Original Articles

A quantitative analysis of extraocular muscle cooperation and squint

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The mechanical properties of human extraocular muscles have recently been described. This allows one to use the force-balance equation of mechanics to solve for the complete mechanical state of all six extraocular muscles and the passive orbital tissues in any eye position. This includes each muscles' force, length, innervation, and unit action vector, which describes how its force is distributed to act horizontally, vertically, and in torsion. Most important, this calculation method allows one to predict the tropias that occur in an eye subject to any imaginable form of peripheral pathology. It also permits, by calculation, an estimation of the correction that might be expected from any sort of muscle surgery. The tight lateral rectus syndrome is used to illustrate the application of the method in diagnosis and surgery. It also offers, for the first time, a quantitative estimate of the multitude of ways in which muscles can interact and interfere with each other when they hold the globe. Two of the results are interesting: because muscles have different lengths and sizes, their innervational participation in a movement can appear to be quite different than their mechanical participation. From an innervational standpoint, the vertical recti and obliques participate equally in vertical gaze. Muscles interfere with each other a good deal and necessitate changes of innervation to counteract these cross-couplings. This causes unexpected dependencies of innervation on eye position and leads, for example, to the fact that the superior rectus innervation in up gaze is just as large in adduction as in abduction.

Key words: extraocular muscles, strabismus, squint, muscle mechanics, mechanical analysis of muscle, eye movements, ocular tropias.

The way in which the extraocular muscles cooperate to hold the eye in various positions is a problem of mechanics. The orbital tissues obey certain mechanical laws which can be expressed mathemat-

ically so, in theory, one should be able to calculate the lengths, tensions, and innervations of all six muscles for any gaze position. More important, it should be possible to predict, by calculation alone, the deviations of an abnormal eye produced by any sort of peripheral neuromuscular or mechanical pathology. The ability to do this could be of some help in the diagnosis and surgical correction of strabismus. It might provide a basis, founded on the laws of mechanics, for what is now a practice based, more or less, on empiricism.

Solutions have been proposed for parts of this problem. For example, one must
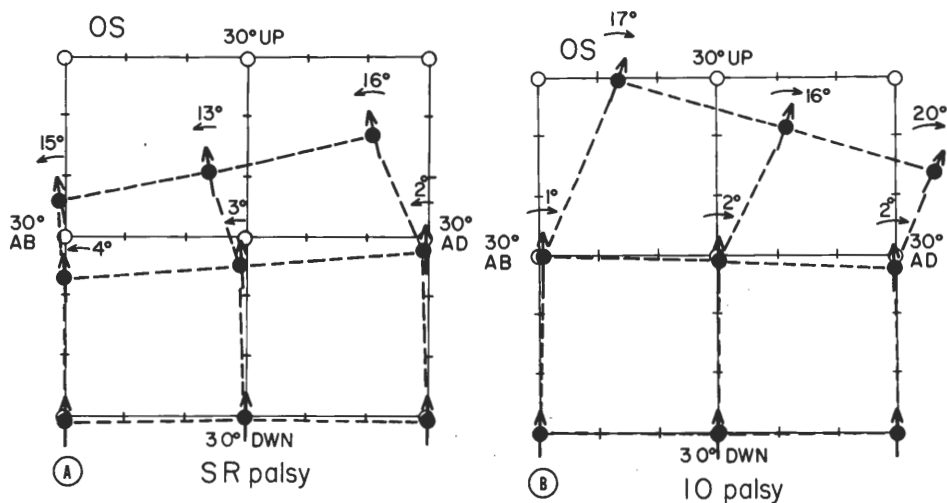
Fig. 1. An example of the use of the theoretical calculations described in this paper to predict the tropias produced by complete SR palsy (A) and IO palsy (B). The field of gaze is shown, viewed from behind. The right eye looks by 30° in the nine principal positions (open circles). The filled circles indicate the position of the abnormal left eye moving under cover. The arrows and numbers indicate cyclotorsion when it exists. This is similar to a Hess chart except that the coordinates of eye position used in this study (Fig. 2) are slightly different that those normally used in the Hess test. (Specifically, the tertiary positions, 30°, 30° in this illustration would appear 30° up but a horizontal deviation of only 25.7° on a Hess chart.)

know how much each muscle lengthens or shortens when the eye moves to a new position. Boeder[1] has presented an approximate solution to this problem. One must also know the axis about which each muscle would act to rotate the globe from any given position. Krewson[2] has given an approximate solution to that problem for a few gaze positions.

But the major obstacle to a complete solution has been that little was known about the mechanics of the muscles themselves. A muscle is a mechanical device that produces a force which depends on its length and innervation. A quantitative description of that dependency is essential if we are to proceed. Those data were obtained in collaboration with A. Jampolsky, C. C. Collins, A. B. Scott, and D. M. O'Meara of the Smith-Kettlewell Institute of Visual Sciences in San Francisco.[3] With those data a complete mechanical solution to this problem became possible.

The problem is solved in two stages. The first is this: if the eye is normal, what innervation must be supplied to the six extraocular muscles to hold it in any given position? This exercise, in itself, is interesting. It is often said that every eye movement involves the activity of every muscle but, having said this, it is usually forgotten because of the great complexity of all but the simplest types of muscle actions. In a complete mathematical analysis, every force must be accounted for. The results of such calculations then allow one to observe all possible muscle interactions which provides a deeper understanding of the mechanics of normal eye movements.

However, the major purpose of this study is the quantitative prediction of the tropias produced by any orbital abnormalities such as palsies, fibrotic muscles, or abnormal innervations. In this case, one assumes that muscle innervations are known (from the solution of the first problem) and one applies the laws of mechanics to calculate the deviations produced by the pathology of that eye, moving under cover, as the patient tries to look in different directions. This is similar to the

sort of data produced clinically by tests of the Hess or Lancaster type. Fig. 1 is an example: it shows the tropia patterns produced by complete left superior rectus (SR) and inferior oblique (IO) palsies. The data in this illustration were calculated by the method to be described. Its purpose at this point is only to illustrate the nature of the solutions being sought and the fact that they can be expressed in a familiar way for comparison with clinical observations.

When disorders are simple, one scarcely needs calculations to make a diagnosis. But often complex problems arise that admit to several interpretations, and differential diagnosis could be aided by the ability to mathematically simulate the consequences of any imaginable type of pathology. Since muscle insertions can easily be relocated in the analysis, one can also try to predict the magnitudes of the corrections obtained by any type of surgical procedure. The experienced surgeon is well aware of the pitfalls in diagnosis and the variability that can occur in response to surgery and some will feel that, because of this, a mathematical solution would be a waste of time. If surgical correction by current methods were totally effective, it would be. But they are not and a review of the literature indicates a deep-seated feeling in this discipline that its problems should be amenable to mechanical analysis.

The purpose of this report is to propose a model, or mathematical method, rather than a verified technique suitable for immediate use in the clinic. The emphasis here is on the theoretical aspects of the influence of the mechanics of the muscles and their attachments on eye position and deviations. However, the model appears to have a very real potential of being developed into a useful clinical tool. What is needed for future research is a refinement of the model, in the clinic, so that it can predict the deviations produced by simple, well-understood disorders, not only qualitatively but with sufficient quantita-
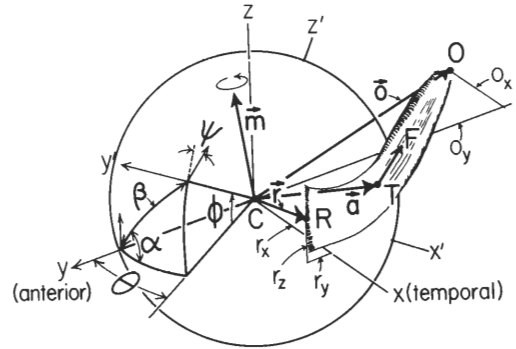

Fig. 2. Coordinates and muscle vectors for the left eye. The primary position of gaze is along the + y axis. The horizontal component of eye rotation is the angle Θ, the vertical component is the angle ϕ. Torsion of the eye, ψ, is measured from the local vertical meridian. Each muscle has an origin, O, and is inserted on the globe at some point, R. A vector from the center of the globe, C, to O is the origin vector $\bar{o}$ with coordinates o_x, o_y, and o_z, A vector from C to R is the insertion vector $\bar{r}$ with coordinates r_x, r_y, r_z. The muscle takes some path from R to O and leaves the globe's surface at T, the point where muscle force F acts. The vector $\bar{a}$ from C to T is the moment arm of the muscle. The moment exerted on the eye is directed along the unit moment vector $\bar{m}$ which is perpendicular to both $\bar{a}$ and $\bar{o}$. Axes x, y, and z are fixed in the orbit; x', y', and z' move with the eye. Angles α and β, used in the Appendix, are associated with Listing's law.

tive accuracy that it can be reliably extended to more complicated situations. The model is presented in its present form to make it available to others who may wish to explore its use in a variety of specific clinical situations. The reader should also be warned that, because of the enormous amount of calculations involved, this method requires the use of a digital computer. However, the calculations to be described have been written as a computer program which I would be happy to make available to anyone interested.

Methods

The following is a simplified account of the steps needed to solve this mechanical problem. A complete mathematical treatment is given in Appendix I. There are two parts to the problem. The first is the innervation problem: it assumes that the eye is normal, some eye position is specified and we are to find the six muscle innervations needed
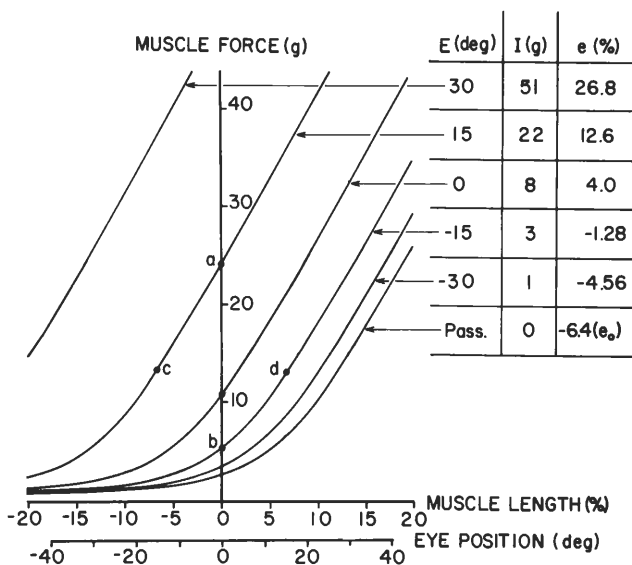
Fig. 3. Length-tension curves of a detached human extraocular muscle for various innervation levels. Muscle length was measured in millimeter changes from the primary position length and converted to per cent change for the lateral rectus using the muscle length values in Table II. These curves represent the mean behavior of five muscles studied. E is the angle, left and right of zero, of the target lamps viewed by the intact eye. The innervation, I, is taken as the isometric developed tension at primary position length which is a measure of the neutral activity created by the gaze angle effort E. The variable e, an alternate way of expressing innervation for calculation purposes, is described in the Appendix.

to hold it there. The second is the position problem: it assumes the innervations are known but the eye is now made mechanically abnormal and the deviation in eye position that this will cause is to be found.

The innervation problem. Assume the eye moves from the primary position horizontally by the angle Θ and vertically by ϕ (Fig. 2). From Listing's law, one can find the angle of torsion, ψ.[4] The eye will stay in this position (Θ, ϕ, ψ) only if all the forces acting on it add up to zero.

Force balance equation. To determine the forces, or moments in this case, one starts with the location of each muscle's origin and insertion. These data, determined by Volkman[5] and modified by Helmholtz,[6] appear in Table II in the Appendix. Each muscle goes from insertion to origin along some path across the globe's surface and leaves it at some point, T (Fig. 2). The muscle acts on the globe as though it were inserted there. The force, F, exerted by the muscle is directed from T to the origin O. Therefore, the moment created by this force is a vector perpendicular to the plane formed by C, T, and O and directed in the sense of a right-hand screw. The unit action vector, $\vec{m}$, is directed along this axis which is the one about which the globe would rotate if acted upon by F alone. This vector is important because its co-

ordinates, m_x, m_y, and m_z indicate the relative amounts by which that muscle's force is distributed to act on the globe vertically, in cyclotorsion, and horizontally, respectively.

The vector $\vec{m}$ only indicates the direction of the moment whose magnitude is F. Therefore, the moment on the globe created by each muscle is F $\vec{m}$. (Actually, this should be multiplied by the radius of the globe but since that is common to all terms, it has been left out for simplicity.) There are seven moments in all, one from each of the six muscles and a seventh, the passive moment, $\vec{P}$, created by the combined nonmuscular tissues (e.g., Tenon's capsule, optic nerve, suspensory ligaments) which act to return the globe to a mechanical neutral point near the primary position. Thus, the force-balance equation is:

$$\vec{P} + F_1\,\vec{m}_1 + F_2\,\vec{m}_2 + --- + F_6\,\vec{m}_6 = 0. \quad (1)$$

The problem, now, is to find the innervation required for each muscle to satisfy this equation so that the eye will remain in the position (Θ, ϕ, ψ).

Muscle force. The force of a muscle, in the steady-state, depends only on its length, L, and its innervation, I. Thus, it may be written F (L, I). This relationship has been measured experimentally[3, 7-9] for several human horizontal recti during

corrective surgery. Briefly, the horizontal recti were detached under a topical anesthesia and one of them (usually the lateral rectus [LR]) was attached to a strain gauge to measure isometric muscle tension, F. The gauge could be moved to change length, L. Innervation, I, was changed by the patient in looking at an array of target lamps with the other eye which, by Hering's law, would cause a neural signal to be sent to the muscle under study that would have turned it by the same amount as the eye which could move. This neural traffic cannot be measured directly but it can be measured indirectly by the tension it produces. As shown in Fig. 3, each level of innervation, or nervous effort, creates a state defined by a length-tension relationship which moves up or down as innervation is changed. We can measure innervation by the force it creates, if we agree on a fixed L at which to measure it, in this case the primary position, muscle length, L_p. Therefore, I can simply be defined as the isometric developed force of a muscle if it were set at its primary position length. (Developed force is total force less passive force.) Thus, I, although it is measured in grams, is always a direct measure of the intensity of the neural message coming to a muscle.

Individual graphs of F (L, I) have been published.[3, 7, 9] The one shown in Fig. 3 is an average of all results to date provided by C. C. Collins and D. M. O'Meara of the Smith-Kettlewell Institute of Visual Sciences, San Francisco, where the experiments were done. The averaged data were fit with a mathematical function for purposes of calculation. The curves have also been normalized so that they apply to all other extraocular muscles. The length-tension curves of a muscle are determined not by length or even change of length, ΔL, from some reference length such as L_p in this case, but by the change in length, Δl, expressed as a per cent of L_p. The strength of a muscle is proportional to its cross-sectional area. The ratio of strength compared to the LR is λ. The values of λ and L_p as reported by Volkmann[5] are given for each muscle in Table II in the Appendix. Consequently, the curves in Fig. 3 are expressed as F (Δl, I) and the actual force of any particular muscle is λF (Δl, I).

Therefore, Equation 1 may be rewritten:

$$\vec{P} + \sum_{i=1}^{6} \lambda_i \, F \, (\Delta l_i, \, I_i) \, \vec{m}_i = 0.$$

(2)

The problem now is to find the passive tissue moment, $\vec{P}$, and, for each muscle, its unit action vector, $\vec{m}_i$, and length change, Δl_i. All this may be done, as we will show, once one knows eye position Θ and ϕ. Then one must find those values of innervation, $I_1, I_2, \ldots, I_6$ which will make the total moment in Equation 2 equal to zero. One then has the desired solution.
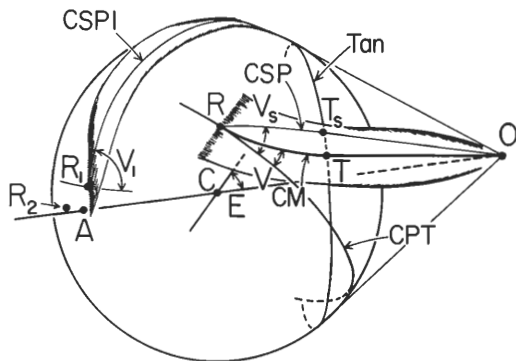

Fig. 4. Possible paths a muscle could take from insertion to origin O. R_1 and R_2 are possible locations of the LR muscle insertion near its antipodes point, A, which lead to absurd muscle locations, such as CSP1, if the muscle is allowed to take the shortest path to O. R is a more general insertion location. Great circle CPT is the track on the globe's surface that the tendon took in the primary position. Great circle CSP is the great circle path the muscle would take if it took the shortest path. CM is a nongreat circular path proposed as a more realistic path than CSP. V_s is the tendon twist angle demanded by the shortest path. V is a proposed, permitted, twist angle. Tan is the tangency circle of O. All muscles leave the globe's surface on their way to O when they cross circle Tan (e.g., points T, T_s). E is the angle between the line from C to O and a perpendicular to the plane of CPT.

Muscles do not take the shortest path. The path taken by the muscle over the globe will determine Δl and $\vec{m}$. Previous analyses[1, 2] have assumed this was the shortest path which is a great circle. That cannot be correct. In Fig. 4, a line from the origin, O, extended through the globe's center comes out the other side at point A, the point of antipodes. When the eye adducts 36.3°, the LR insertion is at its antipodes point. If the insertion now elevated slightly (R_1 in Fig. 4), the shortest path lies over the top of the globe (CSP1, Fig. 4). The LR would be required to slide a quarter of the way around the globe where it would become a pure elevator. In this position the tendon is twisted by 90° (V_1, Fig. 4) with respect to its line of insertion. Obviously this does not happen. To drive the point home, when the eye adducts by, say, 45° (R_2, Fig. 4), the shortest path lies across the cornea, the LR would be expected to slide half way around the globe and adduct the eye! Thus, it is very clear that, in any mathematical model, one cannot allow the muscles to take the shortest (great circle) path.

Although the LR is the only muscle that can reach its antipodes over the oculomotor range of ± 40°, the inferior rectus (IR) comes within 11°,

and the SR and IO come within 15° of it and create similar situations. Clearly, there are elastic restraints on the muscles (e.g., between Tenons capsule and the sclera toward the back of the globe) that prevent undue side-slipping. No physiologic data exists to describe these restraints and it is necessary to guess how they will act to shape the actual path taken by the muscle. To do this it is necessary to obtain some measure of the force acting to twist the muscle sideways and what twisting that will create on the tendinous insertion.

Tendon twist angle and twisting force. Tendon twist is the angle, V (Fig. 4), through which the tendon fibers move, relative to their line of insertion, as the eye moves from the primary to any other position. In the primary position, it is assumed that the muscle path is a great circle and that the line of insertion is perpendicular to it. Imagine that this path, the primary track, is marked on the globe's surface. When the eye moves, it carries the insertion to the point, R (Fig. 4), and with it, the primary track into circle CPT. The shortest path is the great circle CSP (Fig. 4) which lies in the plane containing C, R, and O. If the muscle took this path the tendon fibers would twist through the angle V_s. The muscle would have side-slipped from path CPT to CSP.

This is unrealistic and a simple solution would be to choose some path, CM, which demanded a twist angle, V, that was only some fraction of V_s. However, this is too simple because, as an insertion passes through antipodes, V_s jumps by 180° and V would also jump, by a lesser amount, which would still allow unphysiologic behavior. The twisting force must also be considered. This can be best visualized by imagining that the globe stays still while the origin moves relative to it. When the origin lies in the primary plane (that is, the plane of the primary track, CPT) the muscle's force will press it radially onto the globe's surface and none of the force will be directed sideways. Consequently, V should always be zero in this case and that includes the situation at or beyond antipodes. When the origin O moves off the primary plane (CPT, Fig. 4), then there is a force acting sideways to pull the muscle away from CPT. The greater the angle between the line from C to O and the primary plane, the greater this twisting or side-slipping force will be. E is the complement of that angle (Fig. 4) so that [cos E] is an index of how much each muscle's force will go into producing side-slipping.

In this study then, the angle, V_s, was used as an index of what the twist would be ideally and [cos E] as an index of how much of that value should be realized. So, the actual twist angle, V, allowed for each muscle was:

$$V = [\cos E]\, V_s. \tag{3}$$

Thus, V is always less than V_s and, near antipodes where E is 90°, V is close or equal to zero. This gave what seemed to be reasonable results, especially near antipodes. It is obvious that Equation 3 is a gross oversimplification. However, even if we knew the mechanics of the elastic restraints, the problem of using continuum mechanics to determine the actual muscle path and twist angles would require a volume of mathematical calculations disproportionate to the probable improvement in the final results. Still, a proper mechanical attack on this problem would certainly be worth doing.

The muscle path. Just as for the twist angle, the actual path over the globe's surface is unknown. In order to proceed, we shall assume it is an arc of a (nongreat) circle because such a path is manageable without unduly complicated mathematics and yet has the reasonable property of changing directions in a smooth, continuous way. The circle is determined by two boundary conditions. The first is that it pass through R making the angle, V, with CPT (Fig. 4). The second concerns the point, T, where the muscle leaves the globe. The circle Tan is the locus of all points where a straight line to O is tangent to the globe. Thus, the muscle, bound for O, will always leave the globe as it crosses Tan. Therefore, the circle CM should cross Tan at right angles to avoid any sudden change in the direction of the muscle just as it leaves the globe. These boundary conditions uniquely determine the muscle path, CM.

Change in muscle length. The muscle length in contact with the globe is the arc length of the circle CM between R and T (Fig. 4). This length is also found for the primary position. The difference is the change in muscle length, ΔL, in millimeters. This is converted to Δl, the length change expressed in per cent of L_p for each muscle. Because muscles do not take the shortest path, values of ΔL will reflect muscle lengths longer than those calculated by Boeder.[1]

Unit action vector. Once the circle CM is found, it is easy to find point T. The muscle leaves the globe at T (Fig. 4) so it acts as if it were inserted there. It will act to rotate the globe around an axis perpendicular to the plane containing C, T, and O. The vector, $\vec{m}$, can be found as a unit vector directed along that axis. Again, because muscles do not take the shortest path, these vectors will differ from those calculated by Krewson.[2]

Passive moment. To find $\vec{P}$, one uses Listing's law. Any rotation to a point (θ, φ) in Fig. 2 appears to take place as a single rotation in a plane (Listing's plane)[4] containing the visual axis in the primary position (+ y axis) and in the new position (y′, Fig. 2). The passive tissues will act to return the globe to the primary position so that $\vec{P}$ will be a vector perpendicular to Listing's plane.

The rate at which the amplitude of $\vec{P}$ increases with the amplitude of the rotation has been determined experimentally.[3, 8] The only modification is that the mechanical neutral point of the passive tissues has been shifted slightly (7.5°) temporally to produce the mild abduction seen under deep anesthesia and in death.

Reciprocal innervation. We have now found $\vec{P}$, Δl, and m for all six muscles in Equation 2. We must now find the six unknown innervations. Equation 2 is a vector equation so it really represents three equations, one for each of the x, y, and z components. If there were six unknowns, I_1, . . ., I_6, they could not be solved. But muscles act in antagonistic pairs; they obey Sherrington's law of reciprocal innervation and do not normally cocontract. The values of I in Fig. 3 allow us to describe that law numerically for extraocular muscles. When the patient attempts to abduct from 0° to 15° and 30° the LR innervation goes from 8 to 22 to 51 g., respectively. The antagonist medial rectus (MR) simultaneously must decrease its innervation from 8 to 3 to 1 g. because its innervation should correspond to that of the LR when it attempts gaze deviations of −15° and −30°. Thus, the LR innervation may be plotted against presumed MR innervation, and the curve fit with a mathematical expression for computational purposes. It is assumed (there is no evidence to the contrary) that this relationship is a fixed property of the central organization and does not change just because the eye, for example, is in a tertiary gaze position. Consequently, if one knows I for the LR, SR, and SO (superior oblique) one can then calculate I for their antagonists, the MR, IR, and IO. Therefore, in Equation 2, there are really only three unknowns, I_1, I_3, and I_5.

Solving for I. Equation 2 is a set of three non-linear equations in three unknowns. They are solved on the computer by trial and error. One makes an initial guess at I_1, I_3, and I_5 and, performing all the above calculations, determines the value of the left side of Equation 2. This is the total moment on the globe, $\overline{M}$. If it is zero, one has the correct solution. If it is not, a small-signal linearization is used involving the partial derivatives $\partial F/\partial I$ (see the Appendix) to find a better estimate for I_1, I_3, and I_5. The entire process is then repeated again and again until $\overline{M}$ is negligibly small.

One now has the desired solution and, along with the innervations, one also has determined all the muscle forces, lengths, and action vectors. Thus, one knows the complete mechanical state of the system for that eye position. Two details should be mentioned. When the eye adducts beyond 34° the MR loses its contact arc with the globe. It is the only muscle that does this over the range ± 40° for Θ and ϕ. When that happens the muscle is no longer tangent to the globe and

the effective decrease in the moment arm must be calculated. Finally, all vector calculations are performed in a system of axes (x, y, z, Fig. 2) fixed in the orbit. But when we say, for example, that, in adduction, the SO depresses the eye, we mean with respect to the reference frame of the eye itself (x', y', z', Fig. 2) not the orbit. So, it is useful to transform important vectors like $\vec{m}$ into a reference frame that moves with the eye where they are designated as, for example, $\vec{m}'$. All the above calculations, including print-out, require no more than about 10 seconds for each gaze position depending on the computer used.

The position problem. To study ocular deviations, we assume that one eye is normal and fixation with it establishes a normal innervation, I_1, . . ., I_6 (found by the above calculations) in the abnormal eye under cover. One may now deliberately alter the innervations to simulate over- or under-action. By altering F (Δl, I), a muscle may be strengthened or weakened, its length-tension relationship made abnormal, or its passive force may be altered to simulate fibrosis. The muscle insertion point (Table II, Appendix) may be recessed, resected, or moved in any other way. Any other abnormality one can think of may be introduced so long as it can somehow be mechanically described. The problem now is to calculate the abnormal eye position created by this pathology for several attempted gaze positions, usually 30° horizontally and vertically in all combinations for plotting in a fashion similar to a Hess chart as in Fig. 1.

In Equation 2, I_1, I_2, . . ., I_6 are given, but $\vec{P}$, $\vec{m}$, and Δl, which are all determined by eye position Θ, ϕ, and ψ, are unknown. Again, one makes an initial guess of eye position (usually the orthophoric position) and proceeds to calculate all the terms on the left side of Equation 2. Their sum, $\overline{M}$, is the total moment acting on the globe. Its components indicate the amplitude and direction by which this moment is trying to rotate the globe. This allows one to make a better guess for Θ, ϕ, and ψ. The entire process is then repeated until $\overline{M}$ becomes negligibly small. One then has the solution; the new eye position created by the disorder.

The only change is that in this case Listing's law no longer applies. If ψ does not have the Listing value, there is true torsion and a component must be added to $\vec{P}$ to account for the passive tissue reaction to this twist about the line of sight.

Results

The innervation problem was solved for all horizontal and vertical gaze positions over the range ± 40° in 10° steps. From these 81 computations, constant-innerva-
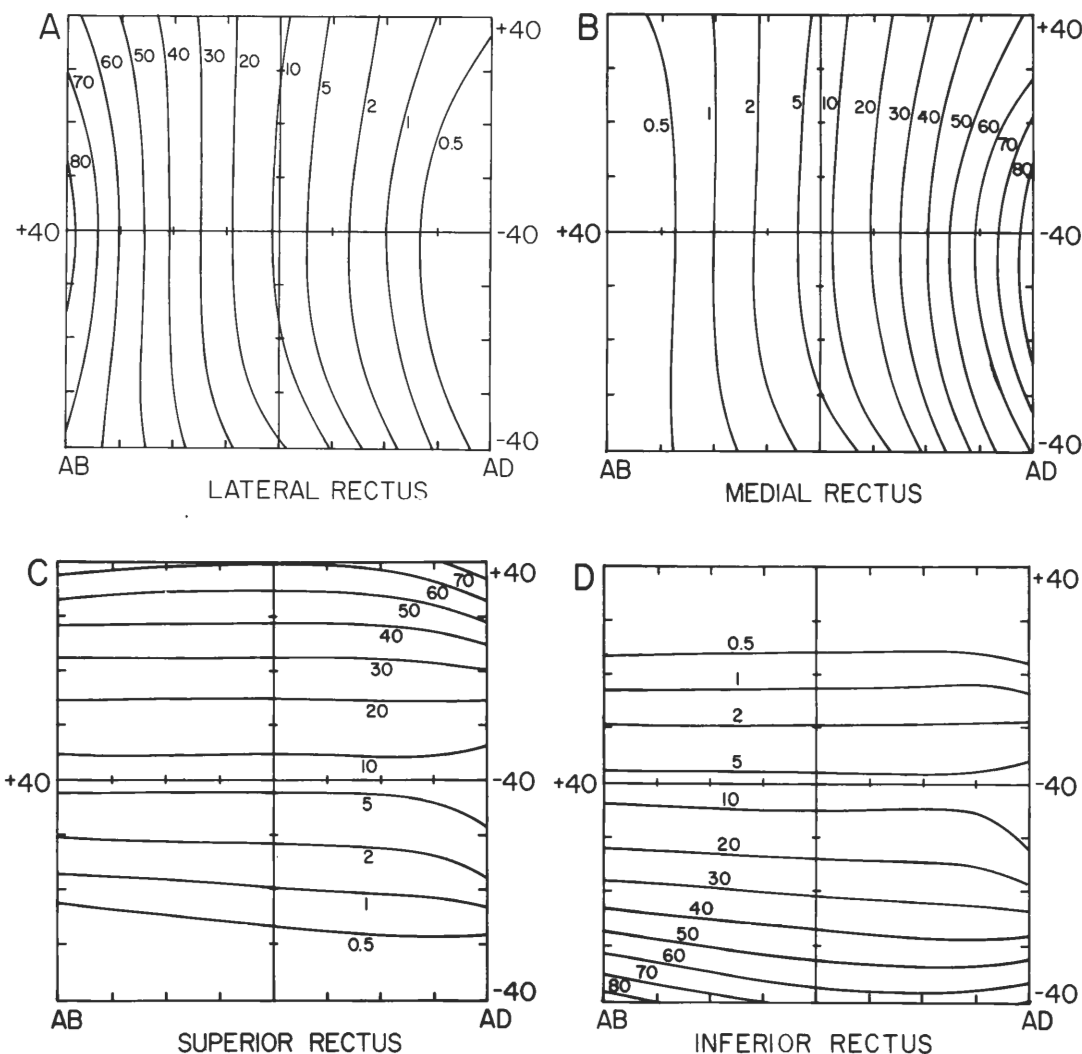
Fig. 5. The behavior of muscle innervation, I, for all six extraocular muscles over the field of gaze out to 40° horizontally and vertically. The contours are equi-innervation lines. Along them, the innervation is constant and its value in g. is indicated next to each contour.

tion contours were plotted over the field of gaze for each muscle. They are shown in Figs. 5, A through F. Table I contains more specific details of each solution such as lengths, forces, and action vectors and how moments break down for muscle pairs for the six interesting positions of 30° abduction and adduction with and without 30° elevation.

One test of the model is to compare the values of I it predicts for the LR in the primary position and for 30° abduction and adduction with the values measured

experimentally in Fig. 3. The curves in Fig. 3 were measured to determine the intrinsic relationship between F, Δl, and I for a muscle and this relationship was used in the model without any reference to the fact that the data applied to any particular gaze effort in degrees. The model would predict I correctly at any eye position only if it correctly estimated the mechanical loads on the LR due to the passive tissues and the other five muscles. For horizontal gaze angles of +30°, 0°, and –30°, the experimental values of

I (Fig. 3) were 51, 8, and 1 g. The predicted values (Table I) were 59.8, 8.9, and 0.3 g. Considering the accuracy of the raw data, the averaging over several patients and the conversion of eye position in Fig. 3 to muscle length change by a fixed globe radius, the agreement is fairly good and suggests that the model should not be too far out in its quantitative predictions.

Fig. 5 shows that, in the primary position, muscle tone (innervation) is about 8 g. for the horizontal recti, 6 g. for the vertical recti, and 4 g. for the obliques. Innervation rises smoothly to about 84, 65, and 40 g., respectively, when the eye is turned about 40° into each muscle's field of action and drops to almost zero in the opposite direction.

The contours for the horizontal recti (Figs. 5, A and B) are as one might expect; near the primary position they are vertical; that is, these muscles do not parcipate in vertical gaze. But, when the eye first elevates and then moves horizontally, the innervation changes less because the amount by which the muscles must shorten is less (by approximately the cosine of the angle of elevation). In Table I, for example, the LR shortens by 6.5 mm. on 30° abduction. While in 30° elevation, it shortens only 5.5 mm. (6.6 less 1.1 mm.) for 30° abduction. This simple geometric factor accounts for the outward curvature of the contours in Figs. 5, A and B in tertiary gaze positions.

The behavior of the vertical muscles (Figs. 5, C through F) is not so simple. For example, one might expect the SR to have more innervation on up gaze in abduction than in adduction but, in fact, the opposite is true. The reason that these contours are not as simple as those for the horizontal recti is that muscles interfere with each other. Whenever the eye moves, muscle pairs are tilted out of their normal planes of action and develop new actions in the planes of the two other muscle pairs. This cross-coupling constitutes an interference which would push the eye away from the

Fig. 5, E and F. For legend, see opposite page.

desired position unless the other muscles readjusted their innervation to compensate. Thus, the muscles spend part of their effort fighting each other.

The horizontal muscles cross-couple onto the vertical muscles more than the other way around (see below) and the latter are also weaker. Thus, the vertical muscles are more affected by cross-coupling. These effects are nonlinear and depend in peculiar ways on eye position. Consequently, the contours in Figs. 5, C through F are distorted in ways that seem impossible to predict by intuition.

This study offers the first opportunity to quantitatively observe all these interactions at once. They are worth a closer look because they can become quite large in pathologic situations and an appreciation

Table I. Details of the mechanical state of the eye for 30° abduction and adduction tendon twist angle if the muscle were allowed to take its shortest, great-circle path; V, the in muscle length in mm.; Δl, change in muscle length in per cent of L_p; I, innervation; strength relative to the LR;* m'_x, fraction of a muscle's force acting vertically (+ down, fraction acting in torsion (+ intorsion, – extortion); m'_z, fraction acting horizontally muscle force; Paris, x', y', and z', the sum of F times the appropriate value of m' for x', y', and z' (e.g., Paris, x' = $F_1 m'_{1x} + F_2 m'_{2x}$ for the LR and MR). The total passive fraction distribution in x', y', and z'. Force balance is shown by the fact that the forces, each row. (The slight failure to do so reflects the fact that the solution was considered than about 0.1 g.)

ABDUCTION–ELEVATION θ = + 30°, φ = + 30°

	LR	MR	SR	IR	SO	IO	P
V_B (deg)	33.2	-36.0	-28.4	30.3	-12.8	39.0	
V (deg)	16.7	-18.0	-13.5	6.6	-2.8	23.2	
ΔL (mm)	-6.6	4.3	-5.6	7.8	2.1	-5.5	
Δl (%)	-13.4	11.2	-13.3	18.3	9.4	-15.5	
I (g)	53.5	0.5	42.8	0.2	0.1	28.9	
I/λ (g)	53.5	0.5	62.9	0.2	0.2	61.5	
m'_x	-0.21	-0.27	-1.00	0.95	0.35	-0.45	0.80
m'_y	0.50	-0.48	0.00	-0.14	0.91	-0.74	0.06
m'_z	0.84	-0.84	-0.05	-0.28	0.21	0.50	-0.60
F (g)	32.6	14.4	28.6	23.2	5.3	17.3	28.7
Pairs, x' (g)	-10.70		-6.45		-5.88		22.97
Pairs, y' (g)	9.47		-3.50		-7.90		1.83
Pairs, z' (g)	15.38		-7.97		9.79		-17.14

ELEVATION θ = 0, φ = + 30°

	LR	MR	SR	IR	SO	IO	P
V_B (deg)	43.5	-26.0	-3.3	-17.5	8.3	22.2	
V (deg)	20.4	-11.3	-0.2	-3.1	1.2	7.5	
ΔL (mm)	-1.1	-1.0	-5.8	5.2	4.1	-4.0	
Δl (%)	-2.3	-2.6	-13.7	12.3	18.6	-11.2	
I (g)	10.7	6.2	41.8	0.3	0.2	27.4	
I/λ (g)	10.7	6.0	61.5	0.3	0.4	58.3	
m'_x	-0.18	-0.25	-0.89	0.75	0.68	-0.53	0.98
m'_y	0.56	-0.37	0.36	-0.58	0.71	-0.82	0.10
m'_z	0.81	-0.89	-0.27	-0.33	0.16	0.22	0.17
F (g)	10.2	6.3	27.1	14.0	12.7	19.3	19.0
Pairs, x' (g)	-3.41		-13.72		-1.50		18.63
Pairs, y' (g)	3.44		1.56		-6.83		1.83
Pairs, z' (g)	2.65		-12.00		6.17		3.17

ABDUCTION θ = + 30°, φ = 0°

	LR	MR	SR	IR	SO	IO	P
V_B (deg)	-0.6	-0.3	-33.7	35.1	-24.2	28.8	
V (deg)	0.0	0.0	-16.1	16.7	-9.3	10.5	
ΔL (mm)	-6.5	6.5	1.0	1.1	-1.6	-0.8	
Δl (%)	-13.2	16.9	2.4	2.6	-7.1	-2.2	
I (g)	59.8	0.3	6.4	6.6	5.1	3.0	
I/λ (g)	59.8	0.3	9.4	6.9	10.2	6.4	
m'_x	-0.01	-0.01	-0.99	0.99	0.18	-0.49	0.0
m'_y	0.01	-0.02	-0.07	0.06	0.94	-0.83	0.0
m'_z	1.00	-1.00	-0.09	-0.11	0.29	0.26	-1.0
F (g)	39.0	23.3	10.7	12.7	2.5	3.1	15.0
Pairs, x' (g)	-0.82		1.95		-1.12		1.0
Pairs, y' (g)	0.16		0.10		-0.26		0.0
Pairs, z' (g)	15.76		-2.34		1.53		-14.96

PRIMARY POSITION, θ = φ = 0°

	LR	MR	SR	IR	SO	IO	P
V_B (deg)	0.0	0.0	0.0	0.0	0.0	0.0	
V (deg)	0.0	0.0	0.0	0.0	0.0	0.0	
ΔL (mm)	0.0	0.0	0.0	0.0	0.0	0.0	
Δl (%)	0.0	0.0	0.0	0.0	0.0	0.0	
I (g)	8.9	7.6	6.4	6.6	3.9	3.9	
I/λ (g)	8.9	7.3	9.4	6.9	7.8	8.3	
m'_x	-0.02	-0.01	-0.87	0.86	0.57	-0.63	0.0
m'_y	0.02	-0.01	0.40	-0.41	0.80	-0.77	0.0
m'_z	1.00	-1.00	-0.29	-0.32	0.17	-0.11	1.0
F (g)	11.7	10.4	8.3	9.2	5.3	5.2	3.7
Pairs, x' (g)	-0.30		0.68		-0.30		0.0
Pairs, y' (g)	0.14		-0.40		0.22		0.0
Pairs, z' (g)	1.26		-5.34		0.33		3.67

Blue := signs corrected by JMM

of their sources is important to anyone trying to understand how extraocular muscles cooperate (or interfere) with each other. It also raises the issue of how a muscle's length, force, innervation, and unit action vector are related to each other and in what way they represent its participation in a given movement.

To gain some insight into this problem, it is convenient to divide a muscle's activity into three components. The first is the innervation needed by a pair of muscles to turn the globe in its plane of action if there were no other sources of interference; that is, if only those two muscles were attached to the globe. I call this the primary innervation. There are two main sources of interference. One is the passive tissue force which tries to pull the eye back toward zero. Innervation shifts required to overcome this force I call secondary innervation. The other is the combined results of cross-coupling from other muscle pairs which I call tertiary forces. The shifts needed to offset these disturbances I call tertiary innervation.

Primary innervation. Because of their length-tension relationships, muscles act like springs. Two antagonistic muscles act like a pair of springs in opposition. If free to do so, the eye will go to the equilibrium position where their forces are equal and opposite. Imagine that two antagonist muscles, each like that in Fig. 3, are attached to the eye. When both receive primary position innervation, each is on the length-

with and without 30° elevation. V_s, the actual permitted twist angle; ΔL, change I/λ, innervation corrected for muscle – up) in the eye's coordinate system; m'_y, (+ abduction, – adduction); F, total pairs of muscle in the action directions force P is given with its actual and Paris x', y', and z', add up to zero across adequate if residual forces were less

ADDUCTION-ELEVATION θ = – 30°, φ = + 30°						
	LR	MR	SR	IR	SO	IO	P
V_s (deg)	80.5	–23.1	21.3	–45.1	32.4	6.5	
V (deg)	35.0	– 8.6	7.6	–26.1	16.9	0.7	
ΔL (mm)	3.2	– 6.7	– 7.3	1.3	5.6	– 4.7	
Δℓ (%)	6.5	–17.5	–17.5	3.1	25.1	–13.3	
I (g)	0.7	45.1	45.8	0.2	0.0	40.1	
I/λ (g)	0.7	43.4	67.4	0.2	0.0	85.3	
m'_x	– 0.21	– 0.27	– 0.62	– 0.32	0.91	– 0.60	0.71
m'_y	0.77	– 0.31	0.65	– 0.82	0.40	– 0.80	0.05
m'_z	0.61	– 0.91	– 0.44	– 0.48	0.04	– 0.01	0.70
F (g)	8.6	16.9	26.5	4.1	17.4	30.2	33.5
Pairs, x' (g)	– 6.36		–15.24		– 2.29		23.88
Pairs, y' (g)	1.38		13.85		–17.15		1.83
Pairs, z' (g)	–10.20		–13.58		0.31		23.43

ADDUCTION θ = – 30°, φ = 0°						
	LR	MR	SR	IR	SO	IO	P
V_s (deg)	7.0	0.0	29.0	–29.3	24.7	–24.9	
V (deg)	0.1	0.0	13.7	–13.8	10.2	– 8.6	
ΔL (mm)	6.5	– 6.5	– 2.6	– 2.8	0.6	– 2.0	
Δℓ (%)	13.2	–16.9	– 6.2	– 6.5	2.7	– 5.7	
I (g)	0.3	61.8	7.0	6.0	1.9	7.5	
I/λ (g)	0.3	59.4	10.3	6.3	3.8	16.0	
m'_x	– 0.01	– 0.01	– 0.53	0.51	0.87	– 0.67	0.0
m'_y	0.04	– 0.01	0.73	– 0.72	0.50	– 0.60	0.0
m'_z	1.00	– 1.00	– 0.43	– 0.46	0.04	– 0.43	1.0
F (g)	16.5	33.6	3.9	3.5	4.7	4.8	22.3
Pairs, x' (g)	– 0.59		– 0.29		0.87		0.00
Pairs, y' (g)	0.20		0.32		– 0.53		0.00
Pairs, z' (g)	–17.16		– 3.29		– 1.85		22.29

tension curve marked 0°, the equilibrium point is at 0°, and each muscle exerts 10.8 g. If the innervation suddenly changes corresponding to an attempted shift of 15°, the agonist muscle would momentarily rise to 24 g. (point a, Fig. 3), the antagonist would fall to 5 g. (point b). When the eye moved to its new equilibrium position 15° away, the agonist would shorten by about 6.5 per cent and its force would drop to about 13 g. (point c, Fig. 3). The antagonist would lengthen by the same amount and its force would rise to 13 g. (point d). Thus, both muscles not only come to the same force (as they must for balance) but that force is not much different from the value they exerted at 0°. Innervation, on the other hand, clearly reflects the gaze

shift. The agonist innervation rises from 8 to 22 g. (Fig. 3) while the antagonist falls from 8 to 3 g.

Two points are clear. Actual muscle force is a poor indicator of what a muscle is trying to do. We tend to associate innervation with a rise in tension which is only correct if a muscle is isometric. We tend to forget about the length-tension relationship. In this example, force is nearly independent of eye position over the range ± 15°. In general, the force increases in the antagonist as well as the agonist (see any muscle, Table I) although not as much. Force is needed to counteract passive tissue and tertiary disturbances to achieve a force balance but, by itself, it is only poorly related to eye position or primary innervation. The second point is that eye position and primary innervation are closely and directly related because, as in the analogy of the springs, the essential feature of a change in innervation is not the force *per se* but the change in equilibrium *position*. (However, it is the differential length change of a pair of muscles [half the difference between the two length changes] that is important, not the change in any one muscle alone. For example, when the eye elevates 30° [Table I] both the LR and MR shorten about 1.0 mm. but these muscles do not shift their primary innervation.)

In short, for any rotation of the globe, muscle force is a poor index of muscle participation, primary innervation is a good index, almost by definition, and differential muscle shortening is also a good index as emphasized by Boeder.[1] Since shortening is greatest when the eye rotates around the unit action vector, that vector, as emphasized by Krewson[2] is also a good index. All of this is simply a restatement of the description of the modes of action of the muscles found in any textbook.

Secondary innervation. Passive tissue force rises by about 0.48 g. per degree for rotation in any direction away from the mechanical neutral point of the tissues. It was found by trial and error that, to account for the slight but persistent diver-

gence (about $3°$)[10] that occurs when innervation in all muscles was reduced to zero (as in death), it was necessary to move that neutral point $7.5°$ temporally. Except for this slight complication, the tissue force acts on each pair of muscles in proportion to the component of the eye deviation in their plane of action. This requires a compensatory increase in agonist innervation and a drop in antagonist innervation. Reciprocal innervation always affects the former more than the latter in eccentric gaze so the load is mostly picked up by the agonist.

This is illustrated most simply in Table I by the horizontal recti in $30°$ abduction where the vertical muscles present very little disturbance (about -1 g., the sum of Pairs z' of the vertical recti and obliques) and the major disturbance is 15 g. of adduction from the passive tissues (P). This must be offset by the LR exerting 16 g. more than the MR. Thus, LR innervation (I, 59.8 g.) is made up of about 44 g. of primary innervation and 16 g. of secondary innervation. The effect of secondary innervation in Fig. 5 is to shift all the contours in the direction of action of each muscle. It does not perturb the contours in ways that depend unexpectedly on eye position as does tertiary innervation.

Tertiary innervation. When a muscle pair is rotated out of its plane of action, it develops moments that act on the other two pairs. There are thus six basic modes of cross-coupling. Since these forces can occur if the eye rotates about any of three axes, there are 18 possible ways for interactions to occur. Since we are only interested in voluntary gaze, we can eliminate torsion, reducing the number to 12. Rotation in the plane of a muscle pair does not cause it to react on the other two, so the horizontal recti may be discounted for horizontal movements, reducing the total to 10.

These modes represent the most onerous problem in trying to understand muscle cooperation or, in this case, lack of it. Even with computer solutions that show them quantitatively, it is impossible to reduce the mass of data into a few intuitive rules. If one could, one would no longer need a computer to keep track of them all. These forces are small compared to primary innervation, but they are large enough to seriously distort the contours in Figs. 5, C through F.

Four of these interactions are fairly simple. When the eye elevates, the horizontal recti develop action components which allow them to elevate the eye further. I call this the bridle effect (German : Zügel) because it resembles the action on a horse's head when the reins are pulled: pulling it up when the head is up and down when it is down. In Table I, the LR develops an 18 per cent elevating action on $30°$ elevation (m'_x is -0.18: the values of m'_x, m'_y, and m'_z do not add up to 1.0 because they are vector components, but it is correct to think of them in per cent because, as in this example, for every 100 g. exerted by the LR, 18 g. goes into elevation). The MR elevates by 25 per cent. Together (Pairs, x') they elevate by 3.41 g. (They similarly depress the eye in $30°$ down gaze.) Thus, the horizontal muscles assist the vertical muscles in both directions but not by much compared to the passive force depression of 18.6 g. But note that in elevation with abduction, this bridle force has risen to 10.7 g., almost half of P. It will be shown later that in pathologic situations, this force can create large disturbances.

The bridle force effect on the SR and IO must be distributed according to their unit action vectors in the x'-y' plane. But the horizontal recti also develop torsional forces. The LR intorts 56 per cent at $30°$ elevation, the MR extorts by 37 per cent. The imbalance is 3.44 g. intortion for the pair (Pairs, y'). The vector sum of these two forces (Pairs x', y') is 5 g. of elevation with intortion, corresponding roughly to the action of the SR. Thus, the horizontal recti, for vertical gaze, interfere little with the IO and assist the SR by 5 g. This illustrates that, when considering any cross-coupling into the vertical muscles, all the x' and y' components must be combined

vectorially to see how it affects the vertical recti and obliques individually.

The other two effects are the horizontal bridle actions of the two pairs of vertical muscles in horizontal gaze. This can even be seen in the primary position. Because the SR and IR are attached to the globe in a parasaggital plane through its center (Table II, Appendix) but have origins medial to this plane, they have adducting actions of 29 and 32 per cent (m'_z, Table I). Together they adduct the eye (Pairs, z') by 5.34 g. The obliques contribute only 0.33 g. so the net horizontal disturbance by vertical muscles is 5 g. of adduction. This is not quite offset by P (3.67 g.). The difference (1.26 g.) causes a tertiary innervation adjustment in the LR (up to 8.9 g.) and MR (down to 7.6 g.).

In abduction the vertical recti bridle force drops to only 2.34 g. adducting (Table I) because these muscles act almost purely vertically (99 per cent) in this position. In adduction their bridle force also drops, to 3.29 g. adducting, because muscle shortening (6.2 and 6.5 per cent) weakens them both here. The oblique bridle force is small, helping to abduct in abduction by 1.53 g. and adduct in adduction by 1.85 g. The total effect of all vertical muscles near the primary position is simply a 5 g. adducting bias which does not depend much on eye position. This is important because, when the passive tissue spring constant, k_p, was determined experimentally,[3] the horizontal recti were detached and the horizontal force-displacement relationship of the globe measured. However, the vertical muscles were still attached and normally innervated. Their bridle effect would have had to be subtracted out of the total measurement to obtain k_p. But, since that part is negligible, the spring constant measured, 0.48 g. per degree, may be taken directly as k_p.

Two of the other six interactions occur when the eye elevates and both vertical muscle pairs exert horizontal forces (Table I, Pairs, z'). The vertical recti exert 12 g. adducting (up from 5.34 g.), the obliques 6.17 g. abducting. This must be offset by

tertiary innervation of the horizontal recti (LR from 8.9 to 10.7 g.; MR from 7.6 to 6.2 g.). Two others occur on elevation. The vertical recti and obliques interfere with each other. The vertical recti elevate and intort by 13.72 g. and 1.56 g. at 30° up. The obliques elevate and extort by 1.5 g. and 6.83 g. (Pairs, x' and y'). These forces must be resolved vectorially into SR and IO components. The vertical recti force component that acts on the SR is part of its primary innervation: only that part acting in the plane of the IO is a tertiary interference and similarly for the obliques. The result is that the vertical recti cross-couple to assist the IO by about 3.5 g., the obliques cross-couple to oppose the SR by 2 g.

Finally, when the eye moves horizontally, the vertical muscles again interact with each other. Their m'_x and m'_y values indicate what is already well known; the SR elevates better in abduction, the IO in adduction. This is only to be expected as the eye turns with respect to the planes of these muscles; what is important is whether the muscle planes move with respect to each other. The Pairs x' and y' of the vertical recti and obliques do not exceed 2 g. in either 30° abduction or adduction so, for horizontal movements, these muscle pairs only negligibly interfere with each other.

In tertiary eye positions, muscle interference often becomes worse. Such interactions generally defy intuition and we will deal with them only on an *ad hoc* basis in the next section.

The relative participation of vertical muscles in ab- and adduction. It is commonly accepted that the SR elevates in abduction and the IO elevates in adduction. This study does not suggest otherwise but Figs. 5, *C* and *D* seem to contradict this idea. Table I shows that for 30° elevation, the SR innervation is even higher in adduction (45.8 g.) than in abduction (42.8 g.). To understand muscle cooperation, it is important to see how this can happen.

From the standpoint of the central nervous system, innervation *is* participation. It is not concerned with force or shorten-

ing, it only knows what neural signal must be sent to accomplish a given task. But strong and weak, long and short muscles translate this message into quite different mechanical forces and length changes. So a muscle's innervational participation can be different from its mechanical participation.

For example, when the eye elevates 30° (Table I) the SR shortens by 5.8 mm., the IO by only 4.0 mm. This would suggest that the SR is 1.4 times more active than the IO. Their elevating components, m'_x, are also 89 per cent and 53 per cent, a ratio of 1.7. But the SR is a longer muscle than the IO (by a factor of 1.4; Table II, Appendix) so that each millimeter of shortening requires more innervation by the latter. When muscle shortening is expressed in per cent, the SR shortens by 13.7 per cent, the IO by 11.2 per cent. Using this more physiologic criterion the muscles participate almost equally.

Their actual innervations in this position are 41.8 g. and 27.4 g., a ratio of 1.5. However, the IO is a weaker muscle (by a ratio of 1.45; Table II, Appendix). One can obtain a number proportional to the per cent of maximum motor nucleus activity by scaling each innervation by that muscle's relative strength λ (Table II, Appendix). This number, I/λ (Table I) enables one to directly compare the activity, relative to maximal, of one muscle with any other. This relative innervation is 61.5 g. for the SR and 58.3 g. for the IO so that prenuclear structures must excite about the same proportion of motoneurons in each motor nucleus to elevate the eye. Thus, whether one considers relative shortening or relative innervation, to those central structures it appears that the two muscles participate equally. It may be that this is why these muscles have developed to their present size, lengths, and locations in primate evolution.

It is convenient to lump secondary and tertiary forces together in 30° up gaze to see how they disturb the elevating muscles. The passive force is 19 g., primarily depressing. The horizontal recti exert 4.8 g. equally distributed (vectorially) in elevation and intortion. The combined disturbance of the vertical recti and obliques on each other is about 4 g., extorting. Adding all these vectorially in the x'–y' plane gives a resultant of 14.5 g. of almost pure depression. This is the load to be picked up by secondary and tertiary innervations of the SR and IO. Resolving this vector into the planes of those muscles gives a required increase in SR innervation of 12 g. and an IO increase of 7 g. In short, the tertiary forces are fairly small, partially cancel each other and part of the passive load. The net depression is more or less shared, in the expected ratio, between the two elevators.

Although the relative innervations of the SR and IO are about equal, mechanically, of course, the SR is stronger. This is easily seen when it is removed. The tropias produced by a complete SR palsy ($I_3 = 0$) were calculated using the position program (Fig. 1, A). When the SR is missing, the IO can only elevate the eye 11° on 30° attempted up gaze. When the IO is missing (Fig. 1, B), the SR can elevate the eye 22°.

When the eye abducts 30° in elevation (Table I) there is a slight increase in the innervation of both the SR and IO. This seems odd since the elevating action of the SR (m'_x) has risen from 89 per cent to 100 per cent while that of the IO fell from 53 per cent to 45 per cent. However, these changes are small and it would be a great mistake to assume that the IO has substantially lost elevating power here. Still, one might have expected a larger increase in SR innervation and some drop in IO innervation.

However, because of the higher LR force, the tertiary force of the horizontal recti (vector sum of Pairs x' and y') has risen to 14.3 g., still distributed about equally in elevation and intortion. This unloads the SR and loads the IO. When all the vectors are added up, the net secondary and tertiary innervation of the SR was 7 g.

(down 5), that of the IO, 10 g. (up 3). These changes are not large but they are enough to obliterate the expected increase in SR and decrease in IO innervation upon abduction.

Fig. 1, of course, shows a simpler mechanical picture. In abduction, the SR, without the IO, can elevate the eye 29°. The IO alone can only elevate the eye 6°. By this measure, the SR is the major elevator in abduction.

In adduction and elevation, the elevating action of the SR falls to 62 per cent, the IO increases to 60 per cent. These two muscles are now about equal in this regard and it is certainly not true that the SR has lost most of its vertical action here. One would expect the IO innervation to rise and it does, from 27.4 g. to 40.1 g. But instead of falling, the SR innervation actually rises again. The major reason for this is that the SO and IO become badly misaligned in this position. Their torting actions (m'_y) differ in amplitude by 40 per cent. This causes a twisting action by the obliques which cross-couples and loads the SR by 8 g. The net secondary and tertiary disturbance vector rises to 24 g., mostly depressing, to be shared equally by the SR and IO. The final result is an extra innervation (secondary plus tertiary) by the SR of 20.5 g. (up 8.5 g. from straight elevation) and IO of 14 g. (up 7 g.). This accounts for the unexpected rise in SR activity. Note in Fig. 1 that in adduction the SR, still mechanically strong, can raise the eye 14° by itself (Fig. 1, B) while the IO, by itself, now raises the eye by 17°. Thus, Fig. 1 shows clearly that the SR does best in abduction, the IO in adduction.

This example illustrates two points. Muscles of different sizes and lengths express their relative innervations quite differently in terms of force and length change. By looking at the central (innervation) or the peripheral (mechanical) side of the neuromuscular junction, one can reach quite different conclusions about relative participation by different muscles. To avoid such apparent contradictions, one should state explicitly whether, by participation, one means innervation, length change (in per cent), muscle force (a poor index of anything), or the components of the unit action vector. None of these are uniquely indicative, they are all different aspects of an exasperatingly complex phenomenon.

The second point is that the way in which muscles interfere with each other (tertiary forces) may be small in the normal eye but they are large enough to cause unexpected behavior in the innervation patterns of the vertical muscles over the field of gaze (Figs. 5, C through F). They arise from unsuspected sources and depend in complicated ways on gaze angle. It is mostly because of them that one is forced to resort to computations of tropias by a computer.

The influence of muscle path on its action. Implicit in these calculations is the fact that muscles no longer appear to act as though they were attached at their anatomic insertions (usually taken as the midpoint of the line of insertion). It was shown in the Methods section that absurd results are obtained if muscles are allowed to take the shortest path. This can be seen in Table I: for 30° elevation in adduction, the LR would twist about its insertion line by 80.5° (V_s) if it took the shortest path. Calculations show that, if allowed to do this, its action would have been 14 per cent abduction, 53 per cent elevation, and 83 per cent intortion. If it is restrained to a twist angle of 35° (Table I), its action becomes 61 per cent abduction, 21 per cent elevation, and 77 per cent intortion. This is a large change in action, especially in abduction (14 to 61 per cent). When we say a muscle acts as though attached at some point, we really mean it acts as though it took a shortest, great-circle path from that point to its origin. When forced to take a different path, the muscle then acts as though it were attached to the globe at some other point, an effective insertion point, that can be quite far from its anatomic insertion.
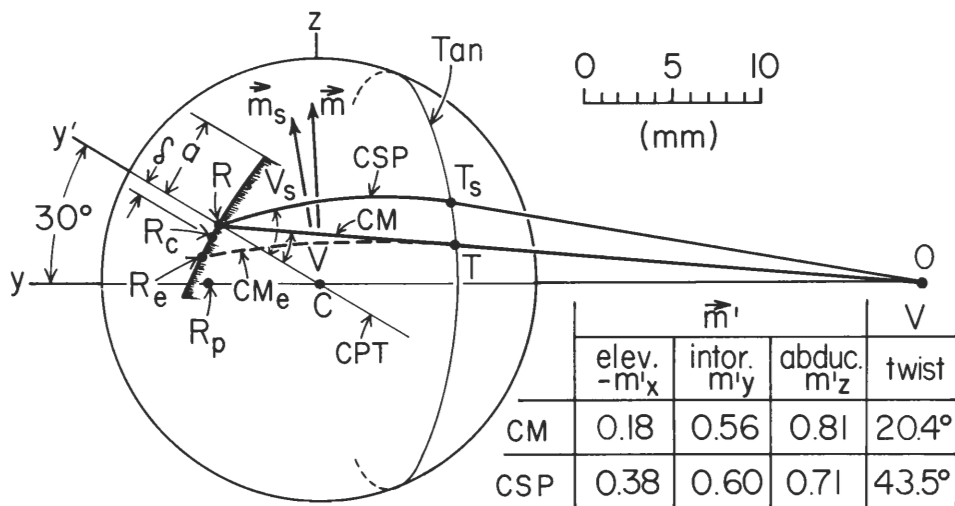
Fig. 6. The effect of tendon twist on a muscle's action. Side view, drawn to scale, of the left eye looking up 30°. The LR is inserted at R. Two possible muscle paths are shown, the shortest path CSP which has a tendon twist angle, V_s, and the path used in this study CM with twist angle, V. The path leaves the globe at point T_s or T which is the point at which the muscle effectively acts on the globe. Each path, therefore, produces a different unit action vector $\vec{m}$. The distribution of those vectors, in the eye coordinate system, is shown in the inset table. CM_e is a great circle extension of the path O-T. It intersects the insertion at R_e, the point where the muscle appears to be effectively inserted. The length of the insertion is 2a. R_e is the centroid; that point at which a twisted tendon effectively exerts all its force. R_p is the insertion point in the primary position.

This is shown more exactly in Fig. 6. If the LR, in 30° elevation, took the shortest path, its tendon fibers would twist (V_s) by 43.5° and its action would be 71 per cent abduction, 38 per cent elevation (bridle effect), and 60 per cent intortion. When restricted according to equation 3, the twist angle (V) is only 20.4° and the muscle's action is 81 per cent abduction, 18 per cent elevation, and 56 per cent intortion (Table I). It now acts as though it were inserted at an effective point, R_e, from which it did take the shortest, great-circle path, CM_e. This point is 2.2 mm. away from the anatomic insertion, R.

The effective insertion point moved back toward the former location of the insertion in the primary position (R_p, Fig. 6). Thus, when the eye moves and carries the real insertion R with it (by 3.4 mm. Fig. 6), the effective insertion R_e stays behind (moved only 1.3 mm.). This means that the tertiary forces caused by the movement which allows the LR, for example, to

develop interfering vertical and torting forces (m_x', m_y'), are diminished from what they would be otherwise. Tertiary forces and innervations are not unimportant and they clearly depend on one's asssumptions about limiting muscle side-slipping. I have chosen a restriction (Equation 3) that seems to give a reasonable compromise but, clearly, physiologic data are needed to allow these calculations to better describe actual muscle mechanics.

This need is illustrated in Figs. 5, C and D where, near 40° adduction, the contours marked 5 and 10 g. suddenly start to spread apart. This is caused by the fact that the LR insertion is beyond its antipodes point. When the eye moves from 10° up to 10° down in this position, the shortest-path, twist angle, V_s, of the LR flips from 112° to –119°. Equation 3 limits actual twist, V, to 18° and –15°, respectively, but even this allows the vertical cross-coupling of the horizontal recti to change from 6.1 g. up to 4.5 g. down, which is

nearly enough to overcome the passive forces. This leaves the vertical muscles with almost nothing to do so their innervation changes very little. This is probably unphysiologic and a proper mechanical treatment of muscle path and muscle side-slipping would probably eliminate it.

When a muscle twists relative to its line of insertion another phenomenon occurs. In Fig. 6, tension will be greatest in the lower tendon and muscle fibers, and less in the upper, shorter fibers. This factor alone will cause a shift of the effective insertion point toward the stretched edge of the muscle. This was well understood and described by Helmholtz in 1910.[11] This point (R_c, Fig. 6) is called the centroid in mechanics. Even if the muscle did take the shortest path in Fig. 6, it would behave as though it were inserted at R_c. The shift δ in mm. between R and R_c may be calculated. It is:

$$\delta = 0.582 \left(\frac{k\ a^2}{F\ L_p} \right) V \qquad (4)$$

where k is the slope of the length-tension relationship in the primary position (1.8 g. per per cent, Fig. 3), a is half the length of the line of insertion (4.6 mm. for the LR), F is total muscle force (10.2 g., Table I for 30° elevation), L_p is 49.1 mm. (Table II, Appendix), and V is the twist angle. If we permit the shortest path, V is 43.5° and δ is then 1.93 mm. This value of V is certainly unrealistically large. If we choose the value 20.4° actually used, δ would be 0.90 mm. This is only half the shift in the effective insertion point (from R to R_e, 2.2 mm.) due to the fact that the muscle path is curved.

Consequently, there are two mechanisms that cause a shift in the effective insertion point: a centroid shift due to a nonuniform stress distribution in the tendon, and another due to side slip restraints which bend the muscle's path. The former was omitted in this study because it is smaller than the latter and, since the two effects are mechanically intercoupled, the mathematical complications that would be created were not felt to be worth the ex-

pected improvement. Nevertheless, the centroid shift is not unimportant and a more detailed analysis could be worthwhile.

Diagnosis and surgical correction. The main purpose of this analysis is to use it in the diagnosis of disorders and the predictions of surgical corrections. This phase of research is only beginning. The calculated tropias associated with SR and IO palsies shown in Fig. 1 qualitatively support the model since they are more or less what one might expect. However, the whole purpose of the model is to be quantitative not simply qualitative. To test it properly, Fig. 1 should be compared with accurate tropia measurements in a series of cases with complete, isolated palsies of these muscles. This has not yet been done.

However, one other example—the tight lateral rectus syndrome—will be given to illustrate that, even qualitatively, the model can be useful in diagnosis and surgery. In this situation, for some as yet unknown reason, the LR refuses to lengthen. It shortens more or less normally and, on visual inspection at surgery, may not appear fibrotic. Nevertheless, it acts like it, and strongly resists extension by forceps after removal from the globe. The patient has a primary position exotropia (Fig. 7, *A*), can abduct, and can elevate and depress the globe normally in abduction. On attempted adduction, the LR opposes the tension in the MR which results in very little adduction but, instead, large cocontractive forces in the horizontal recti. This causes a noticeable retraction of the eye in the orbit. However, the interesting feature of this problem is that on up gaze in attempted adduction the affected eye over-elevates and on down-gaze there is an exaggerated downshoot (Fig. 7, *A*).

While this could be an innervation problem (an overactive IO on up gaze and an overactive SO on down gaze which only occurs in adduction) there is an alternate, mechanical explanation. The bridle effect of the horizontal recti is directly proportional to the forces in those muscles. In at-
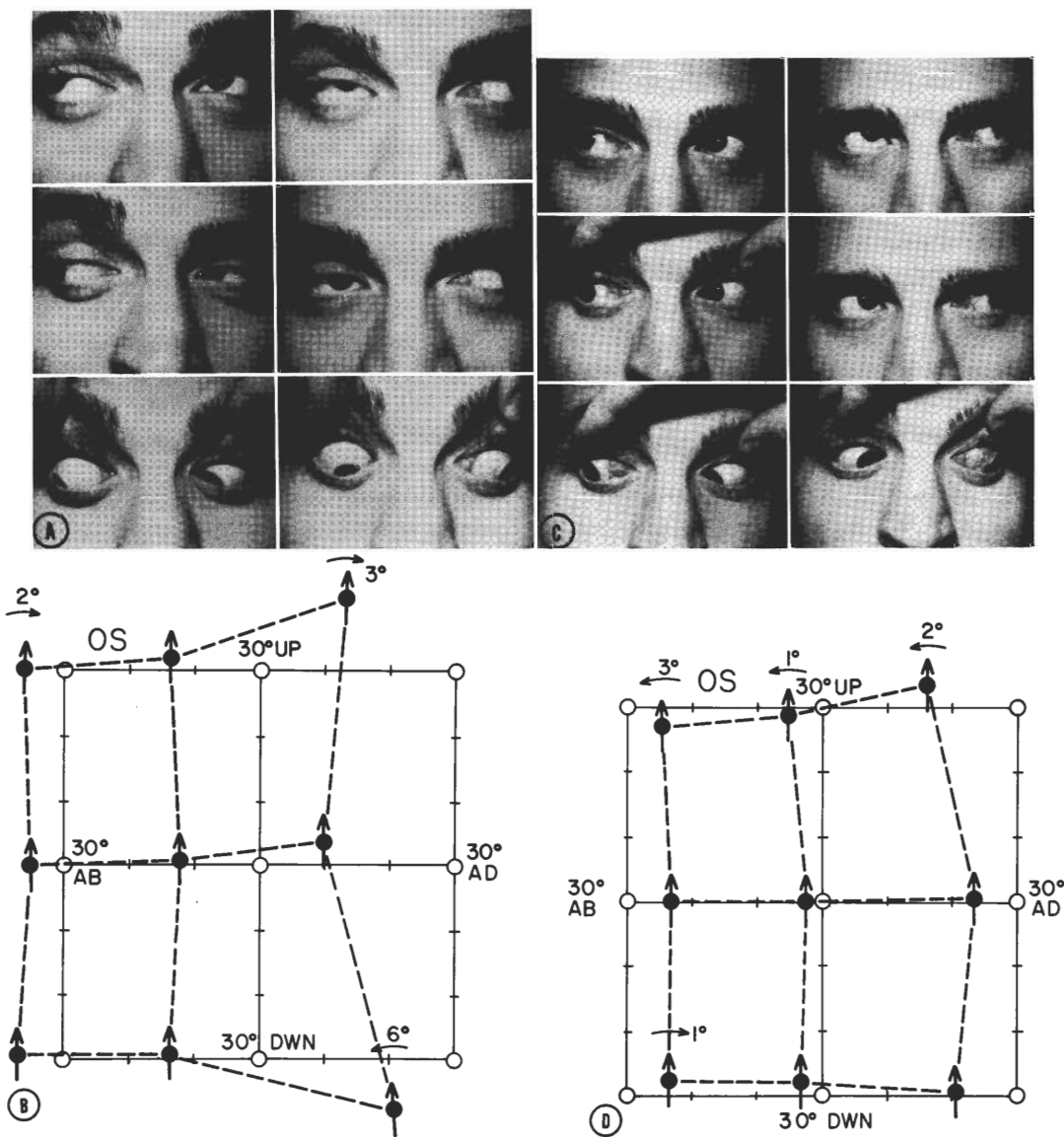
Fig. 7. The tight LR syndrome and its surgical correction. *A*, deviations of a patient with a tight right LR. The right eye cannot adduct and shows an up- and down-shoot with vertical eye movements in attempted adduction. *B*, a simulation of the syndrome. Notation as in Fig. 1. In *A* the right eye is seen from in front, in *B* the left eye is seen from behind so the deviation patterns may be directly compared. Calculated deviations were obtained from the position problem in which the passive LR tissues were greatly stiffened. *C*, the same patient after 9 mm. recession of the right LR and 7 mm. resection of the MR. (Conjunctival markings on the left eye are due to other tests, not surgery. Pictures taken 6 months after surgery on right eye.) *D*, the same simulation as in *B* after a 7 mm. recession of the LR. (Figs. *A* and *C* were provided by A. B. Scott.)

tempted adduction these forces are abnormally large. They will therefore create over-elevation in up gaze and over-depression in down gaze. This explanation has already been proposed by Scott.[12]

To simulate this, the passive force of the LR was increased. Since the actual tropias of this syndrome have not been measured quantitatively, the LR passive mechanical properties were adjusted by trial and error

until the calculated results (Fig. 7, *B*) seemed in best agreement with observation (Fig. 7, *A*). The passive curve (Fig. 3) was moved left by 8.6 per cent, scaled up by three and broadened (a′ in Equation 58, Appendix, was doubled). The developed tension was not changed. In the simulation, the primary position exotropia was 13° and adduction was limited to 10°. Abduction excess was 7°. Vertical movements in abduction are about normal. On attempted 30° adduction, the forces in the LR and MR rose to about 53 g. This excessive cocontraction exaggerated the bridle effect and gave the horizontal recti abnormally large elevating powers on up gaze and depressing action on down gaze. Consequently, with 30° attempted elevation there was a 12° up-shoot and, with depression, an 8° down-shoot.

These data are not presented as an explanation of this syndrome. Its main purpose is to illustrate how simulation might be used in diagnosis and to show what intuition might not: that the bridle effect can, at least qualitatively, offer a simple mechanical explanation of this problem. The actual pathology is clearly more complicated. In attempted adduction, the model permits too much adduction in up and down gaze, not less as shown by the patient. This second-order effect was not pursued in the absence of more quantitative patient data but since this disorder had existed for some time, the other muscles were probably also slightly abnormal, especially the MR which was never allowed to shorten normally.

Fig. 7, *C* shows the same patient six months after a LR recession of 9 mm. and MR resection of 7 mm. Fig. 7, *D* shows the same simulation as in Fig. 7, *B* after a LR recession of 7 mm. In the model eye the primary exodeviation was reduced to 2.5°. The eye could now adduct by 23° and abduction was now deficient as can also be seen in the patient. The vertical tropias were reduced to less than 3°. The main disagreement between patient and model after surgery is that the latter could be corrected by LR surgery alone. Since the MR was assumed normal, it was free to adduct more or less normally once the LR was freed. In the patient, this was inadequate and MR tightening was also needed. This could also be due to an abnormal MR whose shortening range had been permanently affected. Again, without quantitative patient data, this idea was not pursued further. The primary purpose is to illustrate how the method may be used to try to anticipate the results of surgery. A detailed analysis of the etiology of the syndrome would require additional investigation.

Discussion

The purpose of this research is not to present finished results, but to offer a tool for further research. Obviously, careful quantitative comparisons are needed before these calculations can be proved reliable in clinical situations. The examples presented are intended to show that this analytic method is not simply a theoretical plaything. If it can withstand the test of clinical field trials and its parameters are refined to best reflect those of the average eye (probably corrected for age), it should become a useful diagnostic tool in referral centers interested in clinical research in strabismus. In those situations in which the same tropia pattern is produced by two different disorders, quantitative forced-duction tests (which are also automatically calculated by this method) would probably remove all ambiguity. Its usefullness in teaching should not be overlooked. The student can experiment with any type of pathology and any surgical corrections he can imagine and observe their results at once.

The major problem facing the usefullness of these calculations is in predicting the results of surgery. The effects of scarring and atrophy with fibrosis in recessed muscles means that an operated muscle will not behave normally as in Fig. 3. Nevertheless, future experimental research could measure these changes and the calculations could take them into account.

At present, muscle surgery is done on

an empirical basis which is illustrated by the fact that different "schools" of surgery can argue about the efficacy of their procedures without once coming to grips with the quantitative aspects of muscle mechanics. The failure rates indicate that current methods are inadequate. If improvement is to come, it may be best if it comes not from more empirical data but from an attempt to understand the basic problem itself in terms of rational mechanics.

It is unfortunate that the calculations are impossible without a digital computer. This restricts the use of this technique to the few facilities who are willing to take this much trouble. However, the computer in no way pre-empts the judgment of the operator as to which abnormalities will produce which tropias, it merely executes the mindless calculations involved. I again extend my offer to make this computer program available to anyone seriously interested in applying it in further theoretical or clinical applications.

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Appendix

The innervation problem. Given Θ and ϕ (Fig. 2), Listing's law states that ψ should be,

$$\psi = \sin^{-1}\left(\frac{\sin \Theta \sin \phi}{1 + \cos \Theta \cos \phi}\right). \quad (5)$$

Let $\vec{r}_o$ be a vector from C to R_o, the muscle's insertion point in the primary position (Fig. 8). The vector $\vec{o}$ is from C to the point of origin O. The coordinates of $\vec{r}_o$ and $\vec{o}$ appear in Table II, Appendix. The muscle is assumed to take the shortest path to O. Its insertion line is assumed perpendicular to this path. The unit vector $\vec{m}_o$ is perpendicular to $\vec{r}_o$ and $\vec{o}$. It is formed by taking the cross product, $\vec{m}'_o$, of $\vec{r}_o$ and $\vec{o}$ whose components are:

$$m'_{ox} = r_{oy} o_z - r_{oz} o_y$$
$$m'_{oy} = r_{oz} o_x - r_{ox} o_z \quad (6)$$
$$m'_{oz} = r_{ox} o_y - r_{oy} o_x$$

It is normalized by:

$$\vec{m}_o = -\frac{\vec{m}'_o}{|\vec{m}'_o|} = -\frac{\vec{r}_o \times \vec{o}}{|\vec{r}_o \times \vec{o}|} \quad (7)$$

where the absolute value sign indicates the length of the vector which is the square root of the sum of the squares of its components. The minus sign occurs here and elsewhere because the axes in Fig. 2 form a left-hand screw system. Vector $\vec{m}_o$ is parallel to the muscle's line of insertion.

When the eye rotates by Θ, ϕ, and ψ, $\vec{r}_o$ will move to some position, $\vec{r}$, which may be found by multiplying $\vec{r}_o$ by a coordinate transformation matrix, A,

$$
A = \begin{array}{|c|c|c|}
\hline
\cos \Theta \cos \psi & & \sin \psi \cos \Theta \\
+ \sin \psi \sin \phi \sin \Theta & \cos \phi \sin \Theta & - \sin \Theta \cos \psi \sin \phi \\
\hline
\sin \psi \sin \phi \cos \Theta & & - \sin \psi \sin \Theta \\
- \sin \Theta \cos \psi & \cos \phi \cos \Theta & - \cos \Theta \cos \psi \sin \phi \\
\hline
- \sin \psi \cos \phi & \sin \phi & \cos \psi \cos \phi \\
\hline
\end{array} \qquad (8)
$$

Thus:

$$ \vec{r} = A \, \vec{r}_o. \qquad (9) $$

The vector

$$ \vec{m}_1 = A \, \vec{m}_o \qquad (10) $$

is still parallel to the insertion line in the new position (Fig. 8).

If the muscle takes the shortest path from R to O (Fig. 8) it will lie in the plane formed by $\vec{r}$ and $\vec{o}$. Its unit action vector, $\vec{m}_s$, is perpendicular to that plane so:

$$ \vec{m}_s = - \frac{\vec{r} \times \vec{o}}{|\vec{r} \times \vec{o}|}. \qquad (11) $$

If the tendon fibers continued to leave the line of insertion perpendicular to it (the primary track, CPT, Fig. 4) they would be directed along $\vec{t}_1$ which is perpendicular to $\vec{m}_1$ and $\vec{r}$ so that:

$$ \vec{t}_1 = - \frac{\vec{m}_1 \times \vec{r}}{|\vec{m}_1 \times \vec{r}|}. \qquad (12) $$

If they take the shortest path, the fibers will leave along $\vec{t}_s$ which is perpendicular to $\vec{m}_s$.
The shortest-path twist angle, V_s, is the angle between $\vec{t}_1$ and $\vec{t}_s$ or between $\vec{m}_1$ and $\vec{m}_s$. The angle between two vectors may be found from the dot product:

$$ \cos V_s = \frac{\vec{m}_1 \cdot \vec{m}_s}{|\vec{m}_1| \, |\vec{m}_s|} $$
$$ = (m_{1x} \, m_{sx} + m_{1y} \, m_{sy} + m_{1z} \, m_{sz}). \qquad (13) $$

In this case $|\vec{m}_1|$ and $|\vec{m}_s|$ are 1.0. However, cos V_s does not indicate whether V_s is positive or negative. This may be corrected by a trick. Find the angle, W, between $\vec{m}_s$ and $\vec{t}_1$:

$$ \cos W = \vec{m}_s \cdot \vec{t}_1. \qquad (14) $$

Then:

$$ V_s = - (\mathrm{sgn} \, (\cos W)) \, \cos^{-1} (\vec{m}_1 \cdot \vec{m}_s) \qquad (15) $$

where sgn (cos W) is +1 if cos W is positive and −1 if cos W is negative.

The angle E (Fig. 4) is an index of the force on a muscle to pull it sideways. It is the angle between $\vec{o}$ and a perpendicular to the plane of the primary track. Vector $\vec{m}_1$ is perpendicular to the plane of the primary track. E is therefore the angle between $\vec{o}$ and $\vec{m}_1$ so:

$$ \cos E = \frac{\vec{m}_1 \cdot \vec{o}}{|\vec{o}|}. \qquad (16) $$

The permitted twist angle is:

$$ V = |\cos E| \, V_s. \qquad (17) $$

(See Methods and Fig. 4 for justification of this choice.)

The muscle is assumed to follow the arc of a nongreat circle CM (Fig. 9, A) which passes through R and makes the angle V with $\vec{t}_1$. CM intersects Tan at T. The muscle will leave the globe at T and go straight to O. Line O-T is perpendicular to Tan (by construction of Tan). If the muscle path is to be smooth through T, CM must also be perpendicular to Tan so that line O-T is tangent to CM. Therefore, O-T lies in the plane of CM. Then CM may be redefined as a circle whose plane contains R and O and which passes through R, making the angle V with $\vec{t}_1$. Let $\vec{t}$ be the vector tangent to CM at R.

Vectors $\vec{t}_1$ and $\vec{m}_1$ are unit vectors at right angles and $\vec{t}$ lies in their plane (Fig. 9, B). The angle between $\vec{t}$ and $\vec{t}_1$ is, by definition, V. Therefore (Fig. 9, B):

$$ \vec{t} = (\sin V) \, \vec{m}_1 + (\cos V) \, \vec{t}_1. \qquad (18) $$

Vector $\vec{t}$ lies in the plane of CM. Another vector that does so is $\vec{p}$, between R and O (Fig. 9, C):

$$ \vec{p} = \vec{o} - \vec{r}. \qquad (19) $$

These two vectors define the plane of CM.

We now seek the length of the muscle's contact arc with the globe; the arc of CM from R to T. We also need the muscle's moment arm, vector $\vec{a}$ (Fig. 9, A). To do this, find two auxillary vectors, $\vec{A}$ and $\vec{B}$ from $\vec{C}_2$, the center of CM to points R and T. $\vec{A}$ can be found by a linear combination of $\vec{p}$ and $\vec{t}$. The angle between them, X, is found from:

$$ \cos X = \frac{\vec{p} \cdot \vec{t}}{|\vec{p}|}. \qquad (20) $$

Then a unit vector, $\vec{A}'$ directed along $\vec{A}$ is:

$$ \vec{A}' = \frac{\vec{t} \, (\cos X) - (\vec{p}/|\vec{p}|)}{\sin X}. \qquad (21) $$

Table II. The locations of the origins, O, and insertions, R, of the extraocular muscles according to Volkmann[5] and Helmholtz,[6] along with muscle lengths L_p, and cross-sectional area, λ, relative to the LR, also according to Volkmann[5]

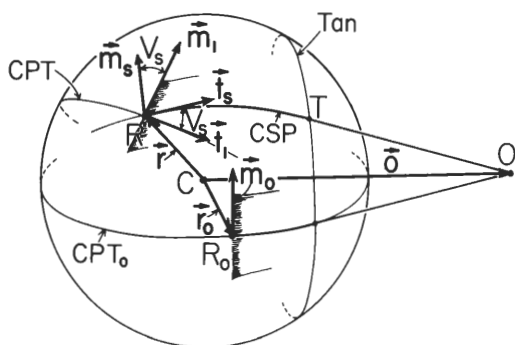|		1, LR	2, MR	3, SR	4, IR	5, SO	6, IO
Location of muscle	r_{ox}	10.08	− 9.65	0.00	0.00	2.90	8.70
insertions	r_{oy}	6.50	8.84	7.63	8.02	− 4.41	− 7.18
R_o (mm.)	r_{oz}	0.00	0.00	10.48	−10.24	11.05	0.00
Location of muscle	O_x	−13.00	−17.00	−16.00	−16.00	−15.27	−11.10
origins	O_y	−34.00	−30.00	−31.76	−31.76	8.24	11.34
O (mm.)	O_z	0.60	0.60	3.60	− 2.40	12.25	−15.46
Muscle lengths, L_p (mm.)		49.11	38.51	41.96	42.49	22.28	35.35
Relative strengths (λ)		1.00	1.04	0.68	0.95	0.50	0.47

Fig. 8. Vectors associated with determining the twist angle, V_s, that would occur if the shortest path CSP were taken. Nomenclature is similar to that in Figs. 2 and 4. R_o, $\vec{r}_o$, CPT_o, $\vec{m}_o$ are points, vectors, and paths associated with a muscle in the primary position. When the eye rotates to a new position they change to R, $\vec{r}$, CPT, and $\vec{m}_1$.

The length of A can be found from the right triangle C, R, C_2 (Fig. 9, D). The angle between $\vec{r}$ and $\vec{A}$ is H:

$$\cos\ H = \frac{\vec{r} \cdot \vec{A}'}{|\vec{r}|}. \tag{22}$$

The length of $\vec{r}$ is the globe radius, Rad. Therefore:

$$|\vec{A}| = (\text{Rad})\ \cos\ H \tag{23}$$

so that,

$$\vec{A} = |\vec{A}|\ \vec{A}'. \tag{24}$$

To find $\vec{B}$, first let $\vec{q}$ be the vector from C_2 to O. From Fig. 9, C:

$$\vec{q} = \vec{A} + \vec{p}. \tag{25}$$

The angle b between $\vec{A}$ and $\vec{q}$ is:

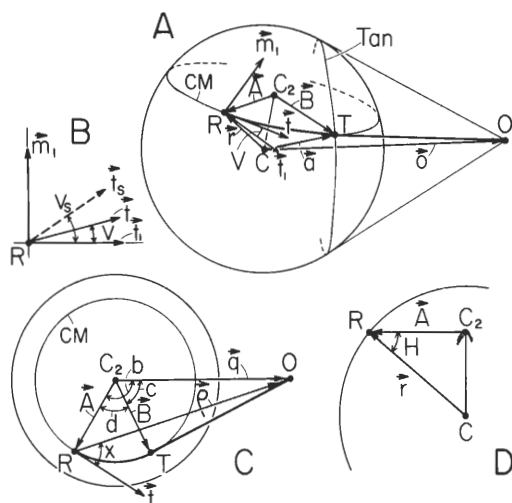$$\cos\ b = \frac{\vec{A} \cdot \vec{q}}{|\vec{A}|\ |\vec{q}|}. \tag{26}$$

Fig. 9. *A*, the muscle lies in a plane containing R and O. That plane intersects the globe and forms circle CM which must be tangent to the tendon vector, $\vec{t}$, which departs from the primary track tendon vector, $\vec{t}_1$, at the permitted tendon twist angle, V. The muscle contact arc with the globe is the length of the arc R to T. The vector $\vec{a}$ from C to T is the muscle's moment arm which determines how it will act to rotate the globe. *B*, a view of the plane tangent to the globe's surface at R which contains vectors $\vec{m}_1$, $\vec{t}$, and $\vec{t}_1$. *C*, a view straight down on the plane of circle CM which contains vectors $\vec{t}$, $\vec{p}$, $\vec{A}$, $\vec{B}$, and $\vec{q}$. *D*, a side view of the plane containing R, C, C_2, $\vec{A}$, and $\vec{r}$.

Since points C_2, T, and O form a right triangle, the angle c is:

$$\cos\ c = \frac{|\vec{B}|}{|\vec{q}|}. \tag{27}$$

Note that $|\vec{B}|$ equals $|\vec{A}|$, already found. The angle d, between $\vec{A}$ and $\vec{B}$ is:

$$d = b - c. \tag{28}$$

Vectors $\vec{t}$ and $\vec{A}'$ are unit vectors at right angles and $\vec{B}$, which lies in the same plane, is a linear combination of them (Fig. 9, C):

$$\vec{B} = ((\cos d) \ \vec{A}' + (\sin d) \ \vec{t}) \ |\vec{A}|. \quad (29)$$

The contact arc with the globe can now be found. It is simply:

$$L_g = |\vec{A}| \ d. \quad (30)$$

In the primary position, $\vec{q}$ equals $\vec{o}$ and $\vec{A}$ equals $\vec{r}_o$ so that, (from Equations 26 through 30) the angles b, c, and d may be found easily, and L_{go}, the contact arc in the primary position, calculated. The change in muscle length is then:

$$\Delta L = L_g - L_{go}. \quad (31)$$

The only exception is when the MR loses tangency with the globe beyond 34° adduction. When this happens, Equations 20 to 31 are no longer valid. This situation can be detected first by finding the angle b_o between $\vec{r}$ and $\vec{o}$:

$$\cos b_o = \frac{\vec{r} \cdot \vec{o}}{|\vec{r}| \ |\vec{o}|} \quad (32)$$

and comparing it to the angle c_o:

$$\cos c_o = \frac{(\text{Rad})}{|\vec{o}|} \quad (33)$$

which is the angle between $\vec{o}$ and any vector from C to Tan (Fig. 9, A). If b_o is less than c_o, the muscle has lost tangency and ΔL may be approximated by:

$$\Delta L = (\text{Rad}) \ (b_o - c_o) - L_{go}. \quad (34)$$

Finally, ΔL is expressed in per cent:

$$\Delta l = \frac{\Delta L}{L_p} \times 100. \quad (35)$$

The unit moment arm vector $\vec{a}$ is (Fig. 9, A):

$$\vec{a} = \vec{r} - \vec{A} + \vec{B}. \quad (36)$$

The unit moment vector, $\vec{m}$, is at right angles to $\vec{a}$ and $\vec{o}$:

$$\vec{m} = -\frac{\vec{a} \times \vec{o}}{|\vec{a} \times \vec{o}|}. \quad (37)$$

If the muscle has lost tangency, this equation is not used and $\vec{m}$ is equal to $\vec{m}_s$.

To convert $\vec{m}$ into the reference frame of the eyeball (instead of the orbit) use:

$$\vec{m}' = A^{-1} \ \vec{m} \quad (38)$$

where A^{-1} is the inverse of matrix A (Equation 8).

To find muscle force, the experimental length-tension curves shown in Fig. 3 were approximated by the hyperbola:

$$F_1 =$$

$$\lambda_1 \left[\frac{k}{2} \ (\Delta l_1 + e_1) + \sqrt{\frac{k^2}{4} \ (\Delta l_1 + e_1)^2 + a^2} \right] \quad (39)$$

where k is the right-hand asymptotic slope and a determines the sharpness of curvature. Experimental data indicates k is 1.8 g. per per cent and a is 6.24 g. Changes in the variable e slide the curve to the left (excitation or + e) or right (inhibition or − e) which simulates a change in innervation. Therefore, although e is only a mathematical device, it is directly related to innervation, I. I is defined as the developed isometric force when Δl is zero:

$$I = \lambda \left[\frac{k}{2} \ e + \sqrt{\frac{k^2}{4} \ e^2 + a^2} \right] -$$

$$\lambda \left[\frac{k}{2} \ e_o + \sqrt{\frac{k^2}{4} \ e_o^2 + a^2} \right]. \quad (40)$$

The first term is total tension from Equation 39, the second term, call it I_o, subtracts out the passive muscle tension. I_o is determined by e_o which is the value of e that fits the passive curve in Fig. 3. The value of e_o is −6.4 per cent. From Equation 40, I can always be found if e is known and *vice versa*. Because subsequent calculations become much simpler, e is used rather than I. Force balance is found in terms of e_1 and finally converted to I_1 by Equation 40.

Reciprocal innervation is also determined in terms of e. From Fig. 3, e of an agonist is plotted against e for its antagonist. The result can be approximated by the curve:

$$e_{2i} = \frac{(h + w)^2}{(e_{2i-1} + w)} - w, \ i = 1, 2, 3. \quad (41)$$

The values of h and w that fit experimental data are 4.0 and 9.7, respectively. From Equation 41, e_2, e_4, and e_6 can be determined if e_1, e_3, and e_5 are known.

To determine the passive moment $\vec{P}$, one utilizes the fact that a normal eye movement obeys Listing's law. Listing's plane contains the primary position visual axis (the + y axis) and the new visual axis, y' (Fig. 2). This plane forms the angle α with the horizontal plane. The slant angle of rotation in Listing's plane is β. $\vec{P}$ will be perpendicular to Listing's plane and its magnitude, F_p, will be proportional to β. One finds α and β from:

$$\beta = \text{sgn} \ \Theta \ \cos^{-1} \ (\cos \phi \ \cos \Theta) \quad (42)$$

and

$$\alpha = \sin^{-1}\left(\frac{\sin \phi}{\sin \beta}\right). \qquad (43)$$

The components of $\vec{P}$ are then:

$$P_x = F_p \sin \alpha \qquad (44)$$
$$P_y = 0$$
$$P_z = - F_p \cos \alpha + P_o$$

and from experimental data[3]:

$$F_p = k_p \beta + k'_p \beta^3 \qquad (45)$$

where β is in degrees and k_p is 0.48 g. per degree, and k'_p is 1.56×10^{-4} g. per degrees.[3]

When all muscles are paralyzed, this determination of $\vec{P}$ would cause the eye to adduct because of the bridle effect of the SR and IR. In fact, the eye should abduct. Consequently, the neutral point of the passive tissues was swung laterally, more in line with the axis of the orbital cone. To do this, a constant abducting moment, P_o, equal to $k_p \beta_o$ was added to P_z in Equation 44. If β_o is chosen as 7.5°, the eye, in death, will then abduct 2.5°.

We now have determined Δl_i (Equation 35) for each muscle. We guess at e_1, e_3, and e_5 (e.g., in the primary position, from Fig. 3, one would pick 4 per cent for each) and, from Equation 41, find e_2, e_4, and e_6. One can then find from Equation 39 all six muscle forces F_i. They are converted to moments by multiplying by $\vec{m}_i$ from Equation 37. By adding $\vec{P}$ from Equation 44 we have the total moment $\vec{M}$:

$$\vec{M} = \vec{P} + \sum_{i=1}^{6} F (\Delta l, e)_i \ \vec{m}_i. \qquad (46)$$

If the guess was wrong, $\vec{M}$ will not be zero. Call it $\vec{M}_o$. Make a linear expansion of M (e_1, e_3, e_5) around the point (e_1, e_3, e_5):

$$\vec{M} = \vec{M}_o + \sum_{i=1}^{6} \frac{\partial \vec{M}}{\partial e_i} \Delta e_i. \qquad (47)$$

$\vec{P}$ does not depend on e and disappears. Setting $\vec{M}$ equal to zero:

$$-\vec{M}_o = \sum_{i=1}^{6} \vec{m}_i \frac{\partial F_i}{\partial e_i} \Delta e_i \qquad (48)$$

(note $\vec{m}_i$ is a constant and does not depend on e_i). Group the muscles into pairs and write e_2, e_4, and e_6 in terms of e_1, e_3, and e_5:

$$-\vec{M}_o = \sum_{i=1}^{3}$$

$$\left(\frac{\partial F_{2i-1}}{\partial e_{2i-1}} \vec{m}_{2i-1} + \frac{\partial F_{2i}}{\partial e_{2i}} \cdot \frac{de_{2i}}{de_{2i-1}} \vec{m}_{2i}\right) \Delta e_{2i-1}. \qquad (49)$$

The derivatives are (from Equations 39 and 41):

$$\frac{\partial F_j}{\partial e_j} = \lambda_j \left(\frac{k}{2} + \left(\frac{\frac{k^2}{4}(\Delta l_j + e_j)}{\sqrt{\frac{k^2}{4}(\Delta l_j + e_j)^2 + a^2}}\right)\right)$$

$$j = 1, \ldots, 6 \qquad (50)$$

and {Blue = correction by JMM, not in original}

$$\frac{de_{2i}}{de_{2i-1}} = - \frac{(h + w)^2}{(e_{2i-1} + w)^2}, \ i = 1, \ldots 3. \quad (51)$$

When these terms are all evaluated, the terms in parenthesis on the right in Equation 49 are relabeled as components of three vectors $\vec{D}_{2i-1}$ (one for each muscle pair) so that it can be more simply rewritten:

$$\sum_{i=1}^{3} \vec{D}_{2i-1} \Delta e_{2i-1} = -\vec{M}_o. \qquad (52)$$

This set of three equations must be solved for Δe. If Δe is thought of as a vector (Δe_1, Δe_3, Δe_5) and the three vectors D as a matrix, then:

$$D \ \vec{\Delta e} = - \ \vec{M}_o. \qquad (53)$$

Inverting D:

$$\vec{\Delta e} = D^{-1} (-\vec{M}_o). \qquad (54)$$

This gives the desired solution Δe_1, Δe_3, and Δe_5. The next best guess, to make $\vec{M}$ zero, is $e'_1 = e_1 + \Delta e_1$, $e'_3 = e_3 + \Delta e_3$, $e'_5 = e_5 + \Delta e_5$. One now uses these new values of e to recalculate all the items needed for Equation 46. This process is repeated until $|\vec{M}_o|$ is so small (e.g., less than 0.3 g.) as to be considered negligible. One then has the desired values of e (or I) as well as all other variables which describe the mechanical state of the system (e.g., as in Table I).

When the MR has lost tangency (Equations 32 and 33), its moment arm and consequently its total moment, F m_i, must be decreased by the ratio:

$$\rho_a = \frac{|\vec{o}| \sin b_o}{\sqrt{|\vec{o}|^2 + (Rad)^2 - 2 (Rad) |\vec{o}| \cos b_o}} \qquad (55)$$

where b_o comes from Equation 32.

The position problem. In this case, e_1, . . ., e_6 are specified, from the solution to the innervation problem, for the attempted gaze angle Θ_o, ϕ_o. If one prefers to use I_1, . . ., I_6, the e_i values can be found from the inverse of Equation 40:

$$e_i = \frac{\left(\frac{I_i + I_{oi}}{\lambda_i}\right)^2 - a^2}{k\left(\frac{I_i + I_{oi}}{\lambda_i}\right)}. \qquad (56)$$

If one wishes to simulate a true innervation disorder (e.g., abnormal third nerve regeneration) that is not equivalent to a muscular disorder (e.g., hypertrophy), one must alter the normal e_1 values accordingly at this point. After that, the e_1 values are fixed.

If a muscle is to be resected, its insertion vector $\vec{r}$ is not changed but the amount of resection, ΔL_s, in millimeters, is added to the value of ΔL from Equation 31. If a muscle is recessed, its vector $\vec{r}$ is moved back (or in any direction of interest) by ΔL_s in millimeters. Its new coordinates must be found and substituted for the normal values (Table II, Appendix) and ΔL_s must be subtracted from ΔL.

Muscle pathology is introduced by separating F for the normal muscle into the sum of passive, F_{pm}, and developed, F_d, tension:

$$F_{pm} = \lambda \left[\frac{k}{2} (\Delta l + e_o) + \sqrt{\frac{k^2}{4} (\Delta l + e_o)^2 + a^2} \right] \quad (57)$$

$$F_d = F - F_{pm}.$$

The two components can now be made abnormal independently. For example, abnormal passive tension:

$$F'_{pm} = \lambda' \left[\frac{k'}{2} (\Delta l + e'_o) + \sqrt{\frac{k'^2}{4} (\Delta l + e'_o)^2 + a'^2} \right] \quad (58)$$

can be created by changing any of the parameters λ', k', e'_o, and a'. Developed tension can be scaled up (hypertrophy) or down (atrophy) to create F'_d. Total abnormal muscle tension is then $F'_d + F'_{pm}$. Abnormal orbital tissues (e.g., a tight check ligament, an orbital floor fracture, scar tissue) can be created by altering Equation 45. Obviously, any other types of abnormality can be created by appropriately changing other parameters and equations.

One now guesses at the solution, Θ, ϕ, and ψ. Usually Θ_o, ϕ_o, and ψ_o are chosen initially. One then proceeds, as in the innervation problem, to calculate the total moment, $\vec{M}$, on the globe. All the steps starting at Equation 8 are followed to find Δl_i and $\vec{m}_1$. F may then be found from Equation 39 or modified by Equations 57 and 58, if necessary. Step Equation 41 is omitted since all the e_1 are known. $\vec{P}$ is determined and $\vec{M}$ obtained from Equation 46.

Of course, $\vec{M}$ will not usually be zero. Its components indicate which way and how strongly

the force imbalance wants to push the eye. A moment of magnitude $|\vec{M}|$ would push the eye through an angle of $|\vec{M}|/K$ where K is an overall effective spring constant of about 2 g. per degree. (For certain types of pathology, this value could grossly over- or underestimate the angle and a different value of K should be chosen to obtain a reasonable rate of convergence on the correct solution.) The breakdown of this rotation into its components $\Delta\Theta$, $\Delta\phi$, and $\Delta\psi$ are:

$$\Delta\Theta = \frac{1}{K} (- M_x \tan \phi \sin \Theta \quad (59)$$
$$- M_y \tan \phi \cos \Theta + M_z)$$

$$\Delta\phi = \frac{1}{K} (- M_x \cos \Theta + M_y \sin \Theta)$$

$$\Delta\psi = \frac{1}{K} \left(- M_x \frac{\sin \Theta}{\cos \phi} - M_y \frac{\cos \Theta}{\cos \phi} \right).$$

The new estimate of eye position is:

$$\Theta' = \Theta + \Delta\Theta \quad (60)$$
$$\phi' = \phi + \Delta\phi$$
$$\psi' = \psi + \Delta\psi.$$

Using these new values, all the calculations are repeated until the value of $|\vec{M}|$ is small enough to be neglected. One then has the desired solution Θ, ϕ, and ψ.

There is one complication. The angle ψ need no longer obey Listing's law. The eye may twist around the line of sight by ν (true torsion),

$$\nu = \psi - \psi_L \quad (61)$$

where ψ_L is the angle predicted by Listing's law (Equation 5) (sometimes called false torsion). This will create a restoring moment in the orbital suspensory tissues of magnitude:

$$F_{p\nu} = k_p \nu + k'_p \nu^3 \quad (62)$$

just as in Equation 45. This moment will add additional components to $\vec{P}$. The new moment $\vec{P}$ is:

$$P_x = F_p \sin \alpha + F_{p\nu} \cos \phi \sin \Theta$$
$$P_y = F_{p\nu} \cos \phi \cos \Theta$$
$$P_z = - F_p \cos \alpha + F_{p\nu} \sin \phi + P_o$$

which is used in place of Equation 44. This equation tacitly assumes that the eye reaches its final position from the equilibrium point of the orbital tissues by a rotation to the primary position by β_o, a rotation in Listing's plane by β, and a twist about the visual axis by ν. Since rotations of a sphere are not additive, it is incorrect to add the three moments associated with the three rotations. However, since β_o and ν are small, the error in doing so is small and does not justify the complications of finding $\vec{P}$ more exactly.