying causative factors thought by many to be autoimmune in nature or abnormalities in cell-mediated immunity have yet to be confirmed or corrected.[4] The thrust of the treatment regimen still is directed toward amelioration of symptoms and, in many cases, subsequent surgical correction of the disabling exophthalmos, ocular muscle imbalance, or eyelid malfunctions which eventuate.

PURPOSE

The purpose of this paper is to compare the surgical experience in 116 patients who had ocular muscle surgery for Graves' ophthalmopathy between 1968 and 1975[5] with that in 83 new patients operated on subsequently from 1975 through 1977. Some additional surgical procedures that have proved effective will be discussed.

PATHOPHYSIOLOGY

In 1969, Werner[6] published a classification of the eye changes of Graves' disease which has been widely accepted:

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0 No signs or symptoms
1 Only signs, no symptoms (signs = upper eyelid retraction and stare, with or without lid lag and proptosis)
2 Soft tissue involvement (symptoms and signs = eyelid edema, chemosis, congestion)
3 Proptosis
4 Extraocular muscle involvement
5 Corneal involvement
6 Sight loss (optic nerve involvement)

In this discussion, classes 4 and 6 are of prime interest.

Although many authors believed that the hypotropia and reduced abduction of the eyes were the result of a "paralysis" of the superior rectus and possibly the inferior oblique muscles,[7-10] other reports[5,11-13] confirm that there is, in fact, a fibrosis of the inferior and medial muscles which prevents elevation and abduction through a "leash" effect. This finding led Braley[11] to report a change in intraocular pressure on up-gaze in a number of patients. This, too, has been confirmed.[5] A false diagnosis of glaucoma can be avoided by measuring the intraocular pressure with the eyes in down-gaze or in a relaxed position.

Studies of orbital tissues and ocular muscles in Graves' ophthalmopathy[14,15] indicate that the muscles are enlarged and rubbery and that the orbital fat is under increased tension. Histologically, these changes result from interstitial edema caused by an increase in mucopolysaccharides and round cell infiltration (lymphocytes, plasma cells, macrophages, and mast cells). The presence of these inflammatory cells suggests an immunologic basis for this reaction.

DIAGNOSTIC AIDS

Electromyographic studies[16-18] have been inconclusive as to whether the basic ocular muscle defect is myogenic or neurogenic. On the other hand, both A-scan and B-scan ultrasonography[5,19-22] have demonstrated the presence of thickened extraocular muscles and, in conjunction with the sophisticated but more costly computerized tomography (CT scan),[5,23,24] have shown clear-cut evidence of enlarged muscles and proptosis. Saccadic velocity studies[25] give further affirmative evidence of a mechanical, restrictive basis for limitation of ocular movement; there is a normal tracing but a terminal slowing of the eye movement (rubber-band effect),[26]

whereas in other restrictive situations (blow-out fracture with muscle entrapment, for example) there is a sudden leash effect. Clinically, the forced-duction test and restricted rotations are final confirmation of the restrictive nature of ocular muscle involvement.

SURGICAL CONSIDERATIONS

The taut fibrous ocular muscles found during a surgical procedure in patients with Graves' myopathy present a unique test of the surgeon's patience and skill. Because of the inflammatory nature of the disease, postoperative scarring often is excessive and a result that looks promising hours or days postoperatively may change to an overcorrection, a regression to the previous state, or the appearance of an entirely new muscle imbalance as weeks pass. Hence, the patient must be apprised of the fact that a second or third or even more procedures may be required. The primary goal is to achieve some degree of single binocular vision in the primary and reading positions; full rotations rarely are achieved.

Basically, the operation of choice is to relax the fibrous, restrictive ocular muscles — most frequently the inferior rectus muscles, secondly the medial recti, less often the superior muscles, and seldom the lateral recti. Some surgeons suggest complete severance of the scleral insertion (inferior muscles), whereas others recess 6 mm or more[27] as a standard procedure; others suggest a graded recession depending on the severity of restriction[5] or an adjustable suture technique,[28-30] so that a muscle can be advanced or recessed several hours postoperatively. This variety of opinions attests to the fact that there is still no panacea for surgical cure of the muscle imbalance. In my experience, reoperation is required in about 40% of patients (Table I).

Unless very careful and thorough dissection is done along the inferior rectus as far posteriorly as possible to relax or strip away the aponeurosis from the inferior rectus to the lower border of the tarsus, retraction of the lower eyelid may require later surgery (scleral graft). In my experience, any recession more than 4 or 5 mm will result in this complication unless dissection is meticulous. A sharpened lid expressor (Weck) as is shown in Figure 1 is an ideal instrument for separating these tissues meticulously in any muscle procedure. Figure 2 shows extensive fibrosis along the inferior rectus muscle and the tissue dissected away from the muscle. A large recession (that is, more than 5 mm) was my choice formerly;[5]

however, although a complete myectomy or a large recession will relieve the hypotropia or esotropia, it also will create an inability to depress the eyes into the reading position (inferior recti) or will reduce convergence (medial recti) so that bifocals are rendered useless or the patient must hold objects upward in front of his eyes to read. This is particularly annoying when an operation is done on only one inferior muscle.

Other complications, such as exposure of the globes, may occur after the release of fibrotic ocular muscles with an increase in proptosis. In my opinion, if the proptosis is more than 22 or 23 mm (Hertel or Krahn exophthalmometer) serious consideration must be

TABLE I
OCULAR MUSCLE SURGERY IN GRAVES' DISEASE: PATIENT PROFILE AND ANALYSIS OF SURGICAL DATA

	Group 1 (1968-1975)	Group 2 (1975-1977)
No. of patients	116	83
Sex		
Female	85 (73%)	60 (72%)
Male	31 (27%)	23 (28%)
Age (yr)		
Female patients		
Youngest	23	16
Oldest	74	73
Average	51	46
Male patients		
Youngest	26	24
Oldest	75	71
Average	48	49
Orbital decompression		
Total	68 (59%)	57 (69%)
Bilateral		
Transantral	63 (93%)	57 (100%)
Transfrontal	3	0
Combined	2	0
Secondary to muscle surgery	2	1
No. of operations		
1	64 (55%)	52 (63%)
2	33 (29%)	20 (24%)
≥3	19 (16%)	11 (13%)
Scleral grafts		
Primary	0	3 (4%)
Secondary	17 (15%)	7 (8%)
Muscles operated primarily		
Inferior and medial only	35 (30%)	46 (55%)
Inferior recti only	48 (41%)	1 (1%)
Other combinations	33 (28%)	36 (43%)
1 inferior only		12 (14%)
Both medial, 1 inferior		8 (10%)
Both inferior, 1 medial		7 (8%)
Medial and inferior, 1 eye		6 (7%)
One medial only		2
Medial muscle only		1

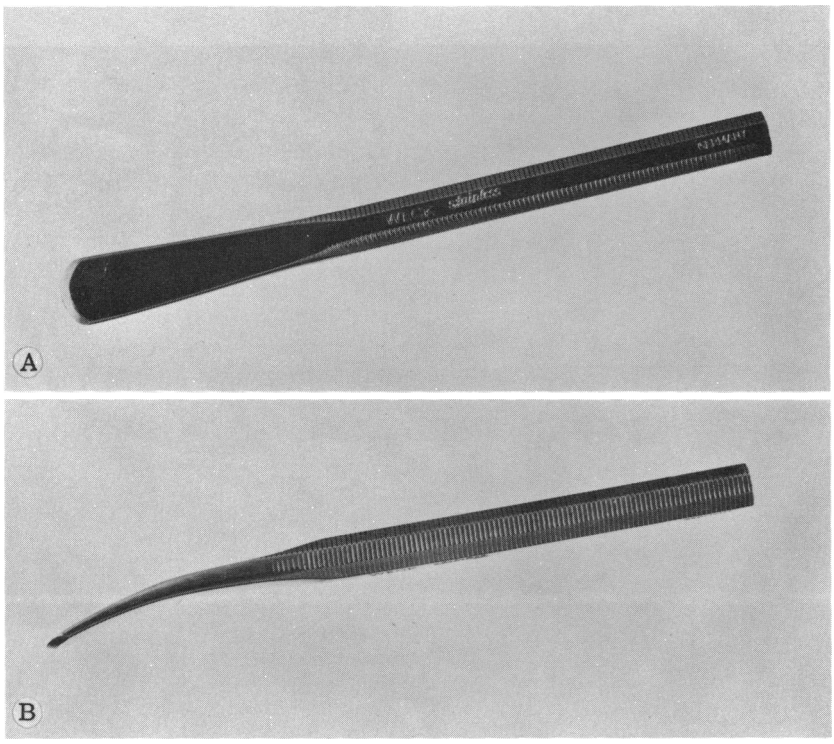

FIGURE 1
A and B, Sharpened lid expressor (Weck) — a useful instrument for separating muscles from surrounding tissues.

given to orbital decompression before muscle surgery even if there is no threat to vision. The patient should be advised that about 50% of those with no ocular muscle imbalance before decompression will have diplopia afterward and that if an imbalance is present before decompression, it will remain or be more severe after surgery.

The prime indications for orbital decompressions are diplopia with proptosis more than 22 to 23 mm, exposure of the globe, cosmesis, and progressive visual loss because of optic neuropathy. In any patient, a surgical procedure is not advised until the thyroid state is stable. Our otolaryngologists perform the transantral decompressions and anticipate from 2 to 10 mm regression in proptosis. A bilateral procedure always is done to prevent asymmetry.

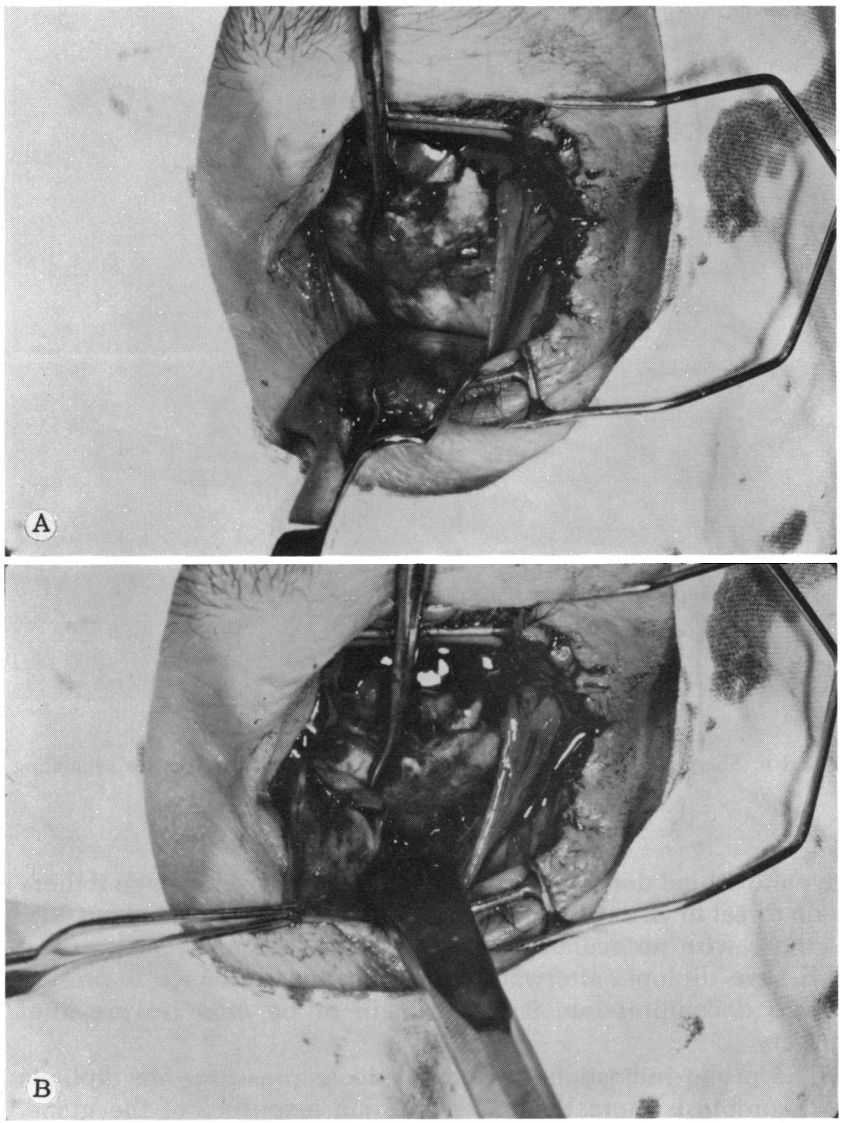

FIGURE 2

A: Extensive fibrosis along inferior rectus muscle. B: Tissue dissected away from inferior rectus muscle.

Muscle surgery is delayed four months or longer to permit any ocular muscle change to occur and all tissue reaction to subside.

If only one inferior or medial rectus muscle is involved with minimal proptosis, a recession of the single muscle may suffice. After decompression, most patients require an operation on both inferior and medial muscles; this should be done as one procedure. From experience, I have found that a minimum recession usually suffices because the eye or eyes will elevate or abduct more as times passes. To relax muscles further secondarily is much simpler than to repair an overcorrection. My results are best when recession of the medial rectus muscle is a standard 5 mm and recession of the inferior muscles is varied from 2 to 5 mm so that the pupillary light reflexes from the operating room light are slightly above center in each eye while the patient is under anesthesia. This smaller recession permits the eyes to elevate adequately, yet depression is maintained for satisfactory reading. Jampolsky,[28] Flynn,[29] and Knapp[30] have advocated the use of an adjustable suture — that is, once the muscle is released, the suture arms are passed through the original insertion close together (no scleral bite) and fastened with a slipknot or bow so that some hours later the suture may be tightened or relaxed to improve the eye position. Usually a maximum recession is performed and the muscle is advanced if necessary. Because the postoperative course is so variable in Graves' disease, I have found the variable recession technique best thus far. I do agree that attaching the suture at the original insertion only (Figure 3) permits a more normal alignment of the muscle. Vicryl or Dexon sutures of 5-0 or 6-0 caliber are preferred; the tensile strength is excellent, and the tissue reaction is minimal.

Many patients have a definite "A" pattern esotropia. Elevation and abduction of the eyes may be poor; the chin is tilted upward so that single vision can be obtained, and on up-gaze the eyes converge excessively. In the past, I attributed this to the taut inferior muscles failing to relax;[5] however, during surgery I noted that, in many patients, the upper half of the medial muscles often was very thick and swollen whereas the inferior portion was very fibrous and tight (Figure 4), as are the inferior muscles. Subsequently, in these patients I have recessed the lower border of the muscle 2 or 3 mm more than the upper portion and displaced the muscles upward at least one muscle width (Figure 4), as one would do in treating a child with "A" esotropia. Although my experience with these pa-

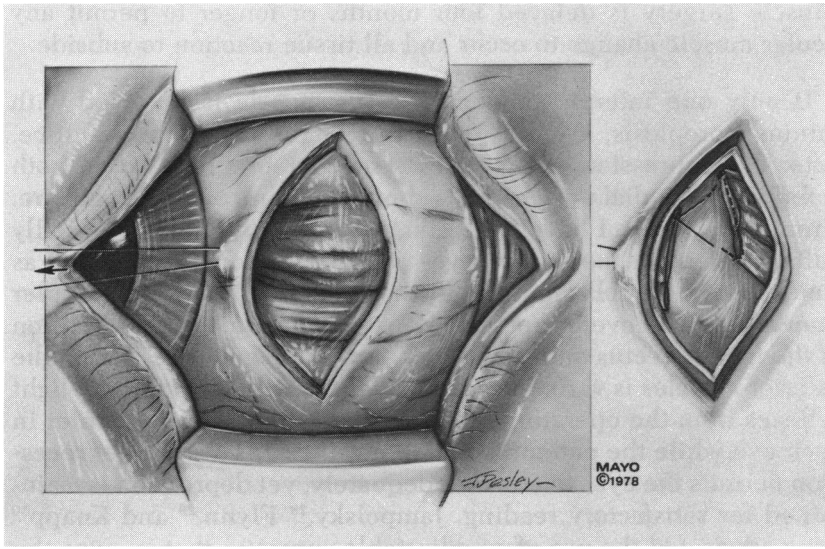

FIGURE 3
Vicryl suture attaching muscle to original insertion only.

tients has been limited, my impression is that the results are definitely more satisfactory.

From experience I have learned also that a secondary hypertropia is best treated in most instances by a recession of the antagonist superior rectus muscle rather than an advancement of the previously recessed inferior muscle because the problem often is contracture of the superior muscle once the tight inferior muscle is relaxed. If the hyperdeviation is severe, the inferior muscle may require advancement also, but only after weakening of the superior muscle usually a maximum of 4 mm. Because of gradual secondary contracture of the antagonist superior or lateral rectus muscles in this disease, one should postpone further surgical intervention for several months. If the inferior muscle is permitted at surgery to retract freely with no measured recession, its secondary recovery is virtually impossible.

In severe cases in which a large recession is desired primarily or in subsequent operations if further relaxation is desired, proper alignment of the muscles often is difficult. The use of homologous sclera as an extension to the muscle has proved useful.[5] The graft is cut the desired length and width, attached to the distal end of the

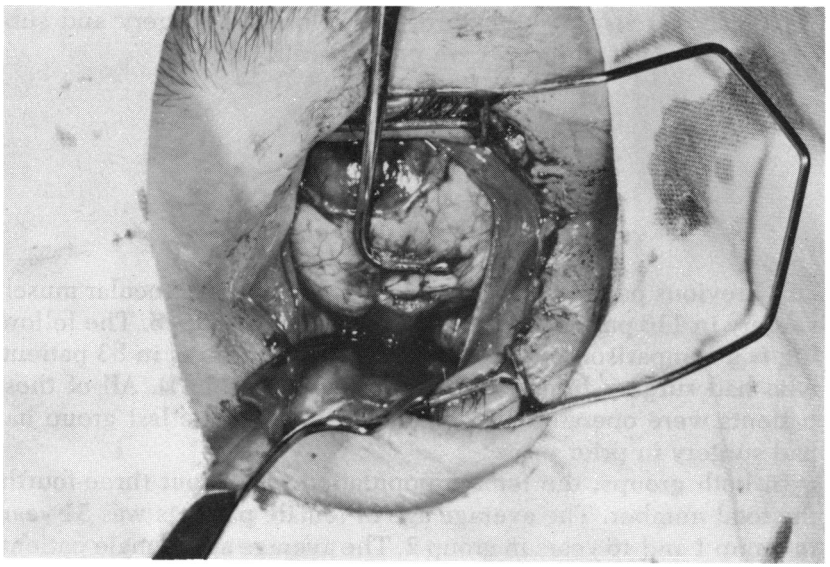

FIGURE 4
Surgical recession with upward placement of medial muscle. Note thickened upper portion and fibrous lower portion of muscle and greater recession of lower border.

muscle, covered by a Supramid sleeve, and attached to the original insertion. This ensures a proper alignment of tissue, blends well with muscle and sclera, and permits easy recovery at a later date if needed. The use of Supramid sleeves and caps, as advocated by Knapp[31] and others,[32] is a useful adjunct in secondary procedures especially.

If lower or upper eyelid retraction results in undue exposure of the eyes, surgery designed to correct these defects is in order. This always follows extraocular muscle surgery inasmuch as excessive upper eyelid retraction may result from the patient's thwarted effort to look up and is reduced when the fibrotic inferior muscles are relaxed.

At the Mayo Clinic, a team approach has proved most satisfactory. This team includes an endocrinologist who certifies that the patient's disease is quiescent, an ophthalmologist, and an otolaryngologist who performs the transantral decompressions on the advice of the ophthalmologist; at least two of these team members must examine the patient and suggest decompression for reasons

previously discussed. Afterward, ocular muscle surgery and subsequent eyelid realignment are performed if needed.

CLINICAL EXPERIENCE

In a previous paper,[5] I reported my experience with ocular muscle surgery in 116 patients operated on from 1968 to 1975. The following is a comparison of these results with the results in 83 patients who had surgery from 1975 through 1977 (Table I). All of these patients were operated on by me, and none of the last group had had surgery in prior years.

In both groups, the female population was about three-fourths the total number. The average age of female patients was 51 years in group 1 and 46 years in group 2. The average age of male patients was 48 years in group 1 and 49 years in group 2. Orbital decompression was performed before muscle surgery in 68 patients (59%) of group 1, 63 (93%) of these having a transantral decompression. In comparison, 57 patients (69%) of group 2 had a decompression and all had a transantral procedure. Decompression was done secondary to muscle surgery in two patients in group 1 and in one patient in group 2. Sixty-four (55%) of those in group 1 had a single surgical procedure compared with 52 (63%) of these in group 2, whereas two procedures were done in 33 patients (29%) in group 1 and in 20 patients (24%) in group 2. Three or more procedures were done in 16% of group 1 patients and in 13% of group 2 patients. Homologous scleral grafts to extend ocular muscles were used in secondary procedures in 17 patients (15%) of group 1; scleral grafts were used in primary procedures in 3 patients (4%) and in secondary procedures in 7 patients (8%) in group 2. Whereas 35 patients (30%) of group 1 had both inferior and medial muscles operated primarily, 46 patients (55%) of group 2 had this as a first operation. Both inferior muscles were recessed in 48 (41%) of group 1 patients, whereas only 1 patient had this procedure in group 2. Other combinations of muscles were operated in 33 patients (28%) of group 1 and in 36 patients (43%) of group 2.

From this comparison it appears that some progress has been made in decreasing the number of procedures required to achieve single binocular vision for patients with severe muscle imbalance.

SUMMARY

When the thyroid state is stable and no further changes in ocular motility are occurring, surgical correction of diplopia is necessary to regain single vision in the primary and reading positions. Preliminary tests including A-scan and B-scan ultrasonography, the CT scan, saccadic velocity testing, and forced-duction tests aid in excluding other causes of muscle abnormalities.

In the absence of proptosis and with minimal eyelid retraction, recession of the taut rectus muscles, most often the inferior and secondly the medial, is required.

If proptosis is moderate (22 to 23 mm) and lid retraction is severe, preliminary orbital decompression (bilateral transantral) is advised inasmuch as prior relaxation of the ocular muscles may cause increased proptosis and exposure of the eyes, which would necessitate emergency orbital decompression.

If severe proptosis creates a cosmetic blemish, or if exposure of the globes or optic neuropathy is a threat to vision, decompression should be done, followed in four months or longer by ocular muscle surgery. After decompression, diplopia may occur in almost 50% of patients with no muscle imbalance preoperatively, whereas all patients with diplopia before decompression will have a more severe imbalance after decompression.

Although I advocated larger recessions of the fibrotic muscles previously,[5] a smaller, graded recession of the inferior muscles after careful dissection is more accurate and prevents lower eyelid retraction. Transposition of the medial muscles upward with recession is helpful in many patients with an "A" pattern esotropia. With time, the antagonist superior rectus muscle contracts and must be recessed secondarily if a large inferior recession has resulted in overcorrection. Advancement of the inferior muscle(s) will not suffice. The use of scleral grafts to extend the ocular muscles and placement of Supramid sleeves or caps are useful adjuncts in reoperations.

These findings have led to a decrease in the number of surgical procedures required to gain useful single vision for these patients. As a general rule, if the pupillary light reflexes from the operating room light are almost centered in each eye at the conclusion of surgery, the eyes will remain in this position when the patient awakens. Because of the fibrotic state of the muscles, full rotations seldom are regained.

When the thyroid state is stable, relieve the proptosis, straighten the eyes, and correct the eyelid retraction for most effective results.

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DISCUSSION

DR PHILIP KNAPP. I want to thank Doctor Dyer for handing me this paper almost two months ago in Honolulu. It is fortunate for me that he did as I just got back from the Far East the night before last.

As you just heard, the author has described what is known about the orbital changes in Graves' disease and he has listed all the fancy and expensive tests available to make the diagnosis. However, as far as the disturbances in ocular motility are concerned, these tests are rarely needed, for the diagnosis is clinical. Nothing else looks like it or acts like it. Moreover, the traction test cinches the diagnosis.

I think the number of cases presented by Doctor Dyer is most impressive. The increase from 113 cases in over 7 years to 83 in the next three years leads to one of two conclusions. Either the Mayo Clinic has built a better mouse trap or the team has gotten more confident so that they are operating on a higher percentage of patients. Certainly, the figure that 69% have had a bilateral orbital decompression by the Ogura method is startling. Our figure is only 10% but we reserve this type of decompression for patients needing a sight-saving procedure either due to the danger of exposure of the cornea or to compression of the optic nerves. This 69% producing 50% of new motility disturbances is one of the reasons so many patients require muscle surgery. We have found that 13% of our patients develop new muscle problems following decompression. We think that many of these patients with exophthalmos of only 22-23 mm can be cosmetically relieved by working on the lids — either recessing the levator or by raising the lower lids with a scleral graft. In such patients one should do the lid surgery first because it will not affect the motility, but we entirely agree that muscle surgery should wait until after the orbital decompressions. We feel the Ogura decompression is a formidable procedure with reported complications including blindness. I would like to ask Doctor

Dyer whether they have had any significant complications besides the disturbed motility?

The author feels that the inflammatory nature of the process leads to excessive postoperative reaction with the resulting scar tissue causing instability of the operative result. I would agree that the results of muscle surgery in Graves' disease are unstable but feel inflammatory reaction causes only part of them. This part can be minimized by recessing the conjunctiva fully, plus using frequent steroid drops alternating with lubricating drops as exposure contributes to the postoperative reaction. Our orthoptic department and I think that some of the postoperative instability is really a failure to evaluate correctly the preoperative measurements. As the ocular myopathy involves all the recti muscles though frequently in varying degree, a marked contracture of one vertical rectus may overshadow other involvements, which then become apparent postoperatively. A marked increase or reduction in a hypertropia in a field where one would not expect such a change may indicate involvement of another or other muscles. Operating to correct these discrepancies at the same time as the main culprit may save subsequent operations, particularly if one uses adjustable sutures which I feel are the best method of achieving a variable recession.

I certainly agree with Doctor Dyer that an excessive recession leading to the crippling of the muscle's action is to be avoided. Not only does this lead to a marked limitation of motion in the field of the crippled muscle, but like any paresis it sets up an ideal condition for subsequent contracture of the yoke or direct antagonist.

I have had no experience with the use of homologous sclera to lengthen the muscle further without losing its arc of contact. If one were to do such a procedure, the use of Supramid Extra would certainly be helpful to prevent adhesions from nullifying the effect of surgery. I have merely added a "Z" or marginal myotomy to the recessed muscle to achieve the same lengthening effect without losing the arc of contact.

I have never been aware of the "A" patterns he reports. The patients I have seen showing this have had congenital fibrosis of the inferior rectus rather than Graves' disease. It has been my thought that releasing the taut inferior rectus was all that was necessary, just as recessing the tight lateral rectus stops the upshoot on attempted adduction with a Duane's syndrome.

There is one frustrating aspect in treating these patients that has really upset me. After a successful result with a good field of single binocular vision, a year or two later a deviation occurs. I remember one woman in whom this happened twice. Has the author also had this miserable experience?

Finally, I would like to congratulate the author on this excellent report of his extensive (probably unparalleled) experience in treating this frustrating problem.

DR JOHN A. DYER. Thank you, Doctor Knapp. Many of the patients have only a moderate degree of proptosis and minimal exposure problems. When lid retraction is the primary problem and diplopia is not present, we will either do a recession of Mueller's muscle or place a scleral graft in the upper or lower eyelids as a primary and perhaps the only procedure.

As to complications of decompression, we have had only one patient in some 350 who had any problem with vision afterwards. This patient had reduced vision in one eye, probably due to postoperative hemorrhage. The vision returned to normal, as I recall. The remainder of the complications are very minor in nature. One or two patients had a fistula that remained postoperatively; these were closed secondarily.

Some patients have inturning of the lower lids because of a "setting sun" appearance to the eyes after liberal decompressions. The otolaryngologist has become a little less vigorous now. If the entropion is severe, a scleral graft to reduce this may be required at a later date. Other than that, the complications are minimal.

I would agree with Doctor Knapp that clinical testing, especially forced-traction testing, is important. If one excludes trauma, there really is no other muscle involvement quite like Graves' disease. One can diagnose this problem with a forced-traction test. This is especially so at surgery with the patient under anesthesia.

As to ischemic iritis, I have had only one patient who showed some cells and flare and a slight clouding of the cornea in one eye the first postoperative day. The remainder of the patients have not had any problem with ischemic iritis. Some of these muscles are so tight that the pull on the muscle for a sustained period of time results in much pressure on the globe. I think many of these eyes have a hypotony and perhaps some degree of inflammation just from the vigorous manipulation of the globe.

Recurrent problems do occur. The patient may have single binocular vision after surgery for nine months or twelve months and return with a new or added muscle problem, without a flare-up of the Graves' disease. I think this is further evidence that there is some continually progressive inflammatory process in the muscles.

The number of decompressions has increased because a greater number of patients who have more severe Graves' ophthalmopathy have been referred to us. Thank you very much.