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Force-length recording of eye muscles during local-anesthesia surgery in 32 strabismus patients

H. J. Simonsz

Strabismus and Neuro-ophthalmology, Department of Ophthalmology, University Hospital Dijkzigt, NL 3015 GD Rotterdam and the Netherlands Ophthalmic Research Institute P.O.Box 12141, NL 1100 AC Amsterdam

Abstract Force-length recordings were made from isolated human eye muscles during strabismus surgery in local, eye-drop anesthesia in 32 adult patients. From each muscle three recordings were made: (1) while the patient looked with the other eye into the field of action of the recorded muscle, (2) looked ahead, and (3) looked out of the field of action of the recorded muscle. Non-innervated eye muscles (state 3) had an approximately exponential relation between force and length. During contraction evoked by letting the patient look ahead or into the field of action of the muscle (states 1 or 2), the relation between force and length was grossly linear. The approximate spring constants of horizontal rectus muscles that had not been operated on before ranged from 2 to 4 g/mm. In palsies, the degree of muscle paresis could be quantified accurately using this method and, accordingly, cases of true superior oblique palsy could be well differentiated from strabismus sursoadductorius (= upshoot in adduction) that may mimic a superior oblique palsy.

In seven patients with Graves' disease of recent onset, affected muscles were found to be very stiff when the other eye looked ahead. It was expected that these stiff muscles would be able to shorten to some extent but would not be able to lengthen, due to fibrosis of the muscle. We found, however, that the affected muscles lengthened considerably when the other eye looked out of the field of action of the muscle. This implies that, in these cases of Graves' disease of recent onset, the raised muscle tension and reduced elasticity of the affected muscles and, hence, the strabismus were primarily caused by active muscle contraction, not by fibrosis.

Key words Eye movements; eye muscles; force-length recordings; superior oblique muscle palsy; Graves' disease

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Introduction The relation between force and length of human eye muscles has been assessed previously by Robinson *et al.*[1] and Collins *et al.*[2] In their experiments, a suture from the lateral rectus tendon, after it was detached from the eye during strabismus surgery (under local anesthesia), was fastened to a strain gauge to measure force. During a single, isometric recording, innervation to the muscle was kept constant by having the patient fixate a target with the other eye. Between recordings, the gauge was moved with a worm gearing to change muscle length: 2, 5 and 8 mm either way from an estimated primary-position length. Between recordings, the patient changed innervation to the recorded eye muscle by looking, with the other eye, at one of several target lights.

In a previous study,[3] we recorded force and length of eye muscles in patients who were operated for strabismus under general anesthesia. Instead of measuring the force at a given length and a given level of innervation repeatedly, we recorded length continuously, very slowly increasing the force applied to the muscle, at zero innervation. We found an exponential relation between force and length for non-innervated eye muscles. Many collagenous tissues, including skeletal muscle, skin, mesentery and sclera obey similar exponential relationships between force and length.[4-6]

These muscles were not innervated and did not contract. Instead, we used intravenous succinylcholine to evoke eye muscle contraction (multiply innervated muscle fibers in eye muscles contract strongly during several minutes on depolarization by succinylcholine). We found a linear relation between force and length when the muscle was contracting.[7,8] In the study described below, the recordings were performed during strabismus surgery under local, eye-drop anesthesia, so that the mechanical characteristics of eye muscles could be assessed in the natural, innervated state.

For understanding the discussion that follows, it is important to realize that a muscle has a different force-length relation at each level of innervation. To put it simple: when a muscle is innervated and contracting intensely it behaves mechanically like a short spring; when it is not contracting it behaves mechanically like a long spring. During eye movement and acceleration, different levels of innervation occur together with different levels of force at any length, but when the eye is not moving, for each particular direction of gaze the force, length and innervation of an eye muscle are fixed. When the eye is not moving, only one single point of the force-length relation at a particular level of innervation applies.

Robinson *et al.*[1] and Collins *et al.*[2] found that the relation between force and length of an innervated eye muscle is linear at higher levels of innervation. In other words, an innervated eye muscle behaves like an ordinary coil spring in that the force rises proportional to the length. They also found that the spring constant, or stiffness, of an innervated eye muscle remained approximately the same whether it was innervated moderately or strongly.

How can the relation between force and length of an innervated eye muscle be linear when its component forces, *i.e.*, force generated by the sarcomeres (active component) and force resulting from stretch of the connective tissue in eye muscles (passive component) are both non-linear ?

Sarcomeres can generate maximal force (active component) at a muscle length slightly longer than the average *in-situ* length, this length varying slightly per muscle-fiber type.[9] For eye muscles, maximal force development is near or at slightly more than primary-position length.[10] At this length the actin and myosin molecules that together constitute the sarcomere, are intertwined over a distance that permits the largest possible number of actin-myosin cross-bridges to contract unencumbered.[11] At shorter muscle lengths, the actin and myosin forks butt; at longer muscle lengths, there are too few crossbridges.

The force resulting from passive stretch of the muscle (passive component) is zero when the muscle is short and slack, starts to rise from zero approximately at primary-position length and rises exponentially thereafter. So when an intensely innervated, short muscle is stretched, first the force rises because of the contracting sarcomeres (active component) and then, when the muscle is stretched further, because of stress of the collagenous parts of the muscle (passive component). Accordingly, whether the relation between force and length of an eye muscle will be linear or not depends upon the amount of connective tissue in the muscle and the structure of the muscle.[6] Eye muscles have very much connective tissue[10] and, hence, the force-length relation of an innervated eye muscle is approximately linear. Only at low levels of innervation does the relation between force and length become exponential as the passive component starts to predominate.

Force and length recording of eye muscles has been applied clinically by many strabismus surgeons.[12-26] Several of these have tried to relate the results of their measurements to the outcome of the surgery. The techniques employed varied and some of them had limitations.

Firstly, continuous recording was never employed when increasing and decreasing force applied to the muscle, making it very difficult to quantify hysteresis. The hysteresis is caused by the viscoelastic properties of the muscle, consisting partly of frictional interaction between active and passive fibers.[9] In our recordings, we found it necessary to increase and decrease force over at least 45 s either way, to avoid artefacts. Secondly, most of the measurements were done with large forces, up to 100 g, while the *in-vivo* force of eye muscles is between 5 and 40 g most of the time. Thirdly, most measurements were done during general anesthesia. Fourthly, in some measurements the muscle was not separated from the globe, resulting in additional forces caused by displacement of the center of the eye.

Patients and methods In 44 patients who were operated for strabismus in local, eye-drop anesthesia force and length were recorded of one or two eye muscles that were separated from the globe during surgery. Data on 32 of these recordings are presented below, the other 12 recordings being unsuited for analysis due to artefacts or apparatus breakdown. Five percent cocaine or 2% tetracaine eyedrops were administered every five minutes during half an hour preceding surgery. In older patients, such surgery is well tolerated if sudden jerking of the muscles is avoided. Additional cocaine or tetracaine eyedrops were usually needed towards the end of the operation, when closing the conjunctiva (the conjunctiva is the most pain-sensitive). All patients gave oral informed consent.

A force and length measuring apparatus was fixed rigidly above the head of the patient. The measuring apparatus has been described previously.[27,28] In short, it had a 2 mm hardened steel shaft, suspended by precision micro-ballbearings and equipped with a shaft-position encoder and an eddy-current motor. The torque generated by the eddy-current motor in the shaft (proportional to the force applied to the muscle) and the rotation of the shaft (proportional to the length of the muscle) could be assessed electronically. The frame on which the apparatus was mounted consisted of vertical 30 mm steel posts fixed to the railing of the operating table, a 20 mm steel transverse round bar above the chest of the patient and a 20 mm steel round bar that could be clamped tight in any position rectangular to the transverse bar. During the recording the apparatus was positioned above the head of the patient, but during the rest of surgery it was withdrawn. The apparatus was covered with sterile drapes. The tip of the shaft, used to attach the suture, protruded through a hole in the drape. To avoid contamination, the tip was covered for at least 20 minutes prior to the recording with a little drape soaked with an iodine disinfectant and care was taken not to touch this part other than with the suture that connected the muscle with the shaft. During the recording, the position of the tip of the shaft was in line with the muscle to be recorded (detached from the globe). In recordings of oblique muscles, the head of the patient was turned contralaterally and the apparatus was positioned such that the muscle was pulled on in a frontal plane.

After detaching the muscle from the globe, it was connected to the shaft of the length-tension measuring apparatus with a 4.0 silk suture. The suture was passed through the muscle to be recorded and knotted around the shaft. Then the torque generated in the shaft was slowly (taking more than 45 s either way) increased and then slowly decreased, resulting in a maximal force of 40 g in the suture. The suture was rolled onto the shaft in a regular coil fashion, and recordings where the suture had crossed itself on the shaft were discarded.

The recording was performed three times: once while the patient looked ahead with the other eye, once while the patient looked between 35° and 45° into the field of action of the muscle, and once while the patient looked between 35° and 45° out of the field of action of the muscle. During the recording the drape on the not-operated eye of the patient was lifted so that both eyes of the patient were visible for the surgeon. The surgeon instructed the patient to look ahead at his hand, at his hand approximately 40° out of the field of action of the muscle or at his hand approximately 40° into the field of action of the muscle. The patient had to look for at least 90 s into the same direction, and this was done with varying degrees of success. The degree of eccentric gaze varied between 30° and 45° in the worst cases. At points were the surgeon reiterated to the patient to keep looking at his hand saccades occurred. During recordings of oblique muscles the patients looked maximally in downgaze, ahead or upgaze.

An xy-recorder (Hewlett-Packard 7035BXY) was used. As the diameter of the silk suture was 0.16 mm and the diameter of the shaft was 1.998 mm, the effective radius was calculated to be 1.08 mm. This value was confirmed by rolling a fixed length of suture on the shaft. The shaft-position encoder (Hohner Elektronik, Tuttlingen, Germany) was described previously.[3,27,28] In short, it consisted of a transparent disc with 150 radial, black lines and two fixed, light-emitting diodes shining through the edge of the disc and through two static patterns of black lines (90 deg out of phase to discern between clockwise and anti-clockwise rotation) onto two light-sensitive transistors. Rotation of the shaft resulted in sawtoothed pulses from the two channels of the shaft-position encoder. These were converted into rectangular pulses and then counted binarily. After digital to analogue conversion, the signal was fed into one channel of the xy-recorder. The AC current through the variable-current coil of the eddy-current motor was proportional to the torque generated in the disc. The voltage across the coil was found to be proportional to the current, within 5% over the entire range. The voltage was amplified, rectified and fed into the second channel of the xy-recorder. The starting friction of the shaft, caused by the precision micro-ballbearings was less than 1 g.mm. Calibration of the force generated in the disc of the eddy-current motor was with weights on a 4.0 silk suture rolled onto the shaft. The small errors resulting from the weight of the suture and the muscle were neglected. To check for displacement of the measuring apparatus and strain of the suture, we let the apparatus pull an extra long silk suture fixed to the steel head support, at full bar extension, which was about 1.5 times the extension used in the experiments. Under these circumstances, the displacement of the apparatus and the strain of the silk suture together were 0.8 mm at 40 g load.

Results The age, diagnosis, short history and angles of strabismus of the patients are listed in Tables 1–6. The patients have been grouped according to diagnosis: (1) horizontal rectus surgery, (2) vertical rectus surgery, (3) oblique muscle surgery, (4) reoperation, (5) palsy and (6) Graves' disease. The patients are coded accordingly with a capital, indicating the group (H, V, O, R, P or G), and a number. An effort has been made to characterize the force-length registrations by estimating, at 10 g force, the approximate slope of the recording (equal to the spring constant at 10 g load) and the relative length of the muscle. This is not an immaculate procedure, but not all of the original recordings can be presented here and there seems no better way to characterize the remaining. The spring constants are expressed in unit g/mm (for your convenience: For the average eye, 1 mm on the surface of the eye equals 4.89° of eye rotation, assuming a radius of the globe of 11.71 mm, which conforms to an axial length of 24.6 mm,[29] so divide by 4.89 to get unit g/deg, used for spring constant in other papers). The force-length registrations of a selection of the patients are depicted in Figs. 1–6.

The hysteresis (difference between recording during force increase and recording during force decrease) in the force-length registrations was considerable. It was kept to a minimum by increasing and decreasing force very slowly, both over a period of more than 45 s. The viscoelastic force is equal, on average, to half the vertical distance between the two limbs of the registration at a given muscle length. Hence the actual relation between force and length, without hysteresis, can be thought of as lying vertically halfway between the two recordings.

When the patient looked out of the field of action of the recorded muscle, the relation between force and length was approximately exponential, as found in previous recordings during general anesthesia.[7] During contraction evoked by letting the patient look ahead or into the field of action of the muscle, the relation between force and length was grossly linear.

Spring constants of (not previously operated) horizontal eye muscles (Table 1 and Fig. 1) ranged from 2 to 4 g/mm. In innervated inferior rectus muscles (Table 2 and Fig. 2), the spring constants were about 2 g/mm in case V2, but about 4 g/mm in case V1.

In case O1 (Table 3 and Fig. 3) force-length recordings were made of the superior oblique muscle while the other eye looked ahead, in adduction, abduction, in adduction-downgaze and abduction-downgaze. Surprisingly, the registrations in abduction, ahead and adduction were the same, as were the registrations in adduction-down and abduction-down gaze. In cases O2, O5, O6, O7 and O8 the superior oblique muscle did not shorten when the patient

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TABLE 1. Horizontal rectus muscles (first surgery)

In Tables 1–6, patient data, angles of strabismus, muscle spring constants and muscle lengths are listed together for each of the six groups of patients. 'VD' denotes vertical divergence, 'DVD' dissociated vertical divergence, 'NRC' normal and 'ARC' anomalous retinal correspondence, 'LS' and 'RS' denote VDs on approximately 45° head tilt to the left or right shoulder (note that, in some cases, the measurement was repeated with simultaneous up- or downgaze). 'LR', 'MR', 'SR', 'IR', 'SO' and 'IO' refer to the six eye muscles, preceded by 'R' or 'L' for right or left eye, respectively. **'H'**denotes horizontal, **'V'** vertical and **'C'** cyclo phoria in nine 25°/25° directions of gaze. The angles were measured during fixation with the non-squinting eye, using alternating cover test whenever possible. Esophoria, right hyperphoria and excyclophoria have positive operating signs. All values in degrees. Following the patient data, the recordings have been summarized by listing the spring constants and relative lengths of the recorded muscle at 10g load. The spring constant was found by measuring the slope of the recording at 10g (10g was chosen rather arbitrarily, it is approximately the resting force in a rectus muscle in gaze ahead). From each muscle three recordings were made: while the patient looked with the other eye into the field of action of the measured muscle (**'on'**), looked ahead (**'ahead'**) and looked out of the field of action of the measured muscle (**'off'**). Therefore, three spring constants and three relative lengths are given for each muscle. The spring constants are expressed in unit g/mm; the relative lengths are expressed in millimeters shorter than **off** length.

H1, 59 years, divergent and right strabismus sursoadductorius, blepharospasm treated with botulinum.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up	-20	15	-23	8	13	-22	4		
ahead	-22	17	-22	13	13	-19	9	14	16
down	-18	22	-18	18	13	-16	14		

H2, 33 years, esophoria, diplopia when tired, especially at near, worsening in recent months. She had 5° of esophoria, increasing to 6° at near fixation.

H3, 67 years, decompensated esophoria since ten years of age, small vertical phoria, small bilateral limitation of abduction, 6 diopters of myopia, TNO: 480", 11° esophoria, 9° at near fixation.

H4, 26 years, accommodative esotropia, treated with occlusion and glasses at age six, now conspicuous strabismus when wearing contact lenses, alternating suppression, 31° esotropia, 29° at near fixation.

H5, 62 years, exophoria, convergence insufficiency, –15° exophoria, –24° at near fixation, accommodation near point at 14 cm, TNO: 480", used antidepressives.

Spring constants and relative lengths

		spring constants			*relative lengths*		
pat	**muscle**	**on**	**ahead**	**off**	**on**	**ahead**	**off**
H1	RLR	3.2	4.3	4.5	17	2	0
	RMR	3.5	4.1	4.1	10	0	0
H2	RMR	2.9	3.1	3.9	15	7	0
H3	LMR	5.1	3.9	4.3	23	5	0
	LLR	8.4	3.2	4.7	24	14	0
H4	RMR	3.1	3.0	2.7	12	6	0
	RLR	3.0	2.6	2.7	13	4	0
H5	LLR	4.1	3.1	3.2	13	5	0
	LMR	5.8	4.1	4.1	10	2	0

TABLE 2. Vertical rectus muscles

V1, 50 years, right convergence, hypertropia and deep amblyopia, RLR resected at age 19, 11° esotropia and 7° right hypertropia measured with corneal reflexes and prisms, no DVD, negative Bielschowsky head-tilt test.

V2, 17 years, torticollis (chin down, towards right shoulder) since two years, no strabismus noted previously; blurred vision when head upright. Titmus: 200″, fundus anatomically slightly intorted, Ct negative.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up	-2	-6	-1	-8	-3	-2	-12	-6	-6
ahead	-1	-10	-1	-13	-3	-1	-16	-14	-10
down	-2	-14	-1	-16	-4	-1	-18	-15	-12

Spring constants and relative lengths

		spring constants			*relative lengths*		
pat	**muscle**	**on**	**ahead**	**off**	**on**	**ahead**	**off**
V1	RIR	5.1	3.9	4.9	16	2	0
	RMR	5.1	4.1	4.1	15	6	0
V2	LIR	2.1	2.0	2.8	12	6	0

TABLE 3. Oblique muscles

As with rectus muscles, three recordings were made from each oblique muscle measured, the patients looking in downgaze, ahead or in upgaze.

O1, 36 years, RE consecutively divergent, right amblyopia and downshoot in adduction of the RE. Force-length recordings of RSO were made while other eye looked into direction adduction-down, abduction-down, abduction, ahead, adduction.

	left		*ahead*			*right*	
	H	**V**	**H**	**V**	**C**	**H**	**V**
ahead		-4	-21	-2			-1

O2, 31 years, right superior oblique palsy, suffered commotio cerebri in 1988 but had had torticollis and strabismus since childhood; no diplopia but NRC with Haidinger brushes and afterimage; Bagolini positive in extreme head-tilt, in moderate head-tilt a small manifest VD was present.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up	-3	18	-3	13	5	0	4		
ahead	0	25	-1	17	5	0	3	12	16
down	4	25	2	17	5	2	7		

O3, 66 years, right superior oblique palsy, vertical diplopia for 17 years.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up		5		5	3		5	5	5
ahead		7		6	5		5	5	7
down		6		7	3		5	4	6

O4, 44 years, strabismus sursoadductorius of the left eye, myopia (S-10/S-10) with macular degeneration (RE: 0.16, LE: 0.5), transient diplopia after accident in 1967, right head-tilt.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up		0		-3	6		-5	-10	0
ahead		-3		-6	6		-6	-14	-1
down		-2		-5	6		-4	-10	-2

TABLE 3. *Continued*

O5, 48 years, right superior oblique palsy, possibly mechanical; eight weeks in hospital for explosion eye injury at age ten. Slight bilateral ptosis, right eyelid elevation on adduction and retraction on downgaze of the RE, negative Tensilon test.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up		3		1	8		0	3	1
ahead		8		5	8		3	3	2
down		6		5	8		4	4	5

O6, 59 years, left superior oblique palsy. After collapse due to cardiovascular disease nine years previously right head tilt started and a diagnosis of an eye muscle palsy was made.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up	-3	-10	-4	-11	-2	-7	-13	-14	-8
ahead	0	-6	-2	-14	-1	-3	-17	-24	-6
down	0	-17	0	-21	2	1	-21	-29	-8

O7, 29 years, partial superior oblique paresis, left head tilt since a long time, diplopia on right head tilt, binocular single vision on downgaze.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up	0	-2	0	-4	5	-1	-7	-4	-2
ahead	2	-3	1	-5	4	1	-8	-5	-3
down	2	-3	1	-3	2	1	-5	-4	-2

O8, 58 years, partial superior oblique paresis, previous temporal plication of the right superior oblique muscle tendon eight years previously.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up	-3	4	-3	0	3	-1	0	0	2
ahead	-3	10	-4	5	8	-1	2	1	7
down	-1	12	-3	9	10	0	4	0	12

Spring constants and relative lengths

		spring constants			*relative lengths*		
pat	**muscle**	**on**	**ahead**	**off**	**on**	**ahead**	**off**
O1	RSO	3.5	2.5	2.5	9	1	0
	abd-down	3.5			9		
O2	RIO	2.6	3.4	5.6	8	2	0
	RSO	2.4	2.4	2.4	2	1	0
O3	RSO	5.8	5.8	5.8	4	2	0
O4	LSO	6.1		6.1	8		0
O5	RSO	2.7		2.7	1		0
O6	LSO	2.5		4.1	1		0
O7	LIO	5.1		3.8	2		0
	LSO	4.3		4.3	1		0
O8	RSO	4.7	4.1	3.9	2	1	0

TABLE 4. Horizontal rectus muscles (reoperation)

R1, 48 years, previous total tenotomies of LMR and of RLR, and partial tenotomy of LLR, all more than 33 years previously, left amblyopia. Two weeks before current operation RLR had been reattached to old insertion

R2, 29 years, secondary divergence after two operations on left eye in 1965 (medial rectus recession and 4 mm lateral rectus resection of left eye) for left convergent strabismus since 6th month of age with failed amblyopia treatment and eccentric fixation of left eye. Manifest angle during fixation at distance –25°, during fixation at near –19°, VD –3°.

R3, 34 years, divergent, two operations for strabismus seven years previously, now bilateral limitation of adduction. Manifest angle during fixation at distance –24°, during fixation at near –31°.

	left		*ahead*			*right*	
	H	**V**	**H**	**V**	**C**	**H**	**V**
up			-27	-1			
ahead	-21	-3	-22	-1		-22	-3
down			-25	-1			

R4, 77 years, secondary divergent strabismus, total tenotomy of LMR at age twelve, deep left amblyopia, left eye could adduct 10°.

	left		*ahead*			*right*	
	H	**V**	**H**	**V**	**C**	**H**	**V**
up			-22	6			
ahead	-11	6	-17	6		-2	5
down			-15	6			

R5, 47 years, esophoria and strabismus sursoadductorius for ten years, RMR recessed five years previously, LMR recessed 3 mm three months before the current operation, RIR recessed eight years previously and LIO recessed one year previously.

	left		*ahead*			*right*	
	H	**V**	**H**	**V**	**C**	**H**	**V**
up	5	2	7	4	5	-1	4
ahead	9	2	8	2	4	3	1
down	11	2	11	1	3	3	0

Spring constants and relative lengths

		spring constants			*relative lengths*		
pat	**muscle**	**on**	**ahead**	**off**	**on**	**ahead**	**off**
R1	LMR	5.8		9.7	9		0
R2	LLR	4.9	4.3	3.0	14	8	0
	LMR	6.1	3.4	4.5	14	3	0
R3	LLR	2.8	3.4	3.6	13	7	0
	LMR	3.0	2.9	2.9	5	2	0
R4	LMR	4.7	3.0	4.7	13	5	0
R5	LMR	4.3	3.5	5.6	8	4	0

TABLE 5. Palsies other than superior oblique

P1, 45 years, partial RLR paresis with multiple sclerosis, diplopia since 14 years, facial palsy, left myokymia.

	left		*ahead*			*right*	
	H	**V**	**H**	**V**	**C**	**H**	**V**
up			3	0			
ahead	6	0	14	0		25	-5
down			7	0			

P2, 70 years, skew deviation after pons infarction 20 months previously. She was being treated for hyperthyroidism and, although she had eyelid retraction and a limitation of elevation of the right eye, a recent orbital CT-scan was negative.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up	-4	-7	-5	-12	13	-5	-15		
ahead	-2	-6	-3	-7	7	-2	-7	-10	-6
down	0	-8	0	-5	7	0	-3		

P3, 82 years, internuclear ophthalmoplegia, approximately 60° divergent since age 66, downbeat nystagmus, diabetes mellitus, bilateral limitation of adduction, optic atrophy with visual acuity 0.3 and 0.5.

Spring constants and relative lengths

		spring constants			*relative lengths*		
pat	**muscle**	**on**	**ahead**	**off**	**on**	**ahead**	**off**
P1	RLR	3.1	3.2	3.2	5	2	0
P2	RIR	2.9	4.1	5.8	9	0	0
P3	RLR	2.9	3.4	4.5	26	6	0
	RMR	4.1	4.1	4.3	2	1	0

TABLE 6. Graves' disease

G1, 50 years, Graves' disease, proptosis since 16 months previously, corticosteroids and radiation of the left orbit (20 Gy) were tried eight months previously, without success. Horizontal angles at that point were +10°, +7° and +4° in left gaze, ahead and right gaze, without cyclophoria. For decreasing visual acuity ethmoidal and maxillary decompression was performed four months previously, and the relative left proptosis was reduced from 5 to 1 mm, but the convergent strabismus increased. Now horizontal angles were +23°, +18° and +13° in left gaze, ahead and right gaze.

G2, 55 years, Graves' disease with bilateral limitation of elevation, slight left hypertropia and upshoot in adduction of the LE. One year previously diplopia and eyelid retraction developed. No hyperthyroidism was present, neither anamnestic, nor biochemically. The angles of vertical strabismus were 1°, −4° and −6° in left, ahead and right gaze, and −6° and −3° in up and downgaze.

G3, 50 years, Graves' disease, strumectomy and thyroid substitution started nine months previously. Diplopia since six months with bilateral limitation of abduction and elevation.

	left		*ahead*			*right*	
	H	**V**	**H**	**V**	**C**	**H**	**V**
up	5	2	9	0		10	0
ahead	9	1	11	0		12	0
down	11	1	13	0		15	0

G4, 61 years, Graves' disease, treated for hyperthyroidism for five years, diplopia for three years, proptosis for two years. Irradiation did not lead to improvement.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up	-3	12	0	15	1	1	14		
ahead	0	11	1	14	1	2	17	6	25
down	2	9	2	12	-1	3	16		

G5, 34 years, Graves' disease treated with strumectomy one year previously, diplopia and extreme proptosis since six months. The left eye could not elevate above the primary position. Curiously, intraocular pressure was about 25 mm Hg in gaze ahead but did not rise on up-gaze. Monocular motility: RE: 45° left, 40° right, 20° up and 50° downgaze; LE: 30° left, 32° right, −5° up and 50° downgaze.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up	-2	26	-3	25	7	-1	21		
ahead	0	23	0	22	7	0	21	14	26
down	1	11	0	15	7	1	15		

G6, 38 years, Graves' disease, swelling of the eyelids, diplopia and headache first noticed 20 months previously.

	left		*ahead*			*right*	
	H	**V**	**H**	**V**	**C**	**H**	**V**
up	3	-12	2	-13	0	1	-12
ahead	1	-10	0	-10	-1	2	-12
down	0	-11	1	-12	0	3	-12

G7, 59 years, Graves' disease, diplopia since one year, hyperthyroidism diagnosed eight months previously.

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up	1	9	3	8	9	1	6		
ahead	5	8	4	8	2	3	9	7	9
down	6	8	6	11	2	5	10		

Second operation, seven months later: At the first operation a 1 mm recession of the LIR and a 3 mm resection of the LSR had been performed. A very thick inferior rectus had been found. Now (second operation) a RIO recession and a LIR anteroposition were performed that, curiously, still undercorrected the secondary left hypertropia.
Two days after first operation:

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up	4	2	2	2	3	1	3		
ahead	4	0	3	0	0	1	1	-1	1
down	4	-1	3	-1	0	2	1		

Shortly before second operation:

	left		*ahead*			*right*		*Bielschowsky*	
	H	**V**	**H**	**V**	**C**	**H**	**V**	**LS**	**RS**
up	-7	-1	-1	-2	14	-4	-5		
ahead	3	-2	3	-1	0	0	-2	-1	0
down	4	-3	4	0	0	3	-2		

TABLE 6. *Continued*

TABLE 6. *Continued*

G8, 71 years, hyperthyroidism diagnosed two years previously, diplopia and proptosis started more than one year previously. A transantral orbital decompression was performed 11 months previously, after which the bilateral limitation of abduction and elevation worsened.

	left		*ahead*			*right*	
	H	**V**	**H**	**V**	**C**	**H**	**V**
up	18	6	21	0	15	17	-7
ahead	19	7	19	5	6	20	4
down	20	4	20	5	3	19	1

Spring constants and relative lengths

In one case, the spring constant could not be assessed with any precision, indicated by parentheses.

		spring constants			*relative lengths*		
pat	**muscle**	**on**	**ahead**	**off**	**on**	**ahead**	**off**
G1	LMR	15.4	15.4	7.5	16	16	0
	LLR	2.8	3.6	7.1	23	8	0
G2	RIR	7.5	7.1	(2.7)	14	8	0
G3	LMR	6.1	4,9	7.9	12	7	0
G4	LIR	6.1	6.4	7.1	16	11	0
G5	LIR	7.9	7.9	7.1	11	8	0
	LSR	2.4	4.7	4.2	12	11	0
G6	RIR	6.1	4.1	7.9	16	9	0
	RSR	4.9	3.1	4.7	13	4	0
G7	LIR	6.1	4.1	4.7	19	14	0
	LSR	8.4	3.9	3.9	11	1	0
2nd	LIR	6.7	4.7	7.5	7	3	0
	RIO	2.8	2.8	2.8	4	2	0
G8	RMR	4.7	4.7	6.7	7	1	0
	RLR	7.5	5.8	5.8	8	2	0

looked down, being indicative of a superior oblique palsy. In case O3 the results were unclear: the spring constant of the superior oblique muscle was high, but the muscle shortened 4 mm. In cases O2 and O7 the ipsilateral inferior oblique muscle was also recorded. In case O2, the result was unclear because the spring constant of the superior oblique muscle was normal, but it did not shorten; the inferior oblique muscle shortened normally. In case O7 the superior oblique muscle did not shorten; the inferior oblique muscle did shorten to some extent but with a high spring constant. In case O4 the superior oblique muscle shortened 8 mm at 10 g load, despite a motility that indicated a superior oblique palsy.

Eye muscles that had previously been tenotomized totally (cases R1 and R4) were stiff, but could still shorten 9 and 13 mm at 10 g load (Table 4 and Fig. 4).

In palsies, the level of paresis could be assessed: of a lateral rectus in case P1, and of a medial rectus in case P3 (Table 5 and Fig. 5).

In Graves' disease (Table 6 and Fig. 6), affected muscles ('affected' meaning that the muscle was clearly thickened in a coronal CT-scan) were found to be very stiff when the other eye looked ahead. Affected muscles lengthened considerably when the other eye looked out of the field of action of the recorded muscle and they did not shorten very much when the other eye looked into the field of action of the recorded muscle. This was particularly true for the medial rectus muscle in case G1. In case G2, some shortening (5 mm) occurred when the other eye looked into the field of action of the affected muscle, but the lengthening from gaze-ahead length (7 mm) was larger (all quoted muscle lengths refer to muscle length at 10 g load). In case G3, the medial rectus shortened by approximately 4 mm, but lengthened 5 mm. In case G4 the inferior rectus muscle shortened 6 mm, but lengthened about 10 mm. In case G5 not only the inferior rectus, but also the superior rectus showed more lengthening than shortening from the length when the other eye looked ahead. Both were thickened in the coronal CT-scan, but the finding remains puzzling as the limitation of elevation was much larger than the limitation of depression in this patient.

Never any adverse effects occurred due to the experimental procedure. No infections or conspicuous over- or undercorrections occurred, except in case G7 (late overcorrection after inferior-rectus recession in Graves' disease).

Discussion Before proceeding to the discussion of the results, the limitations of the method used must be delineated. First, only when the patient looked ahead with the other eye, fixation was well maintained. When the patient was instructed to look at the hand of the surgeon, approximately 40° into or out of the field of action of the recorded muscle, the actual direction of gaze varied between 35° and 45°, because the patients had to maintain this direction of gaze for at least 90 s, and some simply could not. Secondly, even in gaze ahead inadvertent saccades occurred. These were dampened by the mechanics of the apparatus. Thirdly, in some cases cocaine drops had to be given towards the end of surgery, in most cases only during closure of the conjunctiva, but occasionally earlier. In that case the most anterior parts of the second muscle to be recorded may have been anesthetized during the recording. Fourthly, although head movement was restricted by the use of drapes that were adhesive to the skin and fixed to the operating table, it was not possible to fully restrain any movement of the head. Despite these limitations, several conclusions can be drawn.

In horizontal strabismus surgery (cases H1 – H5, Table 1 and Fig. 1) spring constants of (not previously operated) horizontal eye muscles ranged from 2 to 4 g/mm. This is higher than the spring constants in previous recordings in general anesthesia where contractions were induced by intravenous administration of succinylcholine.[7] In those recordings, spring constants of unoperated horizontal eye muscles ranged from 1.7 to 2.7 g/mm. Although being lower, the spring constants during contraction evoked by succinylcholine are still surprisingly high, considering the fact that only 10% of the global-layer muscle fibers and 30% of the orbital-layer muscle fibers are multiply innervated and, hence, contract after succinylcholine.[10]

High spring constants were found for the medial rectus muscle in case H3, with an esophoria, and for the medial rectus muscle in case H5, with a convergence insufficiency. It is tempting to speculate about the reason for this high spring constant but, on the other hand, case H2 also had an esophoria and had normal spring constants.

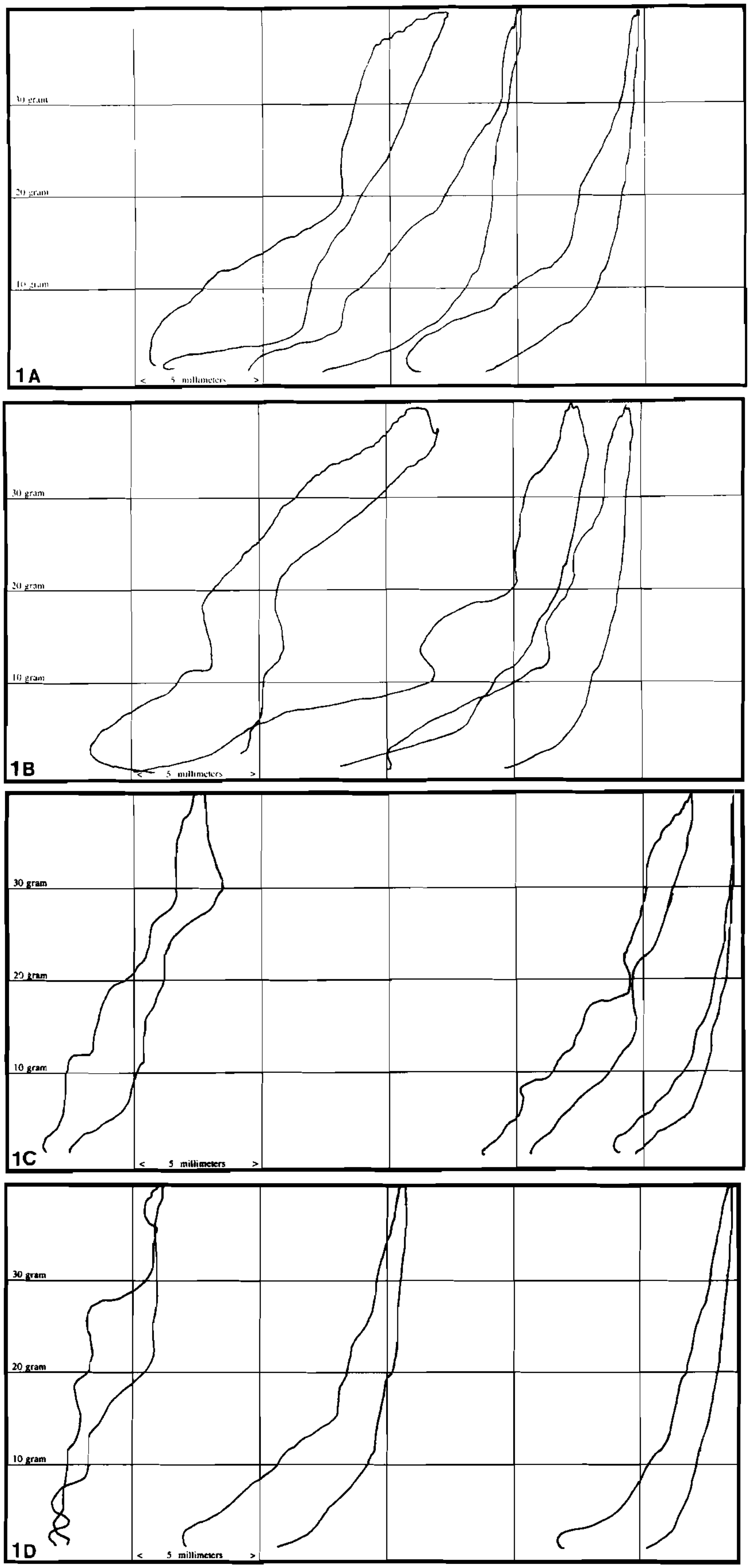


Fig. 1. Force-length recordings of horizontal rectus muscles. All force-length registrations are composed of a left limb (registration during force increase) and a right limb (registration during force decrease). The difference between the two limbs was caused by hysteresis, mainly friction of the muscle in its sheaths and within the muscle. Ordinate: force (unit: g). Abscissa: length (unit: mm). Absolute abscissa values are lacking, because the actual length of the muscle was not known, nor was it possible to determine the primary-position length accurately, after the muscle had been separated from the globe.
Each diagram contains two or three force-length recordings from one muscle. Left curve: during this recording the patient looked with the other, not operated, eye into the field of action of the muscle to be recorded. The surgeon let the patient fixate his hand in approximately 40° eccentric gaze as well as was possible under the circumstances and urged the patient to keep looking into that direction. Middle curve (only if the diagram contains three curves): during this recording the patient looked ahead with the other eye. Right curve: during this recording the patient looked with the other eye out of the field of action of the muscle to be recorded. (A) medial and (B) lateral rectus muscles of case H4, who had 30° of accommodative esotropia. (C) Medial and (D) lateral rectus muscles of case H3, who had 11° of esophoria, a small vertical phoria and 6 diopters of myopia.

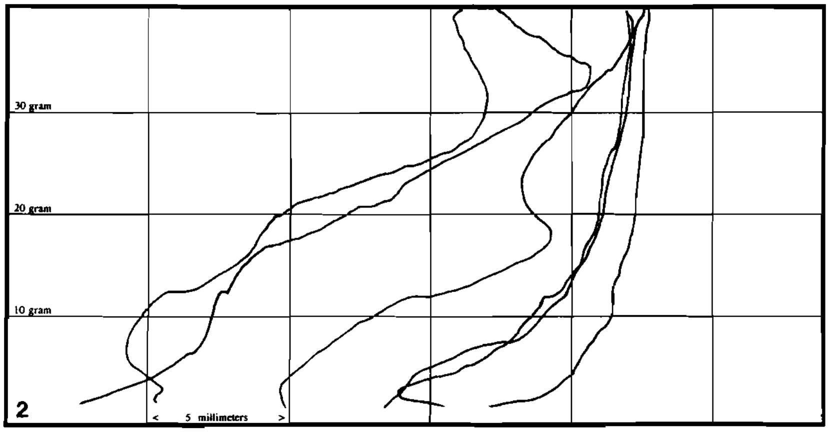


Fig. 2. Right inferior rectus muscle of case V2, who had torticollis since two years and 13° of left hypertropia.

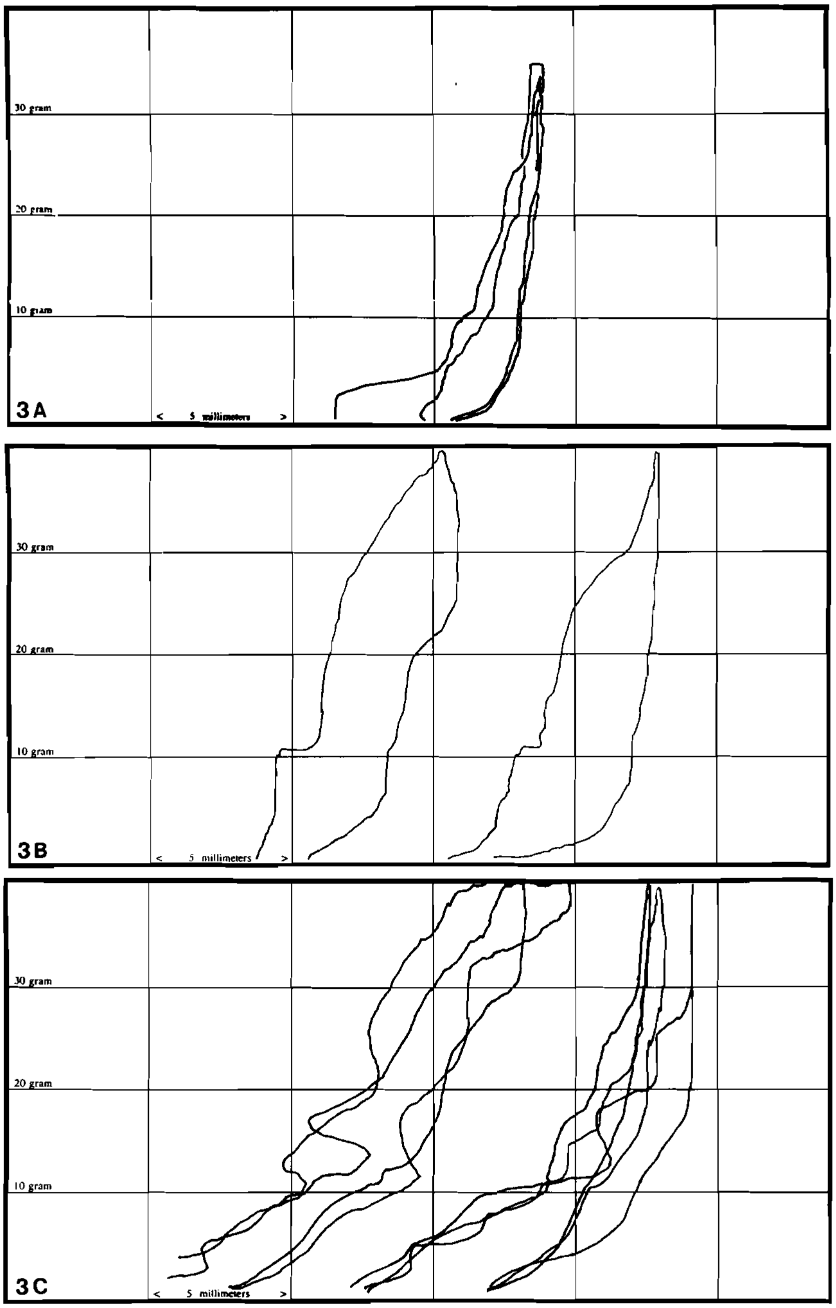


Fig. 3. (A) Left superior oblique muscle of case O6, who had a left superior oblique palsy. (B) Left superior oblique muscle of case O4, with strabismus sursoadductorius of the left eye and myopia. (C) Right superior oblique muscle of case O1, who was consecutively divergent, but also had a downshoot in adduction of the right eye. Force-length recordings of RSO were made while other eye looked into direction adduction-down, abduction-down, abduction, ahead, adduction (in this sequence from left to right in the diagram).

Fig. 4. Left medial rectus of case R4, who was 77 years, had secondary divergent strabismus and had received a total tenotomy of the left medial rectus muscle at age twelve; the eye could abduct only 10°.

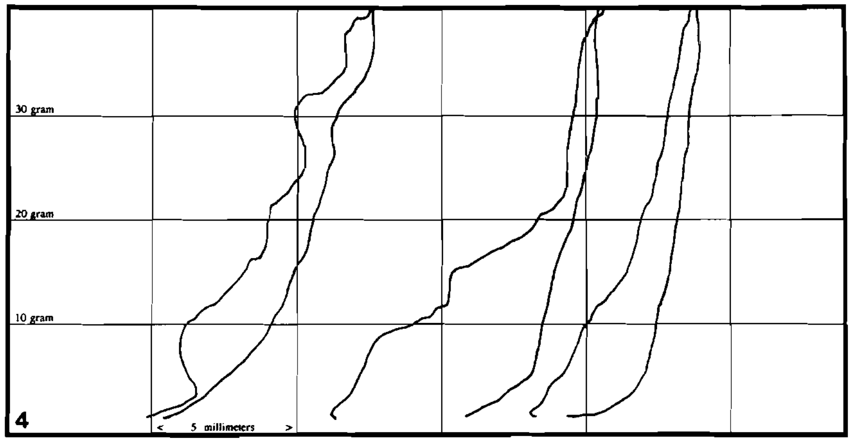


Fig. 5. Right lateral rectus muscle of case P1, who had multiple sclerosis and 14° of esotropia when the other eye looked ahead. Note that the palsy is not complete.

In innervated inferior rectus muscles (Table 2 and Fig. 2), we found a low spring constant of about 2 g/mm in case V2, but about 4 g/mm in case V1. In case V1 strabismus surgery had been carried out on the lateral rectus muscle previously.

Almost all cases in which oblique muscles were recorded (Table 3 and Fig. 3) were diagnosed as strabismus sursoadductorius (= upshoot in adduction) or as superior oblique palsy. Only one case (O1) had strabismus deorsoadductorius (= downshoot in adduction). In that case, force-length recordings were made while the other eye looked ahead, in adduction, in abduction, in adduction-downgaze and in abduction-downgaze. The level of contraction varied exclusively with up- and downgaze, not with ad- or abduction. This may seem amazing because, at first sight, one would assume a higher fire rate for a superior oblique muscle in adduction, where it is more needed for depression. However, the finding that the level of contraction did not vary, in this patient, with ad- or abduction parallels the finding of Henn *et al.*[30] (see also[31]) in monkeys: They even found an even slightly higher fire rate of the superior oblique muscle when the monkey looked into abduction than when the monkey looked into adduction (both without elevation or depression).

In cases O2, O5, O6, O7 and O8 the superior oblique muscle did not shorten when the patient looked down with the other eye, this being indicative of a superior oblique palsy. After elongation, the palsied superior

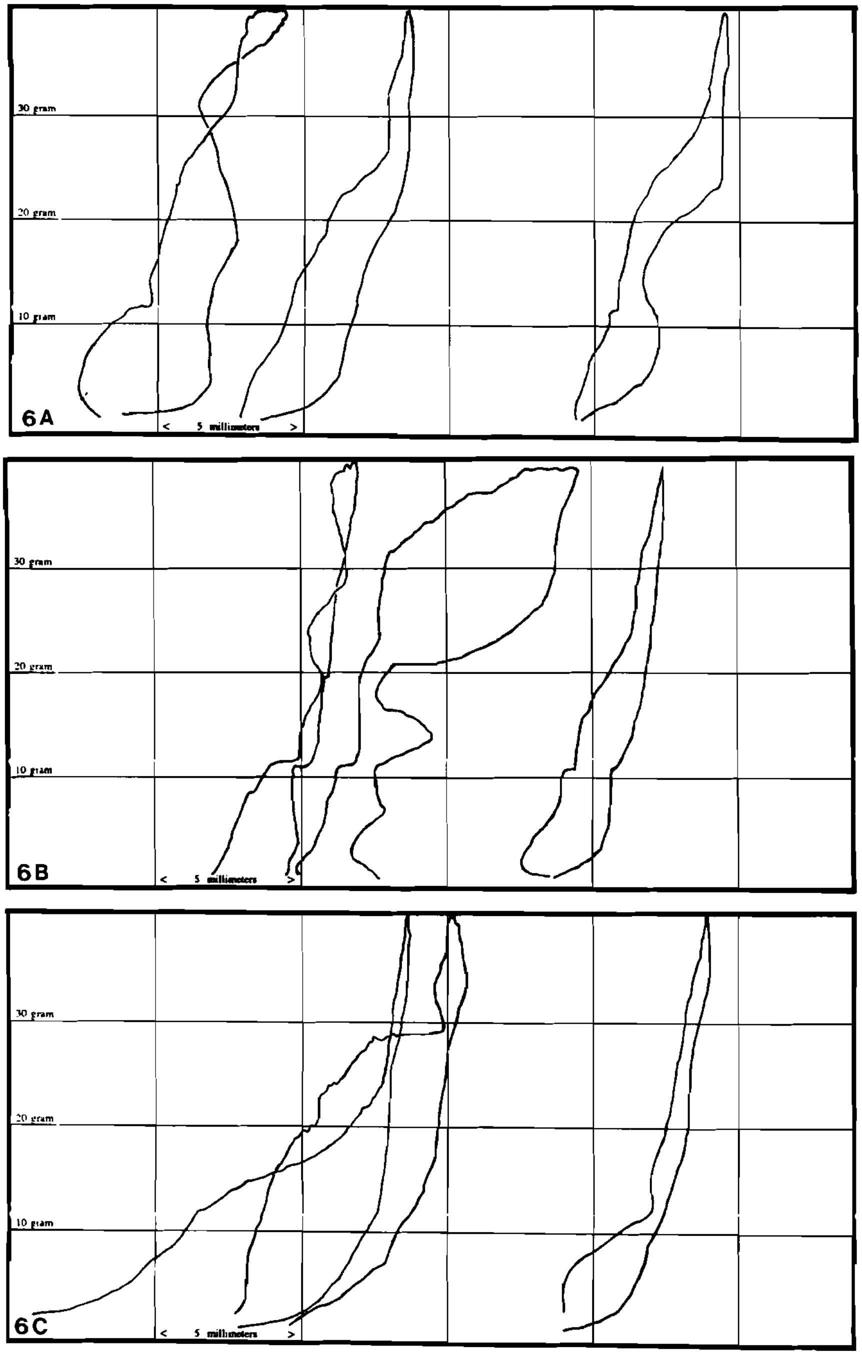
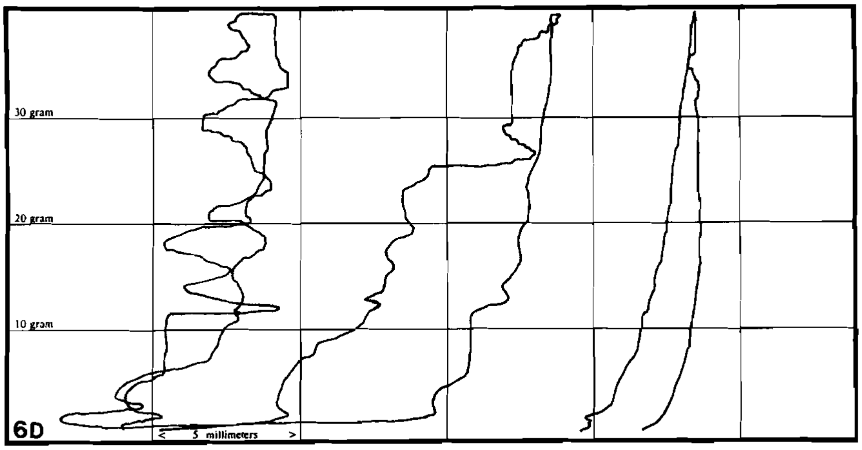
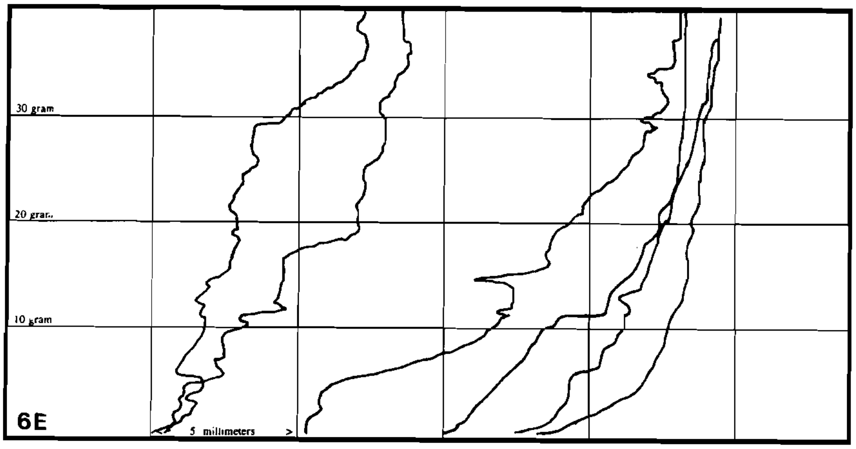


Fig. 6. In seven patients with Graves' disease of recent onset, affected muscles were found to be very stiff when the other eye looked straight ahead. It was expected that these stiff muscles would be able to shorten to some extent but would be unable to lengthen, due to fibrosis of the muscle. We found, however, that the affected muscles lengthened considerably when the other eye looked out of the field of action of the muscle. (A) Inferior rectus muscle of the right eye of case G4, who had diplopia for three years and proptosis for two years; she had 14° of right hypotropia. (B) Inferior and (C) superior rectus of the left eye of case G5, who had been treated with strumectomy one year previously, and had diplopia and extreme proptosis since 6 months; the left eye could not elevate above the primary position. (D) Inferior and (E) superior rectus muscles of case G6, who first noted swelling of the eyelids, diplopia and headache 20 months previously; she had 10° of right hypotropia.
Figs. 6D and 6E, see overleaf.

oblique muscles were very stiff. This could be construed as evidence that the belly of the muscle got stuck in the trochlea at that point but, on the other hand, all muscles with a long-standing palsy are, after elongation, found to be stiff when recording length and tension, like a leash.[7] A normal muscle is elastic, even when not contracting. In case O7, decreased shortening of the inferior oblique muscle was found, with a high spring constant, compatible with the clinical diagnosis 'contracture of the ipsilateral antagonist'.

In case O4 the superior oblique muscle shortened 8 mm at 10 g load, despite a motility that indicated a superior oblique palsy. The patient had a





hypertropia that increased in adduction and on ipsilateral head-tilt (Bielschowsky head-tilt test), torticollis and an excyclotropia. The finding of 8 mm shortening of the superior oblique muscle is incompatible with the diagnosis of a superior oblique palsy. Apparently, some cases of surmised congenital superior oblique palsy that are diagnosed using the criteria mentioned above may in fact be unilateral strabismus sursoadductorius (= upshoot in adduction). These are non-paretic motility disorders that usually accompany a horizontal squint in children, together with V- or A-pattern motility. We have found previously that even in healthy volunteers these motility disorders can be found and made manifest by disruption of fusion by patching.[32] In those experiments, one eye of complaint-free volunteers with full stereopsis was patched for three days. We found that 11 out of 18 volunteers had developed an upshoot-in-adduction (strabismus sursoadductorius) of the patched eye, in five cases no directional change was found, whereas two cases had developed a downshoot-in-adduction (strabismus deorsoadductorius). It seems that strabismus surso- and deorsoadductorius are latent motility disorders that become manifest after disruption of fusion.

What is the reason for this motility disorder? Considering the variability of the anatomy, the physiology and the innervation of the eye muscles[33,34] it seems an unlikely event that both eyes would move exactly in the same direction just by chance. As most normal people have almost perfectly conjugate horizontal eye movements, it seems that some fine-tuning mechanism must be present for this to happen. This fine-tuning mechanism employs binocular vision, and when this is interrupted by patching one eye or by an infantile, horizontal strabismus, the eye movements become disjugate. To what extent they will become skewed is determined by many random variables like the anatomy of the eye, muscles and orbit and the innervational wiring in the brainstem.

It seems amazing that, in unilateral strabismus sursoadductorius, the Bielschowsky head-tilt test can be positive and that torticollis can develop. There are, however, several indications that the change of the vertical angle of strabismus when tilting the head in the Bielschowsky head-tilt test is not only caused by varying otolith input in combination with a superior oblique muscle palsy, as was originally described by Nagel,[35] Baumeister[36] and Hoffmann and Bielschowsky.[37] Certainly, that is the main mechanism, but there is also an adaptive component. Robinson[38] showed in a computer-model study that only secondary overaction of the vertical rectus muscles could explain the very large changes of the vertical angle of strabismus on head tilt, that sometimes occur in combination with a small amplitude of otolith-induced ocular counterrolling of the other eye.[39] In other words, the gain of the otolith-reflex input to the vertical rectus muscles can selectively be turned up or down in the met- or mesencephalon. How this works has not been clarified but, whatever its mechanism, it is certainly advantageous for a patient with a small vertical strabismus, because it allows the patient to have single binocular vision with the least possible head tilt.[40] Apparently, this adaptive component may occur in strabismus sursoadductorius without the main component that is directly caused by a palsy of the superior oblique muscle.

Muscles that had been previously operated upon had somewhat higher spring constants (Table 4 and Fig. 4). Previously totally tenotomized muscles were stiff but they could still shorten considerably. This indicates that, over the years, they had not been fully mechanically separated from the globe, otherwise a full contracture would have developed. Probably, they were still hanging on parts of the muscle sheath or Tenon's capsule.

In palsies the level of a palsy could be assessed: of a lateral rectus in case P1 who had an abducens palsy, and of a medial rectus in case P3 who had an internuclear ophthalmoplegia (Table 5 and Fig. 5).

In Graves' disease (Table 6 and Fig. 6), we expected to find a stiff muscle when the other eye looked ahead, that the muscle would not be able to lengthen due to fibrosis, but could shorten to some extent. What we found, in recent-onset Graves' disease, was the reverse: Affected muscles were very stiff when the other eye looked ahead, showed little additional shortening when the other eye looked into the field of action of the muscle, but lengthened considerably when the other eye looked out of its field of action. The level of contraction of the affected muscle was high when the other eye looked ahead and it seems that the increased muscle force and reduced elasticity, and also the strabismus primarily were caused by active muscle contraction and not by fibrosis. This is not meant to imply that fibrosis plays no major role in Graves' disease. On the contrary, fibrosis of the affected muscles is the end-stage of the disease. But in the initial stage of the disease there is excessive muscle contraction.

In a preliminary report we have presented a hypothesis to account for the high level of contraction.[41] In rats that were made hyperthyroid, Izumo *et al.*[42] found a transition of slower into faster muscle fiber types. The expression of the type of myosin heavy-chain is influenced by thyroid hormones. The thought of a transition of slow muscle fibers into fast fibers is particularly provoking in the case of eye muscles, because eye muscles have several kinds of fibers that do not occur elsewhere in the body. They have at least six,[43,44] but probably even more[45] types of muscle fibers. Two of these, one in the orbital and one in the global layer of the muscle, are multiply innervated and probably do not even contract with normal action potentials.[46,47] Possibly, a transition of one of these or another fiber type particular to eye muscles, might lead to a more forceful contraction, innervation staying the same. A transition of slow into faster muscle fiber types would also explain (1) increased saccadic peak velocity in Graves' disease of recent onset,[48] (2) fast eyelid saccades (Sibony and Evenger, personal communication, September 11th, 1990), (3) fatigability of leg muscles, in climbing stairs, for instance, (4) a positive response to botulinum toxin only in recent-onset cases of Graves' disease,[49] and (5) increase of the diameter of muscle fibers of the levator muscle.[50] Our hypothesis was rejected out of hand by Burde[51] and it is true that our findings must be interpreted with great care because several phenomena remain unexplained. For instance, eye muscles may thicken before the onset of hyperthyroidism. Despite these reservations, the actual finding that affected muscles in recent-onset Graves' disease contract excessively when the other eye looks ahead remains.

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