

***This thesis, having been approved by the  
special Faculty Committee, is accepted  
by the Graduate School of the  
University of Wyoming,  
in partial fulfillment of the requirements  
for the degree of*** Doctor of Philosophy

*Robert H. Bunn*

*Dean of the Graduate School.*

***Date*** October 6, 1967

A DUAL-MODE MODEL FOR THE NORMAL EYE TRACKING SYSTEM  
AND THE SYSTEM WITH NYSTAGMUS

by

Louis Frank Dell'Osso

A Thesis

Submitted to the Department  
of Electrical Engineering and the  
Graduate School of the University of Wyoming  
in Partial Fulfillment of Requirements for the Degree of  
Doctor of Philosophy

University of Wyoming

Laramie, Wyoming

January, 1968

## ACKNOWLEDGEMENT

The author wishes to acknowledge the guidance of his thesis advisor, Dr. F. M. Long as well as suggestions made early in the research by Dr. E. Lonsdale.

Special appreciation is extended to the Westinghouse Research and Development Laboratories where, along with many other lab facilities, the Burroughs B-5500 digital computer was made available. During the programming, the computer staff at the lab provided many helpful suggestions and I am especially grateful for the help of Dr. A. Lavi and G. Reilly who were chiefly responsible for programming this model on the Analog-Algol system, which was still being developed.

The author wishes to dedicate this thesis to the memory of Leonidas Miltiadis Mantgiaris, whose formative years contributed greatly to those of the author, and also to the author's wife, Lynn. Her love and understanding together with the insights gleaned through close friendship with a person of Leonidas' sensitivity have been and continue to be, of immeasurable value in the search for "the good life".

Finally, the author wishes to express his deep appreciation to his parents for providing their love, understanding and encouragement throughout the long process of learning which, at this point, seems to be just beginning.

## TABLE OF CONTENTS

CHAPTER	PAGE
1. HORIZONTAL NYSTAGMUS AND THE OCULOMOTOR SYSTEM . . . . .	1
Introduction . . . . .	1
Anatomy . . . . .	2
Types of Eye Motion . . . . .	5
Saccadic movements . . . . .	5
Smooth pursuit movements . . . . .	7
Compensatory movements . . . . .	7
Vergence movements . . . . .	7
Torsional movements . . . . .	8
Miniature movements . . . . .	8
Nystagmus . . . . .	9
Oculomotor Organization . . . . .	11
Cranial nerves . . . . .	11
Brain sites . . . . .	13
Neural pathways . . . . .	18
Genetics . . . . .	26
II. OCULOGRAPHY AND PHYSIOLOGICAL DATA . . . . .	31
Methods of Oculography . . . . .	31
Method Used . . . . .	34
Target Display . . . . .	35
Response to Predictable Sinusoids . . . . .	35
Transient Responses . . . . .	37

## TABLE OF CONTENTS (Continued)

CHAPTER	PAGE
Pulse response . . . . .	37
Step response . . . . .	37
Ramp response . . . . .	37
Parabolic response . . . . .	41
Responses to various velocities . . . . .	41
Step-ramp response . . . . .	41
Discussion of Data . . . . .	41
III. MODELING THE HORIZONTAL TRACKING SYSTEM . . . . .	50
Introduction to Modeling . . . . .	50
Proposed Models . . . . .	51
Saccadic and smooth tracking models . . . . .	51
Comparative evaluations . . . . .	56
Author's Model . . . . .	67
The saccadic positioning path . . . . .	68
The smooth tracking path . . . . .	71
Complete model for horizontal tracking system . . . . .	75
IV. COMPUTER REALIZATION . . . . .	84
System Used . . . . .	84
Program for the Normal Tracking System . . . . .	84
The non-linearities . . . . .	85
Sampling . . . . .	86
Equivalence to mathematical model . . . . .	86
Alternate program . . . . .	88

## TABLE OF CONTENTS (Continued)

CHAPTER	PAGE
Inputs . . . . .	89
Nystagmus . . . . .	89
Physiology . . . . .	91
Modeling nystagmus . . . . .	93
Complete Dual-Mode Program . . . . .	94
V. RESULTS . . . . .	99
Data From Computer . . . . .	99
Pulse responses . . . . .	99
Step responses . . . . .	101
Ramp responses . . . . .	101
Parabolic responses . . . . .	101
Step-ramp responses . . . . .	106
Discussion of Results . . . . .	106
VI. CONCLUSIONS . . . . .	110
Limitations of the Model . . . . .	110
Future Work . . . . .	112
BIBLIOGRAPHY . . . . .	114
APPENDIX A . . . . .	123
APPENDIX B . . . . .	127

## LIST OF TABLES

TABLE	PAGE
A.1 Transform Pairs . . . . .	125

## LIST OF FIGURES

FIGURE	PAGE
1.1 The Eye . . . . .	4
1.2 The Eye Muscles . . . . .	6
1.3 The Cranial Nerves . . . . .	14
1.4 Vertical Section Through the Brain . . . . .	15
1.5 The Cerebrum . . . . .	16
1.6 Hind- and Midbrains; Postero-lateral View . . . . .	17
1.7 Areas of the Cerebrum . . . . .	20
1.8 Ludvigh's System . . . . .	26
1.9 Neural Pathways . . . . .	27
1.10 Genetic Histories of Nystagmus . . . . .	29
1.11 Subject's Genetic History . . . . .	30
2.1 Block Diagram of Tracking Device . . . . .	36
2.2 Subject Using the Eye-tracking Device . . . . .	36
2.3 Gain and Phase Plots for Predictable Target Motion . . . . .	38
2.4 Pulse Response . . . . .	39
2.5 Step Response . . . . .	40
2.6 Sketch of Ramp Response (from Young) . . . . .	42
2.7 Sketch of Parabolic Response (from Young) . . . . .	42
2.8 Low Velocity Tracking . . . . .	43
2.9 Tracking a Target Moving at $10^{\circ}/\text{sec}$ . . . . .	44
2.10 Sketch of Step-Ramp Response (from Young) . . . . .	49
2.11 Positional Dependence of Nystagmus Amplitude . . . . .	49



## LIST OF FIGURES (Continued)

FIGURE	PAGE
3.1 Vossius' Model for Saccadic Movements . . . . .	53
3.2 Schematic Eye Movement Control System From Fender and Nye . . . . .	53
3.3 Young's Sampled-data Model of the Eye Tracking System . .	55
3.4 Pavlidis' Modified Model of the Eye Tracking System . . .	57
3.5 Warrneunde's Model of the Eye Tracking System . . . . .	58
3.6 Gain Plot for Unpredictable Target Motions (from Young) .	61
3.7 Ideal Response to Step Input . . . . .	64
3.8 Actual Response to Step Input . . . . .	64
3.9 Saccadic System . . . . .	70
3.10 Composite Signal Flow Graph of the Saccadic System . . .	70
3.11 Saccadic System Transient Responses . . . . .	72
3.12 Smooth Tracking (Pursuit) System . . . . .	74
3.13 Composite Signal Flow Graph of the Pursuit System . . . .	74
3.14 Pursuit System Transient Responses . . . . .	76
3.15 Sampled-data Model for Normal System . . . . .	77
3.16 Preliminary Flow Graph for Complete System . . . . .	79
3.17 Sampler $T_1$ Replaced by "Skip Samplers" . . . . .	79
3.18 Ramp Response of the Complete Model . . . . .	81
3.19 Parabolic Response of the Complete Model . . . . .	82
3.20 Step-ramp Response of the Complete Model . . . . .	83
4.1 Dead-Zone . . . . .	87
4.2 Velocity Limit . . . . .	87
4.3 Pursuit Saturation . . . . .	87

## LIST OF FIGURES (Continued)

FIGURE	PAGE
4.4 Alternate Program . . . . .	90
4.5 Nystagmus Waveshape . . . . .	95
4.6 Position Non-linearity . . . . .	95
4.7 Complete Dual-mode Model . . . . .	96
4.8 Dual-mode Program (with Variables) . . . . .	98
5.1 Dual-mode Pulse Response . . . . .	100
5.2 Dual-mode Step Response . . . . .	102
5.3 Dual-mode Ramp Response ( $10^{\circ}/\text{sec}$ ) . . . . .	103
5.4 Dual-mode Ramp Response ( $20^{\circ}/\text{sec}$ ) . . . . .	104
5.5 Dual-mode Parabolic Response . . . . .	105
5.6 Dual-mode Step-ramp Response . . . . .	107

## CHAPTER I

### INTRODUCTION

One method of studying a system involves the postulation and synthesis of a model to describe it. By studying such a model, one can often gain insight into the actual system of interest. This method of attack assumes greater importance when the system which is to be studied is one which is difficult to work with; due to the problems associated with presenting various inputs and measuring the responses under controlled conditions. The system which is the subject of this paper is the horizontal tracking mechanism of a normal subject and that of a subject with nystagmus. Physiological data will be taken from both subjects so that the responses may be compared. Based on this data and that of other investigators, a model will be postulated which will represent the normal tracking system. This model will then be modified to describe the system with nystagmus. Both models will be programmed on a computer and the transient responses will be compared to those of the biological system.

Throughout this paper previous works will be cited and discussed as each area of the total study is treated. In this way, a continuous transition from past investigations and conclusions to present thought on each particular subject will be presented.

The combination of control theory and extensive use of electronic computers has caused the emergence of the field of modeling physiological control systems so that the intricate balances present in the

living organism may be further studied. While one cannot expect to make an exact correlation between the electronics which comprise the functional black boxes of the model and the neural and muscular structures which make up the biological system, the existence of the functional correlations which gave rise to the model does enable the medical investigator to study a complex control system by considering its various functional sub-systems separately. Going beyond the normal system, the modeling of one which is abnormal extends this mode of investigation into the study of the pathological condition itself and how it affects the operation of the whole system. Whether or not the ultimate goal will be reached this way (that is, understanding the affliction to the point of discovering a cure) is something which remains to be seen. One other thing whose value should be pointed out is the quantitative data collected on both the normal subject and, for the first time (to the author's knowledge) the subject with nystagmus. These recorded responses and the methods employed to obtain them could become a valuable tool in treating this and other eye conditions.

### 1.1 ANATOMY

The eyeball is an almost spherical shell which lies in the orbital cavity in a surrounding bed of fibrous tissue and fat (1). It is filled with a viscous fluid called the vitreous humor. The hard shell is the sclera which is continuous except for the front where the cornea protrudes. The cornea is the front lens and has a power of 42 diopters and a radius of curvature of 8 mm. Behind the cornea is the iris which is a group of muscles whose contractions or relaxations vary the eye's

aperture or pupil. The lens lies just behind the iris and its variable power capability is used to focus an image on the retina which lies on the rear inner surface of the eye. In the retina are two types of photo-sensitive cells called the rods and the cones. The rods, which are predominantly distributed around the periphery of the retina, are used for night vision and the cones, more centrally located, are responsible for day vision as well as color vision. The highest acuity occurs at the fovea which is an area of about 60 microns in diameter (this corresponds to a visual angle of about  $1^{\circ}$ ) and consists entirely of cones. The internal anatomy of the eye is shown in Fig. 1.1 along with notes on the physiology of its parts (2).

Connected to the eye are six muscles. These muscles are the extrinsic eye muscles or the extra-ocular muscles. They enable the eye to rotate and to properly position an image on the fovea of the retina. These muscles are arranged in three pairs. During any eye movement one of the muscles of a pair will contract (the agonist) and one will relax (the antagonist). Thus, the agonist pulls the eye while the antagonist maintains an opposing tension to the motion. The center of rotation of the eyeball is essentially at its geometric center (3). Normally these muscles give the eye a range of motion of about  $45^{\circ}$  left or right,  $40^{\circ}$  up and  $60^{\circ}$  down, and  $30^{\circ}$  in extorsion or intorsion. These muscles are attached at opposite ends of eyeball diameters which give them a large mechanical advantage. Also, they are 40 mm in length and 100 square millimeters in cross section which should give them the capability of exerting a force of nearly 1 kg weight. The actual force necessary for a moderate saccadic movement has been calculated

# EYE

## STRUCTURE

## FUNCTION of PARTS

The Eye ball has three coats:-

### 1. OUTER or SCLEROTIC COAT

### PROTECTIVE LAYER

Tough fibrous tissue ---- Preserves shape of eyeball and protects delicate inner layers.

Transparent CORNEA ---- Allows passage of LIGHT RAYS.

[Extrinsic muscles are attached to sclera. .... Permit and limit movements of eyeball within ORBIT.]

### 2. MIDDLE or VASCULAR PIGMENTED COAT

### LAYER of SUPPLY

Contains main arteries and veins of eyeball.

Circular opening at front - PUPIL.

Coloured muscular ring - IRIS - surrounds pupil.

CILIARY BODY.

CILIARY MUSCLE.

SUSPENSORY

LIGAMENT

suspends

CRYSTALLINE LENS.

CHOROID - Posterior

5/6 of Vascular

Coat.

3. INNER or NERVOUS

COAT - the RETINA

Lines back of eye.

Contains

RECEPTORS for

VISION

Controls size of pupil and amount of light entering eye.

Produces AQUEOUS HUMOUR.

Contracts and moves forwards.

Relaxes to allow curvature of lens to alter for accommodation for NEAR VISION.

Brings light rays to focus on light-sensitive RETINA.

### LIGHT-SENSITIVE LAYER

Highly specialized to respond to stimulation by light. Convert light energy into nerve impulses.

CORNEA

ANTERIOR CHAMBER  
(AQUEOUS HUMOUR)

IRIS

SUSPENSORY  
LIGAMENT

CILIARY  
BODY

LENS

(VITREOUS  
HUMOUR)

RETINA

CHOROID

SCLERA

Main blood vessels  
to retina enter  
and leave here.

CENTRAL ARTERY of  
the RETINA

OPTIC NERVE

Conveys these impulses to  
VISUAL CENTRES in OCCIPITAL  
(posterior) part of BRAIN.

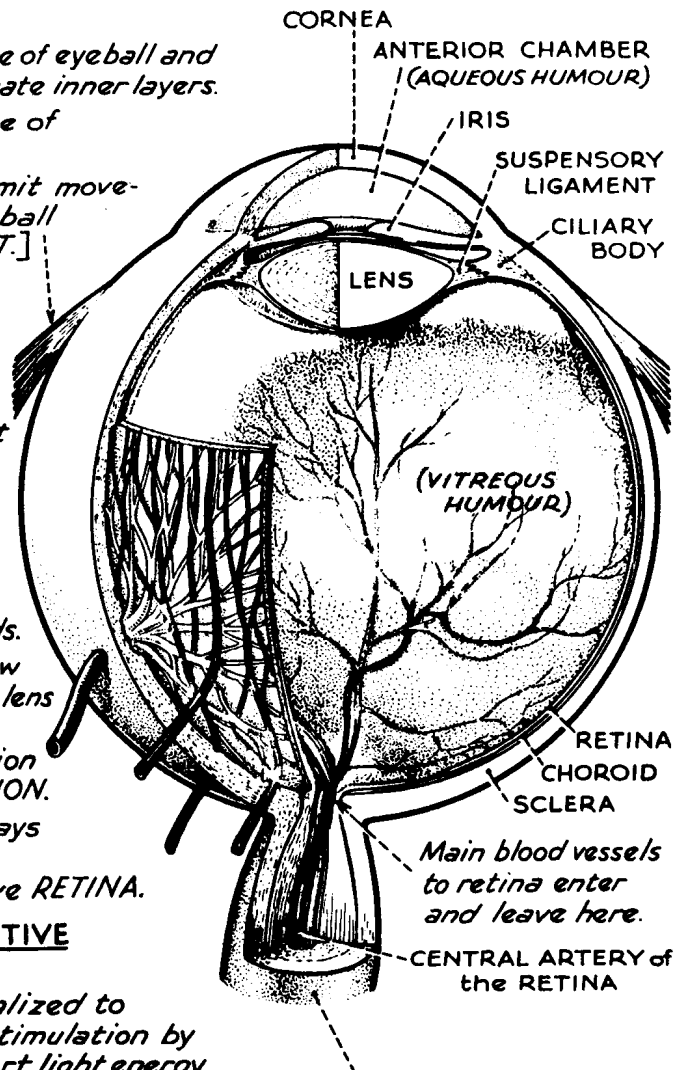


Fig. 1.1 The eye (2)

at 100 g weight. This large reserve allows for extremely large velocities ( $600^{\circ}/\text{sec}$ ) and also explains the fact that these muscles do not normally fatigue (4). With the large number of innervating fibers present in proportion to the muscle size, the brain can exercise very fine control. The extra-ocular muscles have the shortest reaction time (less than 10 msec) of any muscle in the body (5). Fig. 1.2 shows these muscles, their points of attachment and the cranial nerves which innervate them (2). Since this paper is concerned only with movements in the horizontal plane, only the medial (internal) and lateral (external) rectus muscles and cranial nerves III and VI will be considered.

## 1.2 TYPES OF EYE MOTION (6)

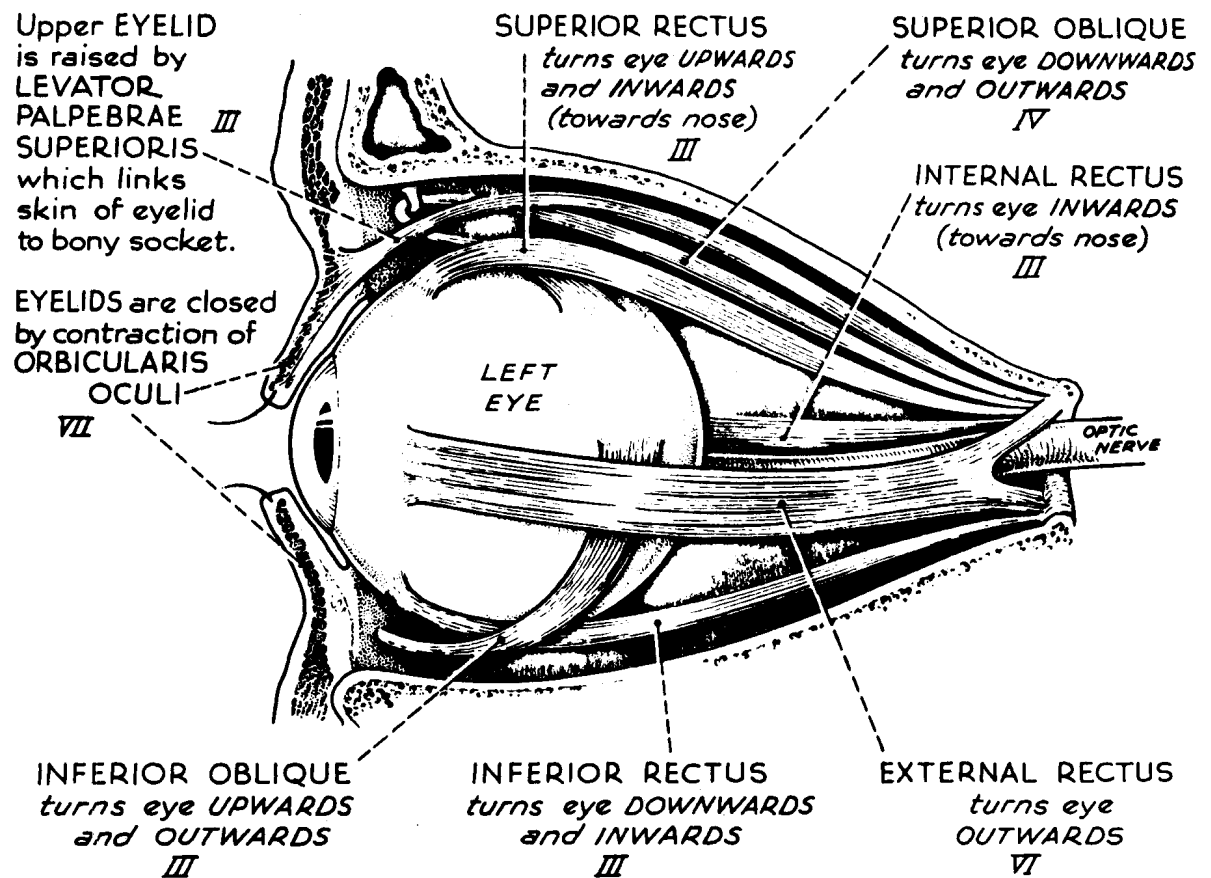
Before proceeding further into the anatomy and physiology involved in the horizontal tracking mechanism of the eye, it would be advantageous at this point to name and define the various types of motion the eye is capable of and to point out those we will consider.

### 1.2.1 Saccadic Movements

A saccadic movement is one in which the eyes are moved conjugately from one position to another in a step-like manner (also referred to as a jerk or jump). Given an initial target displacement, the eye will not respond for 120-250 msec (response time) (7,8). Then there will be a period of acceleration, followed by a constant velocity segment and then a period of deceleration. Many times there will be some overshoot before reaching the final value. At present there is evidence that during a saccade vision is cut off, but this has not been proven. There are two theories concerning the nature of this movement. One school of thought pictures a saccade as a preprogrammed ballistic

## MUSCLES of EYE

The EYEBALLS are moved by SMALL MUSCLES which link the SCLEROTIC COAT to the BONY SOCKET.



*Acting together, the Extrinsic muscles of the Eyeballs can bring about ROTATORY movements of the Eyes.*

*The Extrinsic muscles are supplied by motor fibres from Cranial Nerves III, IV and VI.*

Fig. 1.2 The eye muscles (2)



movement (9). This would be an essentially open loop system. The other concept is that the saccade is controlled in a closed loop manner by position feedback from the extra-ocular muscles. The question of the existence of proprioceptive feedback is one which is widely disputed and will be discussed in the section dealing with neuro-muscular pathways.

#### 1.2.2 Smooth Pursuit Movements

Smooth pursuit movements enable the eye to follow (or track) moving targets with angular velocities of from  $1^{\circ}/\text{sec}$  to  $25-30^{\circ}/\text{sec}$  (10,11). As with saccades, there is a latent period associated with smooth tracking and it is about 120-250 msec. The object of smooth pursuit is to keep the eye focused on a moving target. The angular velocity of the eye is found to be linearly proportional to target velocity and the initiation for this mode is a detection of retinal image motion, whereas the saccadic mode is triggered by a position deviation on the retina. It has been shown experimentally that pursuit is a semi-voluntary mode. One can voluntarily follow a moving target but one cannot move his eyes smoothly from one position to another without such a moving target to follow (12).

#### 1.2.3 Compensatory Movements

These movements help stabilize the retinal image in the presence of head or body movements. They are smooth movements in a direction opposite to the motion. These movements are limited to a velocity of  $30^{\circ}/\text{sec}$  and their latent period is about 100 msec (6,13).

#### 1.2.4 Vergence Movements

These movements are an important part of binocular vision since it is the amount of convergence plus the amount of lens accommodation

which accounts for the perception of distance (14). Vergence movements differ from saccadic, pursuit, and compensatory movements by causing the eyes to move in opposite directions instead of in the same direction as conjugate movements do. Thus, as a target moves toward or away from the subject, his eyes will converge or diverge respectively so that the image falls on corresponding parts of the retina. Experiments have shown that the vergence and tracking systems are independent and that vergence movements are smooth and quite a bit slower than conjugate movements (15,16). Also, a target displacement which would require a step change in convergence gives rise to velocities in the order of  $10^{\circ}/\text{sec}$  and the time necessary for the change is about 1 sec.

#### 1.2.5 Torsional Movements

These movements are rotations about an axis running horizontally from the center of the pupil back through the retina. They are compensatory in nature and they serve to maintain the image on the fovea as the subject rotates his head about the line of sight. The muscles which are mainly involved in this rotation are the inferior and superior oblique muscles (17). It has been observed that the eyes may lag behind the head by as much as  $30^{\circ}$ .

#### 1.2.6 Miniature Movements

During fixation on a stationary target, in the absence of large movements caused by some pathological condition, three types of very small movements (less than  $1^{\circ}$ ) can be observed (18,19). While they may also be present during large movements, both voluntary tracking and involuntary excursions of the eye, it is at present extremely difficult to verify this experimentally.

(1) Drift: This is a random movement of the eyes which occurs during "steady" fixation. The velocity of this random motion is usually less than several minutes of an arc per second (20).

(2) Saccades: Small saccades in a direction opposite to the drift have been observed to occur within 30 msec of each other (21). The purpose of these saccades is to keep the image focused within the foveal region of about  $1^{\circ}$  (22).

(3) Tremor: Tremor is a high frequency vibration of the eyes (23). The range of frequencies of this movement is from 30 to 150 Hz with a peak in energy between 70 and 90 Hz and an amplitude which varies from 5 to 15 sec/arc (24). There is evidence that tremor is independent for each eye. This would suggest that the mechanism involved is not the same one that is responsible for conjugate tracking movements. The purpose of tremor is perhaps brought out by experiments which have been performed using an optical method to negate the effects of tremor on the retina. When such tests were made, the subject reported that the image faded out after several seconds, and then continually appeared and disappeared (25). From this it has been postulated that the purpose of tremor is to prevent the saturation of any one cone by continually sweeping across several cones. Another, or perhaps simultaneous, purpose might be to help one to accurately determine boundaries that would be difficult to perceive because of the random way in which rods and cones are distributed on the retina.

#### 1.2.7 Nystagmus

Movements which are oscillatory or unstable are referred to as nystagmus movements (26). There are several types of nystagmus which have been observed, some of which are described below (27,28,29,30,31).

(1) Vestibular Spontaneous Nystagmus: This type of nystagmus is always of the jerk type and is more prominent with lid closure and tends to diminish during fixation. It is more prominent in a fully aroused subject and diminishes as his fatigue increases (32). Respiration also affects this type of nystagmus. Vestibular nystagmus can arise from either central or peripheral locations. Related types of nystagmus are positional head-shaking, and toxic nystagmus. Vestibular nystagmus can be induced by rotation of the subject (33) or by irrigating the ear canals with warm or cool water (34).

(2) Gaze Nystagmus: There are two types of gaze nystagmus; gaze paretic and gaze direction nystagmus. The paretic type is accompanied by partial gaze paresis in the direction of the quick phase and is usually at frequencies of 1 to 2 Hz. The directional type is usually at frequencies of 3 to 8 Hz and its amplitude increases with increasing gaze deflection from the straight-ahead position.

(3) Fixation Nystagmus: Fixation nystagmus is almost always congenital and is the type of nystagmus that will be of interest in the present study. More specifically, we will be concerned with horizontal fixation nystagmus, which is oscillation in the horizontal plane only. While it is possible for a person with fixation nystagmus to have spontaneous nystagmus with closed eyes, the subject used in this study did not have this additional condition so it will not be investigated at this time. Typical fixation nystagmus does, however, show up in complete darkness when the gaze is directed in a particular direction by a stimulus other than an optical one (i.e. an acoustic stimulus).

Nystagmus can be induced in a subject by directing his gaze to a rotating drum which has alternating black and white vertical bars on

it and which is rotating about a vertical axis so that the bars pass from one side of the subject's visual field to the other. In this way, the movement of the visual field from left to right or right to left will induce the eyes to conjugately track a part of the field as it moves until the eyes reach an uncomfortable position at one end of the range of eye excursion. At this point, the eyes will jump back to the central position and the above sequence repeated. This is referred to as optokinetic nystagmus and it is a valuable diagnostic tool.

### 1.3 OCULOMOTOR ORGANIZATION

This section is devoted to the cranial nerves, neural pathways, and sites in the brain which make up the oculomotor system. An attempt will be made to present the various dissenting views which are associated with this topic and, where possible, try to arrive at the conclusions that are most likely to yield a true picture of this very complex and controversial system.

#### 1.3.1 Cranial Nerves

Five of the twelve cranial nerves are involved, directly or indirectly, in controlling the movements of the eyes. They are the optic (II), the oculomotor (III), the trochlear (IV), the trigeminal (V), and the abducens (VI), nerves.

(1) Optic: This nerve is the nerve which is responsible for vision. It arises from cells in the retina, exits the eye in the rear at a point on the retina known as the "blind spot" and enters the skull through the optic foramen (1,35). The two optic nerves join at the optic chiasma where fibers from the nasal half of each retina cross over to the other side and fibers from the temporal half continue in the

optic tract of the same side. The fibers of the optic tract then proceed to the brain stem where they terminate. From the brain stem other fibers carry the visual impulses to the visual cortex at the back of the occipital lobe.

(2) Oculomotor: The point of origin of the oculomotor nerve is the under surface of the brain stem in front of the pons. From there it goes forward through the superior orbital fissure into the orbit where it supplies the superior rectus, inferior rectus, medial rectus, inferior oblique, and levator palpebrae superioris muscles. The smooth muscles of the ciliary body and iris are also served by this nerve.

(3) Trochlear: The dorsal surface of the midbrain is the point of origin of the trochlear nerve. From there it passes around the side of the brain stem and goes forward through the superior orbital fissure into the orbit where it supplies the superior oblique muscle.

(4) Trigeminal: There are two roots of this nerve, a sensory and a motor, which are attached close together on the side of the pons. The semilunar (Gasserian) ganglion lies on the sensory root at the point where it passes into the middle fossa of the skull. There are three large sensory branches which bring fibers into this ganglion: (a) the ophthalmic, bringing fibers from the orbit, upper eyelid, bridge of the nose, and scalp as far as the crown of the head; (b) the maxillary, with fibers from the lower eyelid, the lower portions of the nose, the cheek, the upper lip, jaw, and palate; and (c) the mandibular, with sensations from the lower lip and jaw, lower part of the face, and front of the ear. The ophthalmic nerve passes through the superior orbital fissure, the maxillary through the foramen rotundum, and

mandibular through the foramen ovale. The motor root lies below the semilunar ganglion and it is continued into the mandibular nerve at the foramen ovale. The fibers go to the muscles of mastication, the masseter muscle, and the temporal muscle. We will not be interested in the motor portion of the trigeminal nerve in this paper.

(5) Abducens: The abducens nerve originates from the under side of the brain stem behind the pons. It passes into the orbit through the superior orbital fissure and supplies the lateral rectus muscle.

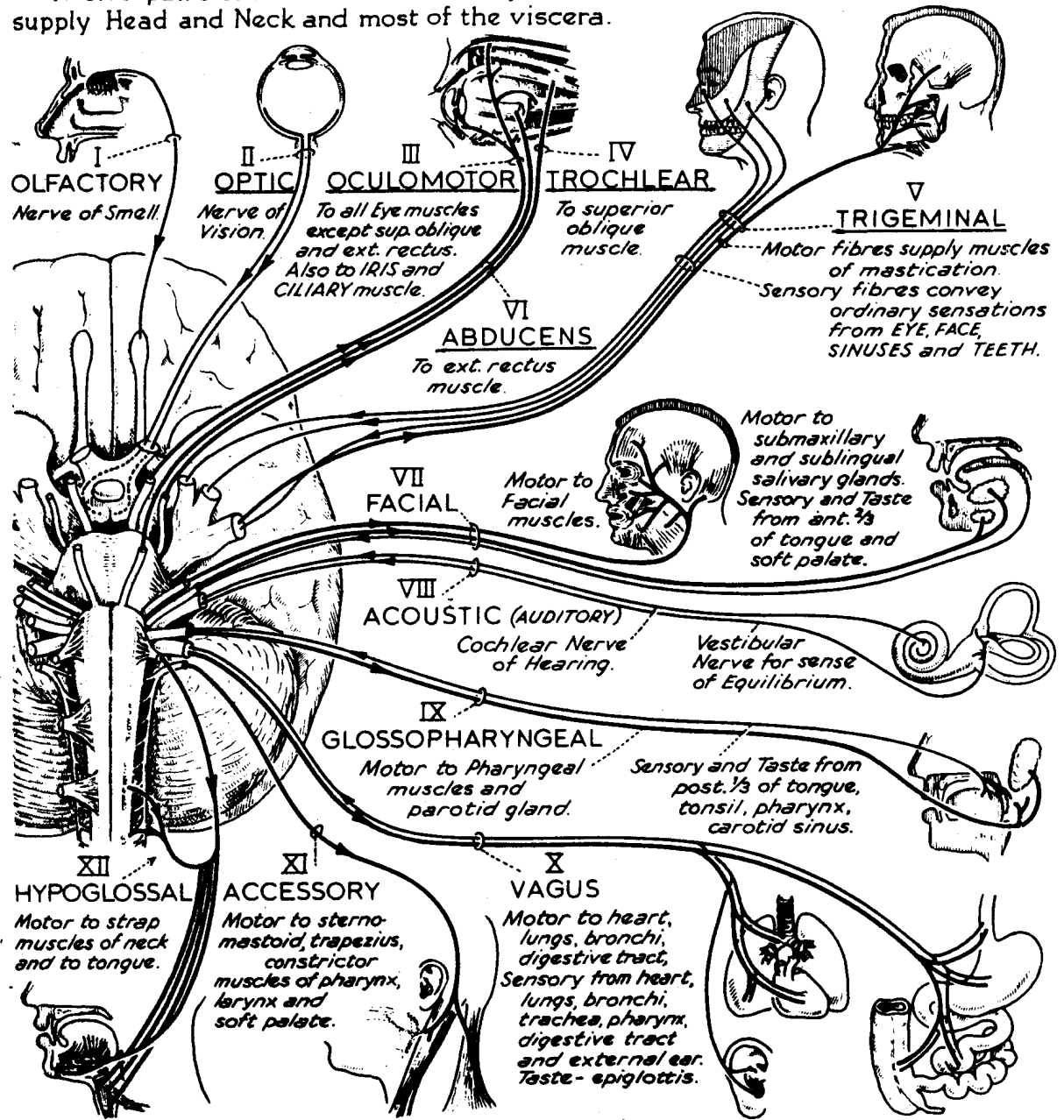
The above descriptions will be more meaningful to the reader if they are used in conjunction with Fig. 1.3 (2). Also shown in this figure are the points of insertion of the nerves into the brain.

### 1.3.2 Brain Sites (35,36,37)

Since the various areas of the brain will be referred to when discussing the neural pathways involved in the transmittance of visual and command information, it will help to become familiar with the relative positions of these areas. To do this most efficiently, Fig. 1.4 and Fig. 1.5 have been provided (2). The areas of most concern are: the frontal lobe, the parietal lobe, the occipital lobe, the pons, the mid-brain, and the cerebellum. Also of interest are the thalamus and corpora quadrigemina, shown in Fig. 1.4, and the various areas of the cerebrum which are shown in Fig. 1.5. Some areas of special interest are: (1) the lateral geniculate body; (2) the superior colliculus; (3) the pretectal region; (4) the motor nucleus of the oculomotor nerve (MN-III); (5) the motor nucleus of the abducens nerve (MN-VI); and (6) the proprioceptive nucleus of the trigeminal nerve (MN-V) which is also referred to as mesencephalic nucleus V.

## CRANIAL NERVES

Twelve pairs of nerves arise directly from the undersurface of the Brain to supply Head and Neck and most of the viscera.



(After Frank H. NETTER, M.D., The Ciba Collection of Medical Illustrations)

Fig. 1.3 The cranial nerves (2)



## VERTICAL SECTION through BRAIN

This is a Vertical Section through the LONGITUDINAL FISSURE — a deep cleft which separates the two Cerebral Hemispheres. At the bottom of this cleft are tracts of nerve fibres which link up the different LOBES of each hemisphere and also link the two hemispheres with each other — the CORPUS CALLOSUM.

### FOREBRAIN

#### Cerebral Hemisphere

#### Thalamus

-relay centres for sensation: pain appreciated here.

#### Hypothalamus

-contains centres for Autonomic Nervous System, e.g. Control of Heart, Blood pressure, Temperature, Metabolism, etc.

### MIDBRAIN

Receives impulses from Retina and Ear. Serves as a centre for Visual and Auditory Reflexes. In the Grey Matter are nerve cell bodies of III, IV Cranial nerves and the Red Nucleus which helps to control skilled muscular movements. The White Matter carries nerve fibres linking Red Nucleus with Cerebral Cortex, Thalamus, Cerebellum, Corpus Striatum and Spinal Cord. It also carries Ascending Sensory fibres in Lateral and Medial Lemnisci, and Descending Motor fibres on their way to Pons and Spinal Cord.

### HINDBRAIN:

[PONS, CEREBELLUM, MEDULLA OBLONGATA]

Pons: Groups of Neurones form sensory nucleus of V and also nuclei of VI and VII Cranial nerves. Other nerve cells here relay impulses along their axons to Cerebellum and Cerebrum. Rubrospinal tract, Lateral and Medial Lemnisci pass through Pons and nerve fibres linking Cerebral Cortex with Medulla Oblongata and Spinal Cord.

#### Medulla Oblongata:

Groups of Neurones form Nuclei of VIII, IX, X, XI, XII Cranial nerves. Gracile and Cuneate Nuclei — second sensory neurones in cutaneous pathways. Tracts of Sensory fibres decussate and ascend to other side of Cerebral Cortex. Some fibres remain uncrossed. The larger part of each Motor pyramidal tract crosses and descends in other side of Spinal Cord.

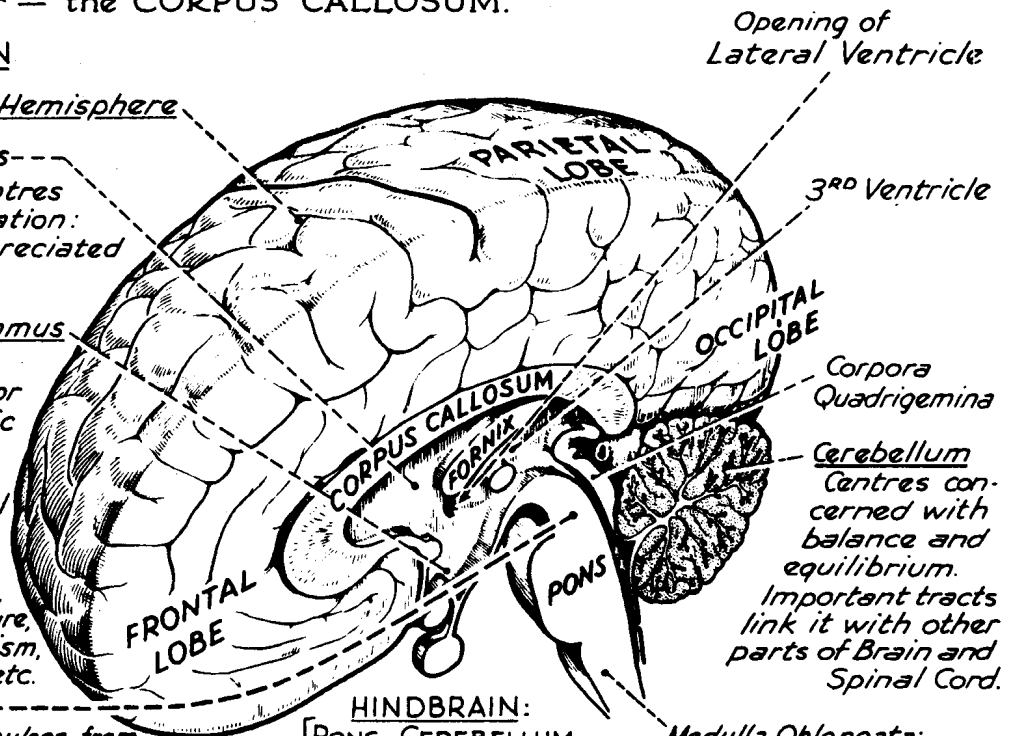


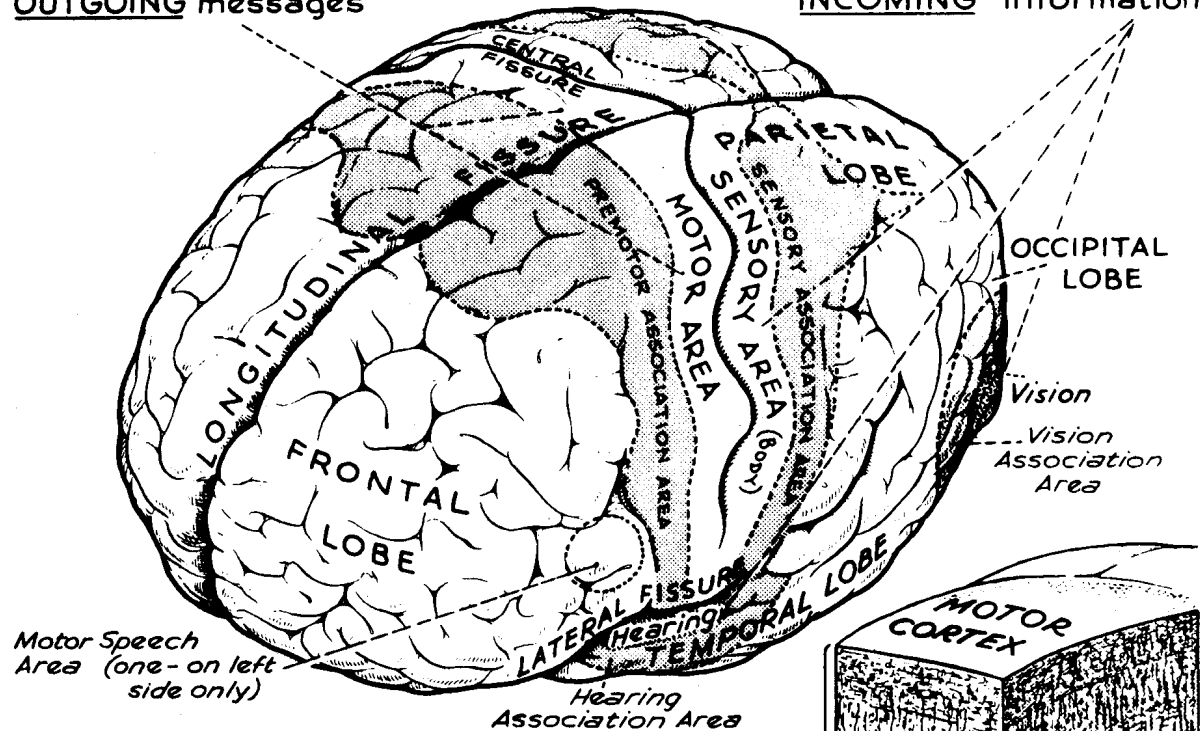
Fig. 1.4 Vertical section through the brain (2)

## CEREBRUM

The largest part of the human brain is the CEREBRUM — made up of 2 CEREBRAL HEMISPHERES. Each of these is divided into LOBES.

INITIATING CENTRES for  
OUTGOING messages

RECEIVING CENTRES for  
INCOMING information



Large uncharted areas of the Cerebral Hemispheres are probably concerned with MENTAL PROCESSES such as *Intelligence, Memory, Judgement, Imagination, Creative and Conscious Thought.*

The surface of the brain shows many folds or CONVOLUTIONS. This has the effect of increasing the amount of GREY MATTER present. The GREY MATTER forms the outer layer or CORTEX. It contains the cell bodies of the NEURONES arranged in many interconnecting layers to form a 3-dimensional network.

About 90% of all Nerve Cells are in the Cerebral Cortex.

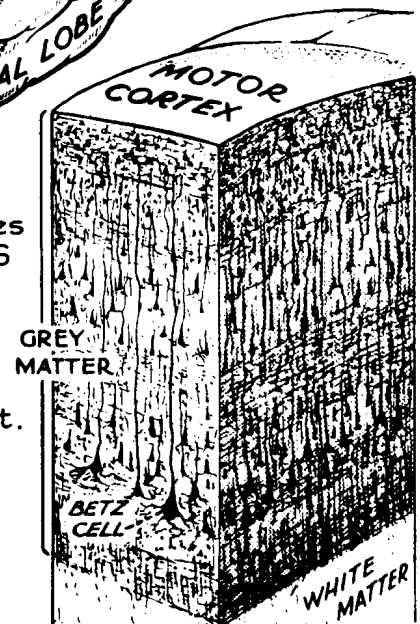


Fig. 1.5 The cerebrum (2)

(1) **Lateral Geniculate Body:** This is an oval-shaped body which is located lateral to the posterior end of the thalamus. It is at the level of the pineal body as can be seen in Fig. 1.6 (35).

(2) **Superior Colliculus:** This body is located on the upper part of the corpora quadrigemina just below the pineal body (see Fig. 1.6).

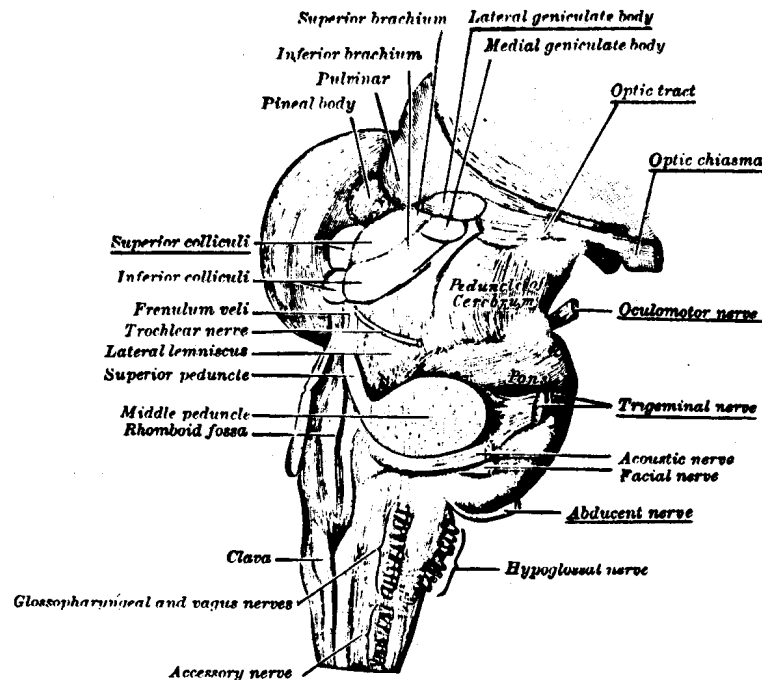


Fig. 1.6 Hind- and midbrains; postero-lateral view (35)

(3) **Pretectal Region:** The pretectal region is a transition zone which lies between the thalamus and the superior colliculus.

(4) **MN-III:** The oculomotor nucleus consists of two parts, the somatic and the autonomic nuclei. The somatic nucleus consists of definite groups of neurons for the individual muscles which can be mapped topographically. The autonomic nucleus lies dorsal to the somatic and is composed of a lateral portion called the Edinger-Westphal nucleus and a medial portion which contains similar small, motor-type cells.

The oculomotor nucleus lies in the gray matter ventral to the aqueduct and is from 6 to 10 mm in length. Adjacent to this, as well as all the nuclei of the extra-ocular muscles, is the mesencephalic nucleus of the trigeminal nerve which supplies the proprioceptive fibers of cranial nerves III and VI.

(5) MN-VI: This nucleus is a small spherical mass in the lower portion of the pons and is the most caudal of the nuclei of the motor nerves to the eye.

(6) MN-V: The trigeminal nerve contains somatic sensory, special visceral efferent, and proprioceptive fibers. Since we are only interested in the proprioceptive fibers we will discuss the nuclei of the mesencephalic root. The mesencephalic root arises from unipolar cells which are located in groups at the lateral edge of the gray matter that surrounds the upper end of the fourth ventricle. It is from this nucleus that the proprioceptive fibers of the III and VI cranial nerves are supplied.

### 1.3.3 Neural Pathways

The tracing of the neural paths involved in the transmission of visual impulses from the eye to the brain, the internal brain-paths which interpret the visual signals and cause motor commands to be sent back to the eyes, as well as the paths for the command signals is a necessary part of this study even though some of the information available is contradictory. An effort will be made to trace only those paths related to the horizontal tracking system and to provide, in those areas where a controversy exists, references in support of and in opposition to the author's contentions.

(1) Visual Information: From the retina both position and rate of change (velocity) information are sent via the optic nerve to the optic chiasma (9,38,39). At the chiasma the fibers of the nerves divide as described in section 1.3.1. The information is now carried in the optic tracts to the lateral geniculate bodies. Most of the fibers terminate in the lateral geniculate body but some go on to the superior colliculus and the pretectal region (35). Wiesel has shown the existence of a mapping between the retina and the lateral geniculate body (40). Fibers go to the visual cortex in the occipital lobe from the lateral geniculate body. The fibers from the pretectal region go to the Edinger-Westphal nucleus where they form part of the pupillary constriction reflex arc which will not be considered in this paper. The superior colliculus is the center for the coordination of eye movements.

(2) Interpretation: Area 17 of the visual cortex is the area in which the visual signals are interpreted for color, form, size, motion, illumination, and transparency. Areas 18 and 19, which surround the visual sensory area, are the visual psychic areas (see Fig. 1.7) (35). It is here that visual impressions are elaborated and associated with past experience for recognition and identification. Eye movements are also related to visual impressions in these areas (35,41).

(3) Motor Commands: At this time there is no definite evidence or agreement as to the layer of the visual cortex in which the motor fibers begin (39,41). Some authors consider the beginnings of the motor path to lie in area 17, but Hines has presented evidence that the efferent path begins in area 18 and area 19 (42). From the area, or areas, of origin the signals pass through the superior colliculus to

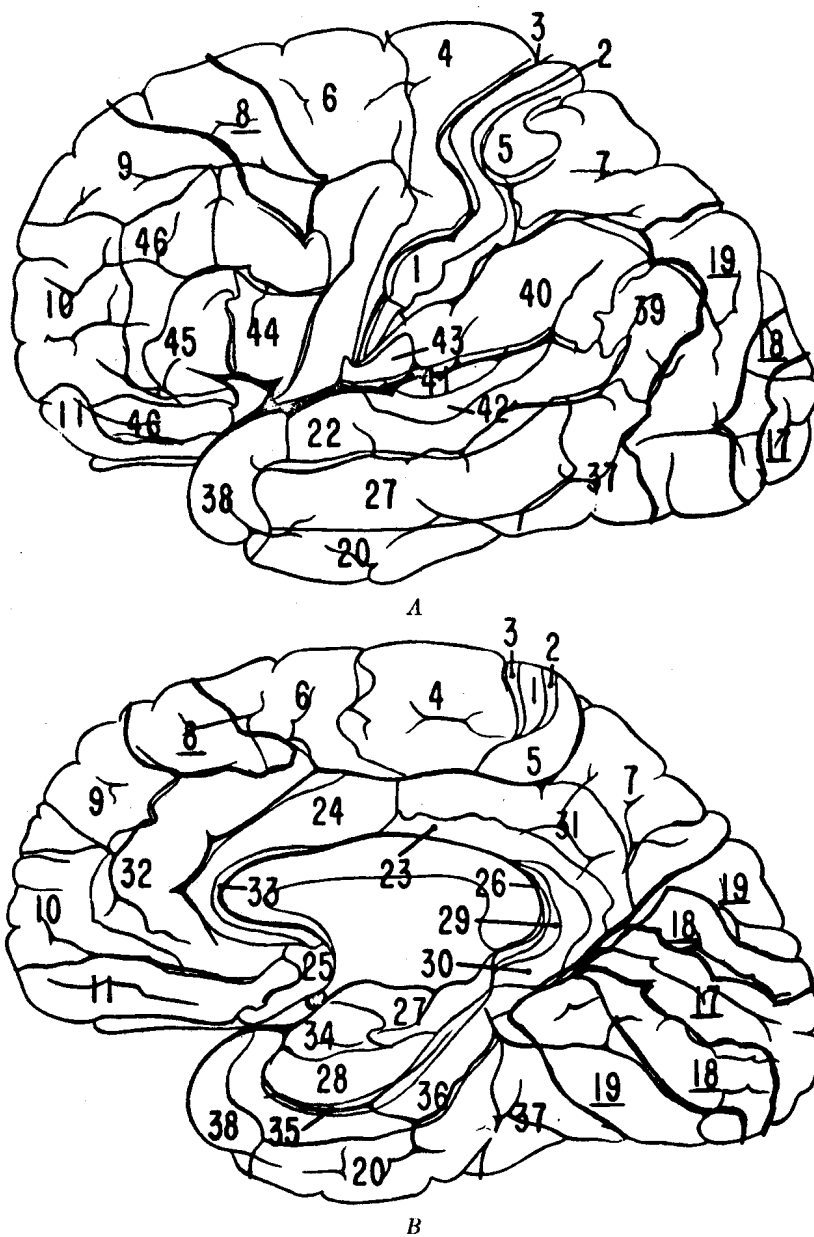


Fig. 1.7 Areas of the cerebrum (35) - A) lateral, B) medial

the vestibular nucleus where the commands for conjugate movement are sent to the motor nuclei of cranial nerves III and VI (43,44). Voluntary movements of the eye are a result of signals from the second and third frontal convolutions of each hemisphere (area 8). The signals then pass through the oculogyric centers in the frontal lobe to the motor nuclei of the oculomotor nerves. Some clinicians feel that there is a pontine center for lateral gaze but there is no histologic evidence for such a center (39). The signals arriving at the motor nuclei are then sent to the oculomotor muscles via cranial nerves III and VI where they cause the eye to move in the desired direction. This completes the forward path in the system along with the main feedback path which is the displacement signal that results from the retinal motion.

(4) Proprioception: The question of whether or not proprioception exists in the extra-ocular muscles and, if it does, what its role might be is one which has been the subject of controversy and discussion since 1867 when H. von Helmholtz published his now famous experiments on the subject (45,46). His observations are subjective and the experiments are simple enough so that anyone may repeat them. He pointed out that if one is looking at stationary objects in the surroundings and moves his point of fixation from one object to another these objects do not appear to have moved even though their retinal images have undergone a displacement. Apparently there is some mechanism which allows for eye and head movements when interpreting retinal images because, if there weren't, the retinal image motion would give rise to the sensation of object movement in the real world. Alternately, if one fixes his gaze on a stationary object and then physically moves his eye

by means of his finger the sensation is one of motion of the surroundings. Thus, retinal image motion gives rise to apparent object movement in the latter case and to non-movement in the former case. This experiment is cited by Helmholtz as proof that the mechanism involved is not one of proprioception from the extra-ocular muscles since in both cases such proprioceptive signals would have been sent to the brain as a result of the muscle movement. The explanation put forth by Helmholtz was that one was aware of eye movements by a knowledge of the innervation sent to the extra-ocular muscles to effect the movement. Experiments with after-images recently conducted by Brindley support this contention (47). Brindley also stated that the movement of one eye does not cause a reflex movement in the other. These results were also found by Merton who used after-images to show the absence of conscious position sense in the dark (48,49). In 1936, Irvine made a histological study of the extra-ocular muscles in which he found little anatomic evidence for a proprioceptive sense (50). The same conclusions were reached by McCouch in 1932 and by Hyde in 1960 although Hyde did concede there was some evidence for proprioception (51,52). In the 1930's, extensive work was done by Irvine and Ludvigh in which they claimed that there was neither a conscious nor an unconscious proprioceptive sense (53). They also stated that proprioception played no part in projection or motion interpretation. Later work by Ludvigh in 1952 substantiated his conclusions concerning the absence of a conscious position sense but he postulated a system with parametric feedback operating at an unconscious level (54,55).



At the same time the above-mentioned investigators were at work trying to support Helmholtz's "outflow" theory of position sense, others were conducting experiments whose results tended to dispute Helmholtz's contentions. One of the first persons to do so was William James (56). He hypothesized that signals from an eye which has moved contain position information and he used this to explain why a person with one paralyzed eye perceives target motion when he tries to move his eye toward an object located in a part of the field to which he has difficulty moving his eye. Since his good eye is being covered, signals from it, according to James, account for the apparent motion and inability to correctly point to the object (subject always points past the object). The reason is that the good eye has moved a greater distance from the center line than would have been necessary in an effort to move the paralyzed eye. Thus, the object seems further to the side than it actually is. Later investigations proved that positions taken up by the sound eye were not related to the false projection. The most powerful arguments to the "outflow" theory, if judged by the impact they had on future study, were those of Sherrington and later, Tozer (57,58,59,60, 61,62). These experiments, published from 1893 to 1918, presented evidence which supposedly verified the existence of a position sense which enabled very accurate direction of gaze in total darkness. Another experiment which Sherrington designed consisted of two sets of three dots on a wall in front of a subject. Each set consisted of the three dots arranged in a vertical line. One set was directly in front of the observer and the other was up and to the right of the straight-ahead position. When the subject directed his gaze straight ahead to the

first set of vertical dots he perceived them just as they were; a vertical array of three dots. He knew this by the fact that their images fell on parts of each retina which, experience had taught him, represented verticality. If the subject then directed his eyes up and to the right to the other set of dots he received the correct impression that they were a set of three dots arranged in a vertical line and situated up and to the right of the first set. It was shown that, due to torsional movements, the images no longer fell on the same receptors in the retina as they did in the previous case. The images actually fell on areas which lie obliquely to the original areas. This should have created the impression that the dots lie on an oblique line from the vertical. Sherrington postulated that proprioceptive position information was responsible for the correct impression of the dot placement. This is an over-simplification. As Irvine and Ludvigh pointed out, just because retinal receptors, which should produce a sense of the oblique, are stimulated there is no reason to suppose that they in fact do so or, that there exists another mechanism which alters their message (53). The factor of experience can modify perception greatly under varying conditions. As is pointed out in Adler's text, a dinner plate viewed from an angle is still perceived as being round even though the retinal image is that of an ellipse (39).

More recently Cooper, Daniel, and Whitteridge have, by a thorough histologic examination, proven the existence of proprioceptive fibers in the extra-ocular muscles (63,64,65,66,67). They found many spiral nerve endings in these muscles. The signals from these endings were traced to MN-V, the superior cerebellar peduncle, the ventral tegumental

region, the central tegumental tract, the superior colliculus, and the medial longitudinal fasticulus. They showed that proprioceptive sense was involved with all eye movements and that the brain stem records all eye motion. It is postulated that this proprioceptive feedback plays an important part in control of efferent nerve fibers. They also traced the impulses back to the brain stem and found that the high-threshold receptors sent back signals via cranial nerves III, IV, and VI, while the low-threshold receptors used cranial nerve V. They showed conclusively the existence of an unconscious proprioceptive feedback loop which plays a part in eye movement control. Graphs, which plotted proprioceptive impulse frequency as a function of time, showed that the signal frequency was proportional to the first derivative of the input signals to the muscles. Also found was a continuous signal which was related to stretch. In 1955, Wolter found six different types of sensory endings in the extra-ocular muscles (68). Thus, while the "inflow" theory of Sherrington seems to be disputed by more recent work and the current feelings favor Helmholtz's "outflow" theory, the presence of the anatomical apparatus and the physiological signals for proprioception increase speculation on the purpose of proprioception in the extra-ocular muscles. In 1952, Ludvigh presented a possible system which would account for present observations (55). As can be seen in Fig. 1.8, the proprioceptive feedback takes place at a level which is unperceived in agreement with the accepted "outflow" theory's assertion that there is no conscious position sense in the extra-ocular system. This feedback does exist however, and does exert some effect on the innervating signals to the extra-ocular muscles.

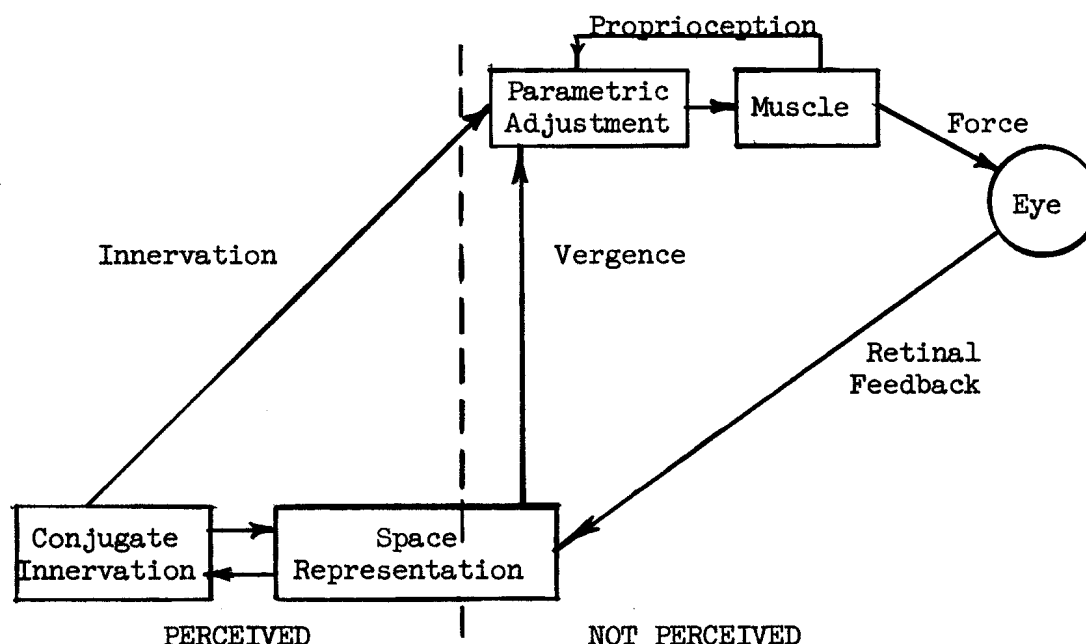


Fig. 1.8 Ludvigh's system (55)

At present, it is generally agreed that the extra-ocular muscles have a well-developed proprioceptive stretch reflex system with muscle spindles and afferent nerves running in the III, IV, and V cranial nerves. The fibers go to the MN-V and from there signals have been traced to the cerebellum and thalamus (69). The arguments concerning conscious proprioception are, in a sense, irrelevant to this type of proprioceptive feedback where the highest level reached is the thalamus. Further discussion of this topic can be found in Christman's paper and in Whitteridge's chapter in the Handbook of Physiology (70,44).

Fig. 1.9 shows, in a schematic way, the main neural pathways involved in horizontal tracking.

#### 1.4 GENETICS (71,72)

Congenital nystagmus is a condition with well-established hereditary characteristics but unknown cause. It probably arises from central disorders in the vestibulo-oculomotor apparatus (71). The actual

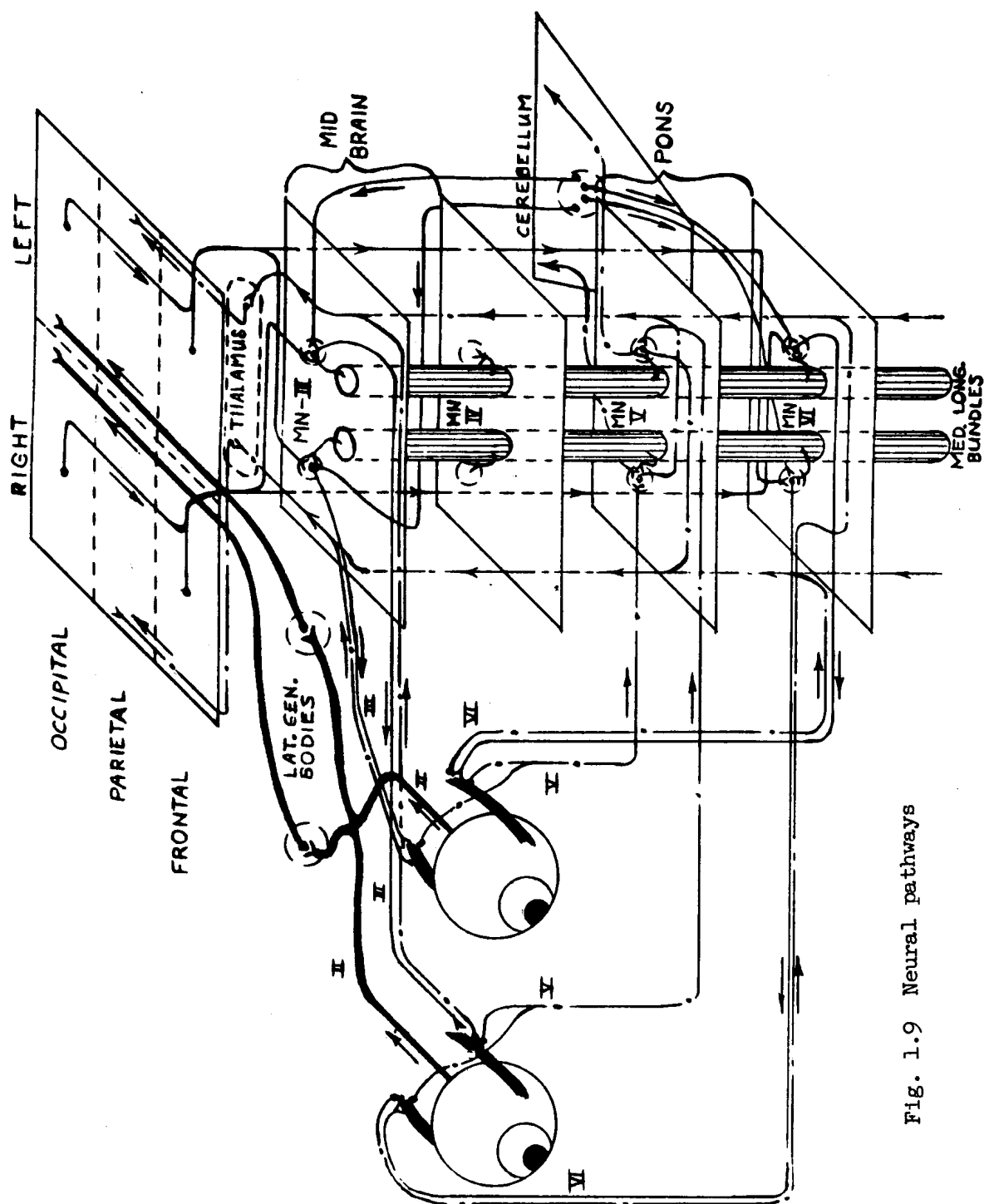


Fig. 1.9 Neural pathways

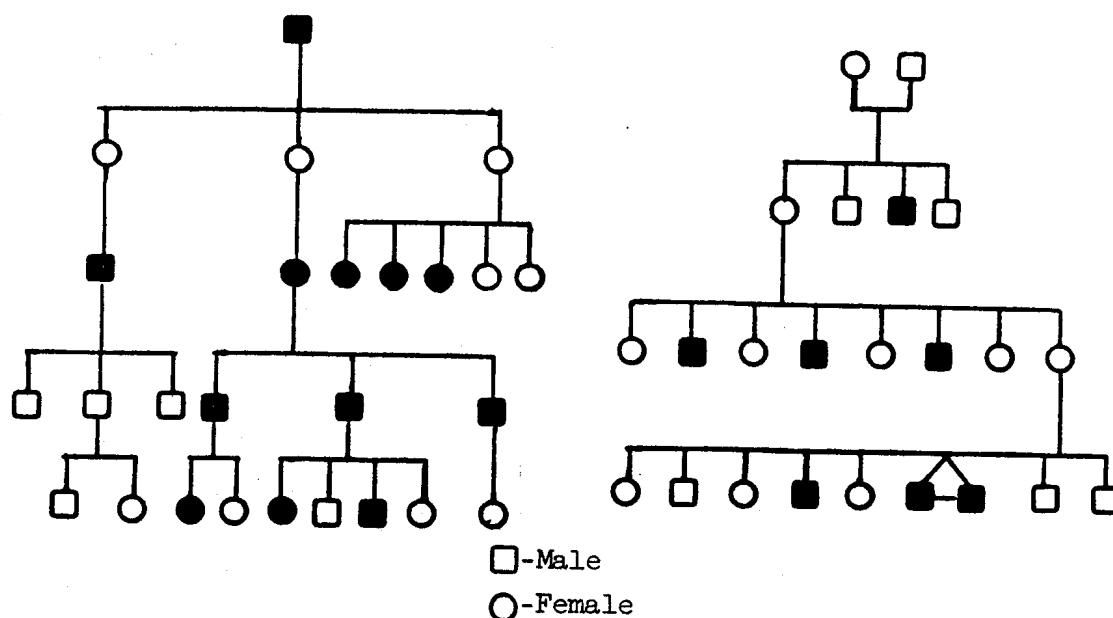
manifestation of the disease varies considerably from one case to another. It could be an imperceptible eye tremor or a large, rapid oscillation which makes vision extremely difficult. It may be unilateral, although in most cases it's bilateral. Amblyopia may or may not occur simultaneously and nystagmus may be the result of other diseases or the subject may be completely normal except for the nystagmus. However, the hereditary element is strong and there are several types of transmission.

(1) Ambi-sexual, irregularly dominant: This affects both sexes with a predominance in the male. In about one half of the cases, head movements occur while pigmentary defects occur only in rare cases.

(2) Recessive, sex-linked, male-limited and gynephoric: In this, a smaller group, head movements are less common but other defects are more common. These include color-blindness, pigmentary failure or optic atrophy.

(3) Simple recessive: This very rare form occurs in one generation, frequently where there is consanguinity in the parentage. It may be unilateral, and the subject may have normal color vision, ocular pigmentation and a normal fovea. This suggests a non-ocular, central origin. Head oscillations may also be present. Fig. 1.10 shows some histories of types (1) and (2).

The genetic history of the subject tested is shown in Fig. 1.11. Since only one generation has been afflicted with nystagmus as of the date of this paper, it is difficult to properly identify the type of genetic transmission involved. It has only appeared in male offspring of three sisters and therefore, it may be the recessive, sex-linked,



(1) Ambisexual dominant (2) Sex-limited  
 Fig. 1.10 Genetic histories of nystagmus

male-limited and gynephoric type or, it may even be the simple recessive type (note the presence of a consanguineous marriage in the parentage).

This is a sex-linked transmission. The most probable offspring to be affected would be the male offspring of the subject's sister. To date, she has had two male offspring and both are unaffected. It will probably take one more generation to accurately classify the type of transmission involved.

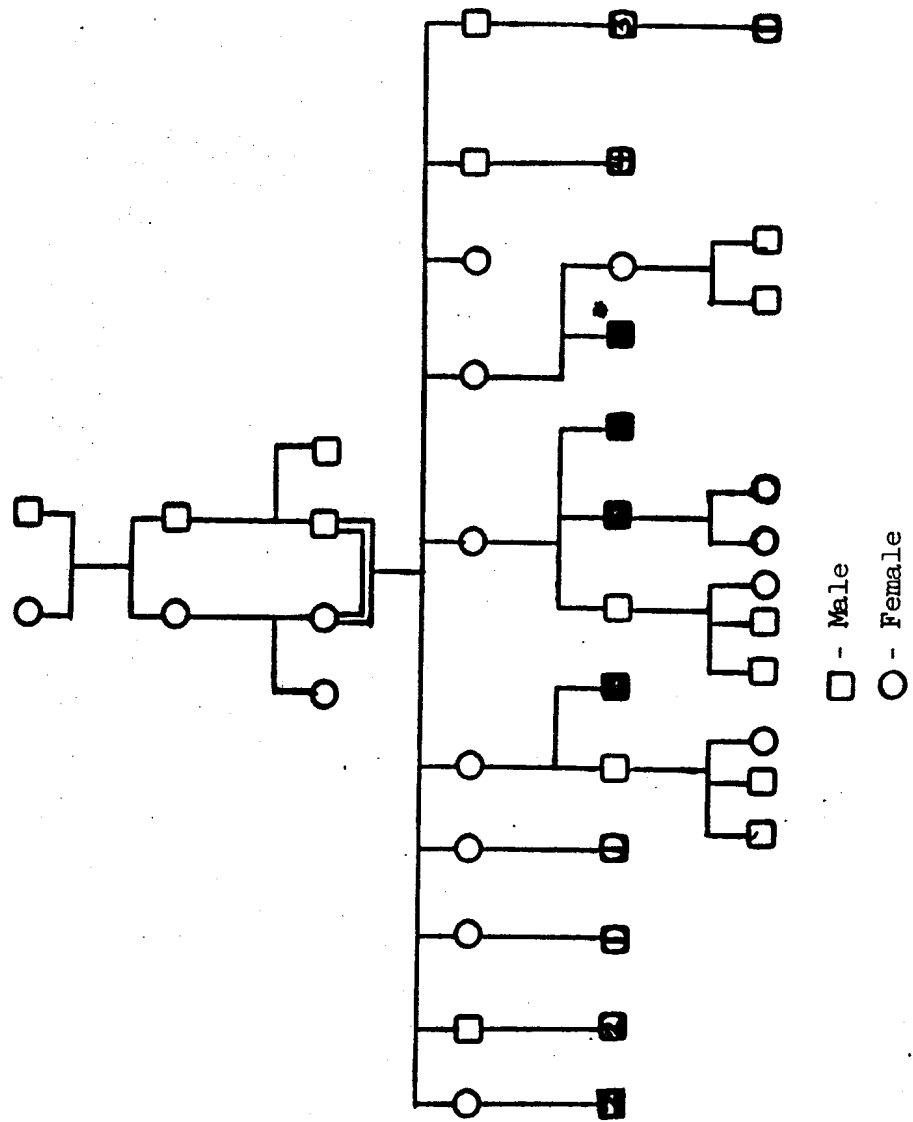


Fig. 1.11 Subject's genetic history



## CHAPTER II

### OCULOGRAPHY AND PHYSIOLOGICAL DATA

#### 2.1 METHODS OF OCULOGRAPHY

Many methods have been developed and used to measure eye position and velocity. The work done in this area will be briefly discussed and those interested in a more detailed history of this area are referred to the works of Young, Carmichael and Dearborn, and Shackel (12,74,75,76). Historically, eye movement monitoring has developed as follows:

(a) The first method, which is still in use clinically, is that of direct observation of the subject's eyes by the investigator. This is usually sufficient to detect any gross abnormalities of the eye tracking system. This method is a subjective one and one which yields no permanent quantitative record of eye movement.

(b) The early work in this field was done using after-images produced by bright light (77,78). A regularly flashing light leaves a series of after-images on the retina and the subject reports their number and spacing. This requires an experienced subject and was a very subjective method. Again, no permanent record resulted.

(c) To satisfy the need for permanent records, several mechanical methods were devised. One typical mechanism consisted of a rod attached to the eye using a plaster of Paris ring. This rod moved levers which recorded motion on the smoked paper of a rotating kymograph (79). Later work used pressure transducers and membranes placed against the

closed eyelid of one eye as the other eye viewed the target. The corneal bulge activated these transducers as the eyes moved (80). Mechanical methods succeeded in providing permanent records. However, they did interfere with normal eye motion and thus caused changes in eye response.

(d) Photography eliminated the mechanical interference associated with mechanical transducers and provided permanent records of eye motion (81). From total eye photography emerged methods employing the photographing of tiny bright objects attached to the cornea. Those used were a small flake of chinese white and small, bright silver beads (15,82,6). These methods consumed large quantities of film and required very rigid head and camera mounting.

(e) An offshoot of direct photography is the corneal reflection method. This technique is based upon the fact that the cornea is actually a bulge on an otherwise spherical shape. Thus it will reflect light, focused on a spot on the cornea, at an angle from its normal at that spot equal to the angle from the normal that the incident light falls. As the eye moves, the normal to the cornea changes position and so the reflected light beam moves also. This light beam can be focused on photographic film for a record. The basic techniques were constantly refined as the need for greater accuracy arose and to suit special conditions (83,84,85,86). Unfortunately, the use of film for a record prevented real-time monitoring of data. This was changed by a method proposed by Young and which was used in this study (12). Corneal reflection also requires rigid head clamping to prevent errors due to lateral motion.

(f) To increase sensitivity of the corneal reflection method a contact lens with a tiny mirror mounted on it was used (87). This method can record eye movements of less than  $10''$  arc. Lamps have also been used in place of mirrors (88). Here, the light is passed through two knife edges and falls on the surface of a photomultiplier whose output voltage is an indication of eye position. These methods involve changing the dynamics of the eye and the results are subject to errors due to slippage of the contact lens.

(g) Work in 1922 showed that eye movement had associated electrical changes (89). It was recently however, 1936, that the electronics were developed to measure and record these changes (90). Electrodes were placed on the skin around the eyes and a potential measured between them. This potential is the result of the potential difference between the retina and cornea which sets up an electrostatic field which rotates with the eye. Due to the higher metabolic rate of the retina it is about 1 mv negative with respect to the cornea. EEG electrodes have been used to record these potentials and since they are fixed to the head, no artifacts due to head motion are present (91,92, 13). The disadvantages associated with this method have been non-linearity of voltage in relation to eye movement and a low signal to noise ratio due to muscle action potentials. Shielding problems have been overcome by balanced input and AC amplifiers (93,94).

(h) Another technique which is in use requires electronic photosensitive devices which detect the position of the limbus (the boundary between the white sclera and dark iris). This information is then sent, in the form of a voltage signal, to a recorder where a record of eye

position results. Several different techniques using this basic idea have been developed and are discussed by Young (95,9,96,97).

## 2.2 METHOD USED

The method used is a variation of the corneal reflection technique which was developed by Richter and used by Young in his work (98). It consists of placing two small lamps in a position which enables them to illuminate circular patches on either side of the pupil of the eye (see Fig. 2.1). At the side of each lamp is a photosensitive device which responds to the reflected light from the eye. The voltage outputs from these photosensors are a function of the angle of gaze and by taking the difference between them a voltage results which is related to the angle of gaze and which is minimally affected by non-linearities or ambient changes. The lamps used were Number 224 which were adjusted to illuminate a spot approximately 1.5 cm in diameter. This spot was mainly on the sclera with a little overlap onto the cornea when the eyes were in the straight ahead position. The photo-sensitive devices were RCA type 1P42 phototubes. Their small size and head-on cathode made them ideal for this application. One other advantage was their fast dynamic response. Young used photo resistors which had a much slower response and he recommended the use of phototubes for studies of saccadic jumps and nystagmus. The bandwidth of the 1P42 is 1 kHz compared to 10 Hz for the photoresistors. The system output is linear to  $\pm 15^\circ$  of horizontal eye movement and the resolution is approximately  $1/4^\circ$ . The lamps were powered by a constant current power supply adjusted to supply 210 ma (Hewlett-Packard model 721A). The phototubes formed two arms of a bridge circuit whose other arms were

5 megohm resistors. The bridge output was fed to a differential pre-amplifier (Argonaut model LRA 042) whose output was recorded on one channel of a Sanborn dual channel, hot-stylus recorder. The target motion was recorded on the other channel to provide real-time correspondence. The lamps, phototubes and bridge resistors were all mounted on a clear piece of plexiglass through which the subject viewed the target. The subject's head was stabilized by means of a bite-bar and a forehead rest which were rigidly clamped down (see Fig. 2.2). A more detailed description of the construction of the apparatus can be found in a paper by Doescher (99).

### 2.3 TARGET DISPLAY

The target consisted of the horizontally moving spot of an oscilloscope. The specific types of motion necessary were generated by a Hewlett-Packard model 202A Low Frequency Function Generator and associated circuitry where needed. The 10 cm screen was positioned 27.1 cm from the eye so that 5 cm on the screen was equivalent to a  $10^\circ$  deflection. Thus, since the radius of the eye is approximately 1.3 cm,

$$\theta = \tan^{-1} \frac{5}{28.4} = 10^\circ \quad (2-1)$$

In both cases, the subjects were instructed to look at the target at all times. No instructions were given regarding anticipation of target motion.

### 2.4 RESPONSE TO PREDICTABLE SINUSOIDS

In an effort to compare the measuring apparatus against other methods, the responses to sinusoidal target motion were recorded for frequencies from 0.1 to 2.5 Hz. The amplitude of the motion was  $20^\circ$

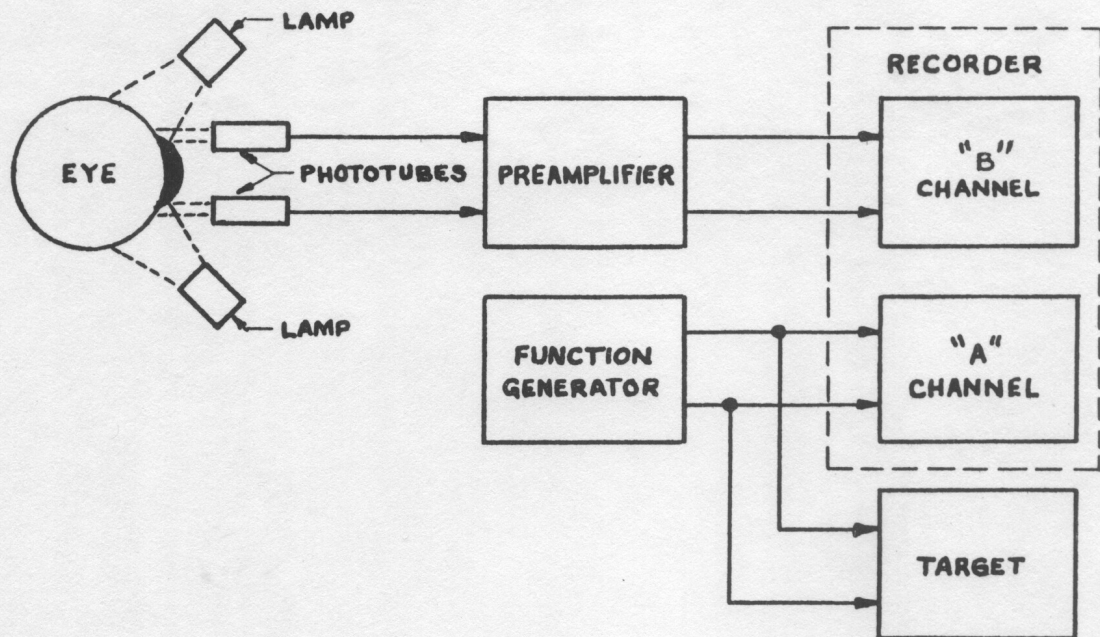


Fig. 2.1 Block diagram of tracking device

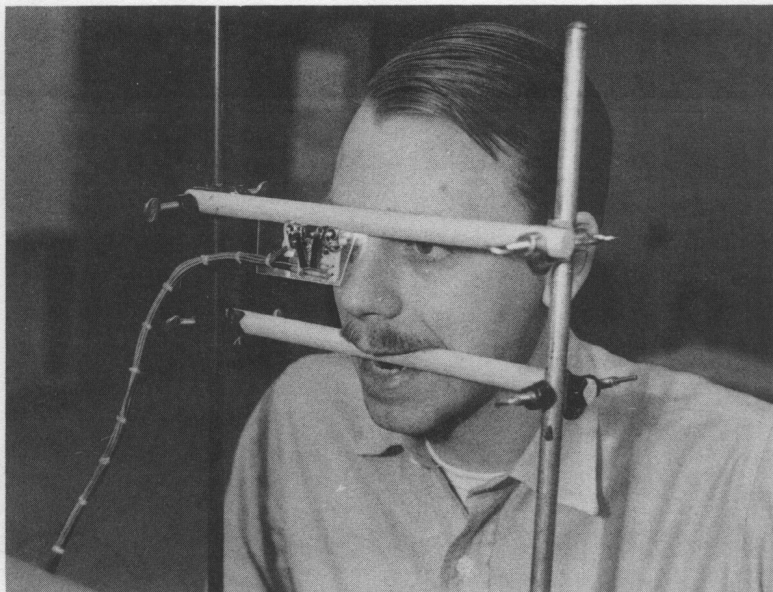


Fig. 2.2 Subject using the eye-tracking device

peak to peak ( $\pm 10^\circ$  from straight ahead). This was done for both subjects to further provide a basis of comparison between the ability of the normal system to follow these predictable target motions and that of the subject with nystagmus. It must be pointed out that the gain and phase plots resulting do not represent the responses to unpredictable target motion which is the topic of this paper and for which a model will be postulated. Fig. 2.3 shows the gain and phase plots for the normal subject tested and the nystagmus inflicted subject. For comparison, the results obtained by Young are included.

## 2.5 TRANSIENT RESPONSES

The following data will illustrate the responses of both subjects to unpredictable target motion.

### 2.5.1 Pulse Response

The pulse in target position was generated using a step change and an R-C differentiating circuit. As can be seen in Fig. 2.4, the response to a short pulse in target position consists of an initial delay followed by a pulse whose width is greater than or equal to the delay. In the case of the subject with nystagmus, we see the 3 Hz sawtooth oscillation present, before, during and after eye movement.

### 2.5.2 Step Response

The step response of the human eye tracking system is shown in Fig. 2.5. Basically it is a delayed step change in eye position. At times some overshoot is noted. Again, a 3 Hz oscillation appears in the response of the subject with nystagmus.

### 2.5.3 Ramp Response

The response to a target that moves with constant velocity at some time  $t = 0$  has been shown by Young to be a delayed ramp of velocity

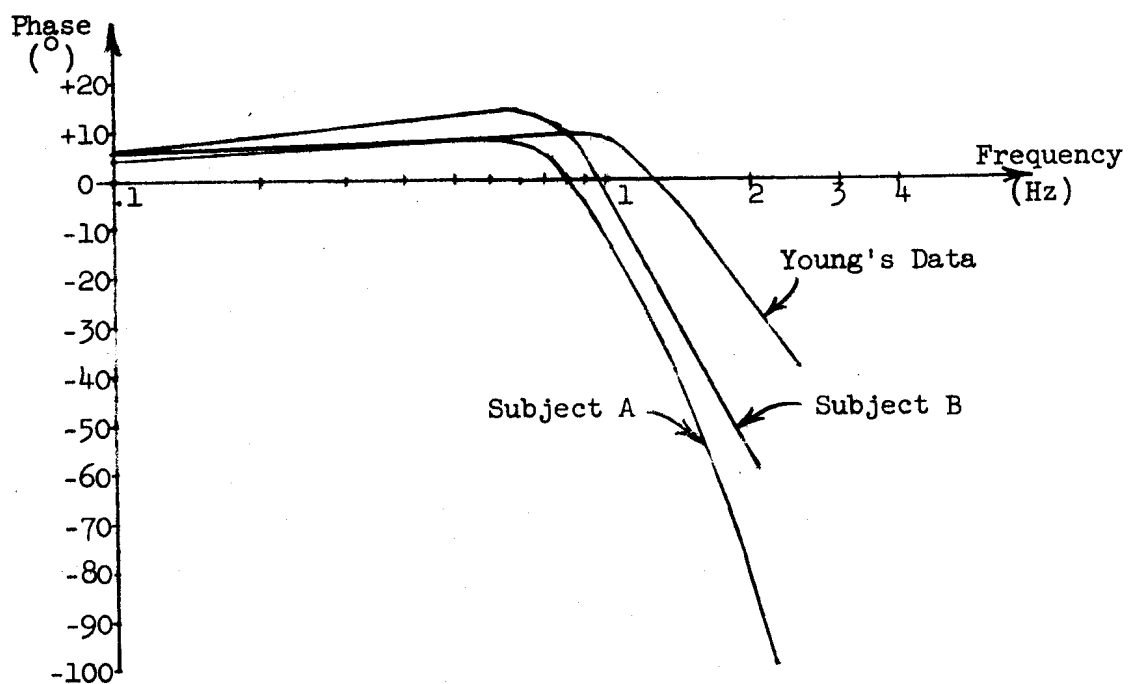
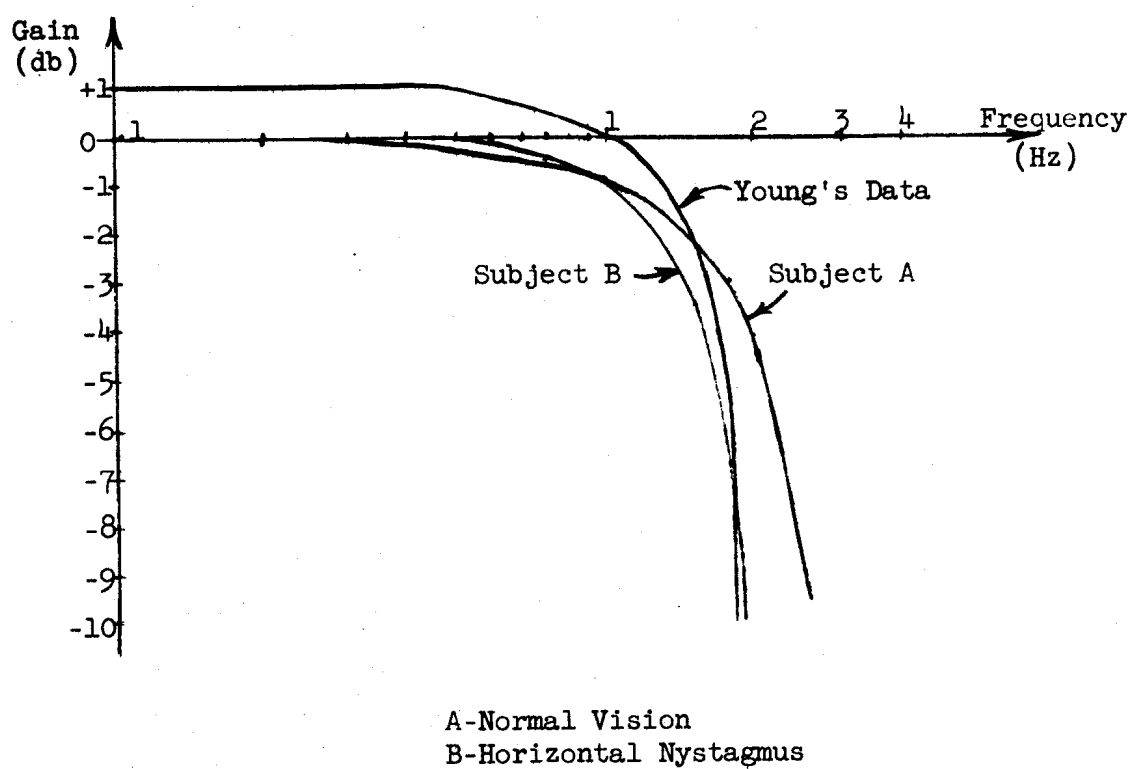


Fig. 2.3 Gain and phase plots for predictable target motion



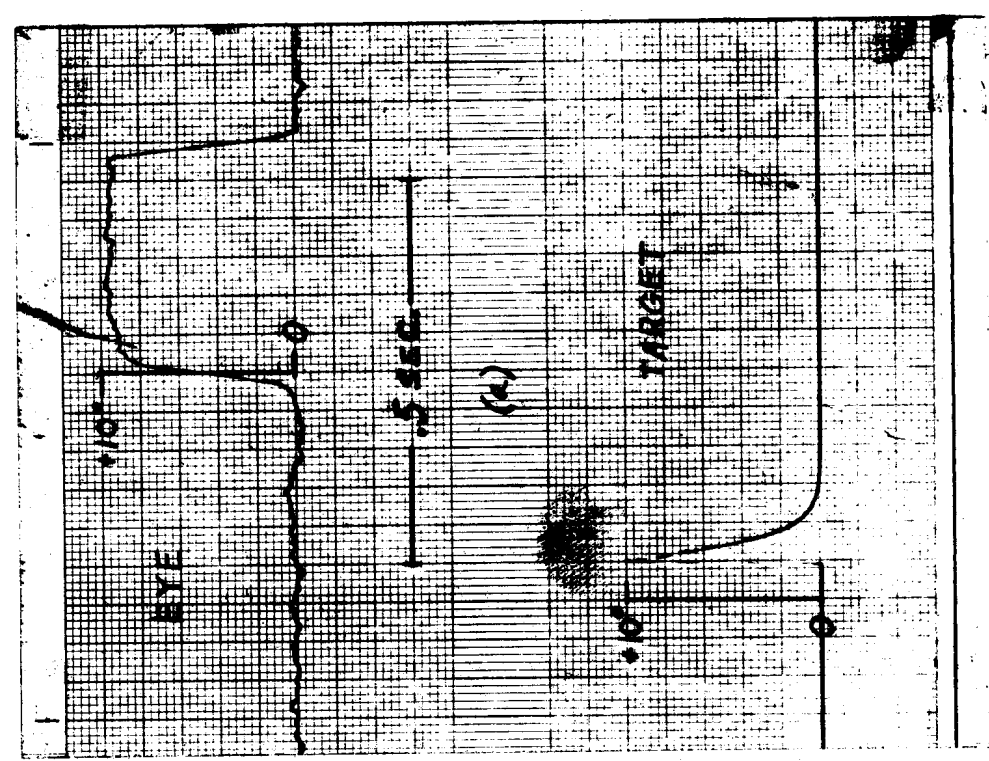
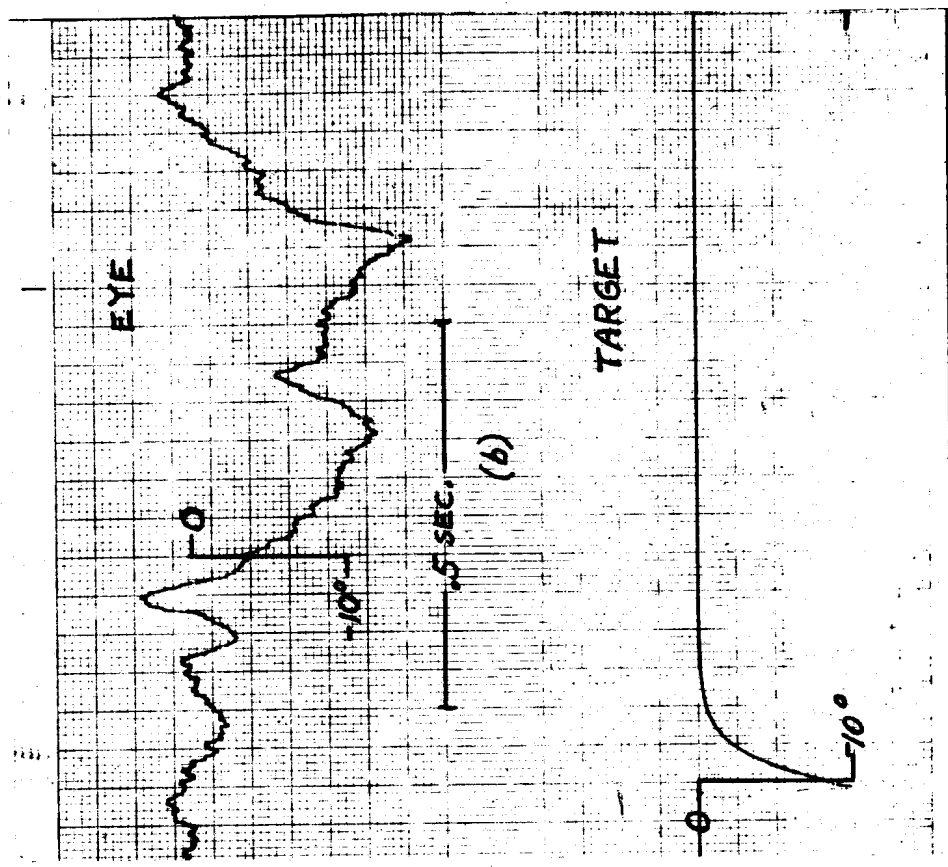
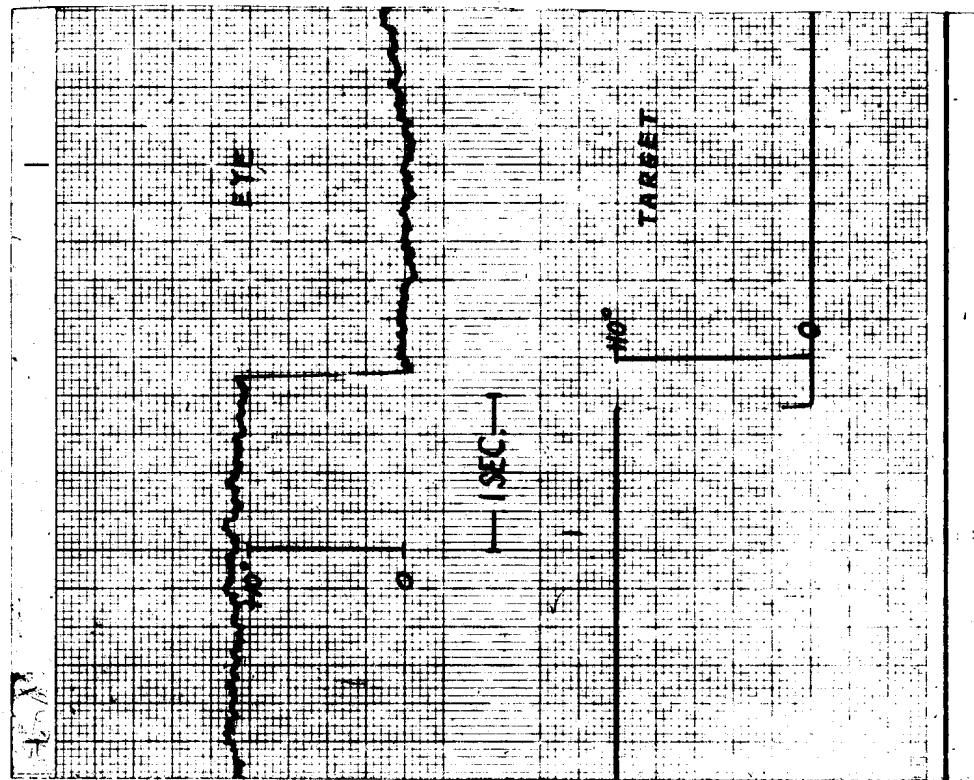
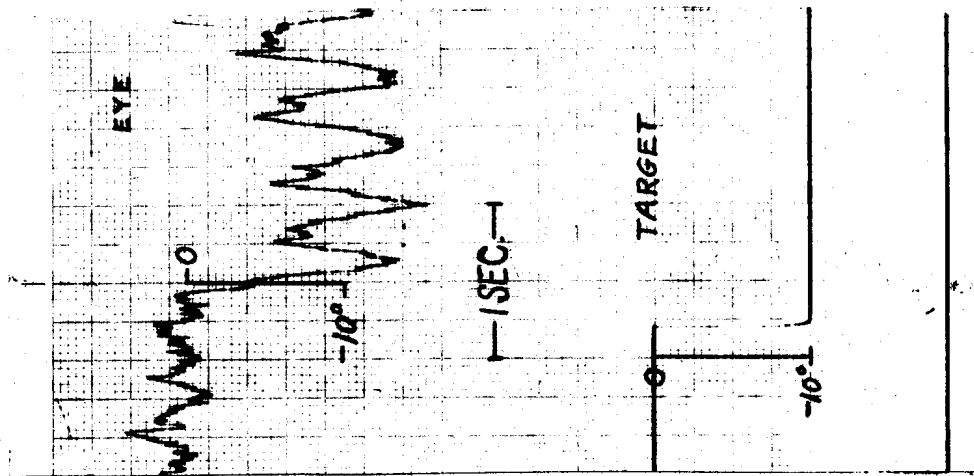


Fig. 2.4 Pulse response (a) normal (b) with nystagmus



(a)



(b)

Fig. 2.5 Step response (a) normal (b) with nystagmus

equal to the target velocity with a step correction occurring approximately one refractory period after eye motion begins (see Fig. 2.6).

#### 2.5.4 Parabolic Response

The response to a constantly accelerating target has been shown by Young to be a series of ramps of increasing magnitude with corrective step changes in position (see Fig. 2.7).

#### 2.5.5 Responses to Various Velocities

To illustrate the difference in responses to slowly moving targets and to those that move more rapidly, both subjects were presented with triangular waveshapes of various frequencies from 0.1 to 3 Hz which corresponded to velocities of from  $2^\circ/\text{sec}$  to  $60^\circ/\text{sec}$  respectively. Fig. 2.8 shows the normal response to  $4^\circ/\text{sec}$  and the response with nystagmus to  $2^\circ/\text{sec}$ . Fig. 2.9 shows both responses to  $10^\circ/\text{sec}$ .

#### 2.5.6 Step-Ramp Response

This target motion was reported on by Young to demonstrate the independence of the saccadic and smooth tracking mechanisms of the eye. It is essentially the superposition of the step response and the ramp response. Fig. 2.10 is a drawing made from Young's data.

### 2.6 DISCUSSION OF DATA

It should be noted at this point that the directions of gaze to the left and right of straight-ahead were given the arbitrary signs of + and - respectively with straight-ahead denoted as an angle of  $0^\circ$ . This applies to the model as well as to the physiological responses. Comparison of the gain and phase plots of the normal subject with those of Young provides the assurance that the measuring apparatus is functioning well. The only difference in the gain curves is at the low

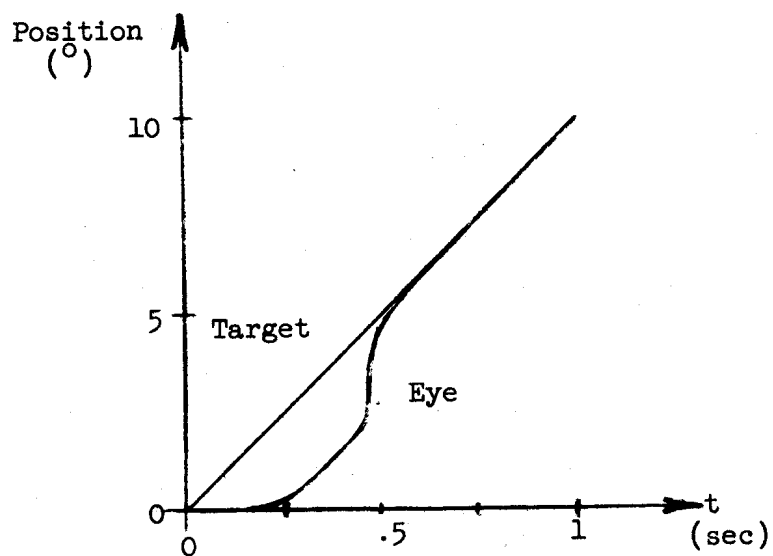


Fig. 2.6 Sketch of ramp response (from Young)

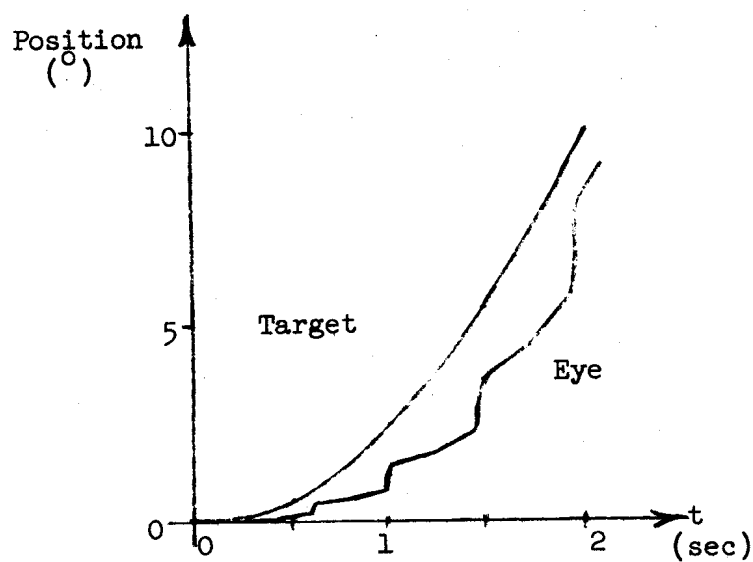


Fig. 2.7 Sketch of parabolic response (from Young),

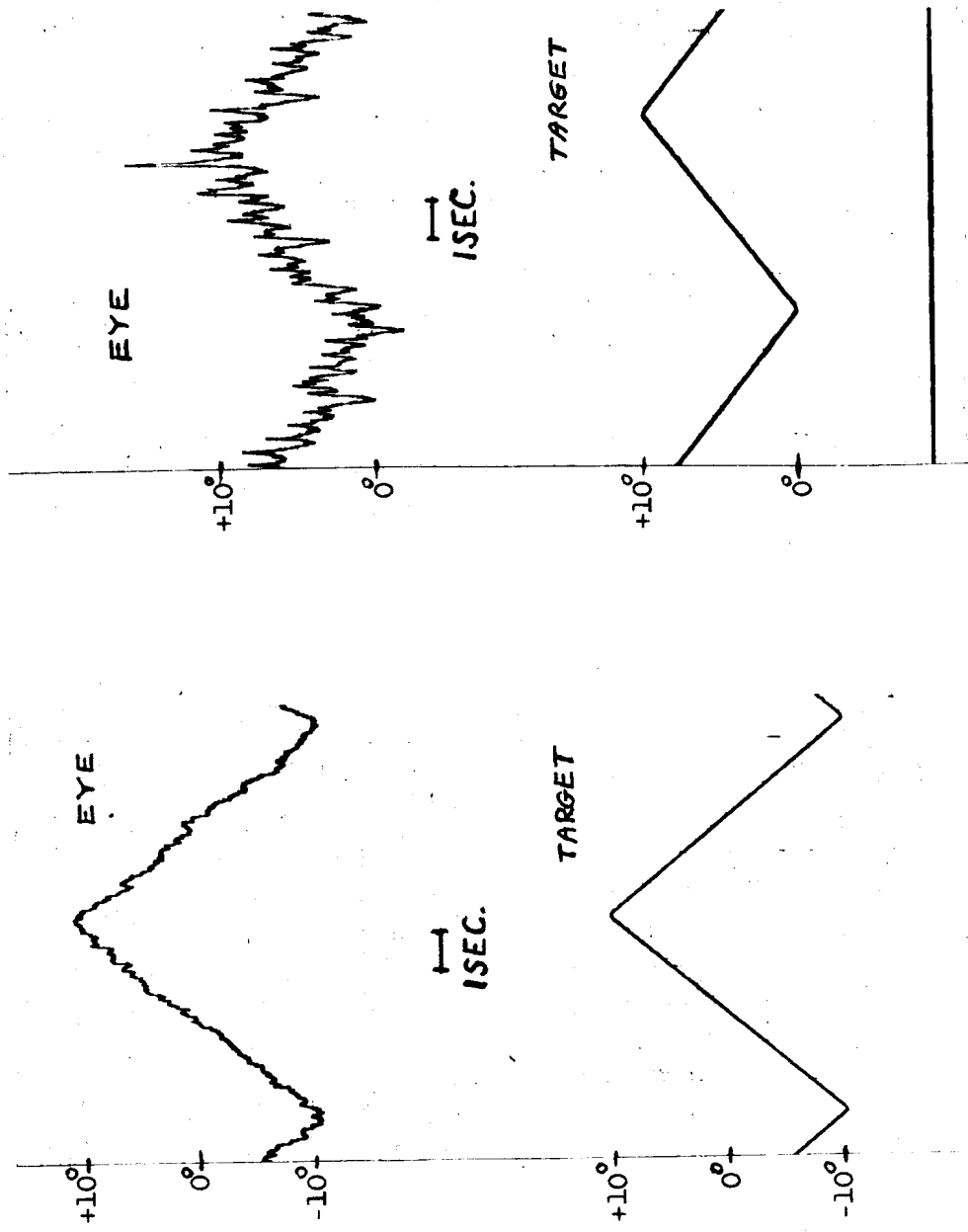


Fig. 2.8 Low velocity tracking (a) normal ( $4^{\circ}/\text{sec}$ ) (b) with nystagmus ( $2^{\circ}/\text{sec}$ )

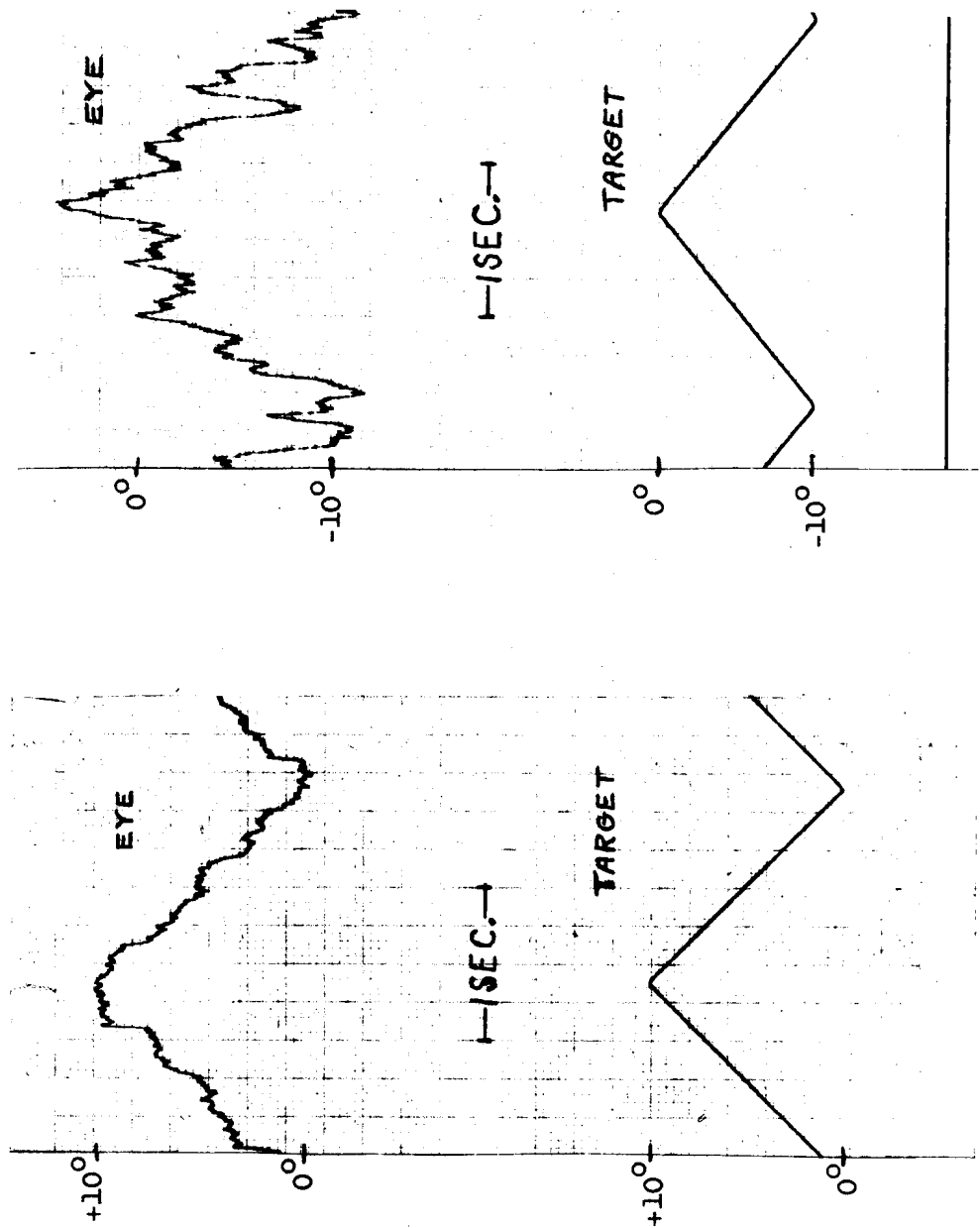


Fig. 2.9 Tracking a target moving at  $10^\circ/\text{sec}$  (a) normal (b) with nystagmus

frequencies where Young reported a gain greater than one and the results of this investigation show a gain of zero. This is midway between Young's data and the value of  $-3$  presented by Fender and Nye (100). In any case, all three sets of data show the ease of tracking low-frequency predictable sinusoids. The phase plots for the normal subject and from Young's data both show a phase lead at low-frequencies which reaches a peak of approximately  $10^{\circ}$  at about 0.8 Hz and then drops sharply. This is the result of a predictive mechanism which is causing the eyes to actually lead the target. It works on past information and expected repetition. The response to unpredictable target motion obviously cannot make use of this mechanism and the gain and phase plots obtained by Young using such target motion reflect this. Fig. 2.3 also shows that the responses of the subject with nystagmus are quite indistinguishable from normal responses. This fact supports the author's hypothesis that both systems are equivalent in response with the only difference being the 3 Hz sawtooth oscillation on the nystagmus response.

The response to a pulse change in eye position illustrates the lack of control present in the system once a decision has been made. The eye sees the target returning to its initial position long before the eye has moved and yet the eye does move to where the target was but is no longer. It can return only when one refractory period has elapsed. The response of the subject with nystagmus shows how the amplitude of this 3 Hz sawtooth varies with position. At the  $0^{\circ}$  position it is smaller than at the  $-10^{\circ}$  ( $10^{\circ}$  to the right) position. For both subjects it was found that the delay varied from 150 to 250 msec

and the pulse width, which was slightly larger, varied from 200 to 300 msec. The term "delay" is taken to mean the time from the start of target motion to that point when the eye has reached 50% of its final value and the term "pulse width" is that period of time between the 50% point on the leading edge of the pulse and the 50% point on the trailing edge. The rise-time varied from 15 to 25 msec. Here, "rise-time" is that period of time in which the eye goes from 10% to 90% of its final value. These figures are for target pulses whose duration is substantially less than one refractory period (about 200 msec). The response to long pulses is merely the superposition of the responses to two step changes in target position.

The response to a step change in target position is a step change in eye position which is delayed approximately 150-250 msec. The movement of the eye appears to be similar to a critically damped second-order system. The rise-time exhibited by the eye was 15-25 msec as would be expected based on the data given for a pulse response. The nystagmus response shows clearly the variation of the amplitude of the oscillation with eye position. The general shape of the sawtooth is also apparent from this trace. The so called "quick phase" (the direction in which the eye moves fastest) is to the left in both gaze positions.

The eye tracks a target that moves with a constant velocity, which starts at some unpredictable point in time, by assuming that velocity after a delay of 150-250 msec. This is followed by a saccadic change in eye position, which occurs approximately one refractory period after the onset of eye motion, to correct for the position error caused by the delay in the onset of eye motion.



A parabolic input, which is equivalent to a constantly accelerating target, produces a response which is a series of ramps (constant velocity segments) with corrective saccades interspaced among them. This type of response will always lag the target since it results from velocity commands which were made one refractory period before. These velocity segments change in magnitude approximately every 200 msec.

As the responses of the subject with nystagmus to constant velocity targets show, the presence of the oscillation masks the corrective saccades. They are clearly visible in the normal response to a target moving at  $10^{\circ}/\text{sec}$ . However, a comparison of the shape of the nystagmus response to this input with the response to a  $2^{\circ}/\text{sec}$  velocity, where no saccades are present, brings out the fact that they are present in the former response. Again, the quick phase is to the left and amplitude increases as the gaze deviates from the straight-ahead position.

If a target has a step change in position in one direction and a constant velocity in the other, at some point in time (a step-ramp) the eye will, after a delay of 150-250 msec, exhibit a typical step response superimposed on a typical ramp response. Thus, there is no interaction of the two. This establishes the fact that the two systems, saccadic and pursuit, are functionally independent. The saccadic mechanism responds to target position and the pursuit mechanism responds to target velocity.

Since it was found that the amplitude of the nystagmus was a function of eye position, data were taken with the subject focusing on

stationary targets placed at points from +12 to -12 degrees. Fig. 2.11 illustrates the peak to peak magnitude of the oscillation as a function of eye position. Measurement of the slopes involved in the nystagmus showed the motion to the left to be approximately twice as fast as the motion to the right. Thus, assuming that the nystagmus is an asymmetrical triangular wave, the slope of motion to the left would be twice that to the right. The period was found to vary between 250 and 400 msec depending upon factors such as fatigue, eye irritation, anxiety, etc. During the tests run, the frequency most prevalent was approximately 3 Hz. No correlation between frequency and eye position was apparent. The actual wave shape consisted of a sharp movement to the left followed (or preceded) by a slower movement to the right. This movement to the right usually had a notch somewhere in it. The size and/or position of this notch appeared to have some dependence on eye position but the experiments conducted were not designed to study such small variations in response but rather the gross movements associated with the tracking of well-defined target motions. Thus, in this paper the nystagmus will be approximated by an asymmetrical triangular wave with no notch.

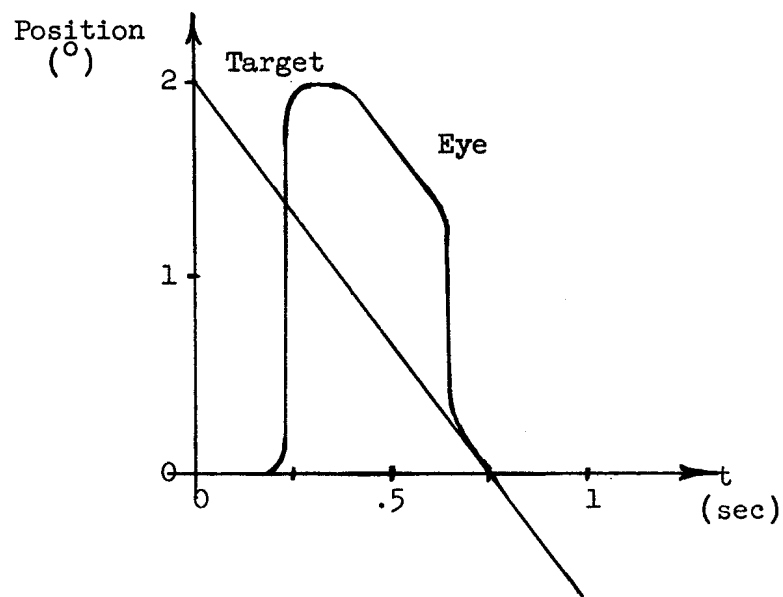


Fig. 2.10 Sketch of step-ramp response (from Young)

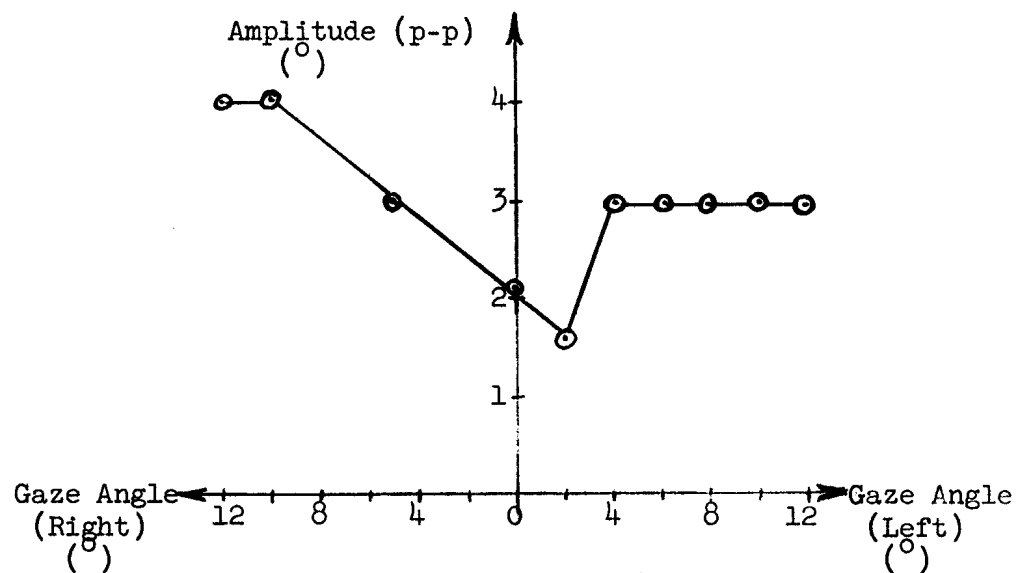


Fig. 2.11 Positional dependence of nystagmus amplitude

## CHAPTER III

### MODELING THE HORIZONTAL TRACKING SYSTEM

#### 3.1 INTRODUCTION TO MODELING

Obtaining a mathematical model for a dynamic system is an art which combines creativity and application of the scientific method. The model is an abstract entity which provides a representation of a physical phenomenon. One of the more important results of the model is the quantifying of the physiological mechanism by providing the numerical values of the parameters as well as the flow paths involved in the system under study. Obtaining the model usually involves hypothesizing, experimentation and re-hypothesizing until the model developed satisfactorily represents the mechanism within some predetermined tolerances. The only departure from the classic method of science is the extensive use of the computer in hypothesizing. This is due to the complexities involved in describing physiological systems which are highly non-linear. While the model, in its simplest sense, only represents the physiological system on an input-output basis, if it consistently conforms to experimental observations of the biological system under various dynamic situations, it is not too presumptuous to suppose that the internal mechanisms of the model may represent a close approximation to those actually involved in the physiological system. This might lead the biologist to search for the anatomical counterparts of the various functional boxes in the model.

There are two basic approaches to modeling. The first is the writing of equations for known physical processes based on a knowledge of the physical laws governing these processes. An example of this is the construction of a model for the motion of a mass on a spring subject to an initial displacement from the equilibrium position. While this is a very simple example, it should not imply that this first approach is an easy one. Many systems whose physical laws are well known still require a great deal of knowledge and creativity to simplify the configurations so that a model may be postulated. The second approach is the one usually used in modeling the biological system. This is the empirical method which relies on carefully controlled experiments which illustrate cause and effect relationships while excluding non-causal artifacts. Due to our lack of understanding of the details of the biological mechanisms their modeling must necessarily be done by this empirical method. This puts a tremendous burden on the instrumentation necessary to measure the biological responses to various inputs since most biological systems have a very low signal to noise ratio.

### 3.2 PROPOSED MODELS

A review of the literature reveals several models which have been proposed for the horizontal tracking system. These models reflect the different approaches taken by the investigators involved and a comparison of one with another will serve to point out the relative merits and shortcomings of each.

#### 3.2.1 Saccadic and Smooth Tracking Models

(1) Vossius (7): By assuming that the saccadic mechanism is a proprioceptively controlled movement instead of a ballistic movement,

Vossius proposed the model shown in Fig. 3.1 for the saccadic mechanism. There are two delays in this system. The first is a brain delay of  $T_1 = 130$  msec and the second a transport delay of  $T_0 = 5$  to 10 msec. A constant represents the transfer function of the eye muscles and the eyeball dynamics consist of its inertia and friction with a time constant  $t_1 = 15$  msec. The presence of a proprioceptive position feedback and rate feedback from eye position is postulated to explain the existence of various classes of the time course of saccadic movements reported by Vossius.

(2) Fender and Nye (100): The model presented by Fender and Nye is the result of the application of linear systems analysis to the gain versus frequency response curve measured by means of a carefully constructed optical system. It is a highly schematic model which attempts to describe the major flow paths in the system. As can be seen in Fig. 3.2, three feedback paths are postulated. Evidence for the existence of these loops is presented as well as arguments to support the regenerative nature of the oculomotor loop. By comparison of observed phase lag and computed phase lag (from Bode equation) the authors show that the system is non-minimum phase. This fact is coupled with the knowledge that opening the main negative positional feedback loop leaves a system with a gain in excess of unity. The only systems which are non-minimum phase and have an overall gain in excess of unity are those with a regenerative loop. A further analysis of stability criteria identifies the regenerative loop as the oculomotor feedback loop. The role of the first derivative feedback is postulated to be that of damping unstable oscillations. The value

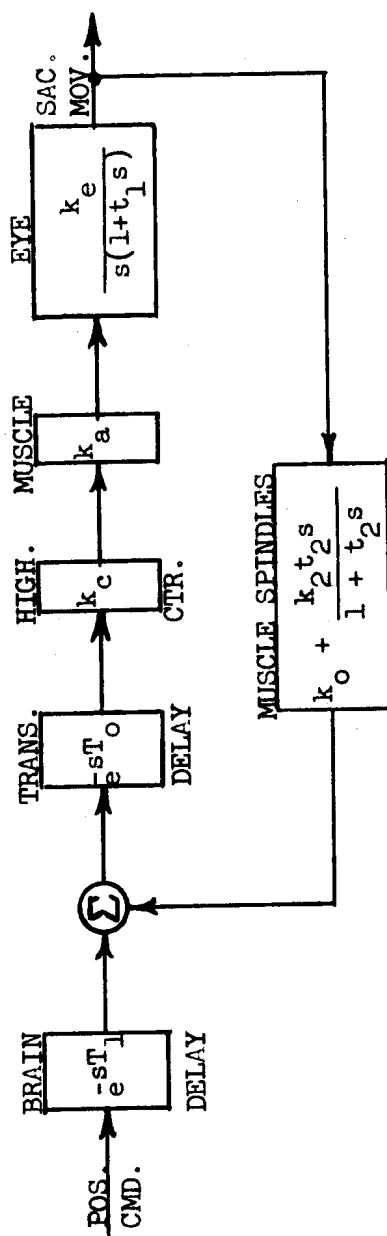


Fig. 3.1 Vossius' model for saccadic movements

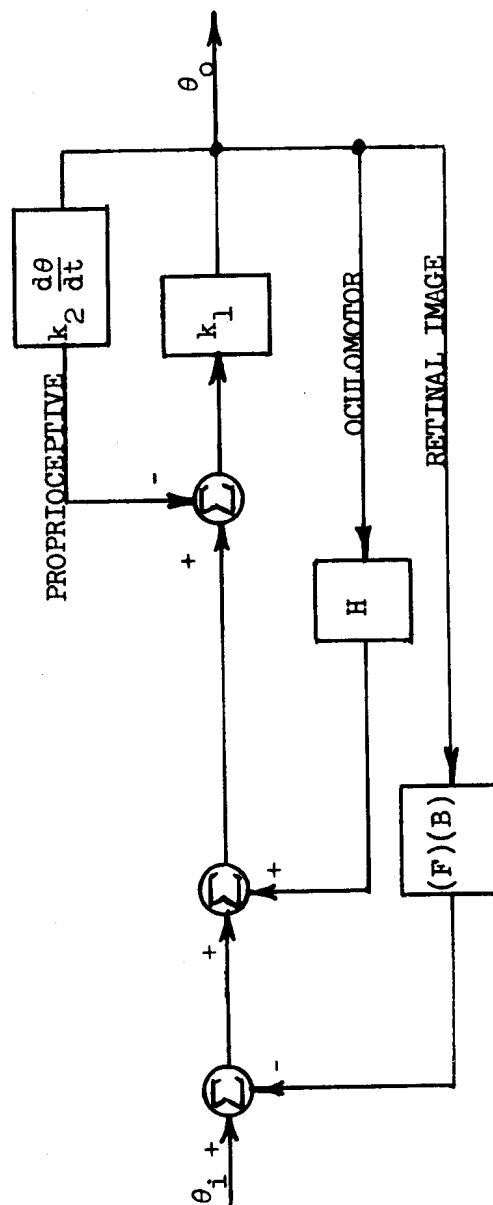


Fig. 3.2 Schematic eye movement control system from Fender and Nye

of  $k_2$  is assumed to vary from small (during saccades) to large (during fixation). A further role postulated for this derivative feedback is that of suppressing visual information during a saccade. The merits of this model in representing the instability of the oculomotor system which causes flicks, drift and tremor are also discussed.

(3) Young (12): A model for the tracking of unpredictable target motion was postulated by Young in 1962. This model was different from any that preceded it because of the assumption that the system is a sampled-data control system instead of a continuous system. Also present are non-linear elements to account for observed physiological data. As can be seen in Fig. 3.3, there are two main forward paths. The upper path is the mechanism for smooth-tracking of moving targets. The velocity of the error is first estimated by taking the difference between the present error and that of the previous sample. This signal then passes into a limiter where it is passed on as received if it falls below a certain rate (25 to 30°/sec) and rejected completely if it is above this value. It has been experimentally determined that the eye will not track smoothly a target moving at a velocity greater than 25 to 30°/sec but will approximate it with saccadic steps. After the first limiter, the signal is integrated to obtain the desired velocity, passed through a saturation non-linearity which insures that the eyes do not move smoothly with a velocity greater than 25 to 30°/sec, and integrated once more to provide a continuous command from the pulsed data. The lower path is the saccadic positioning mode and it consists of a dead-zone of 0.2 to 0.5 degrees followed by a computing delay and an integration to provide the desired position command. It should be



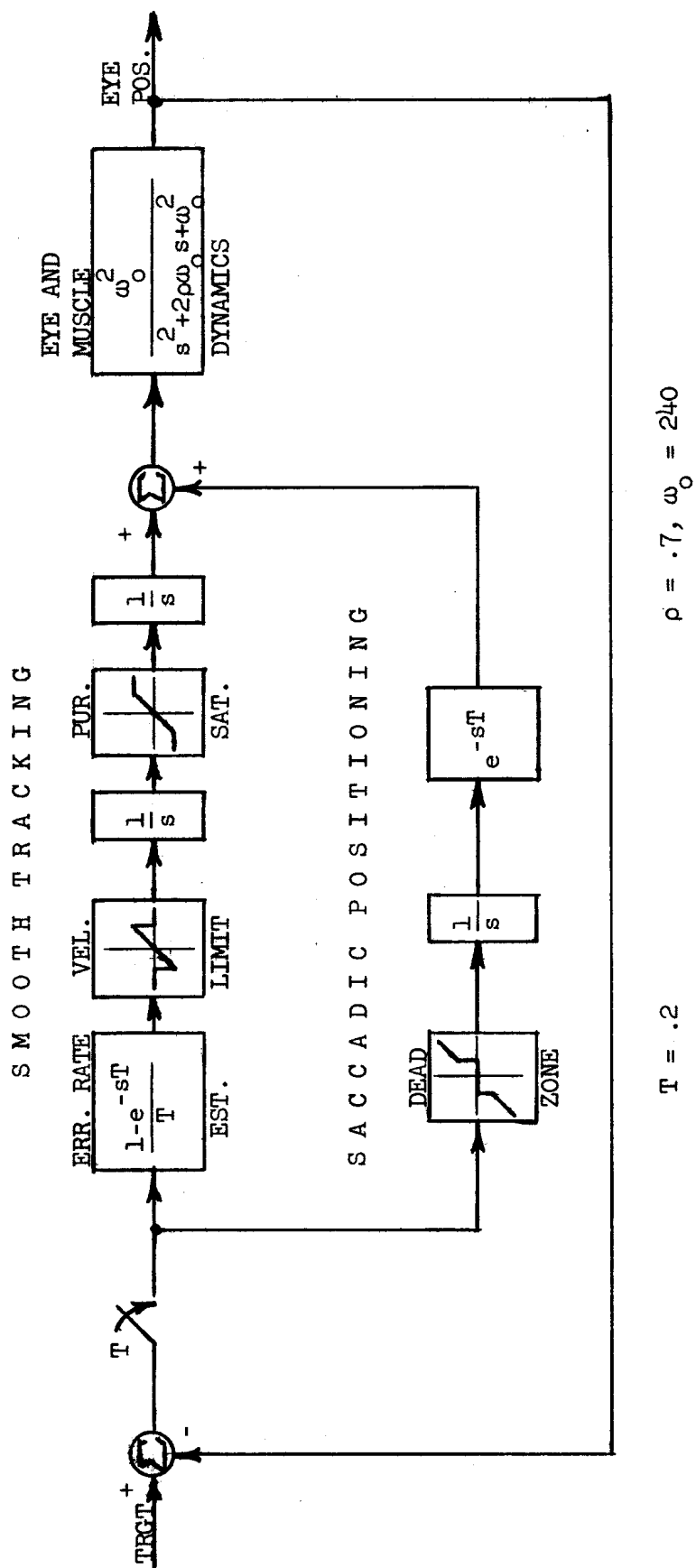


Fig. 3.3 Young's sampled-data model of the eye tracking system

noted that the smooth-tracking path is actually a velocity-segment approximation mechanism whose output is a series of ramps of different slopes (if necessary) every sampling period. The eyeball dynamics are taken from Westheimer's second-order approximation which has a damping constant of 0.7 and a natural frequency of 240 radians/sec (101).

(4) Pavlidis (102): This model is a modification of Young's model which is supposed to correct a discrepancy between the observed saccadic response of the eye and the output which Young's smooth-tracking system would give to a small step-change in target position. Fig. 3.4 shows that Pavlidis' model coincides with Young's in both the saccadic path and the eyeball dynamics. In the smooth-tracking path the error-rate estimating element is replaced by a differentiator which is followed by a sampler, a zero-order hold, a limiter and an integrator. This necessitates the use of two samplers which is in better agreement with experimental results as shown by Rashbass (9).

(5) Warnmeunde (103): The model shown in Fig. 3.5 was presented at the 17th Annual Conference of Engineering in Medicine and Biology. There are several differences between this model and the preceding ones. The smooth-tracking path is continuous while the saccadic path is sampled. The eyeball dynamics are based on work by Miller and consist of a third-order polynomial with positive feedback (104). The smooth-tracking path is not limited to straight line (velocity) segments and the method of sampling in the saccadic path is quite unique in that the dead-zone is present in the controller for the sampler.

### 3.2.2 Comparative Evaluations

The models presented above represent the thoughts of several investigators, each of whom used the work of those who preceded him and

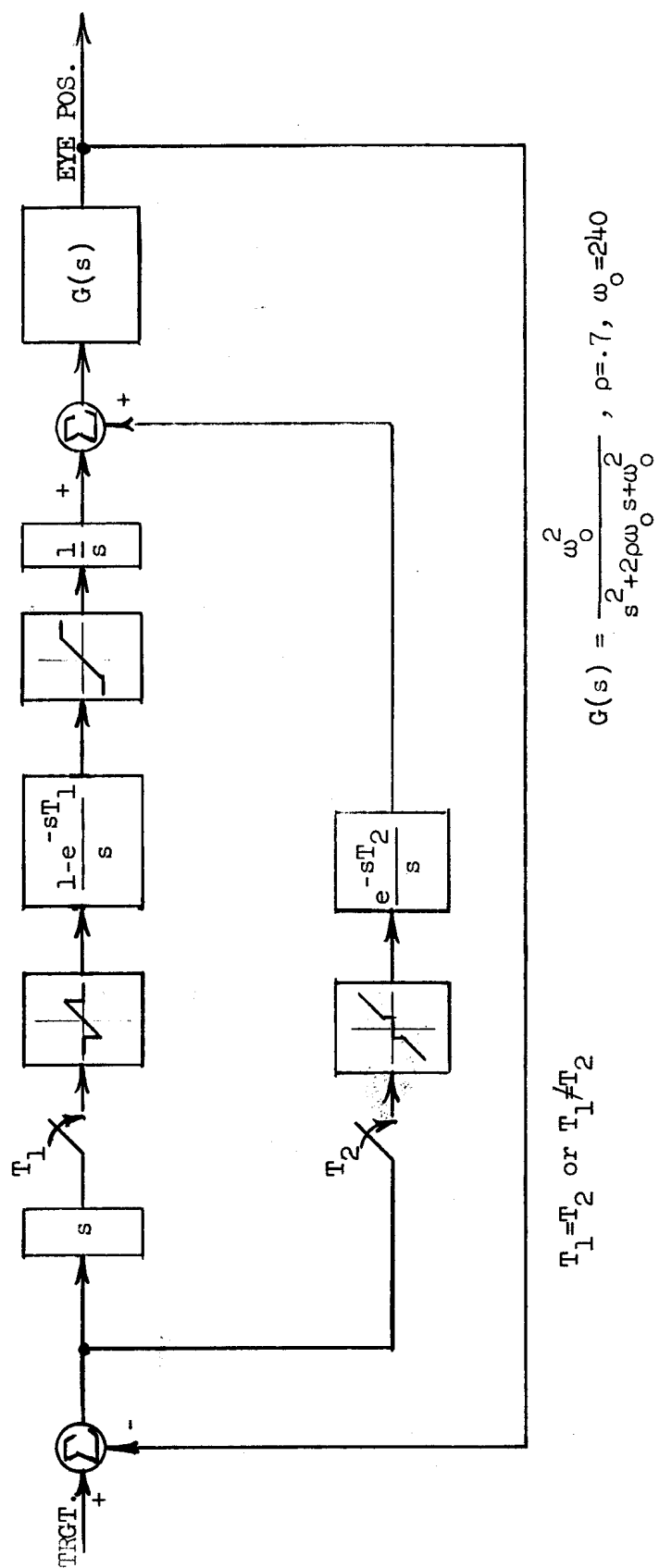


Fig. 3.4 Pavlidis' modified model of the eye tracking system



made modifications in an attempt to better match the physiological data. Since Vossius' model is contradicted by the existing physiological data discussed in the previous chapter, it will not be considered further. It should be pointed out however, that while eye movements are not primarily controlled by position feedback proprioception, there is evidence that such feedback does exist and has some effect on the system (see section 1.3.3 (101)). Vossius' model is linear and thus does not represent any of the non-linearities which have been observed in the biological system. The model of Fender and Nye is also linear and has the same shortcomings. The data gathered by Fender and Nye are applicable to tracking of predictable targets such as sine waves and the model they proposed is better suited for this mode than for the transient target motion with which we are concerned.

Young's model is the first to adequately represent the horizontal tracking system in the transient mode. As can be seen in Fig. 3.3, the non-linearities of the physiological system are represented by piecewise linear approximations in both paths. The major advance made by Young in modeling this system was representing it by a sampled-data control system. The following arguments were used by Young to show the necessity for a sampled system with independent paths for smooth tracking and saccadic positioning:

a) The response to a narrow pulse change in target position is a delayed pulse whose width is greater than and independent of the target pulse width. This type of response cannot be elicited from a linear, continuous system (even with a transport delay) since superposition does not hold.

b) The response to a constantly accelerating target (a parabolic input) consists of a series of constant velocity segments changing in slope at some fixed interval of time. This certainly suggests some form of sampling in the biological system.

c) The following gain versus frequency plot of the response to unpredictable target motion was presented in Young's thesis. It was obtained by constructing the target signal with sine waves of various frequencies (non-low-harmonically-related sinusoids) and analyzing the responses for frequency content (see Fig. 3.6). As can be seen from this plot, there is a sharp rise in gain of about 5-6 db at 2-3 Hz. This suggests a great deal of energy at a frequency with a half-period of 100-200 msec. Given an input spectrum  $F(s)$ , a sampled system would yield an output

$$G(s) = \frac{1}{T} \sum_{n=-\infty}^{\infty} F(s + nj\omega_0) \quad (3-1)$$

where  $T$  is the sampling period and  $\omega_0 = 1/T$ . This energy spectrum would have its first peak at  $\omega = \omega_0/2$ . Thus, if  $T = 0.2$  sec, ( $\omega_0 = 5$  Hz) the energy peak would occur at 2.5 Hz, which agrees with the experimental data. It was also observed experimentally that the tracking of signals with frequencies greater than 2.5-3 Hz degenerated to such an extent that it could not be considered as tracking at all. This is quite understandable when one remembers the sampling theorem which states that we lose information and are unable to reconstruct the data unless we sample at twice the highest frequency present. Thus, since we are sampling at 5 Hz, we should not be able to track any signals with frequency greater than 2.5 Hz.

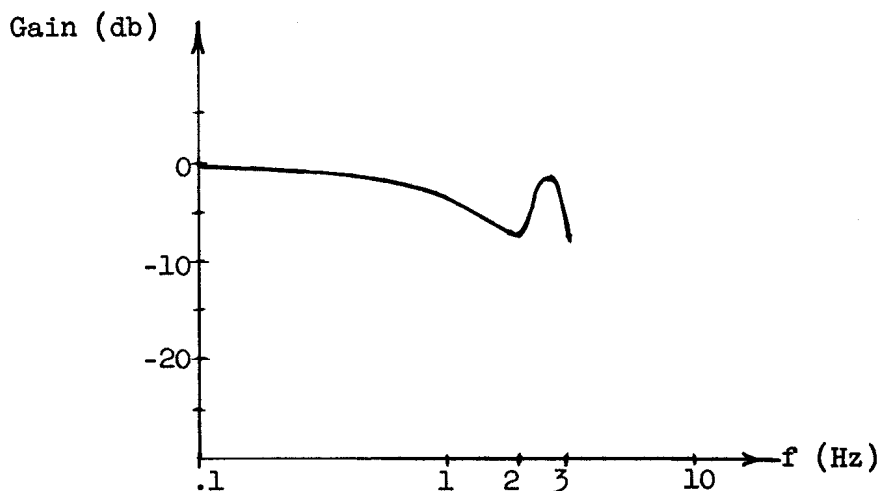


Fig. 3.6 Gain plot for unpredictable target motions (from Young)

d) Another observation in support of a sampled-data system involves the phenomenon of predicting a step change in position. It was found that if prediction occurred, the eye would move to where the subject guessed the target would be. This would happen either before target motion or immediately after it (too soon to be one refractory period). In the first case the subject's eyes moved to a new position based solely on a guess and in the second, the guess was made and then, as a result of the target motion, new visual information was presented which could have modified the original decision. It was found that there was no significant difference in the errors of the guess between the two cases. Thus, the new visual information could not alter the movement command which was made  $T$  seconds before the eye moved. This is also true of pulse targets where, even though the target has returned from a step change in position, the eye will move to where the target was and not move back until another interval of  $T$  seconds has elapsed.

The evidence for the independence of the two paths (smooth and saccadic) comes from the response to a step-ramp target. In this case

the target moves with a step change in position in one direction and at the same time assumes a velocity in the opposite direction. The eye's response to this is a delayed step change in position in the proper direction immediately followed by smooth tracking in the other direction with a velocity equal to that of the target. This is later followed by a corrective step in position to correct for the error due to the initial time lag. Thus, the two paths responded independently, one to the position error and the other to the velocity of the target.

There are however, several areas of disagreement between this model and observed data. The first discrepancy lies in the behavior of the model when presented with a step input. To be consistent with the physiological system, only the saccadic positioning path should respond and the smooth tracking path should remain inactive. This was supposedly assured by the velocity limiter of  $30^{\circ}/\text{sec}$  but if one considers small step inputs of less than or equal to  $6^{\circ}$  it will be found that, in addition to the saccadic response of a step change in position, the pursuit system will act as follows:

a) The input step will be sampled at  $t = 0$  and at  $t = 0.2$  sec and the difference will be taken between these two values in the error rate estimator (multiplied by  $1/T = 5$ ).

b) This signal (for a  $6^{\circ}$  step,  $6(5) = 30^{\circ}/\text{sec}$ ) will be fed into the velocity limiter and will pass through it and on to the first integrator. Thus, for steps of magnitude equal to  $6^{\circ}$  or less, the pursuit system will try to follow. This was first pointed out by Pavlidis and his model represents an attempt to correct for this.

The above error in Young's model is a minor one which could be overlooked since for larger steps the proper path is activated. There



is, unfortunately, an error in Young's model which is of much greater magnitude. In his analysis of the model, Young neglected the effects of the eyeball dynamics and considered only the two paths up to the summing junction before the second-order system which is used to simulate eye and muscle dynamics. His reasoning was that in the interval  $T = 0.2$  sec, the output of this black box would be within  $e^{-33.6}$  of its final value. While this is true, it is not sufficient to neglect the effects of including this subsystem in the overall system. The second-order dynamics exhibit a finite rise-time which affects the eye position at a very critical time, when sampling the error.

To better understand Young's analysis and what happens when the whole system is considered, let us examine the saccadic path when the target is a step (see Fig. 3.7). As the plots indicate, the error is sampled at  $T = 0$  and this impulse is passed by the dead-zone, delayed by  $T = 0.2$  sec, integrated and this output becomes the eye position (neglecting effects of eyeball dynamics). The eye position is fed back, negatively, and the error goes to zero exactly at  $t = T$ . Thus, when the sampler again closes at  $t = T$ , the error is zero, neglecting the ambiguity of the instantaneous drop in the error signal at the instant of sampling, and no further command is sent to the eye. This mechanism is an idealization which is mathematically correct assuming the value taken at any discontinuities is the final value and not any value in between the initial and final values.

Now with the aid of Fig. 3.8, let's examine the same system with the addition of the eyeball dynamics. Here we see the effects of the finite rise time on the sampled error. At  $t = nT$  the eye is always in

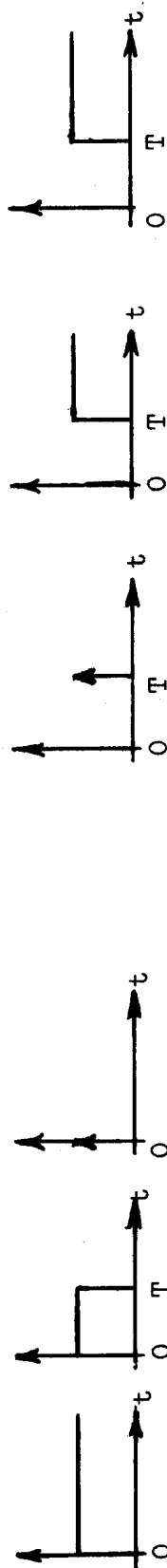
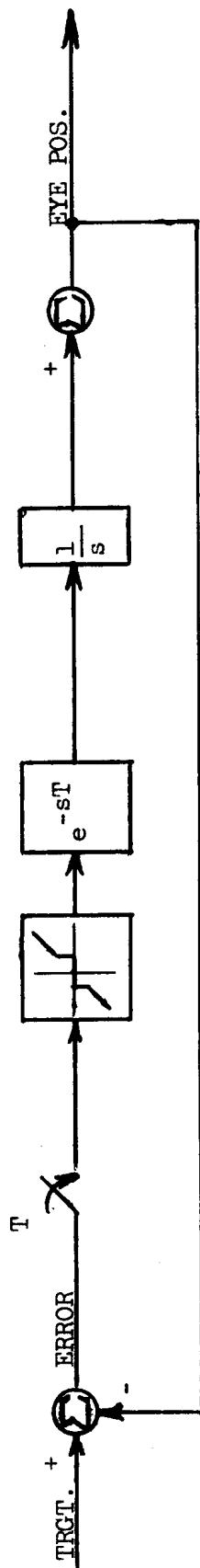


Fig. 3.7 Ideal response to step input

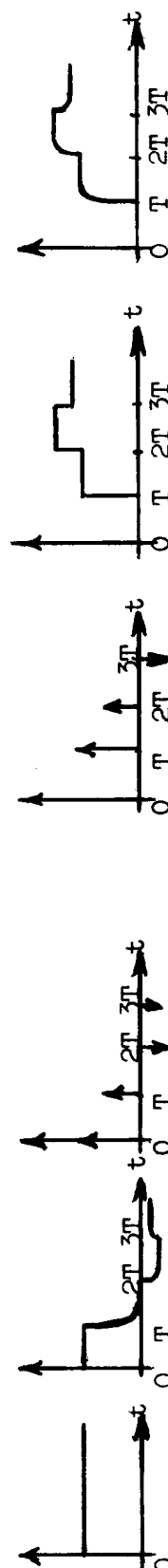
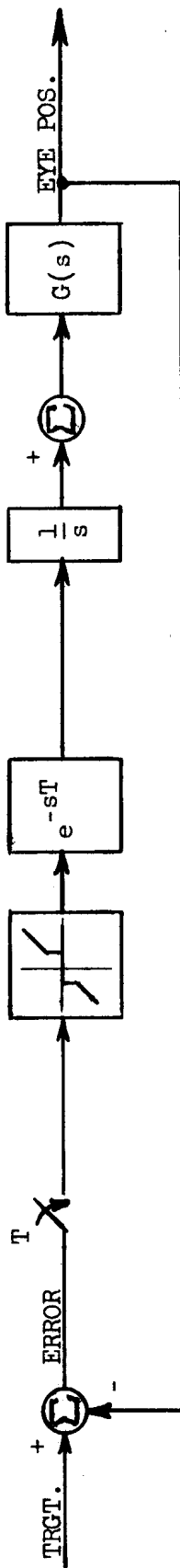


Fig. 3.8 Actual response to step input

transit and thus the system always sees an error and will continue to hunt until the error is small enough for the dead-zone to block the signal to the sampler. This behavior is inconsistent with the observed physiological response. Thus, the model proposed by Young is an excellent mathematical model only under the assumptions of an ideally responding eye (i.e., instant response with zero rise-time) and the value at a discontinuity must always be the final value of the function at that point. Since the aim of this study is to obtain a complete working model for both the normal and nystagmus-inflicted systems, modifications are necessary. Specifically, the system cannot have one sampler for both paths and the sampling period of the saccadic system cannot be the same value as the delay in the saccadic path.

The model proposed by Pavlidis has the same problem as Young's since the saccadic systems are identical. In attempting to prevent small steps from activating the pursuit mode, a differentiator was used in place of Young's error-rate estimator. This indeed does what it was designed to do since the differential of a step will not pass the first limiter. The system will work using the neural-net type differentiator mentioned by Pavlidis because it has an associated delay, but if a differentiating circuit is used the system's ramp response will be a ramp instead of the delayed ramp which is exhibited by the biological system.

Philosophically, Warnmeunde's model is a study in contradiction. He has taken great pains to develop what appears to be a very good

saccadic mechanism and has replaced the second-order approximation to eyeball dynamics with a more sophisticated system developed from the work of Miller. All of this was done to make the model a better representation of the biological system. A look at the pursuit system reveals man's attempt to improve on nature. This pursuit system is much better than the one present in the skulls of mortal men since it can accurately follow velocity changes continuously and thus it can track a moving target more accurately than man's poor velocity-segmented approximations. However, the object in physiological modeling is to synthesize a model that accurately represents the physiological system under study and not to build a better one. Whatever achievement this model represents in the field of control systems is offset by its failure in the field of physiological modeling. The model is presented for its excellent possibilities for further study. A combination of this saccadic pathway and another smooth tracking pathway might yield a very good model.

Recently there have been two other models proposed for the eyeball and muscle dynamics. The first, proposed by Robinson, was the result of a group of controlled experiments which were designed to study saccadic eye movement under abnormal conditions (105). By restraining one eye through a contact lens and measuring the force it applied while the other eye went through a normal saccadic movement he was able to determine that force was not a linear function of eye movement amplitude. Robinson's work was based on that of Alpern (1962) and Bjork (1955) (106,107). Robinson presents evidence that the system is a heavily overdamped one, has no resonant frequency, and is little

affected by eyeball mass. The author of the second study, Cook, went a step further than Robinson by considering the forces generated by the agonist and antagonist muscles instead of just the net forces on the eye (108). An excellent report on both papers as well as a presentation of each can be found in NASA CR-564 (109). Since these models were not used by the author, no further discussion of them will be presented. They do represent, however, significant advances in the modeling of a specific part of the total system under study in this paper. They were not used because the requirements of the model were satisfactorily met by a simpler representation of the extra-ocular system.

### 3.3 AUTHOR'S MODEL

The model developed for the horizontal tracking system of the normal subject is, for the reasons presented in section 3.2.2, a sampled-data control system. It is based mainly on the work of Young with some significant modification to overcome the shortcomings of his model. It is by no means the "last word" in models of this system for, even as this is being written, many different modifications are under consideration by the author for future work. It does represent the best model to date which has been completely synthesized and tested on a computer by the author and it does fulfill the requirements of duplicating the transient response of the physiological system as measured by the eye movement detection equipment discussed previously. For the following analysis the z-transform used was

$$z = e^{-sT} \quad (3-2)$$

The transmission  $z$  is a delay of  $T$  seconds and the region of stability is outside (not inside) the unit circle in the  $z$ -plane. Reference texts for sampled-data systems analysis and  $z$ -transform theory are Kuo (110), Jury (111), and Ragazzini and Franklin (112). Appendix A contains some notes on the transform-pairs used in this paper.

### 3.3.1 The Saccadic Positioning Path

This part of the overall system must be able to produce responses to step and pulse changes in target position which are in agreement with the physiological responses. Specifically, the step response would be a delayed step (150-250 msec) with a finite rise-time (5-25 msec) and the pulse response would be a delayed pulse (150-250 msec) whose width is independent of the short target pulse (50 msec) and is usually slightly longer than the delay (200-300 msec). The saccadic path is shown in Fig. 3.9. As can be seen, the difference between the target position and the eye position ( $X_5$ ) is taken and the result represents the error signal ( $X_1$ ). The error is fed into a non-linear element which provides the  $\pm 0.25^\circ$  dead-zone which will prevent this model from responding to saccades below this threshold or to noise. If the error is large enough it is then sampled by an impulse modulator every 230 msec beginning with the onset of the transient input. The sampled error ( $X_2$ ) is then integrated to give a step command ( $X_3$ ) which is delayed 200 msec ( $X_4$ ) before being sent to the eyeball dynamics,  $G(S)$ , which then act on the command to produce the desired eye position ( $X_5$ ). Although other models for the extra-ocular system have been presented, the author feels that for this system, Westheimer's second-order approximation is more than adequate. The important difference between

this model and that of Young's is the sampling rate of 230 msec as opposed to 200 msec. For a second-order system with  $\rho = 0.7$  and  $\omega_0 = 240$ , this 30 msec delay between the instant the position-change command is given to the instant of sampling amounts to more than 5 time constants. This insures that the eye will have assumed its new position before the error is sampled again and that no sampling can occur during a saccade. Fig. 3.10 is the signal flow graph of the system in the linear region of the dead-zone. From this graph the following can be written by inspection:

$$R(s) = \frac{e^{-sT_1} \frac{1}{s} G(s)C(s)^*}{1 + \frac{z^\gamma G(s)^*}{1-z}}, \quad \gamma = T_1/T_2 \quad (3-3)$$

$$= \left( \frac{1-z}{s} \right) \left[ \frac{z^\gamma}{1-z + z^\gamma G(s)^*} \right] G(s)C(s)^* \quad (3-4)$$

This represents a zero-order hold with a delay term corresponding to the delay of  $T_1$  msec. Similar analysis of Young's system yields,

$$R(s) = \left( \frac{1-z}{s} \right) \left[ \frac{z}{1-z + zG(s)^*} \right] G(s)C(s)^* \quad (3-5)$$

which is a zero-order hold and a delay equal to the sampling interval  $T$ . Young's presentation did not include the effects of  $G(s)$  although he pointed out that where his analysis showed an abrupt step change in position the actual eye would move as a second-order system with finite rise-time and possible overshoot. To arrive at Young's system equation

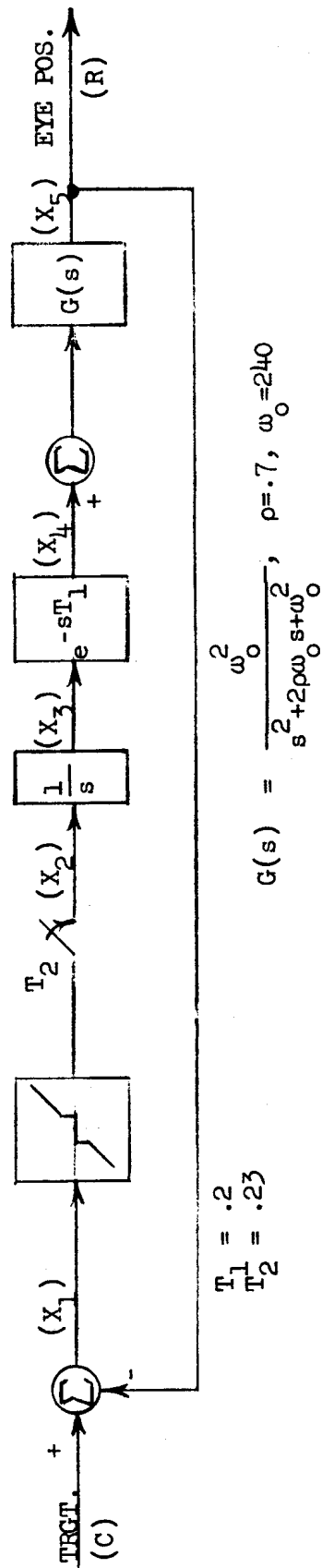


Fig. 3.9 Saccadic system

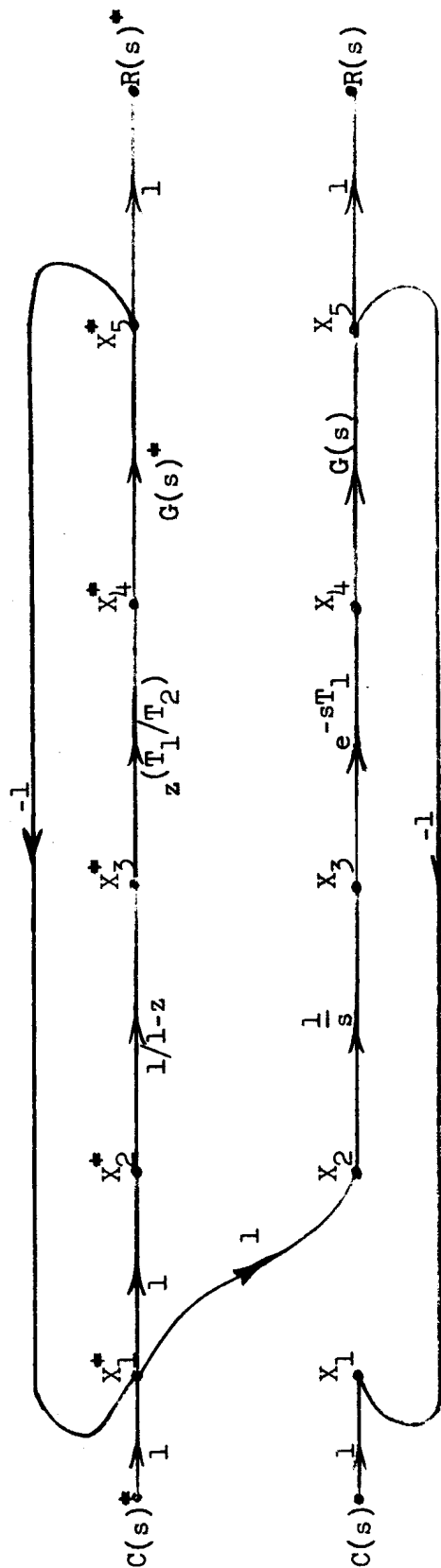


Fig. 3.10 Composite signal flow graph of the saccadic system



set  $G(s) = G(s)^* = 1$  in equation (3-5). The step and pulse responses of this system are shown in Fig. 3.11. As is evident in both cases, the instant of sampling ( $T_2$ ) occurs after the eye has assumed its new position (30 msec after the eye starts to move). This eliminates both the hunting found in Young's model and the sampling during a saccade; the latter, a contradiction to physiological evidence which suggests that vision is suppressed during a saccade (113,114,115,116).

### 3.3.2 The Smooth Tracking Path

The smooth tracking system must be able to track targets with velocities up to  $25\text{-}30^\circ/\text{sec}$ , approximate accelerating targets with a series of constant velocity segments which change in amplitude at a fixed interval and correctly track a moving target regardless of any initial displacement. The ramp response should be a delayed ramp (150-250 msec) and the velocity segments of the parabolic approximation should be changed in amplitude at an interval of every 150-250 msec. A model for this pursuit mode is shown in Fig. 3.12. As in the saccadic system, the error ( $Y_1$ ) must pass the dead-zone before it will be acted upon. If it does, it is first differentiated to obtain the error rate ( $Y_2$ ) which is then passed by a limiter if it is less than or equal to  $25\text{-}30^\circ/\text{sec}$ . This prevents the model from tracking targets with high velocities, which is consistent with physiological evidence. If passed, the signal is delayed by 20 msec ( $Y_3$ ) and sampled every 200 msec ( $Y_4$ ). This sampled error rate is then integrated and must pass through a saturation non-linearity. Thus the eye cannot move smoothly with a velocity greater than  $25\text{-}30^\circ/\text{sec}$ . If it passes it is integrated once more to form the command ( $Y_5$ ) which is sent to the eyeball dynamics,

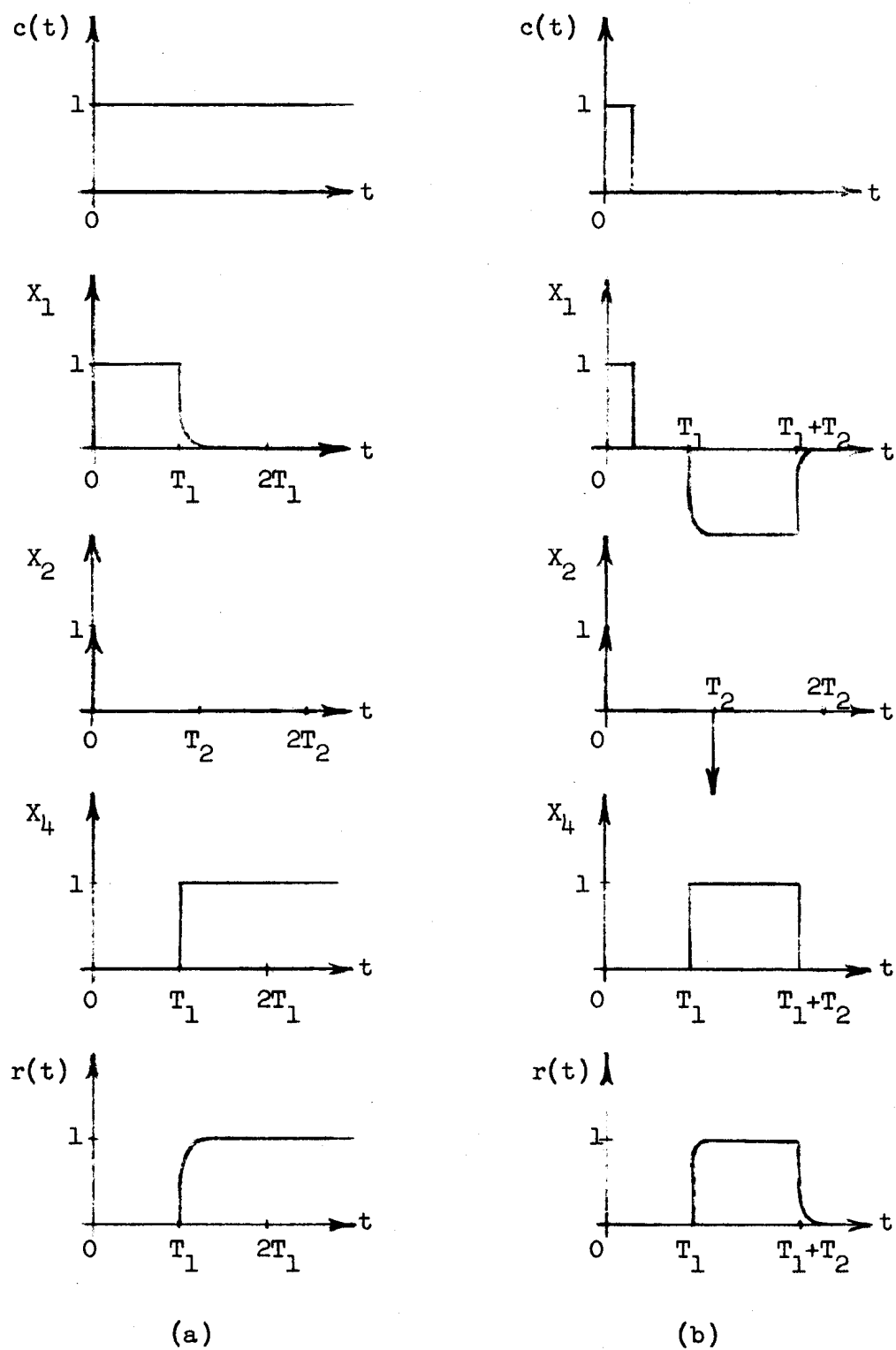


Fig. 3.11 Saccadic system transient responses (a) step response  
(b) pulse response

$G(s)$ , which then cause eye motion ( $Y_G$ ). This system will not respond to any step change in target position no matter how small the amplitude since the differentiator will produce an impulse whose magnitude will pass the first limiter. The 20 msec delay allows the eye to come extremely close to its steady-state value before sampling occurs. For this system the steady-state error to a ramp input of  $S^0/\text{sec}$  is given by,

$$e(\infty) = \lim_{s \rightarrow 0} sE(s) \text{ where,} \quad (3-6)$$

$$E(s) = S \left[ \frac{1}{s^2} - \frac{\omega_o^2}{s^2 (s^2 + 2\rho\omega_o s + \omega_o^2)} \right] \quad (3-7)$$

Thus,

$$e(\infty) = \frac{2S\rho}{\omega_o} = .0058 S. \quad (3-8)$$

For a  $10^0/\text{sec}$  ramp, this amounts to a steady-state error of  $0.058^0$ . Now, the time from the onset of motion to sampling is equal to 180 msec, which is roughly 30 time constants, so that the error at the instant of sampling is equal to the steady-state error. This is truly negligible. Fig. 3.13 is the signal flow graph of this system in the linear regions of the three non-linearities. The eye position would be given by,

$$R(s) = \frac{z^{0.1} \left( \frac{-\ln z}{T} \right) \left( \frac{1}{s^2} \right) G(s)C(s)^*}{1 - \left( \frac{\ln z}{T} \right) (z^{0.1}) \left[ \frac{Tz}{(1-z)^2} \right] G(s)^*} \quad (3-9)$$



$$= \left( \frac{1-z}{s} \right) \left( \frac{1}{s} \right) \left( z^{0.1} \right) \left[ \frac{\ln z}{(z-1)^2 + z^{1.1} \ln z G(s)^*} \right] \left( \frac{1-z}{T} \right) G(s) C(s)^* \quad (3-10)$$

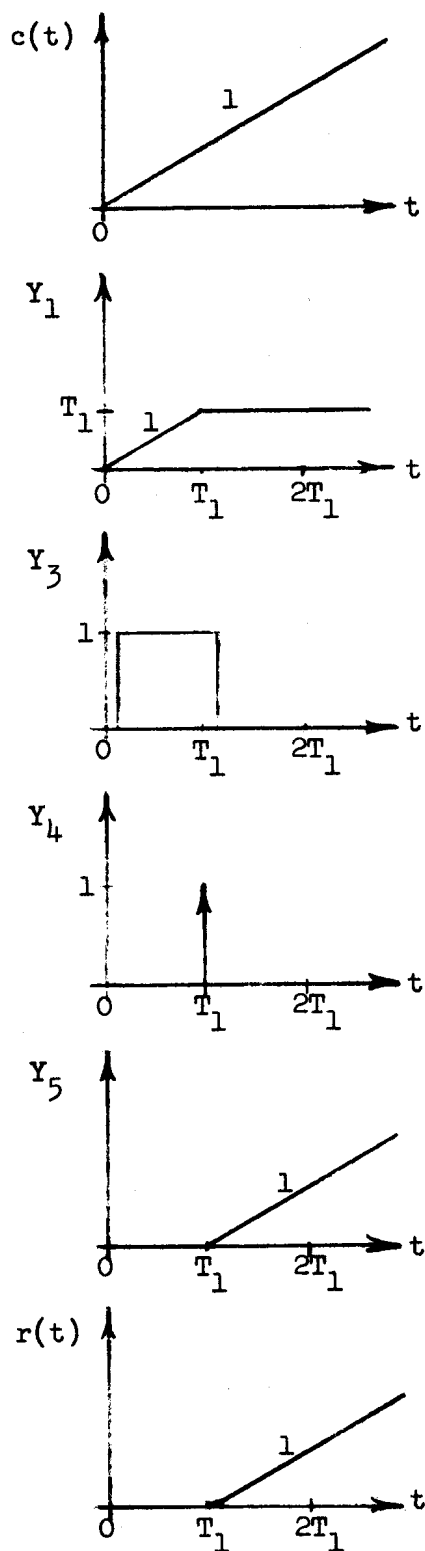
This consists of a zero-order hold, an integrator, a delay term, a term corresponding to the differentiator and a first-difference term. Analysis of Young's system yields,

$$R(s) = \left( \frac{1-z}{s} \right) \left( \frac{1}{s} \right) \left[ \frac{1}{1-z-zG(s)^*} \right] \left( \frac{1-z}{T} \right) G(s) C(s)^* \quad (3-11)$$

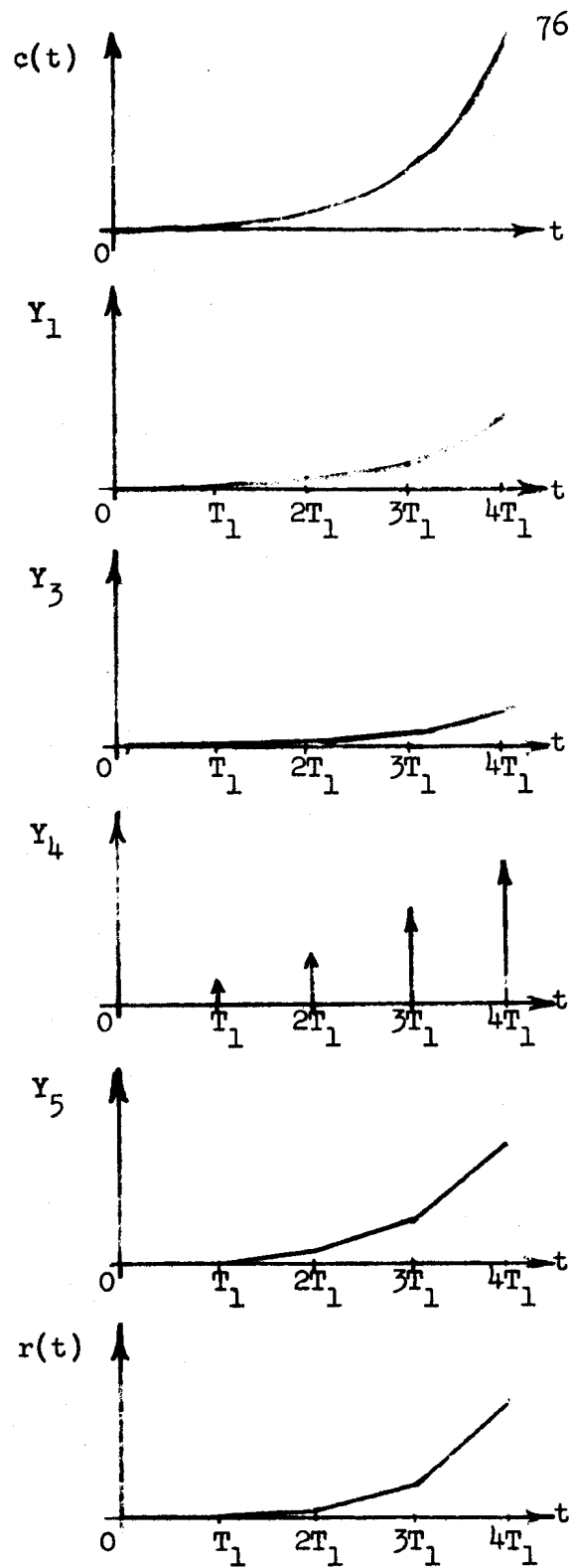
Responses to a ramp and a parabola are shown in Fig. 3.14. Here, as in the saccadic mode, sampling occurs after the eye has had a chance to respond to the previous command. This prevents the inevitable errors associated with sampling at the command instants which would be analogous to sampling during a saccade in the saccadic model. Both will result in responses which differ from the biological responses.

### 3.3.3 Complete Model for Horizontal Tracking System

The model for the whole system involves merely combining the two subsystems discussed above. Unfortunately, this simple little maneuver makes an exact analysis of the system an extremely long and difficult job. Fig. 3.15 shows the complete system for the normal subject. The description of the system merely consists of the two previous descriptions of the subsystems. The dead-zone and retinal feedback paths are now common to both paths. The preliminary flow graph for this multi-rate sampled-data system is shown in Fig. 3.16. The presence of two samplers of different sampling rates calls for an additional step in the analysis leading towards the composite flow graph. In order to



(a)



(b)

Fig. 3.14 Pursuit system transient responses (a) ramp response  
(b) parabolic response

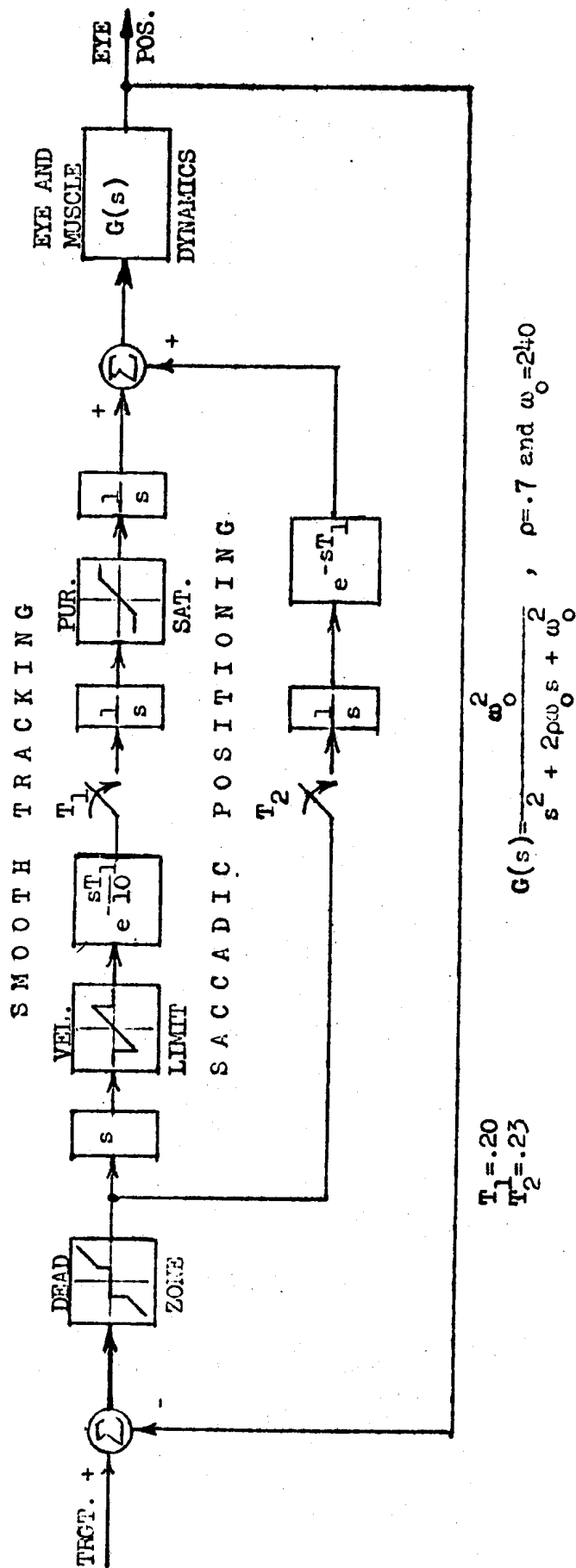


Fig. 3.15 Sampled-data model for normal system

convert this to a system in which all the samplers share a common sampling rate  $T$ , integers  $N_1$  and  $N_2$  must be found such that,

$$T = N_1 T_1 = N_2 T_2 \quad (3-12)$$

For this case  $N_1 = 23$  and  $N_2 = 20$ . The next step involves replacing samplers  $T_1$  and  $T_2$  in the flow graph by 23 and 20 parallel samplers respectively. Each sampler has a time advance preceding it and a time delay following it. These are referred to as "skip samplers". Fig. 3.17 shows the 23 skip samplers which would replace sampler  $T_1$  in the new flow graph. Sampler  $T_2$  would be replaced by 20 such samplers. Analysis of this system now proceeds according to the standard practices for obtaining the sampled flow graph and then the composite graph from which the transfer function is written. For sampling rates related by low integers such as 2 or 3, this analysis isn't too cumbersome, but for the system in question where the integers are 23 and 20, the task is prohibitive; especially due to the probable complexity of the final transfer function which will result. In light of the above, the author feels that a better understanding of the system can be obtained by considering its responses to several standard transient inputs.

(1) Step Input: Since the pursuit system does not respond to a step input, the total system response is identical to that of the saccadic path alone as described in section 3.3.1 and illustrated in Fig. 3.11.

(2) Pulse Input: Here again, the analysis is identical to that in section 3.3.1.



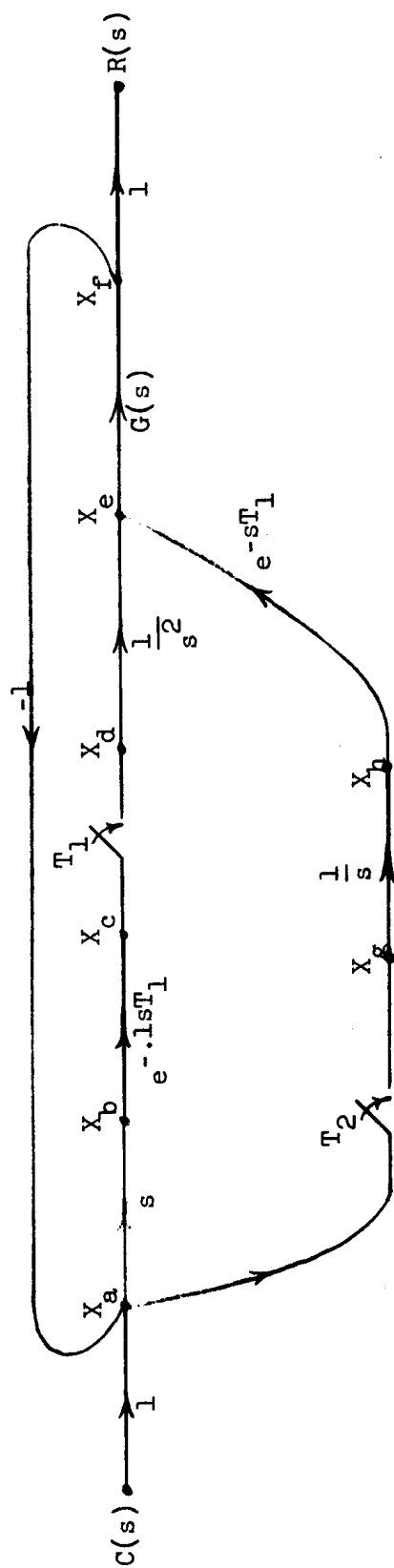


Fig. 3.16 Preliminary flow graph for complete system

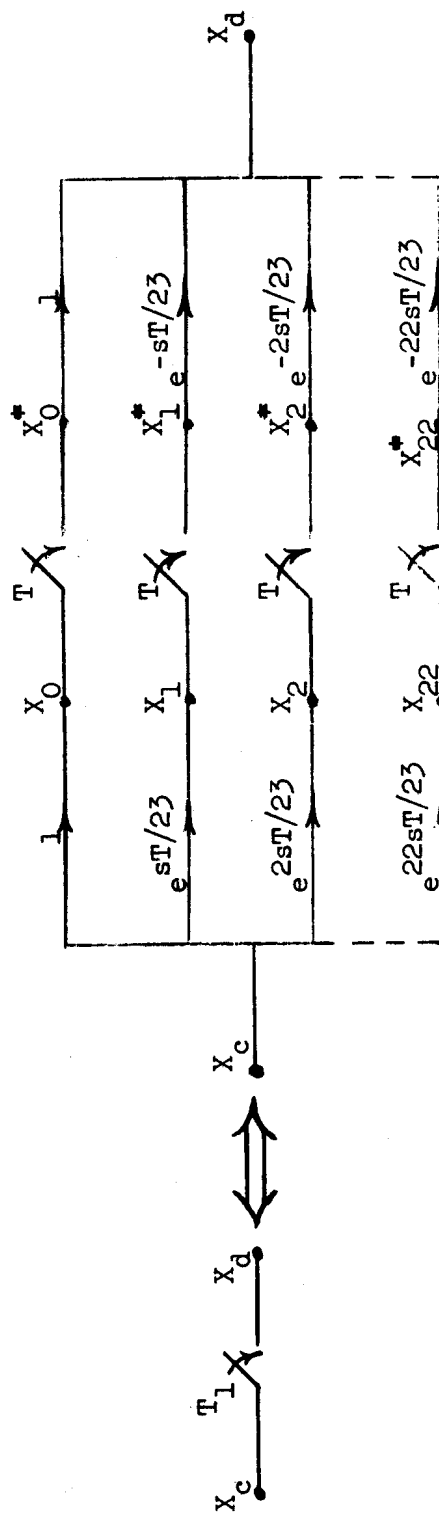


Fig. 3.17 Sampler  $T_1$  replaced by "skip samplers"

(3) Ramp Input: The ramp response of the system is illustrated in Fig. 3.18. For identification of the functions plotted refer to Fig's. 3.9 and 3.12. We observe that the response consists of a ramp delayed by  $T_1$  sec followed by a corrective saccade at  $T_1 + T_2$  sec which corrects the position error due to the delay in the pursuit mode.

(4) Parabolic Input: Fig. 3.19 shows the response to a constantly accelerating target. This can be looked at as an addition of several ramp responses. Note that this velocity-segment approximation to the accelerating target always has a position error.

(5) Step Ramp Input: This input, where the target is initially displaced in one direction and immediately moves with a constant velocity in the other, serves to demonstrate the independence of the two paths. The response of the pursuit path is unaffected by the presence of the step change in position in the direction opposite to the initial velocity of the target (see Fig. 3.20).

Comparison of these predicted responses of the model with the observed physiological responses of the normal subject affirms the validity of this model.

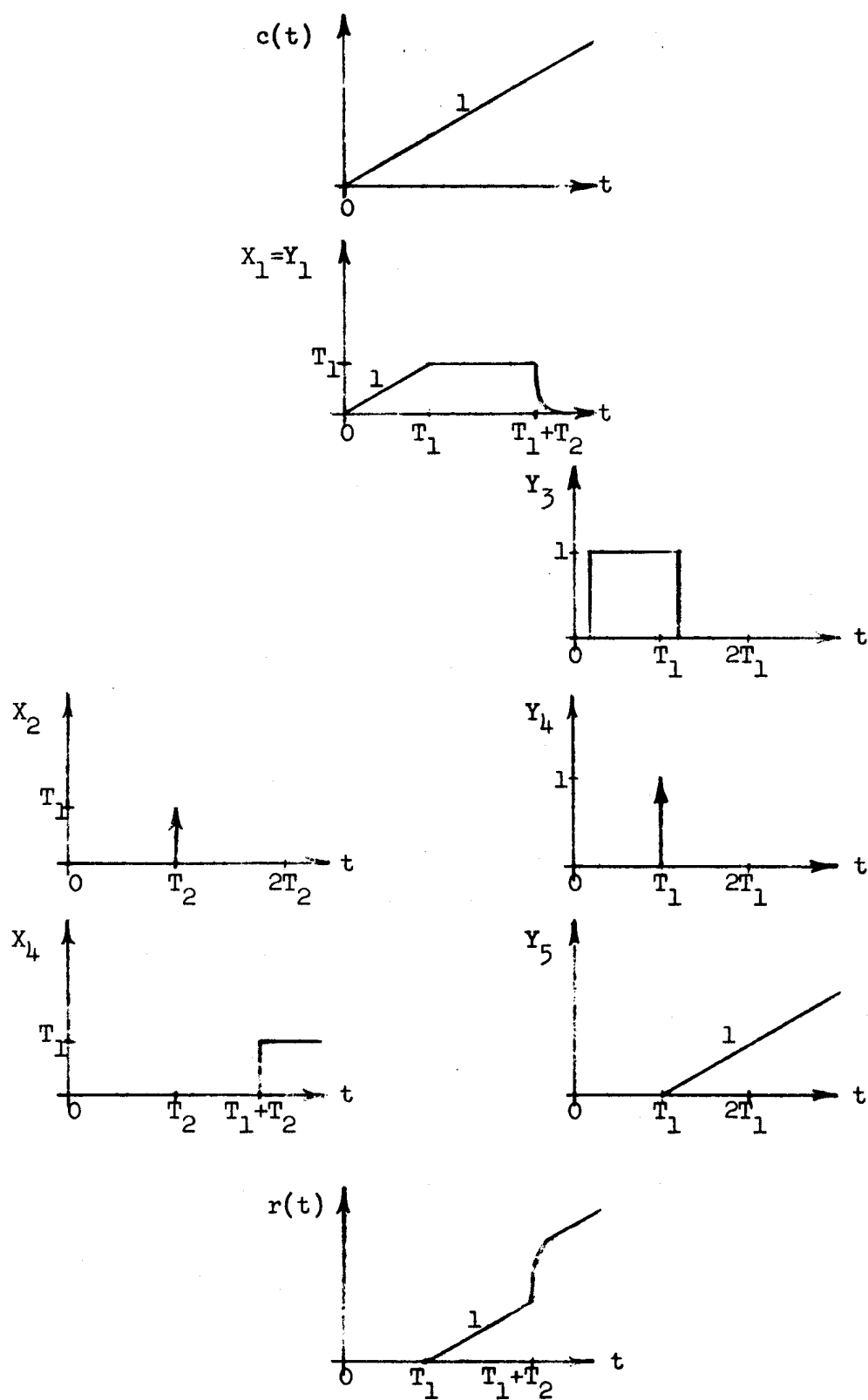


Fig. 3.18 Ramp response of the complete model

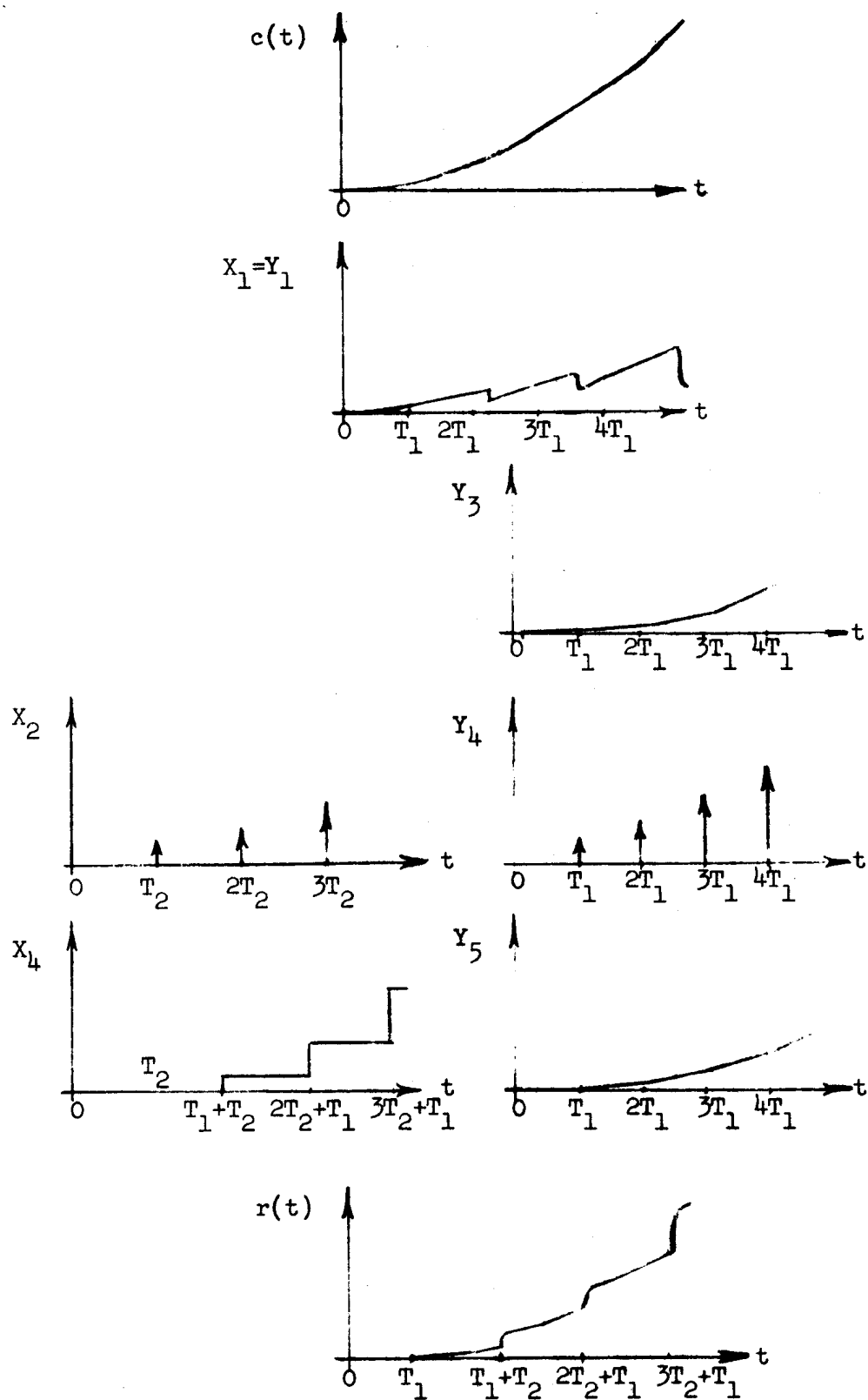


Fig. 3.19 Parabolic response of the complete model

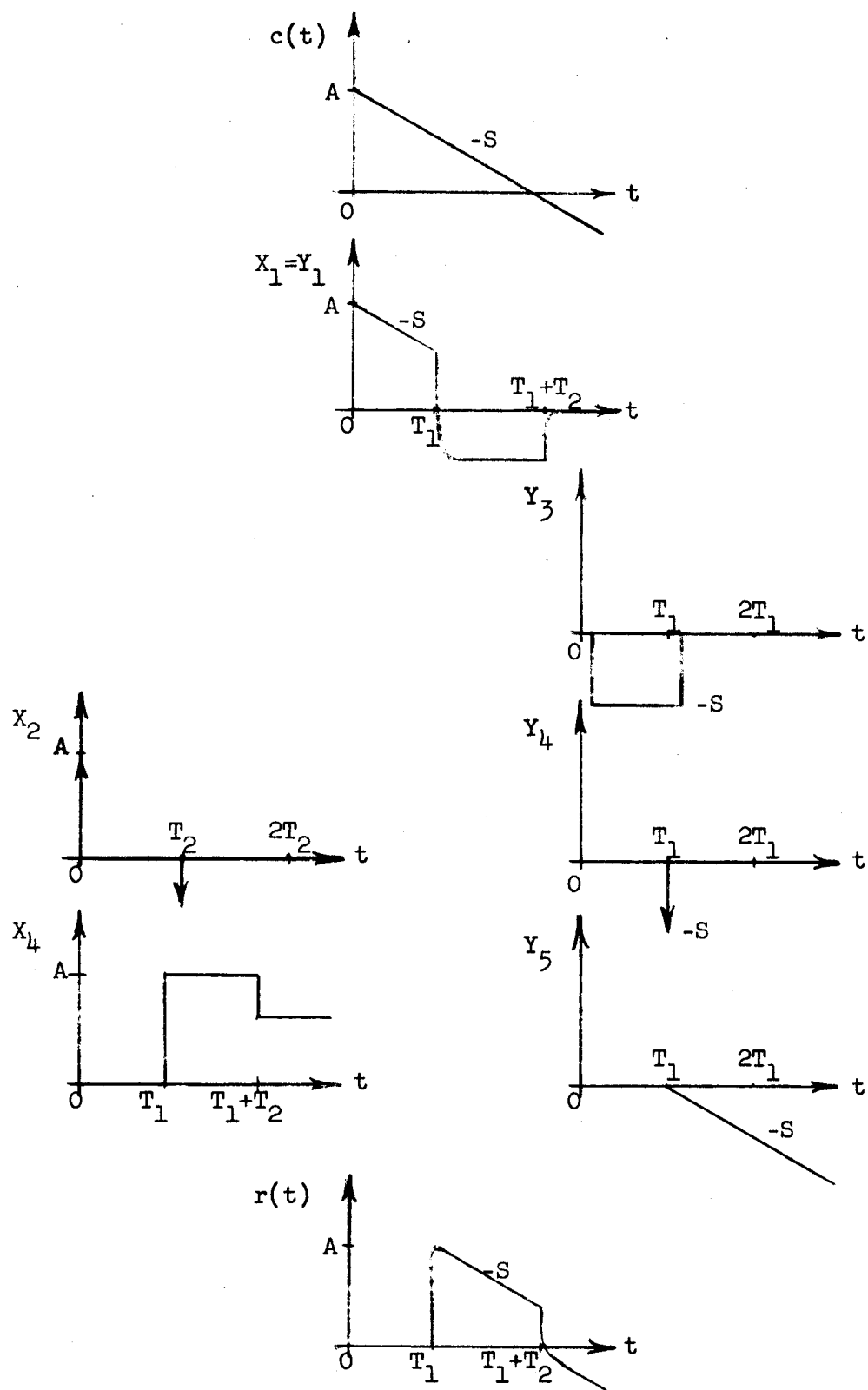


Fig. 3.20 Step-ramp response of the complete model

## CHAPTER IV

### COMPUTER REALIZATION

#### 4.1 SYSTEM USED

The computer used to synthesize the model for the horizontal tracking system was a Burroughs B-5500. The programming was done via the ANALOG-ALGOL system. This is a method of programming, on a digital computer, analog processes which are described by integro-differential equations. Analog-Algol is designed so that a person with no experience on a digital computer can use it to solve complex problems. The commands used are standard English words whose mathematical meaning is common knowledge to engineers and mathematicians. Using Analog-Algol one can go directly from a block diagram to the program. The order of operations, as stated on the program, is not important since the program will run correctly regardless of order. Thus, this system places a powerful tool, namely a large digital computer, within the reach of most engineers and scientists.

#### 4.2 PROGRAM FOR THE NORMAL TRACKING SYSTEM

The model for the normal tracking system as shown in Fig. 3.15 was programmed first. Only after this model was successfully synthesized was a model postulated for the system with nystagmus. Successful synthesis entails the synthesis of a model whose responses, as printed out by the computer, agree with those of the normal biological system when subjected to the same inputs. The actual mechanics of programming the computer to carry out the operations indicated in Fig. 3.15 will

not be discussed here except to give the reader some insight into how it is done by the following brief description of the commands which would accomplish the first operation. This first operation is simply obtaining the error signal from the target and eye position. The command for this would be,

$$\text{SUM } 2(\text{TRGT}, 1, \text{EYE}, -1, \text{ERR}); \quad (4-1)$$

This tells the computer to sum two variables, which are the target (multiplied by 1) and the eye (multiplied by -1) and the result shall be called the error. This simplicity is carried through most of the operations so that one just starts at the input of a block diagram and works his way to the output by writing commands, in abbreviated English terms, for each operation. Appendix B contains a copy of the final program which is a dual-mode program capable of representing either the normal system or the system with nystagmus. Between the commands "BEGIN" and "BEGINANALOG" the variables are defined, operating instructions are given, and the break points of non-linearities are given. After this, the forcing functions are given and the sampler defined. After the command "ENDFORCING;" the operations are listed in the manner just shown. At the very end is the command "ENDANALOG;".

#### 4.2.1 The Non-Linearities

As can be seen in Fig. 3.15, there are several non-linear devices. These were programmed into the system as follows:

(1) Dead-Zone: When the input to the dead-zone is between -0.25 and +0.25 the output is zero. Any input less than -0.26 or greater than +0.26 will produce an output equal to the input. The inputs falling in

the transition regions ( $-0.25$  to  $-0.26$  and  $+0.25$  to  $+0.26$ ) yield outputs which vary linearly from 0 at  $\pm 0.25$  to  $\pm 0.26$  at  $\pm 0.26$  (see Fig. 4.1).

(2) Velocity Limit: This non-linearity will pass on a one-to-one basis any velocity between  $-25$  and  $+25$  degrees per second and will provide a zero output for any input less than  $-26^\circ/\text{sec}$  or greater than  $+26^\circ/\text{sec}$ . Again, the transition zones yield outputs which vary linearly between the two extremes (see Fig. 4.2).

(3) Pursuit Saturation: This is a simple saturation characteristic where any input between  $-25^\circ/\text{sec}$  and  $+25^\circ/\text{sec}$  is passed as received but any input less than  $-25^\circ/\text{sec}$  or greater than  $+25^\circ/\text{sec}$  will produce  $-25^\circ/\text{sec}$  or  $+25^\circ/\text{sec}$  outputs respectively (see Fig. 4.3).

#### 4.2.2 Sampling

The sampling operation, which mathematically is multiplication by an impulse train, was accomplished on the computer by the use of a square pulse whose height was  $1/H$  and whose width was  $H$ , where  $H$  was a variable which could be set to some arbitrarily small number. This pulse of unit area is equal to an impulse in the limit as  $H$  approaches zero. For this problem a value of 0.01 was satisfactory. In the saccadic path the sampling and the integration were combined into a subroutine which performed these operations by another method. A discussion of how this was done within the digital computer, or how any of the other operations were carried out for that matter, is something which is not within the scope of this paper.

#### 4.2.3 Equivalence to Mathematical Model

The construction of the non-linearities was carried out in the above-described manner to eliminate the possibility of an oscillatory



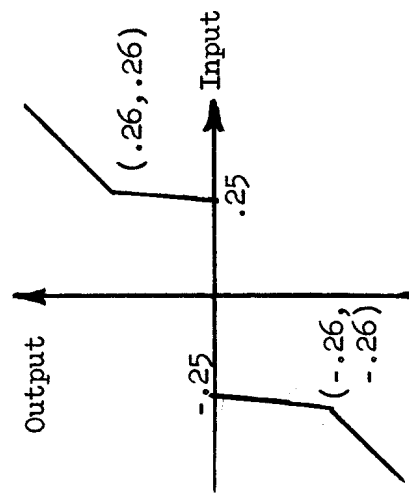


Fig. 4.1 Dead-zone

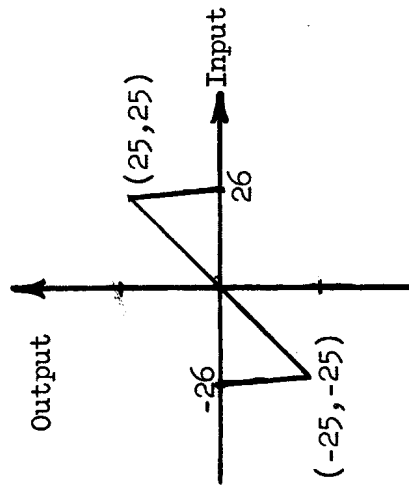


Fig. 4.2 Velocity limit

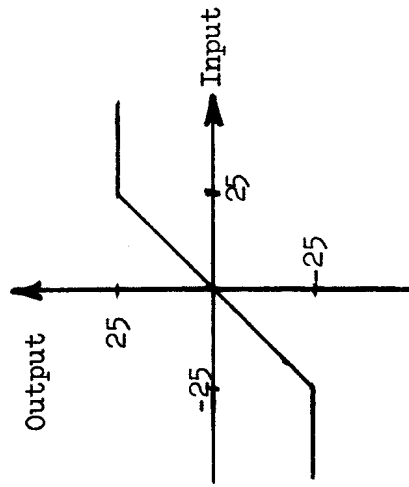


Fig. 4.3 Pursuit saturation

condition which could have developed if they contained the infinite slopes present in the mathematical model. Actually, they represent a closer approximation to the physiological system since it is very unlikely that one would find a step discontinuity in a natural system.

The unit area pulse approximation to an impulse function is a common method of achieving this ideal function, whose height is infinite, width is zero, and area is equal to one. Obviously the area of the pulse is equal to one for all  $H$  greater than zero. The only other criterion that must be met is that it must function in the system as an impulse would. More simply, its duration must be short in comparison to the sampling rate. The choice of  $H = 0.01$  satisfies this since  $T = 0.20$ . Had this proven unsatisfactory,  $H$  could have been reduced to a much smaller value, limited only by the computer's ability to handle the large pulse.

#### 4.2.4 Alternate Program

Since the program used contained a differentiator and many systems cannot handle this noisy operation, the following changes can be made and an equivalent system will result:

- (1) Instead of a target input, feed in the derivative of the target.
- (2) Instead of feeding back eye position feed back the derivative of eye position. This is available since the realization of the eye dynamics consists of two integrations so that the feedback can come from the point just before the final integration.
- (3) Now, feed the difference of these two signals,

$$\frac{d}{dt} (\text{TRGT}) - \frac{d}{dt} (\text{EYE POS.}) = \frac{d}{dt} (\text{ERROR}) \quad (4-2)$$

to the smooth tracking path, which now begins with the first limiter.

(4) Into the saccadic path, feed the derivative of the error and integrate it before sampling it.

Thus, without a differentiator, we now have a system which will yield the same eye position output to various target inputs as the system which was used. The saccadic system will have position error as its input and the smooth tracking system will have error rate. All that need be done is to use the derivative (calculated) of the target of interest as the input (see Fig. 4.4). The computer used had a differentiator available so that this alternate method was not employed.

#### 4.3 INPUTS

The inputs to the computer were: (a) a pulse of variable height (S) and width (ST); (b) a step change of variable height (S); (c) a ramp of variable slope (SL); (d) a parabola of variable acceleration (SL); and (e) a step-ramp of variable height (S) and slope (SL). The values for S, ST and SL are punched in on a data card which is read by the computer along with other variables.

#### 4.4 NYSTAGMUS

Once the above program was completed so that the system responded correctly to the given transient inputs, the model was re-examined to determine the modifications necessary to enable it to reproduce nystagmus responses to these inputs.

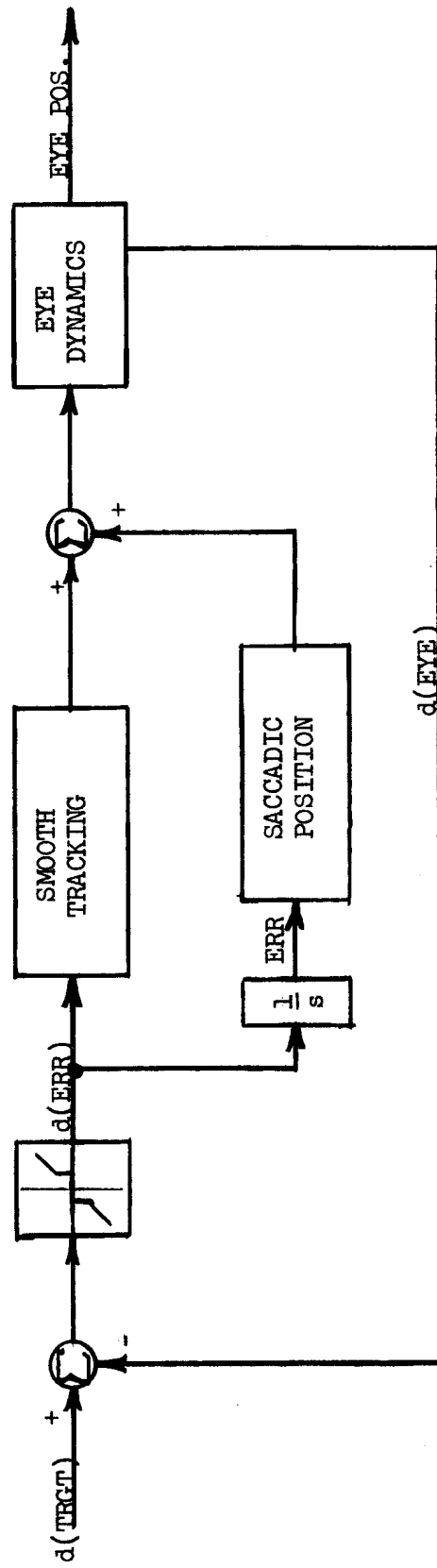


Fig. 4.4 Alternate program

#### 4.4.1 Physiology

Before proceeding with the actual modeling it would be advantageous to review the known facts about congenital, horizontal, fixation nystagmus which exhibits an atypical jerk waveshape to the left and has a saddle in the slow phase. Introductory remarks in Chapter I discussed the type and genetic history of the particular nystagmus of interest and in Chapter II some comments and observations were presented based on the observed waveshape. In order to obtain a clear picture of the particular affliction studied (as present in the subject used) a review of the pertinent literature with a discussion of the case of interest will be presented.

Briefly, the heredity appears to be either recessive sex-linked which affects only males and is transmitted by females or simple recessive. Although simple recessive is very rare, the presence of a consanguineous marriage in the parentage is indicative of such transmission. If it is of the sex-linked, recessive type one would expect no appearance in the subject's offspring and possible appearances in the male children of the subject's daughters. This may not show up for several generations. There have been many family histories traced which illustrate this type of transmission (73,117,118,119,120,121). Nystagmus occurs in males at the rate of  $1/5,032$  and in females,  $1/10,596$  (72). Some common characteristics of congenital nystagmus are: (a) it persists throughout life; (b) head movements, which may be present, are compensatory in nature; (c) it is always bilateral; (d) the eye is anatomically normal; (e) the waveshape may change from pendular to jerky as eye position is varied; (f) it is mainly horizontal; (g) visual acuity

is surprisingly good (20/30 to 20/50); (h) the subject does not see movement of stationary objects; (i) visual acuity may improve with age; (j) head shaking and/or nystagmus magnitude is aggravated by excitement or attempts to see more clearly; (k) many cases show spontaneous nystagmus with eyes closed; (l) in the dark, a non-visual stimulus may induce nystagmus; (m) a rare form exhibits a jerk with an atypical slow phase containing a saddle-like form (notch) or even a rise; (n) the minimal position is often at some angle to the straight-ahead position and the maximum position may not be at the terminal lateral position; (o) it often disappears with lid closure; and (p) the frequency may be from 2-5 Hz (27,31,30,122,123). The subject in question would be well described by statements (a) through (j) and (l) through (p). His vision is 20/40. Other findings which have been reported include: (a) the quick component is in the direction of gaze; (b) some subjects have a hypoactive labyrinth; and (c) nystagmus is worse on one side than the other (28). The subject tested had a quick component which was always to the left, his labyrinths appear to be normal (clinical tests revealed no abnormality) and his nystagmus is worse when the gaze is to the right.

The exact mechanisms involved in congenital fixation nystagmus are still a mystery. However, evidence has been presented to support the conclusion that it is a brain stem mechanism (48,31,124,125,126). Studies have also been made to show that the slow and quick phases are due to separate mechanisms but they have not been conclusive (129). In support of the midbrain theory are various reports of the effects of barbiturates (26,128,129,130). It has been found that intra-venous administration of any of several barbiturates (amobarbital, phenobarbital

or pentobarbital) caused nystagmus to decrease temporarily and visual acuity increase in subjects with nystagmus and when given to normal subjects it caused nystagmus. These substances act on the brain stem (131).

Since the position of minimum nystagmus is often an eccentric one, an operation has been devised and performed which rotates the eyes toward the position of greater nystagmus enough to reset the minimum position to be straight-ahead. This is done by re-positioning the points of attachment of the horizontal recti muscles (72,132).

#### 4.4.2 Modeling Nystagmus

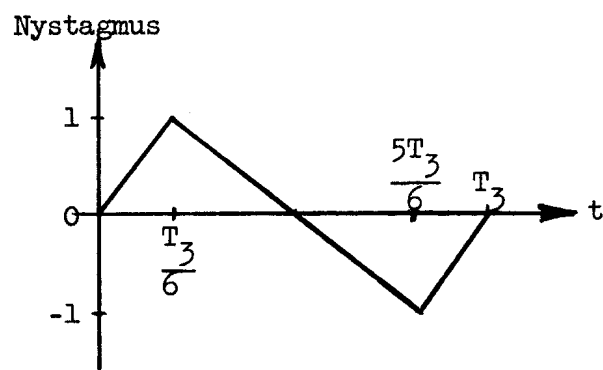
The first item of concern in the modeling of the system with nystagmus is the waveshape which will be considered an acceptable representation of the actual motion. As the data of Chapter II show, the subject had an atypical jerk-type nystagmus with a saddle-like form in the slow phase. The frequency varied about 3 Hz. For the model this will be represented by a simple asymmetrical triangular waveshape. The slope of the quick component will be twice that of the slow component (consistent with the data) and the amplitude must be made to vary with eye position as shown in Fig. 2.11. Since the notch does not always appear and its position varied it will not be part of the simulation. This will not affect one of the main purposes of the model, which is to demonstrate the functional independence of the nystagmus mechanism and the tracking mechanism. The literature cited in section 4.4.1 contained the commonly accepted theory that nystagmus was independent of the normal movements of the eyes. The data presented in Chapter II substantiates this statement since the responses to the various transient inputs were equivalent for both subjects with the exception of the 3 Hz

oscillation superimposed on the response of the subject with nystagmus. If the waveshape shown in Fig. 4.5 is used to simulate nystagmus and it is multiplied by the output of the non-linearity shown in Fig. 4.6 (where the input is eye position) the result will be a 3.3 Hz sawtooth whose magnitude varies with eye position in a manner conforming to the physiological data and whose quick component is twice as fast as its slow component.

#### 4.5 COMPLETE DUAL-MODE PROGRAM

To incorporate the above nystagmus generator into the model, this variable-magnitude waveshape must be fed into the input of the eye dynamics and also to some point in the system before the error signal is fed into the two forward paths. This must be done to prevent the model from acting on this oscillation. The subject does not misinterpret this apparent target motion as real target motion (in fact, he is not even conscious of it) so the model must also act as though it were not present. This is done by adding the same motion (slightly delayed) to the error signal (which carries the negative of this motion) and thereby canceling it out. The final dual-mode model is shown in Fig. 4.7. The value of  $N$  is punched in on a data card as well as the value of GAIN. If  $N = 1$ , we have a nystagmus model. If  $N$  is any other number the model represents the normal system. The value of GAIN is nominally 1 but it can be changed to a higher number to represent an excited or psychologically anxious subject or to a number less than one to simulate an unusually calm subject or the effects of various doses of barbiturates. The program shown in Appendix B is the program for this dual-mode model. With the use of Fig. 4.8, which has all the dummy variables





$$T_3 = .30$$

Fig. 4.5 Nystagmus waveshape

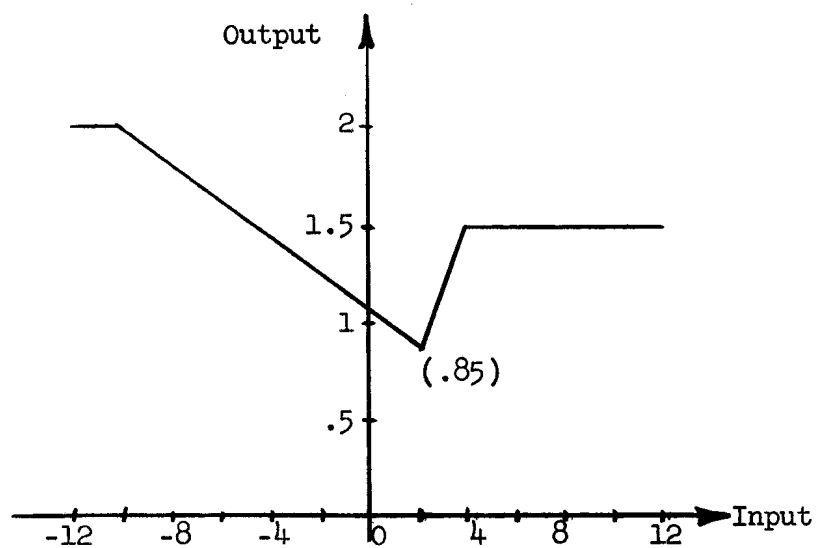


Fig. 4.6 Position non-linearity

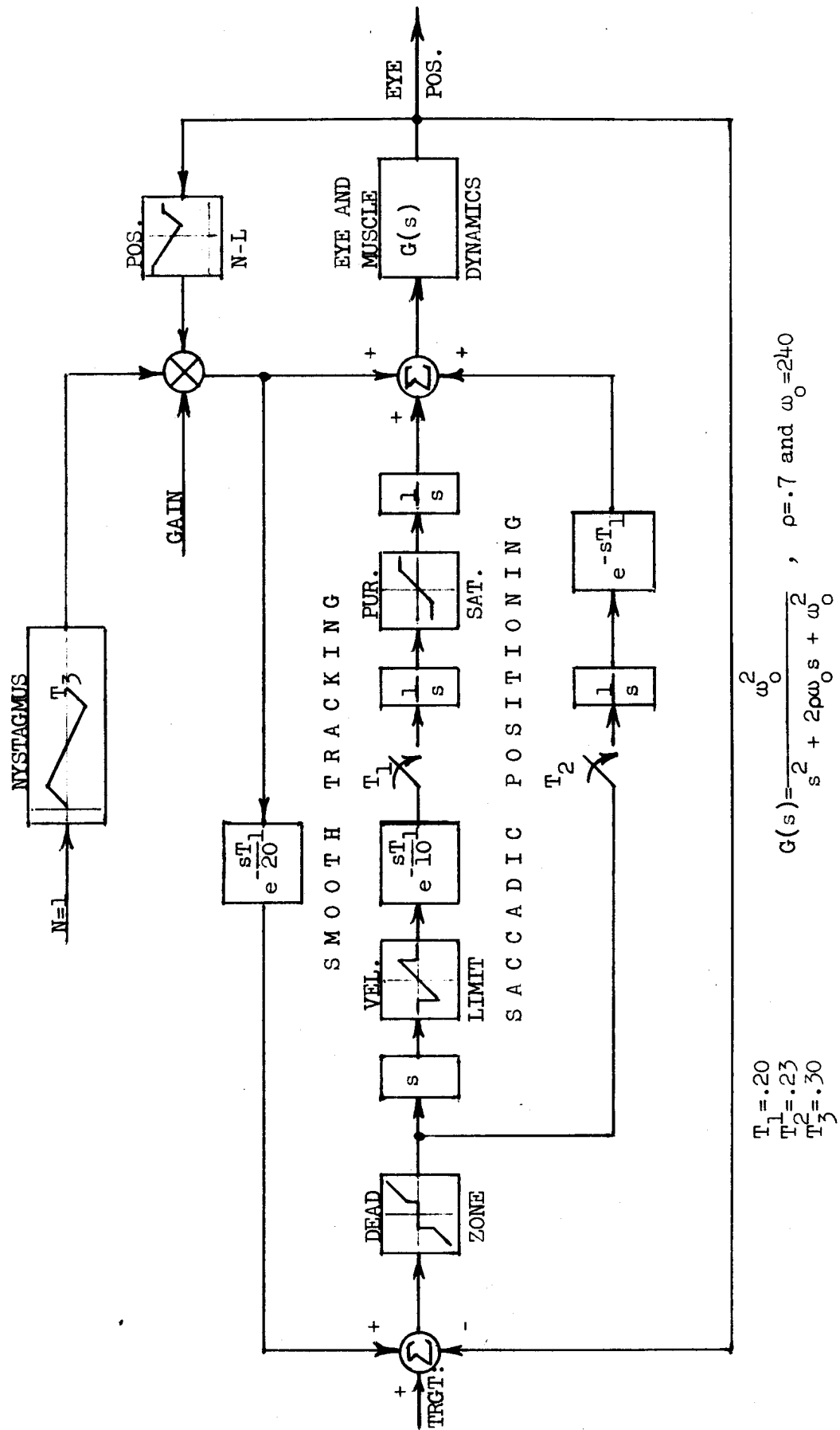


Fig. 4.7 Complete dual-mode model

used in the computer program labeled, the reader should have very little trouble tracing the operations step by step.



## CHAPTER V

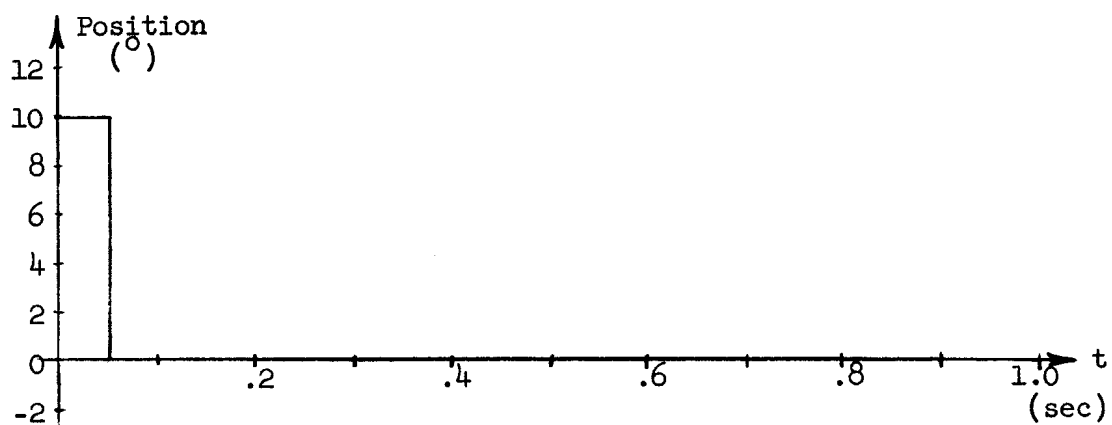
### RESULTS

#### 5.1 DATA FROM COMPUTER

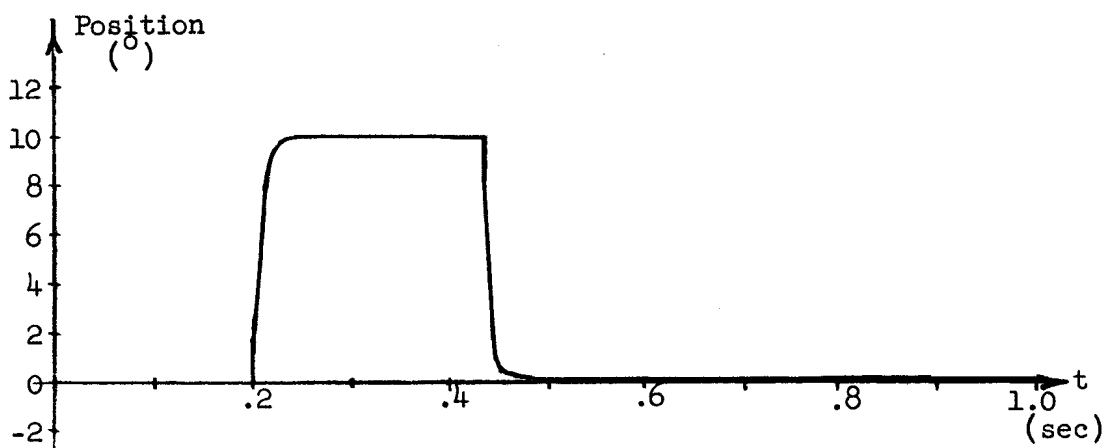
Since the Burroughs B-5500 is a digital computer, the output resulting from a particular "run" consists of a sheet containing a numerical printout of all the variables of interest at discrete time points. These time points are determined by programming in the "step size" that the computer is to take in solving the problem. For the dual-mode program, the variables printed out included the target (TRGT) and the (EYE) as well as nine other variables of interest (see lines 00006700 and 00006800 in the program shown in Appendix B). From this digital data, plots can be made of the variables as a function of time by fitting a curve to the data points. The choice of inputs is controlled by the value of variable FF punched into a data card (see lines 00002600 to 00003500 in the program).

##### 5.1.1 Pulse Responses

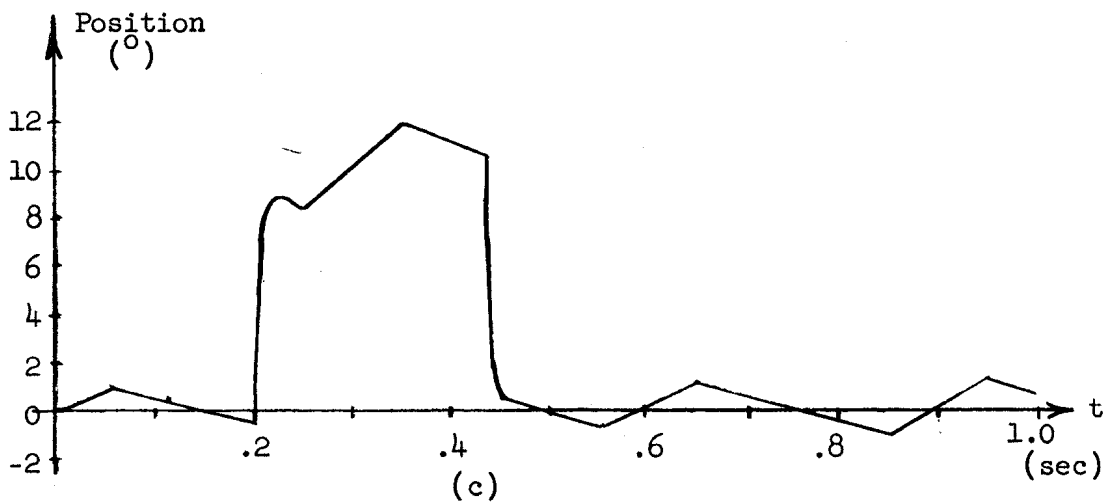
By setting  $FF = 0$  a pulse input is selected. Fig. 5.1 shows the responses to a pulse of height  $S = 10^0$  and width  $ST = 0.05$  sec. The normal response to this narrow pulse is a  $10^0$  pulse which begins 200 msec after the initial target motion. At  $t = 430$  msec the system returns the eye to the zero position. The rise-time exhibited by the model is 15 msec which can be varied by programming in new values of  $\rho$ . In the nystagmus mode the model reproduces this delayed and widened pulse along with the simulated nystagmus. The magnitude of



(a)



(b)



(c)

Fig. 5.1 Dual-mode pulse response (a) target (b) normal (c) nystagmus

the waveshape is a function of position as can be seen in Fig. 5.1 (c). The independence of the positioning mechanism and the nystagmus simulation can be seen by comparing the responses of the model in both modes.

#### 5.1.2 Step Responses

When  $FF = 1$  a step of magnitude  $S$  is the target. Fig. 5.2 shows the responses to a  $10^\circ$  step change in target position. These responses are again delayed changes in eye position. The eye then remains at the  $10^\circ$  position or, in the nystagmus mode, varies about it.

#### 5.1.3 Ramp Responses

At  $FF = 2$  the target is a constant velocity (SL) starting at  $t = 0$ . The values of SL are 10 and  $20^\circ/\text{sec}$  for Figures 5.3 and 5.4 respectively. The normal response to a ramp is a ramp of the same magnitude, delayed by 200 msec, which is followed at  $t = 430$  msec, by a corrective step to bring the eye on target. The nystagmus mode results in a response which has the same basic components plus the nystagmus waveshape. This results in an oscillation about the line described by the target motion.

#### 5.1.4 Parabolic Responses

By setting  $FF = 3$  a target with an acceleration equal to SL is generated. Fig. 5.5 shows the responses to a parabola where  $SL = 10$ . Inspection of Fig. 5.5 (b) reveals the following: (a) the model responds with a ramp starting at  $t = 200$  msec; (b) at  $t = 400$  msec the ramp is replaced by a ramp of greater magnitude since the target is constantly accelerating; (c) at  $t = 430$  msec the first corrective saccade occurs and the eye continues at the velocity assumed at  $t = 400$  msec; (d) at  $t = 600$  msec the velocity is again changed to a higher

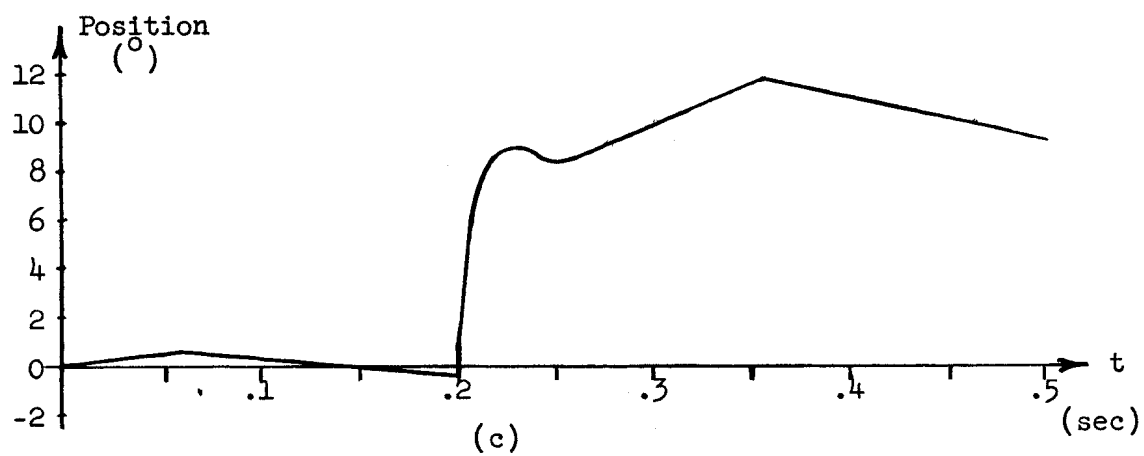
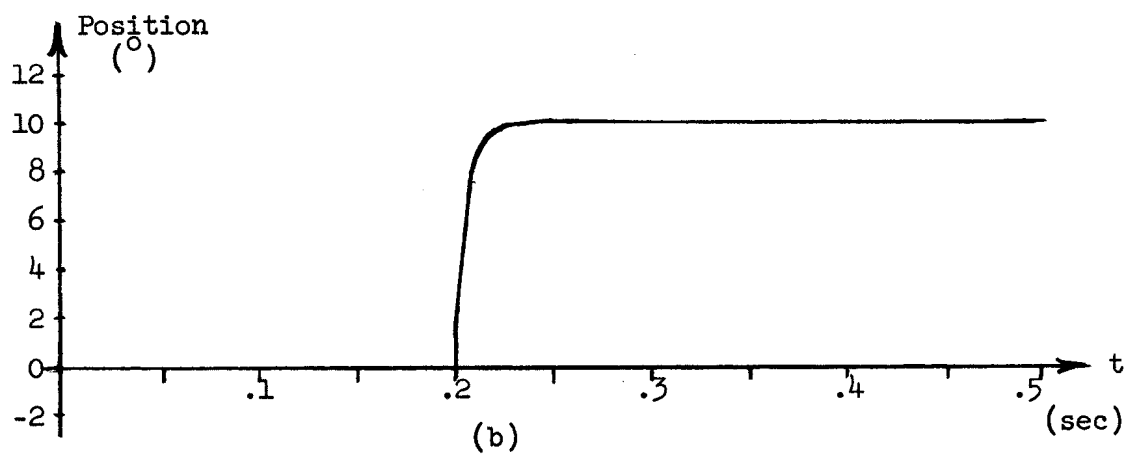
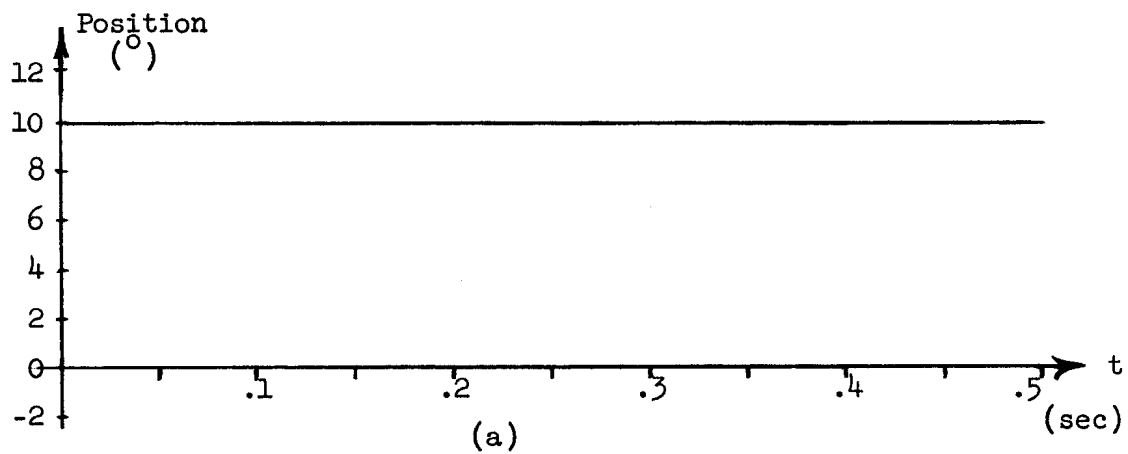


Fig. 5.2 Dual-mode step response (a) target (b) normal (c) nystagmus



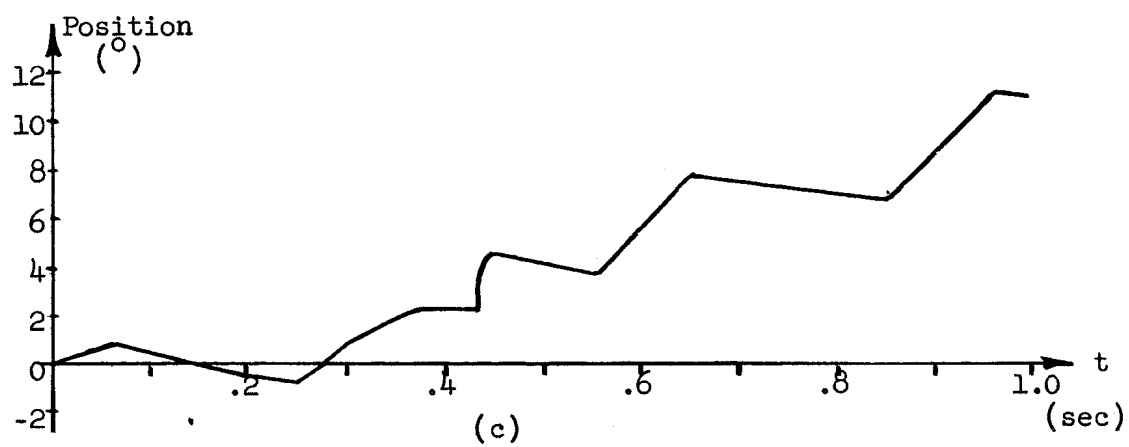
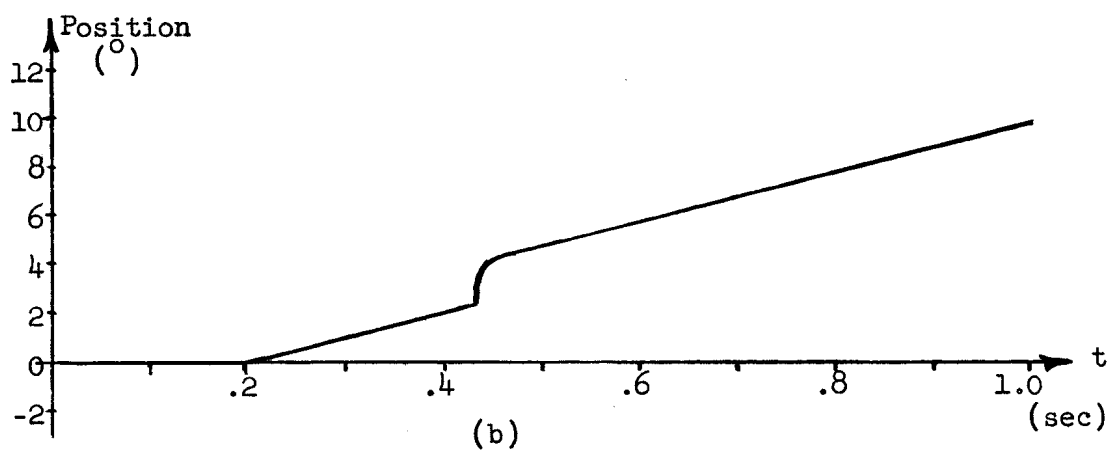
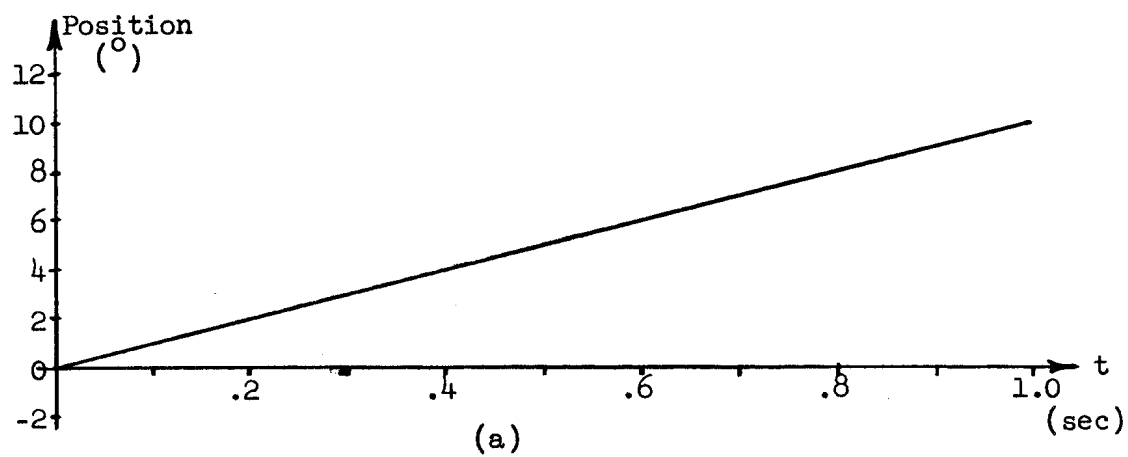


Fig. 5.3 Dual-mode ramp response ( $10^{\circ}/\text{sec}$ ) (a) target (b) normal (c) nystagmus

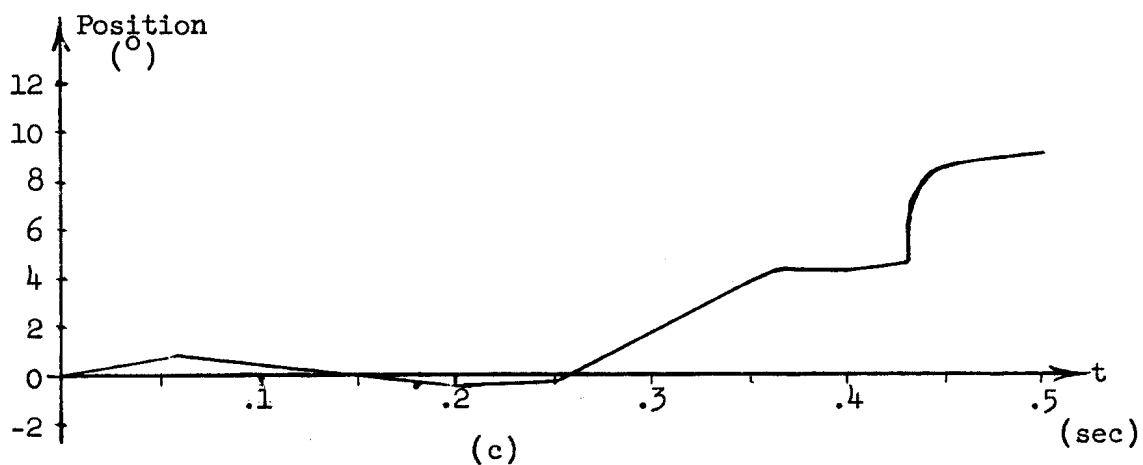
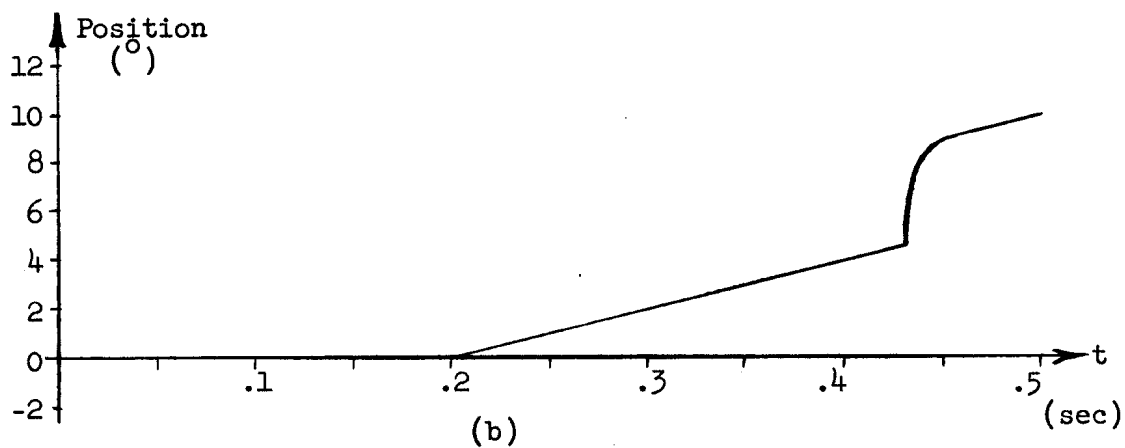
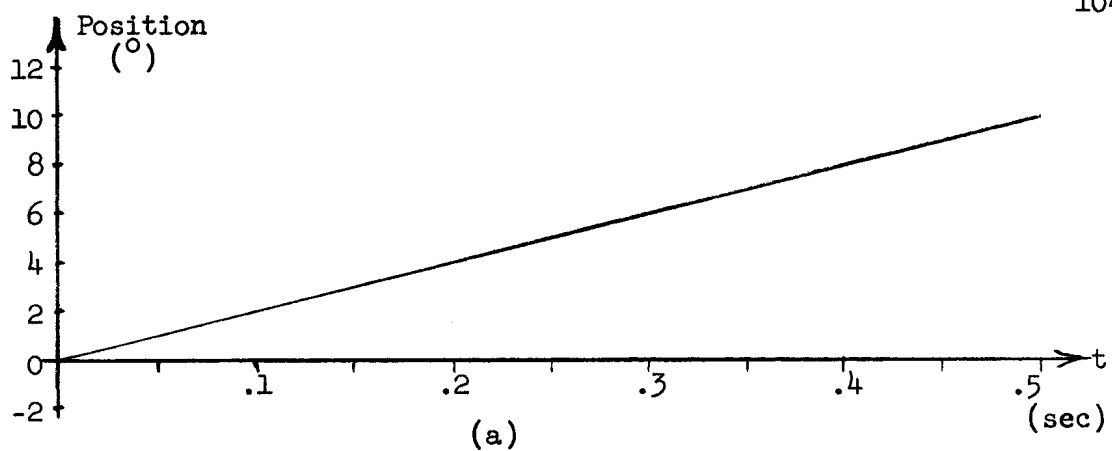


Fig. 5.4 Dual-mode ramp response ( $20^{\circ}/\text{sec}$ ) (a) target (b) normal (c) nystagmus

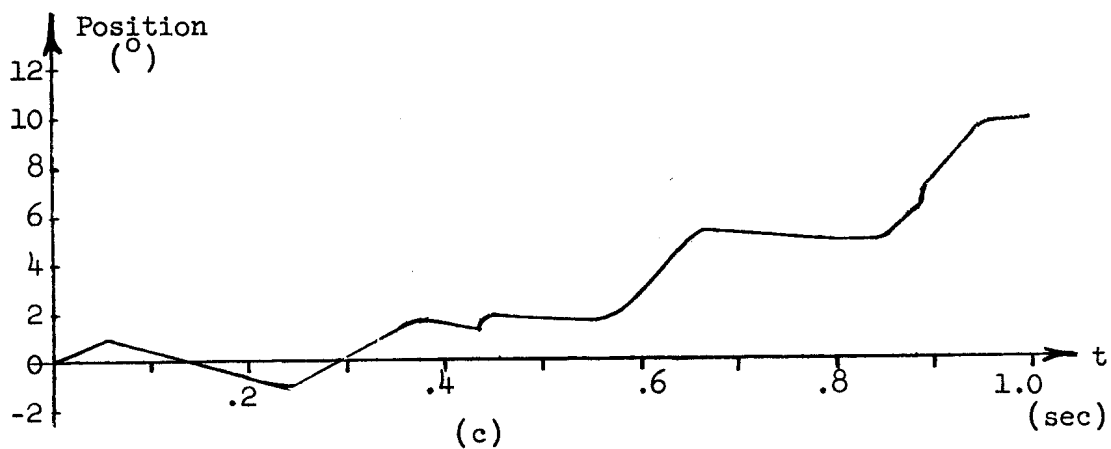
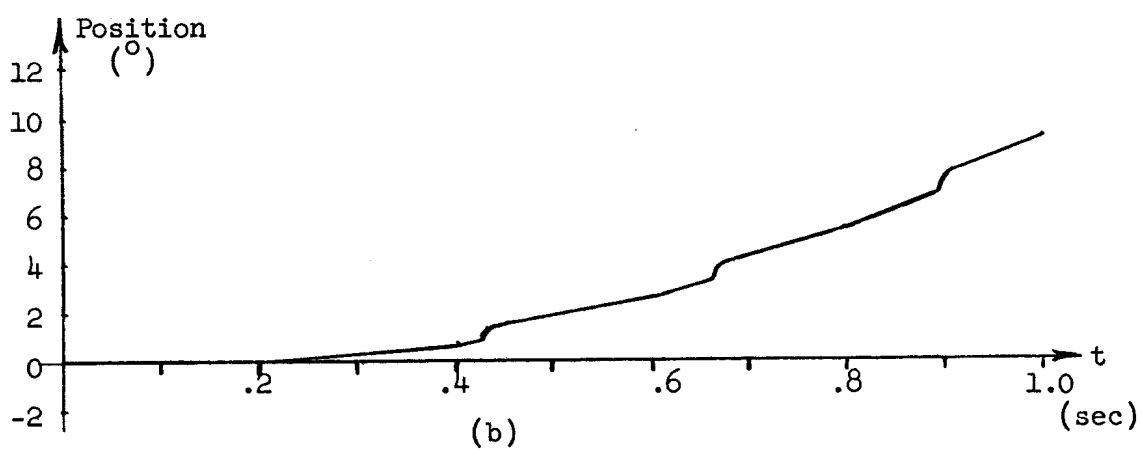
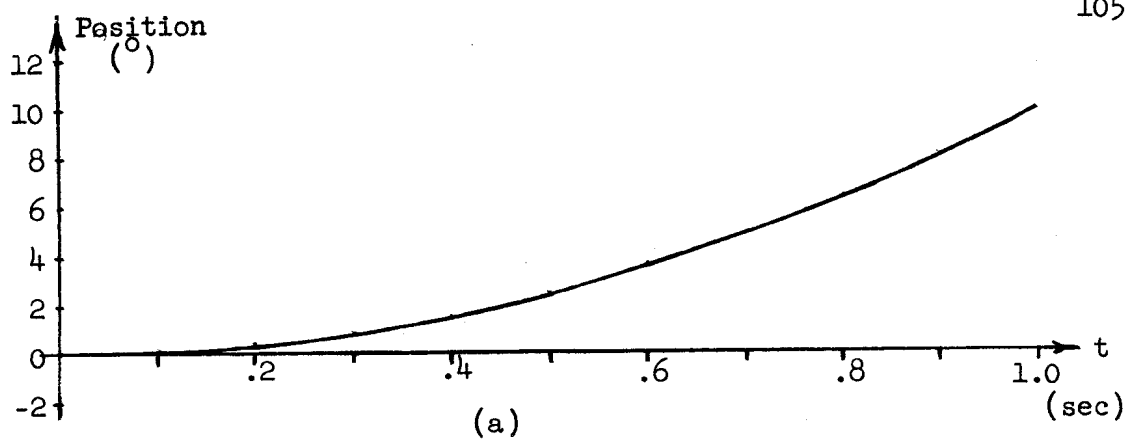


Fig. 5.5 Dual-mode parabolic response (a) target (b) normal (c) nystagmus

value and (e) at  $t = 660$  msec the second saccade occurs and the eye continues with the velocity assumed at  $t = 600$  msec. This continues as long as tracking is required of the model. The nystagmus response, shown in Fig. 5.5 (c) consists of exactly the same components but the oscillation masks them quite well. Two of the three corrective saccades are visible at 430 and 890 msec. Since the computer output consisted of other variables in addition to target and eye position, the equivalence of the two responses was verified. Note that the magnitude of the nystagmus grows as the eye deviates from the straight-ahead position.

#### 5.1.5 Step-Ramp Responses

$FF = 4$  provides a step-ramp input with the magnitude of the step equal to  $S$  and the slope of the ramp equal to  $SL$ . Fig. 5.6 shows the responses to a target where  $S = 10^\circ$  and  $SL = 20^\circ/\text{sec}$ . Both responses demonstrate the independence of the two forward paths in the model. Since the response to a step-ramp is merely the superposition of a step response and a ramp response no additional descriptive comments will be made. It should be noted that the return saccade in the nystagmus response was not large enough and that additional saccades were necessary, at  $t = 660$  msec and  $t = 890$  msec, to bring the eye on target. This is consistent with observed physiological responses.

### 5.2 DISCUSSION OF RESULTS

The data presented in section 5.1 conform to the theoretical responses presented in Chapter III which, in turn, were calculated from a model designed to reproduce the physiological responses that appear in Chapter II. This series of steps has led to the creation of a computer program which will simulate either a normal person's

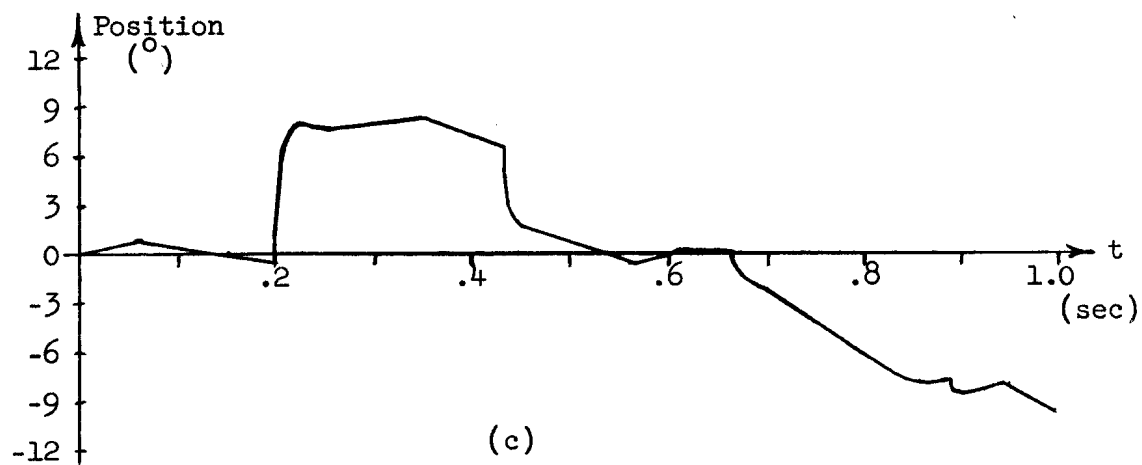
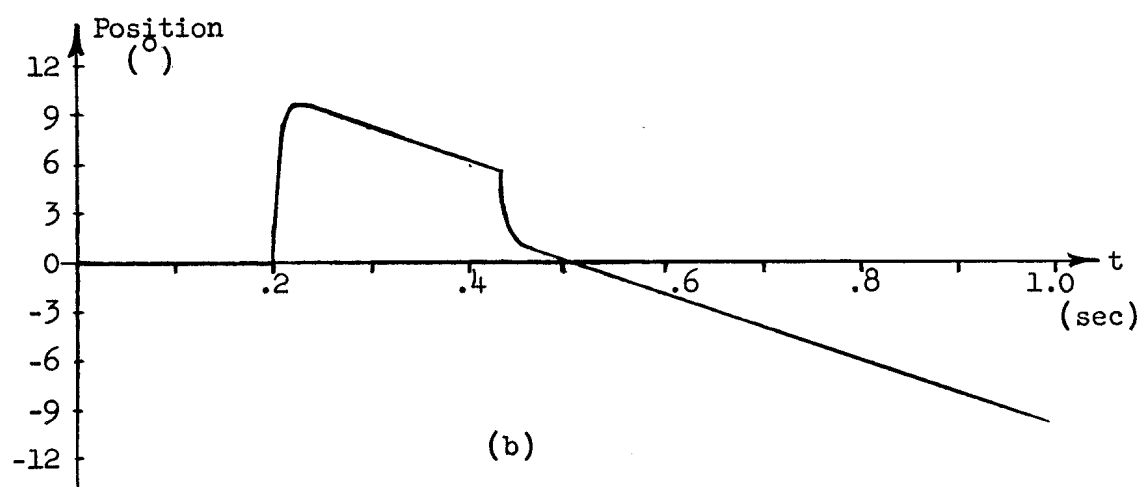
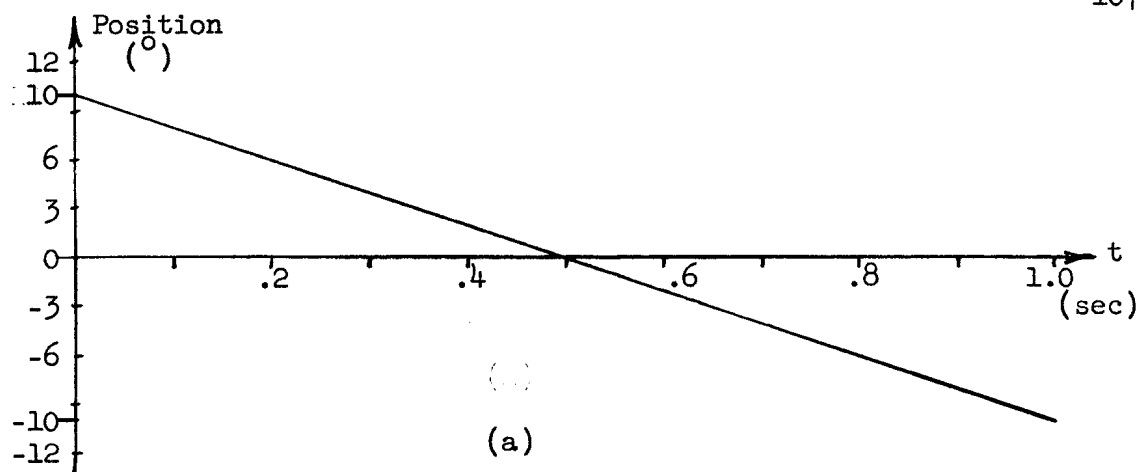


Fig. 5.6 Dual-mode step-ramp response (a) target (b) normal (c) nystagmus

responses to a transient input or those of a person with nystagmus. Provision is made in the program to vary the parameters of each input or to provide additional inputs. Also, several parameters of the model are variable so that the condition of the subject may be altered to observe the effects on the response to a particular input. A method was also presented which would yield the same model but would do so without the use of a differentiator, thereby extending the availability of this model.

The data collected from the subject with nystagmus verify the fact that this affliction has no effect on the ability to track moving targets. The responses of the two subjects were equal, both for transients and in the predictive mode, as evidenced by the gain and phase plots shown in Chapter II.

A mechanism was developed for nystagmus which is in agreement with what little is known about the true mechanism. This mechanism, as programmed into the model, makes use of proprioceptive position feedback at what would correspond to an unconscious level in the physiological system. The position information modifies the amplitude of the nystagmus waveform which is represented by an asymmetrical triangular wave whose slope in one direction (left) is twice that in the other (right). The source of this waveform is a center (unconscious) located in the mid-brain which is affected by higher centers (conscious) that reflect the subject's anxiety, tension, fatigue, etc. By varying the value of GAIN in the model, the effects of these factors can be studied and might lead to a better understanding of the many interconnecting pathways involved in this oscillatory condition.

The mechanism proposed for nystagmus appears to conform to the physiological facts. However, the model's validity as an input-output representation is not dependent on such a correspondence. The object of this study was to produce a black box whose transient responses duplicated those of the biological system. The synthesis of a model whose components were functionally equivalent to those of the living system was undertaken in the hope that such a model would contribute more information about the actual mechanisms involved in horizontal nystagmus.

## CHAPTER VI

### CONCLUSIONS

A dual-mode, sampled-data control system has been synthesized which reproduces the transient responses of both the normal horizontal tracking system and the system with horizontal nystagmus. As stated in the introduction, the object of this study was to gather physiological data from both the normal and nystagmus-afflicted subject, compare these data, postulate a mathematical model which would simulate the responses of both systems to transient inputs and finally, to synthesize this model on a computer and thus, provide a physical vehicle for future studies of both the normal system and of the nystagmus condition itself. The model developed satisfies these requirements as evidenced by the data of Chapter V.

#### 6.1 LIMITATIONS OF THE MODEL

Intelligent use of this model, and most other models, is dependent upon a ~~th~~<sup>o</sup>rough understanding of its theory of operation and, more importantly, the basic assumptions that went into its postulation. The first limitation is a result of the attempt to model a living system with an inherently deterministic model. Since the biological system is not a deterministic one, the responses to a given input are not always exactly the same but vary about some typical response both in time course and in magnitude. By constructing a deterministic model to represent a non-deterministic system we have eliminated this random variation about some "normal" response. The computer output to a given input will always be



the same in every detail. This discrepancy does not detract from the model's usefulness in studying the expected responses of either subject to a given target input.

Two other limitations are due to the methods chosen to represent the sampling mechanism. First, the sampling rates were fixed at some nominal values instead of being allowed to vary randomly about some mean value, as they do in the biological system. Should the variations about a norm be desired, the program could be re-written so that the values of  $T_1$  and  $T_2$  were not fixed but varied randomly about some mean values. The second limitation is due to the synchronization of the samplers with the onset of target motion. Physiological evidence points to non-synchronous sampling of the two paths. These two limitations have the effect of yielding normalized responses to given inputs instead of the observed responses where the time course of various components of a response may not always be the same (i.e., one time a saccade may precede a velocity change and another time, for the same input, the velocity change may occur first).

Another limitation is due to the piecewise-linear approximations used for the non-linearities of the system. The effects of these straight lines with well-defined break points are minimized by the many integrators in the system so that this is a minor limitation when one is looking at gross responses to transient inputs.

The piecewise-linear representation of the nystagmus waveform is another place where a quasi-random variation in waveshape was normalized and represented by a triangular wave of constant shape and frequency. The elimination of the small notch in the slow phase represents a loss

of information, in a sense, since it may be due to factors important in the generation of this oscillation. However, the consideration of the small movements which accompany large responses was not a part of this study and the data taken did not yield enough information to enable the author to attempt to postulate a mechanism for such movements.

The model is also limited, by design, to the tracking of targets whose excursions lie within  $\pm 10^\circ$ . In this region the non-linear effects of movement limitations on the eye are not present.

## 6.2 FUTURE WORK

In spite of the above assumptions and limitations, the model does adequately represent the systems under study and can be a good foundation for elaboration and modification to make it suitable for future studies. One reason the model does a good job of representing the biological system is the incorporation of different sampling periods for the two paths. Although the periods are fixed and the rates synchronized, the refractory periods of the pursuit system (200 msec) and of the saccadic system (230 msec) compare well with the physiological data in both absolute and relative magnitudes. These data reveal that the refractory period of the pursuit system is slightly less than that of the saccadic system. Maintaining this difference in sampling rates and delaying the exact instant of sampling until a saccade is completed has resulted in a model whose methods as well as results agree with the known physiology.

Further study of the nystagmus waveshape and its relation to eye position (if any) appears to be a promising endeavor. If, indeed, there is a relationship between eye position and the size, shape or position of a notch in the slow phase of the nystagmus, the model could be

programmed to reflect this and it might yield new insight into the factors responsible for the disorder.

In conclusion, the study of an abnormality present in a complex biological control system, by methods previously alien to the medical field, has shed some new light on an old problem and perhaps has brought its solution a step closer.

## BIBLIOGRAPHY

1. Francis, C.C., Introduction to Human Anatomy, ed. 3, C.V. Mosby Co., St. Louis, 1959.
2. McNaught, A.B. and Callander, R., Illustrated Physiology, ed. 1, rev. rep., Williams and Wilkins Co., Baltimore, 1965.
3. Park, R.S. and Park, G.E., The center of oculomotor rotation in the horizontal plane, *Am. J. Physiol.*, 104:545, 1933.
4. Westheimer, G., Mechanisms of saccadic eye movements, *A.M.A. Arch. Opth.*, 52:710, 1954.
5. Cooper, S. and Eccles, J.C., Isometric responses of mammalian muscles, *J. Physiol.*, 69:377, 1930.
6. Dodge, R., Five types of eye movements in the horizontal meridian plane of the field of regard, *Am. J. Physiol.*, 8:307, 1903.
7. Vossius, G., Das System der Augenbewegung, *Ztschr. Biol.*, 112:27, 1960.
8. Vossius, G., Der sogenannte "innere" Regelkreis der Willkurbewegung, *Kybernetik*, 1:28, 1961.
9. Rashbass, C., The relationship between saccadic and smooth tracking eye movements, *J. Physiol.*, 159:326, London, 1961.
10. Ludvigh, E. and Miller, J.W., Study of visual acuity during the ocular pursuit of moving test objects, I, *J. Opt. Soc. Am.*, 48:799, 1958.
11. Miller, J.W., Study of visual acuity during the ocular pursuit of moving test objects, II, *J. Opt. Soc. Am.*, 48:803, 1958.
12. Young, L.R., A sampled data model for eye tracking movements, *Sc.D. Thesis, M.I.T.*, 1962.
13. Shackel, B., A pilot study in electro-oculography, *Brit. J. Opth.* 44:89, 1960.
14. Holway, A.H. and Boring, E.G., Determination of apparent visual size with distance variant, *Am. J. Psychol.*, 54:21, 1941.
15. Judd, C.H., Photographic records of convergence and divergence, *Psychol. Rev. Mono.* 8, Suppl., 1:370, 1907.

16. Westheimer, G. and Mitchell, A.M., Eye movement responses to convergence stimuli, *A.M.A. Arch. Opth.*, 55:848, 1956.
17. Duke-Elder, W.S., Textbook of Ophthalmology, vol. I, Henry Kimpton, London, 1932.
18. Barlow, H.B., Eye movements during fixation, *J. Physiol.*, 116:290, 1952.
19. Dichburn, R.W. and Ginsborg, B.L., Involuntary eye movements during fixation, *J. Physiol.*, 119:1, 1953.
20. Cornsweet, T.N., Determination of the stimuli for involuntary drifts and saccadic eye movements, *J. Opt. Soc. Am.*, 46:987, 1956.
21. Lord, M.P. and Wright, W.D., Eye movements during monocular fixation, *Nature*, 162:25, 1948.
22. Hyde, J.E., Some characteristics of voluntary human ocular movements in the horizontal plane, *Am. J. Opt.*, 48:85, 1959.
23. Riggs, L.A. and Ratliff, F., Visual acuity and the normal tremor of the eyes, *Sci.*, 114:17, 1951.
24. Riggs, L.A., Armington, J.C. and Ratliff, F., Motions of the retinal image during fixation, *J. Opt. Soc. Am.*, 44:315, 1954.
25. Pritchard, R.M., Heron, W. and Hebb, D.O., Visual perception approached by the method of stabilized images, *Canad. J. Psychol.*, 14:67, 1960.
26. McIntyre, A.K., Quick component of nystagmus, *J. Physiol.*, 97:8, 1939.
27. Bender, M.B., ed., The Oculomotor System, Harper and Row, New York, 1964.
28. Cogen, D.G., Neurology of the Ocular Muscles, Charles C. Thomas, Springfield, Ill., 1948.
29. Ellis, P.P., Practical evaluation of nystagmus, *Med. Times*, 86(11):1329, 1958.
30. Rucker, C.W., Nystagmus, *Am. J. Opth.*, 36:250, 1953.
31. Walsh, F.B., Clinical Neuro-ophthalmology, Williams and Wilkins Co., Baltimore, 1947.
32. Crampton, G.H. and Schwam, W.J., Effects of arousal reaction on nystagmus habituation in the cat, *Am. J. Physiol.*, 200:29, 1961.

33. Tonnes, J.F., Beschreibung eines Drehstules mit unterschwelliger Anlaufbeschleunigung, J. Psychol. Neurol., 44:468, 1932.
34. Dohlman, G., Towards a method for quantitative measurement of the functional capacity of the vestibular apparatus, Acta Oto-laryng., 23:50, 1935.
35. Gray, H., Anatomy of the Human Body, 100th yr., 27th ed., Goss, C.M., ed., Lea and Febiger, Phil., 1962.
36. Davson, H., The Eye, vol. 3, Acad. Press, New York, 1962.
37. Warwick, R., Oculomotor organization, Ann. Roy. Coll. Surg., Eng., 19:36, 1956.
38. Barlow, H.B. and Hill, R.M., Selective sensitivity to direction of movement in ganglion cells of the rabbit retina, Sci., 139:412, 1963.
39. Adler, F.E., Physiology of the Eye, 3rd ed., C.V. Mosby, St. Louis, 1959.
40. Wiesel, T.N., Integrative action in the cat's lateral geniculate body, J. Physiol., 155:385, 1961.
41. Stark, L., Kupfer, C., Young, L.R., Physiology of the visual control system, NASA CR-238, June, 1965.
42. Hines, M., Recent contributions to localization of vision in central nervous system, A.M.A. Arch Opth., 28:913, 1942.
43. Holmes, G., Cerebral integration of the ocular movements, Brit. Med. J., 2:107, 1938.
44. Whitteridge, D., Central control of eye movements, in Handbook of Physiology, vol. 2, Sect. 1, Amer. Physiol. Soc., pp. 1089-1109, 1960.
45. Helmholtz, H. von, Handbuch der Physiologischen Optik, 1st ed., Leipzig:Voss, 1867.
46. Helmholtz, H. von, Helmholtz's treatise on physiological optics, Eng. trans. by Southall, J.P.C., vol. 3, J. Opt. Soc. Am., Menasha, Wisc., 1925.
47. Brindley, G.S., The discrimination of after-images, J. Physiol., 147:194, 1959.
48. Merton, P.A., The accuracy of directing eyes and the hand in the dark, J. Physiol., 156:555, 1961.

49. Brindley, G.S. and Merton, P.A., The absence of position sense in the human eye, *J. Physiol.*, 153:127, 1960.
50. Irvine, S.E., Histology of the extra-ocular muscles, *A.M.A. Arch. Opth.*, 15:847, 1936.
51. McCouch, G.P. and Adler, F.H., Extra-ocular reflexes, *Am. J. Physiol.*, 100:78, 1932.
52. Hyde, J.E. and Davis, L.M., Extra-ocular proprioception in electrically induced eye movements, *Am. J. Physiol.*, 198(5):945, 1960.
53. Irvine, S.R. and Ludvigh, E., Is ocular proprioceptive sense concerned in vision?, *A.M.A. Arch. Opth.*, 15:1037, 1936.
54. Ludvigh, E., Possible role of proprioception in the extra-ocular muscles, *A.M.A. Arch Opth.*, 48:436, 1952.
55. Ludvigh, E., Control of ocular movements and visual interpretation of environment, *A.M.A. Arch. Opth.*, 48:442, 1952.
56. James, W., The Principles of Psychology, vol. 2, Macmillan, London, p. 507, 1890.
57. Sherrington, C.S., Further experimental note on the correlation of action of antagonistic muscles, *Proc. Roy. Soc. Lond.*, 53:407, 1893.
58. Sherrington, C.S., Further note on the sensory nerves of muscles, *Proc. Roy. Soc. Lond.*, 61:247, 1897.
59. Sherrington, C.S., Further note on the sensory nerves of the eye muscles, *Proc. Roy. Soc. Lond.*, 64:120, 1898.
60. Sherrington, C.S., The muscular sense, in Schafer, E.A., Textbook of Physiology, Edinburgh and Pentland, London, p. 1004, 1900.
61. Tozer, F.M. and Sherrington, C.S., Receptors and afferents in the third, fourth and sixth cranial nerves, *Proc. Roy. Soc. Brit.*, 82:450, 1910.
62. Sherrington, C.S., Observations on the sensual role of the proprioceptive nerve supply of the extrinsic ocular muscles, *Brain*, 41:337, 1918.
63. Cooper, S. and Daniel, P.M., Muscle spindles in human extrinsic eye muscles, *Brain*, 72:1, 1949.
64. Daniel, P.M., Spiral nerve endings in extrinsic eye muscles of man, *J. Anat.*, 80:189, 1946.

65. Cooper, S., Daniel, P.M. and Whitteridge, D., Afferent impulses in the oculomotor nerve, from the extrinsic eye muscles, *J. Physiol.*, 113:463, 1951.
66. Cooper, S., Daniel, P.M. and Whitteridge, D., Nerve impulses in the brain-stem of the goat. Responses with long latencies obtained by stretching the extrinsic eye muscles, *J. Physiol.*, 120:491, 1953.
67. Cooper, S., Daniel, P.M. and Whitteridge, D., Muscle spindles and other sensory endings in the extrinsic eye muscles; the physiology and anatomy of these receptors and of their connexions with the brain-stem, *Brain*, 78:564, 1955.
68. Wolter, J.R., Morphology of the sensory nerve apparatus in the striated muscle of the human eye, *A.M.A. Arch. Opth*, 53:201, 1955.
69. Breinin, G.M., The Electrophysiology of Extra-ocular Muscle, 1st ed., Univ. of Toronto Press, Toronto, 1962.
70. Christman, E. and Kupfer, C., Proprioception in extra-ocular muscle, *A.M.A. Arch. Opth.*, 69:824, 1963.
71. Duke-Elder, W.S., Textbook of Ophthalmology, vol. IV, Henry Kimpton, London, 1932.
72. Francois, J., Heredity in Ophthalmology, C.V. Mosby, St. Louis, 1961.
73. Scheinfeld, A., The New You and Heredity, J.B. Lippincott, New York, 1950.
74. Young, L.R., Measuring eye movements, *Am. J. Med. Elec.*, 2,4:300, 1963.
75. Carmichael, L. and Dearborn, W.F., Reading and Visual Fatigue, Houghton Mifflin Co., Boston, 1947.
76. Shackel, B., Review of the past and present in oculography, *Med. Elec., Proc. of the Sec. Int'l. Conf.*, Iliffe and Sons, London, p. 57, 1960.
77. Cobb, P.W. and Moss, F.K., Eye fatigue and its relation to light and work, *J. Franklin Inst.*, 200:239, 1925.
78. Lamansky, S., Bestimmung der Winkelgeschwindigkeit der Blickbewegung, respective Augenbewegung, *Pfluger's Arch. ges. Physiol.*, 2:418, 1869.
79. Delabarre, E.B., A method of recording eye movements, *Am. J. Psychol.*, 9:572, 1898.



80. Schackwitz, A., Apparat zur Aufzeichnung der Augenbewegungen beim zusammenhagen Lesen (Nystagmograph), Ztschr. Psychol. u. Physiol. o. Sinnesorg., 63:442, 1912.
81. Miles, W.R., Reliability of measurements of the steady polarity potential of the eye, Proc. Nat. Acad. Sci., 25:128, 1939.
82. Totten, E., Eye-spots for photographic records of eye-movements, J. Comp. Psychol., 6:287, 1926.
83. Dodge, R. and Cline, T.S., The angle velocity of eye movements, Psychol. Rev., 8:145, 1901.
84. Jasper, H.H. and Walker, R.Y., The Iowa eye-movement camera, Sci., 74:291, 1931.
85. Taylor, E.A., Controlled Reading, Univ. of Chicago Press, Chicago, 1937.
86. Tinker, M.A., Apparatus for recording eye-movements, Am. J. Psychol., 43:115, 1931.
87. Riggs, L.A., Ratliff, R., Cornsweet, J.C. and Cornsweet, T.N., The disappearance of steadily fixated test-objects, J. Opt. Soc. Am., 43:495, 1953.
88. Byford, G.H. and Stuart, H.G., An apparatus for the measurement of small eye movements, J. Physiol., 159:2p., November, 1961.
89. Schott, E., Uber die Registrierung des Nystagmus und anderer Augenbewegungen vermittels des Saitengalvanometers, Deutsches Arch. Klin. Med., 140:79, 1922.
90. Mowrer, O.H., Ruch, T.C. and Miller, N.E., The corneo-retinal potential difference as the basis of the galvanometric method of recording eye movements, Am. J. Physiol., 114:423, 1936.
91. Kris, E.C., A technique for electrically recording eye position, WADC technical reports 58-660, Research Laboratory of Electronics, M.I.T., December, 1958.
92. Miles, W.R., An early eye movement photograph, Psychol. Monogr., 47:xxxi, 1936.
93. Halstead, W.C., A method for the quantitative recording of eye movements, J. Psychol., 6:177, 1938.
94. Guedry, F.E., Jr. and Lauver, L.S., The oculomotor and subjective aspect of the vestibular reaction during prolonged constant angular acceleration, Report No. 438, U.S. Army Medical Research Laboratory, Fort Knox, Kentucky, 1960.

95. Cornsweet, T.N., New technique for the measurement of small eye movements, *J. Opt. Soc. Am.*, 48:808, 1958.
96. Smith, W.M. and Warter, P.J., Photoelectric technique for measuring eye movements, *Sci.*, 130:1248, 1959.
97. Torok, N., Guillemin, V. and Barnothy, J.M., Photoelectric nystagmography, *Ann. Otol., Rhin. and Laryng.*, 60:917, 1951.
98. Richter, J.R., *Principes de la Photo-Electro-Nystagmographie*, *Rev. Neurol.*, 94:138, 1956.
99. Doescher, R., A tracking device for measurement of the dynamic response of the human eye, Senior Paper, Department E.E., Univ. of Wyoming, Laramie, 1966.
100. Fender, D.H. and Nye, P.W., An investigation of the mechanisms of eye movement control, *Kybernetik*, 1,2:81, 1961.
101. Westheimer, G., Eye movement responses to horizontally moving visual stimuli, *Arch. Opth.*, 52:932, 1954.
102. Pavlidis, T., A modification of a proposed model for the eye-tracking movements, *IEEE TRANS: BME*, BME-11:52, Jan/Ap., 1964.
103. Warnmeunde, J.T., Model of the eye tracking mechanism with random sampling, *Eng. in Med. and Biol.*, *Proc. of the 17th Ann. Conf.*, Session 14, *Biol. Systems III*, 14.7:102, 1964.
104. Miller, J.E., Electromyographic pattern of saccadic eye movements, *Am. J. Opth.*, 46(5,2):183, 1958.
105. Robinson, D.A., The mechanics of human saccadic eye movements, *J. Physiol.*, 174:245, 1964.
106. Alpern, M., The extra-ocular muscles, in The Eye, vol. 3, p. 159, ed. Davson H., Academic Press, London, 1962.
107. Bjork, A., The electromyogram of the extra-ocular muscles in optokinetic nystagmus and in reading, *Acta. Opth.*, Kbh., 33:437, 1955.
108. Cook, G., Control system study of the saccadic eye movement system, Sc.D. Thesis, M.I.T., EE., June, 1965.
109. Young, L.R., Zuber, B.L. and Stark, L., Visual and control aspects of saccadic eye movements, *NASA CR-564*, Sept., 1966.
110. Kuo, B.C., Analysis and Synthesis of Sampled-Data Control Systems, Prentice-Hall, Englewood Cliffs, N.J., 1964.

111. Jury, E.I., Sampled-Data Control Systems, J. Wiley, London, 1958.
112. Ragazzini, J.R. and Franklin, G.F., Sampled-Data Control Systems, McGraw-Hill, New York, 1958.
113. Dodge, R., The illusion of clear vision during eye movement, Psychol. Bull., 2:193, 1905.
114. Dodge, R., Visual perception during eye movement, Psychol. Rev., 7:454, 1900.
115. Holt, E.B., Eye movement and central anesthesia, Harvard Psychol. Studies, 1:3, 1903.
116. Dichburn, R.W., Eye movements in relation to retinal action, Optica Acta, 1:171, 1955.
117. Rucker, C.W., Hereditary nystagmus occurring as a sex-linked character recessive in the female, Am. J. Opth., 29:1534, 1946.
118. Cox, R., Congenital head-nodding and nystagmus, report of a case, Arch. Opth, 15:1032, 1936.
119. Harris, W., Symposium on nystagmus, Proc. Roy. Soc. Med., 7:Part 2, Sec. Neurol., Opth. and Otol., 1914.
120. Maddox, E.E., Symposium on nystagmus, Proc. Roy. Soc. Med., 7:Part 2, Sec. Neurol., Opth. and Otol., 1-xxix xli, 1914.
121. Taylor, J., Symposium on nystagmus, Proc. Roy. Soc. Med., 7:Part 2, Sec. Neurol., Opth. and Otol., 1914.
122. Forssman, B., Study of congenital nystagmus, Acta Oto-Lary., 57(5):427, 1964.
123. Forssman, B., Vestibular reactivity in cases of congenital nystagmus and blindness, Acta Oto-Lary., 57(6):539, 1964.
124. Bergmann, F., Lachmann, J., Monnier, M. and Krupp, P., Central nystagmus, III. Functional correlations of mesodiencephalic nystagnogenic center, Am. J. Physiol., 197:454, 1959.
125. Pasik, P., Pasik, T. and Krieger, H.P., Effects of cerebral lesions upon optokinetic nystagmus in monkeys, J. Neurophysiol., 22:297, 1959.
126. Gutman, J., Bergmann, F., Chaimovitz, M. and Costin, A., Nystagmus evoked by stimulation of the optic pathways in the rabbit, Exp. Neurol., 8(2):132, 1963.
127. Fox, J.C. and Holmes, G., Optic nystagmus and its value in the localization of cerebral lesions, Brain, 49:333, 1926.

128. Gejrot, T., The influence of xylocaine on induced and spontaneous nystagmus, *Pract. Oto-Rhino-Laryng.*, 25(6):361, 1963.
129. Gorman, W.F. and Brock, S., Nystagmus: Its mechanisms and significance, *Am. J. Med. Sc.*, Sec. 5, 220:225, 1950.
130. Greig, D.M., Oxycephaly, *Edinburgh Med. J.*, 33:189, 280, 357, 1926.
131. Bender, M.B. and O'Brien, F.H., The influence of barbiturates on various forms of nystagmus, *Am. J. Opth.*, 29:1541, 1946.
132. Anderson, J.R., Ocular Vertical Deviations and Nystagmus, J.B. Lippincott, Co., Phil., 1959.

# APPENDIX A

## THE Z-TRANSFORM

This appendix will introduce the z-transform, describe its use and list several simple transform pairs which were useful in the analysis presented in Chapter III. The reader who is unfamiliar with this area is referred to the texts cited in Chapter III for a complete treatment of this method of solution to sampled-data control systems.

When a signal  $e(t)$  is sampled by means of an impulse train modulator  $\delta_T(t)$ , where,

$$\delta_T(t) = \sum_{n=-\infty}^{\infty} \delta(t-nT) \quad (A-1)$$

the resulting signal  $e^*(t)$  is given by,

$$e^*(t) = e(t) \delta_T(t) \quad (A-2)$$

$$= \sum_{n=-\infty}^{\infty} e(nT) \delta(t-nT) \quad (A-3)$$

and, since  $e(t) = 0$  for  $t$  less than 0,

$$e^*(t) = \sum_{n=0}^{\infty} e(nT) \delta(t-nT) \quad (A-4)$$

the Laplace transform of  $e^*(t)$  is

$$E^*(s) = \sum_{n=0}^{\infty} e(nT) e^{-nTs} \quad (A-5)$$

The function,  $E^*(s)$ , contains the factor  $e^{-Ts}$ , which makes it a non-algebraic equation. This introduces difficulties in taking the inverse transform to return to the time domain. To alleviate this problem a change of variables is introduced as follows:

$$z = e^{-Ts} \quad (A-6)$$

$$\text{or} \quad s = -\ln z / T \quad (A-7)$$

thus,  $E^*(s)$  becomes the function of  $z$ ,  $E(z)$ . This is called the  $z$ -transform of  $e(t)$  and is equivalent to the Laplace transform of  $e^*(t)$ . Substitution of equation (A-6) into equation (A-5) yields,

$$E(z) = \sum_{n=0}^{\infty} e(nT)z^n \quad (A-8)$$

which is a rational function of  $z$  making it easy to take the inverse transform and return to a function of time.

A disadvantage of the  $z$ -transform is that it contains information about the corresponding time function,  $e(t)$ , only at the sampling instants  $nT$ . Thus, an  $e(t)$  has a unique  $E(z)$  but for a given  $E(z)$  there are an infinite number of functions of time. An example of this would be a sinusoid and a d-c value of zero. If the period of the sinusoid is  $T$  and we sampled both time functions with a sampling period of  $T/2$ , the resulting  $e^*(t)$  would be exactly the same for each and the knowledge of  $E(z)$  would not be sufficient to define the corresponding time function. Since the discrete  $E(z)$  may represent any continuous function  $e(t)$  that has the same values at the sampling

instants as  $e^*(t)$ , we are faced with the problem of loss of information necessary to reconstruct the signal. Looking back at equation (A-5) for  $E^*(s)$ , one can see that it may be re-written as

$$E^*(s) = \frac{1}{T} \sum_{k=-\infty}^{\infty} E(s + jk\omega_0), \text{ where } \omega_0 = \frac{2\pi}{T} \quad (\text{A-9})$$

This is a periodic function with a frequency,  $\omega_0$ , which is the sampling frequency. What this transformation does in the frequency domain is to transform a continuous spectrum into an infinite series where the original spectrum is repeated at intervals of  $\omega_0$ . This being the case, one can see that the original  $E(s)$  must be band-limited with no component higher than  $\omega_0/2$  for the information to be recovered. Only under these conditions does  $E(z)$  carry all the information present in the Laplace transform,  $E(s)$  and, by means of an ideal filter,  $e(t)$  can be recovered. Table A.1 shows some of the elementary transform pairs which were useful in the analysis of the model.

TABLE A.1

Transform Pairs		
LAPLACE TRANSFORM $E(s)$	TIME FUNCTION $e(nT)$	Z-TRANSFORM $E(z)$
$e^{-kTs}$	$\delta(n-k)T$	$z^k$
$\frac{1}{s}$	1	$\frac{1}{1-z}$
$\frac{1}{s^2}$	$nT$	$\frac{Tz}{(1-z)^2}$

The other aid to solution of sampled-data systems is the composite flow graph. This is obtained by combining the continuous flow graph and the sampled flow graph. Once this is done, the transfer function may be written by inspection using Mason's gain formula. The mechanics of this operation are presented very clearly in Kuo's text (110).



## APPENDIX B

### THE DUAL-MODE PROGRAM

The following pages are copies of the Analog-Algol program used to simulate the dual-mode model on a Burroughs B-5500 digital computer. The program appears as it was printed out by the computer. On the left are the command statements and on the right the card identification numbers for the computer.

MONDAY, JUNE 26, 1967                      MRL ALGOL VERSION OF 10/20/66

```

BEGIN
COMMENT A.LAVI - BIOLOGICAL PROGRAM
ARRAY TRGT,ERR,DETR,TEMP1,TEMP2,TEMP3, TEMP4,SERR,DUM7,AUX1,AUX2,EYE,
DUM8,DUM2,DUM3,AUX3,DUM4,DUM5,DUM6,DUM1,DEYE,DOEYE,TEMP2P,TEMP1D(0:19),
DEFTIME ANALOG= TRGT 0 ERR 0 DETR 0 TEMP1 0 TEMP2 0 TEMP3 0 TEMP4 0 SERR
0 TEMP2P 0 TEMP1D 0 DUM2 0 DUM3 0 DUM4 0 DUM5 0 DUM6 0 DUM7
0 AUX3 0 DUM8 0 AUX2 0 AUX1 0 EYE 0 DUM1 0 DEYE 0 DOEYE,
INTEGER FF,SRR,SRC,
ARRAY X1,Y1,X2,Y2(0:5),X3,Y3(0:4),
REAL S, IC,OMEGA,OMEG2,XI,PXI,ST,SL,N,GAIN)

READ(READER,/,FF,S,ST,SL,SRR,SRC,IC,OMEGA,XI,H,NUMBERSTEPS,N),
PXI + XI * OMEGA*2, OMEG2 + OMEGA*2,
FILL X1(+) WITH -30,-26,-25,25,26,30)

FILL Y1(+) WITH 0, 0, -25, 25, 0, 0)

FILL X2(+) WITH -.5,-.26,-.25,.25,.26,.5)

FILL Y2(+) WITH -.5,-.26,0,0,.26,.5)

FILL X3(+) WITH -12,-10,2,4,10)

FILL Y3(+) WITH 2,2,.85,1,5,1,5)

READ(READER,/,MAXIMUMITERATIONS,OPTIONITERATE,GAIN),
WRITE(PRINTER,<
TIME TARGET
AUX3 EYE>))
AUX1 TEMP2 TEMP4
TITLEOFFFORMATS+TRUE;NUMBERDELAYS+2)
BEGINANALOG
START OF SEGMENT ***** 0002
00000000 SC 11 010
00000200 SC 21 20512
00000300 SC 21 20512
00000400 SC 21 20813
00000500 SC 21 30310
00000600 SC 21 30310
00000700 SC 21 30310
00000800 SC 21 30310
00000900 SC 21 30310
00001000 SC 21 30712
00001010 SC 21 30712
00001020 SC 21 30712
00001100 SC 21 30712
00001200 SC 21 33112
00001300 SC 21 33412
START OF SEGMENT ***** 0051
0051 IS 0006 LONG, NEXT SEG 0002
00001400 SC 21 33612
START OF SEGMENT ***** 0052
0052 IS 0006 LONG, NEXT SEG 0002
00001500 SC 21 33811
START OF SEGMENT ***** 0053
0053 IS 0006 LONG, NEXT SEG 0002
00001600 SC 21 34010
START OF SEGMENT ***** 0054
0054 IS 0006 LONG, NEXT SEG 0002
00001610 SC 21 34113
START OF SEGMENT ***** 0055
0055 IS 0005 LONG, NEXT SEG 0002
00001620 SC 21 34312
START OF SEGMENT ***** 0056
0056 IS 0005 LONG, NEXT SEG 0002
00001700 SC 21 34511
00001800 SC 21 35713
00001900 SC 21 35713
00002000 SC 571 35912
00002100 SC 571 35912
0057 IS 0021 LONG, NEXT SEG 0002
00002200 SC 21 36013
00002300 SC 21 36211
START OF SEGMENT ***** 0058
START OF SEGMENT ***** 0059

```

```

CONSTANT S INTO DUM2 ENDIT;

RAMP SLOPE 1 AT 0 INTO DUM3 ENDIT;

IF FF=0 THEN
  CONSTANT(IF TIMESST THEN DUM2(1) ELSE 0) INTO TRGT ENDIT ELSE

IF FF=1 THEN
  CONSTANT S INTO TRGT ENDIT ELSE

IF FF=2 THEN
  RAMP SLOPE SL AT 0 INTO TRGT ENDIT ELSE

IF FF=3 THEN
  MULTIPLY(DUM3,DUM3,SL,TRGT) ELSE
  IF FF=4 THEN
    SUM(DUM2,1,DUM3,SL,TRGT)

CONSTANT(IF TIMEI MOD(SRR)=0 THEN 1/H ELSE 0) INTO SERR ENDIT;

IF M=1 THEN
  LINEAR 8 STEPS 20,.05,-10,.25,20,.35,-10,.55,20,.65,-10,.85,20,.95,-10
  INTO DUM4 ENDIT;

ENFORCING;
SUM3(TRGT,1,EYE,-1,DUM7,1,ERR);
INTERPOLATOR(ERR,X2,Y2,5,2,AUX1);
DIFFRT(AUX1,1,DEHR);
INTERPOLATOR(DEHR,X1,Y1,5,1,TEMP1);
DELAY 2,.02,TEMP1 INTO TEMPID ENDIT;

0059 IS 0037 LONG, NEXT SEG 0058
START OF SEGMENT ***** 0060
0060 IS 0037 LONG, NEXT SEG 0058
00002400 SC 581 5811
START OF SEGMENT ***** 0061
0061 IS 0002 LONG, NEXT SEG 0058
00002500 SC 581 5710
START OF SEGMENT ***** 0062
0062 IS 0003 LONG, NEXT SEG 0053
00002600 SC 581 5810
00002700 SC 581 5810
START OF SEGMENT ***** 0063
0063 IS 0005 LONG, NEXT SEG 0052
00002800 SC 581 6010
00002900 SC 581 6111
START OF SEGMENT ***** 0064
0064 IS 0002 LONG, NEXT SEG 0058
00003000 SC 581 6310
00003100 SC 581 6411
START OF SEGMENT ***** 0065
0065 IS 0003 LONG, NEXT SEG 0058
00003200 SC 581 6610
00003300 SC 581 6711
00003400 SC 581 7013
00003500 SC 581 7210
00003600 SC 581 7513
00003700 SC 581 7513
START OF SEGMENT ***** 0066
0066 IS 0005 LONG, NEXT SEG 0058
00003800 SC 581 7710
00003900 SC 581 7710
00003910 SC 581 7710
00003920 SC 581 7713
START OF SEGMENT ***** 0067
0067 IS 0016 LONG, NEXT SEG 0067
00004000 SC 671 1011
00004100 SC 581 7010
00004200 SC 581 8013
00004210 SC 581 8511
00004300 SC 581 8911
00004400 SC 581 9112
00004500 SC 581 9512
START OF SEGMENT ***** 0068
0068 IS 0005 LONG, NEXT SEG 0068

```

```

MULTIPLY(TEMP1D, SERR, 1, TEMP2P))
INT(TEMP2P, 1, 0, TEMP2))
LIMIT(TEMP2, -25, 25, TEMP3))
INT(TEMP3, 1, 1C, TEMP4))

IF TIME1 MOD(SRC)=0 THEN
  BEGIN
    IF AUX2(0)=0 THEN
      BEGIN
        AUX2(0)=2)
        AUX2(4)=AUX2(1)
      END)
      AUX2(1)=AUX2(4)+AUX1(1))
      AUX2(2)=AUX2(3)+0)
      END ELSE AUX2(0)=2)
      DELAY 0., 20, AUX2 INTO AUX3 ENDT)

SUM3(AUX3, 1, TEMP4, 1, DUM1))
SUM3(DUM1, OMEG2, DEYE, -PXI, EYE, -OMEG2, DDEYE))
INT(DDEYE, 1, 0, DEYE))
INT(DEYE, 1, 0, EYE))
INTERPOLATOR(EYE, X3, Y3, 4, 3, DUM5))
MULTIPLY(DUM4, DUM5, GAIN, DUM6))
DELAY 1., 01, DUM6 INTO DUM7 ENDT)

CONVERGE)

WRITEV TRGT(1), TEMP2(1), TEMP4(1), AUX1(1), AUX3(1), EYE(1),
      ERR(1), DUM5(1), DUM6(1), DUM1(1), AUX2(1) ENDT)
SETINITIALIZATIONFLAG=NOT INITIALIZATIONFLAG)
ENDANALOG)

WRITEV OMEGA, OMEG2, XI, PXI ENDT) FINISH.

A2CTAH      IS SEGMENT NUMBER 0076, PRT ADDRESS IS 0200
CDS          0077

```

```

00004600 SC 5A1 9610
00004700 SC 5A1 9910
00004800 SC 5A1 10112
00004900 SC 5A1 10411
00005000 SC 5A1 10613
00005100 SC 5A1 10613
00005200 SC 5A1 10810
00005300 SC 5A1 10812
00005400 SC 5A1 10912
00005500 SC 5A1 11010
00005600 SC 5A1 11111
00005700 SC 5A1 11210
00005800 SC 5A1 11213
00005900 SC 5A1 11510
00006000 SC 5A1 11711
00006100 SC 5A1 11910
START OF SEGMENT ***** 0070
0070 IS 0005 LONG, NEXT SEG 0058
00006200 SC 5A1 12010
00006300 SC 5A1 12411
00006400 SC 5A1 12910
00006500 SC 5A1 13112
00006510 SC 5A1 13410
00006520 SC 5A1 13810
00006530 SC 5A1 14110
START OF SEGMENT ***** 0071
0071 IS 0005 LONG, NEXT SEG 0058
00006600 SC 5A1 14210
START OF SEGMENT ***** 0072
0072 IS 0049 LONG, NEXT SEG 0073
0073 IS 0066 LONG, NEXT SEG 0058
0074 IS 0066 LONG, NEXT SEG 0058
0074 IS 0081 LONG, NEXT SEG 0058
00006700 SC 5A1 14510
00006800 SC 5A1 14610
00006900 SC 5A1 17111
00007000 SC 5A1 17511
START OF SEGMENT ***** 0075
0075 IS 0076 LONG, NEXT SEG 0058
0058 IS 0216 LONG, NEXT SEG 0002
00007100 SC 21 34110
0002 IS 0185 LONG, NEXT SEG 0001

```

EXP	0078	0210
LN	0079	0215
SIN	0080	0204
SORT	0081	0253
OUTPUT(W)	0082	0032
BLOCK CONTROL	0083	0005
INPUT(W)	0084	0237
X TO THE I	0085	0221
GO TO SOLVER	0086	0214
ALGOL WRITE	0087	0014
ALGOL READ	0088	0015
ALGOL SELECT	0089	0016

NUMBER OF ERRORS DETECTED = 000  
 PRT SIZE=0318; TOTAL SEGMENT SIZE=03686 WORDS;  
 ESTIMATED CORE STORAGE REQUIREMENT = 07095 WORDS.  
 21:23:46 MONDAY, JUNE 26, 1967

LAST CARD WITH ERROR HAS SEQ #  
 DISK STORAGE REQ.=04110 WORDS; NO. SEGS.=0090.  
 PROCESSOR TIME = 25.23 SECONDS I/O TIME = 44.23 SECONDS