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A trilogy of the oculomotor system
Part II – active, passive, and dissipative forces
Part III – diagnostic tests

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ABSTRACT | In Part II, this paper addresses ocular motions, their causes (forces), and the governing laws, beginning with the fundamental question: *Why do the eyes move*? Ocular rotations and different types of translations (ocular, orbital, and corporeal) are reviewed. The discussion then turns to *how* the eyes move, where concepts such as the plane of muscular action, torque, and arc of contact provide possible explanations for the anatomical arrangement of the ocular muscles within the orbit. Sherrington's law of reciprocal innervation is used to explain the distribution of muscular *active forces* in a conservative mechanical system, but in combination with Hering's law, it may *prevent* eye rotation (e.g., isometric contractions of antagonist muscles of an asymmetrical convergence). Normally, however, the *limitation* of an eye rotation is determined by *passive* forces, evoked by muscular activity itself, particularly natural muscular elasticity. Thus, *elongation* of an antagonist muscle may *passively* restrict the active contraction of an agonist. In addition to mechanisms for initiating rotation (*active* forces) and stopping it (*passive* forces), the oculomotor system also requires a means of dissipating energy (*dissipative* forces) to initiate subsequent movements. Hence, it cannot function as a perfectly conservative system of forces. The paper concludes with a review of "selective" effects of muscle function (due to the sparse distribution of fibers), the role of intermuscular membranes (and pulleys), and mechanical considerations of surgical procedures, such as muscular transpositions to alter or abolish actions (e.g., bifid reinsertions). Part III will address the diagnostic complexities of the oculomotor system, general treatment principles, and ocular fixation (eye and head positions). Although the basic concept of the primary position is relativized, the absolute need for referential conditions in defining, qualifying, and measuring strabismus is emphasized. The prim-diopter is challenged due to its lack of "linearity" relative to angular units, and an alternative is proposed. Methods of examining oculomotor disturbances are outlined, including monocular rotations (ductions), and tests to differentiate between muscular deficiencies and opposing forces. Techniques for identifying the site of a rotational restriction are described, followed by approaches to measuring ocular deviations in diagnostic positions. The concepts of muscular overactions and underactions are analyzed before introducing the concept of diagnostic muscle pairs. Classical knowledge about deviations caused by deficient or restricted muscle actions reinforces the theory of distribution of rotational ocular muscles by diagnostic pairs. For vertical deviations, "underactive" muscle pairs must be separately matched (e.g., RSR with LIR, RIO with LSO). Since vertical recti exert stronger vertical actions than oblique muscles, head tilts are recommended to enhance stress on both pairs, mainly by additional stimulation of oblique muscles. Classical diagnostic directions then align with the objective horizontal plane. The article concludes with peropertative oculomotor testing and a broad protocol for evaluating oculomotor imbalance.

Keywords: (Part II): Ocular rotations; Ocular translations, Contractions, Relaxations, Plane of muscular action; Torque; Lever arm; Sherrington's law; Hering's law; Asymmetrical convergence; Isotonic contraction; Isometric contraction; Isotonic relaxation; Isometric relaxation; Active forces; Passive forces; Dissipative forces; Selective muscular functions; Pulleys; Surgical procedures; Muscular transpositions; Bifid transpositions

(Part III): Extraocular muscles; Muscular elasticity; Rotational restrictions; Ocular fixation; Measurement of deviations; Prism-diopter; Unit of measurement; Subjective measurements; Ductions; Muscular overactions; Muscular underactions; Diagnostic positions; Head tilt; Spring-back rotations

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Part II: active, passive, and dissipative forces

A brief revision of the concept of motion and its cause

Motion (or ***movement***) is the concept by which an observer registers a *change in the position* of a body (i.e., the displacement of *matter*) in *space*. Motion takes *time*; hence, it is now generally defined as a *continuous change in the position* of matter *in space–time*. It is one of our most primitive acquired notions and, arguably, one of the most representative landmarks of how we understand and expand our knowledge of the Universe.

For centuries, the Sun was regarded – beyond of any doubt – as moving in the heavens, while the Earth was considered perfectly static. Later, these conceptions were inverted, and today we accept that both the Sun and Earth are in motion. What changed was not their actual *motion*, but rather the *reference point* (in space and-or time) from which their movement was considered.

For example, inside a moving car, if a smartphone is released, the observer sees it falls in a vertical path. To the smartphone itself, the descent is registered as static (the registered movements are of surronding objects), while to an outside observer it follows a parabolic trajectory. These three different descriptions of the same phenomenon highlight that motion is always *relative to the frame of reference* (or "point of view") from which it is observed.

The properties of motion are studied in ***kinematics***, which deals with *velocity* (the relationship between the *amplitude of displacement* in space and the *time* spent) and *direction* (the succession of positions that define a *trajectory*). The cause of motion is a ***force***, a physical entity that not only *initiates movement* (providing velocity and direction) but also *modifies it* by increasing velocity (*acceleration*), decreasing it (*deceleration*), *preventing* it, or *stopping* it. In other words, the existence (or absence) of motion, as well as its variations (in amplitude, speed, or direction), depends on *forces* acting on the body.

These principles form the foundation of ***Newtonian mechanics***, which applies to ordinary bodies but not to very small particles (molecules, atoms, subatomic entities). Mechanics has two main branches, ***statics***, which refers to objects "at rest" (equilibrium of forces), and ***dynamics***, which refers to moving objects.

Matter resists being moved; it has a property – "***inertia***"– that opposes applied force. The amount of inertia is determined by the body's ***mass*** (its quantity of matter). A body's resistance to being lifted, or the force it exerts on a supporting surface, corresponds to its ***weight***, which counteracts the Earth's gravitational pull. Similarly, when a force is applied horizontally, ***friction*** arises between the body's surface and that of the support. These cases illustrate Newton's ***Third Law of Motion***: Every *action* force is opposed by an equal and opposite *reaction* force.

The ***First Law of Motion*** (law of *inertia*), first formulated by Galileo, and later generalized by Descartes, states that if no force acts on a body, its state of motion or rest will not change (*[1]). Thus, a body in uniform motion will maintain the same velocity unless acted upon a force. There is no essential difference between a body at rest (zero velocity) and one moving with constant velocity.

The ***Second Newtonian law*** states that a *force* causes a *variation in velocity* that is, an increase (*acceleration*) or decrease (*deceleration*) in a body's velocity. Such a variation (a) is directly proportional to the applied force (F) and inversely proportional to the resistance (inertia) offered by the body's mass (m, a measure of inertia), expressed as $a = F/m$.

In the case of rotational motion, instead of "linear acceleration" (a) and "linear (or tangential) velocity" (v_T), one uses "angular acceleration" (a_a) and "angular velocity" (ω). These are related as $(a/a_a) = (v_T/\omega) = d$ (*[2]), where d is the "lever arm", or the distance between the point where the force is applied and the body's center of rotation (center of mass, the point where the entire mass of the body is considered to be concentrated).

How the eyes move

The first paper of this trilogy began with the question of *why* the eyes move: Without ocular displacement the visual field would be restricted to a small, fixed frame of space. The present paper addresses *how* the eyes move.

(*[1]) Actually, the first law is expressed as "A body remains at rest, or in motion at a constant speed in a *straight line*, except insofar as it is acted upon by a force". But for a body in an *uniform circular motion* (i.e., with the same speed, – with no acceleration, – with no other force added), although it obeys the law of conservation of energy (the "force" is kept constant), the speed is constant, but the direction is not straight. It changes by the action of another ("centripetal") force.

(*[2]) Such ratio follows the relationship between the linear (curved) length of a circumference ($2\Pi r$) and its respective angle in radians (2Π) that is $(arc's\ length/radians) = (2\Pi r/2\Pi) = r = (S/\Theta)$. So that $(S/t)/(\Theta/t) = (v_T/\omega) = r$, and, or $(S/t^2)/(\Theta/t^2) = (a/a_a) = r$.

There are two basic types of eye movements, both caused by *forces*, which may be classified as *internal* (belonging to the oculomotor system) or *external*. The first type – eye *rotations* – involves movement relative to a "fixed" reference frame (the orbit) and is of special interest to ophthalmologists. ***Ocular rotations*** are caused by the six *ocular* ("extraocular") muscles. (Although the term "extraocular" is widely used, it literally means "outside the eye", which technically applies to all body's muscles – except the "intraocular" group. Thus, the term should be used cautiously when referring specifically to *ocular* muscles.) However, because their amplitudes are limited, full exploration of visual space also requires the second type of ocular movement, ***ocular translations***, in which the eye as a whole moves relative to another reference frame.

Internal forces (those of the rotational ocular muscles) may also cause *ocular translations*, but these are usually so small that they can be neglected for ophthalmological considerations. *Ocular translations* (caused by external forces acting on the oculomotor system) are, essentially, *displacements of the orbits* that is, of the *head* relative to the body, and/or of the *body* relative to an external frame of reference in space. Such displacements may occur in any spatial plane (commonly described as *horizontal*, *sagittal*, or *frontal*), either as a *rotation* around an axis (perpendicular to the plane) or as a *translation* along it.

For example, *orbital (head) rotations* relative to the body, produced by neck muscles, occur around the vertical axis (perpendicularly to the horizontal plane) when the head turns right or left(*[3]); around the transverse axis (perpendicularly to the sagittal plane), when the head rotates up or down(*[4]); and around the longitudinal axis (perpendicularly to the frontal plane) when the head tilts toward the right or left shoulder. Proper *orbital (head) translations* (orbital displacements along the trunk's axes) are also possible, but they have small amplitudes because the head is relatively "fixed" to the neck.

Although *head rotations* may reach large amplitudes, they are nevertheless limited. Additional *ocular translations* may result from *orbital* (head) *translations* produced by *body displacements* relative to the ground, either by rotation or translation. *Trunk rotations* may occur around the vertical axis (turning to the right or left), around the horizontal (or transverse) axis (bending forward or backward), or around an axis perpendicular to the frontal plane (inclining to right or left). *Body translations* (e.g., walking forward or backward, shifting sideways, climbing or sitting) complete the possibilities. Thus, *body* movements (rotations and translations) combined with *orbital* (head) rotations and translations, together with intrinsic *ocular* rotations, allow visual access to any region of the "visible" space.

Common movements of the body also depend on muscular forces, so it is not entirely incorrect to state that contraction of the triceps surae muscle (the gastrocnemius and soleus, responsible for plantar flexion) can influence vision (e.g., enabling the eyes to be elevated to look over a wall). However, the body may also be displaced in space by external means such as a car, a boat, or elevator. In such cases, ocular translations may occur independently of *muscular* forces.

Muscular forces: Causes and consequences

A muscle is essentially an "elastic" structure that can change its dimensions – usually its length – through two complementary components: "*active*" units (sarcomeres) and "*passive*" yet flexible (extensible) tissues. Sarcomeres respond to neural stimuli ("commands") by *altering their length*, a process made possible by both, their proper elasticity and that of the surrounding tissues. When the sarcomere *shortens* (thus *pulling* adjacent structures), the process is called ***contraction***. Conversely, when neural stimuli induce *lengthening* of the sarcomere, allowing it to return to its original state or *push* adjacent structures, the process is called ***relaxation***.

A contraction (or its counterpart, relaxation) is defined as the activation of ***tension***(*[5]) that is, an increase in force. If the developed force is opposed by another of equal magnitude, the muscle length does not change; these are termed ***isometric*** contractions (or

(*[3]) Confusing nomenclatures for the body's axes are sometimes proposed[1] so that the vertical or "longitudinal" axis is considered perpendicular to the "transverse" (horizontal) plane; the "frontal" axis, which passes horizontally from side to side and is perpendicular to the sagittal plane; and "sagittal" axis, which passes horizontally from front to back and is perpendicular to the frontal plane.

(*[4]) Though "head (compensatory of eye deviations) positions" are very commonly found in clinical practice, they call forth a subtle clinical question about the real value of the "true" primary position of gaze, taken as the standard mark of a normal ocular position. In fact, no one would think to classify as "abnormal"–at least from an ophthalmological point of view–someone looking to another with a slightly elevated head position, socially acknowledged as denoting pride, loftiness, and nobleness.

(*[5]) In Physics, the term *tension* (or *traction*) means the force related to pull, or stretch a body, oppositely to *compression*, a force related to pushing against it. Hence, the acknowledged use of *length-tension curves*, of so many papers relating rotations (muscular "lengths") to applied "tensions.".

relaxations). If the applied force results in a *change in muscle length*, the process is ***isotonic***. Unless otherwise specified, the terms "contraction" and "relaxation" in this paper refer to "isotonic" activity that is, variation in muscle length.

From a mechanical perspective, the action of a muscle (*contraction* or *relaxation*) involves variations in *muscle length* determined by the distance between its *insertion* (I) on the eye's surface (which moves with eye movements relatively to the orbit) and its *origin* (O), a fixed point outside the eye (immobile during ocular rotations). A net increase in muscular force shortens the muscle length and pulls the scleral surface toward the origin (force directed from I to O), producing rotation. Conversely, when tension decreases (during relaxation) the force is directed from O to I.

Three anatomical points are fundamental for understanding ocular rotation: ***I*** (***ocular insertion***, where force is applied), ***O*** (muscle ***origin***), and ***C*** (the ***center*** around which ***ocular rotations*** occur). While *O* and *C* can be considered fixed relative to orbital reference points (at least in "pure" ocular rotation), *I* moves with eye movement. At any given moment, however, I, O, and C lie in the same plane, termed the ***plane of muscular action***, with a perpendicular "axis" through C around which rotation occurs. The effect of muscular force is distributed across the three spatial planes according to the projection of this "axis" (vector) in each. Because the effect depends on the position of I, the distribution of muscular force may change during ocular rotation (see also, "The relatively large extension of the muscular insertions").

A) Torque: Quantitative effectiveness of a rotatory force

The *magnitude* of ocular rotational movement in each spatial plane – that is, the ***torque*** plane (*T*, also called "*moment of force*" or "*force moment*") – depends on three factors: (1) the net applied force (*F*) determined by the neural command, (2) the angle of action (α), and (3) the distance (*d*, or "*lever arm*") between the point of force application and the center of rotation. This relationship is expressed by the following equation:

$$T = F.d.\sin\alpha \qquad \text{(F.I)}$$

Because each rotational ocular muscle inserts on the scleral surface, the distance to the center of rotation corresponds to the eye's radius of curvature (*r*). Maximal rotational efficiency occurs when the applied force is tangential to the ocular surface, that is, perpendicular to the eye's radius of curvature (α=90°, sin α=1). In this case, *torque* (*T*=*F*.r.*1*) directly reflects the *muscular force* of contraction or relaxation. This *torque* is distributed across the three fundamental spatial axes according to the projections "of the axis"(vector) – perpendicular to the plane of muscular action.

Determining the position of this vector in space – and, consequently, the rotational components of a muscle's contraction in the horizontal (*H*), vertical (*V*), and frontal/torsional (*T*) planes – requires theoretical consideration of (a) the coordinates of the muscle's origin O (x_o, y_o, z_o); (b) those of its insertion, I at the primary gaze (x_i, y_i, z_i); and (c) their information after a rotation defined by components *H*, *V*, and *T* in each [2-4].

In the primary gaze position ($H = V = T = 0°$), the equations for the horizontal, vertical, and torsional components of muscular pull are expressed as follows:

$$H = x_i \cdot y_o - y_i \cdot x_o \qquad \text{(F. II)}$$

$$V = y_i \cdot z_o - z_i \cdot y_o \qquad \text{(F. III)}$$

$$T = z_i \cdot x_o - x_i \cdot z_o \qquad \text{(F. IV)}$$

Results for *H*, *V*, and *T* may be positive – corresponding to adduction, sursumduction, and excycloduction – or negative, corresponding to abduction, deorsumduction, and incycloduction. These outcomes reflect the specific actions of one of the six rotational ocular muscles. In principle, any "positive" or "negative" rotation (*H*, *V*, or *T*) beginning from the primary gaze position, can be maximally obtained by an appropriate positioning of the applied force (Figure 1).

B) The arc of contact

It is not theoretically convenient that the maximal rotational efficiency of a force be limited to a single *point* of tangential application on the scleral surface, but rather that it extends over a larger superficial area. In other words, the muscle must lie over the eye, that is, with an ***arc of contact*** (Figure 2).

Therefore, although the disposition of ocular muscles to produce eye rotations could in theory be entirely free (Figure 1), the requirement of large arcs of contact limits some alternatives which could otherwise be troublesome. For instance, the existence of a muscle (arc of contact) passing by the anterior or posterior parts of the eye makes the muscle paths (from insertions I to their respective origins O) parallel to the x-orbital axis (Figure 1, second and fourth columns from the left)

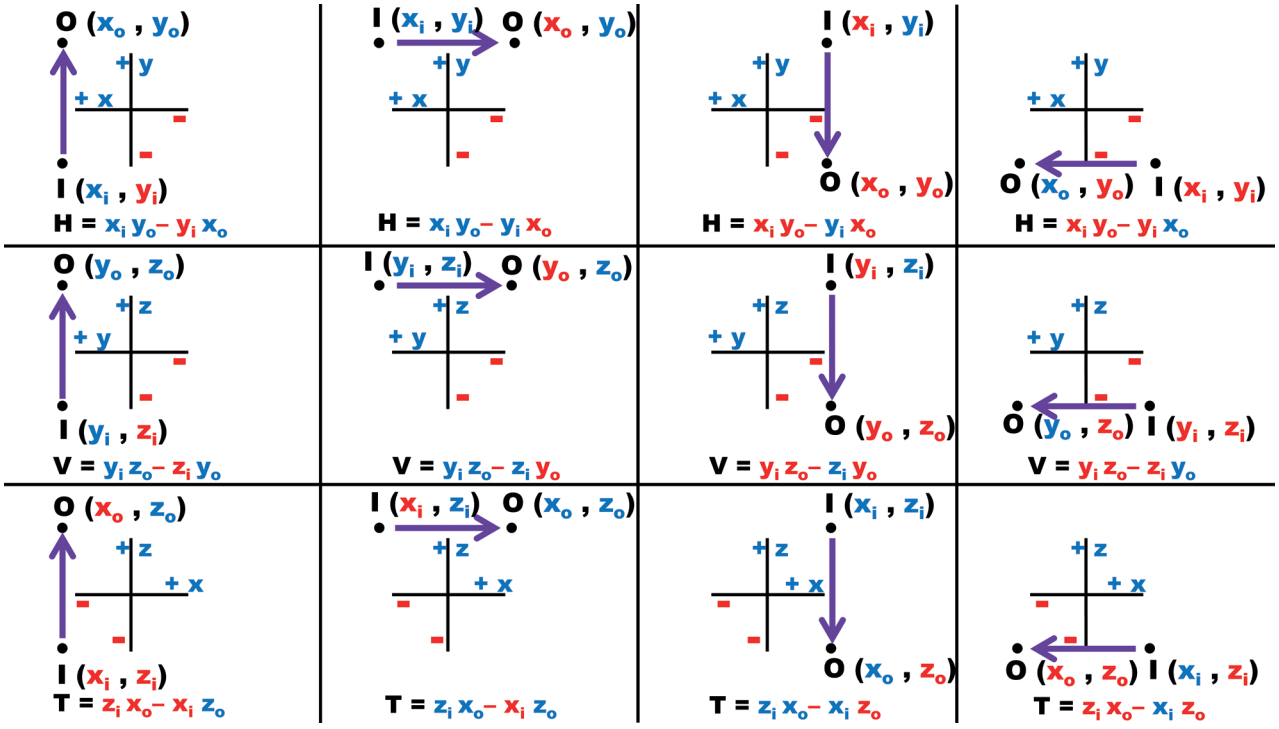

Figure 1. Schematic representations of positive (clockwise) rotations of the left eye, indicated by violet arrows, in the horizontal plane (adduction, top), sagittal plane (sursumduction, middle), and frontal plane (excycloduction, bottom). All possible combinations of ocular insertion (I) and orbital origin (O) satisfy the maximal outcomes of equations (F. II, F. III, or F.IV).

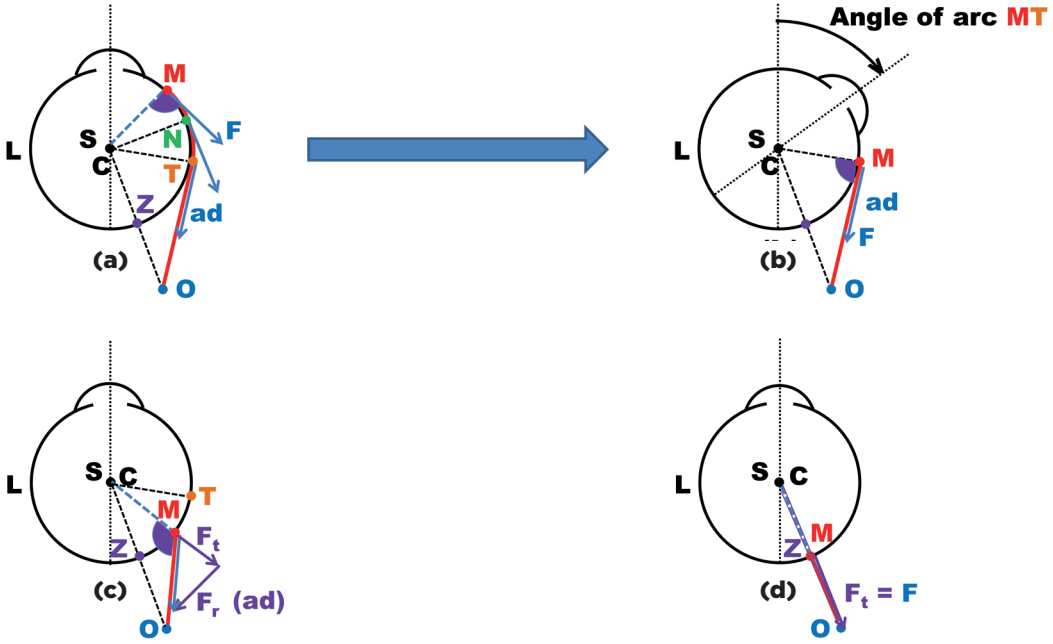

Figure 2. Schematic representation of a section of the left eye (seen from above), containing the plane of action of a muscle (LMR, in red), with the point of its scleral insertion (M), the center of ocular rotations (C), and the origin of the muscle (O). (a) Eye in the primary position: the muscle lies over the ocular surface between points M (*anatomical* insertion) and T (tangential contact to the eye, the *physiological* insertion), forming the so-called "*arc of contact* of the muscle." Tractions from points M, N, T (or any other point between them) always have the same moment of force (angle α=90°), equal to *F.r.* (c) For muscular insertion (M) at points posterior to T, the applied force produces, in addition to a rotational component (F_r<*F*), a translational one (F_t). (d) A force (*F*) applied with muscular insertion (M) coinciding with Z is totally converted into a translational force (F_t=*F*).

which should be avoided. In addition, since the anterior part of the eye must remain free to transmit light, the origins of such muscles should be close to the posterior segment. The actual structure of the human orbit – as the result of phylogenetic evolution – appears to be the natural answer to such needs. In the horizontal plane, a close similarity is observed between the "ideal" (Figure 3a) and the actual (Figure 3b) muscular dispositions.

In fact, the six rotational ocular muscles are arranged in pairs to provide, mainly, actions in each of the three spatial planes. If, by approximation, one assumes for the horizontal recti $z_i=0=z_o$, equation F.II remains unchanged, but Equations F.III and F.IV give $V=0$ and $T=0$. That is, horizontal rotations (H), adduction and abduction, are primarily given by the medial rectus muscle (MR) and the lateral rectus muscle (LR), respectively.

If, also, for the vertical recti, one considers $x_i=0=z_o$, then $H=-y_i \,.\, x_o$, $V=-z_i \,.\, y_o$, and $T=z_i \,.\, x_o$. Considering only the absolute dimension of a measurement (its *modulus*), not its *sign*, since (for the coordinates for a vertical rectus muscle), $y_o > x_o$, then V>T; and as $z_i>y_i$, then $T>H$. Therefore, for the vertical recti, $V>T>H$. By a similar procedure for the oblique muscles (coordinates in Figure 3), $T>V>H$. Thus, the vertical recti and oblique muscles are considered *cyclovertical* muscles, although *vertical* rotations are predominantly produced by the *vertical recti* (*sursumduction* or *elevation* by the superior rectus muscle, SR; *deorsumduction* or *depression* by the inferior rectus muscle, IR), while cycloductions (frontal plane) are mainly produced by the oblique muscles: *excycloduction* (by the inferior oblique muscle, IO) and *incycloduction* (by the superior oblique muscle, SO).

C)The Sherrington's law

The (approximately) parallel forces of each pair of the external ocular muscles act in opposite directions, so that, to maximize rotational efficiency in a specific orientation (clockwise or counterclockwise), they require a special command. Therefore, when a muscle is stimulated for *contraction* (i.e., to initiate a force), its opponent receives a command for *relaxation*. This is the principle of a *conjugated* or *binary* mechanical system, in which the two generated forces act with the *same orientation*. Such a neural relationship, whereby *contraction* of a muscle (the "***agonist***") accompanied by synchronous reciprocal *relaxation* of its pair (the "***antagonist***"), is *Sherrington's law of reciprocal innervation*.

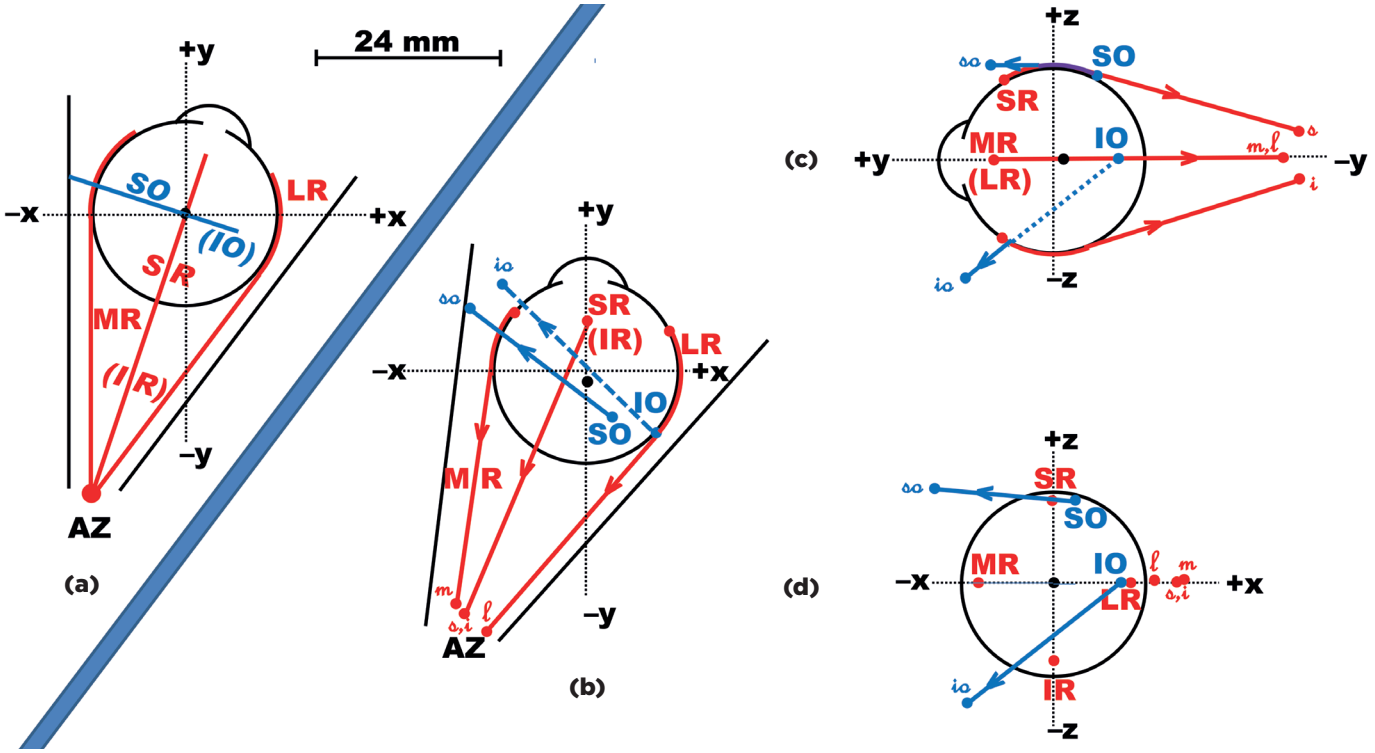

Figure 3. (a) Representation of the horizontal plane of the right eye and orbit (top view) in a theoretical model of the disposition of the ocular recti (horizontal and vertical, in red) and the oblique muscles (in blue). (b), (c), and (d) Actual disposition of the external (or rotational) ocular muscles, according to the classical Volkmann's coordinate values[5] for their insertions and origins (to scale) at the horizontal (top view), sagittal (medial view) and frontal (back view) planes, respectively.

In principle, it might be expected that the variation of forces in a pair of *agonist-antagonist* muscles would be perfectly symmetrical relative to a "median" position (the primary position) but *inverted* – that is, the greater the action of one, the smaller that of the other. In addition, the variation of forces due to *reciprocal* innervation for a desired result (e.g., adduction) and the respective inhibition of the antagonist (relaxation of abduction, therefore allowing more adduction) might be expected to be perfectly linear in the case of a perfectly elastic system. This leads to a graphical and pictorial representation of such forces (Figure 4).

D) Isometric and isotonic contractions and relaxations: Hering's law

Binocularly conjugated eye rotations (***versions***) in each direction are commanded to be symmetrical, according to Hering's law. If the muscles were equal in responding with equivalent forces, their innervation stimuli should also be equal. For muscles with different responses to the same amount of binocular rotation, the stimuli must be "proportional" to guarantee equivalent rotations. In any case, "*fusional*" adjustments of the required rotational amounts are added, if necessary.

For binocular vision directed to points in space requiring *different* amounts of rotation in each eye – or for a "final" fusional correction according to Hering's law – ***disjunctive*** binocular motions (vergences) are provided. Therefore, ***vergences*** and versions may be summed, sometimes resulting in no rotation in one eye and asymmetrical convergence In the other (Figure 5). In the eye without movement, *isometric* contractions ("co-contractions") occur.

The greater the asymmetrical convergence, the greater the increase in stimuli to the horizontal recti of the stationary eye, as confirmed by electromyogram[6].

A similar phenomenon is seen in Duane's syndrome. In type I, affecting the right eye, if levoversion is commanded, the RMR receives a normal stimulus to contract (adduction), while the RLR, instead of relaxing, is also abnormally stimulated to contract. The simultaneous increase of forces in both horizontal recti produces a small posterior translation of the eye (enophthalmos, which involuntarily narrows the palpebral fissure)

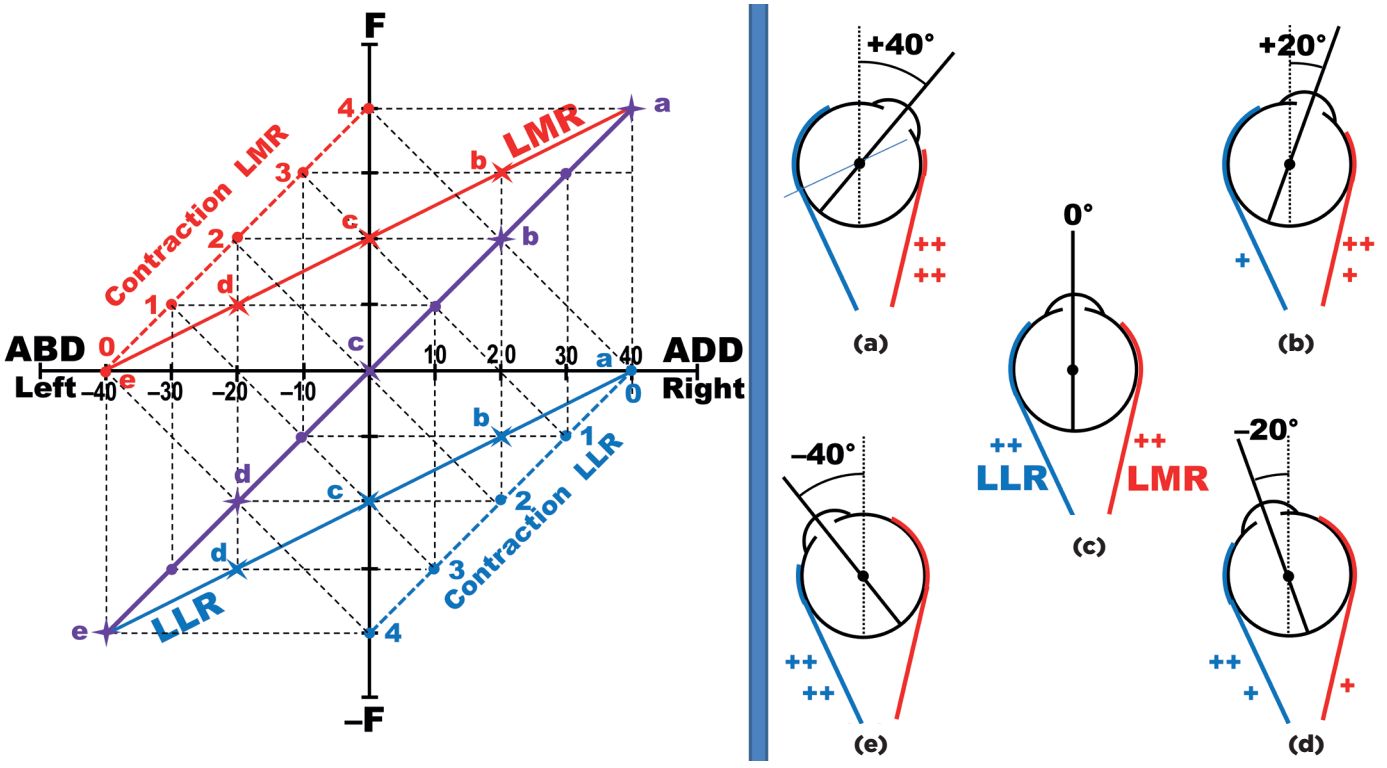

Figure 4. Left: "Active"(generated) forces of the LMR (in red) and LLR (in blue) in ordinates as a function of eye position (abscissas) in adduction (ADD) and abduction (ABD), with the resultant (violet line). Right: pictorial representation of LMR and LLR forces by the quantity of plus signs (+), related to eye positions.

and an *increase* in intraocular pressure[7], as the eye is compressed against orbital contents.

How the eye stops moving

A schematic explanation of how the eye moves – by an *imbalance* of oppositely acting muscular forces of a simple binary system (Figures 4 and 5) – leads to the possibility that, once a rotation is started, it would remain perpetual. On the contrary, however, after a relatively short rotation (maximally around 50°), the eye is braked to a stop. This is not usually due to a new (voluntary or reflex) neural command, though, as seen in asymmetrical convergence and Duane's syndrome, active forces may be stimulated to prevent ocular motion (Figure 5). Of course, *braking* forces could also come from neural commands, but this would appear contradictory to Sherrington's law, or at least suggest a more complex and uneconomical arrangement of stimuli to "go but stop". In fact, what limits ocular movement is a *natural* consequence of the *very* cause that enables *rotation*: the *elasticity* of the muscles and their surrounding structures. Muscular stretching (particularly of the antagonists) absorbs kinetic energy of an ocular rotation and imposes opposing forces, bringing the system back into balance.

The passive forces (of the conservative system)

During an ocular rotation, elastic structures are stretched while others are compressed but both absorb kinetic energy and accumulate it as *potential energy*. Muscular fibers, their surrounding and intermuscular membranes or fascias, conjunctiva, nerves and blood vessels all act as "elastic" structures, but the muscles are the most important.

If the oculomotor system behaved as a perfect (linear) elastic system, *passive* forces (from stretched and compressed tissues) would increase at the same rate as the variation of *active* forces. In a *conservative* system, the *active* forces that initiate rotation are opposed by *passive* forces from elastic tissues; kinetic energy is absorbed, and the rotation stops. Thus, the extent of a rotation is limited by the balance between *passive* and *active* forces (Figure 6).

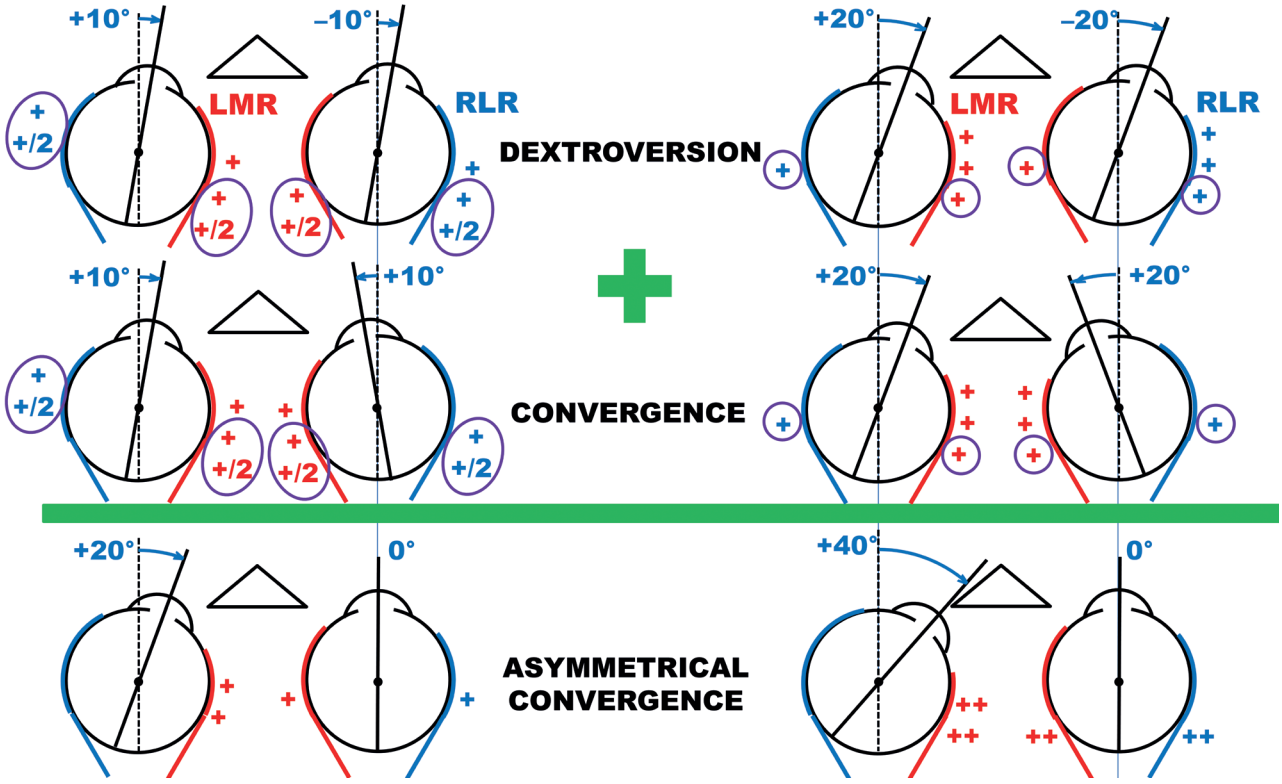

Figure 5. Combinations of dextroversion and convergence of 10° (left) and 20°(right). The activity of the muscles is shown (+), and mutually cancelled forces (violet circles) indicate the resultant variations.

Although periocular structures (including the muscles) could theoretically absorb energy whether shortened (like a compressed spring) or elongated (like a stretched rubber band), they behave "passively" mainly as elastic bands. Experimental data support this: After conjunctival opening (e.g., nasally) or disinsertion of a muscle (e.g., the medial rectus), the eye *deviates to the opposite side* (temporally)[8–10]. If the conjunctiva or muscle had accumulated energy to *push* the eye (like a *compressed spring*), removal of that push should result in adduction. Instead, *abduction occurs*. Therefore, the schematic representation in Figure 4 can be improved by considering the *balance* of forces at the *end* of a rotation (Figure 6) as due to periocular structures – mainly the muscles – acting as *elastic bands*.

Two basic *mechanical* types of eye displacement between two points in space may be considered. One is ***slow***, a smooth *pursuit* movement; the other is ***rapid***, a *saccadic* movement. These are controlled by independent neural systems and have different mechanical explanations. The *smooth pursuit* movement (velocities about 30 – 50°/s) [11] occurs during continuous fixation on a rectilinear target displacement. It depends on a progressive increase in contraction of the agonist muscle and relaxation of its antagonist, automatically accompanied by an increase in opposing passive forces. The *saccadic* movement is produced by a burst of activity in the agonist muscle and complete relaxation of its antagonist. This generates very high velocities (large saccades of about 90° may reach peak velocities near 1000°/s), yet the movement is properly limited by passive forces to the required extent. A similar *saccadic* rotation can also occur if, in a stable eye position (e.g., +40° or –40°) maintained by an external force during general anesthesia, the absorbed energy in stretched tissue (muscles) is released.

The dissipative forces

As shown earlier, although a static eye is necessarily represented by a balance of forces, the state of the muscles – whether *more* activated (contracted) or *less* activated (relaxed) – differs for each position of gaze: The greater the required rotation (e.g., adduction or abduction), the greater the net muscular activity needed. For example, the eye position of +40° (adduction) (Figure 6a) corresponds to strong stimulation of the RMR (++++), while stimulation of the RLR is reduced to 0. The system, however, remains precisely balanced by equal and opposite *passive* forces. As long as this command for activation is maintained,, the eye is held *immobile* at +40°. When the command to the RMR *ceases*, the *balance of forces is lost*. Inactivation (relaxation) thus acts as a reciprocal "activation" that initiates a returning saccadic rotation toward the primary position.

If no other forces acted, the system would continue in perpetual harmonic motion, like a pendulum (Figure 7). On the contrary, after a rapid "return" toward the central position, the rotation *stops* (Figure 8). During *contraction*,

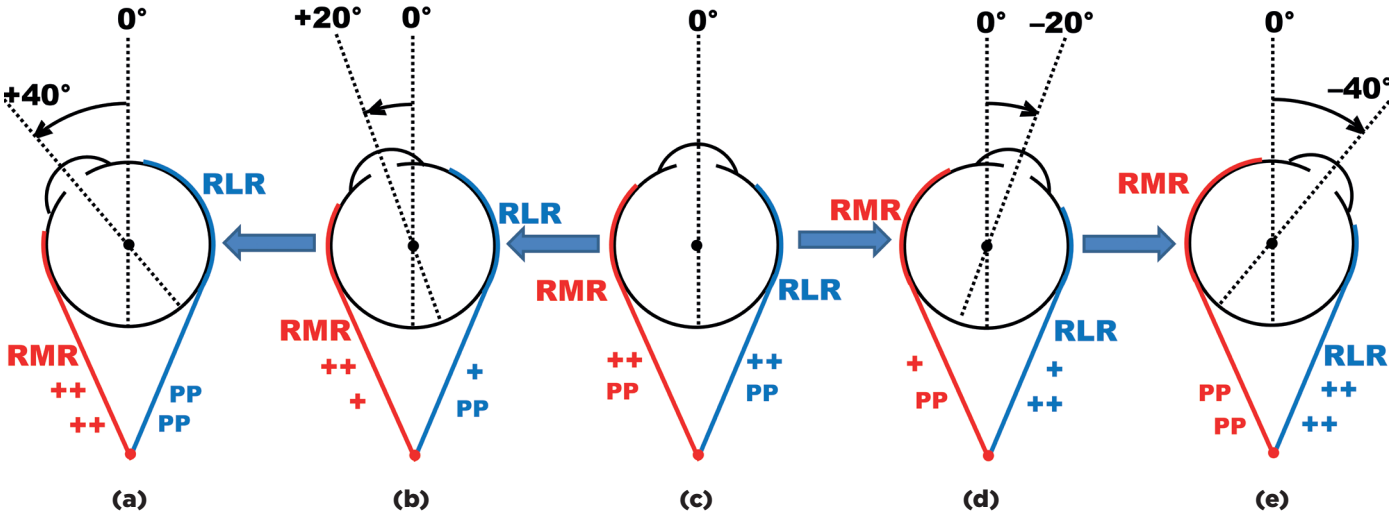

Figure 6. Schematic representation of the horizontal plane of the right eye (top view) in different positions, showing the balance between *active* (commanded) and *passive* (naturally elastic, resistive) forces at the end of ocular rotations. The respective amounts of *active* forces ("activities", "stimuli", "tonicities") and accumulated *passive* forces (absorbed, stored) for the RMR (red) and RLR (blue) are indicated by "+" and "P", respectively.

progressive activation of a muscle, combined with *reciprocal inactivation of its antagonist*, is countered by increasing passive (resistive) forces in stretched tissues (Figure 8a–c). Part of the command energy is converted into motion (kinetic energy), part is stored as potential energy in stretched periocular structures, and the remainder is lost – either dissipated as heat through friction or through inelastic deformation of viscous periocular components (e.g., conjunctiva, soft tissues, or the so-called "check ligaments").

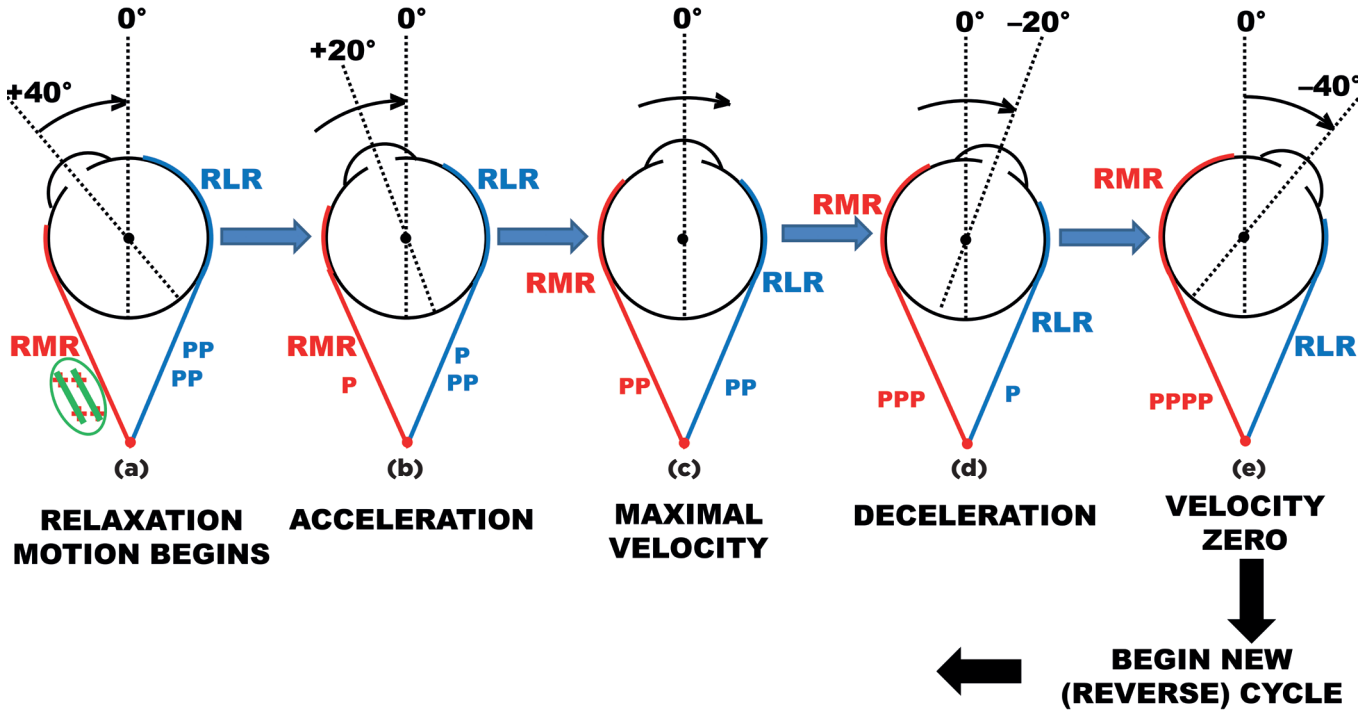

Figure 7. Simplified schematic representation of a theoretical conservative system. After a stable ocular position is reached and the agonist muscle relaxes (a), disappearance of active forces initiates accelerated rotation (b). A maximal velocity is reached (c), followed by deceleration (d), until the rotation stops (e). The accumulated energy in stretched structures (e.g., RMR) then starts a new cycle in the opposite direction.

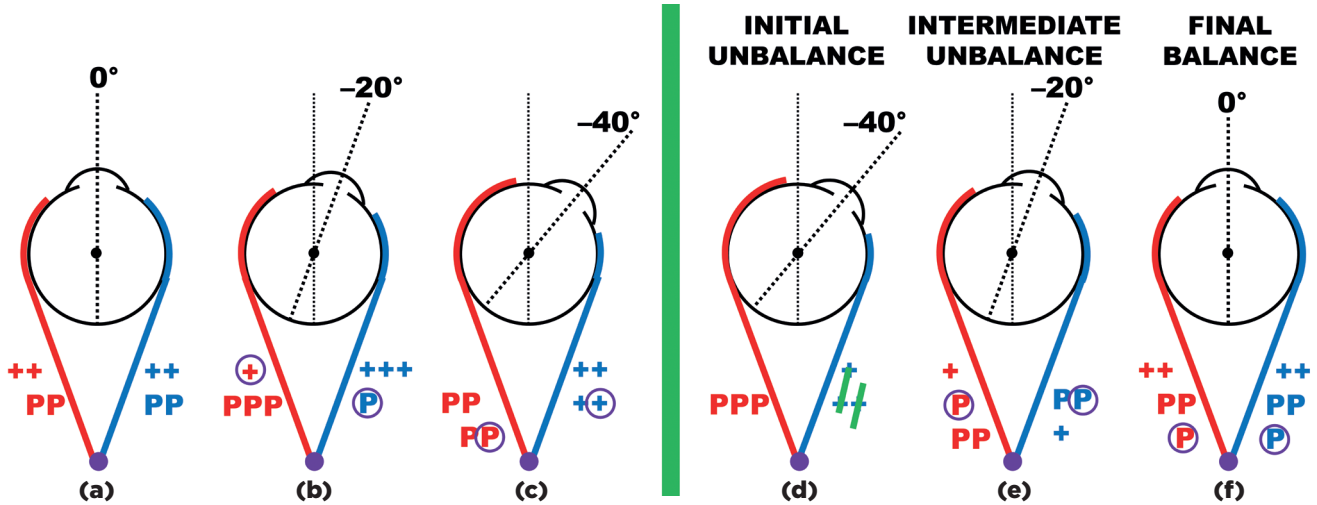

Figure 8. Speculative distribution of forces in the horizontal plane of the right eye (top view): active forces (neural command,"+"), passive – conservative – forces ("P"), and dissipated forces (violet encircled sign, nullifying that force). (a) Balanced primary gaze position; (b) balanced position after abduction –20°; (c) balanced position after abduction –40°; (d) unbalanced position after abduction –40°, when RLR activation (+++) ceases; (e) unbalanced position of abduction –20° (eye moving in "centripetal" adduction); (f) balanced, stable primary gaze position after "returning" adduction. In the spring-back balance test (under general anesthesia), do not consider signs of muscular activation (+): the "centrifugal" rotation is due to exterbally applied, forces; the "centripetal" rotation is due to the stored part of such forces.

If the muscle maintaining a stable position after rotation is relaxed (Figure 8d), the stored potential energy in stretched structures initiates a "returning" rotation. As the rotation progresses, muscular tonicities (+) are gradually restored, passive forces of shortened structures decrease (red P), while those of stretched structures increase (blue P). However, part of the energy is dissipated (Figure 8e), so the cycle ends in a stable position (figure 8f).

Under general anesthesia, when no active forces (+) are present, a similar scheme can be considered (the spring-back balance test, studied later)[8,12]. In both active (neural) and passive (externally induced) conditions, the absorption of received energy has two components: (i) accumulation by elastic structures – the *conservative system* –, and (ii) dissipation through inelastic deformation or frictional heat – loss, the *dissipative system*.

A final schematic view of force distribution during ocular rotation can now be offered. Although the oculomotor system is basically "elastic," it does not behave as a "perfectly" linear (conservative) system. Viscous and inelastic structures *dissipate* part of the received energy. During muscle contraction, a portion of energy is spent as heat or against inelastic resistance; only part results in motion. Consequently, progressively *greater* energy is required for progressive *equal* increments of rotation – for example, the energy to move the eye from 0° to 20° is *less* than that needed from 20° to 40°.The muscle's mechanical behavior is better described by an asymptotic curve (Figures 9 and 10)[13].

The relatively large extension of the muscular insertions

A muscle is composed of numerous fibers that extend over the scleral surface in a line of insertion measuring approximately 9–11 mm. The imaginary *frontal* circle along which the insertions of the recti muscles are distributed has a diameter of about 19.5 mm (broken blue line, Figure 11), corresponding to a circumference of about 61.3 mm. Therefore (4 x 10)/61.3 ≈ 65% of this circle is covered by the recti muscles insertions, a relatively large extension.

Although the *action of an entire muscle* can be simply defined as that of a *single fiber pulling* from the *median point* of the extended insertion, each of the multiple fibers produces *distinct* and specific torques. These

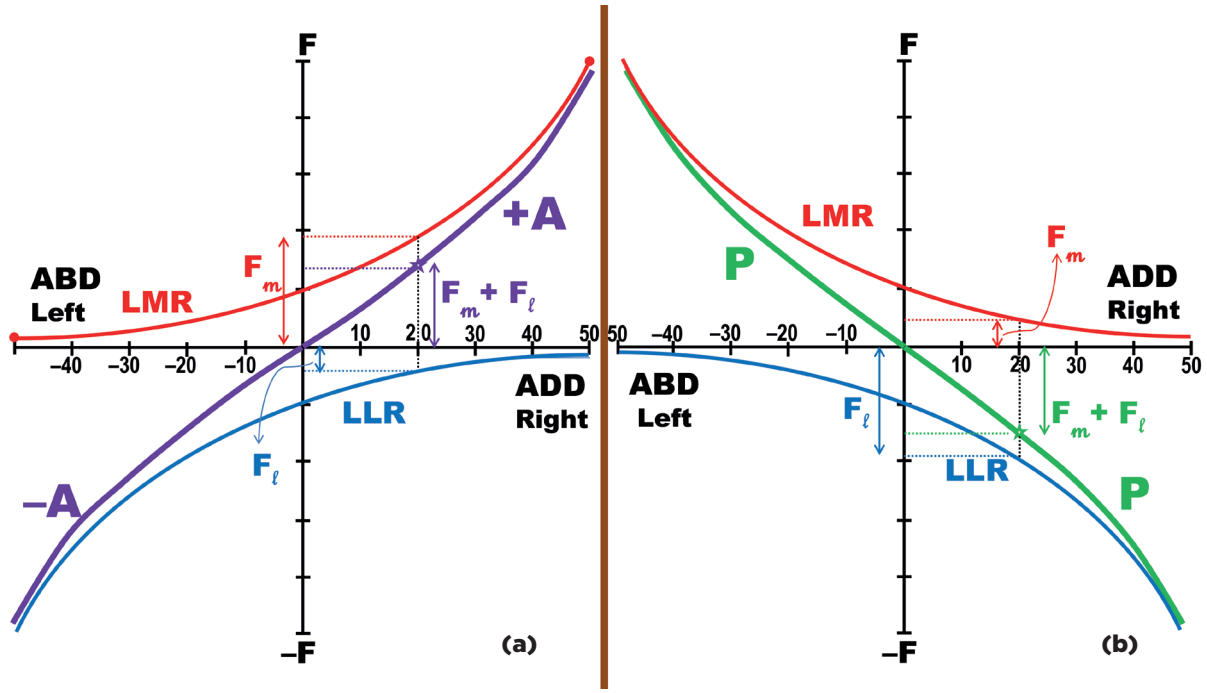

Figure 9. Graphical representation of forces of the horizontal recti muscles of the left eye (ordinates) as a function of horizontal eye position (abscissas).(a) Forces generated by neural activation; (b) Forces of passive elastic resistance. Resultant "S shaped" curves – violet and green – are symmetrical. For active forces (a) an adduction of 20° results from the opposite tonicities of MR (F_m) and LR (F_l), represented by the violet star on the violet curve. For the same amplitude, an equal and opposite passive force develops (b), shown by the green star on the green curve.

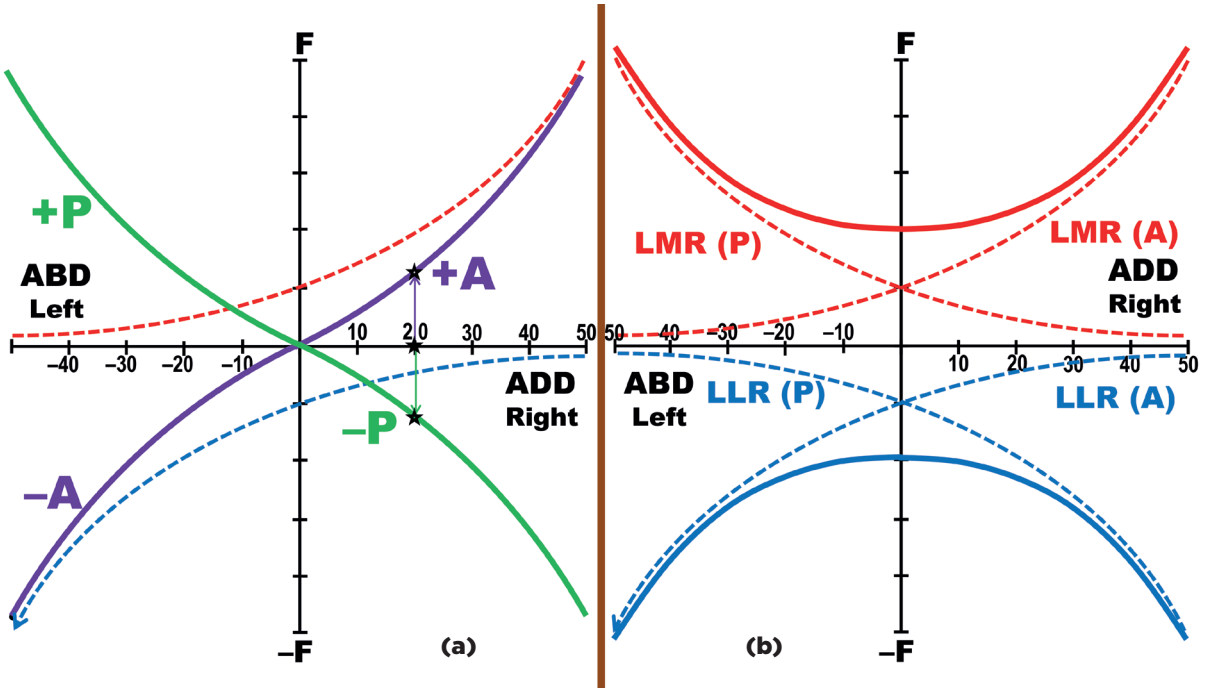

Figure 10. (a) Resultant curves of active forces (violet) and passive forces (green) of the horizontal recti. Normally, their sum balances along the abscissa axis. (b) Separate curves of active (red) and passive (blue) forces for each muscle (dashed lines). The resultant sums (solid red and blue lines), form "U shaped" curves, instead of being parallel to the abscissa, as would be expected in a perfectly conservative system.

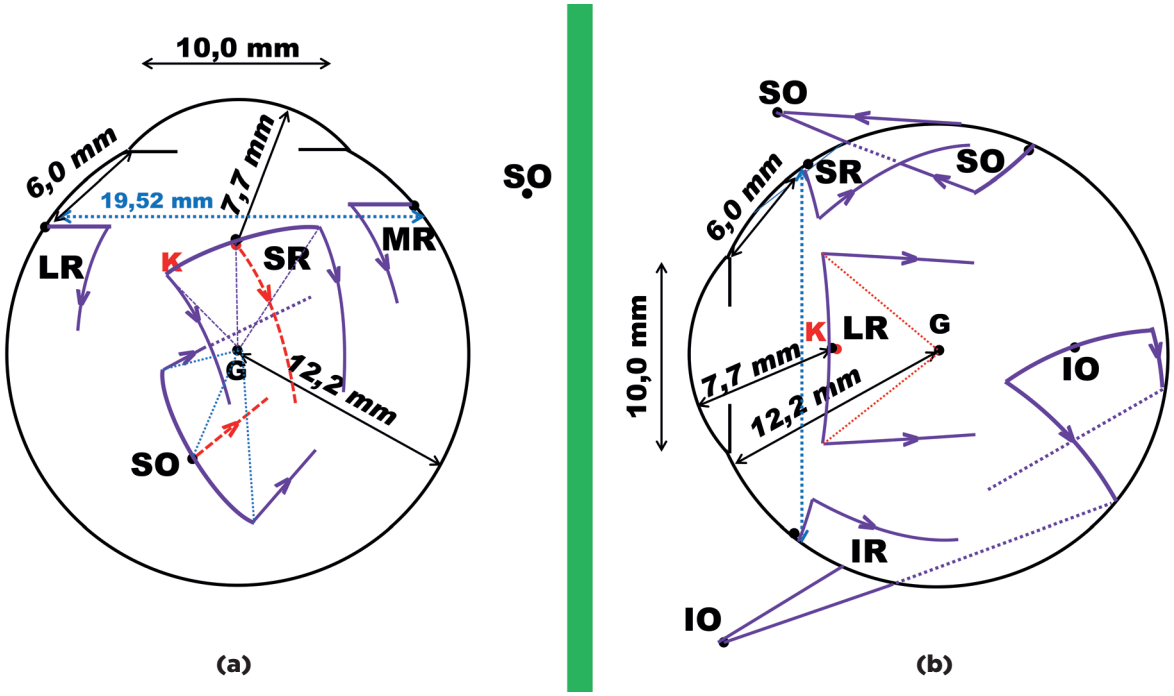

Figure 11. Schematic views of the left eye from above (a) and from the lateral side (b), showing insertions of the medial rectus (MR), lateral rectus (LR), superior rectus (SR), inferior rectus (IR), superior oblique (SO), and inferior oblique (IO) muscles. Projections of the geometric center of the eye (G) and of corneal curvature (K, practically coincident with the projections of he points of the median fibers of the SR and LR) are shown. The radii of curvature of the eye (12.2 mm) an of anterior corneal surface (7.7 mm) are also indicated.

may, in part, oppose the action of the remaining fibers. Consequently, the same muscle may exhibit rotational effects of different qualities and/or magnitudes. For instance, in the horizontal plane, the SR and SO muscles are defined as having the properties of adduction and abduction, respectively (functions attributed to the fibers of their median insertions). These actions are particularly prominent in the *medial* fibers of the SR and the *posterior* fibers of the SO (Figure 11a). In contrast, the *lateral* fibers of the SR (and of the IR) and the *anterior* fibers of the SO muscle are expected to have no horizontal action – or, if present, to contribute to *abduction* and *adduction*, respectively. In other words, lateral and medial fibers of the superior rectus, as well as anterior and posterior fibers of the superior oblique have *opposite* actions

The anterior halves of the SO mainly produce incyclotorsion (and ocular depression). Thus, sectioning these fibers, while weakening the muscle overall, can indirectly and relatively favor *abduction* by liberating the action of the posterior fibers. Selective sectioning of superior fibers of the horizontal recti (MR and LR) favors *depression*, whereas selective sectioning of their inferior fibers favors *elevation*.

Intermuscular membranes - Pulleys[14]

If, during ocular rotations, the rotational ocular muscles were completely free to slide over the eye, they would follow the shortest path between their scleral insertions and their origins (Figure 12b). However, they are enclosed by membranes (fasciae) that interconnect to form a continuous covering. As a result, the pulling directions of a muscle contraction remain almost constant regardless of eye position, as though the muscles passed through *pulleys*. This maintain the same plane of action as in the primary position of gaze (Equations F.II–IV). Therefore, even a *simple section of the intermuscular membranes can alter a muscle's action*. For the same reason, a recession may also modify the expected muscle action. For example, after sectioning the intermuscular membranes, a horizontal muscle may act as an elevator during elevation (Figure 12b); following a very large recession the same muscle may become a depressor (Figure 12c).

Changing actions of external ocular muscles

The majority of medical procedures intended to modify the actions of the rotational ocular muscles are surgical.

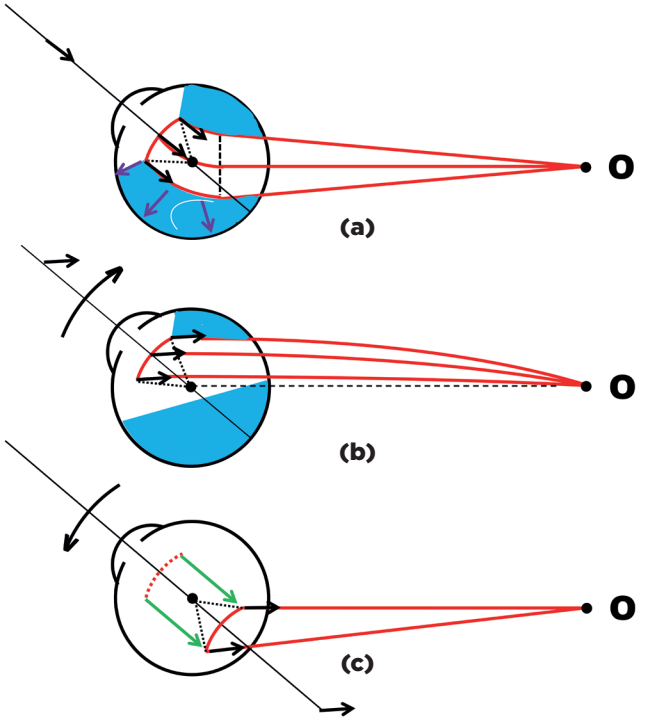

Figure 12. (a) Schematic representation of intermuscular membranes (blue) and the position of a horizontal muscle over the eye. Regardless of ocular rotation, intact intermuscular membranes keep the "physiologic" insertion points (defining the respective planes of muscular action) practically unchanged relative to a fixed reference frame. They maintain the pulling direction of muscle (black arrows, parallel to the axis of visual fixation) and resist vertical rotation (violet arrows). (b) Sectioning the intermuscular membranes allows the muscle to slide over the ocular surface, following the shortest path from its origin (O) to its insertion, thereby altering its pull (now assisting elevation). (c) A simple but large recession (without transposition) may change the muscle's action during rotation (now producing depression).

These are classically divided into two broad groups. The first, ***quantitative***, aims to increase ("strengthen") or decrease ("weaken") the overall action of a muscle without altering the distribution of muscular forces across spatial planes. *Strengthening procedures* include resections, plications (shortening), and advances of the insertion (stretching). *Weakening procedures* include recessions (elongation and slackening), partial or total sections of muscle or tendon (myotomy or tenotomy), with or without excision (myectomy or tenectomy). These may also be "compensatorily" combined such as a "resection" with an equivalent "recession" (Figure 13, the "fadenoperation").

The second group, ***qualitative***, aims to change the spatial distribution of muscular action (e.g., *selective* section of muscle fibers, *transpositions*). These procedures decrease action in one (or two) plane(s) while increasing it in the others[15].

Together, these techniques represent both the *science* (*when*, *which* muscles, and *how much* to operate) and the *art* (the manner of *execution*) of surgical treatment or strabismus and related disorders (Figure 13).

Muscular transpositions

The distribution of a muscle's rotational effect across the spatial planes changes when its plane of action is transposed. This principle is widely applied in surgical treatment of oculomotor disturbances.

It is well established that if the MR muscle is reinserted *below* the horizontal plane, it regains its original horizontal disposition and better adductive effect when the eyes are *elevated*; conversely, it becomes less effective in depression. Stronger action in elevation (greater convergence) and weaker action in depression *produces an "A"* pattern. Thus, transposition of the MR *below* the horizontal plane is designed to *correct a "V"* pattern. Conversely, reinserting the MR *above* the horizontal plane helps correct an "A" pattern.

A similar logic applies to LR transpositions: to correct a "V" pattern (i.e., by producing an opposite "A" pattern), divergence must be greater in depression when the LR muscles are in their original horizontal disposition. Therefore, the LR muscles should be transposed *above* the horizontal plane. For the vertical recti, the prevailing principle is that *medial* transpositions favor adduction: medial transposition of the SR corrects a "V" pattern, while that of the IR corrects an "A" pattern. Conversely, *lateral* transpositions of the SR and IR help correct "A" and "V" patterns, respectively.

Similarly, horizontal recti may be displaced vertically to correct vertical deviations, while horizontal transpositions of the vertical recti can be used to address horizontal deviations.

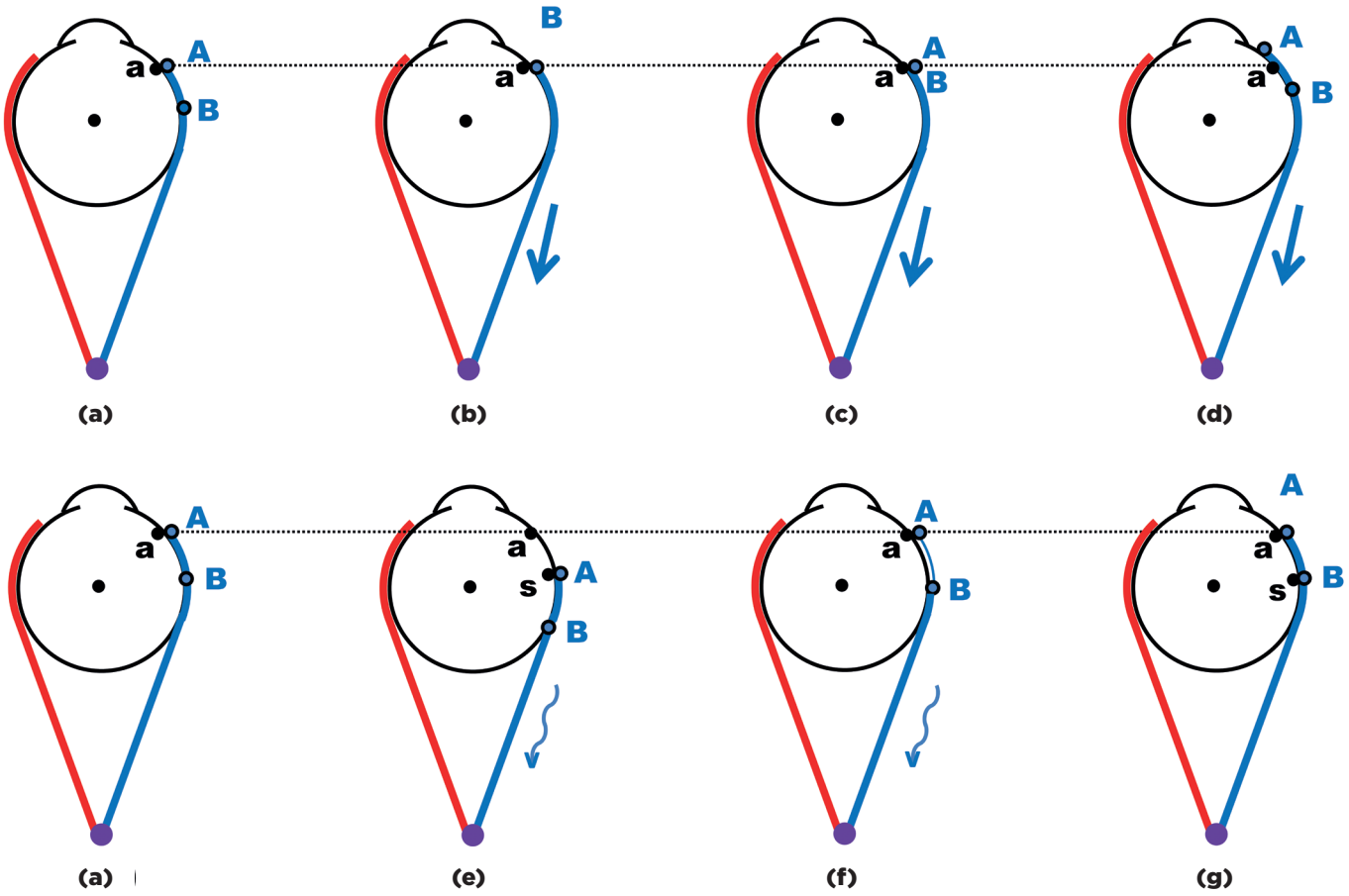

Figure 13. Schematic representation of surgical procedures in an ocular muscle (blue).(a) Original insertion (A, over scleral point "a" of anatomical insertion) and another muscular point (B). Interventions to strengthen (b–d) or weaken (e–g) the muscle: (b) removal of segment (AB) and reinsertion of B at the original position of A (resection); (c) insertion of B over A, without sectioning (plication); (d) advancement of insertion (advance); (e) retroinsertion (recession); (f) longitudinal fiber section (muscle sharpening); (g) suturing part B of the muscle to the sclera (s), preserving the original insertion (A with a) (*fadenoperation*, myoscleral retrofixation).

A widely used procedure is transposition of the SR and IR to the lateral side, either as total transposition (Hummelschein's proposition)[16], partial transposition, or one of its variants. A mechanical effect *preventing the opposite rotation* (adduction) may reasonably be expected, but not the addition of an "*active*" *rotational effect to replace a lost movement* (abduction). Neural control remains unaltered[17]: The vertical recti do not acquire the ability to produce abduction. The SR *contracts* during elevation and the IR during depression, regardless of their insertion site. Thus, after transpositions of the vertical recti to the lateral side, abduction occurs only when *elevation* or *depression* is required.

For example, if the transpositions are performed on the left eye, a levoversion (left-eye abduction) provides no stimulus to the silent vertical recti (LSR and LIR). During dextroversion, activation of LSR and LIR may occur as synergists of left adduction, further *restricting adduction*. In summary, even if the surgery is well executed, the *active* forces of the transposed muscles may result in binocularly abnormal (disjunctive) rotations. Passive tensions of the transposed muscles can, however, contribute to restoring abduction, though at the cost of limiting adduction, much like elastics or springs)(*6). To avoid the adverse effects of active vertical recti transposition, a mechanical alternative for correcting esodeviation due to LR paralysis is simply to shorten (resect) the elastic tissues of the paralyzed but *mechanically* intact LR muscle.

Finally, transposition may also serve to partially or completely *neutralize* the action of a muscle while leaving it inserted in the eye[22]. For example, suppose a rectus muscle is split into two halves and each is reinserted symmetrically on opposite sides of its original plane of action. In that case, the *effective insertion* shifts to an imaginary point *inside* the globe. The lever arm for torque generation is then shorter than the ocular radius of curvature. Torque becomes null when the "effective" insertion coincides with the ocular rotation center, eliminating rotation by that muscle. While technically demanding, partial symmetrical transpositions (reducing lever arm length and decreasing the angle of force application) are ease to perform, effectively *weakening* the muscle (Figure 14).

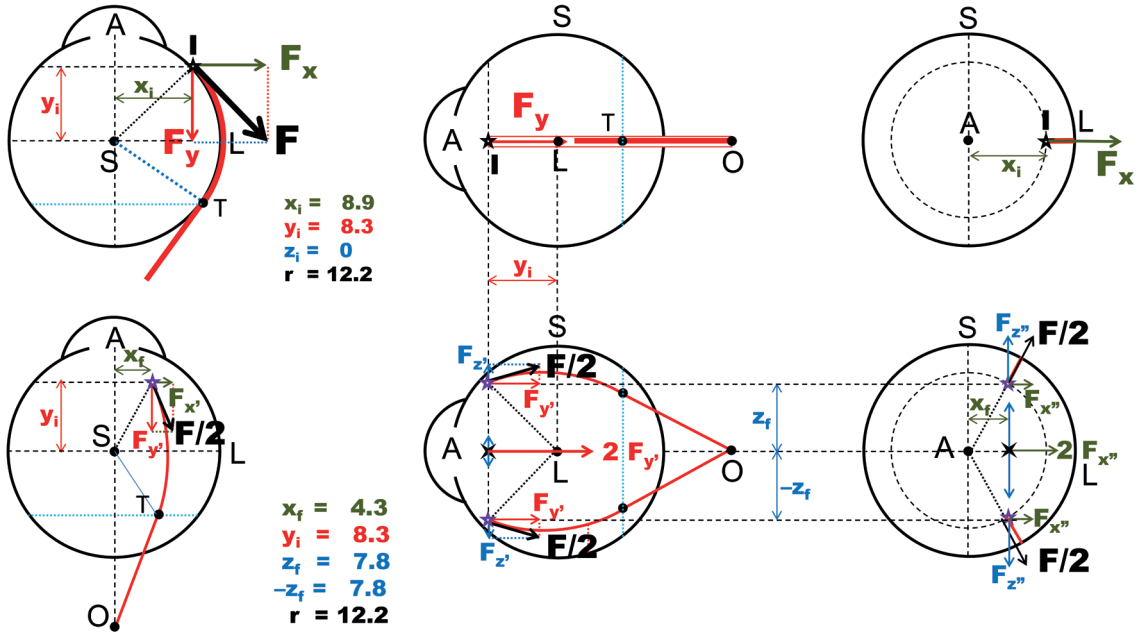

Figure 14. Left to right: horizontal, sagittal, and frontal planes (superior, lateral, and anterior views, respectively) before (top) and after (bottom) longitudinal division of the RLR muscle along its midline, followed by symmetrical reinsertion of each half in a frontal plane (the same *y* coordinate), above (+*z*) and below (–*z*) the horizontal plane. Original insertion coordinates ($+x_i$, $+y_i$) while z_i=0 and reinsertion of superior ($+x_f$, $+y_i$, $+z_f$) and inferior ($+x_f$, $+y_i$, $-z_f$) coordinates are shown. (The sum of squared coordinates equals the square of the ocular radius of curvature.) The original lever arm, equal to the ocular radius of curvature (12.2 mm) decreases from $(8.9^2 + 8.3^2 + 0^2)^{½}$=12.2 mm to $(4.3^2 + 8.3^2 + 0^2)^{½}$=9.3 mm, a reduction of ≈24% = [(12.2–9.3)/12.2].

(*6) Recovery abduction, definitely lost due to a lateral rectus muscle paralysis, may be simply obtained by the mechanical effect of "anchoring" the eye in abduction with an elastic device (spring, distensible silicone band). This has already been shown[18-20], but the alternative has been abandoned, though monocularly effective, since, binocularly, it creates deviations to the opposite side, due to Sherrington's law being contradicted[21]. (The medial rectus works *against* a restrictive device, instead of being helped by the relaxation of its antagonist muscle). It is interesting to mention that one of the propositions for the placement of the abducting artifact was to make its ocular insertion close to the *medial* limbus, and its origin fixed at the superior and inferior margins of the orbit, in front of the eye, by a bifid insertion, one above, other below the horizontal plane, so creating a "reverse" traction (abduction pulling)[20].

For instance, a horizontal muscle such as the RLR (Figure 14) will have its abductive power reduced, while the vertical and torsional effects of the two symmetrically placed halves cancel each other out. If each half is reinserted at the superior and inferior poles, the effective insertion coincides with the center of curvature, rendering the lever arm null in primary gaze (as well as in any other ocular position in the horizontal plane).

A trilogy of the oculomotor system Part III – diagnostic tests

Diagnostic complexities of the oculomotor system

Twelve ocular muscles–each with specific actions, which may vary depending on the fibers involved and on the eye's position relative to its orbit–are commanded by three pairs of cranial nerves and a complex neural organization. Together, they coordinate the spatial and simultaneous orientation of the principal gaze axes of both eyes so that vision is directed to any point in space requiring attention. Broadly, this is the structure and purpose of the oculomotor system–so refined and with such high demands–that it is easily affected by various dysfunctions, some of which have inconspicuous causes and are therefore difficult to detect.

As previously described, the oculomotor system organizes rotational ocular muscles functions in pairs. Thus, even for a "simple" rotation, such as rightward movement of the right eye, contraction of the right lateral rectus muscle (RLR) and relaxation of the right medial rectus muscle (RMR)–Sherrington's law–are required(*[7]). For the left eye to conjugate in this same "right gaze," coordination between contraction of the left medial rectus muscle (LMR) and the RLR–Hering's law–is also necessary, with Sherrington's law simultaneously applying to the left eye. Finally, if this "right gaze" is directed at a *finite* distance, "*fusional*" (binocular) convergence is also required. In summary, even a simple abduction of the right eye depends not only on RLR contraction but also on RMR relaxation, though other simultaneous commands for the left eye are required.

Therefore, even for a "simple" ocular rotation, a defective result (partial or complete absence of rotation) may have different causes (Figure 15):

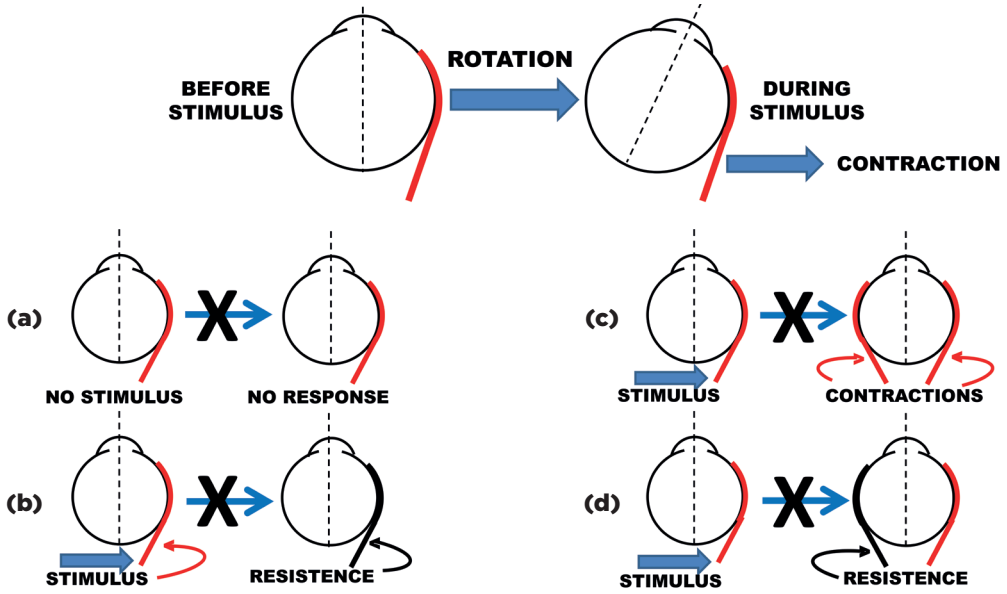

Figure 15. Representation of neural stimulation leading to contraction of a rotational ocular muscle and its subsequent normal response (above), before stimulation (left) and at the end of contraction (ocular rotation, right). Alternatives (a) and (b) illustrate absence of rotation due to muscular paralysis (no stimulation) or inelasticity (loss of compressibility). Alternatives (c) and (d) illustrate absence of rotation due to excessive antagonist action, either from anomalous contraction (instead of relaxation) or from inelasticity (loss of extensibility).

(*[7]) Such a combination of "pairs" of muscles to produce a monocular rotation leads to the apparently paradoxical result of using the force of a medial rectus muscle to obtain *abduction* (its expected *opposite* function). Of course, a *relative* ("centripetal") abduction, that is, a movement from a position of more adduction to one of a smaller adduction–is usually obtained when the medial rectus muscle *relaxes*. But if one, surgically, "anchors" the eye in a position of a "true" abduction, by an elastic artifact, the contraction of a normal medial rectus muscle may produce *adduction*, and, therefore, stretch the elastic artifact (e.g., a band of a distensible material). This may pull the eye to the position of the previous position of passive "anchoring" and make a "real" ("centrifugal") abduction, when the medial rectus muscle relaxes. Such an "anchoring" procedure may be made as a "direct" traction (e.g., fixing the elastic artifact at the lateral ocular surface and backward at the internal lateral orbital surface)[18,19] or as a "reverse traction" (e.g., fixing the elastic artifact at the medial ocular surface) and frontward at the orbital margins[20]. Anyway, though successful by monocular analyses, this method was then discouraged as a practical solution, by the intrinsic *binocular unbalance* during that eye's adduction[21].

a) faulty *stimulation of the agonist* muscle (a true neural paralysis), such as the right eye cannot look right because of RLR paralysis;
b) faulty muscle *response* (an inelastic or "fibrotic" muscle, also–wrongly–classified as a muscular "paralysis");
c) faulty *relaxation of the antagonist* muscle (e.g., limited right eye adduction due to persistent RLR contraction, as in Duane's syndrome–a defective Sherrington's law of the right eye, type I). Such neural defects are now classified as *congenital cranial dysinnervation disorders* (CCDD);
d) excessive *resistance of the antagonist* muscle to stretching (lack of elasticity), as in Brown's syndrome.

In general, ocular rotation results from the simultaneous (active or passive) actions of all rotational ocular muscles of an eye. For example, consider downward gaze. Contraction of the inferior rectus muscle (IR) is required, accompanied by relaxation of its antagonist, the superior rectus muscle (SR). However, the IR also produces adduction and extorsion. To counterbalance these, the lateral rectus muscle (LR) contracts (with simultaneous relaxation of the medial rectus muscle, MR), and the superior oblique muscle (SO) acts (with relaxation of the inferior oblique muscle, IO)(*[8]).

Finally, strabismus *treatment* seldom addresses underlying *causes*; in most cases, it only corrects *consequences*, which explains the frequency of recurrence. For this reason, the best diagnostic approach lies in gathering detailed data about the *effects* of oculomotor disturbances–namely, studying deviations and their manifestations, or in other words, understanding ocular mechanics. In practice, a well-structured examination of oculomotor forces–*active* (muscular responses), *passive* (conservative), and *dissipative* (rotational restraints from periocular structures)–is essential. These examinations constitute *diagnostic* tests, each with different foundations and objectives.

General principles and guidelines of treatments

Disturbances of the ***oculomotor system*** are both *causes* and *consequences* of ***visual dysfunction***. Since good visual function is the natural *finality* of the oculomotor system, it takes *precedence* as the primary purpose of treatment in strabismus and related disorders. In some cases, poor monocular vision (e.g., from a macular scar due to choroidoretinitis or macular retinoblastoma) cannot be corrected, nor can good vision be restored. In other cases, poor vision (such as amblyopia in children) is the *consequence* of strabismus and treatment of vision becomes the absolute priority. Even in the absence of *monocular* visual loss, strabismus must be treated early to prevent loss of good *binocular* vision or to correct diplopia. In summary, *all cases* of strabismus are associated with some degree of visual disturbance.

Therefore, examinations and management of monocular and binocular *visual status* must take priority over those of the *oculomotor system*. The only exception is when strabismus is secondary to a *life-threatening condition* (e.g., brain tumor), which demands urgent attention. Thus, for reasons of importance rather than neglect, examinations of *vision* are not addressed here.

For treatment of *oculomotor* imbalance, the goal is to achieve a "complete" restoration of binocularly adequate eye *positions* (visual axis alignment) and *movements*. However, in some cases, although satisfactory eye alignment is achieved, ocular rotations cannot be fully restored. Muscular *paralysis* ("dead" muscles) and restrictive conditions are the most common causes. Once a rotation is *lost*, it *can* be partially compensated, for example, by anchoring the eye in a desired position through resection of the paralyzed muscle, although such a result (or of *any* other presently available procedure) inevitably reduces movement in the opposite direction. Restrictive scarring can also be surgically removed, though recurrence is common. In some cases, alignment in the primary position of gaze may be the only achievable result, with visual exploration of space then provided by head and body movements.

All rotations are important, but ocular elevation is the least essential and may be sacrificed to preserve other movements. In contrast, downward gaze is critical and should be preserved whenever possible. For example, achieving proper alignment in downward gaze, even at the cost of a slight hypotropia in the primary position, may be an excellent outcome. This can often

(*[8]) The SO produces depression, abduction and intorsion. Thus, in a *very specific direction* of downward gaze (IR + SO) simultaneous actions of adduction and extorsion (IR) may be sufficiently counterbalanced by abduction and intorsion (SO). Although such a theoretical alignment may exist, in *all* other directions of ocular depression, the balancing actions of the horizontal recti muscles are also required.

be compensated by a mild chin elevation–a "proud," socially acceptable head posture (*[9]).

The state of ocular fixations (head and eye positions)

As noted earlier, *vision* intrinsically depends on *ocular motion*. These range from fine, imperceptible micro-movements (essential for detailed visual discrimination) to large eye rotations and translations (necessary for spatial exploration). This intimate relationship between *vision* and *ocular motion*, which cannot be dissociated, is reflected in two main conditions examined in practice:

1. The state of apparent absence of ocular movement, that is, ***fixation*** (attendance to the object of visual attention).
2. The amplitude and quality of eye movements, whether *monocular* (***ductions***), binocularly *conjugate* (***versions***), or binocularly *disjunctive* (***vergences***).

Thus, the most basic information about the *oculomotor system*–and, simultaneously, about fine visual discrimination–comes from examination of ***fixation*** (i.e., eye position or direction), both in temporal and spatial terms.

A) Stability

The *temporal* stability of *fixation* is essential, since gaze on an object must be continuously maintained for fixation to "exist." Poor fixation, manifested as vague or imprecise gaze and an inability to maintain steady attention, reflects impaired vision due to poor ocular reception, neural deficits (e.g., blindness), or severe psychomotor abnormalities. For examination of stability, the subject must attempt voluntary fixation, even if unsuccessful. Rhythmic, involuntary instability of fixation (nystagmus) is another indicator of reduced visual acuity. Minute physiological eye movements are not perceptible to the naked eye.

B) Position (direction)

The expected *spatial state* of fixation is defined as the alignment of the *visual* axis (formed by the *ocular* sagittal and horizontal planes) with the *orbital* longitudinal axis ("straight-ahead" axis, also formed by the sagittal and horizontal *orbital* planes)(*[10])[23,24]. When this coincidence is not achieved, an ***absolute*** *ocular deviation* exists, that is, deviation of the eye relative to its orbit. *Absolute* deviation may be due to limited rotation (from paralysis of an agonist muscle or excessive action of an antagonist, whether active "over-action" or passive restriction from scar tissue), or may occur as a compensatory mechanism in nystagmus. Absolute deviation of the fixating eye (the preferential or unique eye) manifests as an ***abnormal*** (or "*vicious*") ***head posture***. Therefore, correction of abnormal head posture requires correction of the underlying *absolute* ocular deviation–typically by surgery on the affected eye or its muscles–regardless of whether the other eye is blind, amblyopic, or free of strabismus. Any deviation of the poorer eye relative to the better eye must be considered separately.

C) Ocular deviations

Ocular deviation refers to the *difference* between the expected and actual direction of fixation of an eye relative to a reference point requiring attention. In theory, both eyes could be simultaneously deviated from a reference point (e.g., strabismus in a blind individual). In practice, however, at least *one* eye is generally aligned, while the other may be deviated or not. Determining the *reference point* for visual axes is crucial; without it, *no conclusion* about deviation can be drawn, although ocular positions can still be documented (Figure 16).

Measurements of eye positions and deviations

Binocular deviations are measured as the *angles* formed by the fixation lines of both eyes (ocular *positions*),

(*[9]) The below summarized case of an iatrogenic hypotropia, besides exemplifying a successful practical solution, is also a challenge to the paradigmatic concept of the *primary position of gaze* as the standard condition to the measurements of eye positions and deviations[23]. (This ideal concept, though theoretically well defined by "the coincidence of the respective ocular and orbital axes," is of an enormous difficulty to be precisely attainable–mainly due to the notion about what should be a "precisely" erect head)[24]. G.M.S.L, a 65-year-old woman came from João Pessoa (2,600 km NE from my city) by the complains of frequent falls when walking or stepping stairs, and the impossibility of writing or reading due to diplopia and confusion, or loss of stereopsis if one eye were closed, after a cerebral aneurysm disrupted 3 years before. No ocular deviation was noted and a perfectly normal binocular vision was presented at her "presumed" primary position of gaze (the best front facing looking with a "perfectly" erect head). The right eye had a limited abduction, but infra and supra rotations practically absent. In October 17, 2015, she was submitted to a resection of 5.5 mm of the right inferior rectus to (*passively*) correct the *right hypertropia in down gaze* (though with a consequent *right hypotropia at the horizontal plane of gaze*). Naturally spontaneous head postures of adjustments were expected to correct small remaining deviations eventually found. But adjuvant additional prismatic corrections could be helpful to decrease or neutralize them (e.g., a "too proud" head posture–due to the right hypotropia at the horizontal plane of gaze–or a remaining "original" deviation–the right hypertropia in down gaze). At her post-operatory visits (2016, 2018 and 2022) small prismatic prescriptions were then preferred to correct a "too proud" head posture.

(*[10]) It is not sufficient to say that such longitudinal *axes* (ocular and orbital) are *perpendicular* (or *normal*) to the respective frontal (ocular, orbital) *planes*. Such planes may be rotated around those respective lines.

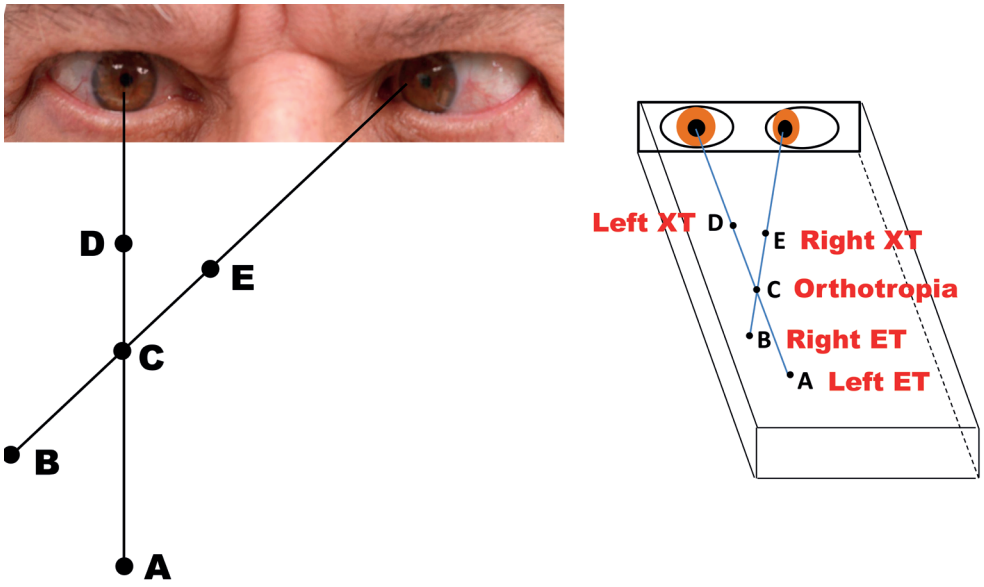

Figure 16. Right: schematic perspective showing two eyes and points (A–E) on the horizontal plane toward which the visual axes may be directed. Left: the same scheme before the horizontal ocular plane is rotated 90° to align with the plane of the page. A photo like this (left) is typically interpreted as representing esotropia of the left eye (assuming the right eye is fixating "straight ahead"). Rigorously, however, any interpretation of horizontal deviation depends on the fixating eye and the point of visual attention. For example, fixation of the right eye on point A (straight ahead) represents left esotropia, but fixation on point D (straight ahead, closer) represents left exotropia. Fixation of the left eye on a distant point B to the right indicates right esotropia (failure of the right eye to abduct), but fixation on a nearer point E in the same direction indicates right exotropia. Finally, if both eyes simultaneously fixate on point C (asymmetrical convergence), no deviation exists.

whether these correspond to normal vergences, abnormal binocular deviations ("true" strabismus), or intermittent states representing both (heterophoria, or "latent" strabismus). Careful differentiation is required.

A rough estimate of ocular deviation may be obtained using Hirschberg's method[25], which evaluates *asymmetries* between corneal reflex positions when a "luminous point" is placed at a large distance. Each 1 mm of asymmetry corresponds to ~7° (von Noorden[26] reports 8°). If the reflex is centered in one cornea but lies at the pupillary margin in the other, the angle is ~15°; if at the limbus, ~45°; midway between margin and limbus, ~30°.

In Krimsky's method, the measurement is based on the prism value placed before the deviated eye that centralizes the corneal reflex. It is important to note that the difference between the *geometric* longitudinal ocular axis and the *visual* (*fixation*) axis–the angle alpha–is normally ~3° but may reach 8°.

In clinical practice, exact measurement of *corneal reflex position* relative to the visual axis is not possible. For the fixating eye, coaxial alignment of the incident light and corneal reflex defines the visual axis. However, for the fellow eye, the reflex is measured from a "dystopic" direction (the visual axis of the fixating eye), introducing parallax error. Moreover, the correct estimation for one eye does not guarantee the same for the other. In summary, Hirschberg's and Krimsky's methods provide only approximate "objective" measurements, reserved for cases where cooperative methods cannot be applied[26].

The preferred ***unit*** for angular measurements is the *degree of arc* (and submultiples: 1=60'=600″), or alternatively, the *radian (rad)*, with 1 rad = 180°/Π. Dennett[27] proposed the *centrad (crad)*, a centesimal part of the radian, where 1 crad=1.8°/Π≈0.57296°, so 1°≈1.74533 crad.

In clinical practice, however, the *prism-diopter* ($^{\Delta}$), introduced by Prentice[28], became preferred. Its advantage is that it directly relates separation (x, in cm) to distance (d, in m): $1^{\Delta}=100\ x/d$.

Since the ratio x/d can be expressed trigonometrically as $a = arctan(x/d)$, conversion between prism-diopters (P^{Δ}) and degrees of arc ($a°$) is given by

$$a = arctan\ (P/100) \quad \text{(F. I) and, or } 100\ tan\ a = P \quad \text{(F. II)}$$

Thus, $1°\approx1.7455^{\Delta}$, or conversely, $1^{\Delta}\approx0.5729°$. The ratio between prism-diopters and centrads for 1° differs by only 0.01%. However, prism-diopters are nonlinear

relative to degrees (and centrads). At 10°, the error is ~1%; at 50°, it is ~36.6%; and at 90°, the value become infinity. Hence, arithmetic operations with prism-diopters are unreliable.

Alternative definitions of a "new" prism-diopter unit (U_k)[29,30] have been proposed. The conversion formulas are as follows:

$$\textit{arctan (P/100) = a = k.arctan (}U_k\textit{/100 k)} \quad \text{(F. III)}$$

$$U_k = 100.\ k.\ \tan(a/k) \quad \text{(F. IV)}$$

For instance, if P=50$^{\Delta}$, then arctan (50$^{\Delta}$/100)≈26.565°≈46.365 cent-radians, which corresponds to a numerical increase of 50$^{\Delta}$/46.365 crad of about 7.84%. This error becomes much greater if the sum 50$^{\Delta}$ (≈26.565°)+50$^{\Delta}$ (≈26.565°)=133.333$^{\Delta}$ (≈53.130°≈92.730 crad) is considered. In this case, the difference from the "linear" value increases to about 133.333$^{\Delta}$/92.730 crad ≈ 43.79%. However, if one considers k=10, using the F.IV for the value of a=26.535° (when P=50$^{\Delta}$), the result is U_{10}=1,000 tan (26.565°/10)≈46.398ˆ, which corresponds to a difference relative to the value in centrads of about 46.398ˆ/46.365 crad≈0.07%. If one considers the double angle (2×26.535°=53.130°), then U_{10}=1,000 tan (53.130°/10) ≈ 92.996ˆ (slightly more than 46.398ˆ x 2 ≈ 92.796ˆ), which compared to the 92.730 crad, gives a numerical difference of about 0.29%. Note also that in prism-diopters, 50$^{\Delta}$+50$^{\Delta}$≈133,33$^{\Delta}$ (an error of 33.33% on the presumed value of the sum), while with the U_{10} units, 46.398ˆ + 46.398ˆ ≈ 92.996ˆ, an error for the presumed sum of only 0.22%.

Qualification of a strabismus depends on the fixating eye

The type and direction of an eye deviation (strabismus) is defined when "fixation" on an object of visual attention is required separately by each eye. This is obtained by the "*cover–uncover test*," in which one may detect a clear dominance of fixation by one eye relative to the other (monocular strabismus) or whether fixation may (spontaneously or artificially) change between eyes (alternant strabismus).

This "qualifying" test is performed when the preferentially fixating eye is directed "straight ahead," an expression not identical to "primary position of gaze", though often — but wrongly — considered as such. Fixation is then prevented (by "*occlusion*"), and the strabismus is classified according to the direction of the ocular movement of the deviated eye–horizontal (eso or exo) or sagittal (L/R or R/L), or a combination of the four possibilities — required to *recover* visual fixation. Although this test characterizes the type, direction, and "basic angle" of deviation to be corrected, it is not strictly necessary or sufficient for diagnosing the paretic muscle or the site of rotational restriction.

Although seemingly simple ("cover the fixating eye–observe possible movement of the *other eye* resuming fixation–then uncover"), the test yields alternative results whose interpretation requires solid clinical experience. In principle:

1. If *no movement* of the uncovered eye occurs, this indicates that the eye either cannot fixate (because of poor vision) or was not previously deviated.
2. If movement occurs, the eye was previously deviated (strabismus). If–when the other eye is uncovered–the eye returns to its former position, the strabismus is "*monocular*." If fixation remains with the previously deviated eye, the strabismus is "*alternant*."

Although the "uncover" step suffices to determine the type (monocular or alternant) of strabismus, repeating the "cover–uncover" test on the newly fixating eye is recommended. This *double* examination not only confirms the type and direction of deviation but also suggests which eye is primarily affected. For example, in a vertical deviation L/R (left eye higher than right) of 15ˆ with fixation by the right eye (REF), if, under left eye fixation (LEF):

a) The deviation is 12ˆ but still L/R → "comitant" L/R vertical deviation.
b) The deviation decreases to 2ˆ but remains L/R → "incomitant" L/R vertical deviation, suggesting paresis of a cyclovertical muscle in the RE (RSR or RIO) or restriction of RE elevation.
c) The deviation increases to 28ˆ but remains L/R → "incomitant" L/R vertical deviation, suggesting paresis of a cyclovertical muscle in the LE (LSO or LIR) or restriction of LE depression.
d) The deviation becomes 20ˆ but changes direction to R/L → "dissociated (or divergent) vertical deviation" (DVD).

Options (a) to (c) follow the clinical rule that ***a deviation*** (ET, XT, R/L, or L/R) ***is greater when fixation is directed by the eye containing the*** (most) ***underactive muscle*** or ***rotational restriction.***

Measurements of deviation at *primary position of gaze*, though often used to define type and "basic" angle (with fixation by the preferential eye), *are not strictly*

necessary for diagnosing the paretic muscle or site of restriction.

Advantages and difficulties of subjective measurements

When a deviation is unapparent (absent or inconspicuous), the "cover–uncover" test becomes more complex, since even temporary interruption of fixation (cover step) may evoke deviation. After uncovering, the eye may recover (heterophoria) or not, and corrective movement may go unnoticed. Thus, distinguishing heterophoria from small-angle strabismus may require additional information (e.g., monocular visions and binocular correspondence).

Subjective tests of deviation (depending on the patient's reports) are *quantitatively* more accurate (able to detect deviations as small as 0.5$^{\Delta}$) than the cover–uncover test, where deviations of about 2$^{\Delta}$ may escape expert detection. However, discrepancies between objective and subjective measurements of strabismus often indicate *anomalous visual (binocular) correspondence*. In addition, subjective tests (e.g., binocular positions using Maddox rods) typically yield *greater* deviations than objective methods, even in normal individuals (heterophorias), likely due to stronger binocular dissociation.

Monocular rotations (ductions)

After testing for deviation at the "basic" straight-ahead position, *monocular rotations* (***ductions***) should be examined. Horizontal and vertical rotations are usually ample but limited by check ligaments and periocular structures to about 50°. In practice, most ocular motion results from combined rotations and *translations* (via head movement), so *pure ocular rotations* are generally smaller (15°–20°).

As shown in Figure 15, two main causes limit rotation: lack of *active* force (*muscle paralysis*, absence of pull) or excessive *(passive* or *active*) restrictive force (excess of opposite pull). Active forces are generally strong, so even when deficient, *monocular* rotations of *normal* amplitudes may occur. However, small defects of neural command are detected by comparing *binocular* rotations (*versions*). Thus, *normal monocular* amplitude is not proof of normality, as it may occur even in muscle palsy. In such cases, end-point stabilization of an "extreme" rotation fails (antagonist pulls eye back), producing a slow centripetal drift followed by a fast corrective saccade. This corrective phase defines the "direction" of nystagmus. For example, right eye nystagmus in abduction (saccadic to the right) indicates RLR underaction.

Incomplete or absent rotation (partial or total) indicates underaction, requiring differentiation between lack of active force and restrictive opposition. Paralysis/palsy (loss of active force) must be distinguished from faulty rotation caused by scar tissue, fibrosis, contracture or passive shortening of antagonist muscle or its overaction (erroneous stimulation, e.g., Duane's syndrome). These *differences* are resolved by *differential tests*: the test of *generated forces* (*active force*)[31] and the *passive duction test* (Table 1).

The duction and generated force tests are complementary: the duction test is "positive" for *restriction* ("negative" for paralysis)(*[11]), whereas the force test is "positive" for absence of forces, or *paralysis* ("negative" for restriction; Table 1)(*[11]).

Strictly speaking, the ***(passive) duction test***–evaluating whether ocular rotation in the expected direction can occur with external traction assisting the subject–is a specific proof of ***rotational restriction***. If *no rotation* occurs, a *restrictive impediment* is confirmed. Conversely, if rotation does occur, *restriction* is excluded. In this case, one could argue that, as restriction is denied, the absence of ocular motion (without external help) indicates muscular paralysis. However, while the diagnosis of paralysis may be inferred as a "negative"

Table 1. Tests for distinguishing the presence (or absence) of ocular rotations by passive ductions and by generated forces in different clinical conditions.

CONDITION / TEST	NORMAL	MUSCULAR PARALYSIS	ROTATIONAL RESTRICTION	PARALYSIS AND RESTRICTION
PASSIVE DUCTION	YES	YES	NO	NO
ACTIVE (GENERATED) FORCE	YES	NO	YES	NO

(*[11]) It is convenient to avoid the ambiguity of meanings "*positive*" (or "*negative*") to designate an *observed result* of a (passive) duction test, since such *result* is ***opposite*** to the respective and consequent *diagnosis*. In fact, if a "*positive*" (passive) duction is referred when an *intended* rotation is *absent* (which means a restricted *motion*), the term ("positive") before "duction" may be erroneously taken as indicative of a "good" (*present*) tested "duction". Conversely, if a "positive" (test of) *generated force* is used to mean ***absence*** of forces *(muscular paralysis)* it may be erroneously taken as a "positive" test of "generated forces".

proof of absent rotational restriction, the test cannot serve as a "positive" proof of paralysis.

The same reasoning applies to the ***test of generated forces***: It may indirectly suggest restriction (if an evoked force is present), but it is specifically designed to confirm or exclude ***muscular paralysis***. Summarizing: if an eye is *fixed* in adduction, the (passive) duction test may confirm a rotational restriction but does not exclude an associated paralysis of the LR. Conversely, the test of generated forces may confirm paralysis but cannot exclude the possibility of a superimposed rotational (mechanical) restriction.

The (*passive*) duction test can be performed in an alert subject or under general anesthesia, while the test of (*active*) generated forces requires a cooperative subject. Alertness and cooperation thus limit both tests in children and certain patients. In practice, indirect clues about generated forces may be observed clinically. A muscular rotational activity in a generated force test is recognized when the examiner feels a sudden pull through forceps holding the eye opposite to the intended movement (e.g., for testing abduction force, the eye is held in adduction). If unrestricted, this produces a sudden, saccadic movement with relatively *high speed* and sometimes *large amplitude*. If paralysis is present, movement may still occur (due to relaxation of the contracted antagonist), but it is *slower*, *smaller* (a centripetal abduction), and usually does not cross the midline (no centrifugal abduction). Note, however that large "spring-back" rotations under anesthesia, entirely passive and due to "extremely" stretched muscles, may reach peak velocities similar to normal voluntary saccades.

Severe restrictions as well as simultaneous paralysis of opposing muscles often cause *inversion* of deviations when fixation changes sides. For example, in horizontal insufficiency (restrictive or paralytic) of RE horizontal rotations, esotropia appears in dextroversion and exotropia in levoversion. With vertical limitations of the RE, L/R deviation appears in supraversion and R/L deviation in infraversion.

Once restriction is diagnosed, its localization may be studied by considering the restriction as a fixed "point" around which the eye "rotates" (effectively *translating*; Figure 17). The larger the lever arm (distance from restriction point to traction point) the greater the obtainable motion. The eye may be *translated* outward (toward the restriction side; Figure 17d, g) or inward (opposite the restriction; Figure 17c,h). A short or stiff antagonist (fibrotic or contracted) may act as the

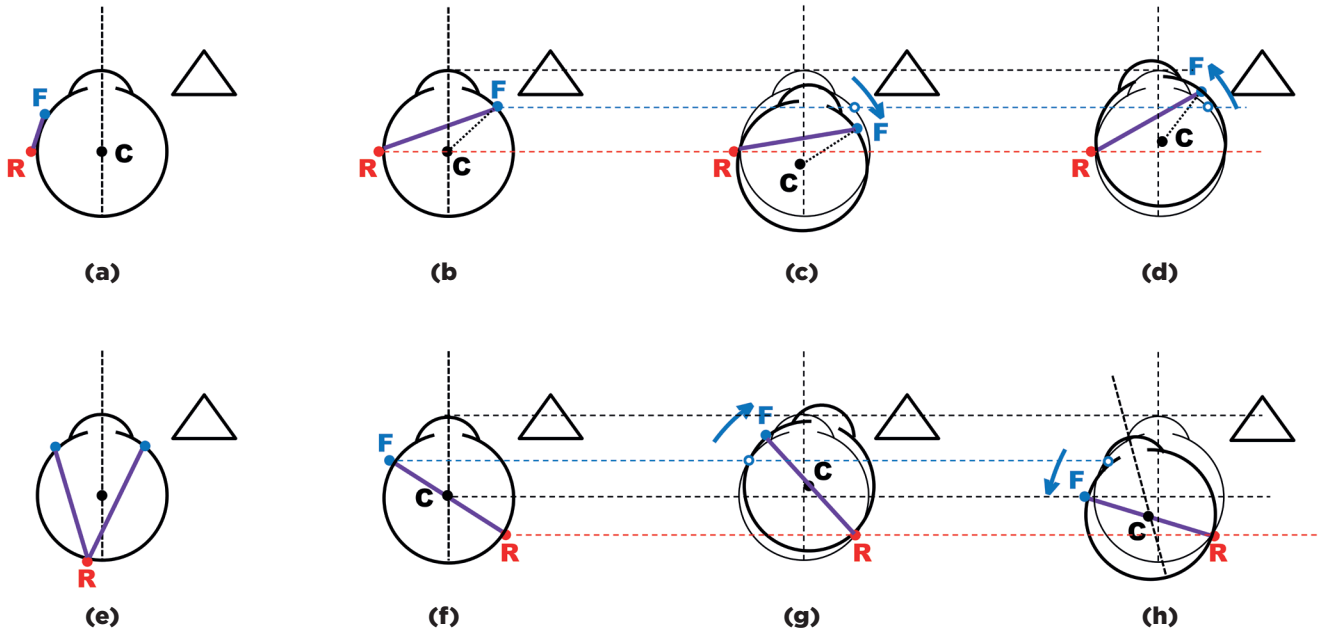

Figure 17. Pictorial representation of the horizontal plane of the eye with restriction points (R) and possible forceps application sites (F). (a) If the distance between R and F (lever arm) is too short, or the restriction point is very posterior (e), displacements are minimal. (b,f) With a large lever arm, the eye may be pulled outward toward R (d,g) or inward opposite R (c,h). Broken lines indicate displacement of the center of ocular rotation (C) and the whole eye around fixed point R.

restricting force. In this case, outward translation is more limited than inward. Large scar tissues may abolish translational rotations entirely.

Measurements of the deviation in diagnostic positions[32]

As noted, slight dysfunctions are not readily detected by *monocular* rotations (*ductions*). Comparing *binocular conjugate* rotations (*versions*) reveals them as (binocular) *deviations*. Causes vary, but all present as defective rotations–whether from *abnormal stimulation* (insufficient or excessive) or *restricted movement* despite normal stimulation.

It is well established that muscle dysfunction manifests in all gaze directions but maximally in the *diagnostic position* of a specific muscle, that is, the direction where its primary action is most required. For example, the left lateral rectus (LLR) muscle, whose primary action is abduction, is maximally tested when the eye looks as far left as possible. While *LLR* underaction appears in all positions, the *greatest esodeviation* occurs in *levoversion*, especially when the *left-eye fixates*. This follows Hering's and Sherrington's laws (Figure 18). Since LLR stimulation decreases progressively from left to right, the *minimal deviation* occurs in *dextroversion*, particularly when the *right eye fixates* (Figure 18). Clinically, this explains compensatory head positions: in LLR paralysis, the head turns *left*. (Mnemonic: the paralyzed muscle "*pulls*" *the head* toward its side.)

Similarly, the right lateral rectus (*RLR*) muscle is maximally activated in abduction (*right gaze*). Here, the esodeviation is greatest when the *right fixates* (Figure 18). Conversely, the minimal deviation occurs when the eye moves to the left, with the fixation of the left eye.

In summary, for an esodeviation supposedly caused by an *underaction* of one or both LR muscles–whether

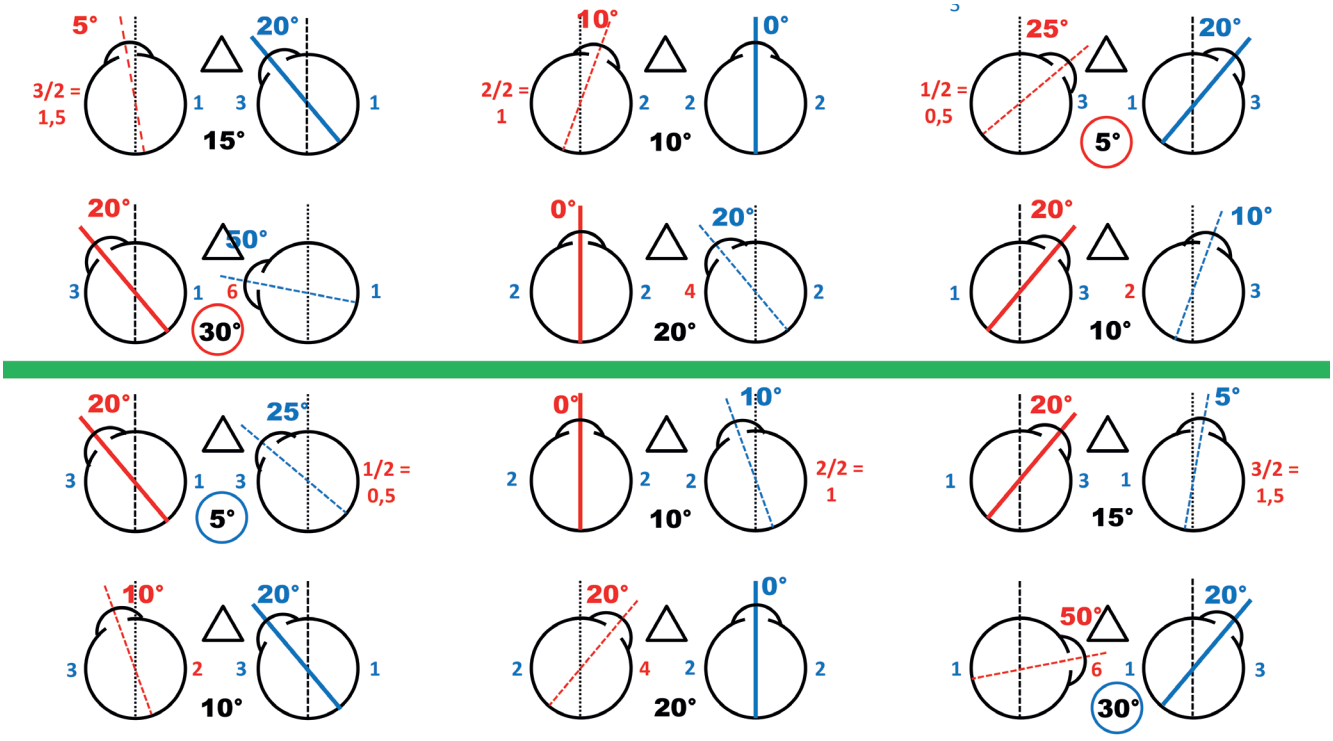

Figure 18. Schematic representation of deviations caused by an underactive left lateral rectus (above) and an underactive right lateral rectus (below). The visual axis of the fixating eye is shown by a bold, continuous line (in blue for the right eye — first and fourth rows; — in red for the left eye — second and third rows). The visual axes of the non-fixating eyes are indicated by thin, interrupted lines (in red for the left eye, in blue for the right eye). Muscular activities are represented by small numbers at the sides of each eye, denoting arbitrary units of 10°. For the affected muscle, the activity is assumed to be reduced to half the expected normal value (shown in red in the first and third rows). When the affected lateral rectus muscle is required to pull the eye into rotation, the conjugate muscle (the medial rectus of the opposite eye) is assumed to receive double stimulation (indicated by red figures in the second and fourth rows). If the affected muscle is the left lateral rectus (above), the greatest deviation (30°) occurs in levoversion when the left eye is fixating, whereas the minimal deviation (5°) occurs in dextroversion, when the right eye is fixating. Conversely, if the affected muscle is the right lateral rectus (below), the greatest deviation (30º) is observed in dextroversion when the right eye is fixating, while the minimal deviation (5°) is seen in levoversion when the left eye is fixating. These conditions are therefore exactly opposite.

due to paralysis, paresis, or mechanical restriction of abduction (as if the LR were "underactive")–*only two measurements* are required to identify the affected muscle:

a) Looking to the right with fixation by the right eye (maximal deviation caused by an affected RLR; minimal deviation caused by an affected LLR);
b) Looking to the left with fixation by the left eye (maximal deviation caused by an affected LLR; minimal deviation caused by an affected RLR).

Thus, the RLR and LLR form a ***diagnostic pair*** in cases of esodeviation. Measuring their maximal actions allows differentiation of the primarily affected muscle. If measurements are *approximately equal*, *both* muscles should be considered similarly affected. Objectively, the limit of measurement error is considered 2$^{\Delta}$; therefore, a difference of about 5$^{\Delta}$ between two measurements cannot reliably indicate i*ncomitance* or prove that one muscle is more underactive than the other. Because other sources of error exist, only differences greater than 10$^{\Delta}$ should be considered reliable criteria for *unequal* measurements.

The rationale for this differential diagnostic test–comparing binocular deviations in opposite gaze directions and eye fixations–is that when one muscle is maximally activated, its counterpart is minimally activated, and vice versa. This *principle* of symmetry between opposed ocular positions is implicit. For example, one can compare the deviation at 40° right gaze with the right eye fixating to the deviation at 40° left gaze with the left-eye fixating (*[12]). If, however, the RLR is paralytic (or right-eye abduction is severely restricted), maximal abduction of the right eye may reach only 0° (absolute abduction failure). This alone is *conclusive* proof of severely limited ocular rotation. Nevertheless, testing the rotation of the left eye is still necessary. If the left eye parting from a position of greater adduction also fails to reach or exceed +20° of adduction (that is, no *relative* abduction is present*)* this indicates an even more severe restriction of left eye abduction. In summary, symmetry of rotational amplitudes is advisable for testing comparative functions and eliciting the greatest deviations.

(*[12]) Out of the respective optical centers, optical glasses present prismatic effects for the eyes. But since they have equal dioptric values, the symmetry with which rotations are compared affect proportionally the respective deviations to be measured. For instance, in an esodeviation and positive lenses, each fixating eye abducts at the back of a base--in prism, the rotation is smaller than that which should be expected (but equal at each side) whereas an equally "compensatory" base-out prism is in front of the adducting eye.

Overactions and underactions

Distinguishing muscular overaction from underaction is not straightforward. A muscular overaction (excessive rotation) of the LLR would produce an exodeviation, the exact opposite of the esodeviation caused by its underaction (reduced or absent action, or excessive resistance from opposing forces). However, when "excessive" or "insufficient" action refers to a different muscle–such as the direct antagonist (LMR) or the yoke muscle (RMR)–further considerations are needed. In practice, underactions and overactions usually occur together, one being the consequence of the other. Primary underactions (paralysis, scarring, or other mechanical restriction) are most frequent, but primary overactions may also occur. A typical example of primary overaction is that of the medial recti in *accommodative* esodeviation.

It is common to assume that if a muscle (e.g., the LLR) is "underactive," its direct antagonist (LMR) is "overactive." Thus, if an esotropia is greater in dextroversion when the fixation is directed by the *right eye*, the diagnosis is of a *RLR underaction;* but if the deviation is greater when the fixation is directed by the *left eye*, the diagnosis is of a *LMR overactiion*. However, when faulty rotation is caused by a mechanical restriction, true muscular dysfunction may not exist. Conversely, when yoke muscles (LLR and RMR) are tested simultaneously, a deviation arises if one action is greater and/or the other smaller. Frequently, a muscular underaction is accompanied (though not always) by overaction of the yoke muscle. For instance, in supradextroversion –when the actions of the RSR and LIO are tested–an L/R deviation may appear, whether the RE or LE is fixating. This may be interpreted either as an RSR underaction or an LIO overaction.

The difficulty arises in interpretation: If the RE is fixating and the LE (deviated eye) appears higher than the RE (L/R deviation), this is typically described as a *LIO overaction*. Technically, however, since the *right eye* controls fixation, the measurement reflects a *RSR underaction*. Conversely, if the *LE is fixating* and the RE is lower (non-fixating), this is often described as a RSR underaction, but in fact reflects relative "*overactivity*" *of the LIO*. Strictly, the diagnosis should be *RSR underaction* (with secondary LIO overaction) when the L/R deviation is greater during RE fixation. Conversely, the dominant condition should be described as *LIO overaction* if the deviation is greater during *LE fixation*. Other differential patterns may also occur (Table 2).

Note that although "underaction" refers to the muscle of the lower eye and "overaction" to the muscle of the higher eye, the determining factor for diagnosis is *which eye's fixation produces the greater deviation*. For example, in left Brown's syndrome, a vertical R/L deviation is observed in supradextroversion (first row of Table 2). Parents often report that the RE "deviates" upward (apparent RSR overaction), whereas the true cause is *underaction of the LIO* (restricted left-eye rotation).

Most commonly, the same type of vertical imbalance (R/L or L/R) occurs in all gaze directions, but *dissociated (or "divergent") vertical deviations* may also be observed(*[13]).

Diagnostic muscular pairs (maximal and minimal deviations)

As noted earlier, underaction of the RLR produces an esodeviation that is *greater* in dextroversion, when the right eye directs fixation ("R_R"). The less the RLR is required, the smaller the consequent deviation becomes. In the early stage of dysfunction, when the muscle is completely relaxed (at left gaze), no deviation may be present. Over time, however, "secondary" defects arise: overaction of the direct antagonist (RMR), overaction of the yoke muscle (LMR), and eventually underaction of the antagonist of the yoke (LLR). These factors *increase* deviations in their respective directions of maximal action and gradually *decrease the differences between them*. Until a fully "concomitant" (equal) deviation occurs in all gaze positions, the deviation remains smaller in levoversion, especially when the left eye directs fixation ("r_L").

The opposite condition applies to the underaction of the LLR. By reversing "greater" and "smaller," "dextroversion" and "right eye," with "levoversion" and "left eye" (and vice versa), the logic holds (Table 3). Thus, an esodeviation caused by LLR underaction–whether primary or secondary–is greater in left gaze during fixation by the left eye (L_L), and smaller in right gaze during fixation by the right eye (l_R).

In summary, in an esodviation (underactions of one, or both, the lateral recti muscles), deviation in *dextroversion* is due to a primary effect caused by the fixation of the right eye (R_R) plus a secondary effect caused by a possible underaction of LLR (l_R), that is, (R_R + l_R). Deviation in levoversion is primarily due to an effect caused by the left eye fixation (L_L) but, also, a secondary effect caused by a RLR underaction (r_L), that is, (L_L + r_L). However, such a difference of deviations at right and left gaze, (R_R + l_R) - (L_L + r_L) equals to (R_R - r_L) - (L_L - l_R), that is, the difference between the maximal and minimal deviations caused by a possible RLR underaction (first term) and that of a possible LLR underaction (second term). Therefore, these muscles form the ***diagnostic pair*** for differentiating the cause of an esodeviation.

Similarly, the *RMR and LMR* constitute a ***diagnostic pair*** for *exodeviation*, the *RSR and LIR* for an *L/R* vertical deviation, and the *LSR and RIR* for an *R/L* deviation (Table 3). The oblique muscles, whose primary actions are torsional (frontal plane), should be evaluated by measuring cyclotorsional deviations. These torsions occur automatically, though not voluntarily, during head tilts to either side (right or left shoulder).

However, observations of cyclorotations are difficult because they cannot be voluntarily elicited and because reliable reference points for measurement are lacking. In addition, torsional deviations cannot be measured by the usual prism method employed for horizontal and vertical deviations. Only subjective reports of cyclodeviation are available, which is a major limitation in many participants. In summary, because the primary functions of the oblique muscles (cyclorotations) and their disturbances (torsional deviations) are not easily assessed clinically, alternative methods are required.

Table 2. Diagnosis of overaction and underaction of a pair of conjugate ocular muscles (acting in supradextroversion), according to the magnitude of binocular deviation and the fixating eye(*[14])

MAGNITUDE OF DEVIATION / FIXING EYE and DEVIATION	FRE > FLE	FLE > FRE
(FRE) (FLE) R/L R/L	Overaction RSR	Underaction LIO
R/L L/R	Over RSR > Over LIO	Over RSR < Over LIO
L/R L/R	Underaction RSR	Overaction LIO
L/R R/L	Under RSR > Under LIO	Under RSR < Under LIO

(*[13]) The term "divergent" seems to be confusing, since, indirectly, it suggests a *horizontal* (associated) *divergent* deviation. "Dissociated" is a better term, but still improper, because it suggests that the deviation occurs by (binocular) "dissociation." A simple and better descriptive name for this condition is *Inverted vertical deviation*. The old name, *alternant hypertropia*, implies the possibility of (spontaneous) *change of ocular fixation*, basically a *sensorial* condition, not imperative in this *oculomotor imbalance*.

(*[14]) The prevailing diagnosis (in blue or red) corresponds to the fixating eye (respectively right or left) with which the greater deviation occurs, whereas its relative position (respectively "above" or "below") estimates its dysfunctional activity ("over" or "under").

Fortunately, the *vertical* actions of the oblique muscles are also clinically relevant (hence their designation as *cyclovertical muscles*). A useful methodological circumstance allows differentiation between vertical recti and oblique actions: the vertical actions of the vertical recti are greater than those of the obliques in *all* positions of elevation or depression. However, vertical rectus action is relatively stronger in abduction, whereas oblique vertical action is stronger in adduction.

Therefore, in elevation combined with right gaze (supradextroversion: abduction and elevation of the right eye; adduction and elevation of the left eye), the RSR and LIO achieve their maximal *vertical* actions. These two muscles are considered *yoke* (or conjugate) muscles, although their functions and purposes differ. Importantly, their *vertical* actions *cannot be matched directly*. For the same reason, the pairs RIR–LSO, LSR–RIO, and LIR–RSO, though anatomically yokes, should not be considered *diagnostic pairs* because vertical actions of obliques and vertical recti are not comparable.

Accordingly, table 3 is replaced by table 4, which lists the clinical *diagnostic positions* for vertical recti and oblique muscles.

The gaze directions and fixations shown in table 4 are the classical "diagnostic positions" for each muscle. However, vertical recti and obliques *cannot be compared directly* because of their different rotational properties. Instead, ***equivalences*** are considered only between diagonally opposite muscles (RSR–LIR, RIR–LSR, RSO–LIO, RIO–LSO).

Testing the muscular diagnostic positions with head tilts

In cases of a vertical deviation (e.g., L/R deviation), measurements taken at the so-called *muscular diagnostic positions* are reliable when comparisons are limited to *pairs of muscles with equivalent functions*. For example, differentiation can be made between vertical recti (RSR and LIR) or between obliques (LSO and RIO). However, comparisons between nonequivalent muscles–such as RSR and LSO (or LIR and RIO), or RSR and RIO (or LIR and LSO)–introduce bias favoring the vertical recti. This occurs because, even in *adduction*, elevation produced by the RSR exceeds that of the RIO; similarly, depression produced by the LIR exceeds that of the LSO, even in *adduction*.

Therefore, in an L/R deviation, diagnostic certainty is obtained only when the deviation produced by a vertical rectus (RSR or LIR) is *smaller* than that produced by the corresponding oblique (RIO or LSO). Such conditions are paradigmatic: For example, elevation of the right eye being worse in adduction than in abduction is a hallmark of right Brown's syndrome. In these circumstances, no complementary duction test is required to confirm the diagnosis. If the RSR were weak, an L/R deviation would be greater in abduction of the right eye. On the contrary, even with complete removal of the RIO, the right eye would still elevate in adduction. Thus, the cause cannot be a paralytic RIO but rather a mechanical restriction preventing elevation, especially in adduction. A similar rationale applies to deficits of depression greater in adduction than abduction, as seen when the inferior oblique is excessively tight after resection and anterior

Table 3. Distribution of diagnostic pairs of ocular muscles according to maximal and minimal horizontal (RLR, LLR, RMR, LMR), vertical (RSR, LIR, RIR, LSR), and torsional (RSO, LSO, RIO, LIO) actions

MUSCLE	GREATER DEVIATION	FIXATING EYE	SMALLER DEVIATION	FIXATING EYE
R L R	Dextro	Right	Levo	Left
L L R	Levo	Left	Dextro	Right
R M R	Levo	Right	Dextro	Left
L M R	Dextro	Left	Levo	Right
R S R	Supra	Right	Infra	Left
L I R	Infra	Left	Supra	Right
R I R	Infra	Right	Supra	Left
L S R	Supra	Left	Infra	Right
R S O	R tilt	Right	L tilt	Left
L S O	L tilt	Left	R tilt	Right
R I O	L tilt	Right	R tilt	Left
L I O	R tilt	Left	L tilt	Right

Table 4. Diagnostic pairs of ocular muscles according to maximal and minimal horizontal (RLR, LLR, RMR, LMR) and vertical actions (vertical recti and oblique muscles)

MUSCLE	GREATER DEVIATION	FIXATING EYE	SMALLER DEVIATION	FIXATING EYE
R L R	Dextro	Right	Levo	Left
L L R	Levo	Left	Dextro	Right
R M R	Levo	Right	Dextro	Left
L M R	Dextro	Left	Levo	Right
R S R	Supradextro	Right	Infralevo	Left
L I R	Infralevo	Left	Supradextro	Right
R I R	Infradextro	Right	Supralevo	Left
L S R	Supralevo	Left	Infradextro	Right
R S O	Infralevo	Right	Supradextro	Left
L I O	Supradextro	Left	Infralevo	Right
R I O	Supralevo	Right	Infradextro	Left
L S O	Infradextro	Left	Supralevo	Right

transposition surgery intended to correct dissociated vertical deviation (DVD).

For this reason, it is preferable to associate head tilts *with the test of diagnostic positions* in vertical deviations. Head tilts not only require greater *activation* of vertical recti and obliques (through stimulation of their *torsional* functions), thereby exposing even subtle dysfunctions, but also introduce a compensatory mechanism favoring the obliques relative to the stronger vertical actions of the vertical recti musclus (Figure19).

In an R/L deviation, the potentially underactive muscles are RIR, RSO, LIO, and LSR (Figure 20). Tilting the head to the right evokes levocyclotorsion: incyclotorsion of the right eye (stimulating RSR and RSO) and excyclotorsion of the left eye (stimulating LIO and LIR). Thus, among the four suspect muscles, two (RSO and LIO) are stimulated with right head tilt (Figures 19 and 20, below left). The remaining two (RIR and LSR) are stimulated by tilting the head to the left (Figures 19 and 20, below right).

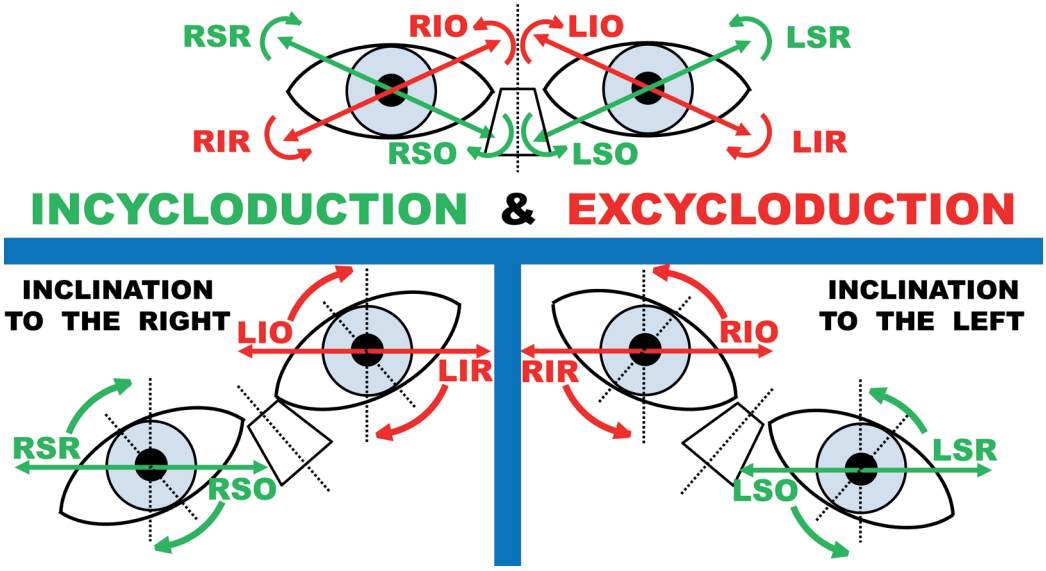

Figure 19. Torsional functions (incycloduction and excycloduction) and their respective causal muscles (above). Half are stimulated by right head tilt and half by left head tilt (below).

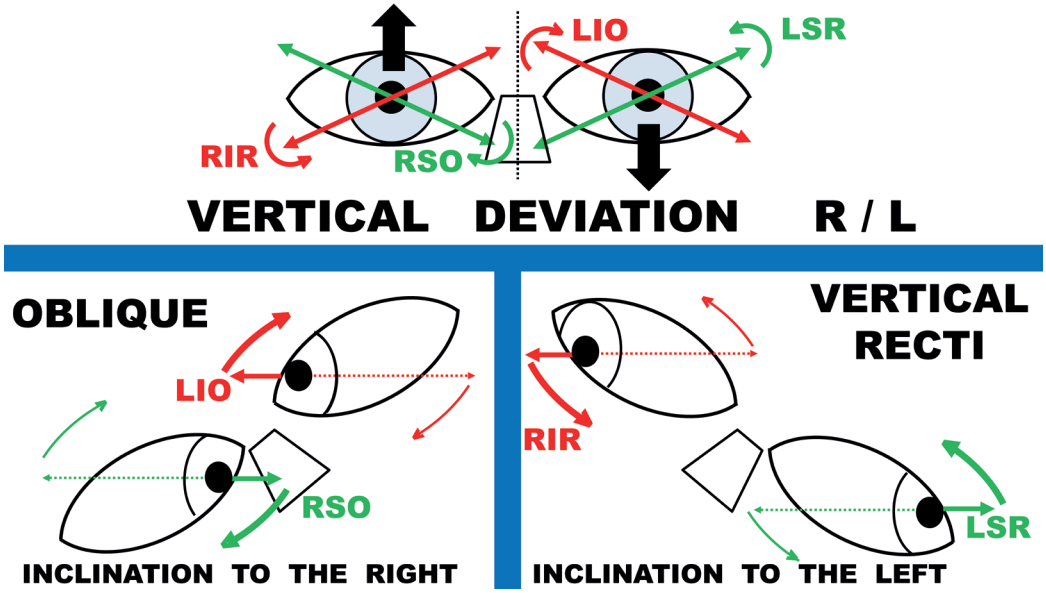

Figure 20. Four possibly underactive muscles in an R/L deviation (above), and their respective stimulation with right head tilt (oblique muscles) or left head tilt (vertical recti).

Accordingly, testing the diagnostic positions of the RSO (infralevoversion) and LIO (supralevoversion) with a right head tilt provides additional stimulation for both their vertical and torsional actions. Conversely, testing the diagnostic positions of the RIR and LSR with a left head tilt adds supplementary stimulation for their vertical and torsional roles.

In an L/R deviation the same logic applies (Figure 21). Here, the potentially underactive muscles are maximally stimulated by combining diagnostic positions with the appropriate head tilt, thereby increasing the likelihood of detecting dysfunction.

Two simple mnemonic rules help determine the appropriate head tilt to maximize stimulation in vertical deviations:

1. The normally *inclined diagnostic positions* of the potentially underactive muscles are shifted *horizontally*.
2. A head tilt that stimulates the *obliques* is directed toward the higher eye (e.g., to the *right* in an *R*/L deviation, Figure 20; to the *left* in an *L*/R deviation, Figure 21). Conversely, a head tilt that stimulates the *vertical recti* is directed toward the *lower* eye (to the *left* in an R/*L* deviation, Figure 20; to the *right* in an L/*R* deviation, Figure 21).

The criteria for comparing maximal deviation versus maximal variation

It should be noted that the strategy of adding a specific head tilt to *increase* the stress on the "diagnostic positions" of each of the four cyclovertical muscles possibly affected in a vertical deviation introduces a new diagnostic criterion. In the classical "diagnostic positions" (with the head erect), the position in which an affected muscle shows its *maximal* deviation (e.g., for the RSR, supradextroversion with the right eye fixating) *coincides* with the position in which its corresponding opponent muscle (the LIR) shows its *minimal* deviation (Table 4).

With the specific position (supradextroversion with the right eye fixating) and a *head tilt to the right* (Figure 21, below, left side), stimulation of the RSR *increases*, so that if underactive, the deviation becomes greater. However, the stimulation of the position of *minimal* deviation of this muscle–while still in infralevoversion with the left-eye fixating–also *increases* with a head tilt to the right. To reach the minimal condition, the head tilt has to be to the *left* (Table 5). Thus, with the addition of the head tilt, the opposed positions of examination of the opponent muscles lose the property of being, respectively, those of *maximal* and *minimal* stimulation. In other words, there are no longer "completely opposed" conditions (Table 5). Consequently, to determine the maximal *and* minimal conditions of stimulation of a muscle (i.e., applying the diagnostic criterion of the greatest *variation* of its deviations), *two* "completely opposed" combinations of conditions (fixating eye, eye position, and head tilt) for *each* muscle would be required. For a specific vertical deviation (say, L/R), eight different measurements

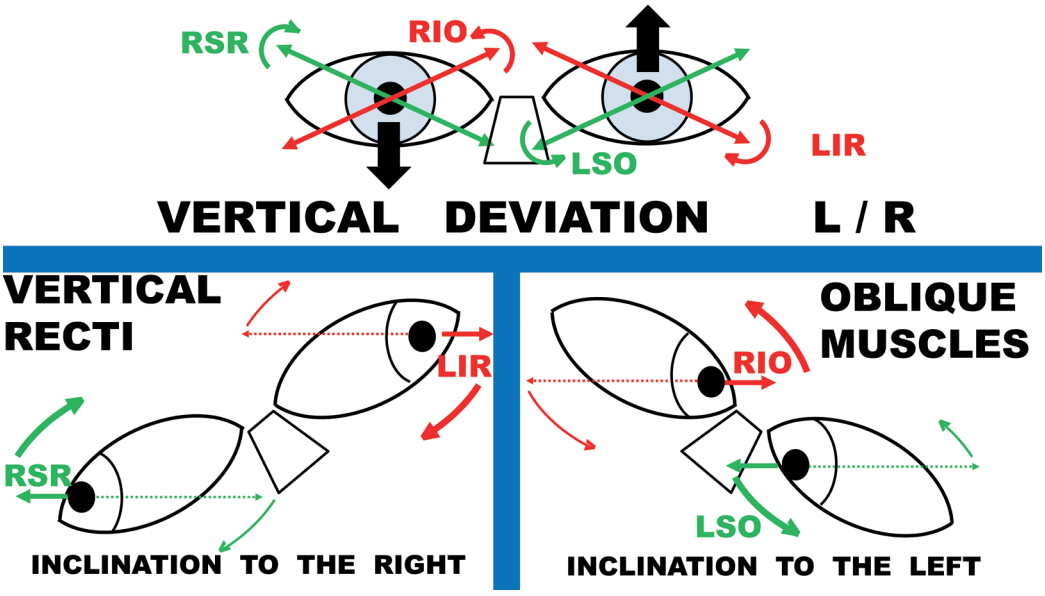

Figure 21. Four possibly underactive muscles in an L/R deviation (above), and their respective stimulation with right head tilt (vertical recti) or left head tilt (oblique muscles).

would therefore be needed–a very time-consuming and impractical proposition. By contrast, with the diagnostic criterion of studying conditions of *maximal* deviations of each muscle, only *four* measurements are necessary.

A possible alternative is to use the head tilt that stimulates the functions of the oblique muscles (e.g., to the left in a vertical L/R deviation; Figure 21, below, right side) and *decreases* the actions of the vertical recti muscles. In this case, the directions for testing the actions of the RSR (supradextroversion, fixating the right eye) and of the LIR (infralevoversion, fixating the left eye) become "vertical," so that measurements in only four different conditions are necessary. However, this introduces a double bias: one favoring the indication of oblique muscles as the most underactive in a vertical deviation (the proper head tilt), and another simultaneously disfavoring the indication of the vertical recti muscles (the same head tilt).

In summary, for the simplicity of a "minimalist" strategy (only four measurements are required) and for ease of execution (a gross comparison of the respective rotational amplitudes is often sufficient, without the need for precise measurements), the diagnosis of the most underactive muscular action in a cyclovertical deviation (say L/R, Figure 21) should be based on measurements of eye deviations in diagonally opposed positions of *equivalently paired* muscles (vertical recti: RSR and LIR; or oblique muscles: LSO and RIO) with the appropriate head tilt (to the right shoulder to test RSR and LIR, and to the left shoulder to test LSO and RIO). In such specific conditions, the possibly *underactive* muscles are *maximally* stimulated. The *maximal* action of a muscle is confronted with the *maximal* action of its "opponent"–a favorable condition of comparison, since both muscles are naturally "equivalent" by their antipodal functions. Thus, the most sensitive and sensible criteria for demonstrating muscular weakness (i.e., its maximal *deviation*) are applied.

Table 5. Conditions of the greater and the smaller deviation caused by the underaction of a specific cyclovertical muscle

DEVIAT.	MUSCLE	GREATER DEVIATION			SMALLER DEVIATION		
		POSITION	FIX. EYE	HEAD TILT	POSITION	FIX. EYE	HEAD TILT
L/R	RSR	Supra dextro	Right	Right	Infra levo	Left	Left
	LIR	Infra levo	Left	Right	Supra dextro	Right	Left
	RIO	Supra levo	Right	Left	Infra dextro	Left	Right
	LSO	Infra dextro	Left	Left	Supra levo	Right	Right
R/L	RIR	Infra dextro	Right	Left	Supra levo	Left	Right
	LSR	Supra levo	Left	Left	Infra dextro	Right	Right
	RSO	Infra levo	Right	Right	Supra dextro	Left	Left
	LIO	Supra dextro	Left	Right	Infra levo	Right	Left

Peroperatory diagnostic tests[8,9,12,33]

In the past, surgical plans for the correction of oculomotor imbalances were considered a direct and unavoidable consequence of clinical data obtained from careful examinations of muscular *activity*. Although such information remains important, it is no longer sufficient for deciding *what procedure* to perform, on *which muscles*, and to *what extent*. The understanding of *passive* forces (the *mechanical* properties of the muscles themselves) and *dissipative* forces has transformed strabismus surgery (and related disorders) from being merely a *therapeutic* step into also a *complementary diagnostic* element of treatment. In summary, strategies and tactics planned before surgery not only can but often *must* be revised based on new data obtained during the peroperatory examination.

1) Passive oculomotor balance (Eye position with no "tonic"–active–forces)

The first "surgical" step, before the procedure itself begins, involves evaluating the "passive" ocular position under general anesthesia. With innervation absent, ocular position depends entirely on mechanical factors and can therefore be considered a passive balance of forces. However, passive forces may also produce motion; thus, their *amplitude* and *velocity* provide additional information. For this reason, duction tests and analyses of the consequences of applied forces are recommended. These are performed by moving the eye into secondary positions of gaze within the *ocular* plane (horizontal or sagittal), where the balance of passive forces is to be assessed(*[15]).

([*15]) As, during an ocular surgery, the subject is in a dorsal decumbent position, the *ocular frontal* plane is parallel to the so admitted *terrestrial horizontal* plane. Therefore, motions at the horizontal and sagittal *ocular* planes in such a condition are subjected to *different* forces when compared to those performed when the subject is standing up. But for practical reasons, since the forces of a studied *ocular* plane suffer, then, the same differential influences, this factor is not considered.

For example, when studying *passive* ocular *motion* in the *horizontal ocular* plane, the eye may be rotated passively (a "duction test") into maximal abduction (e.g., 50°). If this motion is achieved, a restrictive limitation can be excluded. The force required for such a motion is both sensed by the surgeon and, if needed, can be measured(*[16]). More relevant, however, is the portion of that force "stored" by the stretched tissues (primarily the muscle), which can be indirectly evaluated by the *motion* produced upon release. Thus, the surgeon evaluates both the *amplitude* (to a step point) and the *velocity* of the passive return ("centripetal") motion – the so-called ***spring-back ocular rotation***(*[17]).

This first step, a "centrifugal" rotation, essentially represents a simple duction test (Table 1), providing information about *resistance* to rotation and confirming or excluding *restrictive limitations*. The eye is then taken to the symmetrically opposite position (adduction of 50°) and released, producing a passive "centripetal" abduction. The mean of the final positions of these spring-back rotations represents the *balance point of passive forces*. From this, the main causal factor of strabismus can be differentiated. For example, if a deviation of 30° (in the awake state) shows (a) a passive balance equal to 0°, the strabismus is purely *innervational* ("*tonic*"); (b) a passive balance equal to 30°, the strabismus is *restrictive*(*[18]); (c) a passive balance between 0° and 30° (e.g., 12°), the strabismus has a *mixed cause*.

2) Amplitude and velocities of the spring-back rotations

The same *balance* of *passive forces* may arise in different conditions (Figure 22), but the *amplitude* and *velocity* of the returning movements provide further insight. This leads to questions about why the returning motion occurs and why it stops.

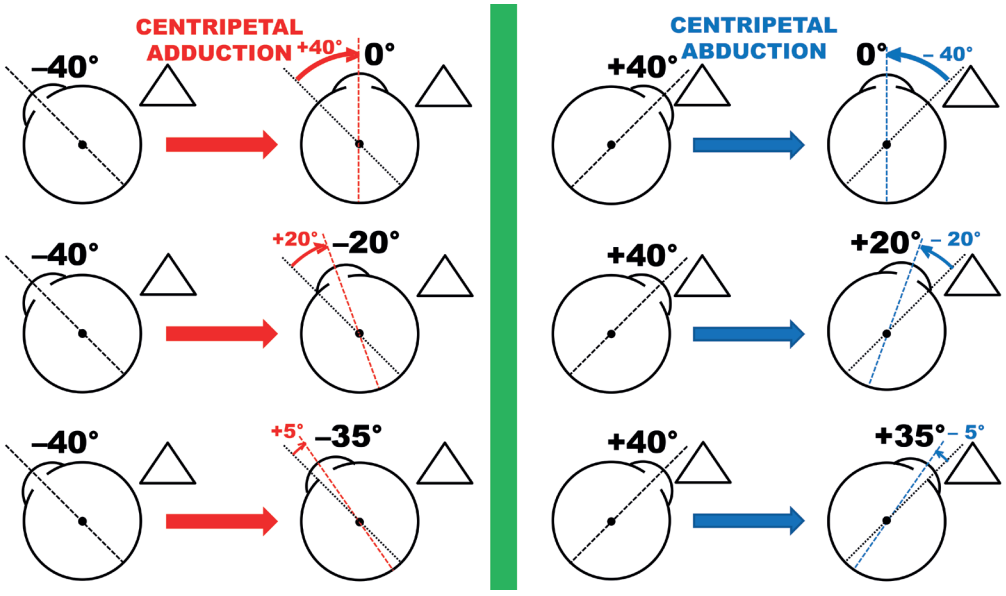

Figure 22. Scheme of spring-back rotations of three different left eyes (top views, each of them in a respective row), showing centripetal adductions (left side) from passive abductions of – 40° (first column) to (after releasing, second column) a passive (centripetal) adduction of +40; before (third column) and after (fourth column) a passive adduction of +40°. The mean value of the final ocular positions reached after the respective centripetal adductions and abductions are *all* the same (0°), whereas the (symmetrically) corresponding amplitudes are quite different (40°, above; 25°, middle; and 5°, below).

(*16) The experiment is very different to that of the classical study of muscular *length-tension* relationships ("curves")[34], since the applied force for a "centrifugal" rotation has to be greater than that applied to an isolated muscle (because besides stretching muscular fibers, it suffers the contrary ("resistive") action of *other* elastic–and inelastic–structures).

(*17) Actually, such a mechanism, described as being similar to that of the retuning motion of an elastic spring being released from its previous stretching tension, is similar to those of other classical examples using the gravitational forces: the descent of a perfectly elastic ball after being vertically thrown; the descent of a ballistic pendulum; but is best compared with the descent of a ball from the top border of a spherical bowl. In all of those cases as well as of others with strings, or elastics, *if no other forces act*, the motion would be harmonic and perpetual.

(*18) Tough "activities" could not be shown (if the eye cannot be moved from that fixed position) one cannot say that innervation ("tonic") state is faulty, since it could be present, but hidden by the restrictive rotations; nor deny that innervation is sound (because it has not been proved).

According to theory, when a force is applied to a body, it moves with a velocity inversely proportional to its mass(*[19]) and if the force continues, the body accelerates(*[20]). In practice, however, other forces intervene: A weak applied force may not move the body at all, or movement stops unless a continuous force is applied. *Resistive* forces, which oppose motion, are called *dissipative* forces and include *inelastic deformations* and *friction*. The kinetic energy of motion is not destroyed but transformed into other forms (e.g., heat and sound), which dissipate into the environment.

Some systems, however, store part of the energy through *elastic deformation*, which can be fully recovered. Thus, motion (kinetic energy) may also be transformed into *conservative energy*. During a duction test, *resistive* forces always occur–sometimes so great that they prevent displacement ("total" restriction). When motion does occur, part of the energy is *dissipated* (friction, inelastic deformation), while another part is stored as *potential energy* in stretched elastic tissues (mainly muscle fibers). The muscles act less like compressed springs and more like extended elastic bands[10].

At the end of a "centrifugal" rotation (the eye held at maximum displacement), the applied force balances the stored potential energy. When released, this energy produces a "*centripetal*" *spring-back motion*, mainly due to the *passive* shortening of the *agonist* muscle. As the motion proceeds, energy *dissipates* until it stops. This storage and recovery follow Hooke's law, expressed as $F=E.\Delta.A/l$, where F is the generated force, E is Young's modulus (elasticity), A is the muscle's cross-sectional area, l is its initial length, and Δ is the extension of the stretch. With a fixed strain or stress (Δ), – the amplitude of the "centrifugal" rotation – a *smaller amplitude* or *slower velocity* of spring-back rotation (that is, a lower value of the corresponding force) indicates a thinner muscle (low A), flaccid (low E), or relatively longer (large l).

Although spring-back rotation does not evaluate *active contractility* (e.g,, of a lateral rectus), Hooke's law provides a mechanical estimate of how stretched tissues may behave (react, shorten) in generating a *passive*, "centripetal" (abducting) force. In practice, this passive mechanism resembles that of a normal rotation produced by muscle relaxation (of the antagonist medial rectus) –similar to a block of wood on an inclined plane where static friction prevents sliding. Thus, spring-back motion reflects the balance between elastic shortening (of stretched tissues) and opposing forces (friction).

During normal isotonic contraction, resistive forces also act, including friction and the conservative distension of antagonist tissues. Therefore, spring-back behavior can *also* predict how a muscle will respond (shorten) in *active* contraction(*[21]). Indeed, spring-back velocities from 50° rotations are comparable with those of normal saccades[9,12,35].

a) Ample (Figure 22, upper row) and/or fast motion: elastic ("good") muscle against normal or weak resistance.
b) Short (Figure 22, middle row) and/or slow motion: weak elasticity ("poor" muscle) against normal resistance, or normal elasticity ("good" muscle) against strong resistance.
c) No motion or very short (Figure 22, lower row): absent or very weak muscle, against normal or strong resistance.

Accordingly, surgical outcomes are expected to be *proportional* to the amplitude and speed of spring-back rotations. A relatively *weaker* muscle (small or slow spring-back) will yield *poorer* results after weakening or strengthening procedures than a stronger muscle.

A broad protocol for examination of an oculomotor unbalance

1) Ophthalmoscopy and ocular refraction

These are cited first, not because they are the initial examinations to be performed, but due to their generic importance in all cases of strabismus and related disorders. Their relevance is so fundamental that it is almost tautological to state they must *always* be conducted. *Ophthalmoscopy* provides valuable

(*19) By definition, *kinetic energy*, $E_k=(m.v^2)/2$, where E_k is the *kinetic energy* (motion) of a body, m its *mass*, and v, its *velocity*.

(*20) Also, by definition, $F=m.a$, where F is the (continuously) applied *force*; m, the body's *mass*; and a ($=v/t$), its *acceleration*.

(*21) Since the dissipative forces may be taken as naturally the same for "centripetal" and "centrifugal" rotations, the spring-back rotations may be taken, also, as analogically similar to a motion produced by the "active" contraction of an ocular muscle (and its consequent and expected response). In case of a normal "active" ("centrifugal") rotation, the origin of the force is the muscular contraction elicited by the neural stimulation, against it works the passive stretch of the antagonist muscle (and of the other periocular elastic structures), besides of dissipative forces. In case of a "passive" spring-back ("centripetal") rotation, the force is the tangential pulling from (mainly) the *shortening* of a stretched ocular muscle, against it only dissipative forces act. From a mechanical point of view, the force of a shortening structure (Δ) be it "actively" elicited (relaxation), or "passively" obtained is the same. Actually, for the same amount of displacement (amplitude of ocular rotation), the passive centripetal motion requires less force than the active centrifugal rotation. Or, conversely, for the same force, the amount of centripetal rotation should be greater.

information about the state of *vision* and, consequently, *visual fixation* (sometimes confirming or ruling out amblyopia). It also yields insights into *retinal status* (e.g., the need to treat a possible recurrence of toxoplasmosis before ocular surgery) and *optic nerve* condition.

The measurement of *ocular refraction* is essential for possible optical corrections, not only to ensure the best visual performance but also due to the unequivocal importance of the synkinetic interaction between accommodation and convergence. This interaction may explain the etiology of certain forms of strabismus (e.g., overaction of the medial rectus muscles triggered by accommodation) or guide *treatment* (reducing accommodation - and, then, *convergence* - for accommodative esodeviations or increasing it for exodeviations). By definition, ocular refraction must be measured with accommodation completely absent, a state that–because it is mediated by the *autonomic* nervous system–can only be fully achieved pharmacologically (*cycloplegia*).

2) Ocular fixations

The state of visual fixation in each eye is such a fundamental condition for diagnosing oculomotor imbalance that it may sometimes be detected even before formal examination begins. For example, the presence of an "absolute" eye deviation (gaze fixation in a position markedly different from the primary position, often associated with a "*vicious*" *head posture*) or different directions of the visual axes in each eye (true strabismus).

Several fixation states exist, each closely related to the quality of monocular or binocular vision. Fixation may be *absent* (indicating poor vision), present but *unstable* (nystagmus), limited to specific gaze positions or present binocularly but *unstable under cover* (latent nystagmus). In monocular strabismus, fixation may be deviated and unattainable even when the other eye is prevented from fixating, or attainable but not maintained once the other eye resumes fixation. In alternant strabismus, fixation is attained and maintained with either eye (as demonstrated by the cover–uncover test). In heterophoria, fixation is displaced under cover but recovers when uncovered, while the fellow eye maintains fixation. Variants of these possibilities also occur. Importantly, alternant fixation does not necessarily indicate "good" visual acuity in both eyes, but rather "*equal*" *acuity*.

From fixation examination, one can indirectly confirm the presence and type of strabismus (monocular vs. alternant), and qualify it according to deviation direction relative to the primary gaze position (eso-, exo-, right/left, left/right).

3) Basic measurements

Conventionally, the *magnitude* of strabismus is defined as the angular deviation between the two visual axes, measured relative to the visually "best" (fixating) eye in the primary gaze position (clinically assumed as "straight ahead" with an "erect head"). In alternant strabismus, repeating the measurement with the fellow eye fixating is also advisable. In monocular strabismus, whether such a second measurement is necessary remains debated.

These measurements help classify strabismus as "concomitant" (equal or approximately equal angles with either eye fixating) or "incomitant" (different angles depending on the fixating eye). However, this classification may be superfluous, as more accurate criteria can be obtained from diagnostic position testing of the responsible muscles.

If the visually "best" eye is itself "*absolutely*" deviated, either movable or fixed within the orbit, measurement of head deviation becomes fundamental in addition to strabismus assessment (relative deviation) of the fellow eye (if present).

4) Ocular rotations

For ***horizontal deviations***, assessment of rotations (and measurement of deviations at their extremes) in *secondary* positions is necessary. These include right and left rotations (horizontal plane) and up and down rotations (sagittal plane). Unequal amplitudes (incomitance) may be revealed, but results must be carefully interpreted–right and left rotations should be tested with fixation by the homonymous eye in esodeviations, or by the heteronymous eye in exodeviations. Vertical rotations may demonstrate *variations in horizontal deviation*, known as *alphabetic* patterns (A, V, λ, Y, X, "diamond").

For ***vertical deviations***, rotations must be assessed in *tertiary positions*, which correspond to the *diagnostic positions of the cyclovertical muscles* (diagonally situated relative to the primary position, between secondary positions).

Together, one primary (for *qualifying* the deviation), four secondary (for *horizontal* deviations), and four tertiary positions (for *vertical* deviations) form a mosaic of *nine* positions. Considering that fixation of each eye must be evaluated (while deviation is measured with prisms before the fellow eye), *18* measurements are required. If head tilts are added, *eight more* are needed. Such an extensive set of measurements is not only fatiguing but also unnecessary, and many can–and should–be avoided. Nevertheless, certain key measurements remain indispensable, as previously discussed.

5) Measurements of deviations in diagnostic positions: Muscle pairs

Although not always required for surgical *indication*, these measurements are usually fundamental in deciding *which muscle(s)* to operate on, the *extent of surgery*, and the *technique to be employed*. In cyclovertical deviations, classical diagnostic position testing of *underactive* vertical rectus and oblique muscle pairs should preferably be performed with the appropriate *head tilts*. This approach better balances the bias of relatively stronger vertical actions of vertical recti muscles.

6) Peroperative tests

Simple tests of passive and active forces cannot always be performed–children, for instance, often do not tolerate them. However, if testing of active (generated) forces is possible, at least passive force tests (peroperative assessments) should *also* be performed. In many cases, these tests lead to changes in the initially planned surgical approach. The potential outcomes of surgical procedures may also be indirectly anticipated by evaluating "spring-back" rotations.

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