STRABISMUS DUE TO FLAP TEAR OF A RECTUS MUSCLE*

BY *Irene H. Ludwig, MD* AND *Mark S. Brown, MD* (BY INVITATION)

ABSTRACT

Purpose: To present a previously unreported avulsion-type injury of the rectus muscle, usually the inferior rectus, and detail its diagnosis and operative repair.

Methods: Thirty-five patients underwent repair of flap tears of 42 rectus muscles. The muscle abnormality was often subtle, with narrowing or thinning of the remaining attached global layer of muscle. The detached flap of external (orbital) muscle was found embedded in surrounding orbital fat and connective tissue. Retrieval and repair were performed in each case.

Results: Fourteen patients had orbital fractures, 7 had blunt trauma with no fracture, and 9 had suspected trauma but did not undergo computed tomographic scan. Five patients experienced this phenomenon following retinal detachment repair. Diagnostically, the predominant motility defect in 25 muscles was limitation toward the field of action of the muscle, presumably as a result of a tether created by the torn flap. These tethers simulated muscle palsy. Seventeen muscles were restricted away from their field of action, simulating entrapment. The direction taken by the flap during healing determined the resultant strabismus pattern. All patients presenting with gaze limitation toward an orbital fracture had flap tears. The worst results following flap tear repair were seen in patients who had undergone orbital fracture repair before presentation, patients who had undergone previous attempts at strabismus repair, and patients who experienced the longest intervals between the precipitating event and the repair. The best results were obtained in patients who underwent simultaneous fracture and strabismus repair or early strabismus repair alone.

Conclusions: Avulsion-type flap tears of the extraocular muscles are a common cause of strabismus after trauma, and after repair for retinal detachment. Early repair produces the best results, but improvement is possible despite long delay.

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INTRODUCTION

Diplopia following head or facial trauma is usually attributed to palsy of a cranial nerve or its branch or to incarceration of an extraocular muscle in an orbital fracture site. Restriction of eye movement by adhesions to scar tissue is also reported to contribute to strabismus.[1-4] Generally, a lengthy delay (months) is advocated before strabismus surgery is undertaken.[1-3,5-8] Repair may consist of ipsilateral muscle surgery and/or a procedure to limit excursion in the nontraumatized contralateral eye[9] to balance the deficit in the injured one.

In a number of post-traumatic strabismus cases, a specific type of avulsion injury to the rectus muscle(s) has been identified and repaired. We propose a mechanism for the development of the flap tear, which is consistent with recently reported anatomic and functional studies of the extraocular muscles.

METHODS

SURGICAL APPEARANCE

Thirty-five patients underwent repair of avulsion-type injuries of one or more rectus muscles. The involved rectus muscle was approached through a standard fornix incision and placed on a muscle hook at its insertion into sclera. The presence of a flap tear was suggested by 3 different appearances:

1. A segment of muscle and tendon was missing, which narrowed the remaining portion of attached muscle (Fig 1).
2. The outer or orbital layer of muscle was missing, beginning at the musculotendinous junction and extending proximally (Fig 2). These muscles appeared thinned and lacked intact muscle capsule. The thinned area involved the entire width of the muscle in some and a smaller portion of muscle in others.

*From the Department of Ophthalmology, LSU Eye Center, Louisiana State University Health Sciences Center, New Orleans (Dr Ludwig) and the Department of Ophthalmology, The University of South Alabama, Mobile (Dr Brown). Supported in part by an unrestricted departmental grant to the LSU Eye Center from Research to Prevent Blindness, Inc, New York, New York.

3. The muscle was encased in adherent orbital fat, requiring careful dissection before disclosure of the avulsion injury, which could appear as either of the two previously described abnormalities.

In each of the 3 presentations, the torn “flap” of tissue was found external to the muscle, scarred into surrounding orbital connective tissue and fat (Fig 3). Sometimes several smaller flaps of muscle were found.

Forced duction testing was performed before and during muscle repair in all cases. Restrictions both toward and away from the direction of the involved muscle’s action were often present. In some cases the forced duction abnormality was subtle, and it only became evident when the procedure was performed gently, with simultaneous comparison to the uninjured contralateral eye.

SURGICAL REPAIR TECHNIQUE

The flap was placed on a small muscle hook and dissected free from its orbital attachments at the distal end. A braided polyester suture was placed through the distal end of the flap, with standard strabismus locking bites (Fig 4). The flap was then attached to sclera at the original insertion, or back to the musculotendinous junction, as necessary to restore anatomy (Fig 5). The rent in overlying Tenon’s capsule, which was always present, was sutured with 6-0 polyglactin after the protruding orbital fat was reposited through the rent.

If the capsule of the repaired muscle appeared complete, it was repaired directly with buried 6-0 or 7-0 polyglactin suture. In some cases, the capsule seemed partly damaged. In these cases, a free graft of Tenon’s capsule was sutured over the traumatized surface of the muscle with running 7-0 polyglactin (Fig 6). The Tenon’s graft was harvested from an uninvolved quadrant of the same eye, usually superotemporally.

Postoperatively no steroids were used. Patients were asked to exercise the muscle frequently by looking in and out of the field of muscle action to prevent adhesions from re-forming between the flap and surrounding orbital connective tissue.

RESULTS

PATIENT CHARACTERISTICS

The mean age of the 35 patients at time of flap tear repair was 40 years (range, 6 to 82 years). The mean delay between the date of injury and repair was 67 months (range, 2 weeks to 46 years). Mean postoperative follow-up was 9 months (range, 1 to 68 months). The left inferior rectus muscle was most commonly involved (22 cases) followed by the right inferior rectus muscle (12), the medial rectus (6), and the superior rectus (2) (Table I). The predominance of affected left inferior rectus muscles is presumed to be related to the right-handedness of assailants delivering the trauma in some cases.

PRECIPITATING EVENT

Fourteen patients had orbital fractures, and 6 had blunt trauma with documentation of the absence of fracture by computed tomographic (CT) scan. In 9 cases, the finding of flap tear was unexpected and orbital imaging was not undertaken. In most cases, a long time had elapsed between injury and repair. In 1 child, CT scan was obtained following development of downgaze deficiency 1 week after seemingly minor blunt trauma to the inferior orbital rim. There had been no external signs of the injury at the time. He had posterior orbital floor fracture and inferior rectus flap tear (Fig 7).

Two patients had no specific history of orbital trauma. One patient had a normal CT scan and questionable, remote history of trauma. Five patients developed flap tears after retinal detachment repair (Table II).

DIAGNOSIS

Motility Defects

In 20 patients, the motility defect was toward the direction of action of the involved muscle, presumably as a result of a tether created by the flap. Twelve of these presented as downgaze deficiencies following documented orbital floor fracture. In 10 patients the presenting deficit was gaze restriction away from the field of action of the muscle, simulating persistent entrapment or muscle fibrosis. One had paradoxical esodeviation on attempted upgaze, along with limitation of elevation and depression after orbital floor fracture repair. One patient had 2 involved muscles in 1 eye. The torn medial rectus caused exotropia with adduction limitation owing to the tether effect, and the superior rectus tear led to hyperdeviation owing to a restrictive effect. Two patients had tears of both inferior rectus muscles and 1 medial rectus muscle each. The inferior rectus muscles caused asymmetric downgaze reduction owing to the tether effects, and the medial rectus muscle tears led to esotropia owing to restrictive effects. Another patient, with idiopathic etiology, had bilateral inferior rectus flap tears, with mild upgaze restriction in 1 eye, and downgaze restriction in the other. There was no difference in the appearance of the flap tear or the difficulty of repair in terms of the various types of motility patterns. Our impression was that an anterior attachment site of the flap led to a tether effect and a posterior flap position led to pseudo-entrapment.

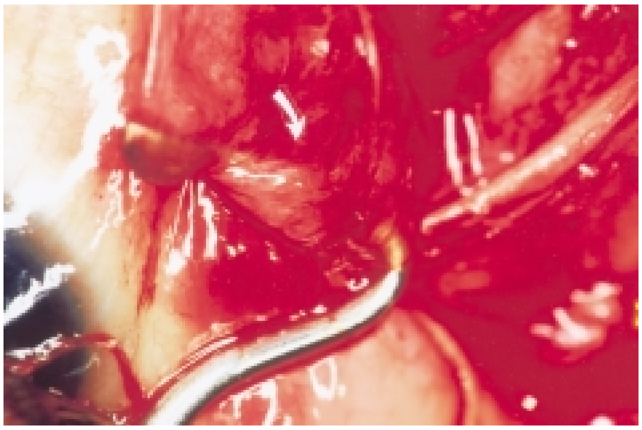

FIGURE 1

Flap tear of inferior rectus, narrowed type (same as in Fig 7A). Arrow indicates missing portion of muscle; hook is pulling on insertion of remaining attached portion of muscle.

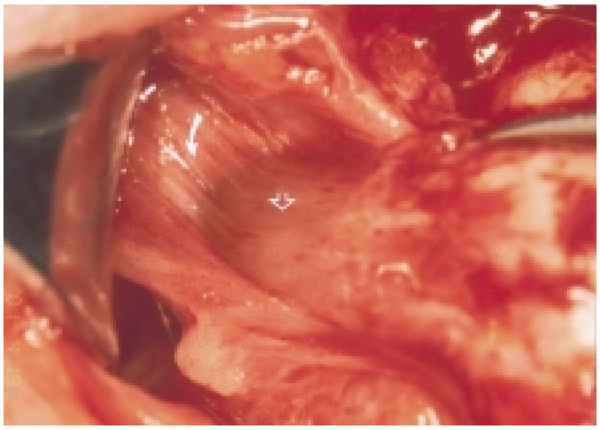

FIGURE 2

Flap tear of medial rectus, thinned type, 2 months after retinal detachment repair. Open arrow indicates thinned remaining portion of muscle, lacking capsule. Solid arrow points to flap, which is pulled outward by retractor.

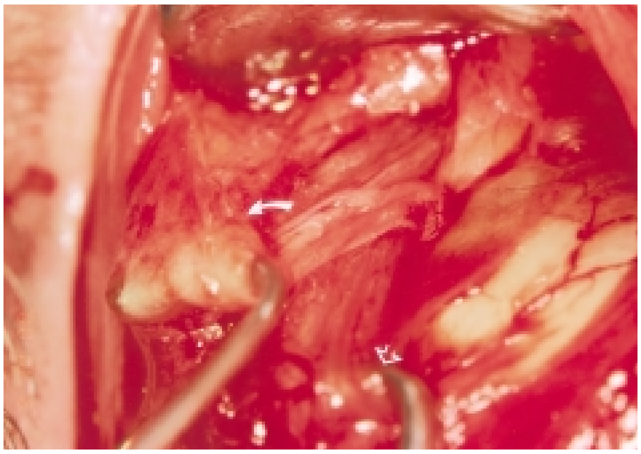

FIGURE 3

Flap tear of inferior rectus, 20 years after motorcycle accident. Attached portion of muscle is held by large hook (open arrow). Flap, held in small hook, is adherent to surrounding orbital tissue (solid arrow).

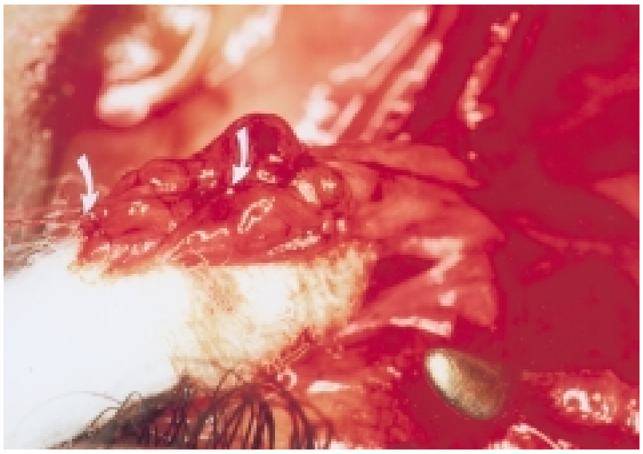

FIGURE 4A

Inferior rectus flap (same patient as in Figs 1, 6, and 7), dissected free, and placed on 6-0 braided polyester suture. Lock bites indicated by arrows.

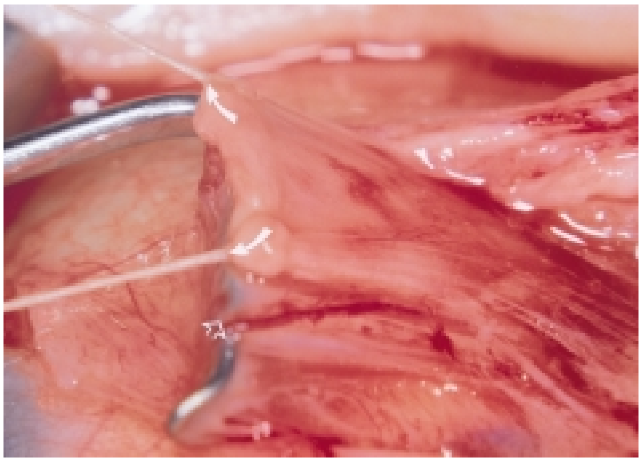

FIGURE 4B

Inferior rectus flap, thinned type. Flap is held on braided polyester suture (solid arrows). Hook (open arrow) holds attached, inner portion of muscle.

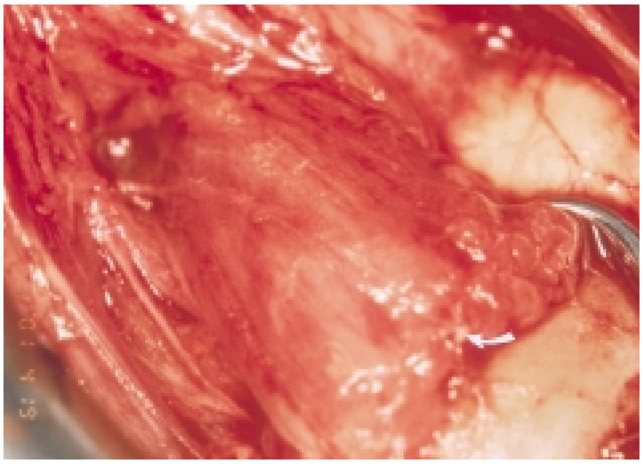

FIGURE 5

Medial rectus from Fig 2, after flap reattachment. Arrow indicates central knot in suture.

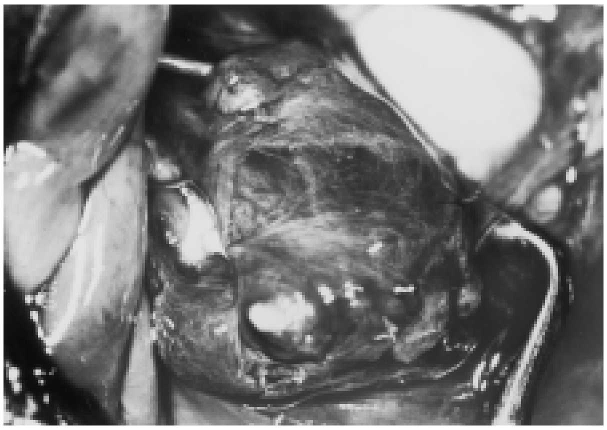

FIGURE 6
Repaired inferior rectus (same as in Figs 1 and 4a), with overlying Tenon's graft. Tenon's fibers are oriented 90° from muscle and tendon fibers.

TABLE I: INVOLVED MUSCLES

NO. OF MUSCLES INVOLVED	NO. OF PATIENTS	MUSCLE			
		LEFT INFERIOR RECTUS	RIGHT INFERIOR RECTUS	MEDIAL RECTUS	SUPERIOR RECTUS
1	30	19	8	2	1
2	3	1	2	2	1
3	2	2	2	2	0

Preoperative Diagnosis

Five patients were misdiagnosed preoperatively as having fourth cranial nerve palsy, 2 as having Brown's syndrome, and 1 as having sixth cranial nerve palsy. In the first patient treated (MC), a lost inferior rectus was suspected because of the absence of downgaze. Many of these patients would have qualified as having isolated palsy of the inferior rectus based on measurements alone.

TABLE II: PRECIPITATING EVENT

CAUSE OF STRABISMUS	NO. OF CASES
Orbital fracture	14
Blunt trauma, no fracture	5
Trauma, possible fracture	10
Retinal detachment	5
Idiopathic	1

Preoperative force generation testing was useful to rule out nerve palsy. Although restricted in excursion of movement, the involved muscles showed normal strength. Forced duction testing was usually performed immediately before surgery, with the patient under paralytic anesthesia.

Since the first case was identified in 1994, every patient presenting to this practice with a preoperative diagnosis of orbital fracture together with limitation of motility toward the direction of the fracture was found to have a flap tear.

SURGERY

Sixteen patients underwent direct repair of the flap alone, with no other muscle surgery (Table III). Two of these required re-repair of the flap: 1 for a small residual flap segment that had been missed at the initial procedure,

TABLE III: PRIMARY SURGERY

	REPAIR OF FLAP ONLY	REPAIR OF FLAP PLUS RECESSION OR RESECTION OF SAME MUSCLE	SURGERY ON ADDITIONAL MUSCLES
Total cases	16	4	15
No. requiring second procedure	2	1	5

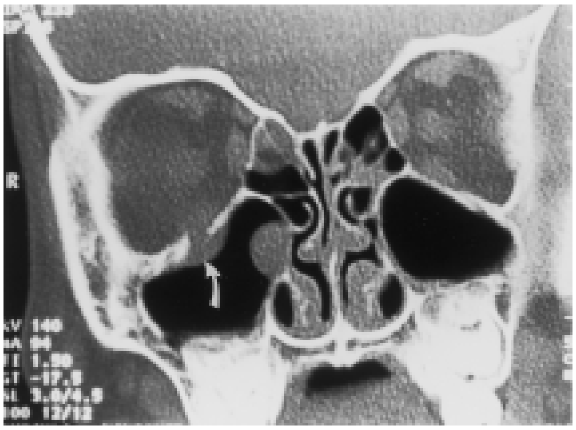

FIGURE 7A
CT scan showing posterior orbital fracture with inferior rectus entrapment (arrow). (Patient's inferior rectus is seen in Figs 1, 4a, and 6.)

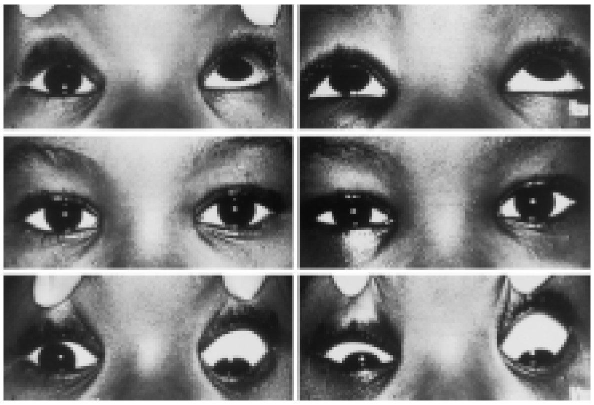

FIGURE 7B
Preoperative (left) and postoperative (right) motility of 6-year-old boy who underwent simultaneous repair of orbital floor fracture and inferior rectus (Figs 1, 4a, and 6) flap tear 2 months after minor blunt trauma.

TABLE IV: CLINICAL DATA OF ALL PATIENTS UNDERGOING REPAIR OF AVULSION-TYPE FLAP TEARS

PATIENT	AGE	DELAY (MO)	MUSCLE	?TETHER	CAUSE	?ADD'L MUSCLE	?2ND SURGERY	ALIGN PREOP	VERSIONS PREOP	ALIGN POSTOP	VERSIONS POSTOP	FOLLOW-UP
KB	12	1	LIR	no	football	no	no	RHT8	1-depr, 2-elev	ortho	nl depr	10
EB	59	3	RIR	no	struck orbit in motorboat	recess RIR6, RLR4	no	LHT25	3-elev OD	ortho	1-depr, 3-elev	19
DB	38	13	LIR	no	RD repair	LIR recessed 4	no	RHT25	2-elev OS	ortho	1-elev, depr OS	3
RB	42	252	LIR, RIR, LMR	yes	motorcycle accident	no	planned	LHT14/ ET20	2-depr OS, 1-elev OD 2-abd OS	LH(T)2/ ET20	2-elev, 2-depr OS	1
MC	24	13	LIR	yes	struck with fist	LSR rec 2/16/94	no	LHT4	3-depr OS	ortho	3-depr OS	68
BC	39	4.5	LIR, RIR, LMR	yes	MVA	recLMR6	resect LLR8, RMR5.5 LIR3	RHT10, ET55	4-abd OS, 1-depr OD, 2-elev OD	ortho	1-elevOU, 1-ad, abdOU	5
PD	29	8	LIR	no	orbit bumped by childs head	LLR, LMR inf transp	yes	ortho large RHT in upgaze	4-elev OS	ortho	3-elev, deprOS	1.5
BD	12	0.5	RIR	yes	bicycle handle	no	no	RHT14	4-depr OD	ortho	1-depr OD	2
TE	82	6	RIR	no	fell on pavement	RSO	no	LHT10	2-elev, depr OD	RHT5	?	1
BE	8	8	LIR	yes	bicycle handle	no	yes	LHT6	1-depr OS	LH2	nl depr	5.5
SE	39	204	LIR	no	RD repair	LLR5	adv LIR to orig insertion	RHT20	2-elev OS	RH2	2-elev OS	1
RG	79	10	LSR	yes	branch struck eye	LIRrec6	adv LIR, LSO	RHT20	4-elev OS	E12	3-elev OU	48
AG	20	228	RIR	no	unknown	RIR rec3	no	ortho. large LHT in upgaze	4-elev OD	ortho	3-elev OD	3
SH	29	24	LSR, LMR	yes	struck with fist	LSR rec4	no	XT10/ LHT10	1-elev, deprOS 1-add OS	X2	1-elev OS	1
CH	42	8	RIR	yes	MVA	no	planned	RHT9	4-depr OD	ortho	3-depr OD	3
WH	82	8	LIR	no	RD repair	LMR4	no	RHT5	3-elev OS	RHT10	4-elev OS	4
PJ	53 ?		LIR	yes	struck with fist	no	no	?	?	ortho	3-depr OD	18
CJ	32	3	LIR	yes	struck with fist	LLR8	no	LHT35	3-depr OS	LH(T)4	3-depr OS	1
JK	68	24	RIR, LIR	yes	?blast concussion Korean War	no	no	ortho LHT6 in downgaze	1-depr OS, 1-elev OD	ortho all gaze	normal	1
SL	15	1.5	LIR	yes	football	no	no	2-elev	2-depr OS, ET on elev	ortho	2-elev OS	8

TABLE IV (CONTINUED): CLINICAL DATA OF ALL PATIENTS UNDERGOING REPAIR OF AVULSION-TYPE FLAP TEARS

PATIENT	AGE	DELAY (MO)	MUSCLE	?TETHER	CAUSE	?ADD'L MUSCLE	?2ND SURGERY	ALIGN PREOP	VERSIONS PREOP	ALIGN POSTOP	VERSIONS POSTOP	FOLLOW-UP
TL	24	87	LIR	yes elbow	struck with	s/p LIR rec	no	LHT10	3-depr OS	ortho	2-depr OS	3.5
JM	38	360	LIR	no	possible fist	LLR	yes	RHT30	1-elev OS	ortho	normal	5
KM	16	6	LIR	yes fist	struck with	no	yes	LHT16	2-depr OS	ortho	2-depr OS	36
SO	28	24	RIR	no	air bag	recSOOU	no RHT10	ET6/	3+LSO	RHT4		0.1
RR	71	7	LIR	yes	MVA Resect LIR	SOOU adv,	yes	LHT5	2-depr OS	ortho	1-depr OS	6
MS	51	552	LIR	yes playground	fell in	no	planned ET18	LHT18/	4-depr OS ET14	LHT14/	4-depr OS	2
TS	8	2	RIR	yes	MVA	no	no	RHT6 2-elev OD	2-depr,	ortho	1-elev OD	7
NS	33	264	RMR	no elbow in playground	struck by	no	no	ET18 2-elev	1-ad, abd OD,	ET6	tr-abd OD	1
FS	56	3	LMR	yes tree branch	struck with	LLR rec10	no	XT25	4-add OS	ortho	3-add OS	4
HV	49	12	LIR	yes	RD repair adv LSO4	recLMR4, res LMR3	res LIR3.5,	LHT25 1-abd OS, 3-LSO	3-depr,	X4 depr OS	3-elev,	8
BW	65	84	LIR	yes ladder	fell off	myot LMR	no	LHT10	2-depr OS	LH(T)3 1-add OS	2-depr,	1
BW	71	2 R	MR,RIR	no	RD repair	no	no	RET25 3-elev OD	2-abd,	RHT6 3-depr OD	2-abd,	0.2
HW	6	2	RIR	yes on bathtub edge	struck orbit	no	no	RHT16 depr OS	4-elev,	ortho 3-elev	1-depr OS	1.5
BW	36	3	LIR	yes pavement	fell on	no	no down)	E4(LHT18	2-depr OS	ortho	normal	25
AW	29	3	LIR	yes bat	baseball LLR6	rec LSR5,	no	LHT25	4-depr OS	ortho	3-depr OS	2

and the other for a complete flap redetachment. The redetachment was thought to have occurred as a result of the child's failure to move the eye postoperatively, as well as damage to the muscle capsule and, possibly, loss of strength of the absorbable suture used to reattach the muscle. At reoperation, the flap was reattached with nonabsorbable suture and a Tenon's graft was placed. The second repair was successful. Nonabsorbable suture has been used for flap reattachment in all subsequent cases.

Forced duction and spring-back testing[10] were used to determine whether flap tear repair alone relieved the gaze restriction and centered the eye. If not, additional surgery was undertaken.

Additional Strabismus Surgery

Fifteen patients underwent simultaneous surgery on other extraocular muscles, and 7 of these required a second strabismus procedure. Four patients exhibited residual restriction of motility away from the direction of action of the muscle with the flap repair, and they underwent simultaneous recession of that muscle alone during the initial surgical procedure. Surgery on additional muscles was needed more often when a longer time had elapsed between injury and repair. Secondary deviations were considered and corrected; the most common was ipsilateral lateral rectus recession for exotropia.

Orbital Fracture Repair

Six patients had undergone orbital fracture repair prior to flap tear repair, and 3 underwent repair of muscle and orbit on the same day by a strabismus surgeon (I.H.L.) and an ophthalmic plastic surgeon (M.S.B.). These 3 cases confirmed the impression that the flap tear is remote from the fracture site and is not the result of bony impingement on the muscle. Five patients with documented fractures did not undergo fracture repair.

POSTOPERATIVE ALIGNMENT

Preoperative and postoperative alignment data were not analyzed statistically for the group owing the heterogeneous population of involved muscles and variety of additional muscle surgeries performed (Table IV). Of the 16 patients who underwent flap tear repair alone, all were improved, most achieved resolution of diplopia during regular activities, and 9 had normal alignment in all gaze positions.

The best results were achieved in those who underwent simultaneous repair of flap tear and orbital fracture and those who did not undergo fracture repair. The worst results occurred in those who had undergone previous orbital fracture repair (MC, CH, SL, TL, KM, AW) and/or strabismus surgery (RB, MC, TL, MS, FS). One patient (CH) had undergone 2 orbital surgeries because of the persistent downgaze deficiency. He had severe motility restriction noted during subsequent flap tear repair, and although the primary position alignment was restored, motility remained poor.

Long delay from injury to repair seemed to worsen the prognosis for some patients, but others did well despite the lapse of many years. Those with smaller flaps and smaller preoperative deviation of alignment had improved chance of resolution with flap-tear repair alone. Greater delay to treatment increased the likelihood of further surgery on additional muscles. No patient was worsened by flap tear repair (Table IV).

CASE REPORTS

Case 1

A 12-year-old boy developed diplopia after striking his right inferior orbital rim on the handle of a bicycle. In the primary position he had a right hyperdeviation of 14 prism diopters (D), which increased to 20D on downgaze. Elevation and depression of the right eye were markedly reduced (Fig 8, left). CT scan showed a narrow orbital floor fracture in the medial portion of the orbital floor, with entrapped orbital tissue and muscle. Orbital fracture and strabismus repairs were undertaken at the same time, 3 weeks after injury, by the authors.

Forced duction testing showed restriction to elevation and depression in the right eye. The fracture site was approached via standard transconjunctival incision. Significant herniated orbital fat and connective tissue were present, and entrapped inferior rectus tissue was identified in the posterior aspect of the fracture (Fig 9). After the tissues were lifted out of the fracture, repair was made with porous high-density polyethylene barrier sheet, and the wound was closed. The inferior rectus was then exposed through a standard inferotemporal fornix

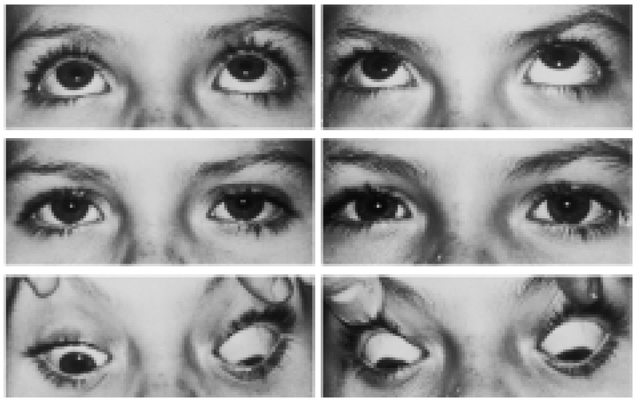

FIGURE 8

Case 1. Preoperative (left) and postoperative (right) motility.

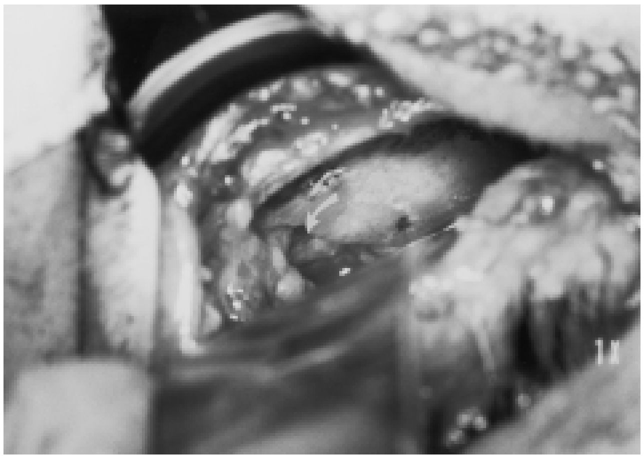

FIGURE 9
Case 1. Orbital fracture (arrow).

incision. The flap tear was identified by the narrowed appearance of the muscle (Fig 10) and repaired as already described (Fig 11), including free Tenon's graft. *The torn segment of muscle was markedly anterior to the entrapped portion and was clearly distinct and separate from the fracture site*. Postoperatively, downgaze gradually improved, and 6 weeks later motility was normal, with orthotropia in all directions of gaze (Fig 8, right).

Case 2

A 36-year-old policeman was hit on the back of the head and fell forward, striking his face on the pavement. He suffered a brief loss of consciousness. When he became aware of his surroundings in the hospital on the next day, he noticed vertical diplopia. An imaging study of the head was reported to be normal. Twelve days later he presented for strabismus evaluation. Alignment was esophoria of 4Δ in the primary position, and left hypertropia of 16Δ with esotropia 4Δ in downgaze. The hyperdeviation was absent on right head tilt, and 6Δ on left head tilt. Left fourth cranial nerve palsy was diagnosed by a strabismus surgeon (I.H.L.) as well as by a neuro-ophthalmologist. There was no improvement by 2 months after injury, and no torsion was found with subjective testing or fundus examination.

The patient recalled that the left lower lid was ecchymotic after the injury. High-resolution magnetic resonance imaging of the orbits and brain, with particular attention to the left inferior rectus, was obtained, but no abnormality could be detected. Nine weeks after injury, he underwent exploration of the left inferior rectus via an inferotemporal fornix incision. A flap tear was found, with thinning of the muscle proximal to the musculotendinous junction and absence of capsule. The repair included closure of the overlying rent in Tenon's capsule. One week after surgery, alignment was orthotropia in primary position with left hypertropia of 10Δ in downgaze. Five weeks after surgery, the patient was orthotropic in all directions of gaze. Two years later he remains orthotropic in all directions.

DISCUSSION

Orbital trauma has been associated with a range of severity of ocular injuries.[11-13] Posttraumatic strabismus has traditionally been attributed to direct muscle contusion by an orbital fracture site, orbital hematoma, or nerve damage.[1-8] Spontaneous improvement in diplopia has been reported,[1,5,6,8,14] and patients are usually asked to wait for some time before strabismus repair is considered.[1-8] Orbital surgery to relieve entrapment is usually undertaken if diplopia persists after 2 to 3 weeks.[6,8] Strabismus repair is then not considered before 4 to 6 months.[1-8] Strabismus procedures that have been advocated include ipsilateral rectus resection and recession,[3] contralateral superior oblique recession,[3] rectus muscle transposition procedures,[8] and the Faden operation to the contralateral eye.[9]

Since we observed the presence of flap tears without orbital fracture, as well as the findings in 3 cases in which orbital and muscle repairs were concurrent, we believe that the avulsion was a related, but separate, finding due to the original trauma.

Recent anatomic studies of the extraocular muscles have shown 2 distinct layers: (1) the global layer, adjacent to the globe, and (2) the orbital layer, which lies externally. The orbital layer has also been shown to be surrounded by dense connective tissue and penetrated by elastin, which effectively inserts the orbital layer of the rectus muscle into the orbital connective tissue.[15]

We hypothesize that the sudden downward force experienced by the orbital contents at the time of blunt trauma may exert traction on the connective tissue insertion into the orbital layer of the muscle, tearing the outer layer away from the inner, global layer (Fig 12). This mechanism could result in the thinned-type appearance of the flap-tear muscle. Other flap tears presented with a narrowed appearance of the muscle, with a full or partial thickness defect of the remaining attached portion of muscle. These may have experienced asymmetric avulsion force, leading to asymmetric flaps. The force might possibly be transmitted to the muscle from the side by the intermuscular septum, which would also produce flap asymmetry.

Perhaps the 2 muscle portions may reunite during healing; this would explain the cases of spontaneous improvement that have been reported.[1-8] Motility findings vary according to the healing pattern of the flap tear. If the flap heals anteriorly, creating a tether, the predominant defect would be a loss of function of the involved muscle (Fig 13). Tether-type motility defects are reported to occur in about one third of orbital floor fractures with

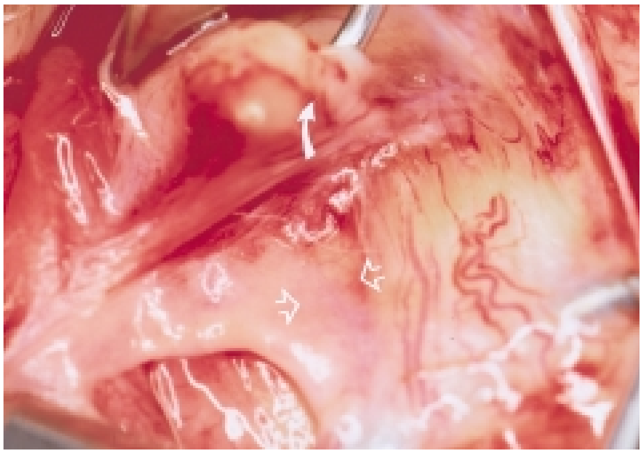

FIGURE 10
Case 1. Inferior rectus showing narrowed type of flap tear. Missing portion indicated by open arrows. Flap is held on small hook (solid arrow).

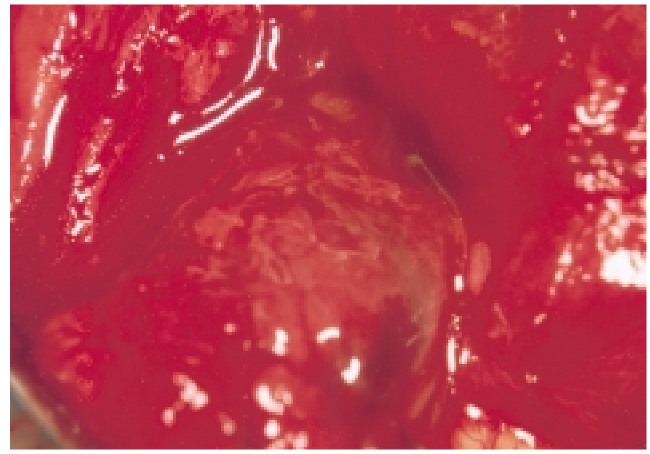

FIGURE 11
Case 1. Repaired inferior rectus, with Tenon's graft.

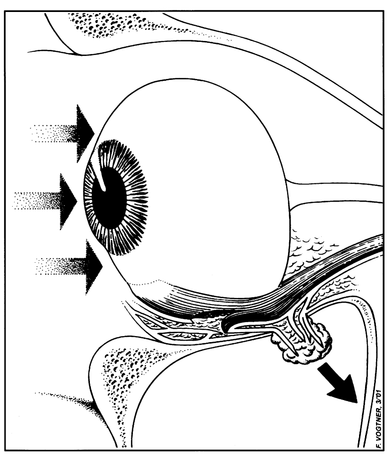

FIGURE 12
Drawing indicating hypothesized traction on inferior rectus outer layer due to orbital fracture, causing avulsion.

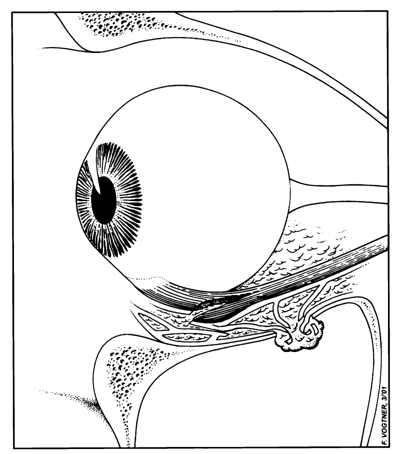

FIGURE 13
Drawing indicating anterior flap position after injury, which would create a tether.

vertical diplopia[14] and have also been reported with medial wall fractures.[16] A small tether effect of an inferior rectus flap tear could mimic ipsilateral fourth cranial nerve palsy. This was seen in 5 of our patients, and a similar motility pattern after floor fracture has been reported.[1,17] A posteriorly healed flap would lead to restriction of gaze away from the site of injury, which was also common. Restrictive strabismus was reported in two thirds of floor fracture patients with vertical diplopia.[14] An intermediate flap location could allow unimpeded motility. This is another possible explanation for spontaneous improvement of diplopia in some orbital fracture patients. Horizontal abnormalities resolved in several patients after flap tear alone, suggesting that horizontally directed adhesions might have contributed to the strabismus.

On the basis of our experience with this series of patients, we believe that a tether-type of motility defect is diagnostic of flap tear. Downgaze deficiency after orbital floor fracture has been attributed to palsy of the inferior branch of the third cranial nerve,[1-3] but in our series all cases with downgaze deficiency after ipsilateral orbital floor trauma had flap tears. The unexpected finding of identical tears in patients with long-standing strabismus, some of whom remembered the trauma only after careful questioning, is of interest. The trauma was sometimes uneventful and not immediately apparent. After one learns to recognize the abnormally narrowed or thinned rectus muscle with missing capsule, the defect becomes readily apparent in many cases. A completely restored muscle capsule confirms that repair is complete. This was even possible in cases repaired many years after injury.

Five patients had flap tears after retinal detachment repair. Perhaps the retinal surgeon's practice of bluntly stripping connective tissue off the extraocular muscle may pull away a flap of muscle tissue and lead to postoperative strabismus.

Immediate and regular eye exercise postoperatively is important to prevent adhesions from re-forming between the muscle and orbital connective tissue. The free Tenon's graft and repair of the overlying rent in Tenon's capsule also may help to reduce adhesions. Nonabsorbable suture is always used to reattach or anchor the flap to sclera.

On the basis of the findings in these patients, it is recommended that all orbital fracture patients with diplopia who are to receive fracture repair undergo simultaneous exploration of the adjacent rectus muscle(s) through separate fornix incision(s), with minimal dissection. If an unrecognized flap is allowed to attach itself to or near the implant material, the motility defect becomes more difficult to treat later. If diplopia is due to flap tear, fracture repair may not be necessary, but if entrapment and flap tear coexist, as in 3 cases of this series, combined repair produces the best outcome.

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DISCUSSION

Dr David L. Guyton. Dr Ludwig's description of "flap" tears of the extraocular muscles is both fascinating and convincing. Since the recent emphasis by Demer and colleagues[1] on the attachments between the orbital portions of the extraocular muscles and the surrounding connective tissue, strabismologists have been looking for practical applications of this knowledge. Drs Ludwig and Brown's flap tear mechanism appears to be consistent with this new view of connective tissue attachments.

Even before the connective tissue "pulley" concept, though, the so-called check ligaments, encountered at the time of surgery on the rectus muscles, have been well known to strabismus surgeons. Especially prominent are the dense attachments to the inferior rectus muscle, representing the origin of the retractors of the lower lid.[2,3] These particular attachments, also known as the capsulopalpebral head of the inferior rectus muscle, are very strong indeed. It is therefore not surprising that orbital trauma from an assailant, or from the cotton-tipped applicator of a retinal detachment surgeon, could tear portions of the inferior rectus muscle via these attachments, producing Dr Ludwig's "flap" tear. Indeed 87% of the involved muscles in her series were inferior rectus muscles.

How were the several medial rectus and superior rectus muscles involved? Their check ligaments are not very strong. Perhaps these cases were the result of locally directed trauma, actually shearing off or avulsing a portion of the muscle. Such injuries have been documented periodically in the literature. In a case of mine several months ago, the superior rectus muscle had been cleanly disinserted from the globe purely by trauma.

Dr Ludwig's contribution is more than just recognition of this "flap" tear mechanism. She has successfully repaired most of her cases. Because she has used several repair techniques, though, we still do not know which of these are necessary. For example, how important is the closing of rents in Tenon's capsule? How important is the free Tenon's tissue graft that she has used over the surgically repaired area? Should a non-reactive suture be used instead of an absorbable one? How important are range of movement exercises postoperatively, and do the patients really do them?

I am confident that Dr Ludwig will continue to research these questions. I congratulate her and her coauthor Mark Brown for an engaging and provocative presentation.

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[Editor's notes] DR EDWARD L. RABB asked why it was necessary to advance the torn flap. DR MALCOLM R. ING asked if an abrupt difference in muscle thickness suggesting a flap tear could be identified on a CT scan prior to surgical exploration.

DR IRENE H. LUDWIG. Regarding Dr Guyton's questions, I am not sure how important all these repairs are. My instinct is to fix a defect when I see it. I don't know if closing Tenon's capsule or employing a Tenon's graft is necessary. I did have 2 cases redetach when I didn't use the method of repair I described earlier. I had used absorbable sutures to reattach the flap, and I had not fully restored Tenon's capsule. Both of these cases were in children, who also did not exercise their motility postoperatively. I then repeated the surgeries the same as the initial repairs, but used non-absorbable sutures for flap reattachment, and free Tenon's graft over the muscles after repair. Their mothers increased the eye movement exercises. The second procedures worked.

Dr Guyton's suggestion of using a non-reactive suture to repair Tenon's capsule is an excellent idea. Perhaps a 7-0 polypropylene would serve well for this.

The patient's parents and/or spouses were charged with the importance of beginning the range of movement exercises immediately upon awakening. Several of those who admitted to poor compliance did not do well, but I do not have enough data to analyze this point. It is easy enough to recommend eye movement exercises, and it may be important.

To answer the question about imaging studies to preoperatively demonstrate a flap tear, I obtained no useful information from any of the preoperative CT and MRI scans I obtained. All were read as normal with respect to the extraocular muscles, excepting several with orbital fractures and posterior muscle entrapment. No anterior abnormality could be specifically identified. A few showed vague scar tissue or edema under the inferior rectus, but these had already undergone orbital fracture repair. One case (case 2 in the manuscript), had several repeat high resolution MRI scans performed of the suspect inferior rectus, with no abnormality seen by the radiologist, despite my insistance that something must be there. I talked myself into thinking the muscle capsule was irregular, but there was really no useful information obtained. His flap was small. The MRI scan would have deterred most from exploring the muscle. I now limit imaging to orbital CT scans to look for fractures.

To answer the question about why I advance the flap to the insertion, when some flaps originate a few millimeters posterior to the insertion, I do this when there is a tether limiting the action of the muscle. This tends to strengthen the muscle. In those muscles with restriction of gaze away from the muscle action, I leave the flap a little further back.