# SCAR REMODELING AFTER STRABISMUS SURGERY*

BY Irene H. Ludwig, MD

**ABSTRACT**

*Purpose*: Patients with overcorrected strabismus (and several patients with undercorrection after extraocular muscle resection) underwent exploration of previously operated muscles, with the intention of advancing their tendons to prevent the need for surgery on additional muscles. Unexpectedly, it was found that, in many cases, an elongated scar segment of variable length was interposed between the muscle and its insertion site on the sclera. Laboratory investigations were carried out to elucidate the underlying mechanism(s) and to create an animal model of the disorder.

*Methods*: Lengthened scars were repaired on 198 muscles during 134 procedures performed on 123 patients. The scars consisted of amorphous connective tissue interposed between the globe and normal tendon. Repair was accomplished by excision of the scar and reattachment of the muscle to sclera, using absorbable sutures in 64 cases and nonabsorbable sutures in 70 cases. Histopathologic examination was performed on 82 clinical specimens, and tissue culture studies were performed on 7 specimens.

To develop an animal model, 10 New Zealand white rabbits underwent bilateral superior rectus resection. Half of the eyes received sub-Tenon's injections of collagenase over the operative site during weeks 2, 3, 5, and 6 postoperatively; the other half received saline solution injections on the same schedule. At 10 weeks, half the sites were studied histologically, and the other half underwent collagen creep analysis. In a second study, the use of absorbable versus nonabsorbable sutures was compared in the rabbit model.

*Results*: In the clinical cases, the mean length of the elongated scar segments was 4.2 mm. A total of 105 of the 134 repair procedures were judged successful. Thirty-one procedures resulted in recurrence of the original overcorrection; 7 of these had documented restretches. Factors that distinguished patients with stretched scars from patients with classic

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slipped muscles included minimal or no limitation of versions, less separation of the tendons from sclera, and thicker appearance of the scar segments. The use of nonabsorbable sutures in the repair procedure reduced the recurrence rate. Histologic examination of the clinical stretched scar specimens showed dense connective tissue that was less well organized compared with normal tendon. In the tissue culture studies, cells cultured from the stretched scar specimens grew rapidly and were irregularly shaped. A high-molecular-weight protein was identified in the culture medium. By contrast, cells cultured from normal tendon (controls) grew more slowly and regularly, stopped growing at 4 days, and produced less total protein than cultured stretched scar specimens.

In the animal model studies, the collagenase-treated sites showed elongated scars with increased collagen between the muscle and the sclera, as well as increased collagen creep rates, compared with the saline-treated controls. The use of nonabsorbable sutures in collagenase-treated animal model surgery sites was associated with shorter, thicker scars compared with similar sites sutured with absorbable sutures.

*Conclusions*: A lengthened or stretched, remodeled scar between an operated muscle tendon and sclera is a common occurrence and is a factor contributing to the variability of outcome after strabismus repair, even years later. This abnormality may be revealed by careful exploration of previously operated muscles. Definitive repair requires firm reattachment of tendon to sclera with nonabsorbable suture support.

## INTRODUCTION

Variable results after strabismus surgery have long been reported. Overcorrection rates of 2% to 8% occur following uncomplicated surgery, often despite good initial postoperative alignment.[1] The true lifetime incidence of overcorrection (or undercorrection following muscle resection) may actually be higher than has been recognized, in that alignment can change many years postoperatively, unbeknownst to the original surgeon.

Pioneering strabismus surgeons experienced difficulty in correcting overcorrected strabismus by readvancing previously recessed muscles. They noticed that the amount of muscle advancement that was required to obtain improvement was greater than expected. Cooper, in his 1961 paper on secondary exotropia,[2] reported improved results obtained by recessing the antagonist lateral recti. He recommended basing the surgical approach to secondary exotropia on the clinical findings at the time, rather than trying to undo the previous procedure. Cooper's dictum has been widely adopted by strabismologists, who usually operate on fresh antagonist

muscles without exploring the originally recessed ones, unless version limitation or strabismus incomitance suggests lost or slipped muscles.[3]

During the author's 12 years of clinical strabismus practice, all patients with secondary strabismus after previous strabismus surgery (by the author or by other surgeons) underwent exploration of previously operated muscles. In the early years, the expectation was that the muscles would be found normally healed at their original surgical attachment sites and that repositioning of the insertions would repair the deviations. In many cases, however, a segment of amorphous scar tissue was found separating the tendon from its attachment site on the sclera.

At first, it was believed that improper technique during the original surgery was the cause of the scar segment complication, and that excision of the scar with secure reattachment of tendon to sclera with absorbable sutures would ensure correction of the defect. Over time, however, several further recurrences were documented, despite what had seemed to be appropriate repair technique. These observations led to a new idea as to the cause of the problem, as well as a change in the approach to repair and investigation into possible mechanisms that might underlie this phenomenon.

In this thesis, the previously unreported observation of an elongated segment of scar tissue interposed between the muscle tendon and scleral attachment site in 134 reoperation cases, as well as related histochemical and tissue culture studies and the development of an animal model, is described. On the basis of the clinical and laboratory evidence, it is suggested that the elongated scars may have lengthened or stretched despite adequate initial surgical reattachment of tendon to sclera, and that this elongation is a function of the stresses of this particular type of surgical procedure combined with individual metabolic and/or wound healing characteristics that differ from patient to patient. The phenomenon of scar lengthening in the extraocular muscles is shown to be similar to known pathophysiology in healing of other tissues under tension. The literature concerning recent advances in wound healing and surgical research is reviewed in terms of new insight into the mechanisms of scar stretch after strabismus surgery (see "Discussion"). Finally, new approaches to reduce the incidence of this complication, as well as to increase the rate of success of repair, are suggested.

## METHODS

### CLINICAL STUDIES

#### *Patient Characteristics*

Only cases with lengthened scar segments interposed between extraocular muscle tendons are included in this thesis. These cases represent a subset of secondary strabismus reoperation cases, encompassing perhaps as much

as 50% of the group. In all, lengthened scars were repaired on 198 muscles in 134 procedures on 123 patients. The mean time between the original strabismus surgery and the scar repair was 122 months (range, 0.5 to 612 months). The median age of the patients at the time of lengthened scar repair was 18.8 years (range, 3.1 to 67.8 years) (Fig 1).

*Time Course of Development of Secondary Strabismus with Scar Lengthening (Overcorrection or Recurrence): Early versus Late Stretch.* A total of 43 patients were able to date the onset of secondary strabismus, and some had original office records to document the time courses of their alignment measurements. Twenty-one cases of secondary strabismus developed within 4 months of surgery, and 20 developed after 18 postoperative months, 1 as long after as 516 months. Only 2 cases of secondary strabismus developed between 4 and 18 months after the original surgery, 1 at 6 months and the other at 10 months. The mean time between the original surgery and the patient's recollection/ documentation of the onset of overcorrection–the historical time-to-stretch–was 60 months (range 0.5 to 516 months) (Fig 2).

Thirty-one patients described "gradual" onset of secondary strabismus, 3 related good alignment for years followed by subacute change, and 57 had no recollection of the time course of their development of secondary strabismus.

*Previous Surgical Procedures.* Records of previous surgery were often unavailable, and a number of patients were uncertain of their original deviation or original procedure(s). Many, however, gave a clear history of strabismus overcorrection, often after bilateral surgery. In other cases, the nature of the previous surgery was determined only by inspection at the

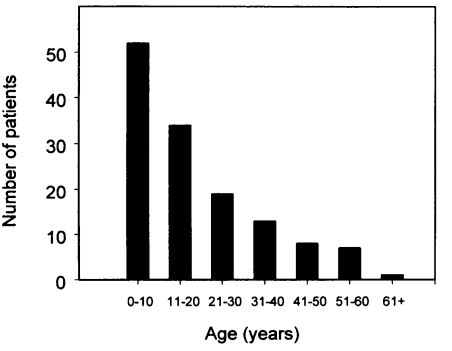


FIGURE 1
Age of patients at time of repair of secondary strabismus due to lengthened scar(s).

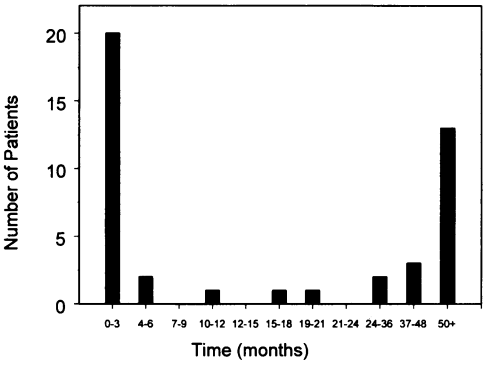


FIGURE 2

Time to stretch based on patient history or previous records (43 patients). Another 31 patients recalled gradual onset of secondary strabismus, 3 were aligned for years followed by subacute change, and 57 had no recollection.

time of the repair procedure.

*Original Surgery by Author.* In 26 cases, the author had performed the original surgery that was followed by secondary strabismus with lengthened scars. In all cases, 6-0 polyglactin sutures had been used, with secure passage of the suture through tendon, 2 lock bites on each tendon, and careful tendon inspection. Loose connective tissue surrounding the insertion, including capsule, had been removed prior to muscle reattachment to sclera (Fig 3). In no case was muscle capsule attached to sclera. Three of these patients had undergone adjustable suture surgery on an inferior rectus.

*Intraoperative Findings*

In all 134 procedures described here, a segment of scar tissue was found interposed between tendon and sclera. The distinction between scar and tendon was often subtle, as the scar closely resembled tendon. The fibers of each scar segment paralleled the tendon fibers, and often blended almost imperceptibly with tendon (Figs 4 through 11). However, the behavior of the scar segment during surgery differed from that of normal tendon. When a scar segment was excised, the linear arrangement of the collagen fibers was lost with the loss of tension, and the segment assumed an amorphous shape (Figs 12 and 13). This change was not seen when normal tendon was excised in resection cases (Figs 14 and 15). Also, when the muscle hook was pulled away from the sclera, a lengthened scar segment could be lifted farther outward (Fig 16) than well-healed muscle (Figs 17 and 18) or normal tendon (Fig 19). Finally, when the hook was pulled

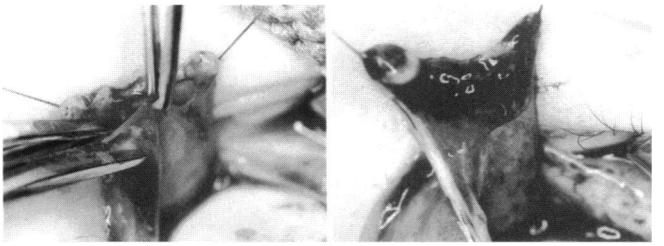

**FIGURE 3**
Removal of muscle capsule internal to muscle prior to attachment to sclera. This is done in all routine cases, as well as during repair of lengthened scar.

anteriorly, an insertion with a lengthened scar usually allowed significant anterior movement (Fig 20), unlike normally healed tendon (Fig 21).

In some cases, the scar segment was so difficult to see that the tendon had to be disinserted and viewed from the underside to discern the transition from normal tendon fibers to scar (Figs 22 and 23). Another clue to the presence of a lengthened scar segment was a greater-than-expected distance (more than the muscle's usual tendon length) from the musculotendinous junction to the sclera. When the lengthened muscle scar was the result of an original resection, muscle blended directly into scar, without intervening tendon (Fig 24). When the tendon was disinserted from the sclera, the lengthened scar tissue could be cut with less resistance than well-healed or fresh tendon.

Most lengthened scar segment cases had less dense scar tissue in the conjunctiva and surrounding the muscle than is generally encountered in reoperation cases. With increased experience, the surgeon was usually able to accurately predict the presence of a lengthened scar by the ease of dissection of surrounding scar tissue, before reaching the muscle.

Sometimes the preoperative history and examination suggested scar stretch, but adhesive syndrome[4] of the antagonist muscle was found instead. The scar tissue in these cases was usually denser and more difficult to dissect.

*Surgical Repair Technique*

Scars that were found to be lengthened or stretched were repaired by excising all scar tissue external to and loose connective tissue (capsule and intermuscular septum) internal to the tendons for a distance of 3 to 4 mm proximal to the insertions (see Fig 3). The muscle tendons were reattached firmly to sclera with suture in standard crossed-swords fashion.[5] In each case, an extra central lock bite was used to support the center of the tendon (Fig 25). Absorbable sutures (6-0 polyglactin) were used in 64

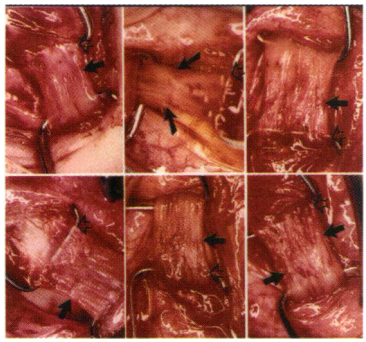

**FIGURE 4**

Composite photograph of lengthened scars of medial rectus in 6 cases, prior to repair. Solid arrows indicate junction between normal tendon and scar tissue. Open arrows indicate attachment site of scar to sclera.

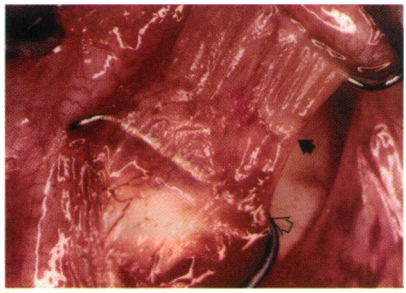

**FIGURE 5**

Enlargement of photograph in Fig 4 (see also Fig 40). Solid arrow indicates junction between normal tendon and scar tissue. Open arrow indicates attachment site of scar to sclera.

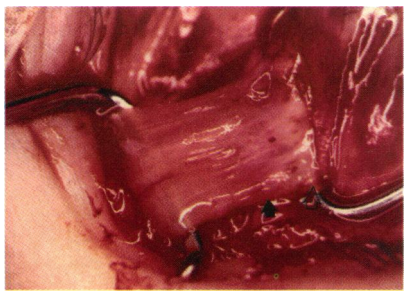

**FIGURE 6**

Enlargement of photograph in Fig 4 (see also Fig 40). Solid arrow indicates junction between normal tendon and scar tissue. Open arrow indicates attachment site of scar to sclera.

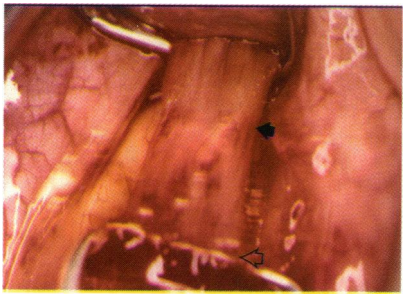

**FIGURE 7**
Enlargement of photograph in Fig 4 (see also Fig 40). Solid arrow indicates junction between normal tendon and scar tissue. Open arrow indicates attachment site of scar to sclera.

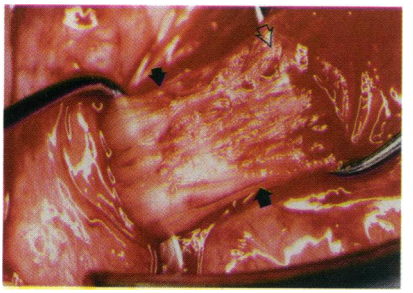

**FIGURE 8**
Enlargement of photograph in Fig 4 (see also Fig 40). Solid arrow indicates junction between normal tendon and scar tissue. Open arrow indicates attachment site of scar to sclera.

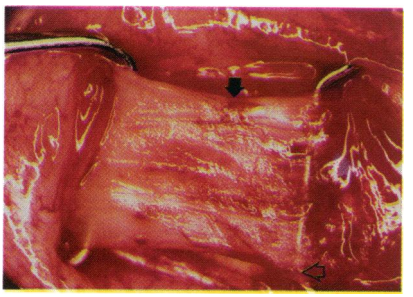

**FIGURE 9**
Enlargement of photograph in Fig 4 (see also Fig 40). Solid arrow indicates junction between normal tendon and scar tissue. Open arrow indicates attachment site of scar to sclera.

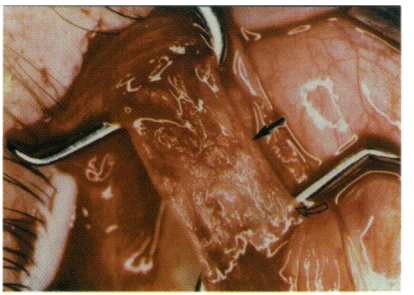

**FIGURE 10**
Lengthened or stretched scar segments of lateral rectus. Solid arrow indicates junction between normal tendon and scar tissue. Open arrow indicates attachment site of scar to sclera.

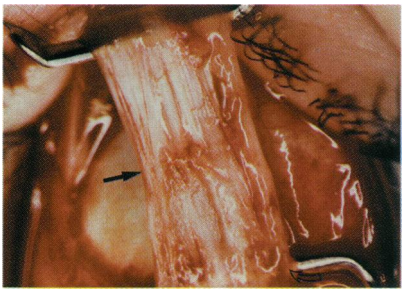

**FIGURE 11**
Lengthened or stretched scar segments of lateral rectus. Solid arrow indicates junction between normal tendon and scar tissue. Open arrow indicates attachment site of scar to sclera.

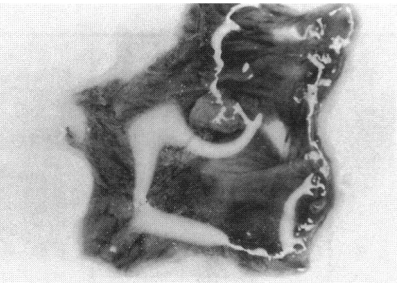

**FIGURE 12**
Amorphous segment of scar tissue after excision. Note that linear arrangement of collagen is lost when tension is eliminated.

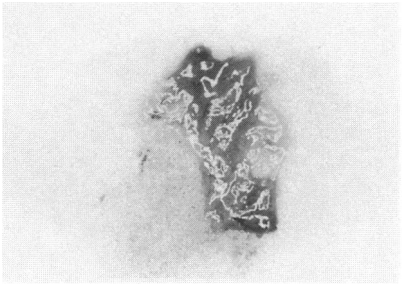

**FIGURE 13**
Amorphous segment of scar tissue after excision. Note that linear arrangement of collagen is lost when tension is eliminated.

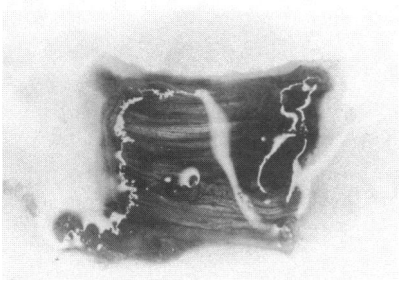

**FIGURE 14**
Normal segment of tendon after excision during routine extraocular muscle resection. Note that regular orientation of collagen fiber bundles is retained in absence of tension.

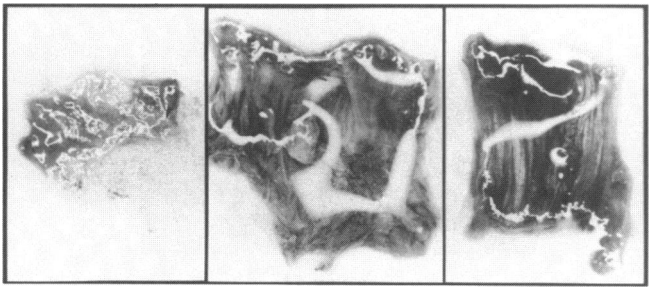

**FIGURE 15**
Composite of Figs 12 to 14.

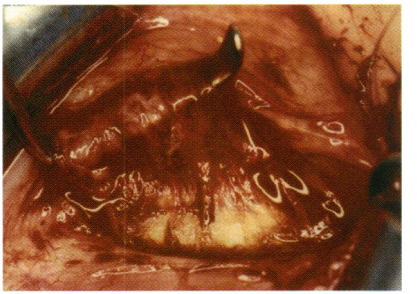

**FIGURE 16**
When muscle hook is pulled outward from sclera, insertion with a lengthened scar is seen to lift up (see Figs 33, 35 and 39).

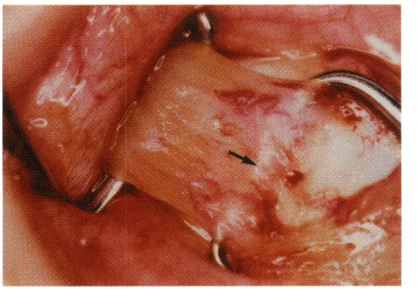

**FIGURE 17**
Despite substantial outward force on normally healed tendon, this lifting of insertion is not seen, as in this reoperation case for strabismus undercorrection. Arrow indicates scleral insertion.

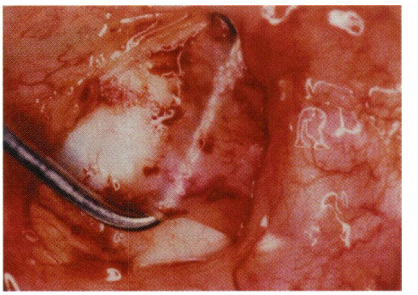

**FIGURE 18**
Despite substantial outward force on normally healed tendon, this lifting of insertion is not seen, as in this reoperation case for strabismus undercorrection.

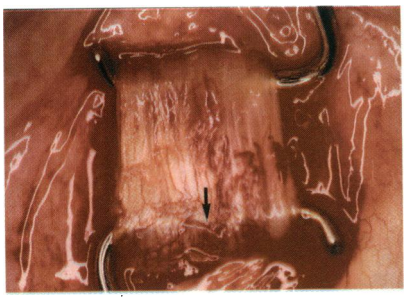

**FIGURE 19**

Normal medial rectus tendon also shows no tendency to lift off sclera with pull on insertion (arrow).

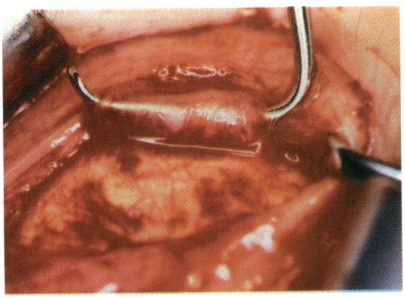

**FIGURE 20**

Anteriorly directed pull on insertion of a lengthened scar muscle causes anterior rolling movement. This was not seen in well-healed tendon during strabismus reoperation due to undercorrection (see Fig 21).

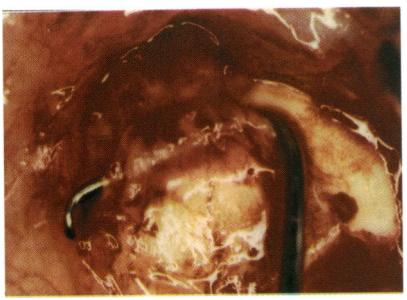

**FIGURE 21**

No anterior rolling movement was seen in well-healed tendon as in this strabismus reoperation due to undercorrection.

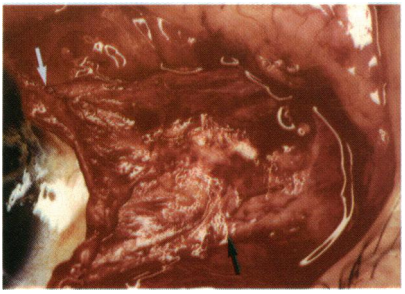

**FIGURE 22**

Although scar lengthening was suspected by history of this strabismus overcorrection case, diagnosis was not apparent after initial inspection of insertion. Scar-to-tendon junction (arrow) of this right lateral rectus is indistinct. Insertion has been detached from sclera and is suspended on 6-0 polyglactin suture (white arrow).

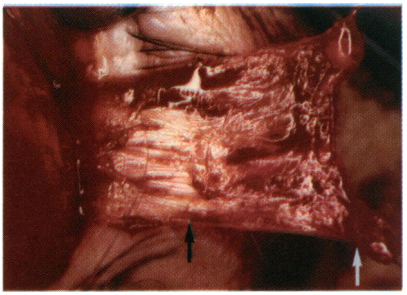

**FIGURE 23**

After inspection of inner surface of lateral rectus, amorphous scar segment and transition to normal tendon (arrow) become clear. White arrow indicates scar insertion, suspended on suture.

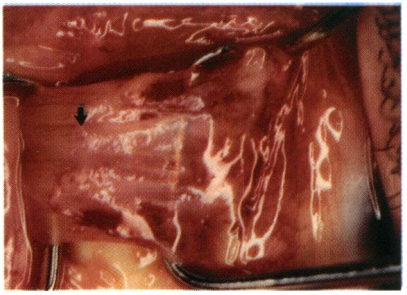

**FIGURE 24**

This medial rectus had originally been resected prior to scar lengthening and recurrence of deviation. Scar blends directly (solid arrow) into muscle tissue, without intervening tendon, which was excised at original surgery. Open arrow indicates insertion of scar onto sclera.

cases. Several recurrences were observed following repair with excision of the scar segment and firm reattachment of muscle to sclera, and it was felt that prolonged support of the attachment site was needed. Beginning in 1995, repairs were performed with nonabsorbable sutures: 17 with 6-0 braided polyester (Fig 26), 1 with 6-0 clear polypropylene alone, and 52 with combined tandem 6-0 clear polypropylene and 6-0 polyglactin (Fig 27). For all cases sutured with polypropylene, after the knot was tied on the muscle, the sutures were passed under the muscle belly and retied 7 to 8 mm posteriorly, to prevent suture end erosion through the conjunctiva (Fig 28). The use of polypropylene sutures in combination with polyglactin sutures was necessary because polypropylene sutures alone are too slippery to tie. Clear polypropylene sutures were used for anteriorly positioned muscles to prevent these sutures from showing through the conjunctiva. Polyester sutures were used for posteriorly attached muscles and inferior and superior recti, which are hidden by the eye lids.

The amount of muscle advancement was determined by preoperative measurements and routine surgical tables (MM Parks, Lancaster Course, Colby College, Waterville, Maine, August, 1985), but the plan was often adjusted intraoperatively to account for factors such as muscle contracture. The goal was a physical centering of the eye intraoperatively as judged by forced ductions, the spring-back test,[6] and corneal light reflexes.

One hundred twenty-four procedures were performed by the author, who used no postoperative corticosteroids, and 10 procedures were performed by another surgeon, who used topical corticosteroids for 10 days postoperatively.

Pathologic examinations were performed in specimens from 82 cases. An additional 7 specimens were used in tissue culture studies.

#### TISSUE CULTURE STUDIES

Cells from 7 excised scar specimens and 3 normal tendon resection specimens (controls) were cultured in Dulbecco minimal Eagle medium (DMEM) with 10% fetal bovine serum. The cells were passaged and medium was collected from each culture after 7 days. The activity of matrix metalloproteinases (MMPs) in the medium was assayed using gelatin and casein zymography.[7,8] Western blot was used for the identification of MMP1, MMP2, and MMP9 in the medium. Levels of MMPs and inhibitors (tissue inhibitors of metalloproteinases, ie, TIMP1 and TIMP2) were determined in cells and tissue extracts by Western blot.

#### ANIMAL STUDIES

##### *Development of an Animal Model*

In an attempt to create an animal model of scar lengthening after strabismus

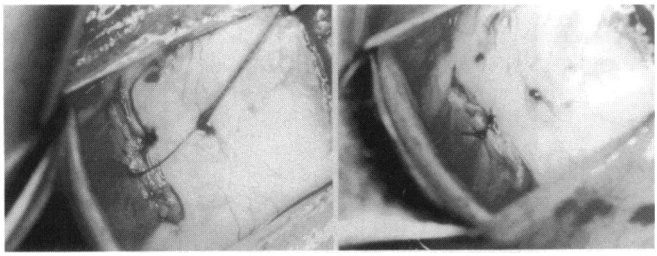

**FIGURE 25**

After reattaching muscle to sclera with standard crossed-swords technique,[5] central knot is tied, and one strand is passed through center of tendon (see Fig 25, left). When this extra center bite is tied (see Fig 25, right), muscle receives additional support, as well as direction of cut end of tendon toward sclera, potentially reducing gap formation.

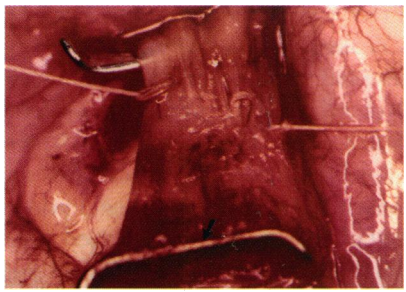

**FIGURE 26**

Braided polyester suture placed in inferior rectus tendon in overcorrection case, prior to excision of lengthened scar segment. Arrow indicates scleral insertion of scar.

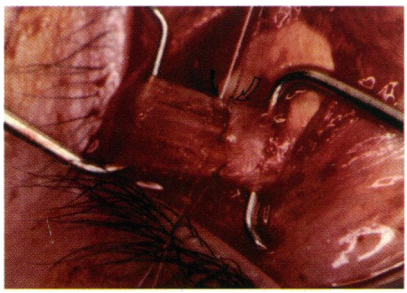

**FIGURE 27**

Clear polyproplyene suture (solid curved arrow) in tandem with polyglactin (open curved arrow) placed in lateral rectus tendon prior to excision of scar segment. Insertion is indicated by a solid straight arrow.

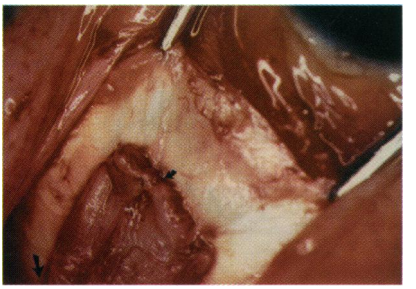

**FIGURE 28**
After scar excision of muscle (also depicted in Fig 38) and reattachment to sclera with polypropylene and polyglactin. Short curved arrow indicates central lock bite. Straight arrow points to site where polypropylene suture ends are retied after being passed under muscle belly. This prevents erosion of suture ends through conjunctiva.

surgery, the superior rectus muscle was resected 7 mm in standard fashion in both eyes of 10 New Zealand white rabbits using 6-0 polyglactin suture. One randomly selected resected muscle of each animal received a subconjunctival injection of 500 U of collagenase in phosphate-buffered saline (PBS) once a week during weeks 2, 3, 5, and 6 postoperatively. The contralateral resected muscle of each animal received injections of saline on the same schedule as a control. The animals were sacrificed 10 weeks postoperatively.

The surgical reattachment sites were excised, examined grossly, and photographed. Half of the collagenase-treated sites and half of the saline-treated control sites were fixed in 3% glutaraldehyde in PBS. The fixed tissue was embedded in HistoResin, sectioned, and stained with hematoxylin and eosin or toluidine blue.

*Creep Analysis*. Two unoperated New Zealand white rabbits were sacrificed, their eyes enucleated, and their normal superior rectus muscles with insertion sites excised. The 5 remaining operated and 4 unoperated muscle insertion sites were analyzed for creep behavior over an 8-week period. Each sclera/scar/muscle junction was marked with 2 parallel black tattoo lines 5 mm apart. The specimens were then suspended in an aqueous environment under a constant 27-g load at 37°C.[9] The distance between the tattoo lines on each specimen was measured with a caliper micrometer several times a week over the 8-week period. Means were computed for the length and width of the specimen test area for the collagenase-injected and saline-injected groups.

*Absorbable Versus Nonabsorbable Sutures in the Animal Model*

The animal model described above was used to compare the use of absorbable and non-absorbable sutures in the prevention of scar lengthening.

The superior rectus muscles were resected bilaterally in 2 rabbits; 1 muscle was attached with 6-0 polyglactin suture and the contralateral muscle with 6-0 braided polyester suture. Collagenase was injected subconjunctivally over each reattachment site once a week during weeks 2, 3, 5, and 6 postoperatively. At 10 weeks, the animals were sacrificed and the attachment sites dissected, examined grossly, and prepared for creep analysis.

## RESULTS

### CLINICAL STUDIES

#### *Preoperative Findings*

*Versions.* Versions were normal in 42 cases, minimally limited (trace to 1- on a scale of normal to 4-) into the field(s) of action of the stretched scar muscle(s) in 64 cases, and moderately reduced (2-) in 21 cases. Five patients had substantial limitation of versions (3- to 4-). Of these, 2 had third cranial nerve palsies, and 2 had severe adhesive syndrome,[4] which explained the version limitations. Versions were undocumented in 2 patients.

*Amblyopia or Other Visual Impairment.* Amblyopia or other visual impairment was absent in 78 cases, mild (visual acuity of 20/40 or better) in 29, moderate (20/50 to 20/80) in 12, marked (20/100 to 20/400) in 7, and extreme (count fingers or no light perception) in 8. Preoperative visual impairment did not significantly affect postoperative success (Table I).

#### *Intraoperative Findings*

A total of 134 stretched scar repairs were performed on 198 muscles in 123 patients. Seventy-three procedures involved 1 muscle, 59 involved 2 muscles, and 1 involved 3 muscles. One patient underwent repair of stretched scars on 4 muscles. One hundred forty-eight medial recti (71 right, 77 left), 41 lateral recti (20 right, 21 left), 7 inferior recti (3 right, 4 left), 1 superior rectus, and 1 inferior oblique were involved.

Symmetrical scar stretch was seen on the same muscle of both eyes in 52 cases, usually due to previous bilateral rectus muscle recession. Forty-five cases had bilateral medial rectus scar stretch, and 7 had bilateral lateral rectus scar stretch.

One patient had definite undercorrection of a previously resected muscle, which was the only muscle previously operated on in that eye. Five cases with documented histories in which the same eye had undergone recession of 1 muscle and resection of its antagonist showed undercorrection; the scars of the resected muscles had stretched, but those of the recessed muscles had not. Three patients developed overcorrection due to scar stretch of the previously recessed medial recti, with well-healed,

TABLE I: AMBLYOPIA*

| | SUCCESSFUL | UNSUCCESSFUL |
|---|---|---|
| Normal vision | 68 | 10 |
| Mild impairment (20/25 to 20/40) | 18 | 11 |
| Moderate impairment (20/50 to 20/80) | 12 | 0 |
| Marked impairment (20/100 to 20/400) | 5 | 2 |
| Extreme impairment (CF to NLP) | 7 | 1 |

* Differences are not significant (exact chi-square test). CF, count fingers; NLP, no light perception

previously resected, lateral recti. Four had scar stretch of both the medial and lateral rectus of 1 eye, suggesting initial recess-resect procedures.

The mean medial rectus scar lengthening was 4.13 mm (range, 1 to 10 mm); lateral recti averaged 4.57 mm of lengthening (range, 1.5 to 9 mm), and inferior recti 3.57 mm (range, 1 to 6.5 mm).

*Incidence of Scar Lengthening in Secondary Strabismus*

An accurate calculation of the incidence of lengthened scars in the author's practice is not possible, as only case records positive for this complication were retained. When compared to all reoperation cases, cases with lengthened scars were relatively uncommon–possibly 10% to 20%. As a subset of overcorrection cases, the incidence rises to about 50%. With experience, the author was able to predict the presence of a lengthened scar with 80% to 90% accuracy on the basis of history and clinical findings.

Incidental scar stretch, that is, scar elongation not contributing to the direction of strabismus being corrected, was found in 9 cases. When the elongation was judged stable and helpful to the desired surgical goal, it was not repaired, but when repositioning of affected muscle(s) was required, the lengthening was repaired and repositioning adjusted accordingly.

Several patients suspected initially of having lengthened scar(s) were found to have adhesive syndrome[4] of the antagonist muscle. This was the most common cause of false preoperative diagnosis of lengthened or stretched scar. It is estimated that this condition was found in about 10% of suspected scar stretch cases.

A few patients who were suspected of scar stretch instead showed migration of the muscle insertion site on the sclera.

*Patients With Previous Adjustable Suture Surgery.* Seven patients had undergone previous adjustable suture surgery on the involved

muscle(s). They had lengthened scars that were clinically indistinguishable from the other stretched scar cases. These scars were not pseudotendons between the tendon and original insertion. Scar tissue was attached to the sclera well behind the original insertion in each case.

One of these patients was especially interesting, as she had undergone 2 previous strabismus procedures. In the first procedure, both medial recti were recessed, with direct suturing of tendons to sclera. During the second procedure, the left medial rectus was advanced with direct suturing, but the left lateral and right superior recti were recessed on adjustable sutures. She gradually developed esotropia and a deficit of upgaze on the right. When the muscles were explored during repair surgery, the directly sutured medial recti showed no signs of scar lengthening, but each of the previously adjusted muscles showed lengthened scars of 6 mm (see Figs 22 and 23).

*Follow-up*

Postoperative follow-up after stretched scar repair averaged 11.4 months for all cases (range, 1 to 69 months). Mean follow-up was 14.8 months for the absorbable suture group and 8.6 months for the nonabsorbable suture group.

*Complications*

*Conjunctival Scar Tissue.* Occasionally, thickened scar tissue of the conjunctiva and Tenon's capsule overlying the repaired post-scar-stretch muscle created a cosmetic problem. This problem was usually the result of adhesive syndrome from the original surgery, with penetration of Tenon's capsule and prolapse of orbital fat. Conjunctival recession was performed at the time of stretched scar repair in 10 patients because of this complication from previous surgery. Two other patients chose to undergo secondary conjunctival recession at a later date.

*Unrecognized Scar Stretch.* Two patients remained exotropic after unilateral repair of a stretched medial rectus. Each was later found to have a stretch of the contralateral medial rectus, which was repaired in a second procedure.

*Secondary Scar Stretch of Antagonist Muscle.* During stretched scar repair, the antagonist of the muscle being repaired was occasionally recessed to relieve contracture. Although nonabsorbable suture was used for the stretched scar repair, it was not initially used for other muscles. In 2 cases, the recessed antagonist muscles later developed stretches, requiring re-repair. The repaired stretched scar muscles did not restretch.

*Pulled-in-two Syndrome.* Two stretched scar patients developed rupture of a medial rectus at its musculotendinous junction intraoperatively. A third patient developed a partial separation that did not pull apart completely. These 3 patients were 6, 14, and 19 years old. In the

literature, this complication is considered rare and usually seen only in the elderly.[10,11]

*Absorbable Versus Nonabsorbable Sutures in Cases of Suspected Restretch* Successful repair was defined in terms of the patient noting substantial correction of the preoperative deviation and being satisfied with the outcome. These patients achieved undercorrection of their preoperative deviations to within 10 prism diopters (Δ ) or overcorrection to 14Δ.

The first 64 stretched scar cases were repaired with absorbable suture. By 1995, however, it became evident that late recurrence was common despite good initial postoperative alignment, and the use of nonabsorbable suture was implemented (Table II).

*Absorbable Sutures.* In the absorbable suture group, 43 of 62 procedures with 1 month or more of follow-up (69%) and 21 of 28 (75%) with 12 months or more of follow-up were judged successful by the patient and the surgeon. One of these patients was an exception to the definition of success as given above; she had an undercorrection of 18Δ, but the preoperative deviation was so large that she was satisfied with the improvement.

Restretch was suspected when there was postoperative drift of 6Δ or more, with the return of the original deviation. Twenty-six cases (41%) had suspected restretch after absorbable suture repair. Six of these were then documented (see below).

Two other patients underwent re-exploration during surgery on additional muscles 1 year after absorbable suture stretched scar repair and were found to have firm re-attachments of the previously stretched muscles (2 medial and 2 lateral recti).

*Nonabsorbable Sutures.* In the nonabsorbable suture group, 62 of 68 procedures with 1 or more months of follow-up (91%) and all 19 with 12 or more months of follow-up (100%) were judged successful. One was overcorrected, 4 showed postoperative drift that indicated possible restretch, and 1 of these 4 had restretch documented (see below). Three underwent repeat surgery for overcorrection of the stretched scar deficit or on other muscles, which permitted documentation of the success of the repair, (ie, no recurrence of scar lengthening). Five cases in which restretch occurred after absorbable suture repair responded well to repair with nonabsorbable sutures (see below). One patient considered to be a success had an overcorrection of 20Δ, which required repeat surgery on the same muscles to achieve orthotropia, but showed no recurrence of stretch.

*Stability of Postoperative Alignment.* Good postoperative alignment is an imperfect measure of the success of repair of the lengthened scar abnormality, because a successfully repaired muscle may have been

TABLE II: OUTCOMES WITH ABSORBABLE AND NONABSORBABLE SUTURES

| PARAMETER | ABSORBABLE SUTURES | NONABSORBABLE SUTURES |
|---|---|---|
| Total cases | 64 | 70 |
| **Cases with 1 or more months of follow-up** | **62** | **68** |
| Successful | 43 (69%) | 62 (91%) |
| Restretch° | | |
| Suspected (not proven) | 20 | 3 |
| Documented | 6 | 1 |
| Mean follow-up (months) | 14.8 | 8.6 |
| **Subset of cases with 12 or more months of follow-up** | **28** | **19** |
| Successful | 21 (75%) | 19 (100%) |
| Restretch° | | |
| Suspected (not proven) | 9 | 1 |
| Documented | 3 | 0 |

° Some patients with suspected restretch remained successfully aligned and thus appear in both the "successful" and "restretch" groups.

unfortunately replaced in a position that resulted in an unplanned overcorrection or undercorrection. The stability of postoperative alignment may be a more significant measure of potential restretch, and this parameter was evaluated (Table III). Only patients who had surgery limited to nonantagonist muscles (eg, both medial recti or both lateral recti or 1 muscle alone), were included in the analysis. The mean difference between the final postoperative total correction and the total correction on the first postoperative day was -4.4Δ in the absorbable suture group and +1.1Δ in the nonabsorbable suture group. In this calculation, a negative value indicated drift toward the preoperative direction and possible restretch. The difference between the absorbable and nonabsorbable suture groups was significant ($P$=.008, $t$ test).

Additionally, there was a greater proportion of patients with large negative postoperative drift (-10Δ or more) in the absorbable suture group [12 (27%) of 44] than in the nonabsorbable suture group [6 (11%) of 55]. The difference was significant (P=.03, exact chi square test). Further analysis revealed that 25 (57%) of 44 patients in the absorbable suture group and 17 (31%) of 55 in the nonabsorbable suture group had at least some negative postoperative drift. This difference was also significant ($P$=.014; exact chi square test).

TABLE III: STABILITY OF POSTOPERATIVE ALIGNMENT

| PARAMETER | ABSORBABLE SUTURES | NONABSORBABLE SUTURES | P VALUE |
|---|---|---|---|
| Cases with single-muscle or symmetrical original surgery | 44 | 55 | |
| Mean of total final correction minus correction on first postoperative day (Δ) | -4.4 | +1.1 | 0.008° |
| Cases with 10 Δ or more negative drift | 12 (27%)† | 6 (11%)† | 0.03‡ |
| Cases with any negative drift | 25 (57%) | 17 (31%) | 0.014‡ |

° *t*-test

† Some patients with marked negative change from the first postoperative visit to the final visit achieved orthotropia and had successful, stable outcomes. They are not likely to have restretched, but their numbers are included in the analysis.

‡ Exact chi-square test

Δ prism diopters

*Absorbable Versus Nonabsorbable Sutures in Cases of Documented Restretch*

*Absorbable Sutures.* Six patients who underwent repair of scar stretch with absorbable sutures and developed recurrence of the original overcorrection had restretch documented on re-exploration.

One patient was an 11-year-old boy who had undergone esotropia surgery at 9 months of age and was aligned (by parental history) for several years before exodeviating. By 4 years of age, he measured an exotropia of 14 Δ (XT=14), and at 10 years, XT=20, with mild bilateral adduction limitation. During surgery by the author in 1994, bilateral stretched medial rectus scars were found (right medial rectus, 2.5 mm; left medial rectus, 6 mm) and repaired as outlined above, with 6-0 polyglactin suture. Two weeks later, alignment was XT=8, but 7 months later, it was XT=20 with adduction limitation in the left eye. The left medial rectus was re-explored, and a 10 mm stretched scar was found and repaired with polyglactin. Alignment was XT=6 at 1 week, XT=10 at 3 months, XT=25 at 8 months, and XT=30 at 2 years after surgery. In 1996, he underwent a third re-exploration of the medial recti, and bilateral recurrent stretches were found [right medial rectus, 3.5 mm, (Fig 29); left medial rectus, 3 mm, (Fig 30)]. One day after bilateral repair with nonabsorbable suture, he measured

XT=18, and 2 months later, XT=10. At this writing, 2.5 years after the final procedure, he is orthotropic, with 400 seconds of stereopsis (Fig 31).

Four patients had their first stretch(es) repaired with polyglactin, followed by recurrence of their deviations within 1 year. In all cases, nonabsorbable suture was used in the second repair procedure, and all have shown stable postoperative alignment. One of these patients has had 19 months of follow-up with no strabismus recurrence; the other 3 have had less than 1 year of follow-up at this writing. The only difference in technique between the first and second stretched scar repair was the use of nonabsorbable suture material in the second procedure.

The sixth recurrent stretched scar patient underwent deliberate overcorrection with polyglactin at the second repair, as described below in the section entitled "Chronic active hepatitis." This case predated the use of nonabsorbable sutures in this study.

*Nonabsorbable Sutures.* In this group, restretch was documented in a young man with partial third cranial nerve palsy. The muscle involved was a very tight medial rectus that had been resected previously. The first stretched scar repair was for a 5 mm scar of the left medial rectus; 6-0 clear polypropylene and 6-0 polyglactin sutures were used. Exotropia gradually recurred (XT=20) despite initial orthotropia. On re-exploration, a 2 mm stretch was found. The polypropylene suture was found securely within tendon and not attached to sclera. A second repair was performed with 6-0 braided polyester. Exotropia recurred, but was less severe (XT=12).

*Sensory Results*

Among the 122 patients in whom sensory testing was performed post-operatively, 52 had no evidence of fusion, 12 had peripheral fusion without stereopsis, 41 had 400 to 1800 seconds of stereopsis, 8 had 100 to 140 seconds of stereopsis, and 9 had 40 to 60 seconds of stereopsis. Exact chi-square testing showed no significant differences in surgical success rate when the groups were subdivided in terms of both stereopsis and fusion (Table IV).

*Interesting Cases and Associations*

*Twins.* Two pairs of twins were operated on. One pair of identical twin boys with juvenile non-accommodative esotropia underwent 4.5 mm bimedial recessions at 12 years of age by the same surgeon on the same day. Initially slightly undercorrected, they then maintained ocular alignment for several years and gradually developed exodeviations. One of the twins measured XT=45 and the other XT=14; both had mild adduction limitations. At surgery, the child with the larger deviation had a 5 mm stretched scar of the right medial rectus (Figs 32 and 33) and a 4 mm stretch on the left. His twin had 3 mm scar stretches of each medial

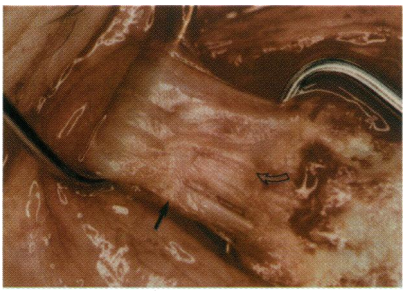

**FIGURE 29**

Right medial rectus of patient after third restretch, before last repair. Solid arrows indicate scar-to-tendon junction. Open arrows indicate scleral attachment site of scar.

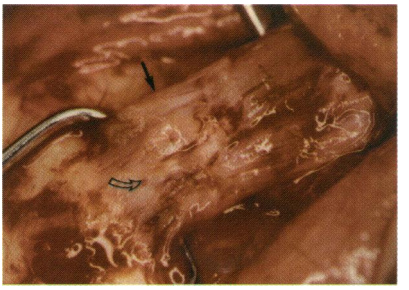

**FIGURE 30**

Left medial rectus of same patient as in Fig 29. Solid arrows indicate scar-to-tendon junction. Open arrows indicate scleral attachment site of scar.

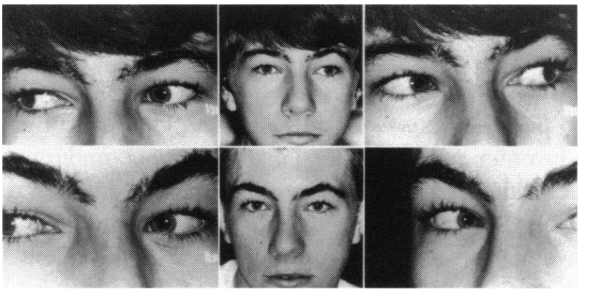

**FIGURE 31**

Patient whose muscles are depicted in Figs 29 and 30. Top 3 photographs are preoperative. Bottom 3 photographs were taken 2.5 years after final surgery. Photographs on left indicate right gaze; photographs on right indicate left gaze.

TABLE IV: SENSORY RESULTS IN 122 PATIENTS

| | SUCCESSFUL | UNSUCCESSFUL |
|---|---|---|
| 40 to 60 seconds | 9 | 0 |
| 100 to 140 seconds | 7 | 1 |
| 400 to 1800 seconds | 34 | 7 |
| Peripheral fusion | 9 | 3 |
| No fusion | 42 | 10 |

Differences are not significant (exact chi-square test).

rectus (Figs 34 and 35). Repair with nonabsorbable sutures produced intermittent esotropia [E(T)] of 10 in the former child, and E(T) of 6 in the latter at 2 months of follow-up. These measurements remained the same 1.5 years after surgery.

A pair of fraternal twins of opposite sexes with congenital esotropia underwent bimedial recession at 1 year of age by the same surgeon on the same day. Both were aligned well initially, but the boy developed gradual exodeviation (25Δ) (Fig 36), whereas the girl did not (Fig 37). Both also developed inferior oblique overaction. The boy had a 5 mm scar stretch of each medial rectus (Figs 38 and 39), and after repair with nonabsorbable suture, maintained an alignment of ET=6.

Finding stretched scars in twins supports the intriguing possibility of genetic predisposition toward scar lengthening.

*Uremia.* A diabetic woman underwent recession of the left lateral rectus for exotropia and remained aligned for 10 months. Shortly after developing renal failure and uremia, which is known to weaken scar tissue,[12] she became diplopic due to esotropia. Her small scar stretch was repaired with nonabsorbable suture, correcting the problem, and the uremia was corrected after renal transplantation. There was no recurrence of strabismus 1 year after surgery.

*Crouzon's Syndrome.* A 14-year-old boy with Crouzon's syndrome presented with gradually increasing exotropia (40Δ). At 3 years of age, he had undergone strabismus surgery for esotropia in his left eye. Surgical reexploration disclosed a 4.5 mm stretch of the left medial rectus scar (Fig 40). The stretched scar was excised and the muscle advanced and reattached with nonabsorbable suture. The lateral rectus was recessed 4 mm. Three months later, exotropia was 8Δ; 33 months after surgery, it was still 8Δ. His mother, a geneticist, was not surprised to hear of the scar tissue abnormality, pointing out that Crouzon's syndrome is associated with an abnormal fibroblast growth factor receptor. She then demonstrated the widened scalp scars from his original craniofacial surgery (Fig 41).

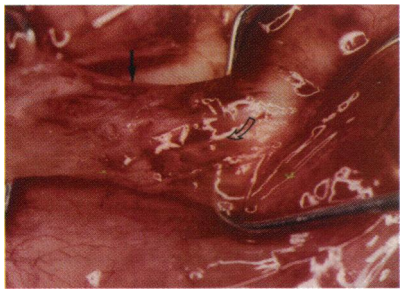

**FIGURE 32**

Right medial rectus of identical twin with larger deviation. Solid arrow indicates junction between tendon and scar. Open arrow indicates scar insertion on sclera

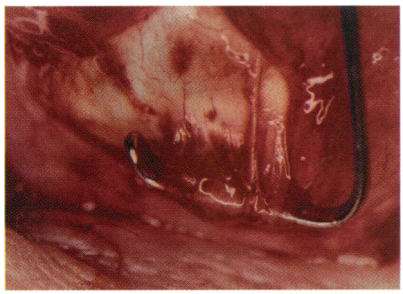

**FIGURE 33**

Insertion of muscle depicted in Fig 32, showing lifting away from sclera with tension on hook.

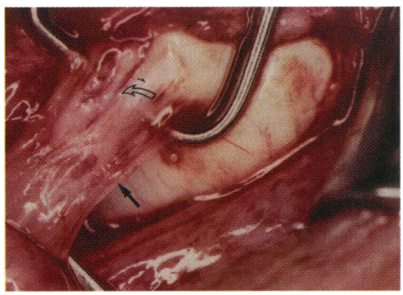

**FIGURE 34**

Right medial rectus of second twin with smaller deviation. Solid arrow indicates junction between tendon and scar. Open arrow indicates scar insertion on sclera.

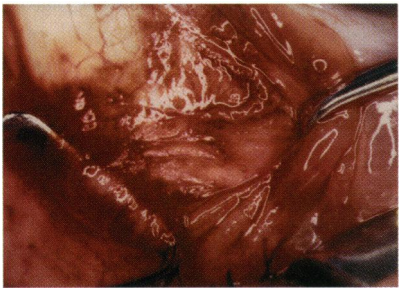

**FIGURE 35**
Same muscle as in Fig 34, showing pulling away from sclera with outward tension on hook.

*Chronic Active Hepatitis.* One of the documented restretch cases occurred in a 5-year-old girl with chronic active hepatitis. She underwent 6 mm recession of each medial rectus in June 1991 for deteriorated accommodative esotropia. Alignment was ET=6 the day after surgery, orthotropia the following week, and XT=6 1 month later. One year later, she measured XT=18, and at reoperation was found to have a scar segment between tendon and sclera of 3 mm on the right and 2 mm on the left. After scar excision and reattachment of each tendon to sclera with absorbable sutures, she was realigned, but 3 months later she measured XT=8, and 16 months postoperatively, XT=20. Surgical exploration revealed restretch, which was repaired with absorbable sutures and deliberate overcorrection to ET=25 on the first postoperative day, in the expectation that restretch would occur again. Ten months later she measured ET=12. This patient was 1 of the 10 cases treated with postoperative topical corticosteroids and was operated on before the use of nonabsorbable sutures was adopted in this study.

*Possible Ehlers-Danlos Syndrome.* An 18-year-old woman presented with a history of gradually increasing esotropia and diplopia. She had undergone bilateral 6 mm recessions of the medial recti for esotropia at age 4 years, followed several months later by bilateral 7 mm lateral rectus recession for consecutive exotropia. She felt that her eyes had remained aligned for 8 years, then began to esodeviate.

She measured a comitant esotropia of 30Δ, with mild limitation of adduction and abduction of both eyes. At surgery there was a striking lack of scar tissue. No incision lines from the previous surgeries were visible, and there was no resistance to hooking or exposure of any of the muscles. No scar tissue was present around the muscle insertions, and the only evidence of the previous surgeries was that the insertions were found close to the prior reported recession sites. A segment of amorphous

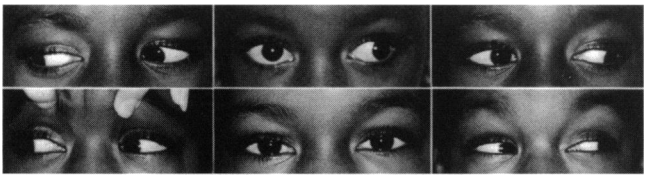

**FIGURE 36**
Boy with gradual consecutive exotropia due to lengthened scar of medial recti, preoperatively (above) and postoperatively (below). Photographs on the left indicate right gaze; photographs on the right indicate left gaze.

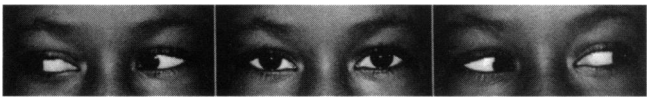

**FIGURE 37**
Twin sister of boy in Fig 36, who did not develop exotropia despite identical surgical history. Photograph on the left indicates right gaze; photograph on the right indicates left gaze.

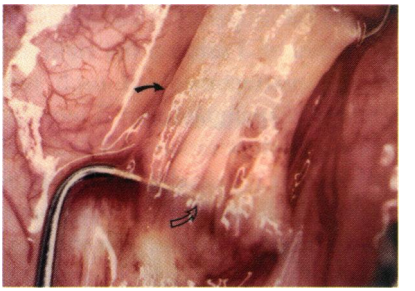

**FIGURE 38**
Medial rectus of child in Fig 36. Solid arrow indicates junction between tendon and scar; open arrow indicates scar insertion on sclera (see also Fig 28).

scar tissue was found interposed between the scleral attachment and normal tendon of each muscle. The scleral position of each medial rectus attachment was 7 mm behind the original insertion (1 mm behind each reported surgical attachment site of 6 mm), and the lateral recti were found 8.5 mm behind the original insertions (each 1.5 mm posterior to the 7 mm reattachment site reported by the previous surgeon). The stretched scar segment was 6.5 mm on the right medial rectus, 4 mm on the left medial rectus, 3 mm on the right lateral rectus, and 3.5 mm on the left lateral rectus. Each was repaired with 6-0 polyglactin suture, as described above. Each medial rectus was reattached 7 mm behind its original insertion, and the lateral recti were advanced to 4 mm behind each

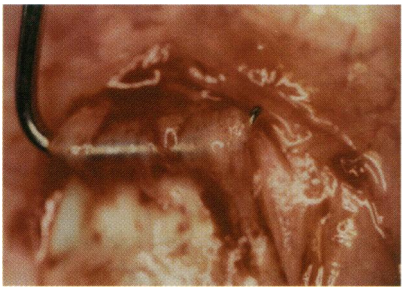

**FIGURE 39**
Pulling up with hook on insertion on same muscle as in Fig 38. Lax scar lifts away from sclera.

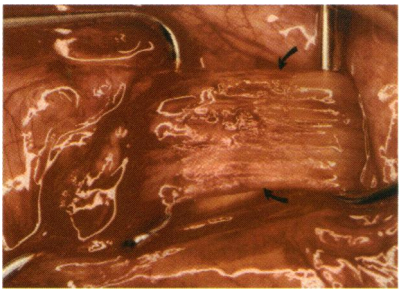

**FIGURE 40**
Medial rectus of Crouzon's syndrome patient (also seen in Fig 4.) Solid arrows indicate junction between sclera and tendon.

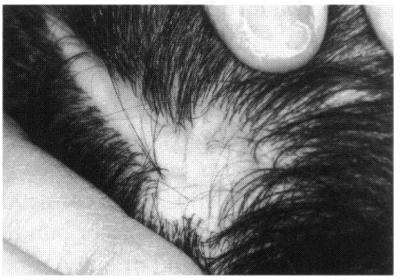

**FIGURE 41**
Scalp scars of patient with Crouzon's syndrome, which have widened over many years since craniofacial surgery.

original insertion. While the left medial rectus was engaged on the muscle hook, it pulled in two at the musculotendinous junction (a complication usually associated only with advanced age),[10,11] but was successfully retrieved and repaired.

One week after surgery, alignment was exophoria (X)=4. Eight months later, she measured esophoria (E)=4, with 800 seconds of stereopsis. Three and a half years after surgery, she measured E(T)=6, 800 seconds of stereopsis, and mild limitation of abduction and adduction of each eye, suggesting possible restretch. Pathologic examination of the excised segments showed fibrous tissue and no muscle fibers.

Although this patient was healthy with no overt abnormalities, her connective tissue was abnormal, and she may have a subclinical variant of a collagen disorder such as Ehlers-Danlos syndrome.[13]

*Thyroid Ophthalmopathy Treated With Orbital Irradiation.* A 53-year-old woman developed severe active thyroid ophthalmopathy and diplopia, with orbital pain that was controllable only by daily prednisone. Orbital irradiation was performed to control the active disease, after which the patient experienced resolution of pain and periocular inflammation. The diplopia persisted, however, and 9 months later, she underwent recession of the right medial rectus and both inferior recti. Postoperatively, she gradually developed an A-pattern exotropia with bilateral incyclotorsion. Two months after the first surgery, she underwent repeated surgery, during which bilateral scar stretches of the inferior recti were repaired with nonabsorbable sutures and the muscle insertions were displaced temporally to improve the torsion. Both the A-pattern and primary position torsion resolved and had not returned at 2.5 years of follow-up.

*Other Disorders.* One patient had Crohn's disease, another had Down's syndrome, and 3 had severe cerebral palsy. One had undergone a glaucoma shunt procedure that caused vertical strabismus requiring strabismus repair, which was followed by overcorrection due to inferior rectus scar stretch.

One patient who underwent repair of congenital ptosis and strabismus developed recurrence of ptosis concurrent with strabismus overcorrection due to scar stretch.

*Histopathology*

The specimens from 82 clinical cases underwent histopathologic examination. All showed diffuse dense connective tissue consistent with scar (Figs 42 and 43), without skeletal muscle fibers. The fiber bundles of normal resected rectus muscle tendons were larger and oriented more regularly (Fig 44) than those in the scar segments, but because normal tendon is also composed of dense connective tissue, there was little diagnostic information obtainable from these examinations.

**TISSUE CULTURE STUDIES**

In culture, the cells from clinical stretched scar specimens were oriented more randomly and contained larger amounts of extracellular matrix (Fig 45) than normal extraocular muscle tendon cells (Fig 46). Cultures of normal rectus tendon cells reached maximal numbers of cells after 4 days, whereas the stretched scar cultures showed cell numbers continuing to increase through day 5 (Fig 47). In most specimens tested, the total protein release into the medium was higher from stretched scar cells than from normal muscle tendon cells (Fig 48).

Total protein extracts from some stretched scar tissue specimens showed higher levels of metalloproteinases, including MMP1, MMP2 (Fig 49), and MMP9, by Western blot analysis, compared with extracts of normal rectus tendon. At later passages, the stretched scar cultured cells also showed higher levels of MMPs than the normal muscle tendon cells in culture (MMP2, Fig 50). Similarly, the medium from stretched scar cell cultures at later passages showed higher levels of MMP activity than the medium from normal cell cultures, and this correlated with higher levels of MMP2 (Fig 51) and MMP9 as determined by Western blot analysis. Levels of tissue inhibitor of metalloproteinase (TIMP1) were also higher in the stretched scar cells than in the normal cells, but the normal cells appeared to release more TIMP1 into the medium (data not shown). Levels of TIMP2 in the medium were similar for both cultures.

**ANIMAL STUDIES**

*Development of an Animal Model*

All the study (collagenase-injected) and control (saline-injected) eyes remained healthy throughout the 10-week observation period. Upon enucleation, gross examination of the collagenase-treated insertion sites showed increased connective tissue at the wound healing sites, similar in appearance to human stretched scar cases, compared with the control sites (Figs 52 and 53). Histologic analysis of the collagenase-treated muscle sites showed an increase in the amount of diffuse collagen at the insertions and decreased muscle integration with underlying sclera (Fig 54), compared with the saline-treated control sites (Fig 55).

Creep analysis of experimental and control insertion sites showed significantly greater creep, or stretch, in the collagenase-injected sites, compared with the controls (Figs 56 and 57).

*Absorbable Versus Nonabsorbable Sutures in the Animal Model*

Gross examination revealed longer and thinner scars in the eyes where the surgery was performed with absorbable sutures (Fig 58) compared with

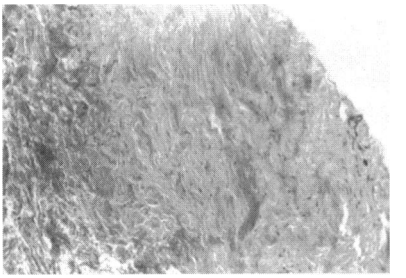

**FIGURE 42**
Histology of scar segment, showing wavy bundles of dense connective tissue.

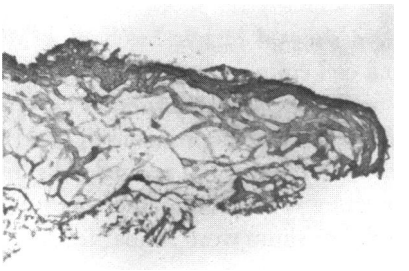

**FIGURE 43**
Histology of lengthened scar segment stained for type III collagen. Note irregular arrangement of collagen fibers.

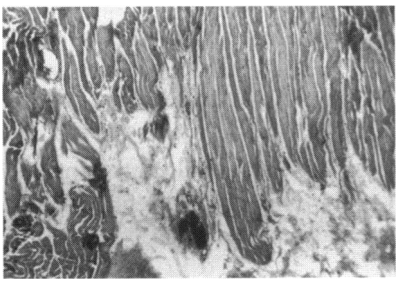

**FIGURE 44**
Normal extraocular muscle tendon histology following resection. Collagen bundles are larger and more regularly oriented.

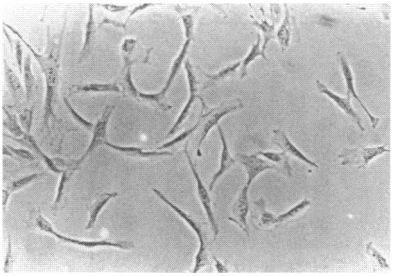

**FIGURE 45**

Cells from stretched scar specimens are irregularly oriented and demonstrated increased production of extracellular matrix compared to cells from normal tendon.

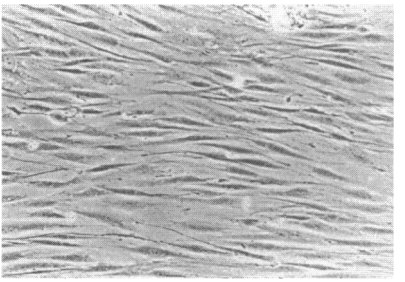

**FIGURE 46**

Cells from normal tendon arrange themselves more regularly in culture.

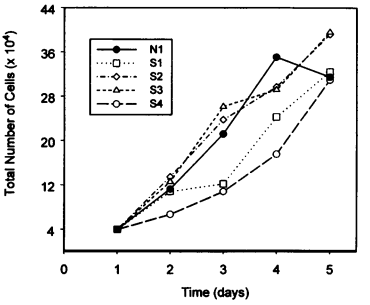


**FIGURE 47**

Normal (N) and stretched scar (S) cells were seeded at 4 x $10^4$ cells per well. Total number of cells in each culture was calculated for days 1 to 5.

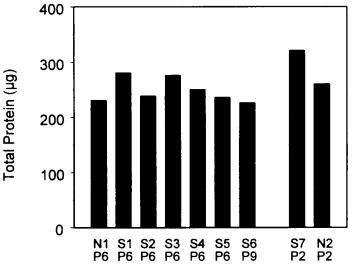


**FIGURE 48**

Total protein released into medium from normal (N) and stretched scar (S) cells at specific passage numbers (P).

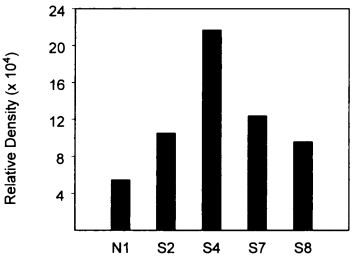


**FIGURE 49**

Western blot analysis of MMP2 in total tissue extract from normal (N) and stretched scar (S) tissues.

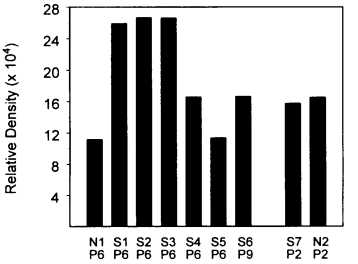


**FIGURE 50**

Western blot analysis of MMP2 in total protein extract from normal (N) and stretched scar (S) cultured cells at specific passage numbers (P).

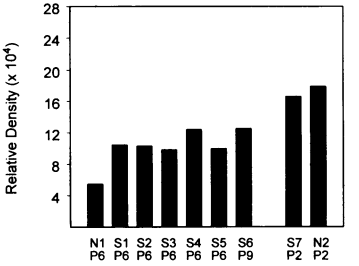


**FIGURE 51**
Western blot analysis of MMP2 released into the medium from normal (N) and stretched scar (S) cultured cells at specific passage numbers.

the surgery sites in which nonabsorbable sutures were used (Fig 59). Creep analysis is underway at the time of this writing.

## DISCUSSION

The lengthening or widening of a scar under tension is often termed stretching in the literature,[14-18] but perhaps should more properly be referred to as scar remodeling.[19] The underlying mechanisms are complex and probably include many aspects of wound healing, including collagen breakdown and synthesis, reorientation of fibers, and other processes, in addition to stretch or creep.

### PRINCIPLES OF WOUND HEALING

To understand the changes that may occur in the scar that secures the extraocular muscle to the sclera after strabismus surgery, an understanding of the principles of wound healing and the behaviors of tissues and scar under tension is needed.

#### *Normal Wound Healing*

The field of wound healing research has exploded in recent years. Despite greatly increased knowledge, however, many questions remain about the interrelated processes involved and how they are coordinated. Abnormalities of wound healing, as well as manipulations to correct them, are another complex area of current and future study. A comprehensive discussion of the intricacies of wound healing is beyond the scope of this thesis, but a brief synopsis is provided.

*Phases of Wound Healing.* Under normal circumstances, wound healing follows a predictable pattern of events, although the time course of these stages is variable depending on the type of tissue and the species.[19]

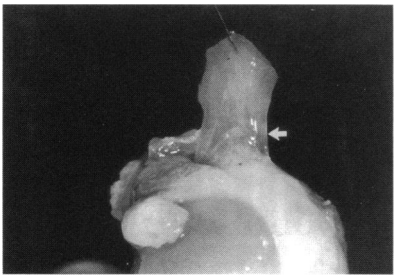

**FIGURE 52**
Insertion site after subconjunctival injection of collagenase following superior rectus resection. Thinned scar (arrow) separates muscle from sclera.

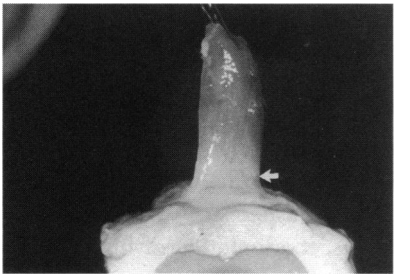

**FIGURE 53**
Insertion site (arrow) in control (saline-injected) superior rectus, which was also previously resected. Scar is shorter and denser with less separation of muscle from sclera.

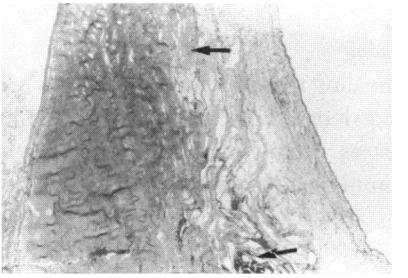

**FIGURE 54**
Histology of a collagenase-treated insertion site. Excess collagen (between arrows) separates muscle (bottom arrow) from sclera (top arrow).

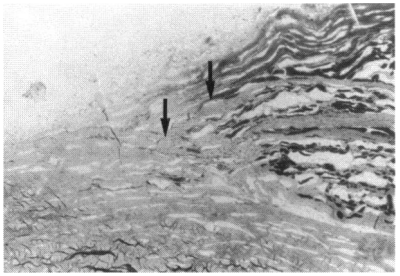

**FIGURE 55**

Histology of control, saline-injected insertion site. Muscle (right arrow) is separated from sclera (left arrow) by minimal scar.

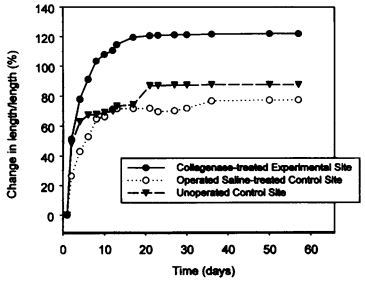


**FIGURE 56**

Creep behavior of explanted muscle/sclera junction shows a nearly 40% increase in creep/elongation of collagenase-treated experimental sites, compared with saline-treated operated controls and unoperated control. Values are means of 3 specimens; standard deviations are less than 5% in all cases.

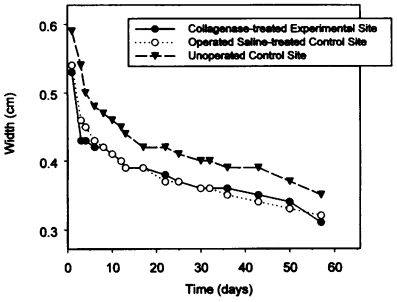


**FIGURE 57**

Decrease in width of muscle/sclera junction during creep analysis indicates that a thinning stretch occurred over time for all samples. Values are means of 3 specimens; standard deviations are less than 5% in all cases.

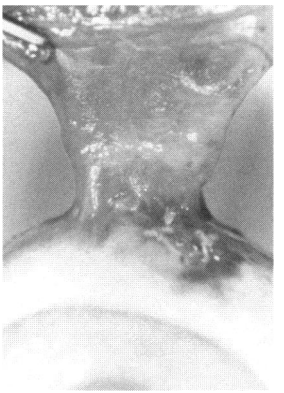

**FIGURE 58**
Insertion site after subconjunctival injections of collagenase. Original resection performed with nonabsorbable suture.

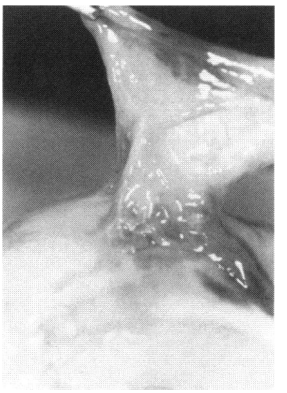

**FIGURE 59**
Same as Fig 58, but with original sugery performed with absorbable sutures. Scar tissue at insertion is thinner and longer than in Fig 58.

Following tissue injury, *coagulation* occurs, with involvement of catecholamines to produce vasoconstriction, vasoactive compounds that stimulate the movement of intravascular cells into the extravascular wound area, and platelets, which form the clot and release fibrin and several cytokines. The *inflammatory* phase begins with the migration of polymorphonuclear leukocytes (PMNs) into the wound at about 24 hours, followed by the appearance of macrophages, which peak in number by day 3.[20] Neutropenia does not impair ultimate wound healing,[21,22] but

macrophage depletion markedly reduces wound healing because macrophages are required for both wound debridement and fibroplasia.[20] *Fibroplasia* is the next phase, in which fibrous protein collagen is synthesized by fibroblasts that migrate from adjacent tissue.[23,24] During the fibroplastic phase, the tensile strength of the wound begins to increase as a result of cleavage of procollagen peptides and formation of cross-linkages. During *remodeling*, inflammation decreases, angiogenesis ceases, and collagen is remodeled; increases in the size of the collagen bundles and alteration of collagen cross-linkages[25] lead to the development of an increasingly strong scar.[13,26] Remodeling continues over a long period, until the equilibrium between collagen synthesis and breakdown is restored.[13] Wounds may continue to gain strength even after 1 year,[19,27] and for as long as 2 years or more in children.[28]

*Mechanisms of Wound Healing*. Although the important mechanism of contraction is not thought to apply to tendon repair,[13] it could play a role in the forward movement of scleral reattachment reported during healing after strabismus surgery in animals.[29]

**Cytokines**. The cytokines, or growth factors, are important proteins that act as wound hormones. Some are synthesized by cells distant from the target cells, some are synthesized locally by adjacent cells, and others are synthesized by the same cells they act upon. Cytokines regulate cell proliferation, enhance cell migration to the wound site, and stimulate cells to produce substances needed for healing.[13]

Platelet-derived growth factors (PDGFs) serve as chemoattractants and mitogens for fibroblasts, neutrophils, macrophages, and smooth muscle cells. They also stimulate production of fibronectin and hyaluronic acid, as well as collagenase production by fibroblasts. Transforming growth factor beta (TGF-β) increases collagen synthesis and inhibits collagenase activity. TGF-β accelerates healing of incisional wounds in rats[30] and in fetal wounds and transforms the normal regenerative repair process into an adult-like fibrotic scarring process.[31] TGF-β is thought to be involved in fibrotic disease states, including keloids, hypertrophic scars, and hepatic fibrosis.[32,33] Fibroblast growth factor (FGF) stimulates angiogenesis and wound contracture. Tumor necrosis factor alpha (TNF-α) inhibits wound healing[34] by inhibiting fibronectin and collagen production and stimulating collagenase.[35] It is thought to mediate cancer cachexia,[36] and the poor wound healing seen in chronic sepsis.[37] In adriamycin-impaired mice, TNF-α improved wound healing, indicating that it serves a complex role in the modulation of this process.[36] Combinations of TGF-β, PDGF, and epithelial growth factor eliminate the healing defect seen in adriamycin-impaired animals.[38] Many other cytokines exist, and their complex

interactions are still being uncovered. Perhaps cytokines may be used in the future to manipulate wound healing.[13,26,33]

The effect of increased TGF-β on extraocular muscle healing could possibly increase scar formation and therefore lengthen the scar segment. A deficiency in TGF-β could cause a weak scar, which would then be prone to stretch. The effect of this cytokine on scar lengthening is therefore difficult to predict.

**Extracellular matrix**. Fibronectin is an adhesive protein that is the first substance to be laid down in a wound.[26] It acts as scaffolding for cell migration and collagen matrix deposition and is essential for wound healing. Addition of fibronectin may enhance wound healing.[39]

Glycosaminoglycans and proteoglycans are important components of wound and scar extracellular matrix. Hyaluronic acid, a glycosaminoglycan, is thought to contribute to early cell migration into a wound and is found in large quantities in fetal wounds, which heal by regeneration.[40]

Collagen is the main component of extracellular matrix and the source of strength of all connective tissue, including scar.[13] Early in wound healing, collagen is synthesized by fibroblasts and then converted from soluble procollagen to collagen. Hydroxylation of hydroxyproline is essential to early synthesis; the lack of oxygen or ascorbic acid, which are required for this step, severely compromises wound healing. Initially, type III collagen is laid down, but it is gradually replaced with type I collagen as the wound healing response stabilizes.[41]

The low wound strength at the early stages of healing (up to day 21 to 28) is associated with small diameter collagen fibers.[25,42,43] During remodeling (day 28 to years), collagen becomes organized into larger bundles, cross-linkages develop, and wound strength increases.[12] Remodeling involves a complex balance between collagen breakdown by collagenase[44] and synthesis. The formation of cross-linkages is initiated by lysyloxidase, and an increasing number and greater stability of cross-linkages are found with increasing age.[45] Cross-linkages increase resistance to collagen breakdown and increase tensile strength.[12,25] Resistance to creep is provided by a mix of small and large fibers, with the smaller fibers providing maximum elasticity,[46] while the larger ones resist tensile force.

*Fetal Wound Healing*

Fetal wounds heal rapidly by the process of regeneration, without scar tissue formation. Tissue healed by regeneration has none of the reduction of preinjury strength found with adult scar tissue. It is hoped that by understanding fetal wound healing, it may be possible to reproduce those conditions in the adult wound in order to improve the ultimate strength and

function of the repaired tissue.[13,26] The potential ability to create a fetal wound-healing response could prove useful in the prevention or repair of scar stretch after strabismus surgery.

*Healing of Normal Tendon*

During healing of normal tendon and ligament in the extremities, scar tissue organizes parallel to tendon fibers. Biochemical differences between normal tendon and its scar tissue in terms of collagen type, arrangement of collagen fibrils, water content, DNA, and glycosamine content persist indefinitely after healing. When stress is applied to tendons, microscopic failures occur in collagen fibrils before gross separation, causing lengthening.[47] If these microbreaks then fill in with scar tissue, the lengthening would be expected to become permanent. A similar mechanism could occur in scar lengthening in strabismus surgery.

During tendon and ligament repair, gap formation should be kept to a minimum, as longer scars are weaker.[48,49] Promising prosthetic materials for tendon repair include polyester textiles and expanded polytetrafluoroethylene fiber material. Electrical stimulation enhances ultimate tendon healing. Nonsteroidal anti-inflammatory drugs may improve the quality of healing. Fibronectin coating may enhance fibrosis to prosthetic devices such as polyester mesh.[47]

*Factors That Influence and Impair Wound Healing*

Manipulation of factors that influence wound healing may reduce the incidence of scar lengthening as well as improve repair results in stretched scar cases.

*Nutrition*. Nutritional factors that influence wound healing are numerous. Protein deficiency markedly impairs healing.[50,51] Glucose; essential fatty acids; vitamins A, B complex, and C; iron; zinc; calcium; copper; magnesium; and essential fatty acids are all required for normal healing.[13,26,50] Vitamin C is especially important, as it is required for collagen cross-linking.

Excess vitamin E delays wound healing and increases postoperative adhesions.[50] Vitamin A reverses the effect of cortisone[46,50] on wound healing and also increases collagen accumulation and postsurgical intra-abdominal adhesions in rats.[52] Vitamin D deficiency causes collagen cross-linking abnormalities.[12]

*Oxygen*. Oxygen is required for hydroxylation of proline and lysine in collagen formation as well as for the energy needed for all the cellular activities. Hypoxia, anemia, and poor tissue perfusion all impair healing and can be improved by oxygen administration.[13,26]

*Chronic Disease*. Chronic diseases, including diabetes, uremia, cancer

(as well as its treatment with chemotherapeutic agents), sepsis, and radiation, impair wound healing.[19] Smoking, advanced age, jaundice, and alcoholism are also factors in impaired healing.[53] Uremia interferes with collagen cross-linking.[12]

*Chronic Inflammation*. Chronic inflammation leads to pathologic tissue destruction and impaired wound healing.[19,54]

*Corticosteroids*. Corticosteroids markedly reduce wound and ultimate scar strength by affecting all components of healing, in particular by inhibiting collagen synthesis.[19,27,46,53]

*Hormones*. Hypothyroidism leads to poor wound healing, which improves with thyroid replacement.[52] Growth hormone increases wound strength. Growth hormone has been shown to inhibit the reduction in wound healing caused by glucocorticoids.[55]

*Abnormalities of Collagen*. As collagen synthesis requires many complex steps, there are numerous potential sites where genetic abnormalities may interfere with collagen production. Ehlers-Danlos syndrome is a group of collagen disorders in which poor wound healing is common.[45] Although clinically diagnosed patients may have severe connective tissue disease, there are probably subclinical variants that will become better known as collagen biochemistry techniques improve.[13]

Marfan's syndrome and osteogenesis imperfecta are associated with deficient collagen cross-linking.[12]

## PROPERTIES OF SCAR TISSUE

### *Strength of Normal Scar Tissue*

Most scar tissue never achieves the original strength of native collagen.[27] The strength of the healed tissue varies relative to the strength of the original, unimpaired tissue, depending on the type of tissue and factors affecting wound healing.

The tensile strength of a healed tendon is about the same as the strength of the musculotendinous junction, or about 8% to 10% of normal tendon strength.[48] Healed medial collateral ligaments of the knee achieve a maximum strength that is only 40% of normal.[56] A corneal scar may never restore the original strength of the injured tissue.[57] Rat skin wounds achieve 80% of normal skin strength[52] and guinea pig skin wounds reach only 25% of normal.[58] Abdominal fascial wounds in rabbits ranged from 50% to 80% of normal strength at 1 year, but occasionally reach 90%.[59] Stomach and duodenal wounds generally do achieve the strength of normal tissue.[60]

### *Metabolism of Mature Scar Tissue*

Scar tissue is metabolically active, with equilibrium existing between

collagen breakdown by collagenase and active collagen synthesis.

The disruption of healed, mature scars in vitamin C deficiency was noted centuries ago, when it was observed in sailors with scurvy. It is known that ascorbate deficiency results in a decrease in collagen synthesis.[61] Collagenase activity in normal scars of healthy patients was found to be markedly greater than its activity in normal skin. The investigators postulated that the equilibrium between normal collagenolysis and collagen synthesis in scar tissue becomes imbalanced in scurvy, causing scar weakening.[61]

Vitamin C deficiency any time after strabismus repair could cause scar weakening and lengthening, which would result in the clinical picture of a stretched scar case.

**EFFECTS OF TENSION**

No studies of the effects of chronic tension on the extraocular muscle scar are found in the literature. However, extensive knowledge exists concerning the effects of tension on materials, cells, tissues, and scar tissues elsewhere in the body. These principles should have general applicability to the extraocular muscles.

*Material Response to Tension (Creep)*

From an engineering and materials science standpoint, catastrophic failure or failure by fracture occurs when high stress is applied to a material over a short period of time. Creep, on the other hand, is characterized by the deformation of material resulting from continuous lower levels of stress applied over relatively long periods of time.[62,63]

All previous studies of wound and suture strength in strabismus surgery have been studies of the biologic equivalent of failure by fracture-rupture strength.[29,64,65] The behavior of strabismus wounds under lower levels of continuous stress, which is more indicative of the true environment of the strabismus scar, has not been evaluated prior to this study.

Viscoelastic studies of human connective tissue show complex behaviors.[66,67] Collagen, as a complex macromolecular structure, is subject to creep. The alternating fiber diameters in the arrangement of collagen of normal tendon increases resistance to creep,[46] but this structure is lost in scar tissue, which, therefore, may be substantially less creep-resistant.

*Cellular Response to Tension*

A study of cultured fibroblasts from chick embryos demonstrated that mechanical stretching induced an increase in cell division and DNA synthesis.[68] Cultured rabbit aortic medial cells subjected to cyclic stretching showed twofold to fourfold increases in synthesis of types I and III collagen as compared to controls of agitated, but unstretched, cultured cells.

Hyaluronate and chondroitin 6-sulfate synthesis was also increased.[69] In tissue cultures of fibroblasts on 3-dimensional collagen lattices, freely contracting lattices showed down-regulation of production of collagens I and III. When the matrices were maintained under tension by fixing the rims of the gel, contraction was inhibited and fibroblasts continued to replicate and to synthesize collagen without down-regulation.[70]

Epithelial cells cultured from porcine periodontal ligament significantly increased their synthesis of DNA within 30 minutes of being subjected to stretching forces. This effect was seen both during active stretching as well as 2 hours after stretching had been discontinued. Stretched cells showed an increased number of desmosomes and greater volume fraction of filamentous structures.[71] Cultured bone cells also showed increased DNA synthesis along with increased protein synthesis after 2 hours of applied stretch.[72]

Prostaglandin synthesis was shown to increase when cultured bone cells were subjected to stretch.[73] Cells cultured from rabbit coronal sutures responded to stretch by increasing their synthesis of collagenase, 2 neutral metalloproteinases, and collagenase inhibitor. Additionally, protein synthesis increased and collagen production doubled.[74] Both collagen and the enzymes that degrade it, as well as the inhibitors that control degradation, are necessary for scar formation and remodeling.

Externally applied mechanical tension was also shown to change the arrangement of occluding junctions of cultured mammary epithelial cells.[75]

These tissue culture studies all demonstrated increases in cellular activities when cells were specifically subjected to stretch, but not with the application of other types of mechanical stress. The findings in these studies suggest that the lengthened scars seen in strabismus could result from mechanisms far more complex than mere stretching and may actually involve increased scar tissue production. Such a hypothesis is in agreement with our clinical impression that stretched scars are not just thinned and that a greater total volume of scar tissue is usually present in stretched scar cases.

*Tissue Response to Tension*

Skin under tension is known to stretch, and this property is used intraoperatively by plastic surgeons when closing large defects.[76-78] Skin expander prostheses may be used preoperatively to generate increased tissue for use in reconstructive surgery. A silicone bag is implanted under the skin to be stretched, and saline solution is injected weekly (beginning 2 weeks postoperatively) to gradually expand the bag and stretch the skin. The stretched dermis shows fibroplasia, increased collagen production, collagen fiber realignment, and increased vascularization.[43]

In a study of the biomechanical properties of skin, load deformation

curves were obtained by applying tension to skin.[79] The data were difficult to explain mathematically until it was shown that several mechanisms were involved because skin is not homogeneous.[79] Histologic analysis showed that the collagen fibers of relaxed dermis were arranged haphazardly, but that when skin was held stretched during fixation, the fibers oriented along the line of stretch.[79,80] These results explained the ease of stretching relaxed skin, in contrast to the increasing difficulty of achieving more stretch once the fibers had aligned themselves. Gibson and Kenedi described the phenomenon of stress-relaxation, in which the force required to hold a stretched piece of skin in the same position decreased over time.[79] They also found that creep occurred, in that a constant load applied to skin caused the skin extension to increase over time. They reported that conditions that cause gradual skin stretch, including lymphedema or obesity, can cause a permanent fourfold increase in skin length. Perhaps the very gradual stretching in these conditions is accompanied by collagen synthesis (in addition to creep), as when expander prostheses are used.

When mouse skin was subjected to externally applied stretch forces, an increase in the cellular mitotic index was seen, compared to controls. Initially, the epidermis thinned under tension (day 1), but at 4 days hyperplasia was seen, with increased thickness of the cellular layer compared to unstretched controls.[81] In another study, increased numbers of desmosomes were found in stratified squamous epithelium subjected to severe mechanical stress, especially in the epidermis of the bovine muzzle, which is repeatedly stretched.[82]

Increased tension on vascular walls under conditions of experimental hypertension was found to increase the aortic diameter, wall thickness, and cross-sectional area. Increased deposition of collagen in the rat aortic media was demonstrated.[83]

*Effects of Tension on Early Wound Healing*

Wound strength early in healing is dependent on suture strength as well as the strength of the tissues anchoring the sutures at the wound margin. A study of early healing and wound margin strength in rats showed that tissue within 1.5 mm of an incision was markedly weakened all along the wound, not only at suture placement sites. Tissue strength was not decreased 3 mm from the incision, **except in incisions sutured under stretch, which had wider zones of weakening**. Midline rat laparotomy wounds closed under tension showed a 77% decrease in wound breaking strength at 72 hours, even when the stretch was removed and the wound was resutured prior to testing. This early decrease in postoperative wound strength was reduced by oxygen, free radical scavengers, and collagenase inhibitors, and was eliminated by induced neutropenia and a proteinase inhibitor (soybean trypsin inhibitor). The author felt

that these factors may therefore play a role in the stretch-induced reduction of early wound strength.[22] Collagen content did not change during early healing phases. Late gain in strength during the fibroplasia phase of wound healing was unaffected by these early manipulations.[22,84]

Perhaps antioxidants or antineutrophilic agents may have potential to prevent scar lengthening after strabismus surgery.

*Effects of Tension on Scar Tissue*

In scars under tension, collagen fibers remodel and reorient themselves along the lines of tension.[22,79] When tension is equal in several directions, the result is a hypertrophic scar, and when the tension is applied mainly in 1 direction, a stretched scar results.[85]

Histologic analysis of skin scars showed that with stretched scars, collagen fibers at the periphery of each scar were aligned longitudinally and central fibers were oriented transversely. The scars without tension remained narrow, and all the fibers were oriented longitudinally. The authors hypothesized that the first fibers laid down during healing orient along a scar. When tension across the scar is present, after external or suture support is removed, the scar under tension responds by laying down new collagen centrally, and these fibers orient along the line of tension, across the scar.[18] **The alignment of collagen fibers along tension lines explains the tendon-like appearance of lengthened scar segments and the frequent difficulty in distinguishing the exact demarcation between normal tendon and scar.**

Increased fibroplasia as well as greater collagen phagocytosis by fibroblasts occurs when the tension across a healing wound is increased.[86] It is thought that this may provide a mechanism for the reorientation of fiber bundle direction seen in tension-stressed scar tissue. It appears that collagen remodeling, as well as increased collagen cross-linking, contributes to the progressive increase in tensile strength measured in normal scars over the first year.[87] A study in rats demonstrated that high closing tension impaired wound healing and strength in the abdominal wall.[88]

### SURGICAL SCAR STRETCH/REMODELING IN OTHER TISSUES

Wherever scar tissue is subject to tension, "stretching," widening, or lengthening has been reported. The findings of the studies described below support the use of nonabsorbable sutures and materials to prevent or repair these complications. Extrapolation of these findings to similar conditions in the extraocular muscle scar would predict improved results with nonabsorbable sutures.

*Skin*

Scar stretch is described in plastic surgery, often in wounds without apparent tension. Late stretching of nonfacial scars is frequently seen.[89] Stretch is a

common cause of recurrence of brow ptosis after repair.[90]

Three studies have been performed of skin wounds closed under tension, mostly after tattoo excision. In each study, a randomly selected half of each incision received a test suture method, with the other half serving as the control. Each study showed decreased scar stretch with more prolonged suture support.

In the first study, scar halves supported for 3 weeks by nonabsorbable, nylon subcuticular suture were 73% narrower 1 year later than control halves supported by interrupted silk sutures removed at 10 days. Wounds closed with deep dermal catgut were not different from the silk suture "controls." Wounds supported for 3 months only by superficial tape closure initially had narrower scars, which later widened to the same dimensions as the silk "control" scars by 6 months. Although final scar widths were variable, the rate of stretch was similar in all groups, with about 90% of stretch occurring in the first 6 months.[18]

A similarly designed study compared 2 absorbable sutures, polyglycolic acid and polydioxanone; polydioxanone suture is known to retains its tensile strength twice as long as polyglycolic acid suture. When the 2 types of suture were placed subcuticularly in skin wounds under tension, the polyglycolic acid–sutured scars were 33% wider than the polydioxanone–sutured scars.[17]

The third and most extensive study compared 6 months of subcuticular, nonabsorbable, polypropylene suture support to interrupted percutaneous nylon sutures removed at 12 days (controls). Variations tested included 3 weeks of subcuticular polypropylene or polyglycolic acid suture support. The scars supported for 6 months with nonabsorbable polypropylene sutures stretched 37.5% less than those with 12 days of nylon suture support and 15.7% less than the scars with 3 weeks of nonabsorbable suture support. The absorbable sutures produced no difference in scar widths as compared to the nylon "controls."[16]

*Scalp*

In a study of scalp reduction surgery in 13 patients, in which midline scalp was excised and the wounds were closed under tension with absorbable suture, the rate of stretch-back was accurately measured over time, using the distances between 4 pairs of tattoos placed at surgery. It was shown that most stretch occurred in the wound area and not in adjacent skin. Stretching was clinically complete by 12 weeks, and averaged 34% to 52% of the original excision width.[15] A similarly designed study compared postoperative stretch-back in wounds closed with permanent subcutaneous nonabsorbable polypropylene suture in 8 patients versus absorbable polyglycolic acid suture in 13 patients. The wounds closed with absorbable sutures had scars that were on average 60% to 76% wider than the scars for wounds closed with

nonabsorbable sutures; the absorbable suture closures were also associated with significantly greater average total wound area and significantly greater scar depression, compared with the nonabsorbable wound closures. Again, the greatest stretch was observed in the first 12 weeks, after which no change was seen. The author felt that equilibrium was reached at 12 weeks, by which time the scars had achieved sufficient strength to resist stretching.[14] In this study, measurements were made for only 5 months, and the possibility of later stretch is not discussed. Additionally, the surgical procedure included extensive undermining of scalp. It is likely, therefore, that these wounds would ultimately be supported by scalp-to-galea adhesions, which may reduce tension across the midline scars. This is not the case in properly performed strabismus surgery, in which the entire tension of muscle pull is brought to bear on the single scar between tendon and sclera.

*Fascia*

An incisional hernia develops after abdominal surgery in 3% to 11% of cases.[91-96] It may develop long after seemingly successful healing has taken place, sometimes years after the original surgery.[94,96,97] Midline incisions, which are subject to higher tension across the wound, have higher rates of incisional hernia than transverse abdominal incisions.[92] Predisposing factors are those which increase tension across the wound (obesity, pulmonary disease with coughing, abdominal distension) or decrease the initial inherent wound strength (wound infection, use of steroids, jaundice, increased age, malnutrition, chronic illness).[92-94,98] One group of investigators found that absorbable polyglycolic acid–sutured wounds had a 12.5% failure rate as compared to a 4.7% failure rate with nylon sutures.[91,99] A study of the longer-acting absorbable polydioxanone suture showed the development of incisional hernias in about 3% of cases by 1 year.[93] Incorporation of absorbable meshes did not provide the long-term tensile strength needed to prevent incisional hernias in predisposed patients.[100]

Recurrence rates following primary repair of incisional hernias are high (30% to 50%).[92,97,101,102] The use of permanent, nonabsorbable polypropylene mesh for additional support was associated with reduced recurrence rates of 0% to 10%.[92,98,101,103,104] Nonabsorbable internal retention sutures may also reduce recurrence of incisional hernias by eliminating the tension on the midline sutures.[92,105]

The late-developing incisional hernia is less well understood. It seems to develop independently of known predisposing factors.[96,106,107] Nonabsorbable sutures did not reduce the risk of late incisional hernia, although they markedly lowered the incidence of early ones.[106] Mudge and Hughes[96] found that serious disease, such as cancer, predisposed to the late development of incisional

hernia. An initially wider scar appears to increase the risk of late hernia.[94,108] Ellis and colleagues[106] found no associated clinical factors to explain late-developing incisional hernias, and felt they were due to stretching of mature scar tissue. Harding and associates[107] hypothesized that either scars were stretching or scar tissue may be more metabolically active than has been recognized, and that endogenous patient factors could disrupt the equilibrium of continuous resorption and laying down of new collagen. Urschel and coworkers[94] noted that scar tissue continues to undergo active remodeling, including production of collagenase, at well after 1 year. They suggested that chronic mechanical stress, as is associated with heavy physical exertion or chronic constipation, may play a role in late development of abdominal hernias.

The distinction between early and late developing incisional hernias suggests a parallel to the early and late development of secondary strabismus with stretched scars of the extraocular muscles (see "Methods" and "Study Conclusions" sections).

*Tendon and Ligament*

Stretching has been described after both tendon[48] and ligament[56] repairs. A study of factors affecting the strength of canine flexor tendon repairs concluded that the braided nonabsorbable suture Supramid produced the greatest final tensile strength. A suture technique that prevented gap formation at the repair site was crucial. The best technique was found to be one that prevented tissue damage at the cut end of a tendon by suturing along the length of the tendon at its periphery. Sutures that traumatized the cut end of the tendon caused tip necrosis and a wider scar. Wider scars were found to be more prone to stretch. An initially minimal gap between tendon ends filled more quickly with collagen and allowed the scar to mature more rapidly, producing a stronger union. Active tension across the site in the early postoperative period markedly weakened the final result, presumably by creating a larger gap and longer scar. It was also demonstrated that tendon microcirculation was compromised by tension on the tendon during healing. Triamcinolone injections at the time of repair significantly reduced wound strength at 6 weeks.[48] However, passive mobilization during healing increased tendon scar strength, compared with immobilized tendons.[109]

The principles of tendon repair advanced by these studies could prove applicable to strabismus surgery.

*Blood Vessels*

Microvascular anastomoses in rats were seen to stretch with progressive separation of the ends of the vessels and pseudoaneurysm formation. Sutures were found within each scar and no longer through the vessel medial layers by 10 days, and the gaps between vessel ends were bridged

by scar. One group of investigators suggested that the sutures had pulled through the vessel ends,[110] but another group believed that the portions of vessel wall contained within the loops of suture had become necrotic, such that the sutures were no longer attached to firm tissue, thereby allowing the separation.[111]

*Eyelid (Ptosis)*

The recurrence rate of ptosis after levator aponeurosis dehiscence repair with long-acting absorbable polydioxanone sutures was significantly greater than when nonabsorbable polypropylene sutures were used.[112]

**PRINCIPLES OF SURGERY AND SUTURE TECHNIQUES**

Injury is the stimulus for wound healing, and successful healing requires the apposition of wounded tissue. The healing of cut tendon to intact tissue, such as the intact sclera of strabismus surgery, is a situation not encountered elsewhere in general surgery. Nevertheless, the direct suturing of tendon to sclera may induce enough scleral injury to allow proper tendon-to-sclera scar formation (E.E. Peacock, Jr., MD, oral communication).

Surgical technique and the choice of suture strongly influence the outcome of wound healing. Electrocautery causes tissue necrosis that interferes with healing, and sutures tied too tightly cause necrosis in the incorporated tissue.[26,113] Tying knots too tightly has been shown to reproducibly reduce wound-breaking strength in rats.[84] Irregular tissue injury during incision, as well as excessive needle-induced trauma during suture placement, hinders healing.[113] In wounds closed under tension, sutures can be seen to pull through their supporting tissues.[22]

Sutures that cause reactive inflammation, such as absorbable sutures, predispose to infection,[113] which in turn reduces wound strength. Absorbable polyglactin suture loses almost 50% of its strength by 2 weeks, has negligible strength at 4 weeks when collagen cross-linking is beginning, and causes an increasing chronic inflammatory response from days 10 to 28 after placement.[114] Absorbable monofilament polydioxanone suture retains 70% of its strength after 28 days and produces little inflammatory response.[113,114] Among the nonabsorbable sutures are polyester, which is a stable braided multifilament, and polypropylene, which is a low-reactivity and long-lasting monofilament.[115]

During tendon repair, the need to prevent gap formation is critical. To prevent tissue necrosis at the cut ends of tendon, a lateral trap[48] or end weave[49] suture is used, which places the suture along the tendon periphery, widely distributing the tension.

Incorporation of these surgical and suture technique principles into strabismus surgery may improve results.

**EXTRAOCULAR MUSCLE SURGERY: A REVIEW OF THE STRABISMUS LITERATURE, CONSIDERING THE POSSIBILITY OF STRETCH**

*Overcorrection After Strabismus Surgery*

Variable long-term results after strabismus surgery have been reported by strabismologists. Early reports of consecutive exotropia following esotropia repair often lumped together different types of surgery, including medial rectus recessions and lateral rectus resection procedures, in the same study population, leading to difficulty in interpreting the data with regard to scar stretch.[116-120] Overcorrection rates of 5% to 41% were reported.[2,116,120,121] A recent pure study of bilateral medial rectus recessions showed an overcorrection rate of 12%, with a mean follow-up of 27 months.[122]

Many surgeons have reported that correction of secondary exotropia by undoing the original esotropia surgery often required more extensive surgery than was done initially.[2,118,119,123-125] This observation suggests that scar stretching may have been involved, with reoperation performed on scar tissue rather than tendon. Readvancement in the presence of a stretch would require a greater distance of surgery to accommodate the length of the stretch.

Interestingly, Cooper[2] noted that consecutive exotropia usually developed following medial rectus recessions but was rarely seen after bilateral lateral rectus resection alone. Scar stretch following a resection procedure would be expected to cause undercorrection, and such a case would not be included in an investigation of overcorrections. The effect of scar stretch on the results of an initial uniocular recess-resect procedure could be variable: none if the stretches were balanced, or an undercorrection or overcorrection, depending on which scar was stronger and which was subject to greater muscle pull. This variability of effect seems to be borne out by the lower reported incidence of overcorrection following recess-resect procedures.[2,117]

Progressive overcorrection after inferior rectus recession was reported to be common, especially after adjustable suture surgery. It was most common (50%), and difficult to treat, in patients with thyroid disease. The authors described the possibility that in some cases the muscle had slipped, but mentioned that the transition between tendon and muscle was difficult to distinguish and that diagnosis of slip was difficult.[126] Their observations are not unlike the stretched scars described in this thesis.

The contribution of adhesive syndrome to variable results after strabismus surgery is frequently mentioned,[4,127] but not systematically analyzed in most case series of strabismus overcorrections. In our series, excessive scarring and adhesions of antagonist muscle(s) was the most common cause of false preoperative suspicion of scar lengthening.

*Normal and Elevated Forces of the Extraocular Muscles*

During unrestrained eye movements, tension in the horizontal rectus

muscles was reported to be 8 to 12 g at 15 degrees away from each muscle's field of action, and 40 g at maximal gaze into the field of action, in strabismic patients undergoing surgery.[128] In another study of 29 normal human subjects, maximum medial rectus force during active contraction was 74.8 g, with 1 subject achieving 103 g. Mean maximum active lateral rectus force was 59.1 g, with a high of 92 g in 1 individual. In normal subjects, average lateral rectus active force was 40% lower than average medial rectus force.[129] Superior rectus tension exceeding 90 g has been measured in thyroid ophthalmopathy with inferior rectus restriction.[130]

*Normal and Postoperative Extraocular Muscle Disinsertion Forces*

Disinsertion forces of the musculoscleral junction have been reported in rabbit studies. After superior rectus recessions and resections with 6-0 silk, the tension required to disinsert each muscle was measured between days 2 and 9. By day 4, average disinsertion tension was less than 50 g, but by day 5, it was 100 g, and on day 8, 300 g. The investigators concluded that suture support was no longer needed after day 5, as a 100 g disinsertion tension was probably sufficient to prevent rupture.[64]

A second study evaluated disinsertion forces over a longer postoperative period (weeks 1 through 5) and compared conventional inferior rectus recession to suspension inferior rectus recession. The ipsilateral superior rectus of each eye underwent resection; 6-0 polyglactin sutures were used in all cases. The disinsertion forces of normal unoperated control superior recti and inferior recti ranged from 420 to 450 g. Resected superior recti showed close to normal disinsertion force at each of weeks 1 to 5, and conventionally recessed inferior recti achieved a disinsertion force close to 300 g by 1 week. Suspension-recessed muscles had a rupture strength of less than 50 g at 1 week and 200 g at 2 weeks. The investigators felt that the reduced rupture strength of the suspended muscle was not clinically significant in that normal rotational forces averaged 30 g.[29]

In the animal studies described above (see "Methods" and "Results" sections), significant scar lengthening occurred postoperatively with a constant 27 g load in both the collagenase-treated and saline-treated control superior recti in rabbits. It appears that scar lengthening during remodeling develops at lower tensions than those required for rupture, well within normal extraocular muscle tensions. In pathologic states such as thyroid ophthalmopathy, extraocular muscle tensions may greatly exceed normal, thus further predisposing to scar stretch.

*Wound Healing in Strabismus*

Wound healing after strabismus surgery in animals has been studied.[29,131,132] A detailed study of histologic changes after eye muscle surgery in rhesus

monkeys documented that the final tendon reattachment site is not always at the location where it was attached surgically. Histologic study of muscle reattachment sites showed that they did not lose their cellular and less organized collagen features and begin to resemble normal tendon until several months after surgery.[131]

A histologic study of healing after strabismus surgery in rabbits showed posterior movement of the tendon insertion at 1 week, followed by 2 mm anterior movement of the insertion by 5 weeks. This phenomenon was seen after both conventional and suspension-type inferior rectus recessions. Resected superior recti moved posteriorly 0.5 to 1.0 mm.[29]

Hang-back recessions of the superior recti of cynomolgus monkeys were performed and re-evaluated surgically at 8 weeks postoperatively. One animal was also studied histologically. Six recessions were found an average of 1.1 mm anterior to the intended site. Two were posteriorly located. Three muscles were attached to the eye by a pseudotendon of scar tissue bridging between the original scleral insertion site and the cut end of tendon.[133]

Polyester mesh and polytetrafluoroethylene sheeting were tested as extraocular muscle tendon prostheses in rabbits. Mesh created a marked fibroblastic response, but was cosmetically acceptable. The sheeting created less reaction.[134] The severe adhesions seen with the mesh caused the investigators to reject it as not useful for their purposes, but adhesion formation would be a useful feature if the mesh was used to improve reattachment strength in scar stretch repair.

*Eye Muscle Sarcomere Changes*

In 3 monkeys, eye muscle length was shown to change in response to induced eye position change, initially by shortening of the sarcomeres of the shortened lateral rectus muscle and lengthening of the antagonist medial rectus muscle sarcomeres. After 2 months, however, sarcomere lengths had returned to normal, while the antagonist muscles had shortened and lengthened, respectively. The author presumed that addition or removal of sarcomeres had occurred.[135]

This phenomenon explains the variability of strabismus seen with a given length of scar stretch. If a scar lengthens slowly, the muscle may shorten at the same time, decreasing the effect of the stretch. All stretched scar muscles in this study were tight at the time of repair, suggesting that muscle shortening and possible sarcomere loss may have occurred.

*Slipped Muscles*

The slipped muscle has been described as postoperative detachment of the muscle insertion from sclera, due to improper suture placement, with only capsule, but no tendon, incorporated onto the suture. The capsule retains

its attachment to sclera, and the muscle retracts posteriorly within the capsule, often to the site of the muscle's penetration through Tenon's capsule. Direct repair is reported to correct the defect. The postoperative ocular deviation is large, and versions are usually markedly affected by the ineffective action of the involved muscle(s). Palpebral widening is seen during attempted movement of the eye into the field of action of the slipped muscle, due to its lack of pull, along with relaxation of its antagonist. [3,136,137]

A study of suture tensile strength in the extraocular muscle tendon after surgical placement showed that 130 to 610 g of force was required to cause slippage of the suture through the tendon end in 25% of muscles. In 75%, the muscle itself ruptured and the suture held.[65]

In our series, patients with lengthened scars did not have the history or findings of slipped muscle. Versions were minimally affected, and palpebral fissure changes were not seen. The time course of strabismus overcorrection was gradual in most cases. Strabismus overcorrection did not occur in any patients immediately after surgery, as would be expected with an improperly attached muscle. Stretched scar segments were frequently bilateral and symmetric, which would be unlikely in a true slipped muscle case. The cases of documented restretch despite repair with firm reattachment of tendon to sclera also support the argument that a lengthened scar is different from a slipped muscle. Scar segments were shorter than the long capsule described with a slipped muscle. The dense connective tissue of scar was clearly present and documented histologically in these cases. Slipped muscles would be expected to show predominantly the loose connective tissue of capsule, which would be histologically different from scar.

*Sensory Factors*

Changing alignment in strabismus is commonly seen over time in the absence of surgery. The accommodative esotrope who years later becomes exotropic and the gradual exodeviation of the nonseeing eye are 2 examples.[138] Clearly, the involvement of sensory factors is an important, nonmechanical variable to consider, when alignment changes after strabismus surgery.

In our study, postoperative fusion was achieved by 70 stretched scar patients, with return of stereopsis in 58 patients. Some had complained of diplopia during their periods of strabismus overcorrection and had developed overcorrection despite seemingly intact fusion. Their ocular alignment changes were probably primarily mechanical, owing to the scar lengthening.

## SCAR STRETCH/REMODELING IN STRABISMUS: IMPLICATIONS OF STUDY FINDINGS

*Implications of Clinical Studies*

The lengthening of the scar between tendon and sclera during remodeling

or even years later may be due to a number of mechanisms. The natural tendency of collagen to creep under tension could contribute to postoperative scar lengthening in humans.

The increase in cellular metabolism and collagen synthesis that develops when tissue culture cells are subjected to stretch[68-72,74] suggests another possible mechanism of scar lengthening after strabismus surgery, resulting from actual growth of the scar.

Individuals with delayed, but ultimately normal, collagen synthesis and cross-linkage formation could have insufficient scar strength to resist creep by the time the 6-0 polyglactin sutures lose their strength 3 to 4 weeks after surgery.[114] This explanation would correlate with the fact that collagen cross-linking usually begins at about 28 days into the wound healing process.[12] In such patients, a gradual change in alignment would occur in the first few months after surgery, with eventual stabilization. This type of history was given by and seen in the patients described above as early stretchers. Clinically, they responded well to nonabsorbable suture repair.

By contrast, the late stretchers demonstrated 2 different time courses of the development of their deviations. Some claimed that their deviations had developed very gradually, over years, but could not date the onset of their problem. They seemed to have continual scar lengthening, which could have been caused by gradual creep or continued excess collagen synthesis, possibly due to an imbalance in regulation between collagen breakdown and synthesis.

Other late stretchers claimed to have good ocular alignment for many years, followed by a subacute change which they could date to within a few months to 1 or 2 years. These patients may have had strong, stable scar tissue that broke down or weakened owing to an underlying metabolic abnormality, disease, or nutritional deficiency.

Although stretched scar repairs with nonabsorbable sutures had lower recurrence rates, absorbable suture repairs were successful in some cases, several of which were documented at re-exploration. This suggests that surgical technique at the time of original surgery did play a role in some patients. Several technique problems may serve to weaken the final scar. Incorporation of any loose connective tissue, such as muscle capsule, into the scar will weaken it (E. E. Peacock, Jr., MD, oral communication). Excising capsule and other loose connective tissue surrounding distal tendon should prevent this problem. Partial suture pull-out through tendon or sclera or loosening of the knot could allow the muscle to pull away from the sclera. If this occurs after fibrin clot formation, the muscle may partially separate from sclera, and the gap would then fill in with scar tissue. Clinically, the appearance would be the same as that of a stretched scar, but the history would probably indicate an acute onset of deviation.

Because a longer scar is weaker,[48] steps should be taken to prevent gap formation during reattachment of tendon to sclera. The author uses an extra central bite of tendon in addition to the usual double crossed-swords technique[5] to roll the cut edge of tendon into direct apposition to sclera. This approach also puts the cut tendon in direct contact with traumatized sclera, allowing the necessary two-sided wound (E. E. Peacock, Jr., MD, oral communication). Adjustable suture surgery does not allow fine control over scleral reattachment and scar length. If a pseudotendon attachment to the original insertion develops, which has been shown to happen in "hang-back" surgery in animals,[133] the resultant long scar could be especially prone to lengthen. Necrosis of the tendon tip due to aggressive handling or too-tight suturing would also lead to a long reparative scar and possible later predisposition to stretch.

The use of topical corticosteroids after strabismus surgery is a common practice. However, because steroids are potent inhibitors of collagen synthesis and repair, one possible result of steroid treatment (weakening of the scar) could lead to stretch. The author does not use steroids after strabismus repair.

The commonly held opinion that suture-induced inflammation is necessary to incite wound healing[132] does not coincide with current knowledge of wound healing. Tissue injury is all that is required to initiate healing,[19] and the chronic inflammation induced by the dissolving suture may actually hinder healing[19,54] and predispose to scar stretch.

Despite perfect surgical technique, patient healing abnormalities may cause scar lengthening during remodeling. Genetic abnormalities of collagen, cytokine deficiencies or excesses, chronic disease, nutritional deficiencies, and smoking could all lead to scar stretch.

Although surgical results with nonabsorbable sutures seemed better than the results obtained with absorbable sutures, our series is not a randomized clinical trial, and the controls are historic. Because the use of nonabsorbable sutures has only recently been adopted, follow-up is shorter for these cases, which could have biased the results favorably. Additionally, follow-up in this group may be reduced because adult patients who undergo strabismus repair rarely return to this practice once they consider themselves cured.

*Implications of Animal Studies*

The animal model of scar stretch was designed for 2 purposes: to test whether scar lengthening can occur despite proper surgical technique when healing is impaired, and to create a model for the systematic evaluation of surgical techniques, sutures, and other materials, which may better repair scar stretch and possibly prevent the complication from developing in the first place.

The results demonstrate that an animal equivalent of human lengthened scars can be created with collagenase. A greater volume of scar tissue was present in the collagenase-treated scars, indicating the possibility of increased collagen synthesis. Collagen creep under tension was demonstrated in all the extraocular muscle scars but was markedly increased when scar formation was hindered by collagenase. The creep analysis supports the hypothesis that weak scars may be subject to lengthening by a true stretch mechanism.

Comparison of absorbable versus nonabsorbable sutures in this animal model is still in progress at the time of this writing. Preliminary results suggest that nonabsorbable sutures reduce scar lengthening. This model could lend itself to testing of other suture materials or techniques.

Collagenase is a powerful scar-weakening agent and probably weakens these scars well beyond most naturally occurring in vivo disorders. If a suture prevents scar lengthening in this severe situation, it should probably hold in the clinical setting. However, if a more natural simulation of stretched scar pathology is desired, it may be useful to try more subtle inhibitors of scar strength, such as beta aminopropionitrile or penicillamine, which inhibit collagen cross-linking, in this model.[139]

*Implications of Tissue Culture Studies*

The amount of extracellular matrix in tissue cultures of stretched scar cells was increased, as was the synthesis of metalloproteinases, which attack extracellular matrix. Without their normal scaffolding, the cells would constantly rearrange themselves and divide excessively. This would then cause scar growth, as well as weakening.

The likely source of these tissue culture abnormalities is cytokine (growth factor) abnormalities, as they regulate the above-described abnormal processes.[140-142] Excess growth factor, such as TGF-β, is suspected in this study, but has not yet been demonstrable.

## RELATED SCAR REMODELING PHENOMENA IN STRABISMUS

*Migration of Scleral Reattachment Site*

Strabismus surgeons have noticed that sometimes the final muscle reattachment site on the sclera is not the same as the original surgical attachment site[119,127] (Fig 60). Migration of the reattachment site has also been shown in animal studies.[29,131-133] The author has frequently observed profound migration of scleral attachment toward the original insertion after Hummelsheim[5] transposition surgery. This complication has been repaired successfully by retransposing the tendon halves with nonabsorbable sutures. Since the use of nonabsorbable sutures was instituted for all transposition procedures, new cases with this complication have not been seen. Anterior transposition of the

inferior oblique has also occasionally failed gradually, owing to posterior migration of the scleral reattachment site during remodeling, and nonabsorbable sutures may prevent this. The mechanism of scleral site migration is unknown, but it could be hypothesized to be related to greater tension on the more anterior fibers of the scar, with gradual lengthening, as well as increasing scar strength of the shorter, less-stressed posterior fibers. Because there is no corresponding scleral wound to direct the scar position, collagen fibers will attach themselves anywhere, and will adhere better where they are under less tension. As remodeling progresses, the position gradually shifts posteriorly. Nonabsorbable suture should reduce tension on the scar, reducing position shift.

*Faden and Jensen Procedures*

Two patients who had undergone previous Faden suture placements elsewhere underwent reoperation for recurrent strabismus. All the operated muscles were intact, in their normal anatomic positions, with no adherence of the muscle bellies to sclera. Similar observations have been made by another strabismologist (D. R. Stager, MD, oral communication).

Another patient underwent a Jensen procedure for diabetic third nerve palsy. Afterward, postoperative alignment was excellent, but the original exodeviation recurred over several months. On reoperation, the superior, inferior, and medial recti were found to have returned to their normal anatomic positions; although the muscles were surrounded by scar tissue, they themselves bore no evidence of the original surgery. The nonabsorbable sutures were found embedded in connective tissue at the sites where they and the muscle halves had originally been placed, midway between the normal muscle positions. A similar case was described to the author by another colleague (E. C. Price, MD, oral communication).

The practice of suturing into muscle tissue is common in strabismus surgery, but not in other disciplines. Muscle must be repaired by repairing its connective tissue, as one cannot suture globular protein (E. E. Peacock, Jr., MD, oral communication).

During natural remodeling of the surgical site, sutured muscle fibers can pull out of their sutures, and the muscles may return to their normal positions and repair their defects. If an adhesion forms between sclera and the muscle, the desired surgical effect may persist, but the suture alone is unlikely to hold its position under tension in muscle.

## WOUND HEALING IN STRABISMUS

With the marked expansion of research and understanding of mechanisms in wound healing, clinical application of these insights should translate to

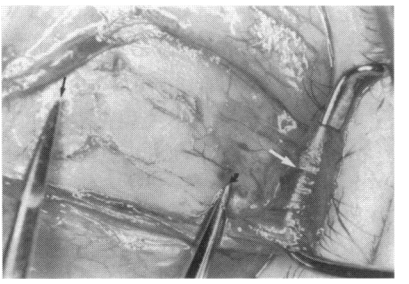

**FIGURE 60**
Caliper indicates original insertion (straight arrow) and previous surgical reattachment site (curved arrow) in this case of overcorrection after lateral rectus recession. Scleral reattachment site has migrated (white arrow).

improved surgical results. Basic general surgery texts usually devote at least one detailed chapter to the principles of wound healing.[102,143] Major ophthalmology textbooks generally contain no dedicated chapters, usually only short sections on corneal wound healing.[144,145] As ophthalmology is a surgical specialty, knowledge of wound healing principles should be included in ophthalmology and strabismus training. Surgical techniques should be based on understanding gained by research and subject to the same scientific scrutiny as medical therapies. This approach should allow us to improve the reliability and stability of postoperative results.

**FUTURE MANAGEMENT**

It may be possible to reduce the rate of occurrence of scar stretch by preventive steps during routine strabismus surgery. The refinements of surgical technique described above, avoidance of steroids, use of vitamin supplementation preoperatively and postoperatively, and avoidance of smoking may also help. When muscles are expected to exert greater than normal tension on the scar, as in patients with thyroid disease, inferior rectus surgery, or muscle transpositions, nonabsorbable sutures may be preferable at the outset. Patients with known collagen abnormalities or chronic diseases that impair wound healing should probably also be operated on with nonabsorbable sutures.

Although it is not common practice to create a scleral wound in order to precisely direct scar position, this is another possibility to consider. Perhaps weaving the suture in and out of sclera during passage of the scleral tunnels would increase scleral injury and allow more directed healing.

A longer-acting absorbable suture, if used in routine strabismus surgery, could reduce the incidence of scar stretch. Manipulation of wound healing by the use of cytokines (growth factors) may ultimately

allow regenerative healing and better scar strength.

Under tension, nonabsorbable sutures can pull out of tissue, and in some situations, a broader surface area of nonabsorbable support is required. Because nonabsorbable meshes are known to reduce the recurrence of incisional hernia, polyester surgical mesh reinforcement was used in superior rectus surgery in rabbit eyes to determine whether the mesh would be tolerated in the subconjunctival space (Fig 61), in the hope that this material could be used to strengthen the muscle-to-tendon adhesion in cases of severe stretched scar. The mesh was well tolerated for a year (Fig 62), with integration of the mesh and the underlying sclera and muscle (Fig 63). With the development of finer-gauge meshes and improved surgical technique, mesh reinforcement may have future utility in human stretched scar cases that recur after nonabsorbable suture repair.

## CONCLUSIONS

This thesis has detailed the intraoperative finding of a segment of lengthened scar tissue between extraocular muscle and sclera in a number of strabismus reoperation cases. The frequency of occurrence of lengthened scar is especially high in strabismus overcorrection cases.

The scar segment can be difficult to see despite careful dissection and inspection of the muscle, as the scar often blends indistinctly into tendon. Features that suggest scar stretch include laxity at the insertion site, the amorphous nature of the scar as compared to tendon, and longer-than-expected tendon. Inspection of the internal aspect of the muscle after disinsertion can be helpful in confirming the diagnosis.

On the basis of the clinical and animal studies and a literature review of similar phenomena in other surgical situations, it is concluded that:

- The scar segment complication can develop despite properly performed strabismus surgery, and the time course of development of secondary strabismus caused by lengthened scars is variable, with many occurring during the first few months after surgery, and others years later. Physiologic processes must therefore contribute to scar lengthening or stretching.

- The stresses to which these operated muscles are subject contribute to the development of this complication in predisposed patients. Studies demonstrating the effect of tension on cells, tissues, and the wound healing process suggest a high probability that scars under tension may actually grow by synthesizing extra collagen, and also stretch due to creep mechanisms when a scar is weak.

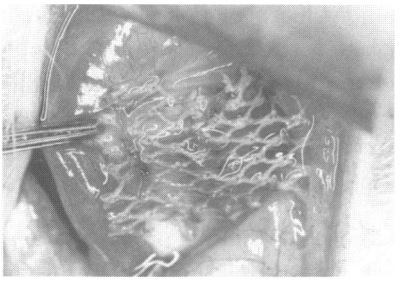

**FIGURE 61**
Polyester mesh is surgically implanted over superior rectus and sclera of rabbit. Superior rectus was disinserted and reattached.

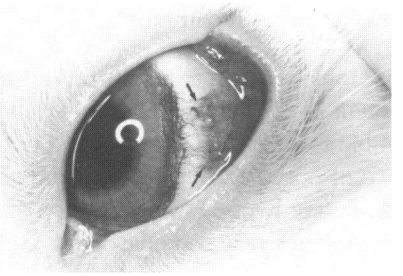

**FIGURE 62**
Superior conjunctiva of rabbit in Fig 61 after 1 year. Edges of mesh indicated by arrows.

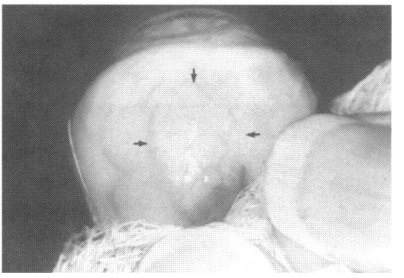

**FIGURE 63**
After enucleation, mesh is seen to be completely covered by conjunctiva, without erosion. Mesh outlines are indicated by arrows.

- It is likely that individual metabolic factors, including alterations in collagen metabolism and possibly nutritional deficiencies, play a role in the development of these lengthened scars. Although most patients were healthy, some had possible predisposing factors, including chronic disease, collagen abnormalities, prior orbital irradiation, and genetic predisposition.

- The use of nonabsorbable sutures provides the necessary support to prevent or repair this complication of strabismus surgery in most cases. The frequency of suspected restretch after stretched scar repair exceeded 40% when absorbable sutures were used, whereas nonabsorbable sutures reduced the suspected restretch rate to 6%. The use of nonabsorbable sutures allows definitive repair of the stretched scar abnormality, as seen in Fig 64. In the future, the use of an anchoring mesh may provide additional support in cases where even nonabsorbable suture repair fails.

- Among the numerous factors that impair healing and may be involved in the development of this complication of strabismus surgery, some may be susceptible to intervention, including nutritional supplementation, avoidance of postoperative steroids and smoking, and control of underlying disease or hypoxia. Manipulation of wound healing with factors such as cytokines may someday allow better control over the wound healing process and more predictable long-term results after strabismus surgery.

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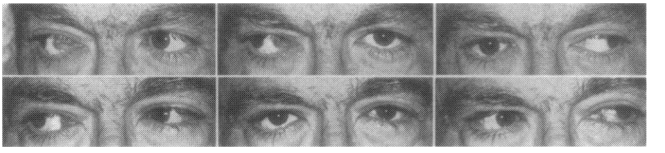

**FIGURE 64**

A 48-year-old man who had previously undergone more than 6 procedures to repair consecutive exotropia. Top, before stretched scar repair; Bottom, after stretched scar repair. Photographs on left indicate right gaze; photographs on right indicate left gaze.

work possible. Alan Y. Chow, MD, who had independently recognized the stretched scar phenomenon before the first of our many discussions, contributed his own case records, which are included in this series. Dr Herbert E. Kaufman encouraged and provided the resources for this work. Jean T. Jacob, PhD, and her associates performed the rabbit care, creep studies, and rabbit histopathology. Roger W. Beuerman, PhD and Doan Nguyen, PhD, performed cellular studies. Alma Baird, Paul Falkenstein, and the suture development team at Ethicon Corporation created and provided sutures, as well as supplies and financial support. Most of the intraoperative photographs were taken by anesthesiologists H. Sharron Carson III, MD, and Ralph Fillmore, MD, and the histopathologic interpretation and photographs of human tissues were contributed by pathologists Arthur R. Summerlin III, MD, and John Rodenbough, MD. Hilary W. Thompson, PhD, provided statistical analyses, and Maxine Haslauer created the photographic layouts. Paula Gebhardt provided editorial services, and Lisa Curole spent many hours in the library searching for references.

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