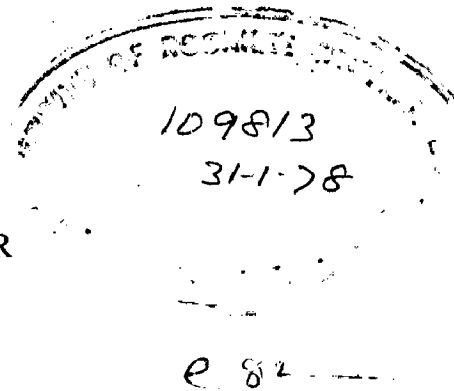


DYNAMICS OF EXTRAOCULAR MUSCLE ^{ASH} AND ITS OPTIMIZATION

A DISSERTATION
submitted in partial fulfilment of
the requirements for the award of the Degree
of
MASTER OF ENGINEERING
in
ELECTRICAL ENGINEERING
(Measurement and Instrumentation)

by
ASHWINI KUMAR

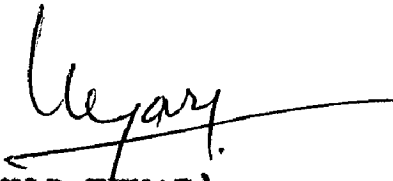


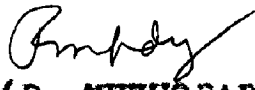
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C E R T I F I C A T E

Certified that the dissertation entitled "DYNAMICS OF EXTRAOCULAR MUSCLE AND ITS OPTIMIZATION", which is being submitted by Shri Ashwini Kumar in partial fulfilment for the award of degree of M.E. in Electrical Engineering (Measurement & Instrumentation) of the University of Roorkee, is a record of bonafide work carried out by him under our supervision and guidance. The matter embodied in this dissertation has not been submitted for the award of any other degree or diploma.

This is further certified that he has worked for 6 months from Jan.77 to June.77 for preparing this dissertation.


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9.7.77


(ASHWINI KUMAR)

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A B S T R A C T

A model for the human vertical eye tracking system is proposed. This is based on the dynamic characteristics of a variety of eye movements as well as the anatomical and physiological aspects of the extraocular mechanism. It includes the nonlinear force-velocity relationship of active muscle and considers separately the distinct asymmetrical properties of the agonist-antagonist muscle pair. The inertial and visco-elastic forces of the globe and orbit on the muscles is also included. The nervous activity which causes the muscles to move the eye was approximated from studies of the extraocular motor nuclei. Responses of the model accurately depict position, velocity and acceleration of saccades. The model also reproduces, faithfully, the "main sequence" of saccadic velocity versus amplitude. It has been shown that saccades are time optimal.

1. INTRODUCTION

Human visual system is one of the most complex and interesting biological systems. It has been extensively studied by neurologists, vision scientists, physiologists, anatomists and engineers. These studies have provided valuable information in the fields of medicine, physiology, neurology and psychology. Eyeball movement pattern has been used to indicate ^hophthalmological conditions, neurological lesion, behaviour pattern etc. and many more uses of oculography are listed in literature.

The work on modelling of eye movement mechanism - a neuro-muscular servomechanism, was initiated in early fifties and has gradually progressed. This has of course included the modelling of extraocular muscles - muscles attached to the eyeball and responsible for its movement. Latest studies are based on anatomical and physiological characteristics of the globe and orbit and static and dynamic characteristics of the extraocular muscles. But all these models are for horizontal movements of the eye. In the present study a model for eye positioning mechanism involved in VERTICAL eye-movements is being proposed. The dynamic characteristics of the model have been studied and found to correspond to experimental results existing in literature.

2. EYE MOVEMENT

2.1 General

The visual system is extremely important for man's normal functioning. It has the following three main aspects.

(i) Pupillary Light Reflex : It is the reflex regulation of pupil area by the iris. The iris provides an opening for the passage of light. As the light intensity increases the pupil area decreases in a logarithmic manner and vice versa. Area of the pupil is varied by muscles attached to the iris.

(ii) Focussing : The corneal convex lens provides effective, but optically complicated means to focus the image of a perceived object upon the retina. The process of changing the lens power until the image is in focus, when the object is at varying distances is called accommodation. Accommodation too is achieved by action of muscles which modify the convexity of the lens. So far as focussing is concerned the eye behaves like a camera.

(iii) Eye Positioning : When a target or object is moving the eye must also move. This is called eye tracking. Each eyeball must be positioned in its orbit for any given target position. Three antagonistic pairs of muscles work in coordination to achieve this eye fixation.

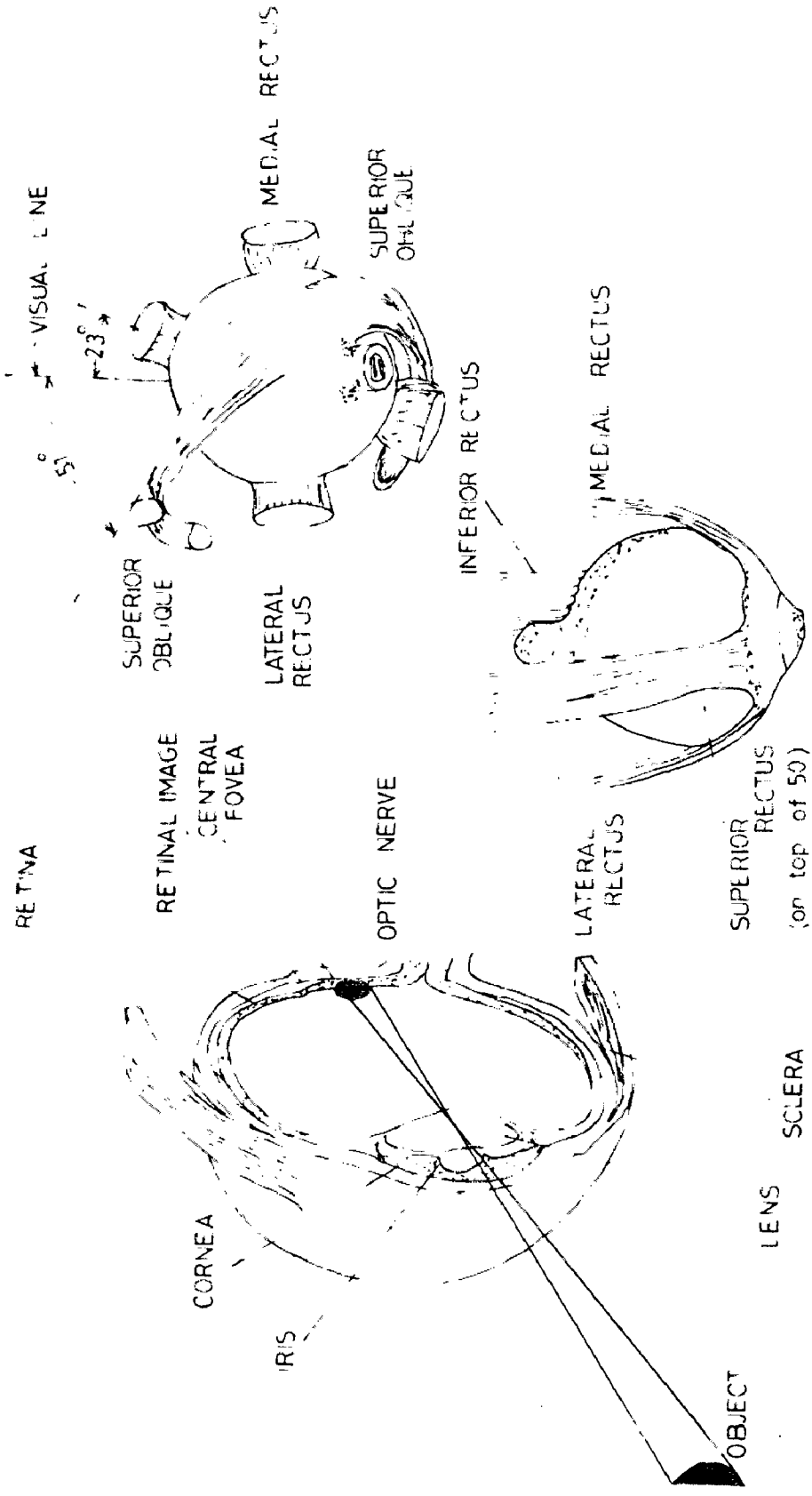


FIG 1 - CONTROL SYSTEM OF THE EYE POSITIONING THE IMAGE OF AN OBJECT ON THE CENTRAL FOVEA. (left) (CENTRE) - RIGHT EYE BALL FROM ABOVE AND FROM REAR (right)

We, thus, see that all the three aspects of visual system depend for their functioning on muscles. The muscles are actuated by various nervous signals, originating in visual cortex and the cerebellum which in turn are parts of the central nervous system (CNS). Viewed in totality all three aspects are neuro-muscular servomechanisms. In this dissertation our attention will be confined to the last - eye positioning, of the three aspects.

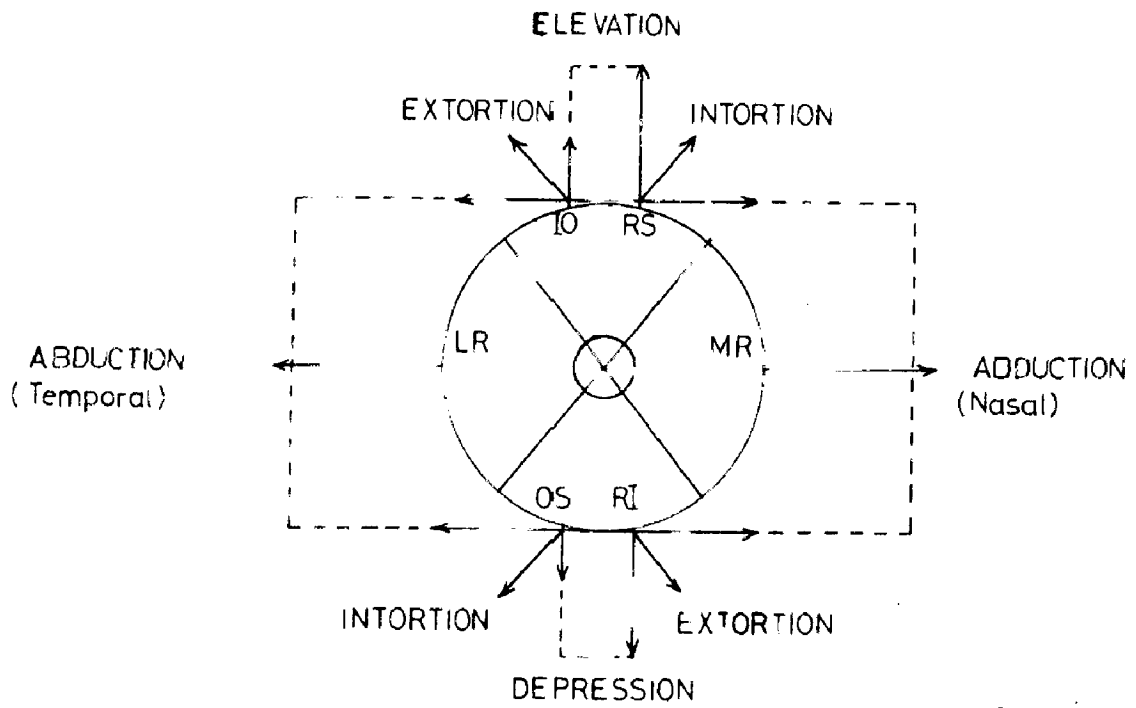
2.2 Eye Movement Mechanism :-

2.2.1 Extraocular muscles

The movement of the eyeball in the socket is actuated by six extraocular muscles attached to the eyeball. These six muscles constitute three pairs of agonist-antagonist muscles (Fig. 1) and are named as below :

- (i) Lateral rectus - Medial rectus
- (ii) Superior rectus - Inferior rectus
- (iii) Superior oblique - Inferior oblique

The action of each of these muscles is shown in Fig. 2. The muscle plane of medial and lateral recti is exactly in the horizontal plane of the globe. When the globe is in the primary position i.e. looking straight ahead, the contraction of these recti results only in internal rotation of the globe; the medial rectus moving the eyeball nasally and the lateral rectus temporally when the visual line is directed above the horizon,



- RS - Rectus superior
- RI - Rectus inferior.
- OS - Oblique superior
- OI - Oblique inferior.
- LR - Lateral rectus
- MR - Medial rectus

FIG. 2 - OCULAR MUSCLES (right eye) - DIAG. OF ACTION
 (NOTE - ACTUAL LOCATION OF IO AND SO ARE REVERSED.)

contraction of these muscles will aid in further elevation and when the visual line is directed below the horizon, their contraction will further depress the globe.

The superior rectus makes an angle of approximately 23° with the visual line when the eye is in the primary position. Its contraction will, therefore, result in a rotation of the globe around several axes resulting in an oblique motion which contains three components, elevation, nasal rotation and intorsion (Fig. 2). Thus the torsion is clockwise in the right eye and counter clockwise in the left eye.

The muscle plane of the inferior rectus is the same as that of the superior rectus and hence its contraction will result in a compound movement of the globe consisting of three components, depression, nasal rotation and turning outward of the 12 O'Clock position i.e. extorsion.

The plane of the superior-inferior obliques forms an angle of 51° with the visual line when the eye is in the primary position. Their contraction also causes oblique movements consisting of three components. Contraction of superior oblique causes depression, intorsion and temporal rotation, whereas contraction of inferior oblique will produce elevation, extorsion and temporal rotation.

It can thus be seen that different muscles are involved in horizontal and vertical movements of the eye.

From the foregoing it is clear that certain muscles are AGONISTS i.e. act in the same direction whereas some are ANTAGONISTS in the sense that each acts in opposite direction. For example superior rectus and inferior oblique are agonists in that they both elevate the globe. Inferior rectus and superior oblique are also agonists because both of them depress the globe. Similarly superior oblique and superior rectus are agonists because both produce intorsion in the primary position of the gaze. In a similar fashion we may group various muscles of each eye that act against other muscles. Thus the medial rectus is the direct antagonist of the lateral rectus, the superior rectus of the inferior rectus, and the superior oblique of the inferior oblique.

With a careful look on Fig. 2 one can pick up many more pairs of agonist and antagonist muscles. It is important to pick correct agonist and antagonist muscles because both are modelled, as will be shown subsequently in a slightly different manner.

2.2.2 Howe Muscular Servomechanisms :

As the observer looks at an object an image is formed on the retina. Now if the object is moved, the image too is displaced. This error information due to retinal image motion

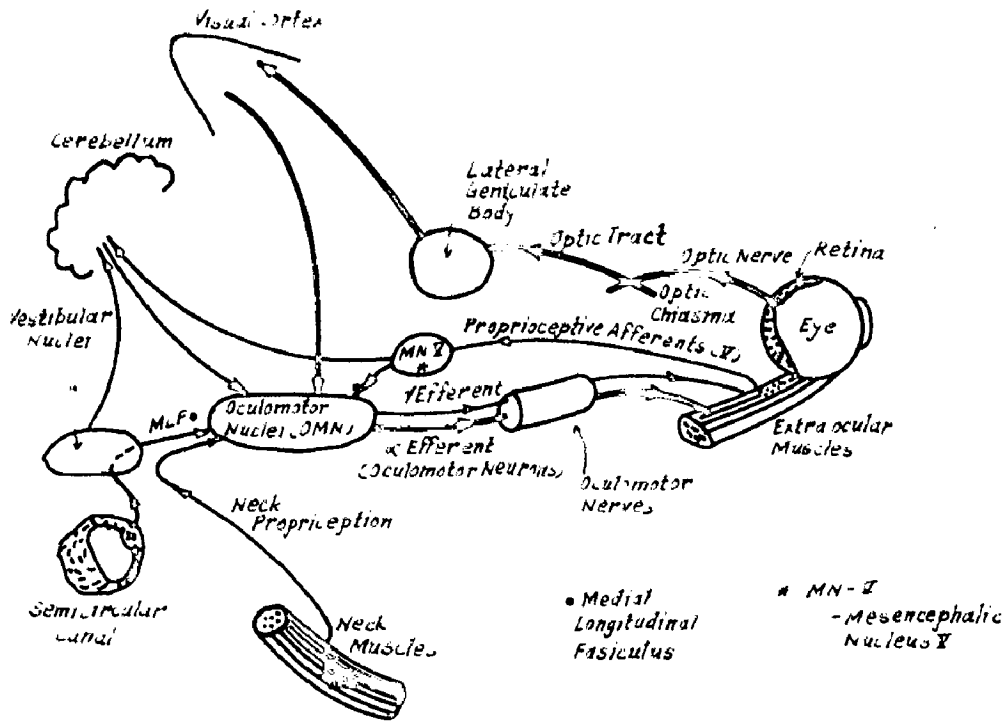


Fig.3. Simplified anatomical schematic of visual-fixation system.

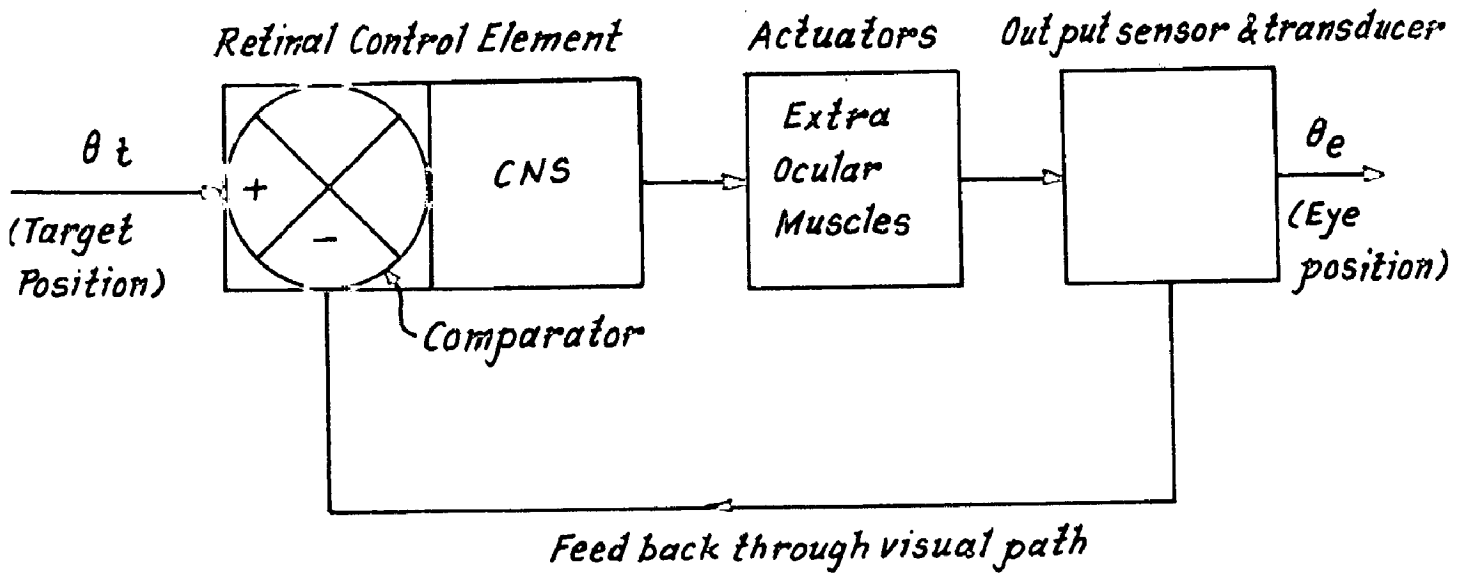


Fig. 4

motion, is transmitted to the visual cortex through optic nerve, optic chiasm, optic tract and lateral geniculate body (11. 3). Visual cortex which is a part of the CNS, then sends a command to extraocular muscles, through oculomotor nuclei and oculomotor nerves. Extraocular muscles in turn, acting in agonist-antagonist pairs, rotate the eyeballs in their orbits to re-adjust the retinal image onto fovea which is the maximum visibility area of the retina. The function of the control system governing eye movements is thus to acquire visual targets rapidly and once acquired, to stabilize their images on the retina despite relative movements between the target and the observer. Thus vision, and hence, perception, is achieved by stabilizing the retinal image by means of feedback of retinal image motion. The eye servomechanism is shown as block diagram in Fig. 4.

Open - and closed-loop behaviour of the eye movement mechanism or control system has been studied by Fender and Hyc (15) and also by Fender (16). They characterised the eye servomechanism in terms of linear operators and studied the frequency response of the eye tracking mechanism by varying the stimulus amplitude and the amount of visual feedback. By giving the external loop a gain of + 1, total feedback could be reduced to zero and thus a stabilized image was obtained (16). This enabled the study of the open-loop transfer function characteristics of the subject and the open-and

closed-loop gain and frequency response characteristics of the eye servomechanism could be compared. This way Fender (16) has shown that eye servomechanism is a negative feedback control system.

Eyeball servomechanism has also been studied by Milsum and Jones (18) with special reference to vestibular stabilization. It had been shown earlier by Van Egmond et al (bibliography), that semicircular canals constitute an angular inertial accelerometer. Over the bandwidth of 0.017 to 17 Hz the semicircular canals act like integrating accelerometer and the normal discharge (signal) to CNS is a frequency coded analog of head velocity. Milsum and Jones have taken into account four main feedback channels (Fig. 3) as enumerated below:

(i) Visual feedback : The visual channel consisting of optic nerve, optic chiasm, optic tract and lateral geniculate body is considered the main feedback path. From visual cortex an efferent signal via oculomotor nuclei and oculomotor nerves is provided to the extracocular muscles for actuation.

(ii) Vestibular feedback : Any movement of the head is converted by the semicircular canal, into a frequency coded signal to the cerebellum. The vestibular channel receives its information from the cupulae of the semicircular canals, The vestibular nerve carries the information to the vestibular nuclei in the brain stem where it proceeds via the medial

longitudinal fasciculus to the oculomotor nuclei. The semi-circular canals provide the main dynamic drive to compensate the eyes for the short, sharp, angular head movements of every day life.

(iii) and (iv) Proprioceptive feedback paths : Eye movement in space can also be brought about by movement of the head on the body and of the body in space. These two movements that of eye in skull and skull on body provide two proprioceptive feedback paths for stabilizing retinal image.

Thus we have an overall feedback system consisting of visual, vestibular and two proprioceptive feedback channels. These are the main sources of information with which the oculomotor nuclei synthesize the actuating drive signals for eye-in-skull control. The overall system is shown in Fig. 12.3 of Milsum's book (bibliography).

Milsum and Jones placed more emphasis on vestibular feedback and they found a second order transfer function between skull acceleration and cupulae deflection and the role of semicircular canals as an angular velocity transducer was proposed.

2.3 Eye Movements

Various types of eye movements have been classified in literature. But clinical tests demonstrate four basic

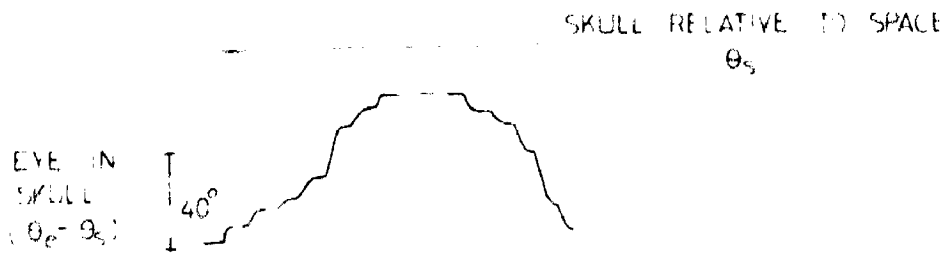


FIG 5 - PURE SACCADIC MODE OF EYE MOVEMENT - SKULL STATIONARY.

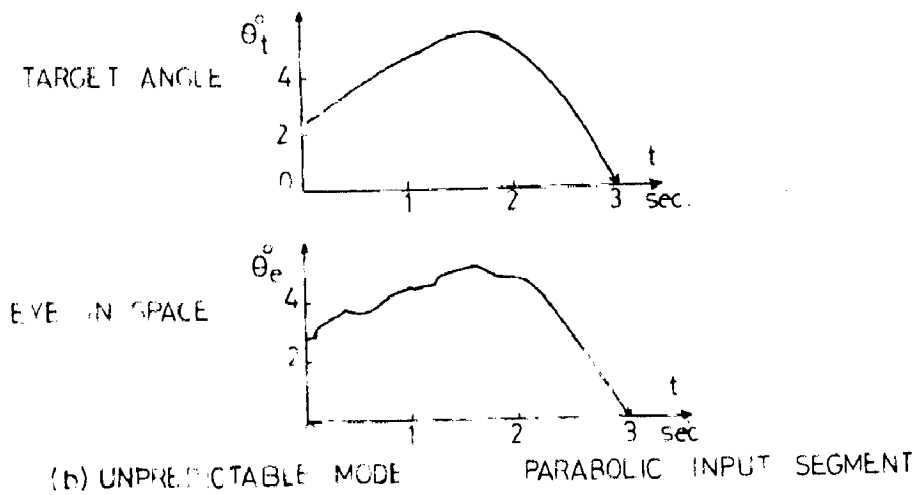
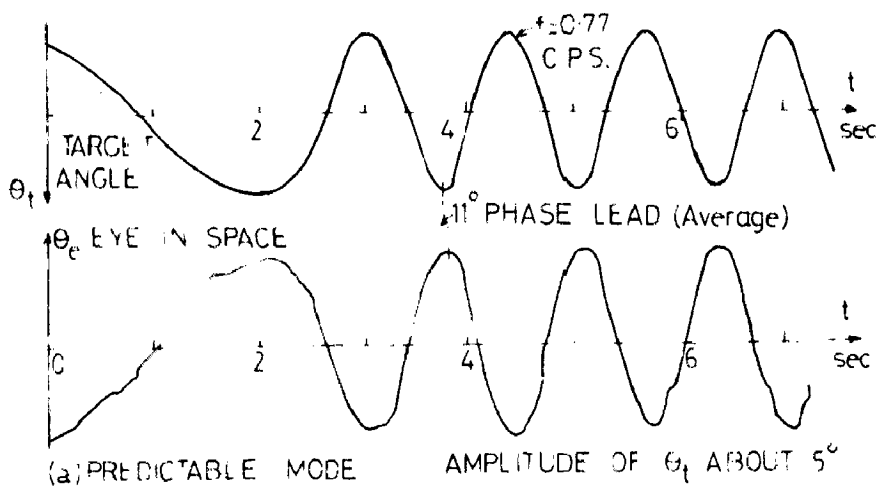


FIG 6 - THE SMOOTH-PURSUIT MODE OF EYE MOVEMENT - SKULL STATIONARY

different types of eye tracking movements.

2.3.1 Versions

Two of these movements are version movements. These types of eye tracking movements are conjugate i.e. both eyes move equally in the same direction. They are described below :

(i) Saccadic : These are step like (Fig. 5) movements found in reading and are standard movements of every day life, carrying the eyes from one fixation point to the next. A fixation pause lasts typically for about 250 ms to 1 s and then a saccade (jump) lasting for 20 to 100 ms depending upon the length of the movement, jerks the eyes to their next position. The normal speed of saccadic movements varies from 100 deg/s to 500 deg./s but can be as high as 1000 deg./s. The reaction time or latency for saccades is 180 ms to 250 ms.

(ii) Smooth Pursuit : These are ramp like movements observed during the following of a smoothly moving stimulus e.g. a pendulum. Latency of these movements is the same as for saccades i.e. 180 ms to 250 ms. Speed-closely follows the speed of the moving objects within a range of 1 deg./s to 30 deg./s. Some subjects can follow smoothly upto 60 deg./s. Beyond this range the eye, usually falls back i.e. lags the target motion.

Smooth pursuit movements can be further subdivided into three categories.

(a) Predictable movements (Fig. 6a) are in response to a known stimulus at a constant frequency e.g. a moving pendulum generates a sine wave input and sudden movement of a target from one side to another generates a square wave input. In this mode the periodic wave form is followed essentially "exactly", except for the rather unusual feature, reported by Fender (16) and Stark et al (29) that the eye actually shows phase lead relative to the target and slight amplification of its magnitude. This is due to the learning behaviour of the eye tracking systems.

(b) In unpredictable mode the eye tracks an unknown target such as obtained by combining nonharmonic sinusoids or by generating a random signal (Gaussian noise). In this case the eye lags the target and the tracking system reverts to using the visual loop for generation of its primary drive signal in a "watch-and-follow" type of action.

(c) Third type of smooth pursuit movements are compensatory movements of the eye for passive body movements and are caused by vestibular stabilization as described in 2.2.2.

2.3.2 Vergence Movements

Still two more eye movements are vergence movements. These are movements of the eyeballs inwards or outwards relative to each other to maintain adequate binocular fixation, when changing from far to near objects and vice versa. Vergence movements are slow-speed about 6 deg./s. to 15 deg./s. No data about latency is available and these movements are less well controlled than other voluntary eye movements and seem to have received less attention at the hands of the researchers.

(i) Fusional Vergence eye movements are disjunctive and are caused by a stimulus which moves directly towards or away from the eyes; both eyes move equally in the opposite direction.

(ii) Accommodative Convergence movements are vergence movements of one eye in response to a target that moves towards or away from the other eye along its line of sight.

In addition to the main four types of eye movements described above there are involuntary miniature movements like 'flicks' and 'drifts' (16). Blinking too is present at 2 to 10 s intervals. But these movements are 'tolerable' disturbances and can be easily ignored for purposes of modelling the eye positioning mechanism.

Most of the work being presented in this dissertation

pertains to saccadic movements.

2.4 Measuring Eye Movements :

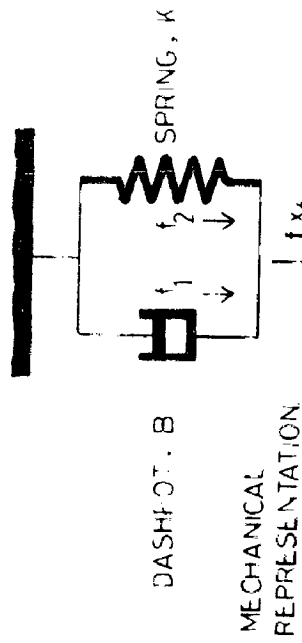
The eyes can be moved sufficiently and fix on objects within a circular area having a diameter of equal to 100° of visual angle. Rotations to the left and right are approximately equal (50°) in extent but vertical upward movements are more limited (50°) than vertical downward movements (60°). The range of eye movement is tested with a tangent screen and is an important diagnostic sign in neurology.

Methods of measuring eye movements have also been classified in literature. One of the commonest methods is the one used by Fender (16) where large eye movements are recorded by photographing the reflection of light from the corneal surface. A tiny medical lamp is placed in the stem of a mirror attached to a subject's eye. This lamp shines down onto a photo-multiplier tube. As the eye moves, the amount of light striking the tube fluctuates and the output current of the tube is, therefore, a measure of the motion of the eye. By combining two tubes we obtain data about side-to-side and up-and-down movement of the eye.

A very significant method is the one developed by Robinson (21) still another common method is to use infrared eye movement monitor (29). Actually many researchers are reporting new methods using electronic circuitry. A few of these are

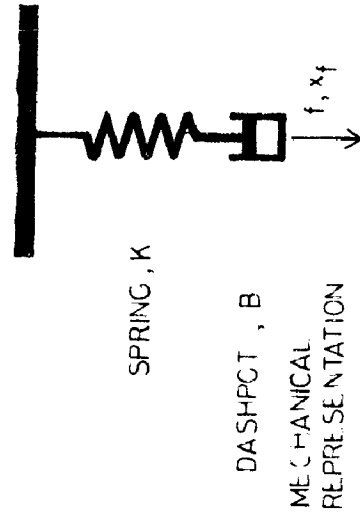
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listed in bibliography. Because we are not directly involved or interested in recording or measuring eye movements, this aspect is not elaborated any further.



APPLIED FORCE = spring force + friction force
 $f = f_1 + f_2 = B\dot{x}_f + Kx_f$

(a) VOIGT ELEMENT



DEFLECTION OF $f =$ spring deflection + dashpot deflection
 $x_f = x_s + x_D = \frac{f}{K} + B \int f dt$

(b) MAXWELL ELEMENT

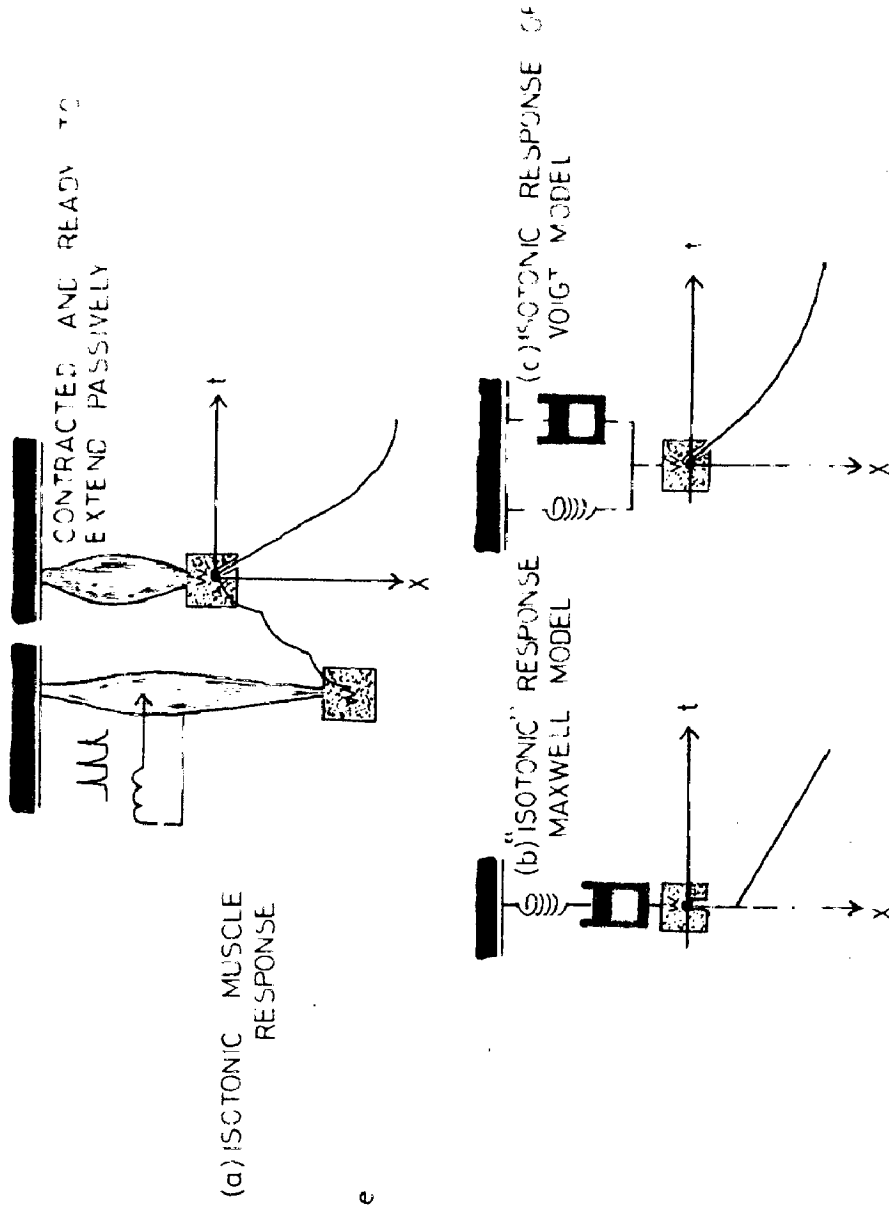


FIG 7 REPRESENTATION OF ELASTOMERIC TISSUE INCLUDING FRICTIONAL EFFECTS

FIG 8 COMPARISON OF MUSCLE AND MODEL ISOTONIC RESPONSES

3. MODELLING THE EXTRAOCULAR MUSCLES

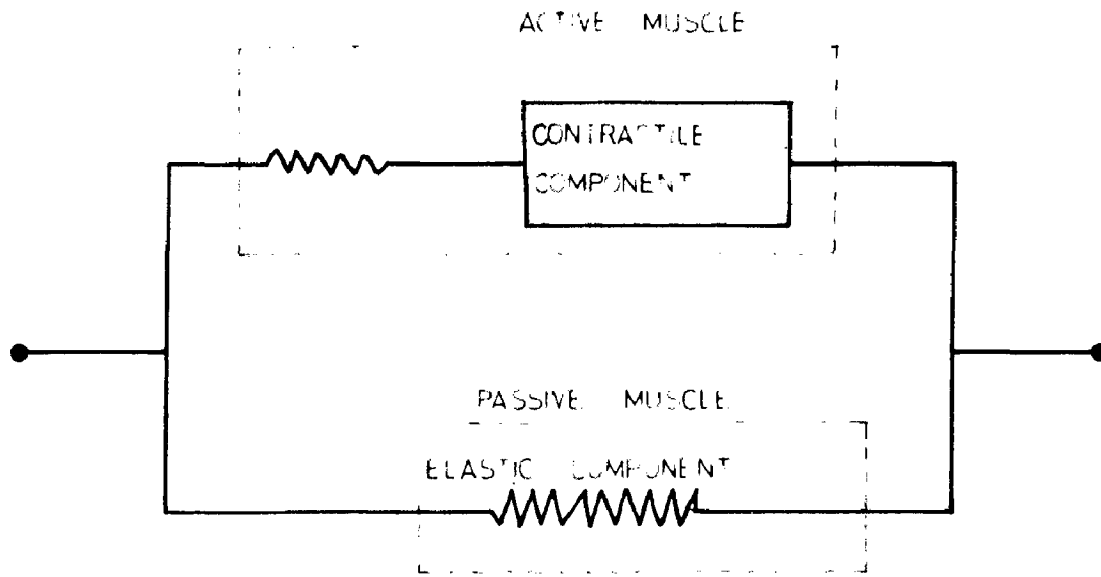
3.1 Modelling of a Muscle :

A muscle is ordinarily thought of as an active system or a source generator and its prime function is to generate force. However, if the muscle is deactivated i.e. its force-generating mechanism remains inactive, it still retains passive mechanical properties. An externally applied force, for example, will cause a deenergized muscle to stretch giving rise to a force-displacement relationship. The ratio of the force-displacement gives us the storage property of the muscle, which is nothing but the mechanical property of elasticity or springiness. The slope of the force displacement characteristic defines the spring constant of the muscle. It is, therefore, reasonable that a model simulation of a muscle should include a mechanical spring to represent the elasticity of the muscle.

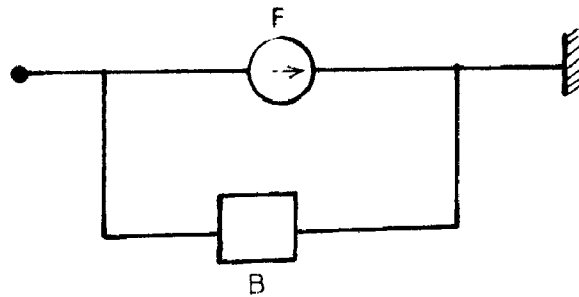
At this stage we realize, intuitively, that this representation must be incomplete as we have not accounted for frictional or resistive loss. No mechanical system is frictionless and muscle tissue is no exception. Muscles have significant resistive properties and this together with springiness, has given rise to two elementary models for the muscle. These are Voigt and Maxwell models (Fig. 7). Both these models contain a spring to represent elastic properties and also include a

dashpot (damper) to represent frictional element. A dashpot usually represents linear viscous friction. In case of a muscle we are justified in accounting for viscous friction as the muscle tissue contains fluid constituents, i.e. the mechanical friction in physiological materials is essentially of the fluid or viscous variety. Elastic tissue is often referred to as viscoelastic material.

Now, if a smooth muscle is loaded i.e. energized to contract, and then allowed to expand passively at constant load (i.e. isotonically, if, however, shortening of the muscle is prevented, stimulation of the muscle results in an increase in tension. When this is recorded, it is spoken of as an isometric contraction. In isometric contraction the muscle performs no external work, and all the energy of its contraction is converted into heat.) the response will be approximately that shown in Fig. 8(a). If the Maxwell model is allowed to respond isotonically it will immediately drop a short distance ~~to~~ until the spring extends (the dashpot cannot instantaneously acquire a velocity); it will, theoretically, then extend ad infinitum at a constant rate (Fig. 8b). This response does not conform with the response of the physiological system as shown in Fig. 8(a). Under isotonic conditions the Voigt model will initially fall at a constant rate. As the spring becomes stretched, the rate of extension is reduced until finally motion will entirely cease. This response, Fig. 8(c), is more



(c) GENERAL STRUCTURE OF THE MUSCLE



(d) CONTRACTILE COMPONENT

FIG 9 REPRESENTATION OF A MUSCLE

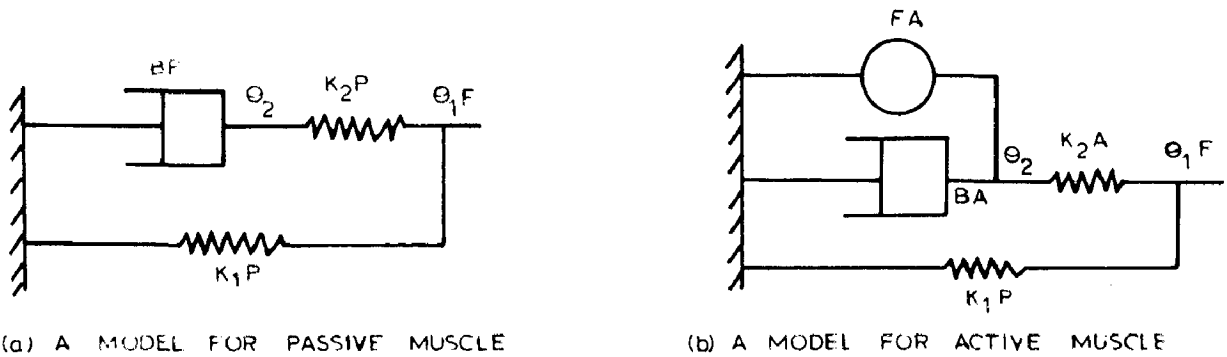


FIG. 11

in keeping with that of the physiological system (Fig. 8a). We, therefore, accept the Voigt model as a preliminary representation of the physiological system i.e. the muscle. With isometric contraction, it can be similarly, ^s shown that the Maxwell model is nearer to the actual muscle system.

As has been mentioned earlier an active muscle is a force generating element. Now incorporating this aspect into our muscle model (Voigt model) we get a force source in parallel with the dashpot. The two together form what is commonly called a contractile component (Fig. 9). Hill (17) has shown that active muscle is composed of a contractile component in series with an elastic component. The contractile component is the portion altered by the nerve signal and is capable of developing active tension. He has also shown that even with zero activation i.e. for a passive muscle when the muscle is stretched beyond a certain length, it exhibits tension even though there is no nerve signal. This characteristic can be represented as a passive elastic component in parallel with the active muscle. This is illustrated in Fig. 9.

3.2 Parameters of the Extraocular Muscle

A number of studies have been conducted on extraocular muscles of the cat, the monkey and the human being to determine their parameters. Perhaps the first, most detailed and comprehensive results have been presented by Cook & Stark (11) taking into account the then available data. Their model is

for horizontal eye movements and is constructed for lateral and medial recti. We shall take up the design of parameters one by one.

3.2.1 Resting length :

Wilkie (37) has shown that muscle length for maximum active tension, L_0 is twice the length at which the active tension first becomes nonzero. Measurements by Robinson (22) on the lateral rectus of a cat revealed its rest length to be approximately 25 mm. Robinson further shows (Fig. 8, page 254 of his paper) a stretch versus tension curve of the same muscle. By extrapolating this curve we find the distance from zero tension to maximum tension to be 12 mm. According to Wilkie, this value is equal to $L_0/2$. We, therefore, have,

$$L_0/2 \cong 12 \text{ mm} \quad \text{or} \quad L_0 \cong 24 \text{ mm}$$

It is assumed that medial rectus too behaves like lateral rectus and that the muscles of human eye movement system also have their operating point at L_0 , the length of maximum tension. For the human recti, this places L_0 at approximately 40 mm, the rest length (11).

3.2.2 Passive Muscle

The static characteristic of passive extraocular muscle is a relation between muscle length and tension (Fig. 10). The elastic force increases with extension in the manner of a hard

spring, but over a fairly wide range of lengths the forces are small enough so that approximating the static properties by a linear spring does not introduce serious error.

Linearization of the curve of Fig. 10 yields elastic coefficients ranging from 0.2 at 0° (or primarily length i.e. resting length) to 1.0 g/deg at 40° away from the field of action of the muscle. Robinson et al (27) found an elastic coefficient of 0.5 g/deg. Data due to Barmack et al (bibliography) leads to a coefficient between 0.1 and 1.25 g/deg. While Cook and Stark (11, 12, 13) arrived at 1.9 g/deg. for both extraocular muscles. Cook & Stark erroneously assumed a maximum force of 500 g. If this is reduced to the observed maximum of 100 g (27), the coefficient becomes 0.38 g/deg for both muscles at 0° . Assuming both muscles contribute $\frac{1}{2}$ equally, the result becomes 0.19 g/deg. for one muscle. Researchers have thus shown variations of K_{1P} , the passive spring constant (Fig. 11a) ranging from 0.1 to 0.5 g/deg. at 0° and from 0.67 to 1.25 g/deg at extreme lengths. It has been shown by Clark and Stark (6) from dynamic quick release experiments that K_{1P} of about 0.4 g/deg yields good static and dynamic results. Similarly they have determined K_{2P} to be 1.6 g/deg with a time constant T_p of B_p/K_{2P} sec. The time constant T_p is approximately 0.1 s (6), which yields a B_p of 0.16 g.0/deg.

It must be noted that this model is valid only for length or force at lengths greater than the primary length. It

predicts zero force at this length. However, since it has been shown (27) that the passive muscle force is at most 2 or 3 g at length below the primary length, this assumption does not seriously affect the validity of the model in approximating static or dynamic passive muscle; it also simplifies the analysis considerably.

3.2.3 Active Muscle

Fig. 10 also shows the static properties of active muscle. We must be, of course, careful to subtract forces due to passive visco-elastic element in order to find the change in muscle due to active state. Robinson et al (27) found elastic constants ranging from 0.5 to 0.825 g/deg. Barmack et al (bibliography) found coefficients from 0.33 to 1.6 g/deg. Using Fig. 10 and data on quick release on active lateral rectus of the cat ^CClark and ^SStark (6) have found $K_{1A} = 0.33$ g/deg. $K_{2A} = 2$ and viscosity coefficient $B_A = .10$ g-sec/deg. This value of B_A corresponds to a time constant for active muscle of 50 ms.

Summarizing the parameters for active and passive muscles we have

$$\begin{array}{ll}
 E_{1P} = 0.4 \text{ g/deg.} & K_{1A} = 0.33 \text{ g/deg. } \text{ (0.33)} \\
 E_{2P} = 1.6 \text{ g/deg.} & K_{2A} = 2.0 \text{ g/deg. } \text{ (2.0)} \\
 B_P = 0.16 \text{ g-sec/deg.} & B_A = 0.10 \text{ g-sec/deg. } \text{ (0.10)}
 \end{array}$$

From the foregoing we can see that $K_{1P} \cong K_{1A}$,
 $K_{2P} \cong K_{2A}$, $B_P \cong B_A$, $T_A \cong T_P$. These results imply that
 active and passive muscles may be approximated by one Maxwell
 model (Refer para 3.1 above) and these have been shown in
 Fig. 11.

Wilkie's (37) quick stretch data was interpreted by
 Cook and Stark (11) and they calculated a value of 1.86 g/deg
 for $K_{1A} + K_{2A}$ which is in approximate agreement with 2.33 g/deg
 found above. This agreement corroborates the values calculated
 here.

Finally, we assume the following parameters :

$$\begin{aligned}
 K_{1P} &= K_{1A} = K_1 = 0.36 \text{ g/deg.} \\
 K_{2P} &= K_{2A} = K_2 = 1.8 \text{ g/deg.} \\
 B_P &= B_A = 0.15 \text{ g-s/deg.} \\
 T_P &= T_A = 75 \text{ ms}
 \end{aligned}$$

For evaluating active and passive time constants two
 methods are available. One relies on taking the slope of the
 quick release data as it rises back to steady state; the other
 involves approximately the time required to achieve 98%
 of steady state. The noise in the data makes the utilization
 of these techniques difficult, therefore, the results are
 only indicative of the approximate time constants. Clark &
 Stark (6) calculated the time constant of 5-10 ms for both

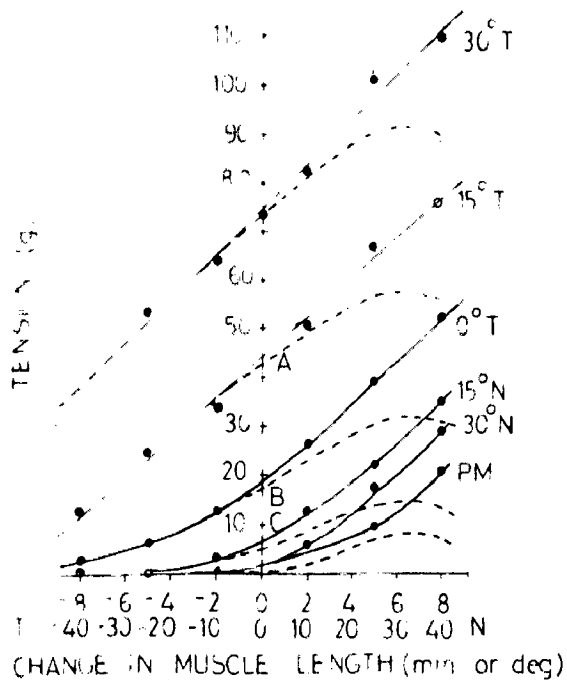


FIG 10 - STATIC LENGTH-TENSION CURVES FOR EXTRAOCULAR MUSCLE. CURVE PM IS PASSIVE MUSCLE TENSION. SOLID LINE INDICATES TOTAL TENSION, AND DASHED LINES INDICATE TOTAL TENSION MINUS CURVE PM (from Robinson, Collins, O'Meara, and Scott. [27])

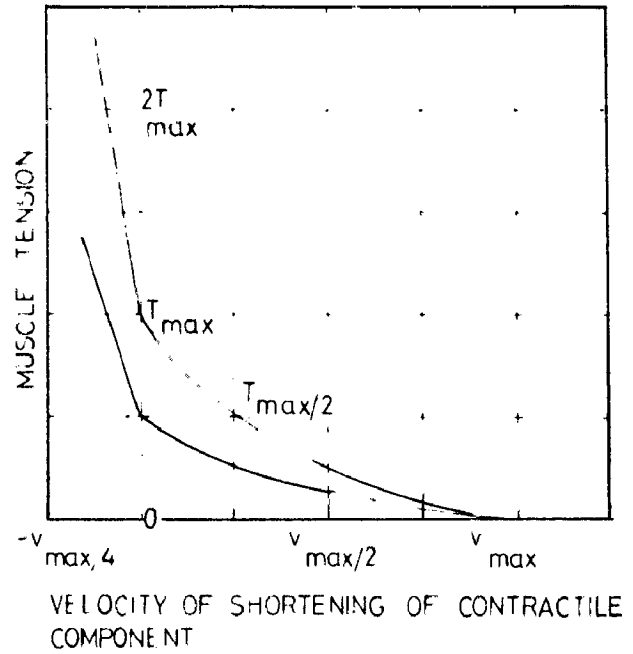


FIG 12 - THE FORCE-VELOCITY RELATIONSHIP FOR ACTIVE MUSCLE (from Cook and Stark (11))

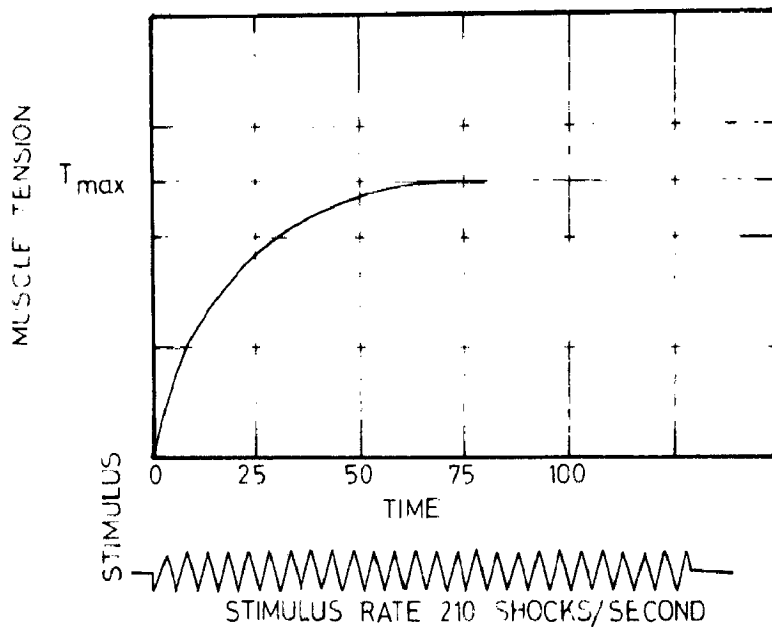


FIG 13 THE RISE OF ISOMETRIC TENSION AS THE MEDIAL RECTUS OF A CAT IS MAXIMALLY STIMULATED TO A STATE OF TETANUS (from Cook and Stark (11) after Wilkie)

active and passive muscle by the first method. The second method yielded values of 100 ms for passive muscle and 50 ms to 150 ms for active muscle. It should be noted that B_A and B_P are also altered proportionately. Although the two methods give different values, the constants are similar in active and passive muscle when one particular method is used.

There is some experimental justification which suggests that this variation is not due only to experimental error. Hill (17) has shown that the force-velocity relationship of muscle, which is related to the viscous force, is non-linear and decreases with increased velocity. This is shown in Fig. 12. Now if we calculate the time constant using the initial slope technique, where the velocity is high and the viscosity low, the time constants would be small. As the velocity decreased the viscosity would increase and so would the time constant; hence the second method would yield a higher time constant.

To calculate B_P , we subtract the passive muscle force at each length, and divide the resultant force by velocity. We find that the viscous coefficient decreases with the increase in velocity. Hence the values of B_A and B_P given above are probably somewhat high because these were calculated with the low velocity characteristics of the quick release experiments.

Hill (17), who has spent many years studying muscle behaviour, advanced the most accepted analytic description of shortening active muscle behaviour in the following equation

$$v(P + a) = b(T_0 - P) \quad \dots \quad (3.1)$$

where, v = Velocity of shortening; P = Total force,

$$a \cong 1/4 T_0 ; \quad b \cong 1/4 V_{\max} ; \quad T_0 = \text{Maximum isometric force at } L_0.$$

Hill's force-velocity relationship will, therefore, be used to describe the viscous properties of the extraocular muscle. Also Abbott and Wilkie (bibliography) have shown that this curve, Fig. 12, is valid not only at L_0 , the length of maximum isometric tension, but at all other lengths if T_0 becomes a function of length. Rearranging Hill's equation given above, we get

$$P = T_0 - \frac{1.25 T_0}{b + v} v \quad \dots \quad (3.2)$$

Therefore the damping coefficient for shortening velocities becomes

$$B_s = \frac{1.25 T_0}{b + v} \quad \dots \quad (3.3)$$

The coefficient b may be calculated with the isometric force-time response as the muscle is stimulated to develop maximum force T_{\max} . This is shown in Fig. 13. Referring to Eqn. (3.2) and Fig. 11(b), we see that $P = F = 0$ and $\dot{\theta}_4 = \dot{\theta}_2 = 0$ at $t = 0$ and that $P = F_A$. Solving for b in equation (3.2) with these assumptions, we get

$$b = 0.25 v(0) = 0.25 \dot{\theta}_2(0) \quad \dots \quad (3.4)$$

Since $\dot{\theta}_1(t) = 0$ for all t in the isometric case

$$\text{Now at } t = 0, \dot{F} = K_2 \dot{\theta}_2(0) \quad \dots \quad (3.5)$$

$$\text{This gives } b = 0.25 \dot{F}(0)/K_2 \quad \dots \quad (3.6)$$

From Fig. 13, $\dot{F}(0) = 75 T_{\max}$, which when substituted into Eqn. (3.6) along with $K_2 = 1.8$ and $T_{\max} = 100$ gives

$$b = 1000 \text{ deg/sec.} \quad \dots \quad (3.7)$$

Will come later

This agrees well with the value of 900 found by Cook & Stark (12).

Katz (bibliography) has shown that the characteristics of lengthening muscles are quite different from those of shortening muscles. This is also shown in Fig. 12. Taking the slope of this curve yields a viscosity for lengthening of

$$B_L = \frac{3 T_0}{b} \quad \dots \quad (3.8)$$

This analysis calls for a correction. It has been assumed that the applied force F in Hill's equation (3.4) is equal to the force of the series spring in the Maxwell model. This is true only if the parallel elastic force is zero at the end of the experimentation, L_0 ; Fig. 10 shows that this assumption does not hold at L_0 , since the passive force is approximately 3 - 10 g. This passive force must be subtracted from the total force exerted on the muscle to yield the

force-velocity relationship for the viscous element depicted in the Maxwell model. Even if we assume a force of 10 g. this is only 10% of T_{\max} and reduces B_v for shortening by 10%. The lengthening viscous force will be more seriously affected by the greater length of K_1 , but even this will not change the value of B_v significantly. In conclusion it is assumed that both passive and active muscles have viscous coefficients which are velocity dependent.

Hence the mechanical components of the model shown in Fig. 11 can be summarized as :

$$\begin{aligned}
 K_1 &= 0.36 \text{ g/deg.} & K_2 &= 1.8 \text{ g/deg.} \\
 B_A &= \frac{1.25 F_A}{1000 + \dot{\theta}_2} & \dot{\theta}_2 &\geq 0 \text{ i.e. shortening muscle} \\
 &= \frac{3F_A}{1000} & \dot{\theta}_2 &< 0 \text{ i.e. for lengthening muscle}
 \end{aligned}$$

3.2.4 Active Tension F_A

Now we must study the statics and dynamics of the active tension source F_A of the contractile element. Fig. 10 shows the static characteristics of the active muscle; the dashed lines show the length tension relation for the active tension source F_A and elasticity K_2 . Fig. 14 displays Wilkie's (37) quick stretch data showing the elastic characteristics of the series element K_2 . By subtracting the length of K_2 at each tension, we can calculate the length-tension curve for the source T as shown in Fig. 15.

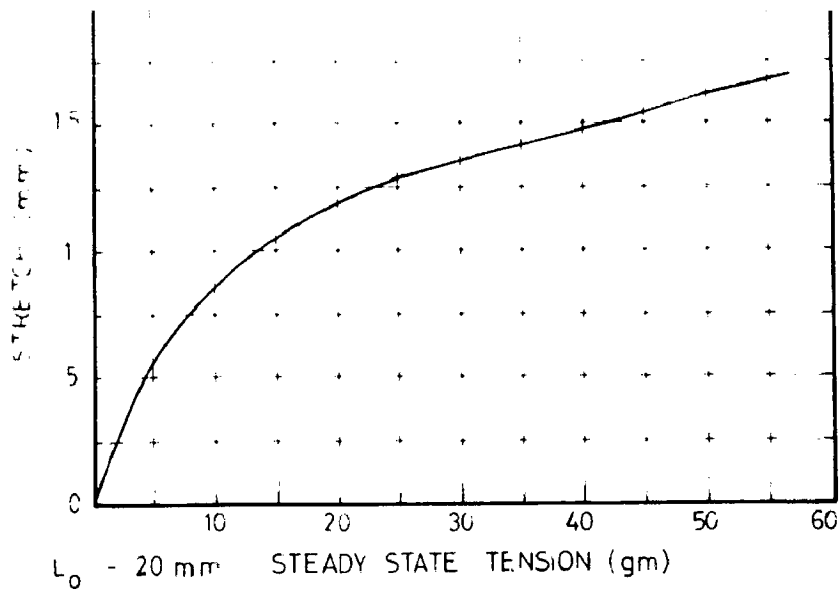


FIG.14 - STRETCH-TENSION CHARACTERISTICS FOR THE SERIES ELASTIC COMPONENT K_2 TAKEN FROM DATA BY WILKIE ON THE FROG SARTORIOUS MUSCLE (from Cook and Stark [11])

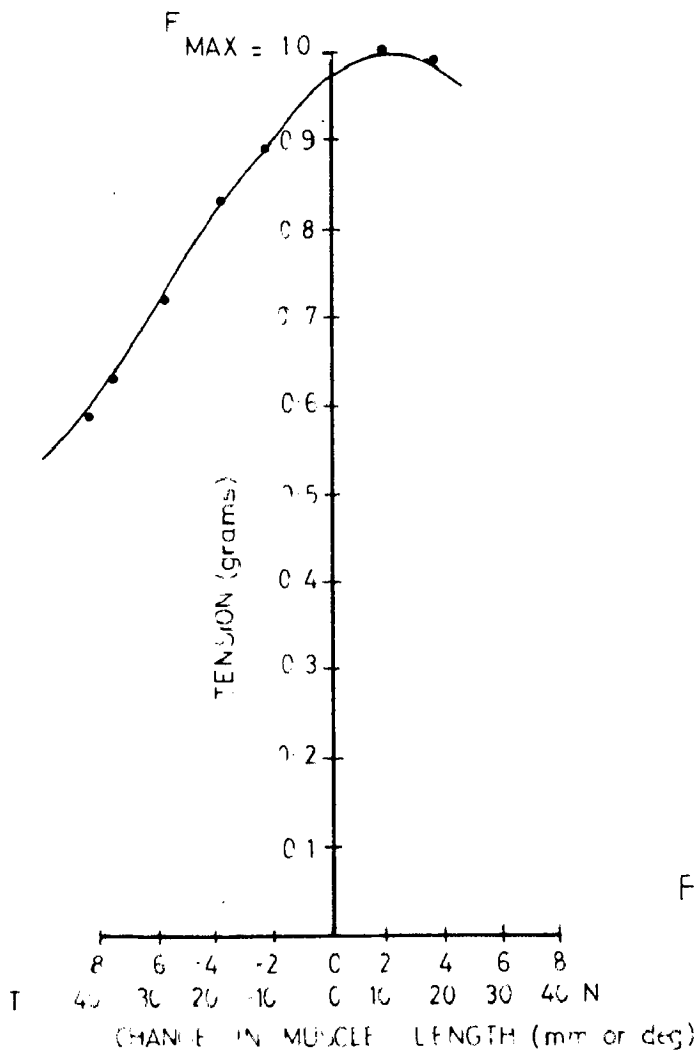


FIG.15 LENGTH-TENSION CHARACTERISTICS OF THE CONTRACTILE COMPONENT DERIVED FROM EQUATION (3.9) USING THE DATA OF FIGURE 14 AND

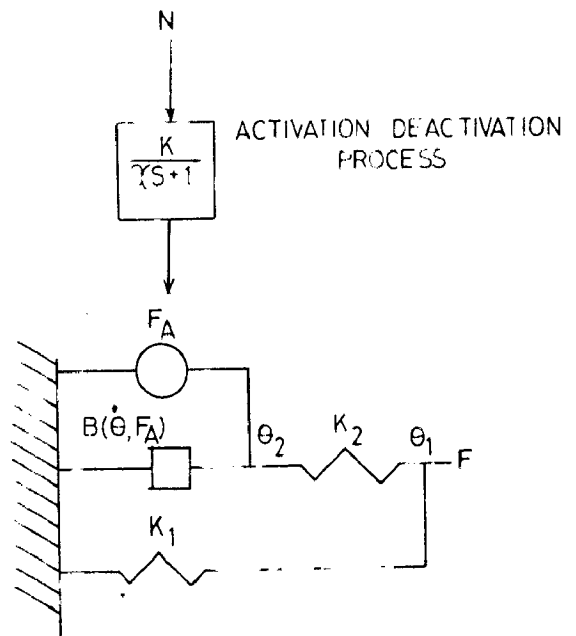


FIG.16 - THE FINAL MODEL FOR ACTIVE AND PASSIVE EXTRAOCULAR RECTUS MUSCLES

The governing equation is

$$L_{AS}(T) = L_{AM}(T) - L_{K2}(T) \quad \dots \quad (3.9)$$

where, L_{AS} = Length of active tension source

L_{AM} = Length of total muscle

L_{K2} = Length of series elastic element K_2

T = Tension of active muscle.

It is evident from Fig. 15 that maximum isometric force of the active tension source occurs when its length is near the primary rest length of the muscle and that the source is length dependent. This length dependence is included in the quick release simulations of the active and passive muscles. A static curve for the force F_A as a function of length has been realized by Clark & Stark (6) (Their Fig. 11) by creating a locus of tonic force on the muscle versus its length.

Ritchie and Wilkie (bibliography) have shown with quick stretch experiments that the onset of maximum tension in the source F_A is very rapid and occurs long before maximum muscle tension is developed. They also showed that the transition to maximum velocity is almost instantaneous, we may therefore conclude that the mass of muscle may be neglected in modelling its characteristics. Wilkie (37) found that the time constant of deactivation for frog sartorius at 0°C

was about 100 ms. For activation it was much shorter. It has also been found elsewhere (6) that for a 10° C rise in temperature the twitch/tension ratio decreased by a factor of 3.5, presumably due to a faster tension source. The active source tension would not be observed, due to the fact that the twitch would be completed before the slower mechanical components could respond. This temperature dependence implies a time constant of deactivation about 8 ms for the extraocular muscle at body temperature (6). It will be assumed that activation is at least twice as fast, or 4 ms. Robinson (bibliography) and other researchers have found a fairly linear relationship between the nervous activity in the oculomotor nuclei and the tension in the active source F_A ; tension is also a function of the length and velocity of the muscle. The muscular response will be dependent on the number of motor neurons active as well as their level of activity - the activation-deactivation process is a simplified representation of this pool of motor neurons. It is assumed that these processes can be depicted as a low pass filter with activation deactivation time constant and a steady state constant of Kg/spike. The tonic nervous activity approximate to different muscle lengths has been studied by many researchers and will be taken up subsequently. Fig. 16 shows the final version of the model of the extraocular lateral or medial rectus.

3.3 A Model of the Muscles for Vertical eye movements

The model developed above in 3.2 is for eye mechanism for horizontal eye movements and is based on the two extraocular muscles - medial and lateral recti involved in the horizontal movement. For vertical movements different set of extraocular muscles is involved and a model for vertical eye ~~mov~~ movements must be based on these muscles only. Before developing this model, let us see as to which are the extraocular muscles responsible for vertical eye movements.

3.3.1 Muscles concerned in elevation of the eye

When the eye is moved straight up, the superior oblique and the inferior rectus are inhibited, whereas the other four muscles maintain their normal tone or contract. The superior rectus and the inferior oblique elevate the eye, the former predominating when the movement starts from the primary position. The abducting and adducting (i.e. temporal and nasal rotations respectively) action of these muscles oppose each other and keep the eye steady in its upward course. The same is true of the lateral and medial recti. The vertical meridian is kept vertical by the counteracting torsional activity of the superior rectus and the inferior oblique (See Fig. 2).

We have said above that the superior rectus plays more part in elevating the eye from its primary position.

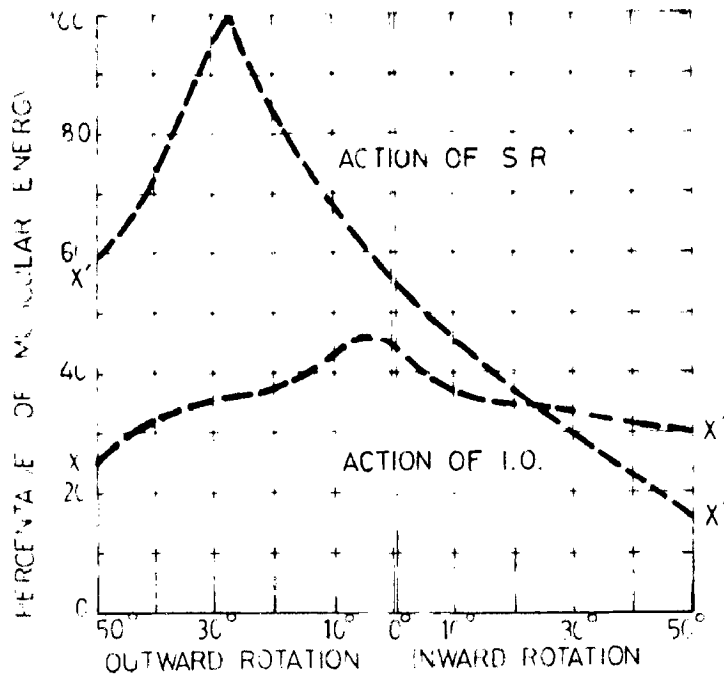


FIG 17. ACTION OF MUSCLES CONCERNED IN ELEVATION OF THE EYE.

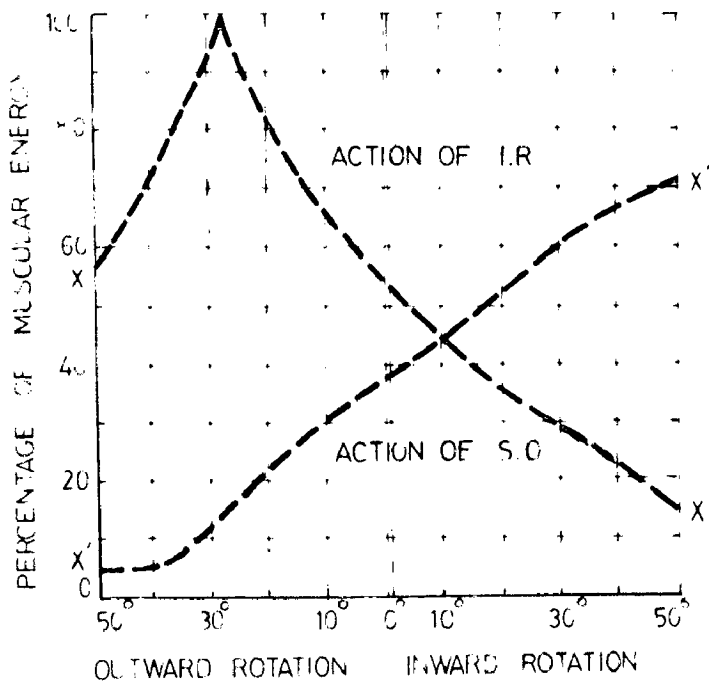
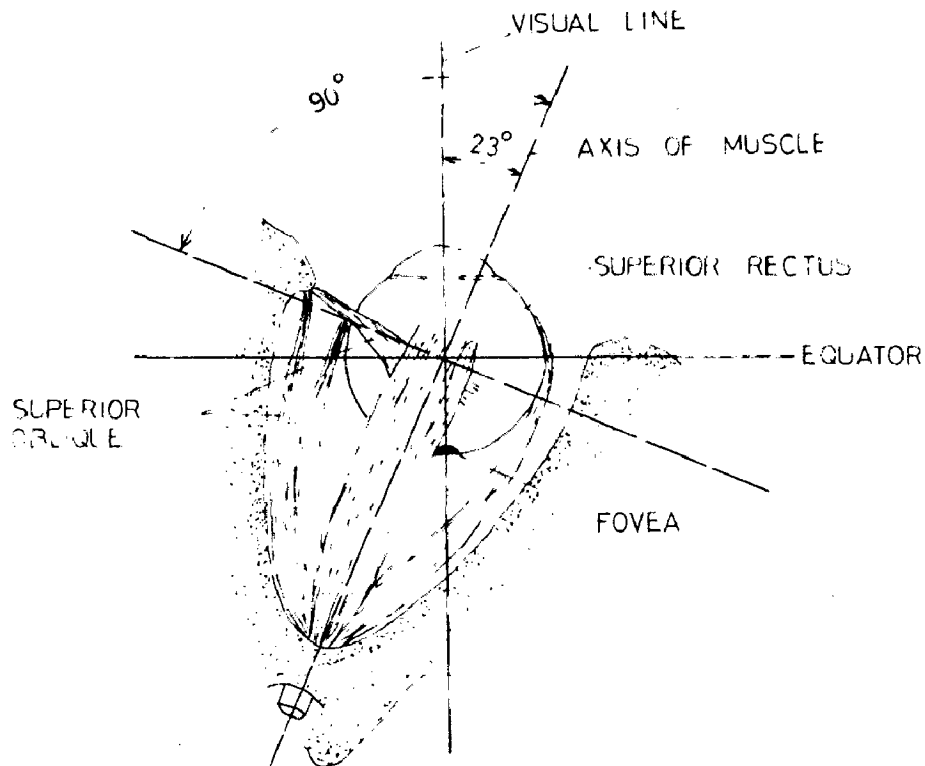


FIG 18. ACTION OF THE MUSCLES CONCERNED IN DEPRESSION OF THE EYE.

But how much? To determine the share of muscular energy supplied by each of the two concerned muscles Fig. 17 has been constructed from data from Adlor's physiology (bibliography). From these curves between extent of eye movement and percentage of muscular energy, we find that superior rectus supplies 55% of muscular energy for elevation. Inferior oblique supplies 42% (The remainder 3% is supplied by medial and lateral recti. This is not shown in Fig. 17). Thus for modelling purposes we can without introducing any significant error, assume that superior rectus and inferior oblique provide muscular energy in the ratio of 60% : 40%.

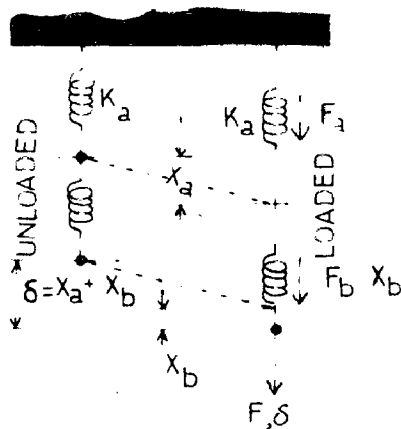
3.3.2 Muscles concerned in Depression of the eye

All the ocular muscles are innervated at the start of the movement except the superior rectus and inferior oblique. These are inhibited. The eye is carried downwards by the inferior rectus chiefly, with the superior oblique adding a lesser share. The abducting and adducting action of these muscles counter act each other (See Fig. 2) as do the abducting and adducting actions of the lateral and medial recti, so that the eye is kept from swerving to the right or left as it moves down. The torsional actions of the inferior rectus and superior oblique nullify each other, and the vertical meridian therefore remains erect.



(a)

DIAGRAM OF MUSCLES SEEN FROM ABOVE I.R. AND I.O. ARE ATTACHED ON THE OTHER SIDE OF THE GLOBE.



(b)

FIG. 19 - SPRING IN SERIES

Referring to Fig. 18 which is again based on data from Adler's physiology of the eye, we find that inferior rectus and superior oblique, provide respectively 54% and 39% of the muscular energy for depression of the eye. These can be approximated to 60% : 40%, thus neglecting the depression caused by medial and lateral recti.

3.3.3 Mechanical model for vertical eye movements :

From the foregoing we see that in case of vertical movements of the eye two muscles are concerned unlike in horizontal movement, where only one at a time is involved. We shall reduce the combination of these two muscles to an equivalent single muscle representation.

A careful reference to Fig. 19(a) will show that when the eye is elevated or depressed the concerned muscles act as if two springs are connected in series on opposite sides of the globe and move the eyeball together. Thus for mechanical representation of these muscles we can draw two springs in series as shown in Fig. 19(b). Both the springs deflect different amounts, although all carry the same load. The deflection of the total system is the sum of the deflection of each spring. As a consequence, the reciprocal spring constant of the total system is equal to the sum of the reciprocal spring constants of the component springs. Thus for Fig. 19(b) we have,

$$1/K_a + 1/K_b = 1/K \quad \dots \quad \dots \quad (3.10)$$

The same applies to damping coefficients.

Now, there is no data available in the literature for various parameters i.e. K_1, K_2, B for superior, inferior recti or superior, inferior obliques. The values of these parameters for the extraocular muscles concerned in vortical movements shall therefore be assumed to be the same as medial and lateral recti i.e. these parameters shall be taken from 3.2. Earlier, we have seen in 3.3.1 and 3.3.2 that out of the two extraocular muscles involved in depression or elevation, one provides 60% muscular energy and the other 40%. Using this fact and equation (3.10) above, we get,

$$1/K_1' = 1/.6 K_1 + 1/.4 K_1 = 1/.24 K_1 \quad \dots \quad (3.11)$$

$$\begin{aligned} & \therefore K_1' = .24 K_1 \quad \left. \vphantom{\begin{matrix} K_1' \\ K_2' \\ B' \end{matrix}} \right\} \\ \text{Similarly } & K_2' = .24 K_2 \quad \left. \vphantom{\begin{matrix} K_1' \\ K_2' \\ B' \end{matrix}} \right\} \quad \dots \quad \dots \quad (3.12) \\ \text{and } & B' = .24 B \quad \left. \vphantom{\begin{matrix} K_1' \\ K_2' \\ B' \end{matrix}} \right\} \end{aligned}$$

Where K_1' etc. are the parameters for the new model for extraocular muscles involved in vortical movements. The values of K_1 etc. are the same as in 3.2.3.

We have thus obtained a mechanical representation for the two extraocular muscles concerned in vortical eye movement. This model has only one muscle equivalent to two concerned and is the same as Fig. 12, but it has different parameters given by Eqn. (3.12).

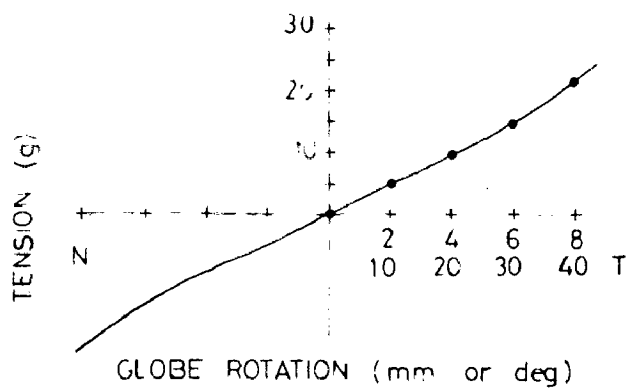


FIG.20. THE ELASTIC CHARACTERISTICS OF THE HUMAN GLOBE IN ITS ORBIT WITH THE LATERAL AND MEDIAL RECTUS MUSCLE DETACHED (from Robinson, Scott O'Meara and Collins.) [27]

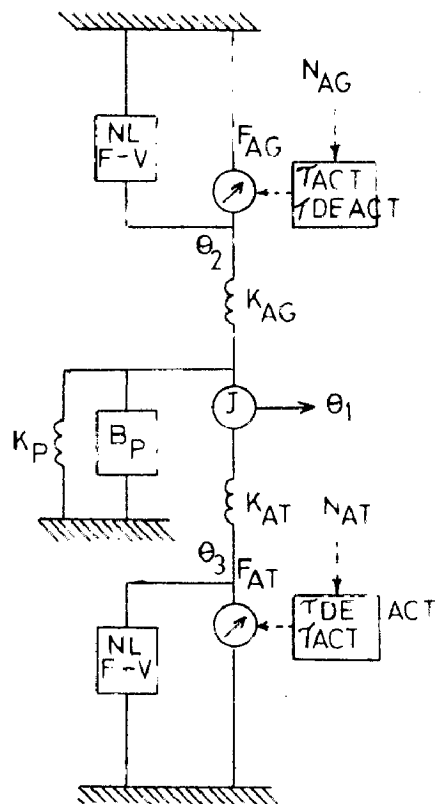


FIG.23. THE FINAL REPRESENTATION OF THE EXTRAOCULAR PLANT FOR SACCADES AND SMOOTH PURSUIT.

4. MODELLING OF GLOBE-ORBIT

It is the globe which is rotated by the extraocular muscles. Therefore, the static and dynamic characteristics of the globe and orbit must be considered and included in an overall eye positioning mechanism.

4.1 Static Characteristics

4.1.1 Elasticity of the globe

The passive properties of isolated globe are shown in Fig. 20. It is evident that the relationship between tension and globe rotation is approximately linear over a range of ± 40 degrees. The vertical eye movements whether elevation or depression rarely exceed this limit. From Fig. 20 we get spring constant for the globe as 0.5 g/deg. This value was taken as zero by Cook & Stark (11).

4.1.2 Inertia of the Globe

The density of the globe is approximately 1 g/cc and its radius is 11 mm (11). Assuming a rigid sphere, the maximum movement of inertia about a horizontal/^{axis}(for vertical eye movements, for horizontal eye movements, inertia about a vertical axis is to be taken) through the centre of rotation of the eye is 2.7 dyne cm sec²/Rad. In terms of tension referred to the muscles, this is 4.3×10^{-5} gm - tension/deg/sec (11).

We know that the eyeball is not rigid, but is composed largely of vitreous humour and that a more accurate model might be a series of spherical shells connected successively by springs and dashpots. However, for simplicity and also because the effect of this inertia is expected to play a very small role in the overall behaviour, we choose to retain the cruder rigid-sphere approximation. The validity of this assumption can be checked when the overall model is completed.

Simulations of saccadic accelerations show a variation in maximum acceleration, from 1×10^4 to 7×10^4 (7). The inertial force that the globe exerts on that extraocular muscle is $J_g \ddot{\theta}$ or from 0.43 to 3 g. Since the extraocular muscles are capable of producing 100 g of tension, the inertial force of the globe is very small, or almost negligible. Due to the fact that the eye is not quite a sphere and part of the vitreous body is left behind during a saccade, the inertial value may be a little high; but it has been shown by Robinson (22) that increasing the mass of the globe a hundred fold only causes slight oscillation i.e. mass of the globe has little effect on dynamic behaviour. Hence this value of inertia, equal to 4.3×10^{-5} g-sec²/deg. will be assumed.

4.2 Dynamic Characteristics - Viscous damping

A typical quick release experiment for the isolated globe depicts a viscoelastic element with a time constant of

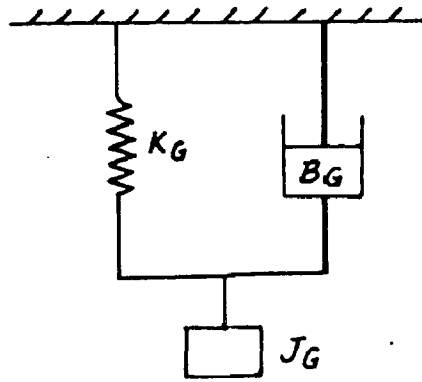


Fig. 21 - model of the inertial and viscoelastic properties of the globe and orbit.

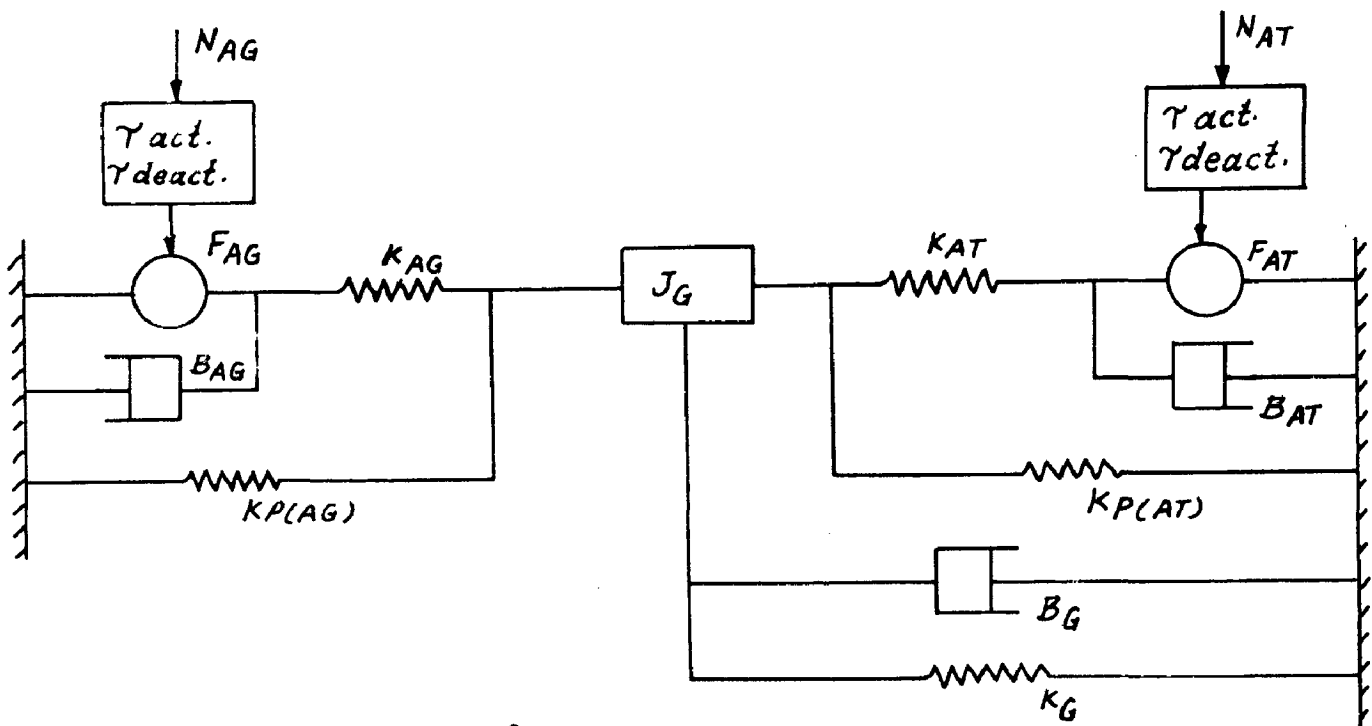


Fig. 22

about 10 ms (7). Since time constant = B/K , it implies a viscosity of about 0.02 g - s/deg. This is in close agreement with value of 0.019 found by Cook & Stark (11) and a value of 0.025 calculated by Robinson (22). There is no evidence for or against the viscous coefficient being velocity dependent. It will, therefore, be assumed that it is a constant.

4.3 A model for the globe and orbit

Fig. 21 shows a model of inertial and viscoelastic properties of the globe and orbit. In summary, the parameters for isolated globe and the orbit are :

$$J_G = 4.3 \times 10^{-5} \text{ g - sec}^2/\text{deg.}$$

$$B_G = 0.015 \text{ g - sec/deg.}$$

$$K_G = 0.5 \text{ g/deg.}$$

The viscous coefficient has been modified to take into account the inertia of the globe while realizing a simulated time constant of about 12 ms (7) if the system is considered to have no mass and be a one-time constant system.

5. AN INTEGRATED MODEL FOR EYE MOVEMENT MECHANISM

5.1 Review of eyeball movement models

Before developing integrated model for eye positioning mechanism, it would be quite in order to review briefly, the attempts made, so far to study eyeball movement system.

5.1.1 Initial Studies

Westheimer (34, 35) was the first to develop a linear approximate second order model for eye dynamics during a saccade in which the forcing function was an instantaneous change of muscle torque on the globe. He noted an effective damping coefficient of 0.7 and an undamped natural frequency of 120 rad./sec. He also observed that the eye tracking system was not strictly linear, due to the non-linear relationship of the velocity and amplitude of saccades. This model does not account for very distinct properties of the agonist and antagonist muscles.

Open-and closed-loop behaviour of the eye movement control system was first studied by Fender & Nye (15) and has been presented by Fender (16). They characterized the eye servomechanism in terms of linear operators. They studied the frequency response of the eye tracking mechanism by varying the stimulus amplitude and the amount of visual feedback.

By giving the external loop a gain of + 1, they could reduce the total feedback to zero and get a stabilised image (16). This enabled them to study the subjects open-loop transfer function characteristics. The composite open-loop transfer function is known experimentally to be one. Therefore, the Open-loop transfer function of the eye movement control system should exhibit minimum phase or greater than minimum phase behaviour, which it does not. Thus internal positive feedback loop cannot mimic the anticipatory behaviour of predictive tracking (i.e. with a known stimulus). Subsequently, Ponder (16) has shown that the predictive mechanism utilizes both the saccadic and smooth pursuit systems.

5.1.2 Predictive and Non-predictive Tracking

These modes of eye movement with respect to the type of stimulus i.e. known - which can be predicted and unknown, which can not be predicted, have been extensively studied by Stark et al (29). They have shown the eye control system to be input - adaptive type, exhibiting anticipation and improved tracking when the input is a predictable waveform. Frequency response curves show important differences in gain and phase for predictable inputs of single sinusoids and non-predictable inputs composed of many non-coherent sinusoids. They reported a markedly increased phase lag for the unpredictable signal as compared to the steady state sinusoidal signal which indicates that the prediction apparatus of the brain is able to compensate

for almost all of the inherent phase lag of the neurological system. Their assertion is that while studying the eye tracking movements, responses to both, predictable and unpredictable inputs must be considered.

In a further study of the predictive behaviour of the oculomotor system Dallos & Jones (14) have shown that the anticipatory behaviour, observed during the tracking of predictive inputs, can be attributed to a prediction operator. This operator is determined by comparing the open-loop transfer function for two types of stimuli, namely Gaussian random noise and sine- and square wave inputs. The transfer function for the predictor, that is what transfer function in cascade with the open-loop transfer function that describes non-predictive tracking will produce the function that describes predictive tracking, was calculated. It contained an element whose gain is independent of frequency and whose phase increases roughly linearly with frequency. This can be crudely approximated by a delay line with a negative delay of about 280 ms. Since the human reaction time (latency) is between 200 to 250 ms the primary role of the predictor seems to be that of slightly over compensating for this reaction time.

5.1.3 Sampled data type of models

Young & Stark's (38, 39) model postulates that eye position is controlled by a sampled-data system with a sampling period

of about 0.2 secs. The oculomotor system seemed to sample the error created by the initial target step and then became refractory to any new incoming information for the next 200 ms at which time it executed a saccade appropriate to the error as it existed 200 ms. before.

Earlier Rashbass (20) had shown that it is necessary to separate saccadic and pursuit systems from functional point of view and this was done by Young & Stark. In their sampled-data model the saccadic system acts as a position servo to direct the eye at the target, and the pursuit system acts as a velocity servo to restore the eye at the same angular rate as the target. Theirs is a nonlinear model and accurately reproduced eye movements in response to different stimuli.

Young-Stark model was, in a way, conducive to further research concerning variation in eye tracking responses and for subsequent modified models. Wholmes et al (36) have shown that when the step input time is reduced from 200 ms downwards to 50 ms, in steps, the saccade response gradually fell from Young's postulate and occurred less and less. It can, therefore be said that when the system observes a position error it waits before shutting the gate and if the position error changes, the system may (with decreasing probability as time goes on) refrain from acting upon the earliest error information but may choose to act upon later information. Thus an impulse-sampling model will not do and some sort of probabilistic finite - sampling - time model is closer to the actual case.

Boeler (2) and Docker & Fucho (1) also studied saccadic variations for pulse and double step stimuli.

Murthy and Dookshatula (19) proposed a more sophisticated and accurate model than Young's (39). Young neglected muscle dynamics and various non-linearities such as dead zone and saturation. Moreover sampled-data system is not pertinent to smooth pursuit because the sampling period must be less than 60 ms in that case. Murthy and Dookshatula take into account both, the mechanics of the muscle and force developed and the sampled data nature of saccadic movement. The pursuit system was incorporated as a continuous system in the model which was subsequently confirmed by Eredkoy and Stark (4).

5.1.4 Experimental Models

Most significant experimental work has been done by Robinson (22, 23, 24). His models based on his own experimental observations were also linear and did not consider the agonist and antagonist muscles separately. He (22) used isometric, isometric and loaded globe experiments to generate a linear representation of eye dynamics during a saccade. In later (23, 24) but similar experiments during smooth pursuit and accommodative convergence movements, he included another visco-elastic element. The overall transfer function of the original model was fourth order; the revised one was fifth order. He correctly concluded that the eye was heavily

overdamped and little affected by the mass of the globe. It had a dynamic saccadic band width which decreased with increasing saccadic amplitude, and had a tracking band width of 1.1 Hz. The force generated in the active tension source was a brief excess force followed by a decrease in force to a tonic level. Although these representations depicted a wide variety of eye rotations under normal and abnormal conditions, they did not represent velocity characteristics correctly.

Thomas (32, 33) devised an ingenious method for measuring accelerations of the eye when it was driven by a small sinusoidal force. He attached an accelerometer to a contact lens on the human eye and passed current through an electromagnet to cause the eye to oscillate. The results showed that the system was under-damped above 10 Hz and could be characterized by a third order linear system. Thomas' model was linear and considered only small saccades (5°) where overshoot is common, larger saccades do not have this characteristic and appear critically damped. He did not consider the agonist and antagonist muscles separately. It is doubtful if his model can be used for larger saccades or for smooth pursuit and vergence movements.

St. Cyr & Pender (30, 31) recorded eye movements of subjects and studied the gain and phase of eye tracking with Fourier analysis techniques in response to noise or to a sum of

sinusoidal functions; they observed variations in responses to different sets of sinusoids and also proposed an alternate explanation for anticipation in terms of time delays.

5.1.5 Vestibular System

Eye-ball servomechanism as studied by Milsum & Jones (18) with special reference to vestibular stabilization has already been discussed in 2.2.2.

Robinson (25) has shown that all version eye movements, saccades, quick phase pursuit and vestibular start as velocity commands and enter a common processing circuit which is nothing but a neural integrator and this converts the signals into eye position commands.

5.1.6 Mechanical Models of Eye Movement Control Mechanism

All four systems of eye movement, saccadic, smooth pursuit, vergence and vestibular system, utilize the same common path made up of the oculomotor nuclei, their cranial nerves, extra ocular muscles and orbital-globe suspensory tissues. These elements each form a part of the over all transfer function between nervous activity in the motor neuron pools of the six muscles of each eye and the subsequent movement of the eyeball. The three dimensional aspect of the action of all six extra-ocular muscles has been considered. Attention is limited to dynamics in which case it is sufficient

to consider the action on the globe of a single antagonistic pair of muscles such as the horizontal recti in one degree of freedom.

Childress and Jones (5) conjectured a model based on anatomical and physiological considerations as well as the characteristics of saccadic eye movements. They considered the agonist and antagonist muscles separately and included the asymmetrical force-velocity relationship of muscles, but they did not show any simulated results.

Cook and Stark (11, 12, 13) presented a non-linear model similar to that of Childress & Jones (5). It depicted the position of velocity curves accurately over a wide range of saccadic amplitudes as well as matching Robinson's (22) isometric and isometric results. The model consists of an agonist and antagonist muscle pulling on the globe in its orbit. The globe orbit is represented by a parallel spring-mass-dashpot-combination, while each muscle consists of a passive elasticity in parallel with another elasticity connected to a tension source and a non-linear viscous element.

Later models proposed by Robinson (26) following Cook and Stark were of higher order and considerably more complicated. They incorporated the agonist and antagonist separately while including the non-linear force-velocity relationship of active muscles. These representations were

not simulated to yield quantitative results; they only presented schematic approximations to the plant. Robinson (26) showed that his results were compatible with Thomas' (32) by noting that the eye seems over damped under 16 Hz and under damped above this frequency.

Subsequently Robinson et al (27) conducted length-tension and quick release experiment on the isolated globe and showed that the steady state tonic forces as well as maximum available forces assumed by Cook & Stark (11) were higher. This yielded a passive elastic coefficient which was too great.

5.2 Integration of the Eyeball and its Muscles

Now that we have separate models of the eyeball (Ch. IV) and its extraocular muscles (Ch. III), we may combine these elements and consider their interaction while constructing a model for the overall plant of extraocular system for vertical eye movements. Fig. 22 represents a composite model. It shows separately the properties of all the elements. One muscle shortens and is the agonist, while the other lengthens and is the antagonist. Of course, in this development we have reduced the two muscles concerned in vertical eye movements to a single equivalent muscle (Sec. 3.3.3). The muscles which are involved here are summarized below.

	Agonists	Antagonists
Elevation	1. Superior Rectus 2. Inferior oblique	1. Inferior rectus 2. Superior oblique
Depression	1. Inferior rectus 2. Superior oblique	1. Superior rectus 2. Inferior oblique

Each one of the above combinations of two muscles has been reduced to one muscle and this has been represented in Fig. 22.

The first important thing is whether we can reduce this model without affecting its functional characteristics. The active tension generators F_{AG} and F_{AT} of the agonist and the antagonist muscles respectively have different characteristics and, therefore, may not be combined. The series springs K_{AG} and K_{AT} must assume the tension of F_{AG} and F_{AT} in steady state. These cannot therefore be simplified. The force-velocity relationship for shortening agonistic muscle and lengthening antagonistic muscle is clearly different; thus D_{AG} and D_{AT} may not be combined. The remaining two elements are the passive elasticities $K_{P(AG)}$, $K_{P(AT)}$ of the agonist and the antagonist. They represent the elastic resistance to steady deviation of the globe from the rest position. Although the passive coefficient for each muscle is non-linear, becoming approximately zero for shortening muscle and increasing to become fairly linear at about 20° , the net effect of the combined passive elasticities is

roughly constant and equal to that of a single spring (7). It will be further assumed that the passive coefficient of both muscles and the globe can be added to give a linear spring constant.

$$\begin{aligned}
 K_P &= K_{P(AG)} \text{ (or } K_{P(AT)}) + K_G \\
 &= .24 \times .36 + .5 = .586 \text{ g/deg.}
 \end{aligned}$$

With this simplification the model is reduced to the one shown in Fig. 23. It has the following parameters.

$$K_P = .586$$

$$K_A = K_{AT} = K_{AG} = .24 \times 1.8 = .432 \text{ g/deg.}$$

$$B_{AT} = 1.25 F_{AT} / (1000 - \dot{\theta}_3) \quad \dot{\theta}_3 \leq 0$$

$$= 3 F_{AT} / 1000 \quad \dot{\theta}_3 > 0$$

$$B_{AG} = 1.25 F_{AG} / (1000 + \dot{\theta}_2) \quad \dot{\theta}_2 \geq 0$$

$$= 3 F_{AG} / 1000 \quad \dot{\theta}_2 < 0$$

$$J_g = 4.3 \times 10^{-5} \text{ g - sec}^2/\text{deg.}$$

$$\tau_{act} = 4 \text{ ms}$$

$$\tau_{deact} = 8 \text{ ms}$$

The dynamic characteristics of this model shall be studied in the next chapter.

6. DYNAMIC CHARACTERISTICS OF THE MODEL

6.1 Activation - De-activation Process

Activation and de-activation of the muscle in the model of Fig. 23 is a transient process of which the inputs are the nerve signals, which we have called E_{AG} and E_{AT} and of which the outputs are levels of the tension F_{AG} and F_{AT} respectively.

Wilkie (37) describes an experiment designed to illustrate this process. The muscle used was a frog's sartorius at a temperature of 0° C. Wilkie's findings were that the time constant for deactivation was approximately 100 ms and the time constant for activation many times shorter. It is difficult to know how to adapt these results to our situation. No doubt the extraocular muscles operating at body temperature are much faster than this. For lack of a better estimation, Cook & Stark (11) assumed the time constant for deactivation of the extraocular muscles as 10 ms and that for activation as less than one ms.

referred to
primary

Later Clark & Stark (6) improved these time constants to 8 ms and 4 ms respectively. These values shall be assumed here too.

6.2 Dynamic behaviour of the model

Having drawn up a complete and simplified model (Fig. 23) for the eye movement mechanism for vertical eye movements, we

are now in a position to study the dynamic characteristics of the model.

With reference to Fig. 23, we may write the system equations as follows :

$$F_{AG} = K_A (\theta_2 - \theta_1) + B_{AG} \dot{\theta}_2 \quad \dots \quad (6.1)$$

$$K_A (\theta_1 - \theta_3) = F_{AT} + B_{AT} \dot{\theta}_3 \quad \dots \quad (6.2)$$

$$K_A (\theta_2 - \theta_1) = K_P \theta_1 + B_G \dot{\theta}_1 + J_G \ddot{\theta}_1 + K_A (\theta_1 - \theta_3) \quad \dots \quad (6.3)$$

Rearranging Eqn. 6.3 and taking Laplace Transforms

$$\theta_1(s) = \frac{K_A}{J_G} \pi \frac{\theta_2(s) + \theta_3(s)}{s^2 + (B_G/J_G)s + (2K_A + K_P)/J_G} \quad \dots \quad (6.4)$$

Assuming that $\theta_2(s) + \theta_3(s) = T(s)$ has no dynamics and can change to yield a step forcing function, which as will be shown subsequently, is nearest to nervous activity corresponding to oscilladic movements; and substituting the values for various constants we have :

$$\begin{aligned} \theta_1(s) &= \frac{.432}{4.5 \pi 10^{-5}} \pi \frac{T(s)}{s^2 + s \left(\frac{.015}{4.5 \pi 10^{-5}} \right) + \left(\frac{2\pi \cdot 432 + .586}{4.5 \pi 10^{-5}} \right)} \\ &= \frac{10^4 T(s)}{s^2 + 350 s + 33700} \quad \dots \quad (6.5) \end{aligned}$$

The roots of the Eqn. $s^2 + 350 s + 33700 = 0$
are $s = -175 \pm j 55$

Thus this second order system is having poles of $-175 \pm j 55$, natural frequency of oscillations $\omega_n = 183.5$ and damping coefficient $\xi = 0.95$. It is, therefore, a stable system which is highly overdamped. It is well known from data by Robinson (22) that the eye is overdamped; increasing the mass of the globe a hundred fold only causes slight oscillations. The highly overdamped nature must be caused by fast movements either in the antagonist as θ_3 changes or in the agonist as θ_2 changes.

To decrease the damping coefficient ξ , we take $s = -175 \pm j 125$. This gives $\omega_n = 215.3$ and $\xi = 0.81$. Still we find that damping coefficient is high, so we take $s = -175 \pm j 150$. Now we get $\omega_n = 230$ and $\xi = 0.76$. This value of ξ is nearer the one found by other researchers. Looking back to equation 6.4 we see that to get a proper value of the damping coefficient the values of K_P and K_A must be modified. We, therefore, have

$$\frac{2K_A + K_P}{4.3 \times 10^{-5}} = 53125$$

$$2K_A + K_P = 2.284$$

$$2K_A = 2.284 - K_P \quad 1.7$$

$$\therefore K_A = 0.85$$

We have not altered K_p deliberately as it is an equivalent spring constant and largely depends upon the spring constants of the globe. The effect of these values of $\dot{\epsilon}_q$ on dynamic characteristics will be shown subsequently.

A typical 10 degree saccade takes 40 ms and the average velocity is about 250 deg/s (7). Again referring to Fig. 23 and from section 5.2 we have

$$B_{\Delta T} = 1.25 P_{\Delta T}/1000 - \dot{\epsilon}_3 \quad \dot{\epsilon}_3 \leq 0 \quad \dots (6.6)$$

$$= 3 P_{\Delta T}/1000 \quad \dot{\epsilon}_3 \quad \dot{\epsilon}_3 > 0 \quad \dots (6.7)$$

$$B_{\Delta G} = 1.25 P_{\Delta G}/1000 + \dot{\epsilon}_2 \quad \dot{\epsilon}_2 \geq 0 \quad \dots (6.8)$$

$$= 3 P_{\Delta G}/1000 \quad \dot{\epsilon}_2 < 0 \quad \dots (6.9)$$

It may be clarified that elevation of the eye is assumed as motion in the positive direction and depression as motion in the negative direction. For elevation of the eye superior rectus and the inferior oblique are the agonists and inferior rectus and superior oblique are antagonists and vice versa for depression. The agonists are shortening muscles and the antagonists are lengthening muscles.

The tension in the agonist is about 100 g and in the antagonist about 2 g (7). Using Eqn. 6.8 and 6.7 we get

$$\begin{aligned}
 B_{AG} &= \frac{1.25 \pi 100}{1000 + 250} = 0.1 \text{ g} - \text{o/dog.} \\
 B_{AT} &= \frac{3 \pi 2}{1000} = 0.006 \text{ g} - \text{o/dog.}
 \end{aligned}
 \left. \begin{array}{l} \\ \\ \end{array} \right\} \dots (6.9)$$

The time constants for the muscles can be found by dividing those values of B_{AG} and B_{AT} by K_A to give an agonist time constant τ_{AG} of 118 ms and an antagonist time constant τ_{AT} of 7 ms.

Clark and Stark (7) have shown that the equivalent time constant for the agonist muscle must include the deactivation of the antagonist and is given by the following relation

$$\begin{aligned}
 \tau_{eq}(AG) &= \sqrt{\tau_{deact}^2 + \tau_{AG}^2} \\
 &= \sqrt{8^2 + 118^2} \\
 &\approx 118 \text{ ms} \quad \dots (6.10)
 \end{aligned}$$

$$\begin{aligned}
 \text{Similarly } \tau_{eq}(AT) &= \sqrt{\tau_{deact}^2 + \tau_{AT}^2} \\
 &= \sqrt{8^2 + 7^2} \\
 &= 10.5 \text{ ms.} \quad \dots (6.11)
 \end{aligned}$$

It can be thus seen that the agonist is the part of extracellular system which slows down the response (even in spite of excess force programme) and causes the plant to be overdamped.

6.3 Nervous activity for eye control

Erwinin (3) shows a graph of integrated muscle potential versus eye position for a human extraocular muscle (lateral rectus). This graph has been redrawn in Fig. 24. Negative angles mean that the eye was turned nasally that is away from lateral rectus and positive angles mean that the eye was turned temporarily i.e. towards the lateral rectus.

Muscle potential is measured by means of electrodes inserted in the muscle. If the probe is bipolar, the signal which is measured is quite localized and may or may not represent the overall muscle behaviour. A monopolar probe inserted into the muscle with a reference attached to some distant portion of the body gives a less localized measurement than the bipolar probe; but nevertheless, it is somewhat localized.

Another disadvantage of these measurements is the difficulty in reading the records. The raw electromyograph looks much like a saw tooth with varying amplitude and frequency. It leaves a good deal to the imagination of the reader. However, if the emg is rectified, integrated and chopped at regular intervals, we get a more readable graph. The peaks of the integrated and chopped emg are proportional to the frequency and envelope amplitude of the pulses composing the emg.

It has been shown elsewhere (11) that a linear relationship exists between integrated (and chopped) muscle potential

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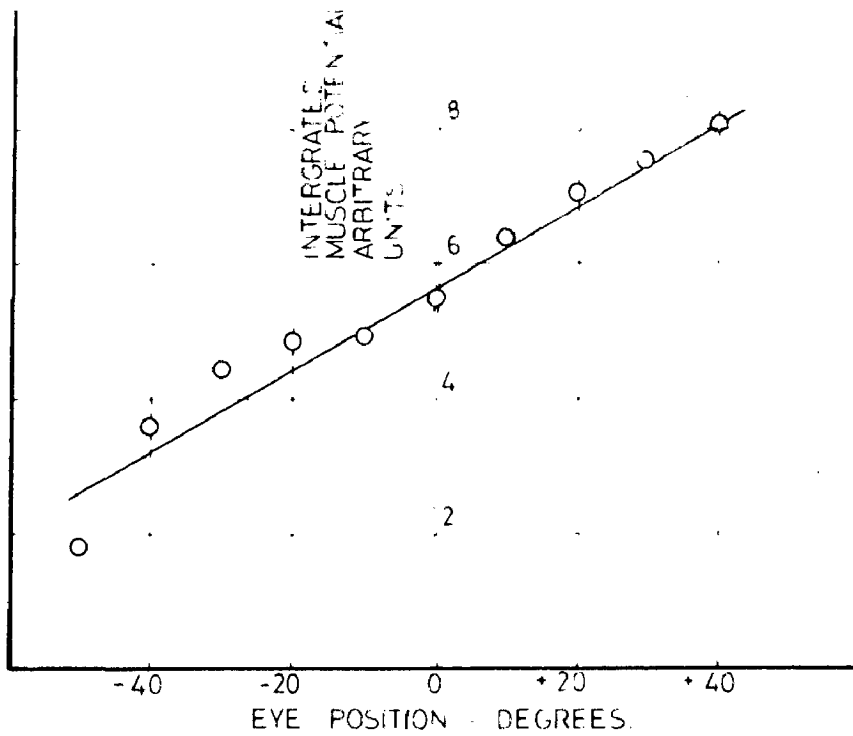


FIG 24 _ INTEGRATED MUSCLE POTENTIAL AS A FUNCTION OF STEADY STATE EYE POSITION (Breinin, 1958)

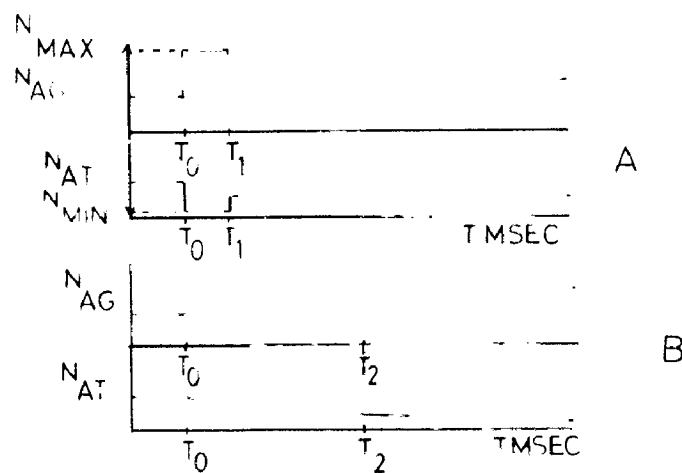


FIG 25 _ NERVOUS ACTIVITY IN THE EXTRAOCULAR MOTOR NUCLEI DURING DIFFERENT HORIZONTAL EYE MOVEMENTS. N_{AG} -NERVOUS ACTIVITY IN THE AGONIST MUSCLE; N_{AT} -NERVOUS ACTIVITY IN THE ANTAGONIST MUSCLE; T- TIME IN MILLISECONDS (a) NERVOUS ACTIVITY DURING A SACCADE (b) NERVOUS ACTIVITY DURING A SMOOTH PURSUIT MOVEMENT IN RESPONSE TO A RAMP STIMULUS

and steady-state tension with fixed muscle length and zero velocity. But our muscle model says that at fixed length and zero velocity the tension is equal to the tension sources. In steady state conditions N_{AG} and N_{AT} are respectively equal to F_{AG} and F_{AT} . Hence N_{AG} and N_{AT} are proportional to integrated muscle potential.

A direct proportion between tension and integrated muscle potential was found by Biglad and Lippold (Bibliography), during constant velocity of shortening or lengthening. These measurements were made with the tension at steady-state. Under this condition, the elastic component was at fixed length or else the tension would have been changing. Thus we know that the velocity of the contractile component was equal to the velocity of muscle shortening or lengthening and therefore constant. The equation for tension in a shortening (agonist) muscle is :

$$F_{AG} = F_0 \left(1 - \frac{1.25}{b + \dot{\theta}_2} \dot{\theta}_2 \right) \quad \dots \quad (6.12)$$

Where, F_0 is maximum isometric force at L_0 . This equation is the same as equation 3.2. Similarly for a lengthening (antagonist) muscle

$$F_{AT} = F_0 \left(1 + \frac{3}{b} \dot{\theta}_3 \right) \quad \dots \quad (6.13)$$

Since the terms within the brackets are constants (velocity

is constant here) F is proportional to F_0 . With F also proportional to integrated muscle potential F_0 is proportional to integrated muscle potential. However, in steady state F_{AG} and F_{AT} respectively equal N_{AG} and N_{AT} which means that N_{AG} and N_{AT} are proportional to integrated muscle potential. This provides us with a means of measuring directly the control signals. In interpreting the unintegrated electromyogram. N_{AG} and N_{AT} are proportional to the density of the pulses on the emg record and to the peak to peak amplitudes of those pulse envelopes.

Referring again to Fig. 24, we can say how the tension of the tension sources vary with steady state eye position. According to our model (Fig. 23), for eye to be held θ^0 from centre, a tension differential of $K_p \cdot \theta$ g is required from the tension sources. We use the linearized approximation of Fig. 24 which tells us that the increase in tension in the muscle which has shortened is equal to the decrease in tension in the muscle which has been lengthened. This also gives the linearization a slope of $K_p/2$ g/deg.

If we assume $\pm 40^\circ$ as limiting eye position for vertical movement and that the lengthened muscle is completely inhibited at this angle, then our curve for integrated muscle potential should go to zero at -40° . The tension at zero degrees is then

$$F_o(0^\circ) = 40 \frac{K_P}{2} \theta \quad \dots \quad (6.14)$$

With K_P equal to 0.58 g /deg.

$$F_o(0^\circ) = \frac{40 \times 0.586}{2} \theta \quad 12 \text{ g} \quad (6.15)$$

In general the steady state tension of the sources for a resting position is given by the following:

For shortening muscle

$$F_{AG} = 12 + K_P \frac{\theta}{2} \quad \dots \quad (6.16)$$

For lengthening muscle

$$F_{AT} = 12 - K_P \frac{\theta}{2} \quad \dots \quad (6.17)$$

Where θ is the extent of eye movement from primary position. Equations 6.16 and 6.17 show that resting tension of an extraocular muscle is in contrast to that of a skeletal muscle, such as leg or arm muscle which have zero or nearly zero resting tension.

Recent research has described the nervous activity of the extraocular motor nuclei during a wide variety of different eye movements. These results help one to model the nervous activity responsible for rotating the eye. A number of researchers have studied EMG activity during the saccade. Robinson, Fuchs and Luschei, and ~~Hom~~^{Wm} and Cohen studied single unit activity in the extra ocular ~~net~~

motoneurons during eye movements (10). All showed a brief optimal burst in agonist firing rate accompanied by an inhibition of the antagonistic firing rate. The duration of this pulse determined the amplitude of the saccade. The agonist then reduced its activity while the antagonist increased its firing rate to levels representative of the new tonic position. Fig. 25(a) shows the results graphically.

Robinson (23) observed the isometric force of the human eye in response to a ramp stimulus. He found an initial but brief excess force about that maintained subsequently to move the eye at a constant velocity. He postulated a similar initial excess burst rate to account for the excess force development. Robinson (Bibliography) also observed single unit firing in response to a smoothly moving target and found a smoothly changing firing rate. Fig. 25(b) shows the integrated nervous activity assumed during the response to a ramp.

6.4 Position Response of the Model

6.4.1 Saccadic Movement

Consider equation 6.5

$$\theta_1(s) = \frac{10^4 T(s)}{s^2 + 350s + 33700}$$

$$\theta_1(s) = \frac{10^4 T(s)}{(s + a)(s + b)} \quad \dots (6.18)$$

*you have changed the
value of the parameters
+ the value of the gain
... for analysis*

where, $a = 175 - j 55$

and $b = 175 + j55$

As has been shown in Fig. 25(a) and explained in Sec. 6.3 the nervous signals N_{AG} and N_{AT} corresponding to saccadic eye movement may be approximated by the first-order time optimal control signals. At the start of a saccade N_{AG} achieves a high level of activity and N_{AT} drops to a level of almost zero activity. Combining the two activities, we get a step like input to the eye movement system, so replacing $T(S)$ by a step function of amplitude A

$$E_1(S) = \frac{10^4 \cdot A}{S(S+a)(S+b)}$$

Taking inverse Laplace Transform

$$e_1(t) = \frac{A 10^4}{ab} \left(1 + \frac{1}{a-b} (be^{-at} - ae^{-bt}) \right)$$

substituting for a and b and simplifying we get

$$e_1(t) = \frac{A \pi 10^4}{33650} (1 - e^{-175 t} (3.2 \sin 55 t + \cos 55 t)) \dots \quad (6.19)$$

Now the only unknown in this equation is A the amplitude of the step input force. For a saccade force in the agonist is 100 g and that in the antagonist is 2 g. Thus the net force is 98 g. We can therefore take A approximately 100.

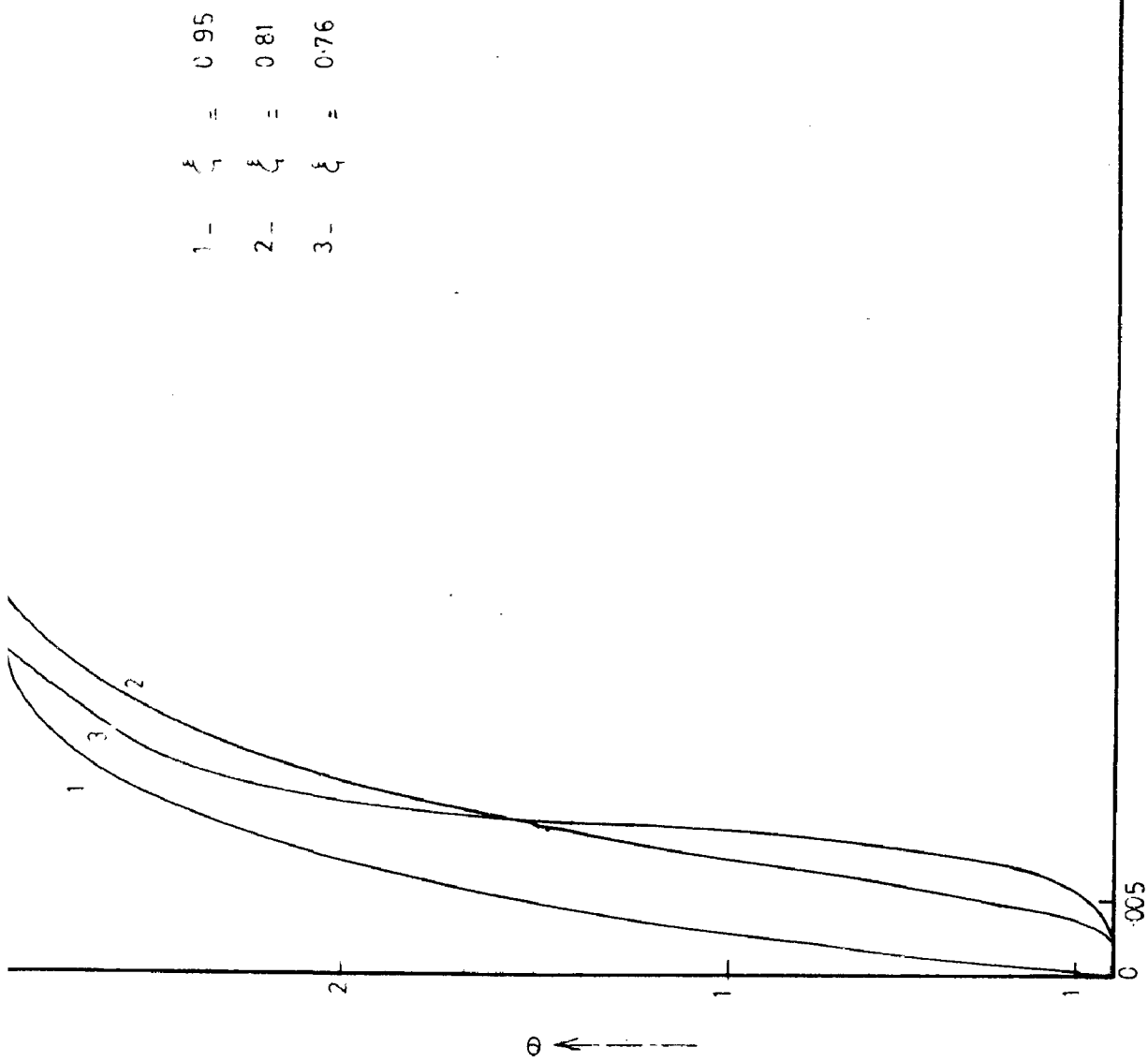


FIG.26 - EYE POSITION (θ) VS TIME FOR SACCADIC EYE MOVEMENT.

A computer programme (See Appendix) was prepared for the above equation to plot eye position e_1 versus time. The time was varied from 0 to .1 s in steps of .001 s. The programme was run on TDC-12 digital computer. The result is plotted in Fig. 26. Also plotted on the same graph are the curves corresponding to $\xi_c = .81$ and $\xi_c = .76$. The output data of computer shows (though it is not prominent in the curves) that lowering damping coefficient introduces oscillations. We therefore conclude that it is not desirable to reduce the damping coefficient and the vertical eye movement system is highly overdamped.

6.4.2 Smooth Pursuit Movement

Again take equation 6.18

$$e_1(s) = \frac{10^4 K T(s)}{(s+a)(s+b)}$$

The nervous activity corresponding to smooth pursuit movement of the eye is like a ramp as shown in Fig. 25(b). so replacing $T(s)$ by a ramp function

$$e_1(s) = \frac{10^4}{s^2(s+a)(s+b)}$$

$$= 10^4 \left(\frac{-1}{ab} \left(\frac{1}{a} + \frac{1}{b} \right) \frac{1}{s} + \frac{1}{ab^2} + \frac{1}{a^2(b-a)(s+a)} + \frac{1}{b^2(a-b)(s+b)} \right)$$

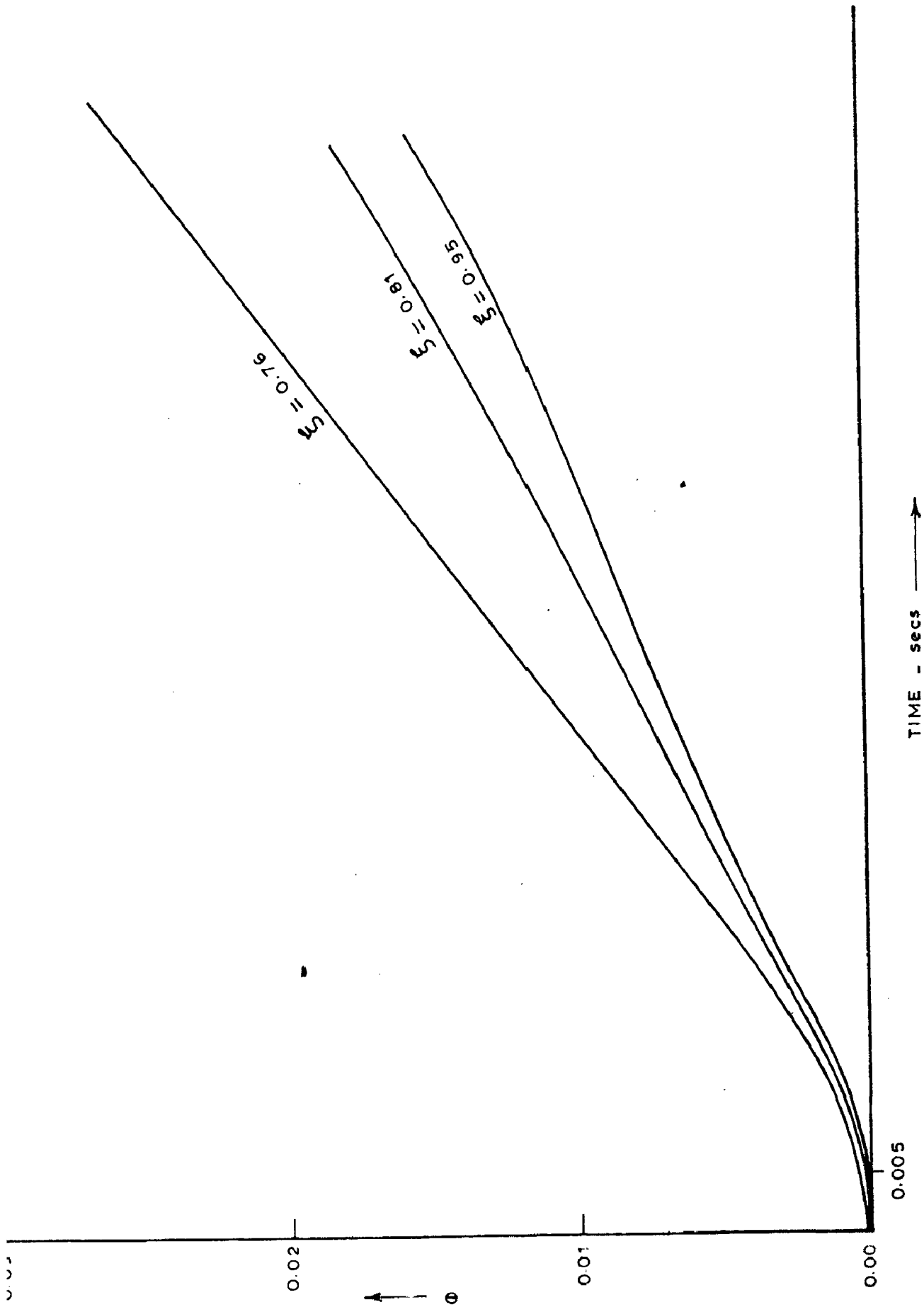


FIG. 26 (a) - EYE POSITION (θ) VS TIME FOR SMOOTH PURSUIT EYE MOVEMENT

Taking inverse Laplace transform.

$$\theta_1(t) = 10^4 \left(\frac{-1}{ab} \left(\frac{1}{a} + \frac{1}{b} \right) + \frac{1}{ab} t + \frac{1}{a^2(b-a)} e^{-at} + \frac{e^{-bt}}{b^2(a-b)} \right)$$

Substituting for a & b and simplifying

$$\theta_1(t) = \frac{t}{3.365} - .0031 + .01 e^{-175t} (.308 \cos 55 t + .445 \sin 55 t) \dots \quad (6.20)$$

This equation too was programmed and run on TDC-12 to plot eye position versus time. Fig. 26(a) shows smooth pursuit curves for various values of .

6.5 Velocity Curve

We have 6.18 as

$$\theta_1(s) = \frac{10^4 T(s)}{(s+a)(s+b)}$$

$$\dot{\theta}_1(s) = \frac{10^4 s T(s)}{(s+a)(s+b)}$$

To plot velocity curve for saccadic movement we replace $T(s)$ by a step function

$$\therefore \dot{\theta}_1(s) = \frac{10^4 A}{(s+a)(s+b)}$$

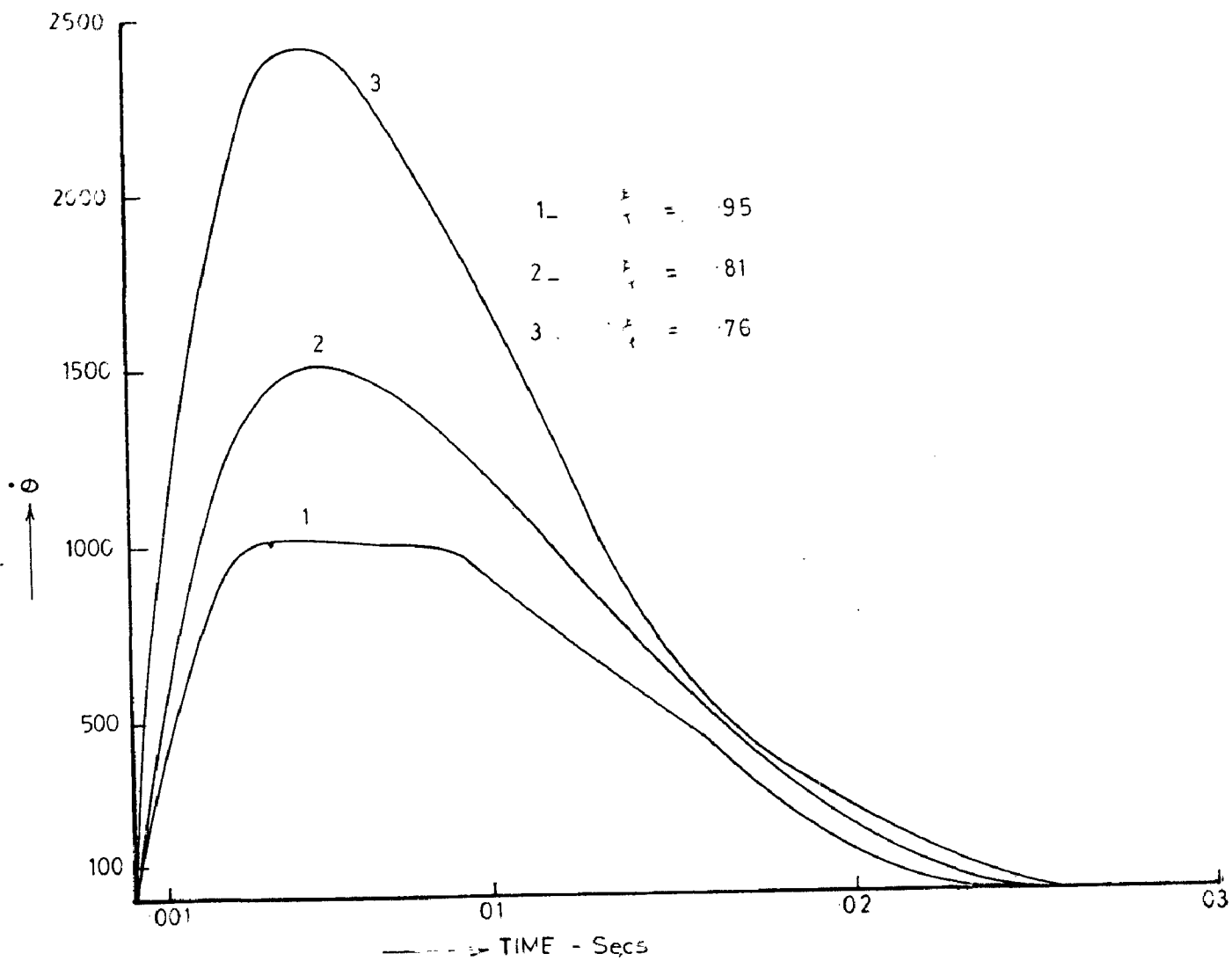


FIG. 27 - SACCADIC VELOCITY ($\dot{\theta}$) VS TIME

Taking inverse Laplace Transform

$$\begin{aligned}\dot{e}_1(t) &= \frac{10^4 \Lambda}{b-a} (e^{-at} - e^{-bt}) \\ &= \frac{10^4 \Lambda e^{-175 t}}{55} \times \sin 55 t \quad \dots \quad \dots \quad (6.21)\end{aligned}$$

This curve i.e. velocity versus time has been plotted in FIG. 27 for all values of ξ_q . We find that lower values of ξ_q give a very high value of velocity, whereas values of velocity for $\xi_q = 0.95$ are comparable to those found by Clark and Stark (8, 10). It is again confirmed that eye movement mechanism for vertical movement is highly overdamped. It may be noted that the model depicts an asymmetry in acceleration and deceleration phases, with a quick acceleration followed by a longer deceleration. This is in conformity with experimental results of Hyde quoted by Clark & Stark (8).

6.6 Acceleration Curve

Equation 6.18 is

$$\begin{aligned}e_1(s) &= \frac{10^4 T(s)}{(s+a)(s+b)} \\ \therefore \ddot{e}_1(s) &= \frac{10^4 s^2 T(s)}{(s+a)(s+b)}\end{aligned}$$

Replacing $T(s)$ by a step function for saccadic eye movements

$$\ddot{e}_1(s) = \frac{10^4 s \Lambda}{(s+a)(s+b)}$$

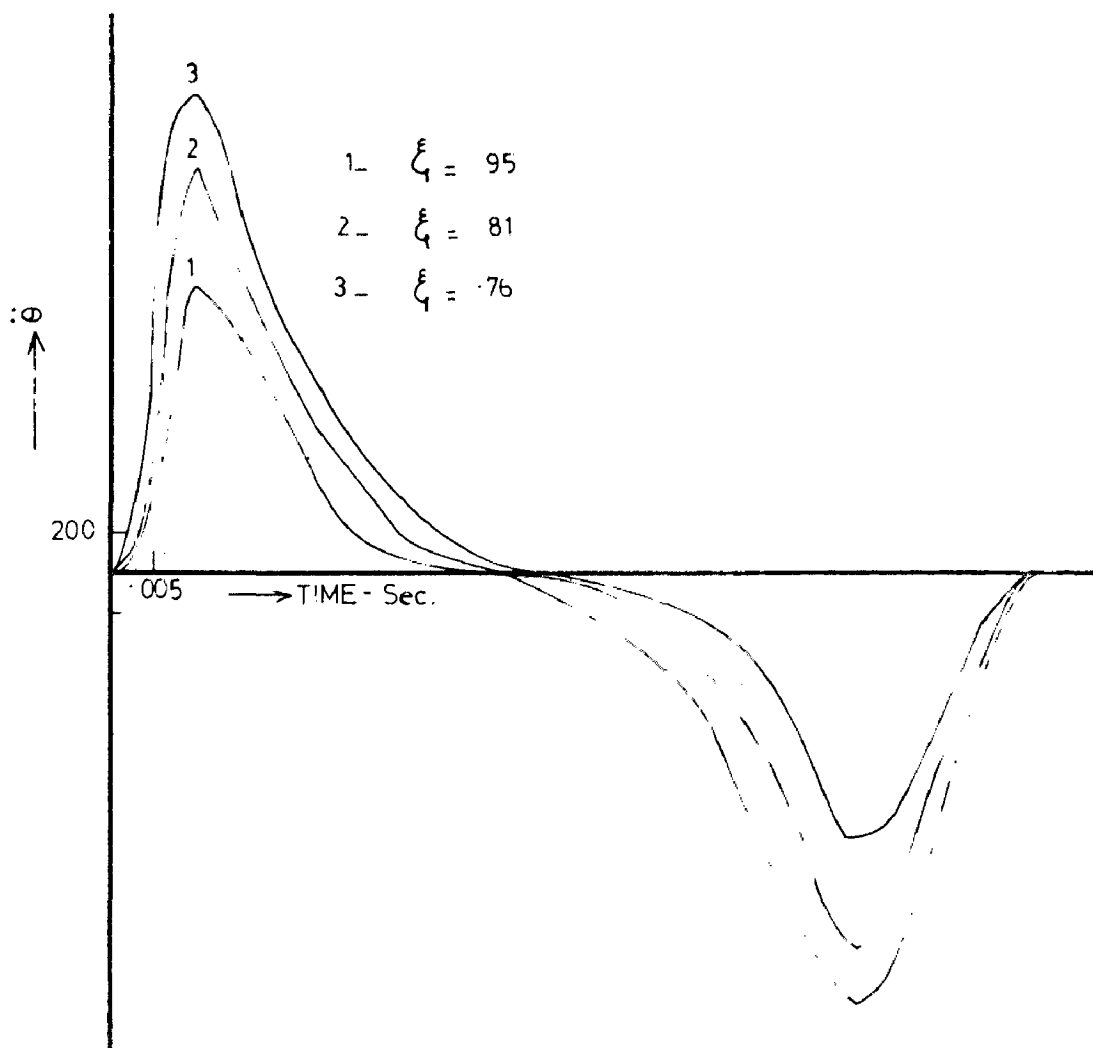


FIG 28_ SACCADIC ACCELERATION VS TIME .

Taking inverse Laplace transforms

$$\begin{aligned}\ddot{\theta}_1 (s) &= \frac{10^4 \Lambda}{b - a} (b e^{-bt} - a e^{-at}) \\ &= 10^4 \Lambda e^{-175t} (\cos 55 t - 3.2 \sin 55 t) \dots (5.22)\end{aligned}$$

Saccadic acceleration versus time is plotted in Fig. 28 for all values of damping coefficient.

6.7 Phase Plane Relationship

From Figs. 26 and 27 we can derive a relation between saccadic velocity and position. This is called phase plane curve and is shown in Fig. 29. It is evident from these plots that the peak velocity of saccades increases as the amplitude of the saccades increases, but beyond a certain point of saccadic amplitude, the peak velocity starts decreasing. This shows that the ocular plant is fast for saccades of certain amplitudes only and not for very large saccades. Larger saccades take longer time.

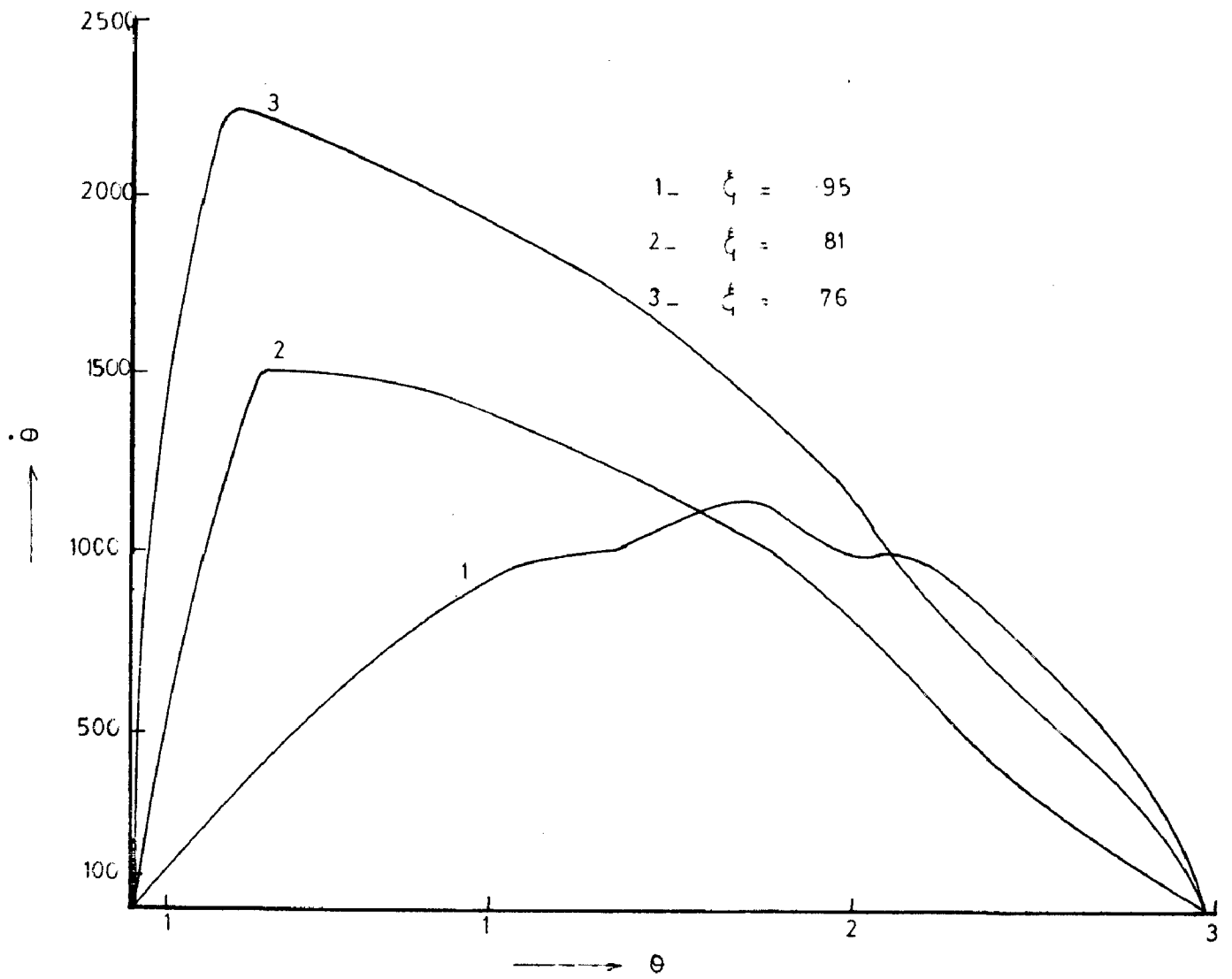


FIG. 29 - PHASE PLANE PLOT - VELOCITY VS POSITION.

7. SACCADES ARE TIME OPTIMAL

Optimal control theory takes into account constraints such as energy and time economics which are relevant to the understanding of biological design. The versional eye tracking system responsible for the extremely rapid and precise movements - saccades, seems a likely biological system in which to test for time optimality.

7.1 Saccades

Saccadic eye movements are produced by an exceedingly elegant control system which drives conjugate eyeball rotations with velocities of up to 1000 deg/s in versions lasting less than a twentieth of a second. Various researchers, such as, Physiologists, Psychiatrists and Clinicians have all been intrigued by these movements responsible for positioning the image of an object on the high resolution foveal region of the retina. The saccade is characterized by a rapid acceleration to a relatively constant high velocity followed by a less rapid deceleration to the new eye position. These movements are very quick; a 10 deg. saccade may take as little as .04 s (9). This time excludes the well known random delay time of 150 - 200 ms. This latency may be neglected because it does not affect the shape of the dynamic trajectory, (15, 35, 38). A nonlinear relationship exists between saccadic amplitude and velocity with a saturation of 1000 deg/s at about 50 deg (9); thus larger eye

movements take a longer time. Saccades which reduce slip between the image and the retina, are outputs of a dual-mode control system in the brain which is responsible for the executing^o_λ of complex versional eye movements.

Recently, the techniques of system analysis have been employed to develop models which approximate the dynamic trajectories, of saccadic eye movements (5, 11, 12, 22, 23, 32, 35). As discussed in 5.1 using physiological data alongwith powerful engineering tools such as frequency and time-domain analysis, several mechanical analogs to the extraocular muscles and the eyeball have been constructed. One branch of analysis, aside from modelling, synthesis, simulation or stability, is that of optimal control theory (Bibliography) which is primarily concerned with the minimization or maximization of such quantities as time, energy, or distance in order to achieve a certain objective. To this end, the techniques of optimal control theory have been applied to a model for saccades to see if these eye movements are time optimal. The only previous application of time optimal control theory in biology are by Smith (28) for hand movement and Cook & Stark (11) for eye movement. Cook and Stark used an erroneous assumption as to maximum muscle force and drew the false conclusion that the eye movements were not time optimal.

7.2 Nervous Signals

Experimental results from human and animal neurophysiology have shown that the nervous signals N_{AG} and N_{AT} may be approximated by the first-order time optimal control signals as shown in Fig. 30 (10) (See also Sec. 6.3). At the start of a saccade, N_{AG} achieves a high level of activity and N_{AT} drops to a level of almost zero activity. This is completely compatible with the time optimal control signal for a first-order system where one would want as large a driving force and as small a resisting force as possible to move the system quickly. After a certain time, N_{AG} decreases and N_{AT} increases its activity to a steady tonic level required to maintain the eye at its new position; Dashed lines in Fig. 30 show typical nervous signals observed experimentally. The slight delay between the end of N_{AG} and the end of N_{AT} is an interesting subtlety of saccadic control reflected in neurophysiological data (27). This nervous activity was first measured by Frazier et al (Bibliography), but Cook and Stark (11) postulated the following relationship between tonic activity and steady state eye position θ for horizontal eye movement as :

$$F_{AG} = 37.5 + \theta K_P/2$$

$$F_{AT} = 37.5 - \theta K_P/2$$

For vertical eye movement we have earlier developed similar relations in Sec. 6.3. These are given by equations 6.16 and

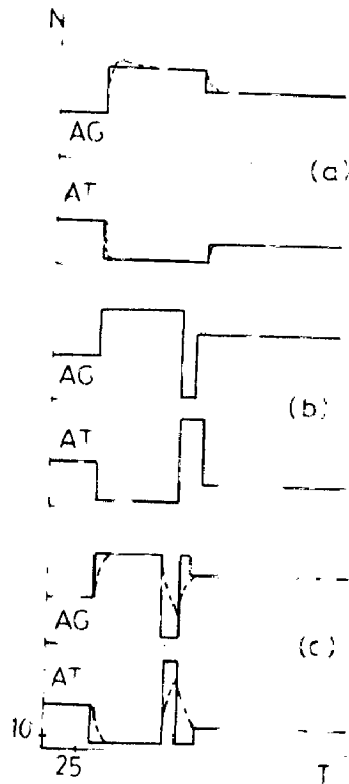


FIG. 30 - PAIRS OF FIRST, SECOND AND THIRD ORDER TIME OPTIMAL CONTROL SIGNALS SHOWING THE ABRUPT CHANGE FROM INITIAL AND FINAL LEVELS TO MAXIMA FOR N_{AG} AND N_{AT} , RESPECTIVELY. SCALES: ABSCISSA - TIME IN MILLISECONDS; ORDINATE - PULSES PER SECOND WITH "10" BEING EQUIVALENT FORCE IN GRAMS. DASHED LINES IN (a) SHOW TYPICAL SIGNALS OBSERVED EXPERIMENTALLY. NOTE ESPECIALLY THE INCREASED NUMBER OF SWITCHINGS IN THE HIGHER ORDER PATTERNS.

6.17 which are reproduced below :

$$P_{AG} = 12 + K_p \theta/2$$

$$P_{AT} = 12 - K_p \theta/2$$

The fact that the controller signals are very similar to the optimal control for a first-order system has led several researchers (22, 23, 32, 33, 35) to utilize a first order approximation for modelling the plant. This has proved quite satisfactory in models of the overall torsional eye control system where the controller is much slower than the plant; the plant is then simply represented. However, in a model of the plant alone where minute facets of its dynamic responses are of interest, one must recognize and include a more accurate realization which includes the asymmetrical nonlinearities of the extraocular muscles.

7.3 Mathematical Representation

In order to achieve a time optimal response, one must consider the properties of the control set N_{AG} and N_{AT} as well as the elements of the ocular motility model. The modified model (Fig. 23) is basically a nonlinear sixth-order system consisting of T_{AG} , T_{AT} , a second-order globe orbit model and two first-order muscle approximation.

The basic form of the optimal controller signals for the simulations depend on :

(i) The differential equations describing the saccadic system, the Hamiltonian functional formed from the differential equations and their adjoint form and

(ii) The performance criterion, which in our case is minimum time.

The mathematical equations governing the extraocular system of Fig. 23 are given below :

Let $\theta_1 = \theta$, the angle of the eye in degrees.

$\theta_2 =$ the angle of the agonistic nonlinear force-velocity relationship in degrees.

$\theta_3 =$ the angle of the antagonistic nonlinear force-velocity relationship in degrees

$\dot{\theta}_1 = \theta_4$, the eye velocity in deg/s

$P_{AG} = N_{AG} = \theta_5$

$P_{AT} = N_{AT} = \theta_6$

The governing equations are

$$\dot{\theta}_1 = \theta_4 \quad \dots \quad (7.1)$$

$$\begin{aligned} \dot{\theta}_2 = 1000 (K_A (\theta_1 - \theta_2) + \theta_5) / (0.25 \theta_5 \\ + K_A (\theta_2 - \theta_1)) \quad \dots \end{aligned} \quad (7.2)$$

$$\dot{\theta}_3 = 333 (K_A (\theta_1 - \theta_3) / \theta_6 - 1) \quad \dots \quad (7.3)$$

$$\dot{\theta}_4 = \frac{1}{J} (\kappa_\Delta (\theta_2 + \theta_3) - D_P \theta_4 - (2 \kappa_\Delta + \kappa_P) \theta_1) \dots \quad (7.4)$$

$$\dot{\theta}_5 = (1/\Upsilon_{\Delta Q}) (-\theta_5 + \Pi_{\Delta Q}) \dots \quad (7.5)$$

$$\dot{\theta}_6 = (1/\Upsilon_{\Delta T}) (-\theta_6 + \Pi_{\Delta T}) \dots \quad (7.6)$$

$$\theta_2 \geq \theta_1 \geq \theta_3, P_{\max} \geq P_{\Delta Q} = \theta_5 \geq P_{\Delta T} = \theta_6 \geq P_{\min} > 0 \dots \quad (7.7)$$

$$\left. \begin{aligned} \Upsilon_{\Delta Q} &= \Upsilon_{\text{act}} & \dot{\theta}_5 \geq 0 \\ &= \Upsilon_{\text{doact}} & \dot{\theta}_5 < 0 \end{aligned} \right\} \dots \quad (7.8)$$

$$\left. \begin{aligned} \Upsilon_{\Delta T} &= \Upsilon_{\text{act}} & \dot{\theta}_6 \geq 0 \\ &= \Upsilon_{\text{doact}} & \dot{\theta}_6 < 0 \end{aligned} \right\} \dots \quad (7.9)$$

The parameters for these equations are already given in Sec. 5.2

The activation-deactivation time constants are initially omitted in this development; thus $\Upsilon_{\Delta Q} = \Upsilon_{\Delta T} = 0$. The optimal policy with their inclusion will then be developed heuristically. The adjoint equations for this simplified fourth order are formed from the Jacobian Λ of the original equation 7.1 to 7.4 in the following manner :

$$\dot{\hat{p}} = -\Lambda^T \hat{p} \quad \text{---} \quad 7.10$$

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where,

$$A = \begin{bmatrix} 0 & 0 & 0 & 1 \\ b/c^2 & -b/c^2 & 0 & 0 \\ \frac{333 K_A}{F_{AT}} & 0 & \frac{-333 K_A}{F_{AT}} & 0 \\ \frac{2K_A + K_P}{J} & \frac{K_A}{J} & \frac{K_A}{J} & \frac{-B_P}{J} \end{bmatrix}$$

where, $b = 1000(1.25)K_A F_{AG}$ 71

$$c = .25 F_{AG} - K_{A1} + K_{A2}$$

$$\therefore A^T = \begin{bmatrix} 0 & b/c^2 & \frac{333 K_A}{F_{AT}} & \frac{2K_A + K_P}{J} \\ 0 & -b/c^2 & 0 & \frac{K_A}{J} \\ 0 & 0 & \frac{-333 K_A}{F_{AT}} & \frac{K_A}{J} \\ 1 & 0 & 0 & \frac{-B_P}{J} \end{bmatrix}$$

$$\begin{aligned} \dot{P}_1 &= -1.25(1000) K_A P_{AG} P_2 / (0.25 P_{AG} - K_A \theta_1 + K_A \theta_2)^2 \\ &\quad - 333 K_A P_3 / F_{AT} + (2 K_A + K_P) P_4 / J \quad \dots (7.11) \end{aligned}$$

$$\begin{aligned} \dot{P}_2 &= 1.25(1000) K_A P_{AG} P_2 / (0.25 P_{AG} + K_A \theta_2 - K_A \theta_1)^2 \\ &\quad - K_A P_4 / J \quad \dots (7.12) \end{aligned}$$

$$\dot{P}_3 = 333 K_A P_2 / F_{AT} - K_A P_4 / J \quad \dots (7.13)$$

$$\dot{P}_4 = P_4 B_P / J - P_1 \quad \dots (7.14)$$

The Hamiltonian functional is the inner product of the vectors $\dot{\theta}$ and P , plus the performance criterion; this functional must be maximized and equal to zero in order for optimality to be achieved.

$$\begin{aligned} H &= 1 + P_1 \theta_4 + 1.25(1000) P_{AG} P_2 / (0.25 P_{AG} + K_A \theta_2 - K_A \theta_1) \\ &\quad - 1000 P_2 + 333 (K_A \theta_1 - K_A \theta_3) P_3 / F_{AT} - 333 P_3 \\ &\quad + (K_A (\theta_2 + \theta_3 - 2 \theta_1) - K_P \theta_1 - B_P \theta_4) P_4 / J \quad \dots (7.15) \end{aligned}$$

In order for H to be optimal, let us consider it as a function of P_{AG} and F_{AT} . Maximizing the Hamiltonian implies

$$P_{AG} = \begin{cases} P_{max} & P_2 > 0 \\ P_{min} & P_2 < 0 \end{cases}$$

$$F_{AT} = \begin{cases} P_{\max} & P_3 < 0 \\ P_{\min} & P_3 > 0 \end{cases}$$

The number of switchings of P_2 and P_3 determine the exact force programme. It can be shown that P_4 does not change sign (9). Thus the coefficients of P_1 are positive. Thus if $-P_4(0)$ and $P_2(0)$ or $-P_4(0)$ and $P_3(0)$ are of opposite sign, then either P_2 or P_3 can change sign. However, once $-P_4$ and P_2 become of the same sign, they do not change sign again. Thus P_2 and P_3 can change sign only once implying F_{AT} and F_{AG} change only once.

It can be further shown (9) that the proper optimal control signals are of the same form as in Fig. 30(b). The activation deactivation time constants add one more switching of each controller signal so that the tension sources F_{AT} and F_{AG} approximate as closely as possible the optimal controls depicted in Fig. 30b. This is shown in Fig. 30c where the tension sources are shown with dotted lines; note how they approximate the signals shown in Fig. 30b when activation deactivation is ignored.

The responses of the model to first order control signal have already been given in Chapter VI. As has been shown there, these responses closely match the observed experimental human eye movements. It is, therefore, hypothesized

that human saccades are time optimal. The control pattern found in human electromyograms and animal nerve signals during saccadic eye movements also appear to be the same first order time-optimal sequence.

8. CONCLUSION

A model for the human eye tracking mechanism for vertical eye movement has been developed which includes the nonlinear and asymmetrical dynamic characteristics of the agonist - antagonist muscle pair (superior rectus plus inferior oblique - inferior rectus plus superior oblique) driven by nervous signals from the extraocular nuclei and which moves the eye to overcome orbital viscoelastic forces. The basic structure and function of this system was obtained from anatomical and physiological results on the extraocular muscles, the globe and the orbit.

The model is a realistic mathematical representation of the eye tracking mechanisms in which each parameter (of the model) can be identified with a physical counterpart. Its responses accurately depict the position, velocity and acceleration of saccades. It also reproduces, faithfully, the well known "main sequence" diagrams of saccadic velocity versus amplitude. Smooth pursuit movement has also been successfully simulated.

Lastly it has been shown that the human eye saccadic movement is time-optimal.

8.2 Scope for Future Work :

The model presented can also be tested for vergence eye movements and relevant dynamic characteristics can be studied.

The comparison so far given in this dissertation, of the model results, was in respect of data pertaining to horizontal eye movements. Data on recording of vertical eye movements is non-existent in literature. The model can, therefore, be tested against actual data of vertical eye movements.

The model can also be tested for dynamic stability by 'innore' technique(Bibliography).

A more accurate model for vertical eye movement can be constructed by considering the four extra ocular muscles concerned separately, instead of lumping them together to reduce them to two effective muscles, as has been done in this dissertation.

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A P P E N D I X

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CC      ASHWINI KUMAR      SACCADIC
READ    AKM, X Ø, XØM, DØX
1:      CONTINUE
2      =  (AKM/3.365) * (1. - EXPP(-175. * X Ø) *
          (3.2 * SIN F (55. * XØ) + COS F (55. * X Ø)))
IF (X Ø - XØM) 5, 5, 20
5 :     WRITE 10, 2, XØ
10:     FORMAT ( / , E, E)
XØ     =  XØ + DØX
GO TO 1
20:     STOP
END
```

10. , .001 , 0.1 , .001

All the other programmes were of the same type except for the value of 2.
