COMPARISON OF A PURSUIT GENERATED DECAY WITH THE FIRST COMPONENT OF A TWO COMPONENT OKAN DECAY IN EYE MOVEMENTS

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ABSTRACT

This was a study designed to compare a pursuit generated decaying eye movement with the first component of a two component OKAN decay proposed by Jell et al., (1984) in man. The first component is hypothesized to be due to the pursuit input in OKAN. Pursuit was elicited in humans by tracking of a red LED in a dark room for 30 degrees at a speed of 20 deg/sec. Eye movements were recorded using dc - EOG methods. The LED was switched off in front of the subject, and a decaying eye movement ensued. A one exponential decay was fitted to the pursuit decay by a computer program. The best fit to the decay started at 130 msec after light out. Light intensity and repeated exposure to the pursuit stimulus had no significant effects on the pursuit decay. OKAN was produced at 20 deg/sec with full field stimulation for 1 min followed by 1 minute of darkness. A two component decay was fit to the OKAN in a standard fashion (Jell et al., 1984). The pursuit decay and the first component of the two component OKAN decay had different time constants ($p < .001$). These findings could mean that the same integrator is being charged differently in pursuit vs OKAN, or two different integrators are involved. Since the time constant of the pursuit decay was less than the 500 msec latency response of the eye due to the viscoelastic properties of the eye, isolation of a neural integrator was not possible.

ACKNOWLEDGEMENTS

One never really learns how hard a task is until it is completed, and by then, the daily vicissitudes are becoming forgotten, and the whole affair is shrugged off and taken in normal stride.

I'd like to thank all my friends who served as subjects and gave shoulders to lean on and especially my parents who had to put up with me. I'd like to especially thank Dr. Jell, my supervisor, who wisely told me that all it takes is to work a bit everyday, and eventually it will come. I'd also like to give special acknowledgement to:

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I will say that I definitely will never again go through a time like I did when I was finishing my Master's, as it was probably more of a normal stride than most.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

INTRODUCTION

A. GENERAL INTRODUCTION

The human eye is driven by the oculomotor system, which allows it to focus on the visual world and track objects of special interest. The oculomotor system consists of the muscles controlling the eye, along with their innervation from different neural centers. These different neural centers process different inputs to the eye muscles from the outside world, thus controlling their various movements. For an easier understanding of the many diverse movements the eye is capable of, the oculomotor system has been hypothetically broken down into a number of functional subsystems. It has been assumed that each subsystem is responsible for a distinct eye movement in response to a discrete stimulus. These hypothetical subsystems are, in order of phylogenetic occurrence:

1) the vestibuloocular reflex - this reflex produces eye movements in response to head movements sensed by the semicircular canals in the inner ear, and drives the eyes in the opposite direction of the head movement.

2) the optokinetic system - this system is responsible for producing eye movements in response to large visual movements sensed by the eye. This moving image causes eye movements in the same direction as the image. The optokinetic system complements the vestibuloocular reflex in natural surroundings.

3) the pursuit system - this system produces eye

movements for following small objects across the visual field. The most common hypothesized input is a small moving image on the retina around the area of the fovea (the area of the eye specialized for fine visual discrimination).

4) the saccadic system - this system is responsible for rapid eye movements that fixate an object on the fovea, the region of highest acuity.

5) the vergence system - this system focuses both foveas on the object of interest. This allows depth perception.

All these hypothetical systems are found in man. Only the vestibuloocular reflex is found in all animals (Robinson, 1981).

This thesis concerns itself with the pursuit system and its functional relevance to the optokinetic system. Stimulation of the optokinetic system has been hypothesized to also stimulate the pursuit system (Hood, 1967; Robinson, 1981). The optokinetic system is stimulated by visual image slip across the whole retina, including the fovea, while the pursuit system is stimulated by visual image slip of a small part of the retina around the area of the fovea (Robinson, 1981).

It is important to distinguish the hypothetical pursuit input in optokinetic stimulation. In the clinical setting, there is a test that measures the performance of the optokinetic system. It is currently assumed that the measured response of the test is due only to the optokinetic

system. Patients with inner ear and balance disorders are diagnosed using this optokinetic test. The inner ear not only senses head rotation, but also any head movement. Balance disorders can thus be sensed by the inner ear through unnatural head movements, and can themselves be initiated by disorders of the inner ear (ie. unnatural signals from the inner ear due to fluid build-up in the semicircular canals giving inappropriate posture commands). The optokinetic system adjusts for gross instabilities in vision (ie. head rotation), but for finer control (ie. due to natural tremor), the pursuit system could stabilize the visual world on the fovea (Robinson, 1981). In an optokinetic stimulation situation, the pursuit input (hypothesized visual image slip on the fovea) might "fine tune" the eye movement to closer match the stimulus movement (Ireland and Jell, 1982). Thus, differentiation of the hypothetical pursuit input in the optokinetic test results and its subsequent mal response could specify lesions of the inner ear by indicating the area of the brain and brainstem hypothetically responsible for optokinetic eye movements or the pursuit input in optokinetic stimulation.

Dr. Jell and staff, in previous work in the laboratory, were able to standardize an optokinetic test and an analysis that theoretically showed the contribution of the pursuit system to the optokinetic system. If proven true, this would make finer diagnosis more possible and accurate, and the test could further be used as an investigative tool. This Master's thesis is an attempt to independantly verify

the contribution of the pursuit system to the optokinetic test by stimulating the pursuit system alone in a manner similar to that used in the optokinetic test, and show how this contribution compares to the theoretical contribution in the optokinetic test.

It would be incomplete to discuss the optokinetic and pursuit system without putting them into their theoretical current perspective. For this purpose, a discussion of the vestibuloocular reflex is included, as there is a straight logical graduation in the maintainance of balance from the vestibuloocular reflex to the optokinetic system to the pursuit system (Robinson, 1981). All these systems deal with vision and the moving world. The vestibuloocular reflex deals with sight and head shaking, the optokinetic system deals with sight and large visual field motion, and the pursuit system deals with fixating small moving objects. Head movement is sensed by the inner ear, which has connections to the oculomotor system to form the vestibuloocular reflex and connect to the optokinetic system. The optokinetic system in turn theoretically has some pursuit input from the pursuit system. The introduction becomes more understandable if this overview is kept in mind.

B. THE VESTIBULAR - OPTOKINETIC SYSTEM.

1. The Functional Relationship of the Vestibular and Optokinetic System.

It has long been known that the optokinetic system is complementary to the vestibuloocular reflex (ter Braak, 1936; Waespe et al., 1977). Both these systems elicit nystagmus during head rotation in the light. Nystagmus is a characteristic eye movement consisting of repeated slow sweeps of the eye (called slow phase velocity; SPV) interspersed with rapid flicks back in the opposite direction (Fig 1). One beat of nystagmus is equal to one SPV and one rapid flick back. The hypothesized purpose of nystagmus is to stabilize gaze in relation to the moving visual surrounding during head rotation. The theoretical relationship of the optokinetic system to the vestibuloocular reflex becomes apparent in the nystagmic response to rotation. At very low rotational velocities (head rotation), nystagmus is sustained by the optokinetic system, whereas the vestibuloocular reflex is capable of responding to high and low transient rotational velocities (head shaking) (Melvill Jones and Milsum, 1971). Nystagmus is sustained at intermediate rotational velocities by the declining influence of one and the rising influence of the other. This is due to a different stimulus input for each. It has benn demonstrated by: 1) isolating the two systems and showing the nystagmic response of each to different rotational velocities; and 2) showing the effects of one on the other in rotation in light (Raphan et al., 1979).

2. The Stimulus for the Vestibuloocular Reflex.

Nystagmus is elicited by different stimuli for the

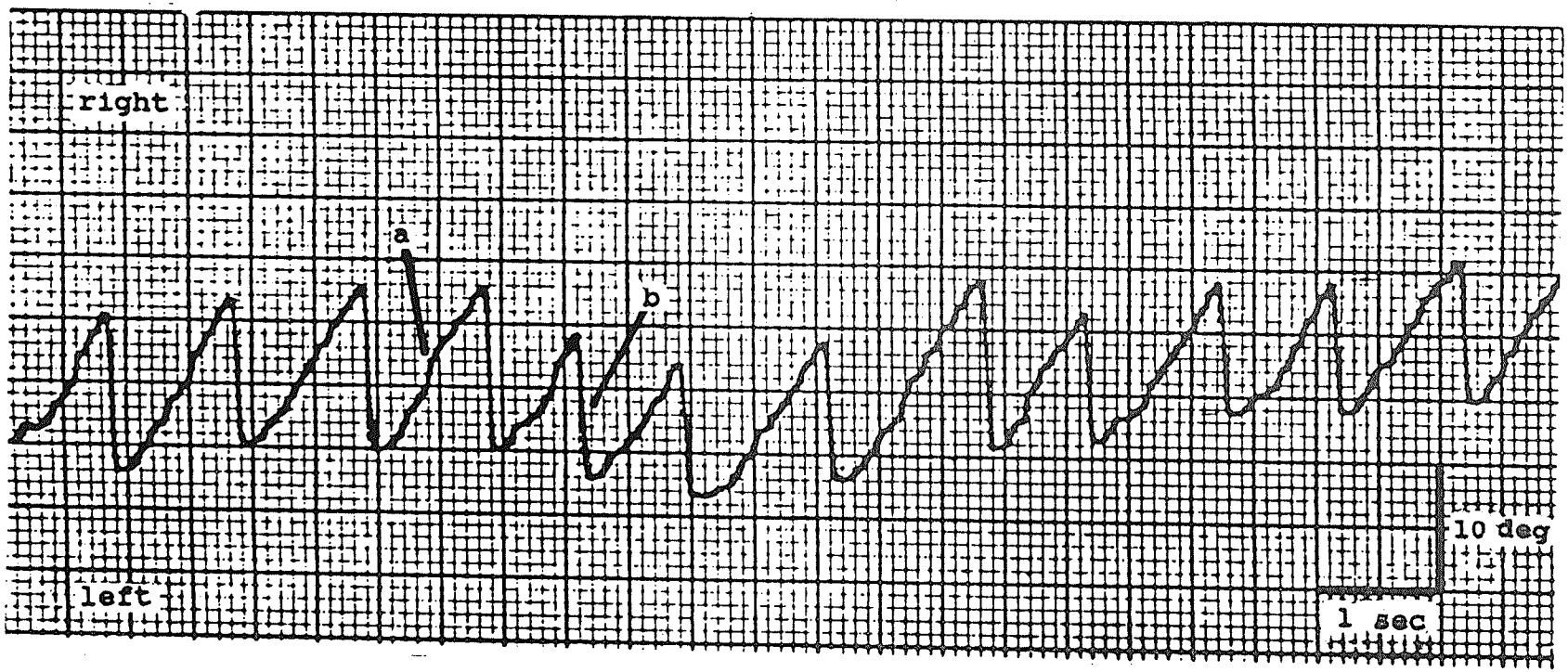

Fig 1. Example of nystagmus generated by optokinetic stimulation at 20 degrees/sec. Rotation was from left to right. Slow sweeps of the eye are depicted by a, rapid flicks back are depicted by b.

vestibuloocular reflex (VOR) and the optokinetic system. The stimulus for the VOR is head rotation in the dark. Head rotation is sensed by the fluid-filled semicircular canals in the inner ear. Activation of the semicircular canals is achieved by a transient deflection of the cupula by the inertial momentum of the fluid within the semicircular canals. The cupula is a diaphragm extending across the lumen of the canals within the ampulla. Within the cupula is the crista ampullaris, which contains hair cells that, when bent (due to the inertial momentum of the fluid in relation to the semicircular canals), send signals down the vestibular nerve to the vestibular nucleii. The vestibular nucleii are a group of nerve cell bodies found in the brainstem. The signals from head rotation then go from the vestibular nucleii to the oculomotor nucleii, and hence to the eye muscles and cause nystagmus (Wilson and Melvill Jones, 1979). When the fluid in the semicircular canals re-equilibrates with the head rotation, the cupula is again at rest, no signal is generated from the hair cells, and the nystagmus generated dies out.

The dynamics of the system can easily be demonstrated. If a subject has been accelerated in a rotating chair in the dark in one direction, nystagmus is seen in the opposite direction of the chair rotation (ie. the slow sweeps of nystagmus are in the same direction) until it dies out due to the cupula coming to rest. If rotation is stopped suddenly, nystagmus occurs in the opposite direction of rotation due to the cupula being deflected the other way by

the inertial momentum of the fluid. This is called postrotatory nystagmus, and it also decays gradually (Goldberg and Fernandez, 1981).

A practical self demonstration of the VOR can easily be shown. If a person was to hold his head still and shake a newspaper back and forth in front of him, he would not be able to read it. If instead he was to hold the newspaper still and shake his head, he would be able to read the newspaper. This is due to the VOR stabilizing gaze in relation to head shaking. This procedure will not elicit nystagmus, but it does demonstrate the VOR.

3. The Stimulus for the Optokinetic System.

The stimulus for the optokinetic system alone is rotation of a moving visual surround (images moving past a person). In the laboratory, this is usually effected by seating the subject in a lighted room, and surrounding him with a rotating optokinetic curtain. The inside of the optokinetic curtain is usually covered with vertical black and white stripes to provide a high contrast, contour rich stimulus. Check patterns have also been used (Dubois and Collewijn, 1979). The subject is instructed to look at or follow the stripes one by one. Nystagmus immediately develops with the slow phase in the direction of the curtain rotation (Cohen et al., 1981; Koenig and Dichgans, 1981) (See fig 1).

The above explanation only describes how we elicit

optokinetic nystagmus (OKN), but not why it is elicited. In the vestibuloocular reflex, the bending of the hair cells elicited nystagmus. In OKN, things become more complex, and it has not been entirely established exactly what mechanisms elicit OKN.

One of the major theories proposes that slip velocity, or images of the visual environment moving across the retina, is the input to the optokinetic system (Robinson, 1977). Slip velocity is the error signal, or ratio, between the stimulus (visual) velocity and the eye velocity. This error signal causes a controller in the brain to attempt to keep the eye velocity equal to the stimulus velocity, so little visual image slip occurs. There is always an error signal in eye movements. Without the error signal, the controller is inactivated. The slip velocity input has been studied in the cat (Stone and Fukeda, 1974) and the rabbit (Oyster et al., 1972).

Another theory proposes that efference copy, or knowledge of the movement of the eye muscles, could be responsible for the tracking motions of OKN (Holtzman et al., 1978). This theory has been difficult to study in OKN, but has been more thoroughly studied in smooth pursuit (See C.2. The Stimulus for Smooth Pursuit).

A practical self demonstration for producing OKN can be readily achieved. If a person is in a car on the highway and looks at passing fenceposts, he will find that his eyes seem to flick up to the next fencepost in the direction he is

going almost unconsciously, with little will involved in the physical effort. This is an example of OKN. The same can be done while watching a parade on the street, or watching railway tracks while on a train.

Unlike the vestibuloocular reflex, the stimulus for optokinetic nystagmus does more than excite the optokinetic system in man. It also theoretically excites another visual subsystem, the smooth pursuit system (ter Braak, 1936; Cohen et al., 1977; Leigh and Zee, 1983), which is found in all foveate species. The presence or absence of the fovea in different species changes the character of OKN in ways that reveal how it is elicited. The presence of the pursuit system has a characteristic theoretical effect on OKN.

C. THE PURSUIT SYSTEM.

1. The Physiology of the Eye and Its Relation to Smooth Pursuit.

The human retina will be taken as an example of a foveate eye. The human retina can be divided into two parts, the fovea and the periphery. The fovea is found in the center of the retina. It subtends an arc of 5 degrees and consists of only densely packed cone photoreceptors, which detect the light signal (Daveson, 1976). The peripheral retina surrounds the fovea and consists of rods and cones. These photoreceptors are less densely packed, and their signal processing is markedly different from that of the

foveal cones. The rods are used for night vision.

The human retina not only has a horizontal organization (fovea and periphery), but also has a layered organization. Light is detected by the photoreceptors, which in turn send their signals up to bipolar cells, amacrine cells and ganglion cells. These cells process the signal by (among other processes) polysynaptic inputs from many photoreceptors. Foveal cones tend to make single synapses with the processing cells, while peripheral photoreceptors make many diffuse synapses. Due to the greater density of foveal cones and the canalization of their signal, the fovea effects the highest resolution of the eye (Ruch and Patton, 1965). The primate retina is very similar to the human retina, and all foveate species are organized along similar lines.

With one part of the eye having higher resolution than the other, the foveate animal would preferentially use this area for viewing objects of interest. The saccadic system assists in target acquisition for the viewing area. A saccade is a very rapid eye movement that positions a small object of interest on the fovea. Due to an apparent refractory period of 0.2 secs. (Westheimer, 1954b), and a sampled data-like behaviour that makes the saccade a ballistic motion where it cannot change its intent until the motion is completed (Westheimer, 1954a), the saccadic system is unable to continuously keep a small smoothly moving object foveated. The optokinetic system is also deficient

for this purpose, as it responds to full field rotation of a moving visual surround. Foveation of a small moving object entails ignoring the stationary visual surround. To accomplish this tracking, the pursuit system developed to allow smooth tracking eye movements for continuous foveation of a moving object, as no other oculomotor system allows this (Lee and Zee, 1983). Afoveate species, such as the rabbit, do not have this ability (Collewijn, 1977).

The eyes themselves tend to move slowly off target due to what is called oculomotor noise. Oculomotor noise is perhaps due to muscle tremor or programmed inaccuracies designed to transmit meaningful data. A function of the smooth pursuit system here may be to produce controlled eye drift during fixation of a stationary target. In the human the standard deviation of eye drift in the light is 0.13 deg/sec (Steinman, 1965). In the dark, it has been estimated to be less than 1.0 degs/sec (Robinson, 1981). It appears that vision is being used to help control gaze, as the slow drifts are greater in the dark than in the light. Image stabilization has been hypothesized as the function of a separate ocular stabilization system (Steinman et. al., 1973), as it is present in afoveate species, such as the rabbit (Collewijn and van der Mark, 1972). An alternative hypothesis presented is that stabilization may be a special case of smooth pursuit or optokinetic nystagmus, where the image slip on the retina elicits corrective motions (Leigh and Zee, 1983).

2. The Stimulus for Smooth Pursuit.

The principle stimulus for smooth pursuit eye movements is a target moving across the fovea. The target can be remarkably small (even smaller than the fovea) and the eyes will still move to track the target and ignore the non-moving surround (Murphy et al., 1975). The target need not even be foveated. In cases where ambient light is poor, rods (that are not found in the fovea) will preferentially be used to track the target (Winterson and Steinman, 1978). This demonstrates that the part of the eye used to pursue an object is not determined by anatomy, but rather by a central selection process in higher centers of the brain concerned with pursuit (Robinson, 1981).

Another stimulus that is capable of eliciting smooth pursuit movements is extraretinal (muscle) information on how the eye is moving. Studies have shown that the path of motion of spots of light moving in the dark is often strikingly miscalculated when the subjects are asked to move their eyes and pursue the object (Festinger and Easton, 1974), with perceived extent of eye movement being much less than the actual extent of eye movement (Holtzman et al., 1978). Perception of movement in that situation has been hypothesized to depend on extraretinal information, since this is the only source of information with a reference point (movement of the eye as compared to the stability of the head). The extraretinal information in this instance has been called efference copy, or corollary discharge. The

extraretinal information can be combined with retinally obtained information to give the perception of the target motion (Holtzman et al., 1978). Efference copy may be effected either of two ways. One is proprioceptive feedback from the muscles that move the eye (Matin, 1975); the other is monitoring of the motor commands to those muscles (Skavenski et al., 1972). While evidence is not totally unequivocal, it appears to favor the monitoring of the motor command as the main source of extraretinal information (Holtzman et al., 1978).

The existence of efference copy has been demonstrated by a number of investigators. Steinbach, (1976) showed that subjects could smoothly track their own outstretched finger in the darkness with their eyes, probably by using their knowledge of the motor command to the limb and proprioceptive input from the eye muscles. Patients with acquired blindness have been shown to be able to do the same (Leigh and Zee, 1980).

The perception of movement itself can elicit following eye movements. Pursuing the imaginary center of a rolling wheel has been shown to be an adequate stimulus for pursuit eye movements (Steinbach, 1976), demonstrating that perceived velocity of a target of interest may generate pursuit eye movements. Tracking the afterimage of a pendular moving object (which is stationary on the retina) was even succesful in eliciting pursuit eye movements (Heywood and Churcher, 1971; Yasui and Young, 1975). Pursuit of an eccentric afterimage on the eye was also successful in

eliciting pursuit eye movements (Steinbach and Pearce, 1972).

This demonstrates that pursuit eye movements may be generated by various stimuli, although the most common stimulus is retinal image slip. This is similar to the stimulus for optokinetic nystagmus, except while the retinal image slip is across a major part of the retina for optokinetic nystagmus, in smooth pursuit, the image slip is very small and is mainly across the fovea.

3. Smooth Pursuit in Optokinetic Nystagmus.

Though pursuit eye movements have probably developed for pursuing small objects against stationary backgrounds, any visual image moving across the fovea, such as full field visual stimulation as in OKN, causes the eyes to make pursuit movements. Strong evidence suggests that the pursuit system plays an important part in eliciting OKN in different species.

In man, after a latency of 0.1 - 0.2 sec presumably due to signal processing (Rashbass, 1961; Robinson, 1965), upon optokinetic stimulation eye velocity jumps quickly within one beat to follow stimulus velocities of a moving target of 60 - 100 degs/s (Cohen et al., 1981; Howard and Ohmi, 1984). Beyond these velocities, human pursuit movements are unable to track the moving object accurately (Meyer et al., 1985), and nystagmus declines and deteriorates (Dichgans and Brandt, 1978). In the foveate monkey, eye velocities are

characterized by an initial rapid and later slow rise to a steady-state level, with peak velocities between 120 - 200 deg/s (Raphan et al., 1979). In the afoveate cat, there is a small initial jump followed by a slow rise in eye velocity to a peak velocity of 20 degs/s (Haddad et al., 1980). The afoveate rabbit has a similar response to that of the cat, but the eye reaches lower peak velocities (Collewijn, 1969).

The difference between species in the initial and slow rises in OKN velocities probably reflects the extent to which the smooth pursuit system is used to elicit OKN (Cohen et al., 1981; Robinson et al., 1981). Man and monkey have a well developed fovea from which the central processing areas of the pursuit system preferentially target, while the afoveate cat and rabbit have no smooth pursuit system. The fast initial rise of OKN in man and monkey is interpreted as being due to the pursuit system, while the slow rise is interpreted as being due to the optokinetic system (Raphan et al., 1977; Cohen et al., 1981). This interpretation is supported by the fact that in patients with deficient pursuit but preserved optokinetic responses, the build-up of slow phase eye velocity is as slow as in the rabbit (Yee et al., 1979). A small initial rise in eye velocity is seen in the rabbit and cat, even though they lack a smooth pursuit system. The simplest explanation for this small rise is that it is due to the hypothetical stabilization system (Robinson, 1981), mentioned before.

Though man and monkey both have fast initial rises in

OKN slow phase velocities, a slow rise is also evident in the monkey (Cohen et al., 1977). This may be because the pursuit system is more tightly coupled to the optokinetic system in man than in monkey. It has been hypothesized that the absence of a slow rise in OKN velocities in man is due to the predominance of the pursuit system over the optokinetic system , allowing an instant response to decrease the image slip brought about by the optokinetic stimulation. This tight coupling in man allows the instant increase in slow phase velocity, but at the expense of reaching lower optokinetic-induced slow phase velocities than monkey, where a looser coupling of the pursuit system from the optokinetic system allows the optokinetic system to compensate for the pursuit system and bring up the slow phase velocity. This could explain the appearence of the slow build-up of slow phase eye velocity in monkey as opposed to none in man (Raphan et al., 1977; Cohen et al., 1981). Differences in coupling have been suggested to be the reason for the different peak slow phase velocities reached. In man, they range from 60-100 degs/s, while in the monkey, they range from 120-200 degs/s, suggesting the pursuit system has less input into OKN in monkey than man, hence allowing the more reflexive optokinetic system to have the major input (Raphan et al., 1977; Cohen et al., 1981).

More and perhaps stronger evidence suggests that the pursuit system plays an important role in optokinetic stimulation. The normal optokinetic test not only records OKN in the light while the subject watches the rotating

stripes, but after a period of stimulation in the light, the subject is abruptly placed in darkness and eye movements are still recorded for a period of time. What appears is a nystagmus with beats which die out over time (in terms of slow phase velocity). This is called optokinetic after-nystagmus (OKAN) and has many characteristic properties of its own that differentiate pursuit effects from optokinetic effects.

D. STORAGE IN THE OPTOKINETIC SYSTEM (OKAN).

1. The Practical Basis for OKAN.

Optokinetic after-nystagmus (OKAN) is elicited after continuous stimulation of a full field rotating visual surround. If continuous stimulation is abruptly stopped with sudden onset of darkness, OKN is seen to continue but in a decaying manner until there is no nystagmus left (Koenig and Dichgans, 1981). This after - effect is called OKAN (Fig. 2). As there are no outside reference points in the dark, OKAN is largely undetected by the individual. OKAN is not present in the light.

The practical basis for OKAN can only be discussed in terms of its interaction with the vestibuloocular reflex. Continuous stimulation by a rotating visual surround is an unnatural situation. In natural settings, this is usually accompanied by whole body or head rotation as well,

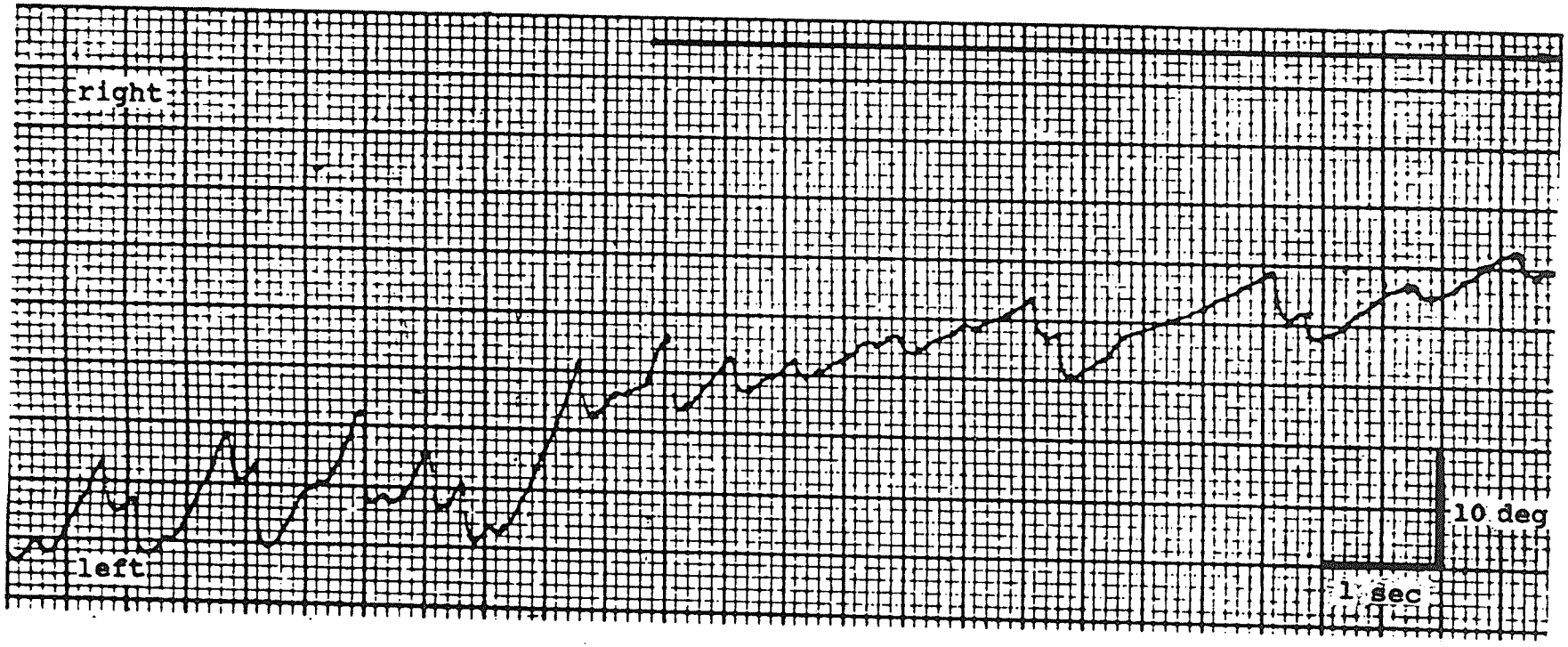

Fig 2. Example of OKN and OKAN with a stimulus of 20 deg/sec. OKAN and period of darkness are represented by black bar at top. Note diminishing velocity (slope) of slow phases as OKAN progresses.

therefore stimulating the vestibuloocular reflex (as it senses head movement). Body rotation in light causes nystagmus generated by the optokinetic system and the vestibuloocular system to go in the same direction as rotation. Abrupt halting and onset of darkness allows post-rotatory nystagmus and OKAN to become evident. Post-rotatory nystagmus occurs in the opposite direction to rotation, due to the dynamics of the cupula (see B.2. The Stimulus for the Vestibuloocular Reflex). OKAN continues in the same direction as OKN. It would appear then that OKAN helps null inappropriate post-rotatory nystagmus, as they occur in opposite directions. This would give a more realistic view of the world, as rotation has stopped. Experimentally, these conclusions have been demonstrated through the response of monitored eye movements (Raphan et al., 1979).

The physiological basis for OKAN is perhaps harder to understand . OKAN is completely due to a stored phenomenon, with storage of information taking place in neurons in the brainstem. It may be useful to go through a similar situation occurring in post-rotatory nystagmus to better appreciate OKAN. Here, there is also storage of information, but the storage can be broken down into discrete parts, one part being shared by OKAN.

2. An Analogy of Storage in the Vestibuloocular Reflex.

The close alliance of the vestibuloocular reflex and the optokinetic system is readily seen in the presence of

after-nystagmus and the storage of information in each (Malcolm and Melvill Jones, 1970; Koenig and Dichgans, 1981). All subsystems mentioned previously have been shown to only respond to direct ongoing stimuli as far as is known. The vestibuloocular reflex and the optokinetic system go further than this. Both these systems also have a release of stored information when the stimulus is removed. Upon first analysis, the reasons for storage in post-rotatory nystagmus may seem to be different from storage in OKAN, but on closer examination, there is one important similarity.

Post-rotatory nystagmus is caused by a deflection of the cupula in the opposite direction to rotation, but the nystagmus generated lasts much longer than the mechanics of the cupular movement allow (Raphan et al., 1979). The decay of the post-rotatory nystagmus has been well defined and described (Fernandez and Goldberg, 1971). It can be described by a mathematical exponential decay consisting of three parts. Two of these parts, the elasticity of the cupula and very fast rotation above 3.3 Hz. (the mechanics of the system), contribute very little to the overall value of the time constant of the decay. The time constant is the amount of time it takes the exponential decay to reach a value 1/e of its original starting value. The cupula time constant for the monkey is 5.7s, while the generated eye velocity time constant is about 12s (Fernandez and Goldberg, 1971). Most of the time constant therefore is not generated by the mechanics of the system, but from another source.

This source, which makes up the third and most important component of the exponential decay originates from stored information in the vestibular nucleii.

The persistence of post-rotatory nystagmus led investigators to believe that the vestibular nucleii were storing information based on eye velocity commands, and, upon stimulation cessation, the storage was released in the form of extended after-nystagmus. Evidence was given correlating the firing frequencies of neurons in the vestibular nucleii to the slow phase eye velocity during post-rotatory nystagmus (Waespe et al., 1977). Afferent pathways from the semicircular canals to the vestibular nucleii have long been known (Lowenstein and Sand, 1940; Goldberg and Fernandez, 1971), and numerous pathways from the eye motoneurons to the vestibular nucleii have been described (Precht, 1977).

One interpretation for the storage of information that has long been held, is that in the unnatural situation of optokinetic and vestibular stimulation, continuous rotation in the light causes the neural mechanism responsible for afternystagmus to assume in the dark that when the body is set in motion, it will remain in motion until acted upon by another force (Newton's first law of motion). There it would continue to produce OKAN in the dark, but following Newton's first law you wouldn't expect it to decay since the law is only true in a vaccuum (Rademaker and ter Braak, 1948; Robinson, 1981). Viewed in this way, extended vestibular

after-nystagmus and OKAN have very purposeful behaviours.

3. Storage and OKAN.

Having seen how post-rotatory nystagmus is produced, and the role of storage in the afternystagmus, it is easier to believe the reason for OKAN. Moving visual fields have been shown to influence vestibular nuclear units, and their firing frequencies have been correlated with OKAN (Henn et al., 1974). Further evidence that the storage of OKAN occurs in the vestibular nucleii comes from the abolition of OKAN by labyrinthectomy, which removes the organs of the inner ear, or by lesions medial to the medial vestibular nucleus (Cohen et al., 1973; Ireland and Jell, 1982), showing that the vestibular nucleii must be fully intact with their connections in order for OKAN to be produced. Lesions of parts of the perihypoglossal nucleii (Uemura and Cohen, 1975), or pretectum (Matsuo et al., 1983), which are intimately connected with the vestibular system, also abolish or greatly affect OKAN, but not OKN.

The vector stored for producing OKAN is eye velocity. Velocity storage in the optokinetic - vestibular system is an idea originated by Cohen, Raphan and Matsuo (Cohen et al., 1977; Raphan et al., 1977), although the storage concept has been in evidence for some length of time (ter Braak, 1936). Velocity storage is most clearly seen in OKAN, but in the monkey and not in man, it is also seen in OKN

initiation where there is a fast and then slow rise in OKN, while in man, there is only an initial jump.

Cohen et al., (1977) showed that in monkeys upon stimulus cessation, OKAN had a fast initial decline followed by a more gradual decline in slow phase eye velocity, approaching zero over 20-60s. They described the slow decline by an exponential decay with a characteristic time constant. They also demonstrated that the slow rise in slow phase eye velocities in the monkey upon stimulus initiation, which had always been thought to represent the optokinetic input, could also be described by a time constant. These time constants were shown to be the same, and hence it was concluded by them that both were due to the same storage mechanism, or a modelled integrator. Further evidence supported the finding. Upon bilateral labyrinthectomy, OKAN was lost and OKN slow phase eye velocities were irregular, and showed only a fast initial rise with no slow rise (Raphan et al., 1977). Firing frequencies of neurons in the vestibular nucleii were found to follow slow phase eye velocities during OKAN and OKN (Waespe and Henn, 1979). Velocity storage was later confirmed in man (Cohen et al., 1981).

Raphan et al., (1977) also gave evidence that the same modelled integrator was responsible for OKAN and storage in the vestibuloocular reflex. They showed: 1) the time constant of the two decays to be similar; 2) if directional assymetries were present in one, they were present in the other; and 3) if OKAN was habituated by repeated

stimulation, the vestibuloocular reflex was also habituated in a similar manner.

These above conclusions led Raphan et al., (1977) to formalize a mathematical model for OKAN, OKN and the vestibuloocular reflex. This model is similar to one described by Robinson, (1981), which we will present. As we are now only concerned with optokinetically mediated effects,we will disregard the input of the vestibuloocular reflex.

4. Modelling of OKAN.

Conceptually, it is useful to formulate a model and working hypothesis to offer explanations for behavior shown, and make predictions to guide future research. Cohen et al., (1977) formulated a mathematical model capable of producing the characteristics of OKAN and OKN previously described for the monkey. Though the model is basically valid, another was chosen to be discussed. Other models have been proposed with basic similar structures (Collewijn, 1981; Robinson, 1981).

For this discussion the Robinson, (1981) model will be discussed. Figure 3 diagrams the hypothetical Robinson 1981 model. Basically , the system has an input of the visual world through the eye that takes two separate paths; a direct pursuit path and an indirect path through the modelled integrator (S) (which produces OKAN). The paths rejoin after different processing mechanisms to give an

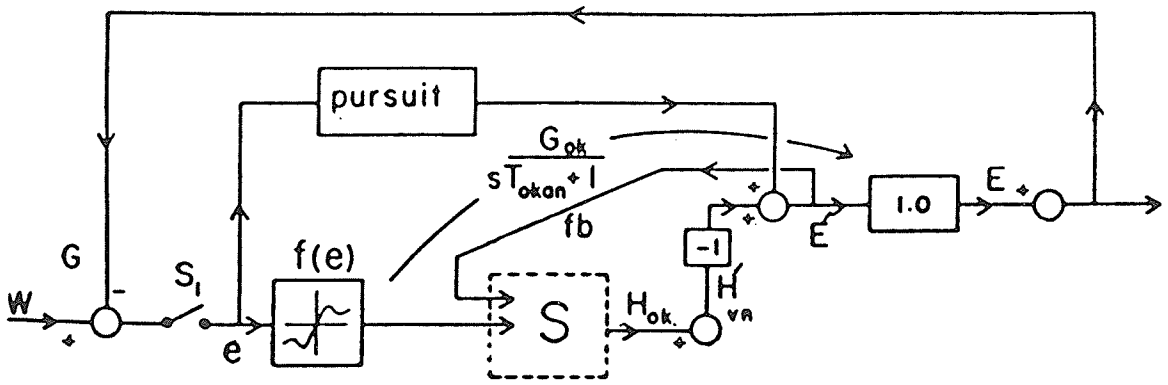

Fig 3. Robinson, (1981) model of the neural mechanism responsible for producing OKN and OKAN. Salient features are S, the integrator responsible for OKAN and the undetermined PURSUIT component (after Robinson, 1981).

output (E), which is eye velocity in the head.

The basic concept in the modelling of OKAN is the integrator (Raphan and Cohen, 1981). Briefly, its characteristics allow it to store a "charge" from visual stimulation by the moving optokinetic image (eye velocity) that is released in a manner that can be described by a one component exponential decay having the equation:

$$Y = A\, e^{-bt}$$

where A is the initial value of the decay, and 1/b is the time constant of the decay, or amount of time needed to discharge the integrator to a level A/e, and e is the natural logarithm.The model is currently based on one integrator having one time constant, but recent evidence has shown the possibility of one integrator having more than one time constant (Waespe et al., 1985).

The input to the system is (W - G), or velocity of the visual world minus the velocity of the eye in space following the moving visual world (Fig 3). This gives (e), a retinal error slip signal that accumulates in (S), the storage integrator, or splits to go through the pursuit path. From (S), the signal changes to an eye velocity command (H_{ok}) and appears in the vestibular nucleii (vn). From the vestibular nucleii, the eye velocity command from the storage integrator (S) meets with the pursuit component and sums to form (E), the eye velocity command in the head. The transfer function between retinal slip (e) and the eye velocity command (E) is characterized by a gain (G_{ok}) and a

long time constant (T_{okan}) which accounts for OKAN. Feedback (fb) is then initiated from the eye velocity command (E) to the storage integrator (S). This is due to the finding that fixation during OKAN can inhibit OKAN and discharge the integrator (Cohen et al., 1977). The eye velocity command in the head (E) is then processed into the eye velocity in space (G), which feeds back to the input (W), the velocity of the visual world. Switch (S1) removes all retinal input in the dark to allow evidence of OKAN. A non-linearity f(e) is present to split the actual visual signal. This is to accomodate the finding that two integrators and systems are hypothetically responsible for OKAN in different directions. Cohen et al., (1977) found that the time constant in the rightward and leftward direction in the monkey had different values. This led them to conclude that two integrators were present. This finding was supported by Jell et al., (1984) in man. They showed that habituation of OKAN on one side did not affect the other. If the same integrator was responsible for OKAN on both sides, habituation on one side would be evident on both sides. As this would duplicate the model from f(e) down, one side is shown for simplicity. It is unknown what route the pursuit pathway would follow.

Thus is presented the Robinson, (1981) model of the optokinetic system, the main features being the storage integrator (which is hypothetically responsible for OKAN), the pursuit pathway, feedback to the storage integrator and OKAN, and feedback to the visual world and OKN.

Though the model has stood in its basic form for some

time, a new controversy is cropping up that needs to be addressed. The pursuit system is known to have an input to the optokinetic system, but its exact nature is unknown. New evidence is presented that appears to give the pursuit system a separate hypothetical storage integrator in OKAN, thus perhaps changing the basic nature of existing models to include the two storage integrators; the existing one and one possibly involved with the pursuit pathway.

5. One component vs. two component decay in OKAN.

It has been noted that OKN and OKAN are produced by two possible inputs in the foveate animal, namely the pursuit input and an optokinetic input (ter Braak, 1936; Cohen et al., 1977,1981; Dubois and Collewijn, 1979). The presence of two components is seen by the initial fast and later slow rises and falls in OKN and OKAN in the monkey (Raphan et al., 1979). Further evidence for two inputs is given by the demonstration of two kinds of visual tracking in optokinetic stimulation. Visual tracking can either be foveal pursuit (conscious tracking) or tracking without conscious effort, termed "active" and "passive" tracking respectively by Hood and Leech, (1974). An alert individual may use either or both mechanisms during optokinetic stimulation to produce OKAN (Hood, 1967; Brandt et al., 1974; Cheng and Outerbridge, 1975; Dubois and Collewijn, 1979; Collewijn, 1981).

Modelling of the optokinetic system takes into account

the largely unknown hypothetical pursuit input and the optokinetic input (Cohen et al., 1977; Robinson, 1981). One storage integrator with one decay is modelled to fit the slow decay seen in OKAN, as the initial drop was not perceived to contain a decay. New computerized techniques with better resolution than previous analysis have shown that the fast initial drop in eye velocity observed in OKAN has a fast exponential decay. Zasorin et al.,(1983) described human OKAN to "have a fast initial decay followed by a gradual decline". Similar findings were described by Jell et al., (1983, 1984) who proposed a two exponential decay equation to describe human OKAN. This equation is in the form:

$$Y = Ae^{-bt} + Ce^{-dt}$$

where A and b would describe the fast initial decay, and C and d would describe the later slow decay. Using non-linear regression analysis and least squares method curve fit, they demonstrated a better curve fit for OKAN with a two decay equation than a one decay equation (Jell et al., 1983). With the above evidence, this led them to propose that the two component decay of eye velocity in OKAN could be caused by two separate mechanisms, one due to the hypothetical pursuit input and the other due to the optokinetic input.

On the basis of the above information, it would be very tempting to attribute one of the two OKAN decay components to "active" tracking, and one to "passive" tracking

mechanisms, corresponding to a direct, pursuit (cortical) pathway (Jell et al., 1984), and an indirect, optokinetic (subcortical) pathway (ter Braak, 1936; Cohen et al., 1981; Hoffman et al., 1982). If this were the case, the direct pathway would be expected to demonstrate the short time decay component for rapid acquisition and holding of the target (Jell et al., 1985). Pure pursuit responses have been demonstrated to have storage effects, and have been modelled containing one storage integrator (EEckmilleer, 1981). What relation this has to OKAN has not yet been determined.

E. STORAGE IN THE PURSUIT SYSTEM

Storage in the pursuit system is a relatively new idea. Mitrani and Dimitrov, (1978) were among the first to give evidence for storage in the pursuit system. Using human subjects, they demonstrated a decaying eye movement after disappearance of a tracked, horizontally-moving spot of foveated light (Fig 4). Similar evidence was given inadvertently by Eckmiller and Mackeben,(1978a) for monkeys and Bahill and McDonald, (1983) for man. Bahill and McDonald were interested in zero-latency tracking of a sinusoidally moving spot of light, the tracking movement being the theoretical output of an "internal predictor mechanism". Diagrams of their eye movement recordings clearly showed decaying eye movements after disappearance of the tracked spot. Becker and Fuchs, (1985) similarly demonstrated in

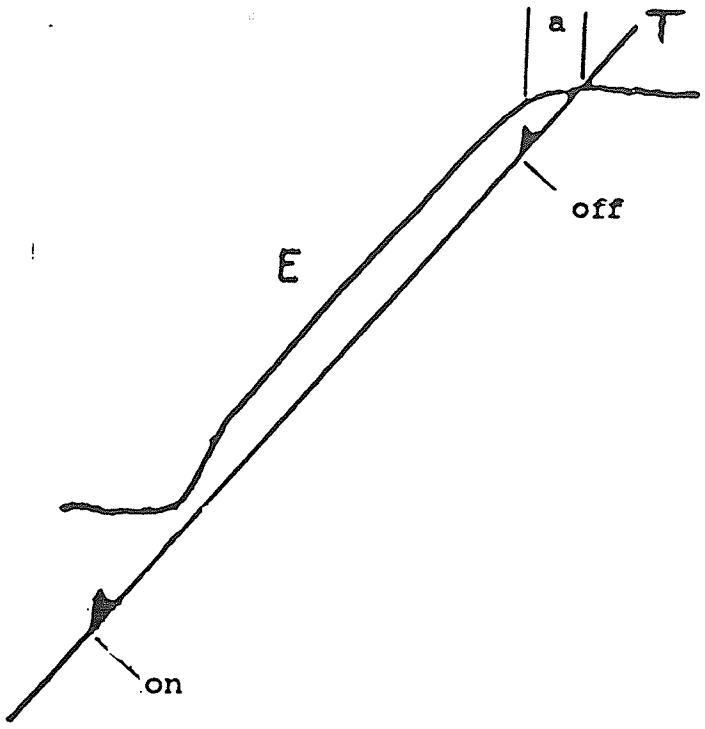

Fig 4. Mitrani and Dimitrov, (1978) demonstration of a decaying eye movement (depicited as a). T is the target motion, E is the eye movement intentionally displaced from T. Appearence and disappearence of foveal light spot is depicited by on and off respectively. Subjects were instructed to fix a point where the light was to go on, then follow it. Velocity of light was 11.4 degrees/sec (after Mitrani and Dimitrov, 1978).

humans continued pursuit of a disappearing moving target.

Decaying eye movement responses after offset of stimulus in natural systems indicate the presence of an integrator (Raphan and Cohen, 1981). The above evidence gives support for a hypothetical pursuit integrator, which has been incorporated into models of the pursuit system (Eckmiller, 1981). The relation of the pursuit integrator to the optokinetic system has not been determined. One attempt was made to relate a pure pursuit generated decaying eye movement to OKAN. Muratore and Zee, (1979) described another pursuit aftereffect in an attempt to elicit a pursuit response similar to optokinetic stimulation. Using a horizontally moving dot that was projected in a repetitive sawtooth ramp pattern that elicited eye movements much like OKN, upon cessation of stimulation in the dark, they produced a decaying eye movement aftereffect that looked somewhat like OKAN, which they called pursuit after-nystagmus. They fitted this decaying after-effect with a one component decay (Muratore and Zee, 1979). Jell et al.,(1984) were able to fit a two component decay with a very small fast decay component and a normal slow decay component to their data. The explanation of this phenomenon is uncertain, and its relation to either pursuit or OKN models is unknown at this time.

Eckmiller, (1981) presented a model that may largely explain most of the above information. Modelling of the pursuit system is at a rudimentary stage, and bears little resemblance to the known neurophysiology (Robinson,1981). In

this case, it is useful to hypothetically integrate the above information.

The Eckmiller model, (1981) is presented in figure 5. Visual movement input is through the pursuit area at top (fovea), which is divided into left and right halves. The signal then goes through a spatio-temporal translator, which is a hypothetical set of neurons that converts the raw eye position error signal to an acceleration signal. The signal leads to the velocity predictors, which deal with changes in eye velocity. From the spatio-temporal translator, the signal splits to two opposing velocity predictors, which can account for prediction in eye movements in the pure pursuit experiments. The signal continues from each velocity predictor to take two paths. One is directly to the pontine reticular formation neurons (PRF), which carries an eye position signal plus an eye velocity signal in one direction only. The other goes indirectly to the PRF via a storage integrator that is responsible for the storage and release of information. From the PRF, the eye signal goes to other systems and reaches the opposing eye muscles (Fig 5). The storage integrators and velocity predictors are opposing since they are assumed to work in a push-pull relationship to minimize position error of the target, so the target will stay foveated. They both have separate interconnections for reciprocal inhibition to maintain constant eye velocity in relation to a smoothly moving target (Eckmiller, 1981). Evidence from Muratore and Zee, (1979) cannot be placed in

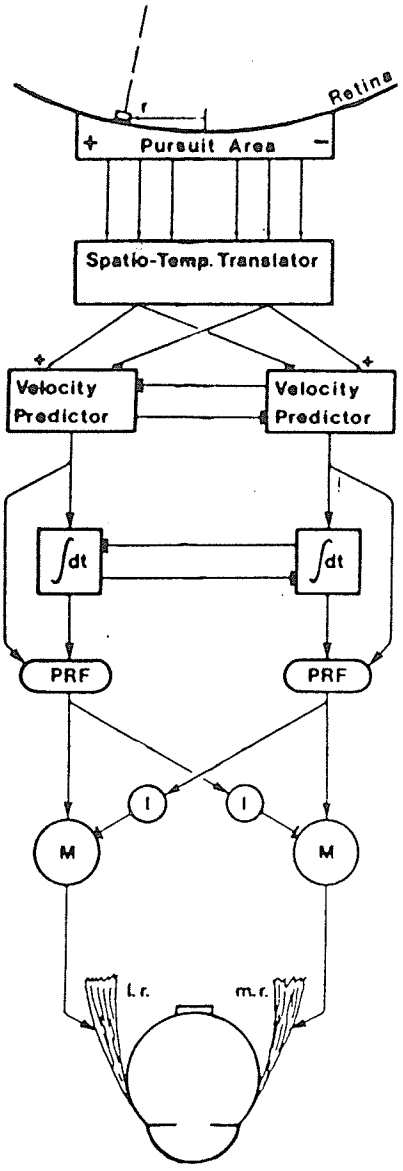

Fig 5. Eckmiller, (1981) model of the neural mechanism responsible for producing pursuit movements and decays (after Eckmiller, 1981).

this scheme, as it has a decay more like OKAN.

It must be stressed that much of this pursuit model presented, unlike the optokinetic model, has unknown neurophysiological parallels (Robinson, 1981). The relation of the Eckmiller, (1981) model to the OKAN model, if any, is unknown at this time.

There is also the possibility that the generated pursuit eye velocity decays in the afore mentioned experiments may have been due to the pursuit of a decaying afterimage on the retina. Tracking the afterimage of a moving object on the fovea, which is stationary on the retina, has been shown to elicit pursuit eye movements (Heywood and Churcher, 1971; Steinbach and Pearce, 1972; Yasui and Young, 1975). An afterimage reflects the continued photoreceptor activity to visual stimulation following termination of the stimulus: as a rule, more intense stimulation generates longer afterimages, which eventually fade (Leguire and Blake, 1982). There are two types of afterimages (or visible persistence). Type 1 is a visible response that continues beyond stimulus offset, which is judged as a subjective offset (ie. the subject cannot judge if he is looking at a real stimulus or its positive afterimage). It decreases in length with increasing luminance. Type 2 is a fading sensory trace sometimes visible as a weak afterimage (ie. the subject is aware of seeing an afterimage). It increases in length with increasing luminance and follows Type 1 afterimages when

present (Brindley, 1959). Since afterimages are capable of eliciting pursuit movements, it may be possible that the decaying eye movement was due to a Type 1 fading afterimage. As the afterimage becomes less apparent, the pursuit eye velocity could concurrently decay to zero. This type of afterimage effect would be unperceived by the individual. Afterimages have been reported with luminances as low as 6 cd/m^2 (Ueno, 1983). Strong room lighting has luminances of 16 cd/m^2 (Ruch and Patton, 1965). The possible production of pursuit decays by afterimages needs to be addressed.

E. NEUROPHYSIOLOGY OF THE OPTOKINETIC AND PURSUIT SYSTEMS.

1. Neurophysiology of the Optokinetic System.

The neurophysiological studies were carried out in the rabbit, cat, rat and monkey. Some species differences do exist as noted, but the general neurophysiology is assumed to hold true for all.

The optokinetic system has been described as the indirect, or subcortical pathway. This is because processing of the optokinetic signal is independant of the visual cortex and has been well mapped in the brainstem (Robinson, 1981). Direct connections with the optokinetic system have been shown to exist in the brainstem with the vestibular nucleii. Other connections from the vestibular nucleii, though, do lead to the cortex.

The optokinetic input signal e is represented by the

discharges of direction-selective cells in the retina (Oyster et al., 1972). The signal then may be carried by some of their thin-axoned, slowly conducting W -cells (Stone and Fukeda, 1974). The most important destination for these signals for the optokinetic system is the nucleus of the optic tract (NOT) in the pretectum (Collewijn, 1975). Stimulation of the NOT produces vigorous nystagmus in the rabbit (Collewijn, 1974). From the NOT, the signal passes through the nucleus reticularis tegmenti pontis (NRTP) to the vestibular nucleii (VN). Lesions of the NRTP greatly reduce or abolish OKN and the response of the VN (Cazin et al., 1980). Alternate pathways do exist through both climbing fiber and mossy fiber activation to the flocculus (Maekawa and Takeda, 1977), and are relayed from there to the VN by Purkinje cells (Ito, 1977). Cerebellectomy, however, does not greatly affect optokinetic responses in the rabbit (Collewijn, 1970), although it does abolish optokinetic responses in the monkey (Westheimer and Blair, 1973). These findings show species differences clearly do exist, and confuse the issue of the relative functions of the brainstem and cerebellar pathways (Robinson, 1981).

The optokinetic signal shows up as an eye velocity in the VN (Dichgans et al., 1973), and from here projects to the eye motoneurons. The cells in the VN respond to optokinetic stimulation (Henn et al., 1974). The optokinetic signal may project to eye motoneurons by alternate routes, but this hypothesis remains unproven (Robinson et al., 1981). Bilateral labyrinthectomy in man (Ireland and Jell,

1982) and in monkey (Uemura and Cohen, 1973) blocks OKAN but not OKN (Zee et al., 1976). This could be due to the input of the pursuit system in monkey and man in OKN. Consequently, after labyrinthectomy, pursuit nystagmus during optokinetic stimulation can still occur (Zee et al., 1976a). In the rabbit, labyrinthectomy does abolish OKN (Collewijn, 1976), although some remnant nystagmus is seen that could be due to the hypothetical stabilization system (Robinson, 1981). From the VN, the eye velocity signal then goes to the oculomotor nucleii, where it is converted to an eye position signal (Skavenski and Robinson, 1973), that reaches the eye musculature.

2. Neurophysiology of the Pursuit System.

The hypothetical pursuit system has been described as the direct, or cortical pathway. This is because the pursuit eye movements have known effects on the visual cortex. The pursuit system though is not solely under control of the cortex. Subcortical (brainstem and cerebellar inputs) are important too.

The input to the pursuit system is presumably retinal image slip, which is detected by direction-selective cells in the visual cortex in the parietal lobe (Lynch et al., 1977). Some selection process must take place that decides which retinal slip belongs to the target and which does not, as the smaller slip is due to the object being pursued, and the larger slip due to the non-moving surround. One

strategy to reduce the selection time may be to track first what is on the fovea, and ask questions later (Robinson, 1981). The signal from the retina to the cortex is carried by the retinostriate pathway, which is generally assumed to be essential for foveal pursuit (Robinson, 1976). Very little is currently known about single unit activity during pursuit eye movements (Eckmiller, 1981; Robinson, 1981). A number of additional representations of the retina in the cortex other than the primary visual cortex have been found (Van Essen, 1979). Their purpose is unknown. There are direct projections from the fovea to the superior colliculus, which so far seem to play a minor role in foveal vision (Cowey and Perry, 1980). Stimulation of regions above the midcollicular levels in alert animals have failed to elicit pursuit (Bender, 1962). Stimulation of the cerebellar hemispheres (Ron and Robinson, 1973) and the PPRF (paramedian pontine reticular formation) (Cohen and Komatsuzaki, 1972) which is found in the brainstem, only produces pursuit-like movements.

Cells that appear to be directly related to pursuit eye movements are found in the flocculus. There, some Purkinje cells in the alert monkey modulate their discharge in proportion to gaze velocity in space (Miles and Fuller, 1975). Cerebellectomy abolishes pursuit in monkeys (Westheimer and Blair, 1973), and vestibulocerebellectomy interferes with it (Zee et al., 1978). It is presumed that the gaze Purkinje cells inhibit cells in the vestibular

nucleii, because it is well known that the flocculus projects there (Ito, 1977), but because cells in the vestibular nucleii do not respond to pursuit mediated Purkinje activity as predicted, it is uncertain where the gaze Purkinje cell axons go (Keller and Kamath, 1975).

In the pons, cells have been found just ventrocaudal to the abducens nucleus that carry a discharge rate proportional to pursuit eye velocity (Eckmiller and Mackeben, 1978b). These cells do not modulate during the vestibuloocular reflex. These cells could carry the pursuit velocity command and could be related to gaze in the Purkinje cells (Robinson, 1981).

F. OUTLINE OF PROPOSAL.

Jell et al., (1984) described a two component decay model for OKAN. They proposed that the first component could be due to the pursuit input in OKAN. This thesis presents the results of an attempt to elicit a pure pursuit response and decay in man in a similar manner to that used to generate OKAN. The pursuit decay is compared with the fast decay of OKAN. The following steps were taken:

1) A pursuit decay was elicited repeatedly in each of a series of human subjects and the possible effects of an afterimage on the decay were investigated. A decaying afterimage could elicit pursuit eye movements in the form of a decay. Pursuit was elicited at three separate light intensities. Pursuit and pursuit decay parameters were

compared to see the effect of different light intensities on each.

2) A single exponential decay curve was fit by a computer program to each pursuit decay at a single light intensity. Its parameters were then compared to the parameters of the fast decay in OKAN that had been similarly fit by a computer program.

3) OKAN was elicited in the subjects and a standard double exponential curve was fit to the OKAN decays (Jell et al., 1984) OKAN has much intersubject variability and in an attempt to reduce this variability, pursuit decays were compared to the corresponding OKAN decays within one subject.

4) Pursuit decay parameters were compared to the fast decay parameters of OKAN within one individual.

5) Results in terms of the pursuit model and OKAN model were discussed.

METHODS

A. EXPERIMENTAL POPULATION.

There were eight human participants in this experiment, ranging in age from 22 to 31 years. None had any known history of vestibular or visuomotor disorders. Three of these subjects wore corrective lenses.

Three subjects, two males and one female, participated in the PURSUIT INTENSITY (afterimage) experiments. These consisted of three pairs of pursuit trials at three different LED light intensities in the dark. Each trial consisted of 1 minute of pursuit stimulation, equalling 13 pursuit events in all.

Five subjects, one male and four females, participated in the PURSUIT STUDY (comparison of pursuit and first component of OKAN decays). It consisted of four pursuit trials consisting of 13 - 20 pursuit events each trial. This was followed after a 5 minute rest period by three OKN - OKAN control trials.

B. EYE MOVEMENT RECORDING - EOG.

Horizontal eye movements were recorded using standard dc-electrooculography (EOG). This method is most suitable for measuring eye velocity and approximate eye position (Schlag et al., 1983). A standard Beckman non-polarizable electrode was applied to the outer canthus of each eye and

on the nasion for monitoring of horizontal eye movements. The EOG was amplified and recorded using a N - 301 ICS standard electronystagmograph. Pursuit testing began after a 30 min stabilization period and dark adaptation with red light (Gonshor and Malcolm, 1971), at which time the subjects sat in a dental chair and made themselves comfortable. The head was supported on an occipital rest and restrained with a Velcro band. Before the beginning of OKN - OKAN testing (which followed pursuit testing), the subject was light adapted for 5 minutes while remaining in the chair (Ruch and Patton, 1965).

Horizontal eye calibrations were performed with a board at eye level and 1 m in front of the subject. The calibration board had operator controlled light emitting diodes (LEDs) at center (0 degrees) and at 20 degree left and right positions. Center was determined by the alignment of the fixed dental chair and the subject's position. The LEDs could be flicked on one at a time by the operator. The subject was instructed to look at each LED as it was turned on. The purpose of the calibration board was to calibrate the EOG signal from the subject to a known scale. Calibrations were performed before and after each trial in the pursuit and OKN-OKAN testing.

All eye movements were displayed on a rectilinear chart recorder, and stored on FM magnetic tape (Hewlett Packard Model 3960).

C. DESCRIPTION OF PURSUIT APPARATUS.

Four equally spaced red LED preparations were mounted at eye level on the inside of a black curtain 120 cm in height and 100 cm in diameter, hanging from a horizontal hoop and forming a cylinder. The hoop could be rotated by a dc electric motor in the leftward or rightward direction around its vertical axis. The motor was powered by a variable - voltage regulator power supply. Drag of the curtain, determined by the vertical orientation of the LEDs, was not noted when the curtain was rotating.

Red LEDs were chosen to optimize foveation by stimulation of the red sensitive foveal cones by a pinpoint source of light (Noorlander et al., 1983; Nygaard and Frumkes, 1982). LED preparation consisted of first cutting the top off the rounded LED to as close to the light emitting element as possible, and then sanding it smooth (Nygaard and Frumkes, 1982). The LED was then mounted on a small piece of electrical hobbycraft board in series with a 200 K resistor. A plug was attached to the back and the preparation was painted flat black. A hole 0.07 cm was drilled in the center of the LED to allow light through. This subtended an arc of 0.13 degrees on the fovea of the subject 30 cm away. The inner foveal pit of the human eye subtends an arc of 0.5 degrees (Daveson, 1976). The LED preparation was then taped to the inside of the curtain with a small hole in the back of the curtain to allow projection of the plug and attachment of the power wire. LED preparations were mounted at 90 degree intervals around the

circumference of the drum.

Figure 6 diagrams the apparatus controlling the LEDs. Power was supplied to the rotating LEDs from a power source with a range of 1 - 12 v. Power was monitored by a dc - voltmeter. Voltage output corresponded to drum velocity. Power was fed through a wire to an assembly above the rotating curtain. This assembly contained 4 stationary spring-loaded brushes with individual variable resistances placed in hard plastic. Power was translated through the stationary brushes to 4 metal slip rings mounted on a hard fiberglass disk fixed in position with the rotating hoop. Each brush and slip ring fed power to an individual LED through a wire. A matched input of 1.8v was fed to each LED, which corresponds to an intensity of around 0.027 mcd.

The power to the individual LEDs was controlled by an automatic switching infrared emitter/detector device. A discrete stationary infrared light emitter/detector source was mounted above the rotating fiberglass disk. The infrared emitter/detector was positioned so reflection of the discrete infrared beam from the emitter off the disk activated the detector and switched on an LED. The disk was painted black to make it opaque to the infrared light beam. Reflection of the light beam was accomplished by pieces of reflective tape. The radial length of the tape determined the amount of time one LED was switched on. Only one LED could be on at a time. Switching was accomplished by an electronic switching circuit. Four pieces of tape

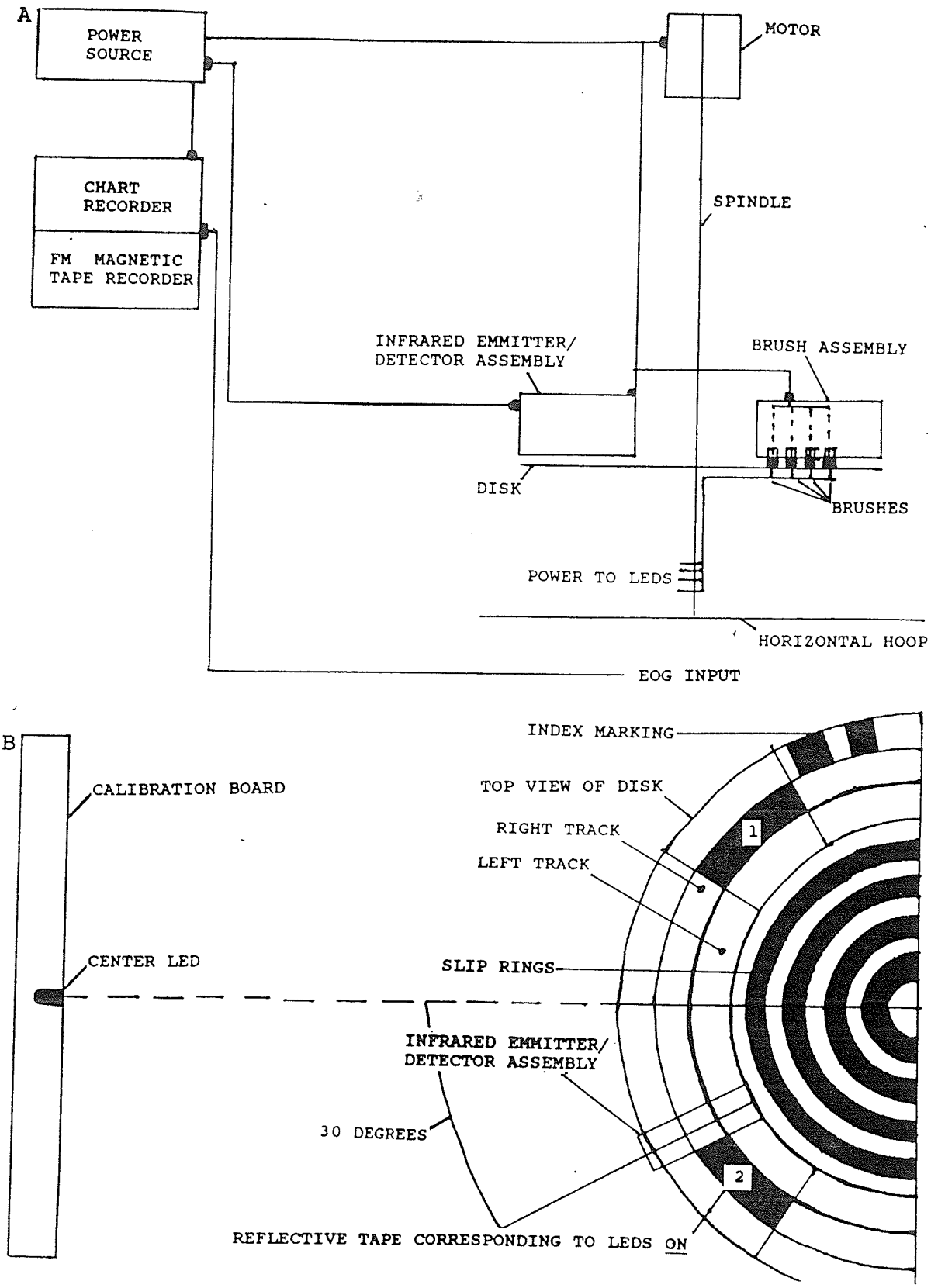

Fig. 6. Diagram of pursuit apparatus. A is the side view and B is the top view.

corresponded to the four LEDs. Leftward and rightward rotations had their own separate tracks and infrared emitter/detector sources. Absolute correspondence of tape to LED was accomplished by one index marking resetting the switching mechanism every rotation. This also made sure that at each rotation the LED was activated in the same place relative to the subject. An index marking indicated one rotation. It had its own infrared emitter/detector system as described above. This made three infrared emitter/detector devices in all, mounted beside each other on different tracks. One was for the index marking, signifying one rotation and resetting the switching component of the LED system. Two were for switching the LEDs on in a consecutive manner one at a time consistently in the same place, one of which was for the leftward and the other for the rightward direction. This assembly also had an ambient light detector, which detected the amount of light present in the room.

The infrared emitter/detector assembly was stationed above the disk at 330 degrees (0 degrees is defined as the line drawn from the center calibration light to the center of the rotating spindle on which is attached the hoop and disk). Radial lines were drawn every 10 degrees across the hoop and disk. 0, 90, 180 and 270 degrees on the disk corresponded to the position of the four LEDs on the curtain. A 30 degree span of the reflective tape on the disk corresponded to a 30 degree activation of the LED it controlled when that tape was under the infrared emitter/

detector assembly. The disk, hoop, infrared emitter/detector assembly, calibration board and the dental chair on which the subject sat were fixed in place relative to each other (Fig 6).

Index markings and LED on/off markings were recorded on one channel of the rectilinear chart recorder and FM magnetic tape recorder. Index markings were recorded as double rectangular pulses of equal height, with one of short and one of long duration. This indicated one revolution and the order of the pulses (long - short or short - long) indicated the direction of rotation. Between the index markings were four pairs of pulses (spikes) corresponding to the four LEDs. Pulses (spikes) in the same direction as the index corresponded to an "LED on" signal (reflective tape appearing to the detector), and impulses (spikes) in the opposite direction to the index corresponded to an LED off signal (non - reflective portion after a reflective portion). Intervals between an on/off pair correspond to periods of darkness. LEDs could be switched off by a switch, inactivating the controller mechanism. This would leave only the index marking on the recording. Room light off was indicated by a gross shift of the index trace in a direction towards the index.

D. PURSUIT STIMULUS.

Pure pursuit was induced by lights on a rotating

curtain, similar in manner to the inducement of OKN - OKAN. Four red LEDs were mounted at eye level on a black curtain, equally spaced at 90 degree intervals around the inner circumference, and were turned on and off in a consecutive manner. Pursuit experiments were carried out in complete darkness in the rightward direction only. Pursuit was induced over a 30 degree arc at a speed of approximately 20 degrees/sec. This combination had been demonstrated to elicit good, continuous smooth pursuit eye movements (Takahashi et al., 1983). LEDs were switched on at 330 degrees and switched off at 0 degrees (center). This corresponded to a taping on the disk for the 0 degree LED from 330 degrees to 0 degrees. Individual activation was determined by individual spans of reflective tape under the infrared emitter/detector assembly. For this 30 degree span, the LED was continuously on and moving towards the right. Each LED turned on at 330 degrees and off at 0 degrees one at a time, resulting in a cycle in which an LED was on for 1.6 sec followed by darkness for 3.2 sec. During periods between pursuit events (one activation sequence of each LED), the subject was in complete darkness. At 20 deg/sec, 13 pursuit events equal 1 minute of stimulation. After 13 - 20 pursuit events, all lights were switched off and the subject was in total darkness for 30 seconds. (For subject instruction, see F.EXPERIMENTAL PROCEDURE).

E. OPTOKINETIC STIMULUS

A standard optokinetic method was employed to induce OKN - OKAN (Jell et al., 1984). OKN - OKAN was induced by an internally lit optokinetic curtain made from a white cloth curtain with 2 degree black vertical stripes at each 18 degree interval. The curtain was 120 cm in height and 100 cm in diameter.

Optokinetic testing was conducted at a velocity of 20 degrees/sec. This speed was chosen to match that of the pursuit stimulus. Optokinetic stimulation consisted of approximately 10 sec of darkness with the curtain rotating followed by 1 min of curtain rotation in the light. During optokinetic stimulation, the subject was instructed to follow the individual black and white stripes one by one as they crossed his field of vision. OKAN was induced immediately after OKN by abruptly switching off the lights in the room, placing the subject in darkness for 1 min. During this time the subject was instructed to look straight ahead, and, to ensure alertness, instructed to count back from 200 by 7's mentally. He was encouraged to not move his head and to keep alert in this period of time. All instructions were given to the subject prior to each test. Three separate optokinetic trials were performed. Before and after each trial an eye movement calibration was performed. Between each trial a 5 min rest period was given.

F. EXPERIMENTAL PROCEDURE.

1. Pursuit Intensity Series.

This set of tests was to determine if the pursuit generated decay (Mitrani and Dimitrov, 1978) was due to the afterimage of the LED on the fovea. The stimulus was a red spot of light at 3 intensities seen in the dark. The hypothesis was that if the pursuit aftereffect (PAE; ie. pursuit generated decay) was due to an afterimage of the LED in the dark, measured PAE parameters would change with different LED intensities, because a stronger, and hence longer lasting afterimage would result from higher intensity stimulii.

LED intensities were changed by applying plastic neutral density filters (Roscolux F stop 2, F stop 3) between the subject and the LEDs. Filters were taped onto a removable helmet placed on the subject, spanning his field of vision. No visual deficit due to the filters was reported. Filters could be changed quickly by taping and untaping. The LEDs were illuminated with 1.8v, corresponding to approximately 0.027 mcds throughout all tests. Three different intensities were used. Highest intensity was the LED with no filter; moderate intensity was with the neutral density filter of F stop 2, corresponding to a 14% reduction in the luminance; and low intensity was with the neutral density filter of F stop 3, corresponding to a 29% reduction in the luminance. All intensities were easily visible to the eye.

Three subjects were used for this series. They were dark adapted in red light for 30 min while EOG electrodes were applied. Subjects were instructed at this time. They were told to look straight ahead when no light was visible. When a light became visible on their left, they were told to pick up the light and follow the light. When the light went off around center, they were told to look straight ahead and count backwards from 20 by 1's slowly to ensure alertness. During the testing, they were told to keep alert but relaxed.

This PURSUIT INTENSITY series consisted of 6 pursuit trials in all. The actual testing was conducted in the dark. Trial 1 was with no filter, trial 2 and 3 were with filter F stop 3, trial 4 and 5 were with filter F stop 2, and trial 6 was with no filter. Each trial consisted of 13 - 20 pursuit events, (one minute equal to 13 pursuit events), followed by 30 sec of darkness, where the subject was requested to look straight ahead and keep alert. Eye calibrations were performed before and after each trial, with a 1 min rest period in between trials. Length of test was 1 hour. Investigator had contact with subject only when the filters were applied.

2. Pursuit Study Series.

This set of tests was to determine if the pursuit generated decay was due to the output of the same neural integrator as the fast decay in the two component model

proposed by Jell et al., (1983;1984). The pursuit stimulus was a red spot of light seen in the dark. LED intensity was kept at 1.8v, or approximately 0.027 mcd. A one component decay was fitted to the pursuit decay. The OKN - OKAN stimulus was the standard full - field rotating curtain. A standard two component decay was fitted to the OKAN data.

Five subjects were used for this series of experiments. They were dark adapted in red light for 30 min while EOG electrodes were applied. Instructions to the subjects were similar to those given in the PURSUIT INTENSITY series, except that they were told to look where they last saw the light when the light went off around center. Subject protocol was changed in this manner to get a smoother PAE without contamination of saccadic eye movements, as had been found in the PURSUIT INTENSITY series. This approach was quite successful and deemed adequate for our purposes.

The series consisted of 4 pursuit trials in all, followed by 3 OKN - OKAN trials. Each pursuit trial consisted of 13 - 20 pursuit events, in an attempt to get as many smooth decays (PAEs) as possible. After pursuit testing, subjects were given a 5 min light adaptation and rest period. During this time, the LEDs were unplugged, the black curtain taken off and the optokinetic curtain put on the hoop. The subjects were then instructed on the OKN - OKAN tests, as described before. Length of testing was 1 1/2 hours.

G. EXPERIMENTAL ANALYSIS.

1. Pursuit Intensity Study.

A number of different parameters were analysed in the PURSUIT INTENSITY series. They were analysed off the raw and differentiated chart record from the rectilinear chart recorder by hand for each pursuit event. All endpoints used were easily discernable. The parameters analysed were:

1) PICK-UP LAG - the amount of time for the eyes to swing to the left and pick up the LED after it appeared. This was measured from the LED on index mark on the chart to the first large saccade used by the subject for picking up the LED.

2) PURSUIT TIME - the amount of time the eye pursued the LED while the target was on. Measured horizontally from the first largest saccade to the LED off index mark.

3) PURSUIT DEGREES - the number of degrees the eye pursued the LED while it was on. Measured vertically from the lowest point of the first largest saccade to the point where the LED off index intercepted the pursuit movement.

4) PURSUIT SPV - The slow phase velocity (pursuit velocity) of the eye while it was tracking the LED. Measured by the angle of the pursuit movement.

5) PAE (TIME) - the amount of time the pursuit aftereffect (PAE) was in effect from the LED off index mark to the commencement of the first saccade.

6) PAE (DEG) - the number of degrees the pursuit

aftereffect (PAE) was in effect. Measured vertically from where the LED off index mark intercepted the pursuit movement to the farthest eye movement.

These parameters were analysed for intensity effects and habituation effects within one intensity.

2. Pursuit Study.

i) Pursuit Analysis.

Different parameters from the PURSUIT INTENSITY series were analysed in the pursuit study. The main object of this analysis was to compare the PAE decay to the first component of the two component OKAN decay fit. For this reason, an exponential decay was fitted to the PAE in much the same way as in OKAN.

A computer program was set up to analyse the PURSUIT STUDY data and fit an exponential decay curve to the PAE. Data from the FM magnetic tape was fed through an analog-to-digital converter into a Hewlett-Packard Model 9836 Desktop Computer. Each trial with calibrations was fed in separately at a 5 msec sampling rate and analysed separately. Two channels were fed into the computer. Channel 1 contained the EOG eye signal and channel 2 gave the lights on, lights off, and index marking signal. The signal was smoothed two times with a 5 point moving average having the equation:

$$y'(t) = \frac{y(t - 2t') + y(t - t') + y(t) + y(t + t') + y(t + 2t')}{5}$$

Smoothing was done to reduce the background noise of the signal.

Interactive data processing began with the calibrations. 15s of the calibration EOG signal before and after each trial were displayed consecutively on the computer screen. Operator controlled moving horizontal cursors were fixed to define the upper and lower (right and left) eye movement calibration volt signal of each. The values were averaged between the two calibrations and the rest of the data was converted into degrees.

Individual pursuit events were displayed consecutively on the screen. Raw pursuit events were picked up by the lights on signal and a 4 second epoch of data was displayed after the lights on point. A vertical line from the lights-off signal was also displayed on screen, showing where the LED went off in time. This occurred approximately 1.6sec after the LED appeared.

Pursuit events could be discarded by the operator. The selection criteria were based upon the best raw smooth and consistent pursuit events. The least number of events chosen in a pursuit trial was 7 out of 13, the most was 13 out 18.

The selected raw events were then differentiated to give velocities and then consecutively displayed. Distinct peak eye velocities were removed as being anomalous eye movements or gross saccadic eye shifts. Operator controlled moving vertical cursors could define the beginning and end of removal.

A decay curve was then fitted to the PAE. The single exponential decay curve was fitted in the form: $y = A \exp(-Bt)$ using conventional linear regression analysis. Start of the decay could be defined by a vertical moving cursor by the operator. Placement of the cursor was helped by a display on the screen of a number that gave the displacement of the cursor from the lights off point. The cursor had an accuracy of 4 msec. The endpoint of the decay was fixed at 1 sec after lights off. At this point, the decay had ended. Three criteria were used to place the start of the decay, since some decays appeared to start at some point after the lights off point. A signal processing time has been described (Rashbass, 1961; Robinson, 1965), of which the best estimate is 130 msec (Robinson, 1981). Decay analysis was therefore carried out 3 times on a pursuit event; once with the decay start at 0 sec (lights off point); once with decay start at 130 msec; and once with decay start at the operator's estimation of where the decay appeared to begin.

The average SPV (slow phase velocity of the eye movement pursuing the red dot) of the pursuit movement was determined by placing a horizontal moving cursor through the median differentiated SPV points of the pursuit eye movement. Statistics of each pursuit event were then printed out giving A, B, T (time constant = 1/B), r^2 (correlation coefficient of scatter points), lights out time and decay start time.

Upon completion of one pursuit trial, selected events

were shown superimposed over each other. Selected events were then meaned and displayed as a raw signal with one standard deviation. The mean pursuit event was then differentiated. The same procedure as described above was then carried out on the differentiated mean event. Data could then be reprocessed to discard more selected events, or new data could be input.

ii) OKAN Analysis.

OKAN was analysed from the raw and differentiated chart recordings. Graphic recordings of OKAN were laid upon a Houston Hipad graphics tablet, serving as a digital input device to the Hewlett Packard 9836 Desktop Computer. Calibrations were used to average the data in a similar manner to that of the pursuit analysis. Start and end points of each slow phase were selected by the operator and coordinates digitized and fed into the computer. Curved slow phases were approximated by linear segments. Slow phases were gradually fed in from the light-out point until the slow phase velocity became indistinguishable from zero. Lights out point (beginning of OKAN) was also fed into the computer. Data was stored on computer diskette. From these data, it was possible to plot cumulative displacement against slow phase number, and to plot the slow phase duration against time, as well to determine the statistics of the slow phase durations and fit a curve to the decay of SPV.

Computer programs had been written for analysis. Non-

linear regression analysis of each OKAN decay was carried out using Marquardt's procedure (Marquardt, 1963) to obtain estimated parameters by re-iteration for an equation of the form $y = A \exp(-Bt) + C \exp(-Dt)$, describing the two component OKAN decay process (Jell et al., 1983; 1984). Cumulative gaze displacement during OKAN was computed and plotted.

H. EXPERIMENTAL CONTROLS

Two experimental controls on the equipment were carried out. One (Control 1) was to determine the consistency of the computer program used to analyse and fit the decay to the PAE (pursuit aftereffect). Known values were fed into the program to synthesize a normal pursuit event, with A = 19.89 deg/sec; B = 4.00; tc(time constant) = .250 sec; r^2 = 1.000. This pursuit event was differentiated to give decay values of A = 19.89 deg/sec; B = 4.00; t = .250 sec; r^2 = 1.000; lights <u>off</u> time = 1.6 sec; and average SPV (slow phase velocity) = 20.00 deg/sec. The other (Control 2) was to determine the amount of inherent noise in the system. One pursuit trial was run without a subject hooked up to the EOG channel. Instead, the EOG input was shorted. Recordings for two rotations were made with the LED power on. The computer program was used to analyse the results. Instead of a curve fit to the shorted EOG signal, the mean X value of the signal was given with the standard deviation and high and low values.

I. STATISTICAL ANALYSIS.

Analysis of variance (ANOVA) split plot design procedures were employed to examine the various hypotheses in the thesis. Split plot procedures were employed to study each factor within the design independently and compare any interactions between the factors. Where necessary, analysis was performed by a student t-test. The student's t-test was only employed for comparison of the PAE decay fits with the OKAN first component decay fits, where non-homogeneity of variances invalidated ANOVA use (Cohen, 1965).

For the PURSUIT INTENSITY series, two hypotheses were tested. The first hypothesis was that decreasing the pursuit light intensity in the dark would change the PAE parameters, demonstrating that the PAE decay was due to an afterimage. Pursuit parameters were similarly investigated to demonstrate that an adequate range of intensities in this experiment had been chosen to elicit a change in PAE response by a subsequent change in pursuit parameters. The statistical design was that two trials consisting of 13 pursuit events had been done at each of the 3 intensities. The two trials were considered as replicates. ANOVA was between intensities, between events and between subjects. Bartlett's test with a chi squared statistic ascertained the validity of replication by testing for homogeneity of variances between the replicates.

The second hypothesis tested in the PURSUIT INTENSITY series was that repeated exposure to the pursuit stimuli

caused a decrement in the parameters studied. This second hypothesis was tested only on parameters found to be significantly different between intensities in the intensity analysis, as a non significance between intensities (and between trials) supports a lack of effect of repeated exposure. Further repeated exposure effects were only looked at in Intensity 1; NO FILTER. The two trials done at this intensity were trial 1 and trial 6, the first and last trials of the experiment. ANOVA analysis was given between trials, between events, and between subjects. A posteriori hypotheses regarding possible changes between individuals or grouped means were tested using the multiple comparison test (Sokal and Rohlf, 1981).

For the PURSUIT STUDY series, two hypotheses were tested. The first hypothesis was that repeated exposure to the pursuit stimuli caused a decrement in the parameters studied. Repeated exposure involved Trials 2, 3 and 4 out of 4 trials. Each trial consisted of 7 chosen events. The events were chosen on the basis of showing smooth pursuit of the target. Seven events were chosen to allow a balanced ANOVA. Data from trial 1 was not used, as it was thought to represent more variability due to first exposure to the stimulus. ANOVA was between trials, between events, and between subjects.

The second hypothesis tested in the PURSUIT STUDY series was that pursuit generated decay and the first component of the OKAN decay were similar. A t-test was employed to test significance in the parameters A and 1/B

between the two populations. Three pursuit trials were compared to three OKAN trials. In this instance, ANOVA was invalidated due to non-homogeneity of variances (Cohen, 1965).

RESULTS

A. PURSUIT INTENSITY SERIES

1. Analysis of Effects of Light Intensity on the Pursuit and PAE Parameters

Two typical pursuit events of this series are shown in figure 7. The top trace shows the eye movements while the bottom trace shows the lights on and off signal. On the first event are superimposed lines to show how the actual measurement of the different parameters was accomplished. Parameters 1-4 are the pursuit parameters and parameters 5 and 6 are the PAE (pursuit aftereffect) parameters. Pursuit events at all intensities looked similar, and followed the same course. The subject was looking straight ahead in the dark when a rotating LED turned on at 30 degrees to the left of the subject (fig. 7). The offset of the eye trace represents the subject's center gaze as compared to the absolute center of the chart strip. After a 250-500 msec delay (Parameter 1. PICKUP LAG), the eyes swung quickly to the left and with a corrective saccade, fixated the light. The eyes followed the LED to the right until it was turned off at center (measured by parameters 2. PURSUIT TIME; 3. PURSUIT DEGREES; 4. PURSUIT SPV). At this point, the eyes continued to move to the right in decaying fashion, until after 250-500 msec and a 4-6 degree movement (measured by parameters 5. PAE TIME and 6. PAE DEGREES respectively), this movement was halted by an abrupt saccade to the left as

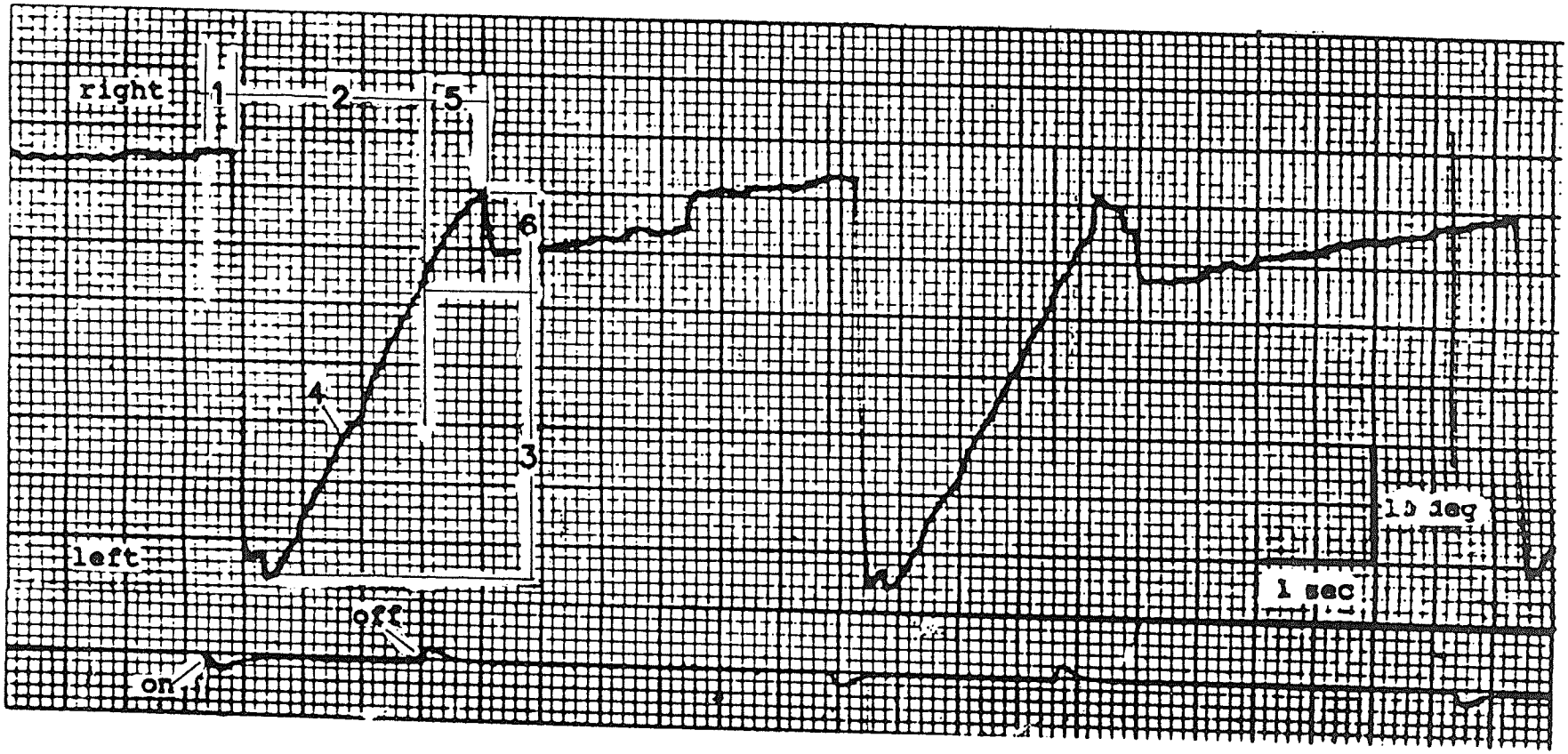

Fig 7. Two typical pursuit events of the PURSUIT INTENSITY series. The top trace shows the EOG eye signal and the bottom line shows the lights on and off signal (depicted on and off respectively). Pursuit parameters are diagrammed as follows: 1. PICKUP LAG; 2. PURSUIT TIME; 3. PURSUIT DEGREES; 4. PURSUIT SPV; 5. PAE TIME; 6. PAE DEGREES. The PAE is depicted by 5 and 6. The first event was used in the analysis, the second event was not.

the subject brought his eyes back to where he thought center was. The subject's eyes then continued to drift towards the right in the darkness after the LED was off. Further corrective saccades to the left were sometimes needed to reposition the eyes at center. The subject was in darkness for 3.2 seconds before the next LED appeared at 30 degrees to his left. LED's were on for 1.6 seconds. Eyes did not drift more than 8 degrees to the right after the LED went off. Subjects reported that the LED was easy to pickup and follow, and that when the LED went off it appeared that it suddenly went behind a curtain. Subjects were unaware that their eyes were drifting right and being corrected. Most pursuit events were like the first event. The second event shown demonstrates an event not used in analysis, as it shows an anomalous eye movement after lights off. Neither of these events show anticipation of the lights on, which would have been demonstrated by an eye movement before the LED appeared.

Table 1 shows the results of the effects of different light intensities on pursuit and PAE parameters over 3 subjects. Values given are means and standard deviations. Overall, as the light intensity is decreased, 1. PICKUP LAG increases (350-485 msec) while 2. PURSUIT TIME (1.36-1.2 sec), 3. PURSUIT DEGREES (22.4-18.5 degrees), and 4. PURSUIT SPV (19.0-15.3 deg/sec) decreases. PAE parameters 5. PAE TIME (313-361 msec) and 6. PAE DEGREES (4.5-5.0 degrees) affected by a decrease in the light intensity. Trends are not obvious as the means are summed over 3 subjects, and

TABLE 1. PURSUIT INTENSITY SERIES: EFFECT OF DIFFERENT LIGHT INTENSITIES ON PURSUIT AND PAE PARAMETERS

INTENSITY LEVEL	PURSUIT PARAMETERS				PAE PARAMETERS	
	1. Pickup Lag (msec)	2. Pursuit Time (sec)	3. Pursuit Degrees (deg)	4. Pursuit SPV (deg/sec)	5. PAE Time (msec)	6. PAE Degrees (deg)
1. No Filter	350 ± 90[1]	1.36 ± .11	22.4 ± 2.7	19.0 ± 1.5	331 ± 95	5.1 ± 1.7
2. F Stop-2 Filter	385 ± 92	1.33 ± .11	23.2 ± 2.7	17.2 ± 2.0	313 ± 97	4.5 ± 1.2
3. F Stop-3 Filter	485 ± 95	1.21 ± .12	18.5 ± 2.8	15.3 ± 1.8	361 ± 101	5.0 ± 1.5

[1] mean and standard deviation summed over 3 subjects

therefore are not taking into account intersubject variability.

Table 2 gives ANOVA results of the effects of different light intensities on the 4 PURSUIT and 2 PAE parameters, taking into account the intersubject variability. There were significant effects on the pursuit parameters 1-4, ($p < .001$ each) but no significant effect on PAE parameters 5 and 6 between light intensities .On pursuit parameters 1-3, there were significant effects (on 1., $p < .01$;2 $p < .05$ and 3; $p < .01$) between pursuit events 1-13 across the intensities, but no significant effects on 4 and the PAE parameters 5 and 6. Intersubject variability was significant in parameter 2 ($p < .005$) and 3-6 ($p < .001$). Bartlett's test for non-homogeneity of variances in replicates was not significant in any parameters. It is concluded that in this experiment, the PAE (pursuit aftereffect) was not due to a decaying afterimage, and that there was a wide enough range of light intensities to elicit changes in pursuit parameters, but not concurrent changes in the aftereffect parameters.

2. Analysis of Effect of Repeated Exposure on Pursuit Intensity Events.

Repeated exposure to pursuit stimuli within one intensity was only looked at in parameters found to have significant effects between intensities, as non-significance between intensities would indicate that repeated exposure to pursuit stimuli was also not significant. PAE parameters 5

TABLE 2. PURSUIT INTENSITY SERIES: ANOVA SPLIT PLOT ANALYSIS OF DIFFERENT LIGHT INTENSITIES ON PURSUIT AND PAE PARAMETERS

ANOVA ANALYSIS	PURSUIT PARAMETERS				PAE PARAMETERS	
	1. Pickup Lag (msec)	2. Pursuit Time (sec)	3. Pursuit Degrees (deg)	4. Pursuit SPV (deg/sec)	5. PAE Time (msec)	6. PAE Degrees (deg)
1. Between Intensities 1 : 2 : 3	$p < .001$	$p < .001$	$p < .001$	$p < .001$	NS	NS
2. Between Events 1 - 13	$p < .01$	$p < .05$	$p < .01$	NS	NS	NS
3. Between Subjects	NS[1]	$p < .005$	$p < .001$	$p < .001$	$p < .001$	$p < .001$
4. Bartlett's Test[2]	NS	NS	NS	NS	NS	NS

[1] NS - not significant
[2] Bartlett's Test - determines any departure of homogeneity of variance of the two replicates of data that was pooled within a subject from the each intensity.

and 6 showed non-significance in the intensity analysis, and were therefore excluded. Table 3 gives the means with standard deviations and ANOVA results of pursuit parameters 1-4 between trials 1 and 6 in intensity 1: NO FILTER. The only significant effect due to repeated exposure was between trials on parameter 4 ($p < .05$). All other ANOVA analysis (between trials on parameters 1-3 and between events on parameters 1-4) were not found to be significant, except for intersubject variability (parameter 1, $p < .005$; 2.NS; 3. $p < .001$; 4. $p < .001$). Due to the significant decrease between trials of parameter 4: PURSUIT SPV ($p < .05$), two multiple comparisons test were carried out to see if the decreases were progressive or random. One was between events within each trial (1 and 6), to ascertain if there were significant differences within a trial (significance $p < .05$). The other was between trials within one event (events 1-13) to determine which events were significantly different (significance in event 9 $p < .05$). On the basis of the above results, habituation to repeated exposure to the pursuit stimuli was concluded not be have any effect on pursuit parameter 1-3 and PAE parameters 5 and 6, and to have minimal effect on parameter 4.

B. PURSUIT STUDY SERIES

1. Analysis of PO, P130, PVAR Decay Fits and General Pursuit Parameters.

TABLE 3. PURSUIT INTENSITY SERIES: EFFECT OF REPEATED EXPOSURE TO STIMULUS ON PURSUIT PARAMETERS WITHIN INTENSITY 1; NO FILTER

ANALYSIS	PURSUIT PARAMETERS			
	1. Pickup Lag (msec)	2. Pursuit Time (sec)	3. Pursuit Degrees (deg)	4. Pursuit SPV (deg/sec)
Trial 1 Means	377 ± 103	1.31 ± .12	24.6 ± 3.2	18.7 ± 1.7
Trial 6 Means	324 ± 78	1.40 ± .11	24.5 ± 2.1	17.3 ± 1.3
ANOVA Analysis Between Trials	NS	NS	NS	$p < .05$
ANOVA Analysis Between Events	NS	NS	NS	NS
ANOVA Analysis Between subjects	$p < .005$	NS	$p < .001$	$p < .001$

A typical pursuit event of this series is shown in figure 8 (subject LW). Fig. 8A shows the raw eye movement and fig. 8B shows the differentiated eye movement. In all figures, the LED appears on the subjects's left (-30 degrees) at 0 seconds, and rotates to the right at 20 deg/sec, where it disappears (dashed line) at center (0 degrees) at approximately 1.6 seconds after appearence. The tracing shows the subject's eye movements. In the raw eye movement (fig. 8A), the subject is initially looking straight ahead ,and after some delay after the light goes on, makes a saccade to his left to fixate and follow the light. When the light is switched off, the subject looks where he last saw the light, and an ensuing decaying eye movement follows until the eyes come to rest. The eye movements in general were smooth. Figure 8B shows the differentiated plot of figure 8A. Each dot represents a 5 msec sampling of the differentiated eye movement. The horizontal line represents the median pursuit slow phase velocity of the eye movement, which in this case was 19.89 deg/sec (fig 8B). The one exponential curve fit to the decay after lights off is shown by the fitted line to the eye movement. In fig. 8B, the decay was started at 130 msec (P130) after lights off. The equation of the line is given below the curve fit, with A, the initial value here being 19.52 deg/sec and B = 2.76. The time constant 1/B is .362 sec and r^2 = .473. The differentiated eye signal was found to be quite noisiey, a finding similar to one made by Zasorin et al., (1983).

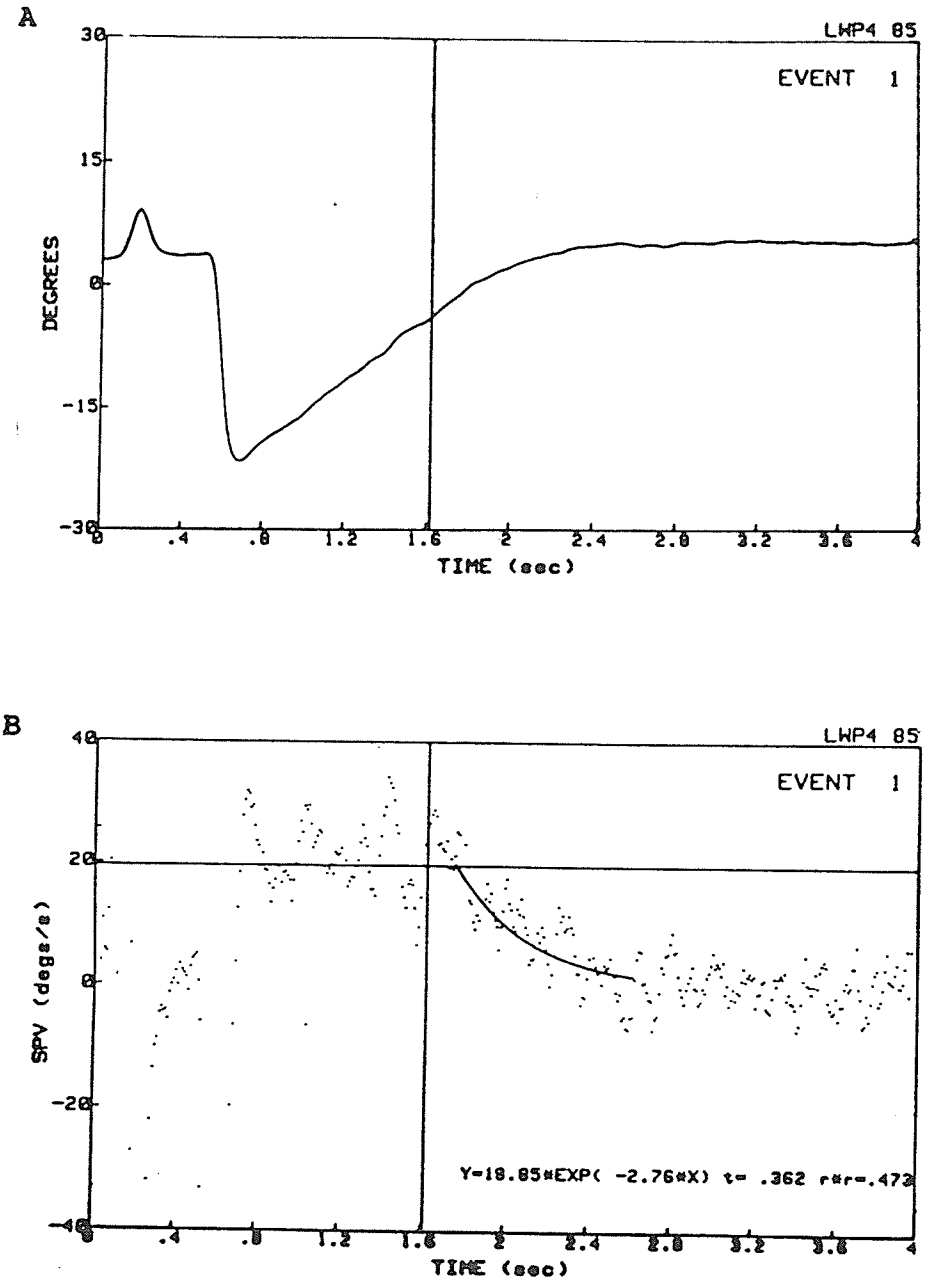

Fig 8. Diagram of a typical pursuit study event from subject LW. A gives the raw pursuit event. Note the smoothness of the movement and the obvious decay after lights out (lights out depicted by a vertical line). B gives the differentiated eye movement with a decay fit starting at 130 msec after lights out (P130). The horizontal line depicts the median pursuit slow phase velocity. Note the noisiness of the signal in B.

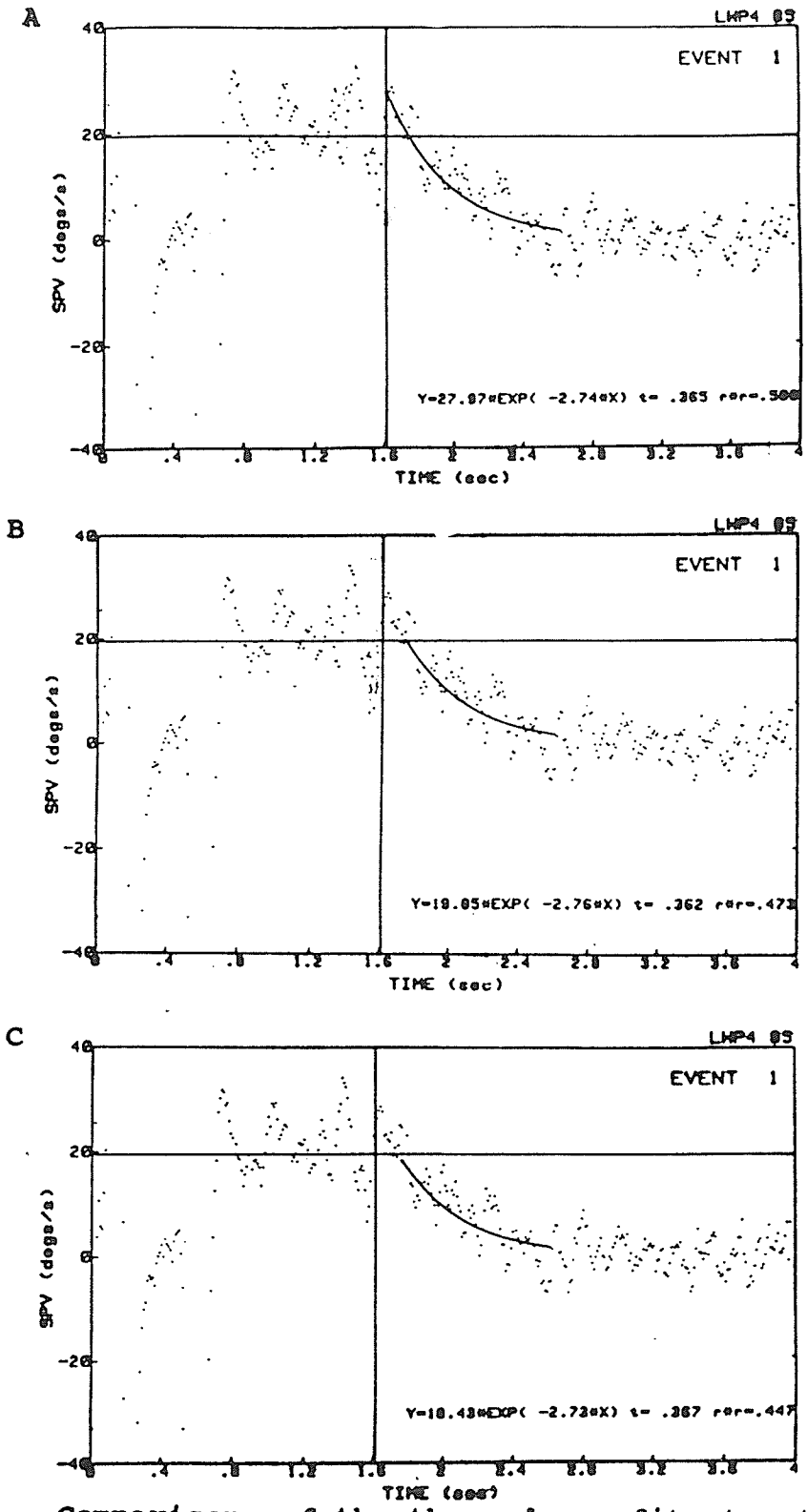

Fig 9. Comparison of the three decay fits to the single pursuit event in fig 8A. A is the decay fit at lights out (vertical line - P0). B is the decay fit at 130 msec after lights out (P130). C is the decay fit at the experimenter's estimation of the decay start (PVAR = 150 msec). The P130 fit is the best fit.

Figure 9 shows the three decay fits to the single pursuit event shown in fig. 8. Fig. 9A depicts the decay fit at lights out (at the vertical line; P0), fig. 9B is the decay fit starting at 130 msec after lights out (P130) and fig. 9C is the decay fit starting at the investigator's estimation of the decay start from the data (PVAR; here equal to 150 msec). All comparisons of decay fits will follow this protocol. Comparison of the decay fits here illustrates the plausibility of any of the decay fits describing the curve. The P0 fit, although appearing to extrapolate over the median pursuit slow phase velocity, looks to be within the range of the scatter of the pursuit velocity. The P130 and PVAR decay fits appear to be little different from each other. The best fit, (being defined as the decay curve with an A (initial) value closest to the median pursuit slow phase velocity), here is the P130 decay fit, with A = 19.85 deg/sec (pursuit velocity = 19.52 deg/sec). Comparison of r^2 values between different fits (P0; P130; PVAR) cannot determine best fit because the r^2 values are determined by the number of points, which are not necessarily the same in the three fits.

Figure 10 shows the consistency of the pursuit events and PAE (pursuit aftereffect) within one trial from the same subject (LW) used in figures 8 and 9. Fig. 10A shows eight events from one trial superimposed over each other. Fig. 10B shows the mean and standard deviations (dashed lines) of the eight events. Figure 11 shows the three decay fits of the mean pursuit event in figure 10. The decay fits (A. P0;

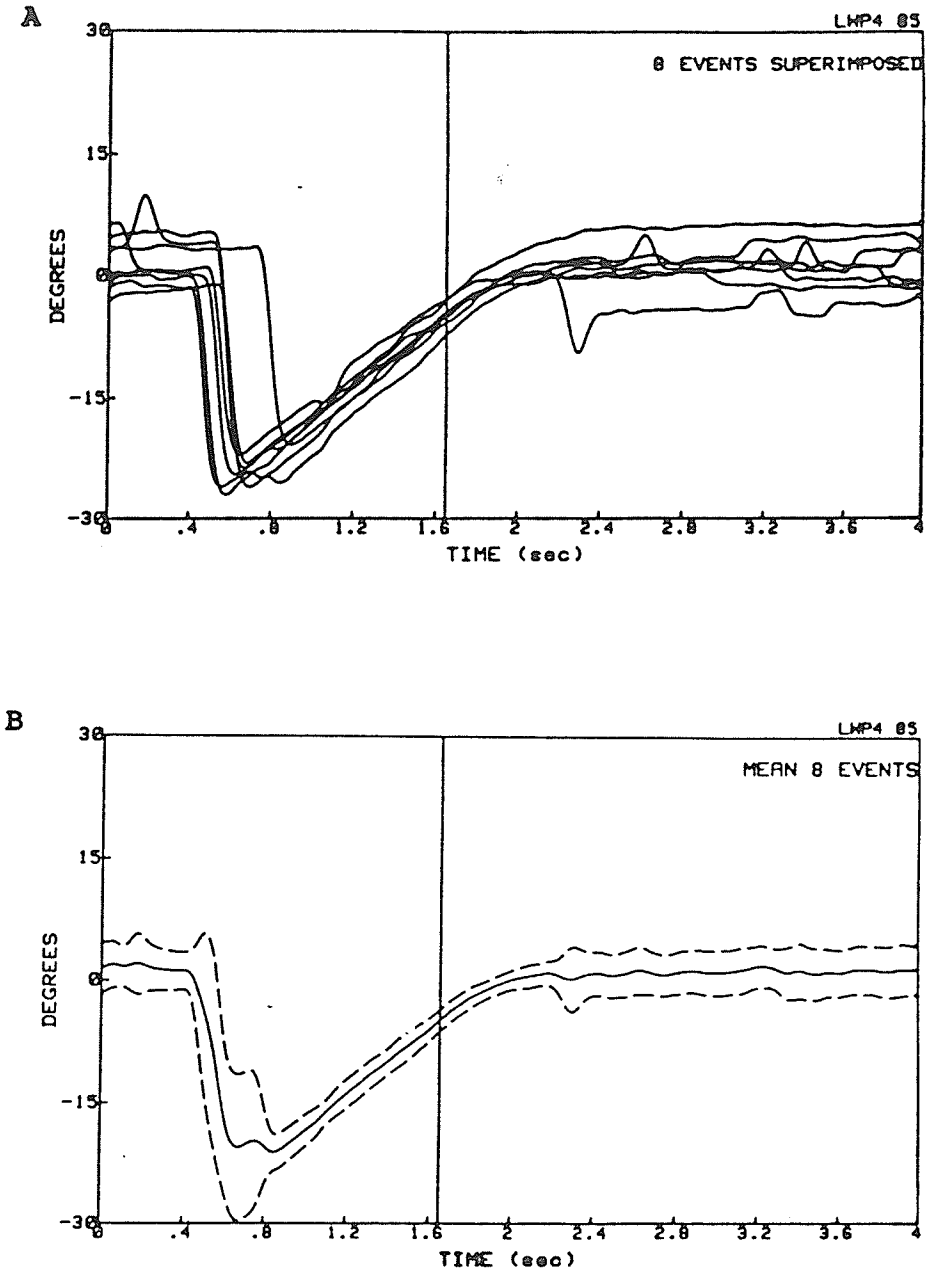

Fig 10. Diagram of the typical consistency of events within a trial from subject LW. A gives the 8 events within a trial superimposed over each other. B gives the mean event with one standard deviation (dashed lines). The vertical line indicates the lights out.

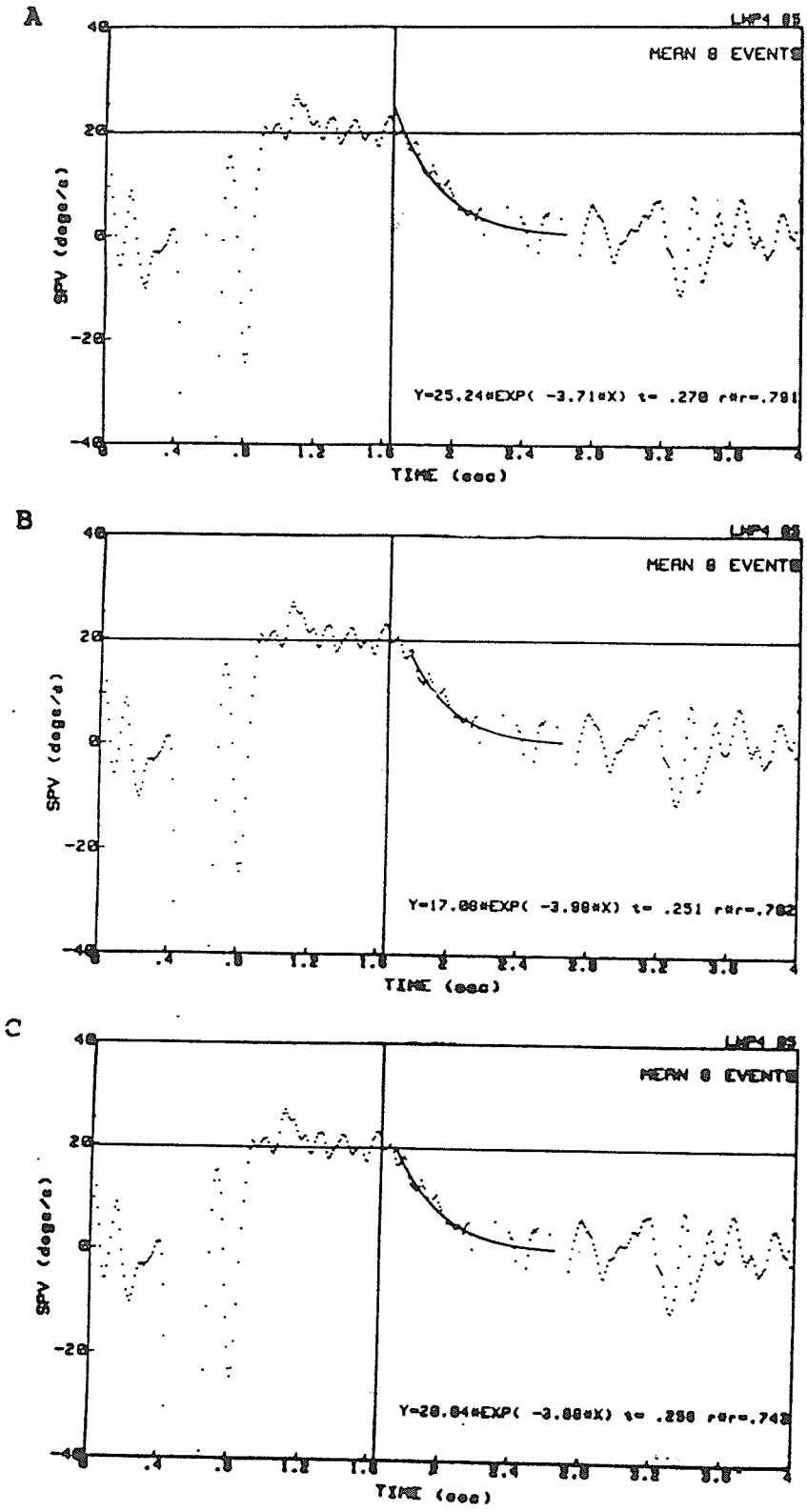

Fig 11. Comparison of three decay fits to the mean event in fig 10. (A. P0; B. P130; C. PVAR = 75 msec). The PVAR fit is the best fit.

B., P130; C. PVAR = 75 msec) here further illustrate the uncertainty of which is the best fit. The PVAR fit here is the best decay fit (A=20.04 deg/sec; pursuit velocity = 19.68 deg/sec). Little difference is apparent between the PVAR and P130 fit. The P0 fit appears not to be plausible here.

Table 4 compares the means and standard deviations of the parameters measured in the three decay fits within a subject, as well as the general parameters of DECAY START (of the PVAR decay), PURSUIT SPV and GAIN (of the pursuit movement). Upon comparison, little difference between decay fit measures of 1/B and tc of P0, P130 and PVAR were found, indicating that the time constant is unaffected by the start of the decay fit. Mean r^2 values changed little between decay fits. Parameter A, the initial value of the fit, does show some differences in means between the decay fits. The mean P0 'A' value (mean = 24.35 deg/sec) is different from the P130 'A' value (mean = 17.77 deg/sec) and the PVAR 'A' value (mean = 17.97 deg/sec), though the P0 'A' value is within the standard deviation of the 'A' value of the other two. The P130 'A' value and the PVAR 'A' value are very similar, and upon examination of the DECAY START (mean = 126 msec), it becomes clear that both decays started at about the same time after the LED was extinguished. The mean PURSUIT SPV was 21.40 $\pm$ 2.12 deg/sec and the GAIN of the system was 1.104 $\pm$ 0.090. From the above data, and from the individual pursuit event data, it was concluded that: 1) there is little difference between the PVAR decay fit and

TABLE 4. COMPARISON OF 3 DECAY FITS (P0, P130, PVAR) OF PURSUIT DATA WITH GENERAL PARAMETERS[1]

DECAY FIT PARAMETERS		SUBJECT		
		1. S.L.	2. A.K.	3. D.F.
A	P0	27.98 ± 5.31	22.24 ± 4.18	22.29 ± 5.77
	P130	22.48 ± 4.82	14.56 ± 4.80	15.27 ± 5.00
	PVAR (deg/sec)	21.78 ± 4.76	16.61 ± 3.58	16.60 ± 5.31
B	P0	3.153 ± 0.952	3.675 ± 1.100	4.631 ± 1.190
	P130	3.569 ± 1.382	3.336 ± 1.214	4.664 ± 1.455
	PVAR	3.538 ± 1.348	3.705 ± 1.113	4.960 ± 1.574
1/B	P0	0.352 ± 0.139	0.305 ± 0.142	0.239 ± 0.074
	P130	0.328 ± 0.137	0.343 ± 0.166	0.239 ± 0.081
	PVAR (sec)	0.323 ± 0.126	0.304 ± 0.148	0.225 ± 0.081
r^2	P0	0.635 ± 0.110	0.548 ± 0.131	0.422 ± 0.139
	P130	0.548 ± 0.121	0.408 ± 0.185	0.323 ± 0.156
	PVAR	0.545 ± 0.120	0.468 ± 0.128	0.352 ± 0.144
DECAY PVAR START (msec)		145 ± 88	91 ± 61	118 ± 82
PURSUIT SPV (deg/sec)		22.83 ± 1.46	22.72 ± 1.16	20.69 ± 3.27
GAIN		1.187 ± 0.073	1.208 ± 0.134	1.096 ± 0.068

[1]mean and standard deviations summed over all pursuit events

TABLE 4. COMPARISON OF 3 DECAY FITS (P0, P130, PVAR) OF PURSUIT DATA WITH GENERAL PARAMETERS (CONTINUED)

DECAY FIT PARAMETERS		SUBJECT		OVERALL MEAN
		4. L.W.	5. A.P.	
A	P0	25.06 ± 5.21	24.43 ± 5.12	24.35 ± 6.16
	P130	19.21 ± 4.47	17.81 ± 4.06	17.77 ± 6.43
	PVAR (deg/sec)	18.54 ± 3.21	17.26 ± 2.91	17.97 ± 4.65
B	P0	3.672 ± 1.320	3.427 ± 0.786	3.712 ± 1.141
	P130	4.009 ± 1.746	3.500 ± 1.026	3.813 ± 1.418
	PVAR (sec)	4.231 ± 1.919	3.633 ± 1.007	4.112 ± 1.396
1/B	P0	0.309 ± 0.130	0.308 ± 0.071	0.303 ± 0.111
	P130	0.309 ± 0.138	0.312 ± 0.099	0.306 ± 0.124
	PVAR (sec)	0.287 ± 0.139	0.302 ± 0.081	0.288 ± 0.115
r^2	P0	0.511 ± 0.111	0.480 ± 0.117	0.519 ± 0.121
	P130	0.450 ± 0.160	0.383 ± 0.126	0.422 ± 0.150
	PVAR	0.441 ± 0.119	0.385 ± 0.116	0.438 ± 0.125
DECAY PVAR START (msec)		142 ± 85	135 ± 70	126 ± 77
PURSUIT SPV (deg/sec)		19.39 ± 3.41	21.38 ± 1.28	21.40 ± 2.12
GAIN		0.980 ± 0.171	1.051 ±0.051	1.104 ± 0.190

the P130 decay fit, and the P130 decay fit was the best representation of the PAE as it was better defined than the PVAR decay (ie. discreet start) and had previous justification (signal precessing time = 130 msec; Robinson, 1981); 2) The P0 decay fit did not adequately describe the decay curves because in many cases, high A values indicated an extrapolation of the decay fits beyond the actual data. Therefore the P130 decay fit was chosen as the best representation of the data and was used for further analysis.

This does not invalidate the decay fits of P0 and PVAR. Some data appeared to start their decay at P0, but more data clearly showed an extrapolation of the curve fit. This is illustrated in figure 12 and 13 by a mean of ten events within a trial with subject SL. Fig. 12A gives the superimposed raw movement while fig. 12B gives the mean event with standard deviation. Fig. 13 gives the three-decay fits (A. P0; B. P130; C. PVAR = 150 msec) for the mean pursuit event in fig. 12. Fig. 13A (P0 fit) clearly indicates extrapolation of the fit, and is not the best fit to the data. The other two fits (B. P130 and C. PVAR) are not clearly indistinguishable from each other. A clear example of a PVAR decay fit (PVAR = 230 msec) is given in fig. 14 and 15 for subject AP. Fig. 14A gives the raw pursuit event and fig. 14B gives the overall preferred P130 fit. Fig. 15 gives the three decay fits (A. P0; B. P130; C. PVAR = 230 msec). Fig. 15c clearly shows the start of the decay to be much later than 130 msec, yet the P130 decay

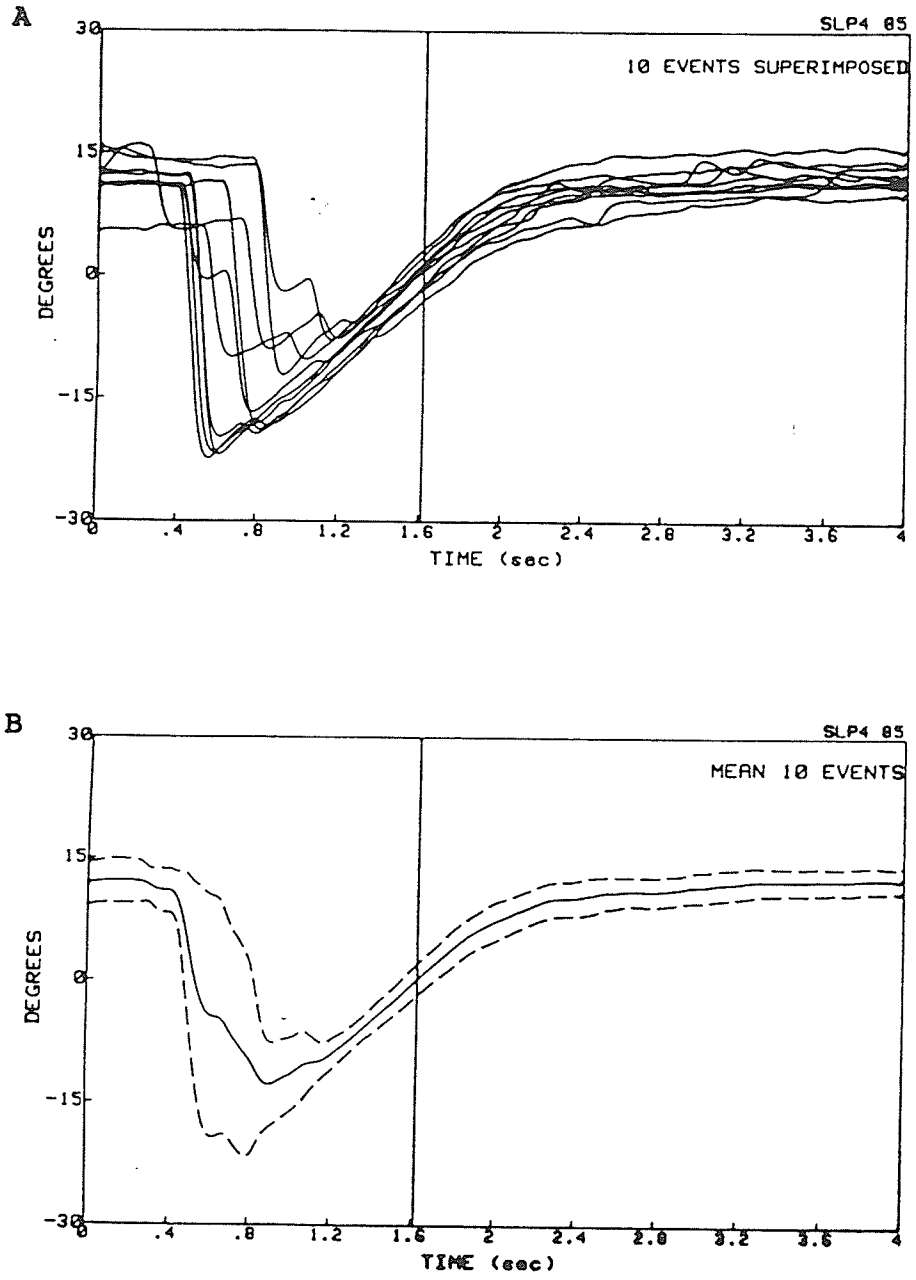

Fig 12. Diagram of the typical consistency of events within a trial from subject SL. A gives the 10 events within a trial superimposed over each other. B gives the mean event with one standard deviation (dashed line). The vertical line indicates the lights out point.

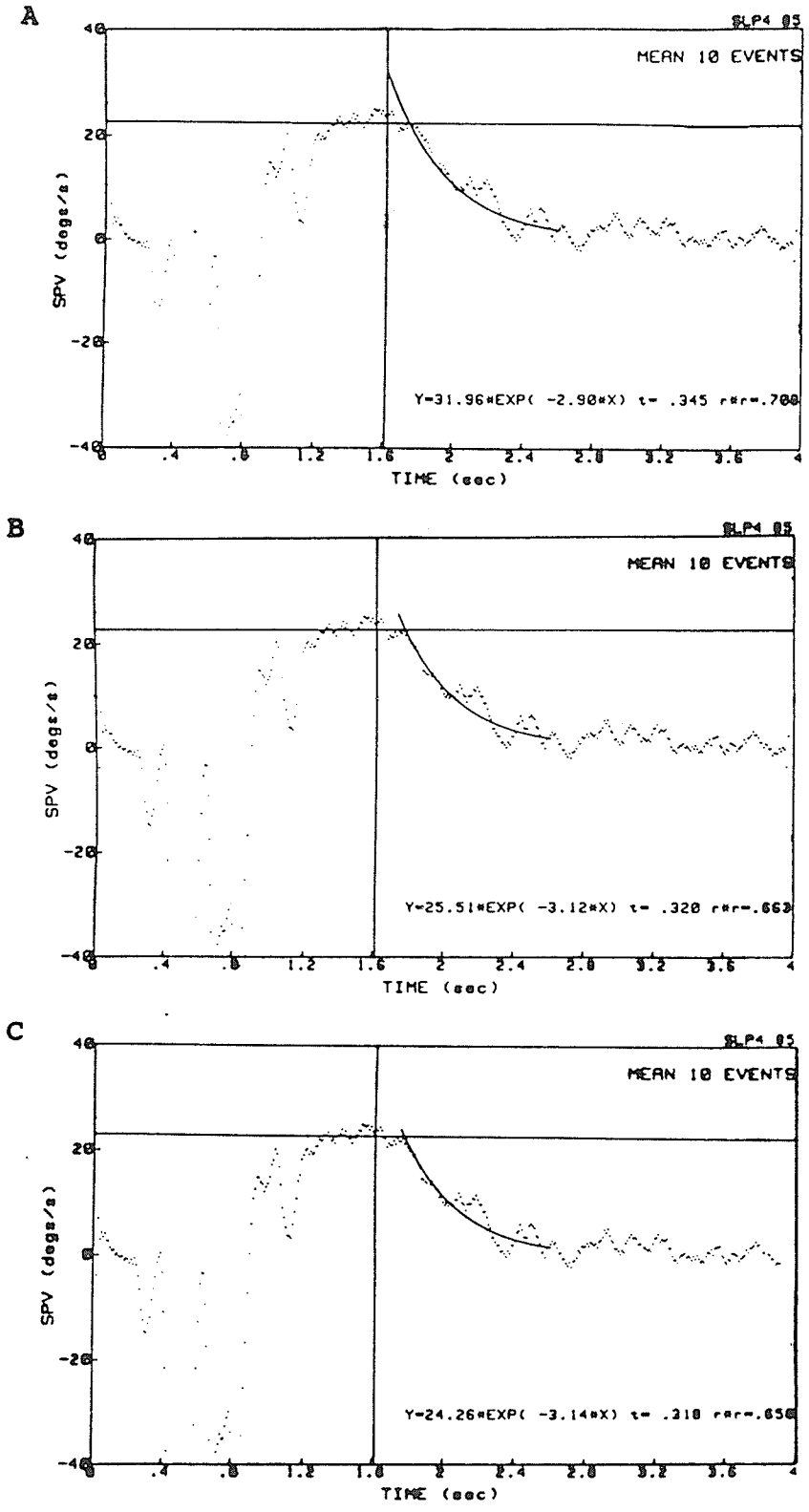

Fig 13. Comparison of three decay fits to the mean event in fig 12. (A. P0; B. P130; C. PVAR = 150 msec). Note the extrapolation of the P0 fit in A.

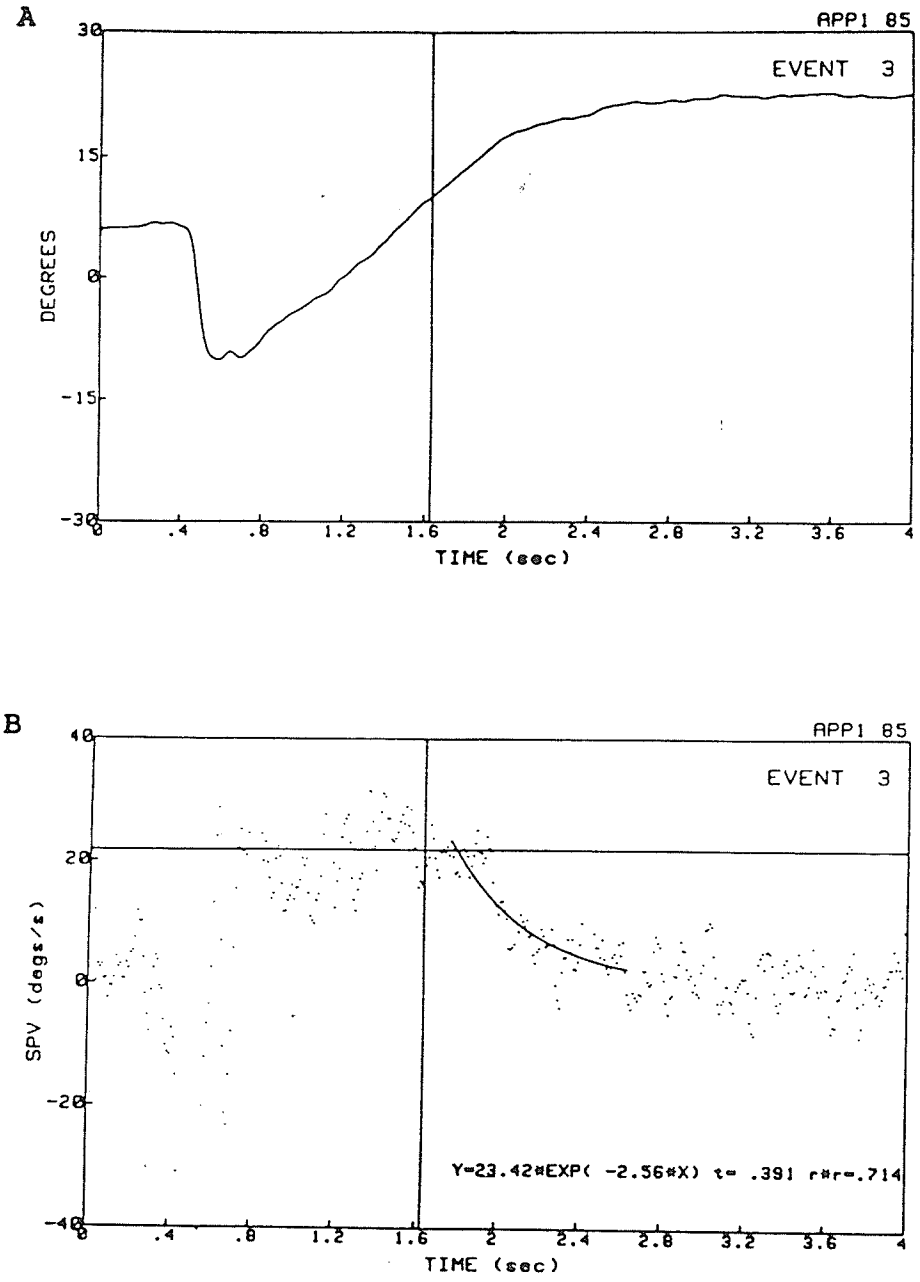

Fig 14. Diagram of a pursuit event from subject AP. A gives the raw event while B gives the preferred P130 decay fit.

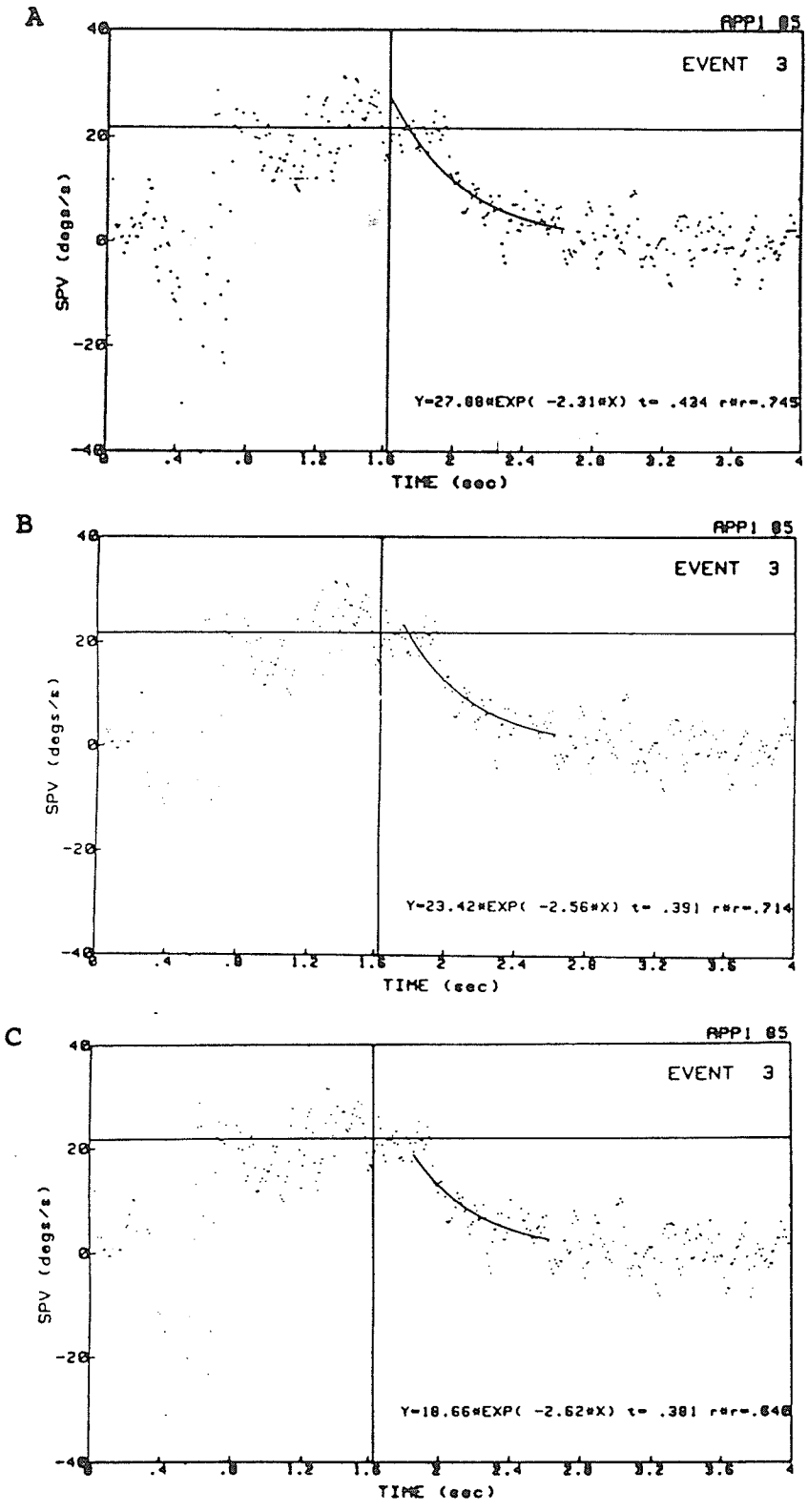

Fig 15. Comparison of three decay fits to the single event in fig 14. (A. P0; B. P130; C. PVAR = 230 msec). Note the clear PVAR decay start in C.

fit, as defined previously, is the best fit.

2. Analysis of Effects of Repeated Exposure on P130 Decay Fits.

Table 5 gives the means, overall means with standard deviations and ANOVA analysis of repeated exposure to the pursuit stimulus. This analysis shows that repeated exposure had no effect on the pursuit parameters studied. Trials 2, 3 and 4 were only analysed for repeated effect, as these trials were only used for further analysis against the three OKN - OKAN runs. Four parameters were studied. All trial means in the decay fit parameters (A and 1/B) and the general parameters (PURSUIT SPV and GAIN) were within one standard deviation of the overall means (A = 16.57 deg/sec; 1/B = 0.296 seconds; PURSUIT SPV = 21.35 deg/sec; GAIN = 1.090). ANOVA results between trials and between events in all parameters were not significant in repeated exposure to the stimulus. Intersubject variability was not significant in PURSUIT SPV, but was significant at a level $p < .001$ in parameter A. Repeated exposure to the stimulus had no effect on the pursuit and PAE response.

3. Analysis of OKAN Parameter.

A number of parameters were studied in OKAN. Table 6 gives the means and standard deviations of these parameters within each subject and the overall means. The first component of the two component decay had initial values A =

TABLE 5. EFFECTS OF REPEATED EXPOSURE TO PURSUIT STIMULI ON P130 DECAY FITS AND GENERAL PURSUIT PARAMETERS[1]

ANALYSIS	DECAY FIT PARAMETERS		PURSUIT PARAMETERS	
	A (deg/sec)	1/B (sec)	PURSUIT SPV (deg/sec)	GAIN
Trial 2	17.85	0.279	21.45	1.098
Trial 3	16.54	0.311	21.47	1.090
Trial 4	15.32	0.299	21.71	1.108
Overall Mean	16.57	0.296	21.35	1.090
ANOVA BETWEEN TRIALS	NS[2]	NS	NS	NS
ANOVA BETWEEN EVENTS	NS	NS	NS	NS
ANOVA BETWEEN SUBJECTS	$p < .001$	NS	NS	$p < .05$

[1] mean of 5 subjects
[2] NS = not significant

TABLE 6. OKAN PARAMETERS[1]

SUBJECT	PARAMETER						
	A (deg/sec)	1/B (sec)	C (deg/sec)	1/D (sec)	CUM DISP (deg)	# SP (#)	SP DUR (sec)
1. S.L.	18.88 ± 2.91	0.776 ± 0.094	3.99 ± 1.05	14.16 ± 6.90	27.7 ± 9.8	34 ± 10	0.254 ± 0.042
2. A.K.	15.28 ± 1.11	0.750 ± 0.292	4.75 ± 1.16	54.71 ± 28.29	84.8 ± 24.3	72 ± 20	0.320 ± 0.006
3. D.F.	6.20 ± 4.31	0.933 ± 0.022	4.54 ± 3.79	10.18 ± 2.73	23.7 ± 14.0	32 ± 8	0.261 ± 0.056
4. L.W.	16.95 ± 7.27	0.870 ± 0.069	5.52 ± 3.05	20.02 ± 4.36	59.9 ± 31.0	66 ± 20	0.267 ± 0.029
5. A.P.	20.41 ± 1.48	0.690 ± 0.026	4.18 ± 1.14	21.52 ± 7.90	43.1 ± 12.3	38 ± 19	0.335 ± 0.009
OVERALL MEAN	15.54 ± 6.05	0.797 ± 0.127	4.48 ± 1.83	24.12 ± 19.4	47.8 ± 27.7	48 ± 21	0.287 ± 0.059

[1]means and standard deviations over 3 OKAN trials

15.54 deg/sec and 1/B = 0.797 seconds. The second component had initial values C = 4.48 deg/sec and 1/D = 24.12 seconds. The cumulative displacement (total number of degrees displaced by the eye) was 47.8 degrees, the number of slow phases (#SP) was equal to 48, and the slow phase duration (SP DUR) was equal to 0.287 seconds. Intersubject variability was somewhat high, but all values fall within normal ranges described by Jell et al., (1983).

4. Comparison of P130 Decay Parameters and OKAN First Component Decay Fits.

The object of this study was to compare the pursuit generated decay (PAE) with the first component of the OKAN two component deay. Table 7 gives the means with standard deviation within one subject, and the overall means of decay parameters A and 1/B in the P130 decay and the OKAN first component decay. The overall parameter 'A' mean in the P130 decay fit was 17.77 $\pm$ 6.43 deg/sec, compared to 15.54 $\pm$ 6.05 deg/sec in the OKAN first component decay fit. The overall parameter 1/B mean in the P130 decay fit was 0.292 $\pm$ 0.120 seconds, compared to 0.797 $\pm$ .127 seconds in the OKAN first component decay fit. A t-test was performed to determine statistical significance of the results as variances were unequal, invalidating split plot analysis. Results were not significant ($p > 3.35$) for A and significant at a level $p < .001$ for 1/B. This indicates that the time constant is different.

TABLE 7. COMPARISON OF PURSUIT P130 DECAY PARAMETERS WITH FIRST COMPONENT OF TWO COMPONENT OKAN PARAMETERS[1]

SUBJECT	PURSUIT P130 PARAMETERS		OKAN FIRST COMPONENT PARAMETERS	
	A (deg/sec)	1/B (sec)	A (deg/sec)	1/B (sec)
1. S.L.	22.48 ± 4.82	0.328 ± 0.142	18.88 ± 2.91	0.766 ± 0.094
2. A.K.	14.56 ± 4.80	0.317 ± 0.131	15.28 ± 1.11	0.750 ± 0.292
3. D.F.	15.27 ± 5.00	0.231 ± 0.079	6.20 ± 4.31	0.933 ± 0.022
4. L.W.	19.21 ± 4.47	0.300 ± 0.146	16.95 ± 7.27	0.870 ± 0.069
5. A.P.	17.81 ± 4.06	0.301 ± 0.104	20.41 ± 1.48	0.696 ± 0.026
OVERALL MEAN	17.77 ± 6.43	0.292 ± 0.120	15.54 ± 6.05	0.797 ± 0.127

[1]mean and standard deviations summed over 3 pursuit and OKAN trials respectively

C. EXPERIMENTAL CONTROL ANALYSIS

1. PAE Computer Decay Fit Program.

This analysis was to look at how accurately the computer program represented the pursuit event and fit the decay curve. Figure 16 illustrates the synthesized pursuit event. Fig. 16A depicts the raw event based upon a 20 degree pursuit event decaying at time zero with a time constant of 0.25 seconds. Fig. 16B depicts the P0 decay fit and fig. 16C depicts the P130 decay fit. Parameter values for the event (A = 19.89 deg/sec; B = 4.00; 1/B= .250 seconds; r^2 = 1.000) determined by the computer program were in agreement with the synthesized values. A value of r^2 = 1.000 indicated a completely accurate fit. Fig. 16C illustrates a P130 decay fit that is a poor representation of the data. Even though the fit is good (r^2 = 1.000), the fit is not the best fit because A = 11.80 deg/sec does not approximate the PURSUIT SPV = 20.00 deg/sec. The time constant (tc) and B value were the same in both fits. We concluded that the computer decay fit program accurately represented the data.

2. NO EOG Signal Control.

This analysis was to look at how much noise was in the eye signal and processing system. Figure 17 illustrates the NO EOG input. The signal was flat and biased by +2.90 degrees. Fig. 17B gives the differentiated signal in the same scale as shown in the differentiated pursuit events.

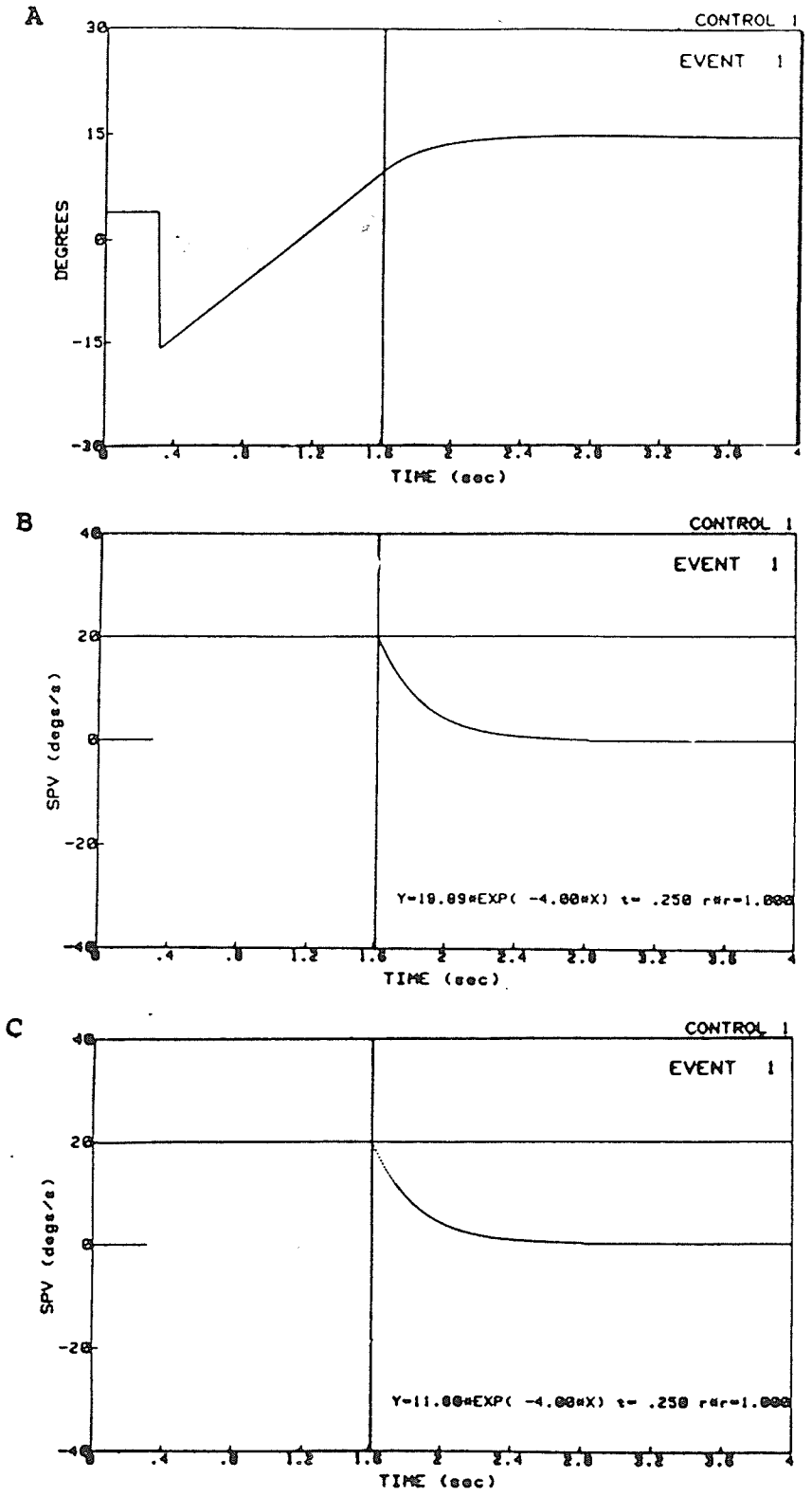

Fig 16. Control 1 depicts the synthesized pursuit event to check the computer decay fit program. A depicts the raw event, B depicts the P0 decay fit, C depicts the P130 decay fit. Note that "best fit" is B, the P0 fit.

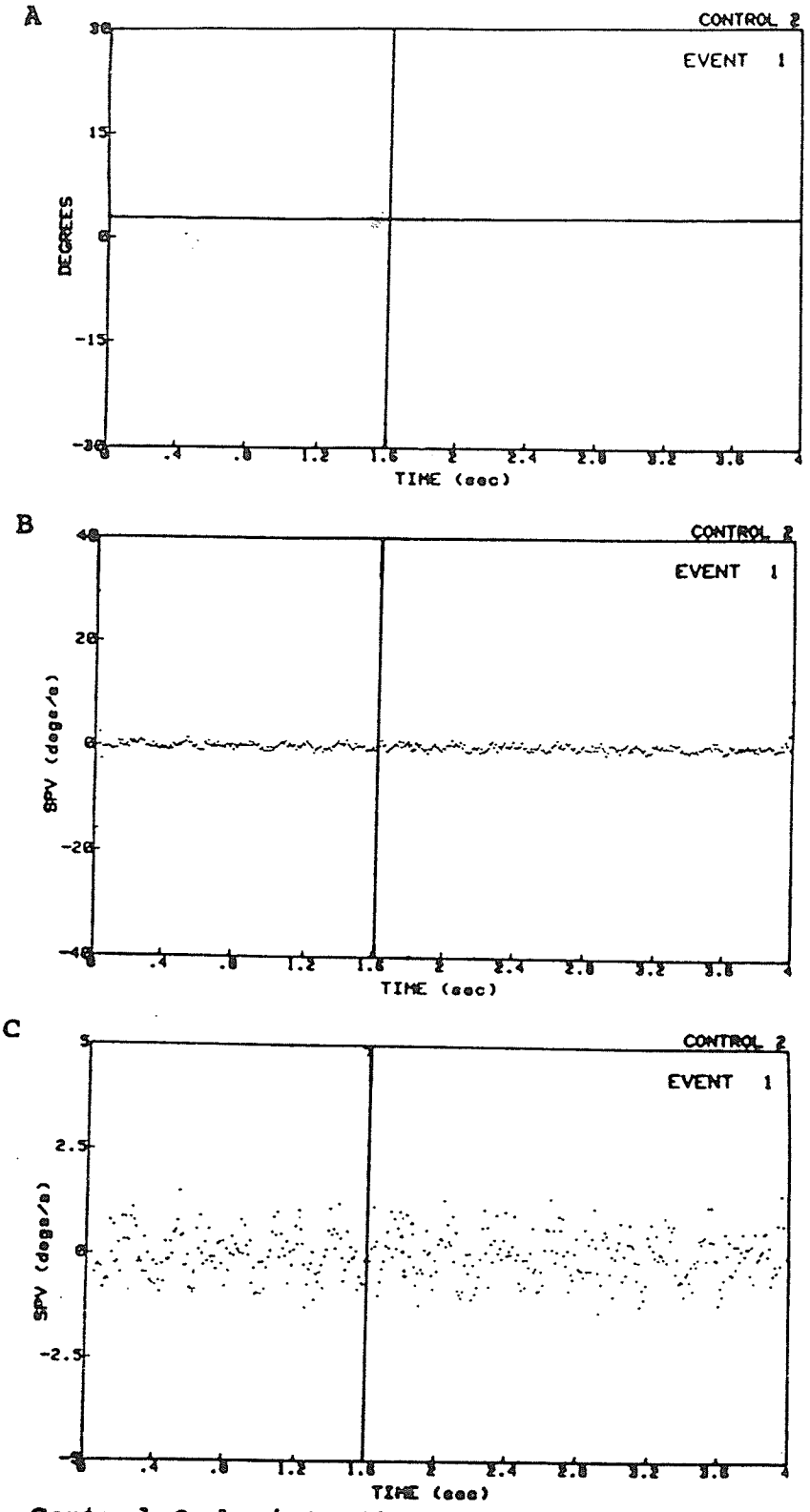

Fig 17. Control 2 depicts the NO EOG signal processed by the computer program. A depicts the raw event. B depicts the differentiated plot comparable with the differentiated pursuit events. C depicts an expanded differentiated plot. The noise of the system was ± 1.5 deg/sec.

This illustrates the magnitude of variability of the signal as compared to the pursuit event. Fig. 17C gives an expanded differentiated signal. This illustrates the variability of the signal. The average velocity (SPV) was less than 0.01 deg/sec. The positive peak velocity was +1.858 deg/sec, the lowest was -1.985 deg/sec, with a standard deviation of $\pm$0.6473 deg/sec. This indicated an absolute range of about 4 deg/sec, and a standard range of about 1.3 deg/sec. We concluded that an error of $\pm$0.65 deg/sec is the experimental error in the measurement system, and did not interfere with our experimental results.

DISCUSSION

A. ANALYSIS OF EFFECTS OF LIGHT INTENSITY ON THE PURSUIT AND PAE PARAMETERS

Our results show that luminance has effects on human smooth pursuit but not on the PAE (pursuit aftereffect). As the luminance is decreased, the reaction time is longer and the amount of time, number of degrees, and eye velocity with which the target is viewed are reduced.

Similar luminance effects on pursuit of a sinusoidal target have been reported by Wheeless et al., (1967). Conflicting evidence that luminance only affects reaction time, and not overall pursuit characteristics, has been put forward by Winterson and Steinman., (1978). Wheeless et al., (1967) attempted to look at foveal pursuit of photopic (cone vision) and scotopic (rod or night vision) targets, and found differences between luminances on gain between photopic and scotopic pursuit and within the range of the photopic stimulus. Winterson and Steinman, (1978) argued that in Wheeless, (1967), the decrease in pursuit characteristics (ie. gain) between the photopic and scotopic foveal targets could be explained by the lack of visibility of the scotopic target on the fovea. When the target was scotopic, its luminance was below foveal threshold, and hence the target would be invisible on the fovea, and invisible at least part, if not all the time to the subject as the target was foveated. They could not reconcile the

differences between their results (decreasing photopic luminance having no effect on gain) and the Wheeless, (1967) data on decreasing photopic luminance decreasing the gain of foveally pursued sinusoidal targets (Winterson and Steinman, 1978).

Our results indicate that we used an adequate range of luminances to show systematic changes in pursuit characteristics but not in the PAE, demonstrating here that our PAE is not due to a visible decaying afterimage. If the PAE had been due to a visible decaying afterimage, a concurrent systematic decrease in PAE parameter as in pursuit parameters would have been expected, as the duration of a visibly occurring afterimage is proportional to the stimulus input (Brindley, 1959). Our pursuit stimulus did not enter the scotopic range of luminance, as at all intensities the stimulus was reported to look similar and equally visible, and did not "disappear" when foveated. As we did not intentionally elicit a visible afterimage through our pursuit stimulus, we do not know the true effect of a decaying afterimage on the PAE. Bahill et al., (1983) showed that pursuit in a lighted room of a moving dot that was abruptly halted and left visible, elicited a smooth PAE decay. The decay had a duration of 400 msec and was ended with a refixation of the still visible dot. This evidence would suggest that any visible stimulus would not affect the PAE. Though pursuit of afterimages has been demonstrated (Heywood and Churcher, 1971; Steinbach and Pearce, 1972), no results have been put forward indicating that decaying of

the afterimage could be responsible for a PAE. On the contrary, the smoothness of the PAE negates pursuit of an afterimage, as all afterimage pursuits were jerky and conscious.

B. ANALYSIS OF EFFECTS OF REPEATED EXPOSURE ON PURSUIT EVENTS.

Previous research has shown that expectations about the direction of future target motion always affects smooth eye movements (Bahill et al., 1983; Kowler et al., 1984). Prediction can occur within presentation of one target motion (Kowler et al., 1984), or within a quater cycle of a sinusoidally moving target (Bahill et al., 1983). Prediction occurs whether or not the target velocity or direction of motion is unpredictable (Bahill et al., 1983; Kowler et al., 1984). Prediction of target motion has been measured by anticipatory smooth eye velocities (Kowler et al., 1984), zero latency tracking of a sinusoidal moving target and predictive tracking of a non-predictive sinusoid target (Bahill et al., 1983), and augmented eye velocity with repeated stimulation (Whittaker and Eaholtz, 1982). Pursuit models have included a predictive component to account for prediction (Bahill and McDonald, 1981; Eckmiller, 1981). The input to the predictive component has been hypothesized to be a corollary discharge (either efference or re-efference copy) and perceived retinal image information (Yasui and Young, 1984), based on a perceptual

feedback hypothesis (Young, 1977), for which there is some neurophysiological evidence (Miles and Fuller, 1975). The corollary discharge input is necessary for prediction, since the retinal image information is normally maintained small, and therefore less predictable (Yasui and Young, 1974).

Our results indicate that there were no significant effects of repeated exposure on pursuit events. Few pursuit events had shown anticipatory eye movements before appearence of the LED, and these events were not used in analysis. Because we found no significant effects of repeated exposure on pursuit events in our experiment, we were able to group all our events together. This does not mean that there were no predictive components involved in our experiment. With the repetitiveness of the stimulus, one would expect some process of prediction to be occurring. This could be in the form of an unconscious attenuation (ie. pre-priming of synapses) to the stimuli, as a conscious attenuation can be defined in the form of an anticipatory saccade before target appearence. There is no evidence to suggest that the predictive component has any effect on the output of the pursuit integrator. On the contrary, Becker and Fuchs, (1985) demonstrated a decaying eye movement followed by a continuing predictive tracking of the disappearing horizontal moving dot, showing no interference of the predictive movement on the decaying eye movement. We concluded that prediction had no significant effects on our pursuit and PAE parameters.

C. CHARACTERISTICS OF THE PURSUIT EVENTS AND THE PAE IN GENERAL.

The pursuit events in both series (PURSUIT INTENSITY series and PURSUIT STUDY series) were standard and reproducible. The pursuit of the LED after foveation was smooth with some fluctuations in pursuit eye velocity. Initial foveation, or foveation with one corrective saccade, of the moving LED indicated the obvious visual nature of the stimulus input (Deubel et al., 1982). Fluctuations in pursuit velocity (ie. eye tracking faster and/or slower than stimulus) indicated that retinal slip was occurring during the experiment. This is to be expected, as the major input to the pursuit system is retinal image slip on the fovea (Young, 1977). Non-foveal following may have occurred due to the variability of the pursuit velocity. We assume this to be minimal, as there were no large catch-up saccades evident in our pursuit movements. The fluctuations in pursuit velocity may indicate that miniature eye movements and saccades were occurring. This has been reported as a normal part of smooth pursuit movements (Steinman et al., 1973; Bergenius, 1984).

The PAE in the pursuit intensity series was elicited by different instructions than in the pursuit study series, yet both showed a similar initial decaying eye movement for the first 300 msec. This would indicate that the same hypothesized pursuit integrator was discharging, and further

eye movements in the pursuit intensity series after a mean of 300 msec were due to subject instruction. In the pursuit intensity events, subjects were instructed to refixate their eyes in the center after the stimulus light went off. Initiation of the first saccade to refixate the eyes to center occurred after a mean of 300 msec. In the pursuit study events, subjects were instructed to look where they "last saw the light". The time constant of the decay was approximately 300 msec. Since the mean initial refixation of the eyes was equal to the time constant of the integrator, our evidence would suggest that involuntary eye movements may be inhibited when the integrator is discharging. The eye movement signal need not be a visual input, as demonstrated by the imaginary refixation in total darkness, but it could be an efference copy signal. Similar results (absence of refixation signal still eliciting a decay) were given by Bahill et al., (1983). They further showed that when there is a visual refixation signal (ie. halting of moving dot), the eyes still show a characteristic decay, similar to when there is no refixation signal. This demonstrates that a voluntary eye movement may also be inhibited when the integrator is discharging, even when there is a larger visual error signal input to the eye movement. The PAE reflex therefore seems to be due to a component that is distinct from other eye movements initiated by the subject.

If efference copy and retinal image signals affect the PAE in a similar manner, what charges the integrator

causing the PAE? The charging up of the integrator in our experiment could be due to either retinal image input or efference copy signals, or both. The primary signal for the eye movement is from the retinal image signal, as the eyes use a saccade to fixate the light (Deubel et al., 1982). The pursuit of the light is maintained by the visual signal, and this signal could charge the pursuit integrator. But the eye muscles are moving concurrently with an efference copy signal, and this signal could also have input into the integrator. Yasui and Young, (1984) hypothesized that both might have input into the predictive component of the pursuit system, and it is easily conceivable to extend this to the pursuit integrator component. Our experiment cannot differentiate these inputs. This point needs further investigation. It is also not certain whether a position signal or velocity signal charges the integrator, although it has been hypothesized that the position signal has a charge input to the optokinetic integrator (Waespe et al., 1985).

The PAE is certainly a real phenomenon. It continued for around 300 msec andit was a consistent and repeatable response. The decay curve fit and parameters represented the data well.

D. ANALYSIS OF THE PAE DECAY FITS.

The results indicate that the best decay curve fit (P0,

P130, and PVAR) was the P130 decay fit for two reasons. One is because the P130 decay fit adequately described the pursuit decay. The fit closely approximated the actual points, and a delay in the start of the decay seemed to be closely approximated by the 130 msec start of the decay after lights out. Another reason is that the PVAR start (variable decay start approximated by the investigator) had a mean start of 126 $\pm$ 77 msec, indistinguishable from the mean P130 decay start (130 msec). The delay in the start of the decay has been reasoned to be due to signal processing, and has been estimated to be 130 msec (Rashbass, 1961; Robinson, 1965), but other values of the delay have been given (150 msec; Yasui and Young, 1984). The P0 fit clearly did not represent the data well.

For purposes of comparison of the pursuit decay fit with the first component of the two component OKAN fit, the P130 decay fit was used. The values of the P130 decay fit were A = 17.77 $\pm$ 6.43 deg/sec; B = 3.813 $\pm$ 1.481; 1/B = 0.306 $\pm$ 0.124 seconds; and r^2 = 0.422 $\pm$ 0.150. 'A' values gave a gain close to 1, indicating the decay started around the stimulus velocity. r^2 values were not as high as expected (0.422 $\pm$ 0.150), but can be explained on a number of grounds. Firstly, the experimental signal processing introduced a variability of $\pm$1.5 deg/sec to the generated signal (see E. EXPERIMENTAL CONTROLS), which would contribute to a lower r^2 value. Secondly, the decay fit procedure is prejudiced towards the lower points in the curve. A greater number of points in the lower part as

compared to the upper part would decrease the goodness of fit of the decay curve due to a greater sum of squares error, and this would give more weight of the curve fitting procedure was given to the lower part. This was demonstrated two ways. One was through a higher r^2 value ($r^2 = 0.519 \pm 0.121$) in the P0 fit than the P130 fit ($r^2 = 0.422 \pm 0.150$) due to more points in the higher part of the curve being taken into consideration in the P0 fit, even though the P0 fit clearly was not a better representation of the data. Another way was when a 1.5 second decay fit gave lower r^2 values due to an abundance of lower points (unreported data). This prejudice would contribute to a lower r^2 value. Thirdly and most importantly, lower r^2 values than expected could be explained by physiological noise from the oculomotor plant. This physiological noise, exaggerated by the digital differentiation process, has been described in pursuit eye movements (Steinman et al., 1973; Deubel et al., 1982; Bergenus, 1984), and similar arguments of unconscious miniature eye movements and corrective saccades can be applied to the pursuit decay. Further, Schalen, (1980) reported randomly occurring square wave forms without any corrective function during eye movements, of which our results did show some rare examples. All these incongrouous eye movements would contribute the major reason for lower r^2 values. r^2 values probably reflected more of the oculomotor noise than the actual decay fit, which could be better judged on its visual representation of the data.

E. CHARACTERISTICS OF THE EXPERIMENTAL CONTROLS.

Control 1 was to determine the consistency of the computer decay program by feeding in a synthesized pursuit event. Processing by the program demonstrated that the decay program was working correctly.

Control 2 gave the No EOG signal control for the error of the differentiated signal. The standard deviation of points with no signal input was $\pm$0.65 deg/sec. Since we assume our eye movement recording system (dc EOG) to have an error of 2.0 deg/sec (Schlag et al., 1983), the deviation of the no EOG signal is within our experimental error. The offset of the signal was +2.90 degrees. This did not affect the results, the analysis was concerned only with measurement of velocity and relative displacement, not absolute displacement. It was concluded that the EOG recording system and the computer decay fit program were adequate for the purposes of this project.

F. CHARACTERISTICS OF THE OKAN.

The OKAN elicited was normal. Values for the first component were A = 15.54 $\pm$ 6.05 deg/sec, 1/B = 0.797 $\pm$ .127 sec. Values for the second component were C = 4.48 $\pm$ 1.83 deg/sec, 1/D = 24.12 $\pm$ 19.4 sec. A is expected to be lower than the stimulus velocity of 20 deg/sec, as A and C (the y-intercept of the short and long time constant) when summed,

should equal to the stimulus velocity. This was found to be so. The variability in the cumulative displacement (47.8 ± 27.7 deg) and the number of slow phases in OKAN (48 ± 21) clearly indicate the wide spread of the data was due to individual variability. Contrasting that is the consistency of the slow phase duration (0.287 ± .059 sec). The consistency of the slow phase duration throughout each OKAN trial could indicate that the eyes are resetting themselves by a spatial reference (Jell et al., 1984).

The two component decay fit to the OKAN described it well. Our values closely resembled those reported by Jell et al., (1985). Habituation through repeated stimulation was not a factor in this analysis, since Jell et al., (1985) reported no significant decrease between trials in one session. Corroborating evidence of a two component decay fit in OKAN was given by Zasorin et al., (1983) and Segal and Liben, (1985), who described their OKAN as having "fast initial drops, followed by a slower decline". Values for their slow decline agree with our long time constant components.

G. COMPARISON OF THE PAE AND THE FIRST COMPONENT OF THE TWO COMPONENT OKAN DECAY

The values for the PAE were A = 17.77 ± 6.43 deg/sec; 1/B = 0.292 ± 0.120 seconds. The values for the first component of the two component OKAN decay were A = 15.54 ± 6.05 deg/sec; 1/B = 0.797 ± 0.127 seconds. "A"

values were not significantly different from each other, but 1/B values (the time constant), were significantly different at $p < .001$.

On the basis of the above information, if one assumes that one physiological integrator can be characterized by only one time constant, then one would have to conclude that the integrator stimulated with the pursuit stimulus is not the same as the one stimulated by the OKAN stimulus (ie first component), or that the charging or discharging are not facilitated in the same manner. Evidence for these assumptions have been demonstrated in postrotatory nystagmus by Schrader et al., (1985). The overall time constant for postrotatory nystagmus is the sum of the output of mainly two components, the peripheral vestibular nerve and the vestibular nucleii. Schrader et al., (1985) demonstrated a reduced time constant in postrotatory nystagmus approaching that of the peripheral nerve time constant by head tilting during the nystagmus, and postulated that head tilt suppressed the vestibular storage mechanism selectively, showing a separate facilitation of the discharge of the vestibular storage mechanism from the peripheral nerve. On the other hand, similar evidence of head tilt suppression on OKAN and post-rotatory nystagmus (decrement of the time constant) has been hypothesized to be due to the output of one integrator with a modifiable time constant (Waespe et al., 1985), the modification due to the conflicting visual and vestibular stimuli. The model in this instance would

therefore include one integrator with more than one time constant. This approach may be plausible, but little evidence is currently available to support it.

It has generally been accepted in the past that in man, pursuit and the immediate onset of OKN are mediated by the cortical pathways, and that they have the same mechanism (Cohen et al., 1977; 1981; Yee et al., 1979; Robinson, 1980; Collewijn, 1981). It has also been accepted that different mechanisms are responsible for cortical and subcortical pathways in OKN. This has been demonstrated by the differences in "look" (cortical; active) and "stare" (subcortical; passive) nystagmus (Hood and Leech, 1974) and by the selective occlusions of the optokinetic field (Brandt et al., 1973). The concept of cortical and subcortical pathways have also been supported by a slow build-up of OKN in humans with central scotomas (no foveal vision)(ter Braak et al., 1971; Yee et al., 1979), which is also seen in patients with hereditary cerebellar ataxia (Zee et al., 1976b). These findings also support a widely held view that the immediate onset of OKN is due to the central area of the eye (ie. area around the eye), but it does not necessarily mean it is due to the pursuit system. It has been demonstrated that pursuit can be dissociated from the fovea (Winterson and Steinman, 1978), and seems to depend more on an internal selection process and secondarily on the fovea, as smooth pursuit is only found in foveate species (Robinson, 1981). "Look" and "stare" nystagmus cannot either be easily classified on the basis of the "active" and

"passive", (cortical vs subcortical), retinal pathways, as there is conflicting evidence in that the stare OKN has been found to be stronger in the central 10 degrees (van Die and Collewijn, 1982), yet OKN has been generally believed to be mostly a subcortical process due to the periphery, (Brandt et al., 1973). This is supported by evidence from patients with central scotomas and slow build-ups of OKN (Zee et al., 1976b). Another viewpoint is that the foveal area acts mainly on the fixation and the foveal following while the peripheral area plays an important role in foveal fixation in OKN (Miyoshi, 1985), from evidence that the "foveal" mechanism in OKN decreases as the stimulating velocity is increased. Conscious and unconscious inputs may be another description of the inputs to OKN. This input has been demonstrated in voluntary and reflexive saccades. Reflexive saccades due to optokinetic and vestibular inputs were faster than voluntary saccades, and not necessarily additive (Henriksson et al., 1980). All the above evidence shows associations between pursuit and OKN, although there are no clear cut classifications of input in OKN.

There is some evidence for dissociation between the onset of OKN and pursuit. Yasui and Young, (1982) showed different levels of predictive capability between pursuit and OKN, and some patients with neurological diseases have been found to have pursuit intact but have poor passive OKN, and vice versa (Baratt et al., 1985).

Our evidence would lend some support for dissociation

of the pursuit and optokinetic systems, but we must address the question of what the inputs are to the optokinetic and pursuit integrators, and whether they necessarily have to be the same. In our pursuit stimuli, analysis of the decay after each smooth pursuit movement meant that the input to the pursuit integrator could have been due to the foveal image slip of tracking the dot, the efference copy signal of the single tracking movement, or both. In the optokinetic stimulus, the input to the integrators could have been due to the repeated retinal image slip of tracking the surround, the efference copy signal due to repeated tracking movements, or both. The difference in the foveal image slip versus full retinal slip, or single versus repeated efference copy signals could have charged a single integrator differently, and perhaps given a different time constant. Evidence for different stimulating techniques affecting pursuit discharge was given by Muratore and Zee, (1979), where stimulation by a foveal stimulus produced a decay much like OKAN (Jell et al., 1984). Though there were some confounding influences, this demonstrates an apparently different charging of possibly the same integrator. Lafortune et al., (1985) did attempt to investigate the charging of first component in the two component OKAN by changing the duration of stimulation, but even at their lowest stimulation duration of 5 sec, the first component was already fully charged. This is not surprising, as Mitrani and Dimitrov, (1978) showed the pursuit integrator was fully charged by 1 sec of stimulation. Pola and Wyatt,

(1985) on comparison of pursuit (active) and optokinetic (passive) following eye movements, concluded from their results that passive eye movements may be involved in normal pursuit movements, and whereas the optokinetic movements respond to a velocity input, pursuit movements involve a position channel in parallel with the optokinetic velocity channel, and perhaps add together prior to a common integrator.

Finally, one must ask why the decays in OKAN and pursuit should be modelled as the output of an integrator. Could the decays be modelled on a damped oscillator, or be described by polynomial equations? Due to the low r^2 values of the decay fits, the answer to the above question is probably yes, but what would the rationale be for the models? Most other models are the product of an active decaying process, while all passive decays found in nature have been adequately described by an exponential decay curve modelled on the output of an integrator. Since the decay described here is a passive process, the integrator model would best describe the decay.

Our conclusions therefore are that our results indicate that the pursuit decay and the first component of the OKAN decay are not the same, but this could be due to differences in the stimuli.

H. FUTURE CONSIDERATIONS

This thesis attempted to answer the question - is the first component of a two component decay model of OKAN due to pursuit? Comparison of the pursuit decay elicited and the first component of the OKAN decay indicated it was not, but there was some controversy on whether or not the two were similar, especially in light of the results of Muratore and Zee (1979) and Pola and Wyatt, (1985). It maynot even be possible to separate the first component from thee second component of OKAN, as they may be coupled in some way. In light of this fact, separation of the two components may only be possible in patients with central scotomas, as described by Yee et al., (1978); Schalen et al., (1982); and Barratt et al., (1985). Fitting of two component decays in these patients may identify the lack of pursuit input. Another technique may be to elicit OKAN with no OKN by a fixation point inhibiting OKN. This has been shown to elicit strong OKAN (Brandt et al., 1973), but has not been investigated with regard to possible diffeerences in pursuit and optokinetic inputs. A two component OKAN decay could be fit to a normal OKAN and compared to the fixated OKAN. Another technique may be to look at differences in two component OKAN between "stare" and "look" nystagmus. "Look" nystagmus may be more pursuit mediated than "stare" nystagmus, as the subject is actively following the stripes in "look" nystagmus. This may be seen as differences in the two component OKAN.

On the question of cell studies, it is unknown where

the hypothetical pursuit integrator is located, or whether or not it is related with the vestibular storage integrator. Finding a group of cells responding to the pursuit decay would be very useful. One area that has been hypothesized to contain the pursuit component is the cerebellar flocculi (Yasui and Young, 1984). It would also be informative to do single cell recordings in the vestibular nucleii during the pursuit decay, to determine whether or not this pursuit input has a pathway to the vestibular nucleii. This would suggest a coupling of the pursuit input in OKAN. Since pursuit and OKN have different predicitive components, it would also be useful to locate these different components in the brainstem. This information would show the possible dissociation between the pursuit system and the optokinetic system by their different locations.

It would also be useful to determine what effect an actual afterimage had on the pursuit decay. Our results indicated it was not a factor, but we did not elicit a afterimage. In light of the Bahill et al., (1983) data, we hypothesize that an after image would have no effect on the pursuit decay, but this remains to be seen.

An integrator model may best describe the pursuit decay, but an interesting question arises of whether it has to be a neural integrator. Robinson (1965) demonstrated that the mechanics of the eye give the eye a 500 msec latency movement after the pursuit command has stopped. This time

span could be due to an integration of the viscoelastic properties of the eye, and would probably hide any neural integration less than 500 msec. In our circumstances, isolation of a possible neural integrator would not be possible.

I. CONCLUSIONS

The conclusions from these experiments are:

1. The pursuit aftereffect is unaffected by changes in light intensity. The decay is not due to an afterimage.

2. The best decay fit to the pursuit decay started at 130 msec after lights out. This gives our best estimate of the signal processing time.

3. There are no significant effects on the pursuit decay due to repeated exposure to the stimulus. Habituation to the stimulus was not an overt factor in our experiments.

4. The pursuit decay and the first component of the two component OKAN decay did not have the same time constant. The time constant in the pursuit decay was 0.292 sec while the time constant in the first component of the OKAN decay was 0.797 sec.

5. Since the time constant of the pursuit decay was less than the 500 msec latency due to the viscoelastic properties of the eye, isolation of a neural integrator using this technique is not possible.

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