SCOTOPIC AND PHOTOPIC CONTRIBUTIONS TO THE HUMAN STEADY-STATE VISUAL EVOKED POTENTIAL

by

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Scotopic and Photopic Contributions to the Steady-State Visual Evoked Potential

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ABSTRACT

The possibility of scotopic activity above the photopic threshold was investigated through two indices of scotopic and photopic function, the steady-state visual evoked potential (VEP) and the psychophysical photopic threshold.

Latency and amplitude measures were obtained from the steady-state VEP over a 5.7 log unit range spanning scotopic and photopic levels in response to a 9.6° circular stimulus which excited both rods and cones. Steady-state VEPs were also recorded to foveal and parafoveal stimulation exciting predominantly cones and rods, respectively.

In all three observers tested VEP latency and amplitude functions obtained from the 9.6° field described two distinct systems. A long latency, single component VEP was observed at low illuminances, the amplitude of which described a growth-decay function. At intermediate to high illuminances the VEP shifted to a short latency response and a second component appeared whose amplitude was also described by a growth-decay function. The latency shift, which occurred between 1.0 and 1.4 log trolands, and the two distinct amplitude functions suggested a transition from scotopic to photopic activity at this point. In contrast, the psychophysical estimate of the least upper bound of the photopic threshold was between -.05 and .4 log trolands. That the photopic system was active at these levels was further supported by a foveally recorded VEP at .4 log trolands. In addition, a large amplitude VEP was recorded to

parafoveal stimulation at illuminances immediately above the photopic threshold, suggesting that rods were active in the photopic range.

Thus, the scotopic system continued to function for about 1.2 log trolands above the photopic threshold. Within this illuminance range when both receptors were activated simultaneously only rod signals were transmitted to the cortex. This result was attributed to an interaction between the different response latencies of the rods and cones and the stimulus repetition frequency. The probable site for this interaction was the internal plexiform layer.

DEDICATION

To my father
for his inspiration

To my mother
for her dedication

To our heavenly Father
from whom these gifts come

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A. Introduction

I. The Duplicity Theory of Vision

In 1866 Schultze first hypothesized the duplex nature of visual function after observing the predominance of one of two types of photoreceptors in nocturnal and diurnal animals. He proposed that rods were chiefly responsible for nocturnal vision, while cones mediated daylight vision. Later, this concept was further supported from evidence gathered by Parinaud (1898) and Von Kreis (1929). Today, the duplicity theory stands as one of the fundamental laws which guide our understanding of vision.

The duplicity theory, broadly stated, is based on differences between rods and cones in terms of their structure, photopigments, spectral sensitivity, retinal distribution, functional dynamics, neural connections, and many other factors. The cone system[1] is responsible for daylight vision, visual acuity, and colour perception, the rod system for the detection of objects and movement in very dim light.

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[1] the term system is used because the two types of vision proposed are not simply the results of a particular type of receptor, rod or cone, but are generated by a neural network, of which the receptor is just one part.

Although it is of great conceptual utility to dichotomize vision into separate photopic and scotopic categories, it is becoming clear that this simple black and white (or chromatic and achromatic, if you prefer) approach, will require some shades of grey if an understanding of all facets of vision is to be attained. For example, it is possible to extend the duplex theory of vision to postulate the mutually exclusive domains of rods and cones. When one system is operating the other is completely shut down. In fact, one of the cornerstones of the duplicity theory is that over most of the visible spectrum there is a level of luminance at which cones cease to function and the job of seeing is relegated exclusively to the rods. However, the converse of this phenomenon has yet to be demonstrated, the absence or the inhibition of rod function at photopic levels. Indeed, it appears that there is a range of illumination, the mesopic range, at which both rods and cones function. In addition, there is considerable evidence, to be discussed later, that under certain conditions rods and cones interact. Thus, the duplicity theory would have to be extended to include joint and interactive rod-cone function. The possibility of rod activity above the photopic threshold is the focus of this dissertation research.

Various methodologies have been employed to investigate rod and cone systems. The following review of the literature examines the contribution of rods to vision above the photopic threshold, which has drawn upon research in psychophysics and

the electrophysiology of the retina, cortex, and the single cell. This review will concentrate, in particular, on studies which bear on the following two questions. First, are the rods at all functional in the photopic range? Put another way, at what level of light adaptation results in a complete cessation of rod function? Second, if rod activity is present at photopic levels, does this activity contribute anything to the visual experience?

In studies of the electroretinogram it has been shown that the a-wave and the b-wave contain both a photopic and scotopic component, which can appear together (Armington, 1964; 1974; Gouras, 1966; Granit, 1963). This situation occurs in the dark-adapted eye at relatively high stimulus intensities. In fact, one of the major problems with using the ERG to investigate cone function is the difficulty in suppressing rod activity (Granit, 1963). Auerbach and Rowe (1966) have shown in the dark adapted eye a large scotopic ERG in rod monochromats under conditions that produced scotopic and photopic ERG components in normals. These studies supported the hypothesis that rods are active at photopic levels.

However, when light adaptation increases rod activity decreases, as indicated by the amplitude of the scotopic b-wave. Riggs and Johnson (1949) made a quantitative study of the effect of light adaptation on the ERG b-wave. They used white test flashes centered on a white adapting field that varied in log unit steps from dark to 1.6 mL. As adapting luminance increased

in the presence of relatively high intensity test flashes response amplitude and latency decreased. An adapting intensity of only 1.6 mL was sufficient to reduce the b-wave amplitude to a barely detectable level. Biersdorf et al. (1965), on the other hand, found that the b-wave amplitude could be maintained at a level of 40 uV over a much larger range of adapting luminances. A 50° test field was flashed on a equally large adapting field. The log stimulus intensity required to elicit a 40 uV criterion response was found to be proportional to the log adapting luminance that extended over 6 log units to a maximum of 55 fL.

Granit (1935, 1963) noted that the size of the scotopic b-wave decreased proportionately with increasing light adaptation, and with very thorough light adaptation the b-wave virtually disappeared. In these studies the luminance level at which rod activity ceased seemed to depend on factors such as stimulus intensity and retinal area. Unfortunately, they do not seem to present a clear picture of the luminance level at which rods cease to function.

Perhaps, an upper limit to rod function in the presence of light adaptation is imposed by the photopigment, rhodopsin. Upon the bleaching of rhodopsin by steady light less of the pigment is available for the intiation of a response. Thus, one might expect that the sensitivity of scotopic vision is directly related to the amount of available rhodopsin. Unfortunately, the experimental evidence has failed to confirm this hypothesis.

It is well known that when rods are exposed to light their thresholds rise. Aguilar and Stiles (1954) have shown that over the middle range of scotopic visicn, the threshold for observing the test light rose in proportion to the luminance of the adapting field. At higher levels of adaptation rod vision saturated so that any further increase in stimulation produced no concomitant increase in rod response. However, it has been demonstrated that, by calculating the amount of light absorbed by the rods, the increased rod threshold was not due to a proportional drop in available rhodopsin, even at saturation levels (Aguilar and Stiles, 1954; Campbell and Rushton, 1955). Rushton (1969) stated that when one per cent of the rods caught an average of only a single quantum of light, the resulting decrease in rod sensitivity was three-fold. In the rat Dowling (1960) found a linear relationship between the amount of rhodopsin bleached and the logarithm of the threshold required to produce a constant amplitude ERG. In other words, unitary changes in bleached rhodopsin produced tenfold changes in rod sensitivity. Granit and co-workers (1938) found that the bleaching of the frog eye by various monochromatic lights was followed by a considerable reduction in the size of the ERG b-wave elicited by a wavelength of 500 nm, but that this reduction was not accompanied by any reduction in the amount of rhodpsin that could be extracted. Thus, the 30-70% reduction in the b-wave amplitude could not be explained by supposing that the quantity of rhodopsin present was the sole determinant of

the size of the b-wave during dark adaptation. Further investigation revealed that during the course of dark adaptation after a significant bleach, there was a considerable delay (10 min. approx.) before an increase in ERG amplitude was noted, which always began when rhodopsin concentration had reached about 50% of its maximum. Granit (1963) also noted a complete absence of a measurable b-wave immediately after a bleach in spite of the presence of 40% of maximum concentration of rhodopsin. Given this evidence Granit (1963) concluded,

> The size of the b-wave is a good expression of the average excitability of a large number of retinal elements and we must therefore conclude that average excitability and average visual purple content can, under certain conditions be largely independent of one another and that the increase of rod excitability lags behind the increase in visual purple concentration during dark adaptation. In the mammalian eye the b-wave can be depressed to the extent of not appearing on the record in spite of the presence of considerable quantities of visual purple (p. 249-50).

Similarly, Sakitt (1976) measured dark adaptation thresholds after an estimated (albeit crudely) 100% bleach. A test flash of 10.5 log units above absolute threshold, shown on a 20° area centered 13° from central fixation could not be detected until after 9 min of dark adaptation. She estimated this time period corresponded to less than 25-30% of pigment left bleached. Barlow (1964) found that the saturation level of rods at approximately 1000 td corresponded to a bleach of no more than 28% of rhodopsin.

It may be concluded, therefore, that the limiting mechanism for rod function was not the amount of bleached rhodopsin, but

rather some other, perhaps physiological, aspect of the rod system.

Gouras (1972) cited research which suggested that this mechanism was completely limited to the rod system, so that it must take place at a point where rods are independent of cones. Since Gouras and Link (1966) have shown that rod and cone signals converge on the same ganglion cell, the mechanism by which rod thresholds are elevated must reside between the inner plexiform layer (the synapse between bipolar and ganglion cells) and the receptor layer.

The exact mechanism that prevents rods from transmitting signals in the light adapted state remains unknown. However, the extent to which the rod system functions in the photopic range has been investigated in the case of the normal retina and in the cone-free retina of the rod monochromat.

Klingaman (1977, 1979) compared VEPs and psychophysical increment thresholds on a normal and a rod monochromat observer. He presented a 7° circular test field on a 24° white background. The background luminance increased over a total of 6.5 log units to a maximum of 5.34 log scotopic trolands. The test flash was 520 nm in wavelength. Klingaman found that the rod monochromat gave both psychophysical and VEP responses at least up to a level of 1000 scotopic trolands. Above this level Klingaman reported that the monochromat became functionally blind, a condition he attributed to the saturation point of the rods, which was about 3-3.5 log units above the photopic threshold of

a normal observer determined under the same conditions.

Other studies have found a similar saturation value of 1000 scotopic trolands for rod monochromats using different parameters for the test and background fields (Blakemore and Rushton, 1965; Sakitt, 1976).

Of considerable significance is the study by Aguilar and Stiles (1954) which measured the saturation point of the rods in four normal observers. These researchers estimated the level of rod saturation to be between 2000-5000 scotopic trolands. This range was extrapolated from the increment threshold function in the intermediate range of backrcund intensities. This was done because the cones began to intrude at higher backround luminances. In this range the Weber/Fechner ratio applied, where the logΔI/log I = K (I = backround intensity; K = constant). They defined the rod saturation level as 100K. Using this criterion the average upper limit of the saturation level of the four observers was 3.7 log scotopic trolands. Thus, the normal eye appears to have a similar saturation level as the pure rod eye. This level is in the range of 1000-5000 scotopic trolands.

Now, I will consider the fate of the rod signal as it passes through the neural network of the retina. Of particular interest is the possibility of the rod signal interacting with the cone signal. Considerable research has been conducted on this question and the concensus appears to be that there is an interaction between rod and cone signals, the final output of which is decided at the retinal ganglion cell.

Several psychophysical studies have demonstrated changes in the sensitivity of rods due to cone activity (Buck, Peeples, & Makous, 1979; Ingling, Lewis, Loose, & Myers, 1977; Makous and Booth, 1974; Makous & Peeples, 1979; van den Berg & Spekreijse, 1977) and changes in cone sensitivity due to rods (Drum, 1981; MacLeod, 1972; McCann, 1972). These studies as well as others (Frumkes et al. 1973; Trezona, 1970; Von Grunau, 1976) have shown that rods and cones may inhibit or facilitate one anothers' activity in either the spatial or temporal domain.

The possible sites of interaction have also been investigated. The first opportunity for interaction, anatomically, is in the outer plexiform layer where horizontal cells receive input from cones and send output to rods (Kolb, 1970). This interconnection by horizontal cells may account for a cone influence on rods but not the converse. However, this seems unlikely because Gouras (1966) has shown in the monkey retina independence of the rod and cone systems at this stage of retinal processing. He found additivity of responses in the intraretinal ERG when rods and cones were stimulated together. Since the part of the ERG examined (the b-wave) represented bipolar cell activity (Armington, 1974; Granit, 1963) it is difficult to argue rod-cone interaction at the horizontal cell layer.

Interactions between rods and cones seem more likely to

take place at the next confirmed[2] site of interaction, the inner plexiform layer. At this level it appears that bipolar cells receiving input from exclusively either rods or cones converge on the same ganglion cells (Boycott and Dowling, 1969; Polyak, 1957) In addition, amacrine cells appear to interconnect rod and cone bipolar cells at this layer (Dowling and Boycott, 1965). Thus, the interaction may be mediated either presynaptically on the bipolars or postsynaptically on the ganglion cells.

Although no evidence has yet become available to distinguish between these two possibilities, evidence has surfaced in support of antagonistic rod-cone interaction at the level of the ganglion cell (Gouras, 1965; Gouras and Link, 1966.) Gouras and Link recorded spike activity from a perifoveal ganglion cell in the dark adapted monkey to monochromatic light which stimulated either only rods (violet) or only cones (red). When the two colored lights were flashed far enough apart temporally, the ganglion cell responded to both flashes, showing independence. However, when the two flashes were brought temporally close to one another the signal that arrived first excited the ganglion cell while the following signal had no effect. This was the case whether a rod or cone signal arrived first. It appeared that the earliest signal left a transitory refractoriness in its wake which made it less likely that the

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[2] Polyak (1957) has suggested that individual bipolar cells receive input from both rods and cones but more recent evidence suggests a private coupling between bipolars and the two receptors (Boycott and Dowling, 1969; Dowling and Boycott, 1966).

ganglion cell would be excited by the second signal. Gouras and Link also noted that cone signals arriving at the ganglion cell always had latencies shorter than rod signals, even at the cone threshold. Gouras (1967) reported that the latency of activation of a ganglion cell by rods was never less than 50 ms and was always more than the response latency to cones. As a result, a ganglion cell receiving input from simultaneously activated rods and cones only responded to the faster cone input. Thus, the resulting output to the brain consisted of just the cone signal.

This finding may help to reconcile the disparate results obtained when the ERG and VEP were simultaneously recorded. Several studies found that the VEP had a predominantly photopic basis while the ERG was relatively scotopic in nature. (Armington, 1966; Nagata & Jacobson, 1966; Perry & Copenhaver, 1966; Ripps & Vaughan, 1969). Apparently rod signals, even when present in the ERG, do not reach the brain when they are in competition with cone signals. It must be noted that other factors, reviewed later, are also involved in the discrepancy between the luminosity characteristics of the ERG and the VEP.

The prefered transmission of cone signals through the retina may also acount for the assertion by Wooten (1972) of an entirely photopic VEP once the cone threshold was exceeded. He found this to be the case when determining the photopic spectral sensitivity of the VEP under conditions which were capable of stimulating both rods and cones. Since a rod contribution was not found in the photopic spectral sensitivity function Wooten

proposed that rod signals were being pre-empted.

It could be argued that Wooten's observation did not negate the possibility of rod activity in the photopic range, but only that rods did not contribute to the measurement of photopic spectral sensitivity. Wooten used a constant criterion implicit time (latency) measure to define the VEP spectral sensitivity curve. The implicit times for the photopic experiment were between 150-250 ms and 225-235 ms for the scotopic experiment with very little overlap between the two. Since Gouras (1967) demonstrated that the response latencies of rods were always longer than cones for stimuli of equal energy, the rods may have made a later contribution to Wooten's VEPs which would not have affected the threshold measurements. His statement, "These data seem to indicate that the VECP is entirely photopic once the cone threshold is exceeded." (p. 1658) does not seem to be justified.

To summarize the research conducted at the retinal level, it appeared that the scotopic system was functional at photopic luminances, up to about 3-3.5 log units above the photopic threshold. Rod signals appeared to remain largely independent of cone sigals at least up to the level of the ganglion cell. At this level interactions between rod and cone signals were possible, with either one capable of being selectively transmitted by the ganglion cells. However, under most circumstances cone signals appeared to be transmitted once the photopic threshold was reached because of their faster response

latencies.

It is clear now, that in order to answer the second question about rod function, i.e. do rods contribute anything to visual perception above the photopic threshold, we must move outside the domain of the retina, to the brain itself, where the visual percept is generated.

Many psychophysical studies have reported changes in visual perception due to interactions between rods and cones. These studies have made use of conditions which tend to elicit both scotopic and photopic activity. Such conditions have included illumination at mesopic levels, stimulation of the parafovea, special phase relations between the onset of rod and cone signals, and the selection of wavelengths to preferentially stimulate rods and cones. Given such conditions studies have reported changes in cone thresholds due to rods (Drum, 1981; Frumkes et al. 1973) and changes in color perception due to the influence of rods on cones (Ball, 1964; MacLeod, 1972; McCann, 1972; McCann & Benton, 1969; Trezona, 1970; Von Grunau, 1976, Walters, 1971).

The utility of visual evoked potentials in the investigation of rod function has been questioned on the basis of the predominantly "photopic" nature of the VEP (see e.g. Wooten, 1972). Thus, the VEP has been relatively neglected as a tool for examining the effects of rod-cone interactions at the cortical level. The research findings that have led to this situation are discussed in the next section.

II. Scotopic and photopic contributions to the transient and steady-state VEPs

Although there is a sizeable body of literature on the photopic and scotopic contributions to the transient VEP, very little work of this type has been done on the steady-state VEP. Most of the work on transient VEPs has centered around the question of whether or not the scotopic system contributes anything to the VEP.

The contention of a predominantly photopic or foveal origin of the VEP rests upon several factors. Foveal cone receptors enjoy a one-to-one correspondence with optic nerve fibers, whereas there is a considerable amount of convergence of the extra-foveal receptors on the ganglion cells. Thus, the representation of the foveal cones on the visual cortex is magnified greatly compared to the ratio of the number of foveal to extrafoveal receptors in the retina (Witteridge, 1973). Moreover, the fovea projects to the convexity of the occipital pole, whereas the peripheral retina projects to the medial walls of the calcarine fissure (Barr, 1974). It is assumed that the closer proximity of the foveal cortical projections to the recording electrode results in a greater contribution of foveal receptors to the VEP compared to the extra-foveal receptors. Thus, it was concluded from initial studies that the VEP was largely foveal in origin.

While these factors explain why such a relatively tiny fraction of the total number of retinal receptors accounts for such a large proportion of the VEP, they do not completely rule out the possibility that the VEP is also sensitive to the peripheral or scotopic portions of the retina. The following review indicates that both the photopic and scotopic systems contribute to the VEP with the major portion coming from the photopic system.

There have been several demonstrations documenting the photopic nature of the VEP. Using either a constant criterion amplitude measure (Armington, 1966; Cavonius, 1965) a constant criterion implicit time measure (DeVoe, Ripps, & Vaughan, 1968; Wooten, 1972), or heterochromatic flicker photometry (Sigfried et al. 1965), investigators have found that the spectral sensitivity of the VEP matched the psychophysicaly determined CIE photopic luminosity function rather well.

With respect to rods or scotopic vision, Wooten (1972) found that the spectral sensitivity of the VEP measured by means of the constant-criterion implicit time method matched the CIE scotopic luminosity function except at wavelengths longer than 600 nm. Since under threshold conditions the photopic system is slightly more sensitive than the scotopic system in this region of the spectrum, Wooten concluded that the photopic system was contributing to the VEP. Taking advantage of the directional sensitivity of the cones (Stiles and Crawford, 1933), Wooten stimulated the retina eccentrically with a Maxwellian beam

thereby stimulating only rods. He found that the VEP matched the CIE scotopic luminosity function at the long wavelength end of the spectrum.

Vaughan and Hull (1965) also found evidence of a scotopic contribution to the VEP. They used a 4° field, centrally viewed, which stimulated both rods and cones over an energy range of 8 log units. They found that the latency of the P1 component increased steadily with decreasing intensity up to 6 log units of attenuation where a distinct break in the slope of the latency function occurred. This break amounted to a change of about 30 ms in each of 3 subjects examined. Also, at this level of attenuation a 1.5° foveally projected stimulus failed to generate an EP nor did the subjects report seeing the stimulus. For these reasons the authors concluded that the VEPs to luminances below 6 log units of attenuation were generated by "a distinct long-latency scotopic mechanism" (p. 721).

Another way to assess scotopic and photopic contributions to the VEP is by examining the behavior of the VEP during dark adaptation. Initial studies reported some success in describing both rod and cone contributions to the VEP during dark adaptation (Huber and Adachi-Usami, 1972; Perry, Childers, Dawson, and Stewart, 1968). Both Fujimura et al. (1975) and Klingaman (1976) reported comparisons of VEP changes and psychophysical measures collected from the same individuals during dark adaptation. Both found a direct comparison of the VEP dark adaptation function with the psychophysical data.

One major problem with collecting VEP data during dark adaptation is that the number of flash repetitions needed to produce an average VEP may significantly influence the level of adaptation in the eye. To control for this problem Klingaman (1976) obtained a large N VEP by pooling a number of small N VEPs collected over numerous replications of the experiment. With this improvement Klingaman (1976) found that if the stimulus conditions during dark adaptation favored the photopic system, changes in VEP amplitude reflected cone activity, but if stimulus conditions favored the scotopic system, then the VEP reflected rod activity.

Photopic versus scotopic contributions to the VEP have also been examined using increment thresholds in normals and rod monochromats (Adachi-Usami, 1974; Huber & Adachi-Usami, 1972; Klingaman, 1977, 1979). Klingaman used a constant criterion latency measure to plot the VEP increment threshclds and compared them to the psychophysical thresholds. In the normal and the rod monochromat Klingaman found that the increment threshold curves derived from psychophysical and VEP data very closely paralleled one another. However, when comparing the two subjects Klingaman found a clear break in both the psychophysical and VEP increment threshold functions for the normal but not for the rod monochromat. The break in the VEP curve was an abrupt change in latency of 40-45 ms within a half log unit. This break was attributed to a changeover from the scotopic to the photopic systems as a result of increasing

backround illumination. The rod monochromat exhibited a smooth scotopic increment threshold function.

Thus, a number of studies have shown that the VEP is sensitive to both photopic and scotopic mechanisms when the eye is stimulated under the proper conditions. Previously, many investigators have cited a predominant photopic contribution to the VEP, the bulk of which stimulated the fovea where the concentration of cones was greatest (Armington, 1966; Cavonius, 1965; DeVoe et al. 1968; Potts & Nagaya, 1965; Ohba, 1967; Rietveld, Tordoir, & Duff, 1965). Although the fovea appeared to make the greatest contribution, the parafovea (which contains both rods and cones) has also been shown to make a sizeable contribution to the VEP (Copenhaver & Perry, 1964; Rietveld et al. 1965). Finally, a purely scotopic VEP has been reliably recorded and the spectral sensitivity curves obtained from the VEP closely matched the psychophysical scotopic luminosity function (Huber & Adachi-Usami, 1972; Klingaman, 1976, 1977, 1979; Wooten, 1972).

Regan (1970) has used heterochromatic flicker photometry as a means of correlating psychophysical and steady-state EP measures of the the spectral sensitivity of the retina. This procedure, as described by Regan (1972) is as follows:

> The subject views a stimulus field which consists of two superimposed patches of light. One light is coloured and the other is a standard white which is left unaltered throughout the procedure. The white and coloured beams are alternated at a sufficiently high frequency that the colours fuse subjectively but the alternation frequency is not so high that brightness flicker disappears. The luminance of the coloured beam is then altered until the

> subject reports that flicker is a minimum. At this point perceived flicker will increase if the coloured beam is made brighter or dimmer; and at the point of minimum flicker the luminances of the white and coloured stimuli are equal (by definition). (p. 97)

The steady-state VEP measure of spectral sensitivity consisted of the point at which the luminance of the flickering coloured light produced a minimum VEP amplitude. Siegfried, Tepas, Sperling, and Hiss (1965) found that EP amplitude fell to a minimum at the same luminance where the point of minimum subjective flicker was reported. However, Regan (1970) who repeated the experiment found this relationship in only one of eight subjects tested.

In a rather detailed analysis of the two conflicting studies (which shall not be dealt with here, but see Regan, 1972, p. 97-104) Regan concluded that for EP components in the 10-30 Hz range the point of the perception of minimum flicker and the minimum EP amplitude could be dissociated by appropriate choices of stimulus intensities and modulation depths. However, Regan pointed out that the situation was quite different for frequency components of the EP in the high frequency range of 40-60 Hz. In the heterochromatic photometry paradigm the amplitude of the high frequency EP components fell to a sharp minimum at the point of minimum subjective flicker. By using wavelengths across the spectrum Regan (1970) found that the EP amplitudes of the second harmonic (48 Hz component) agreed with the relative spectral sensitivity curves of the psychophysically obtained heterochromatic photometry data. In contrast, the fundamental component (24 Hz) of the EP had no corresponding

minimum amplitude at the point of minimum flicker.

Although not explicitly stated, this spectral sensitivity function was determined for the photopic range of luminances as the average luminance of the white and coloured lights varied around 100 td. To date, there have been no data relating the steady-state VEP to a scotopic spectral sensitivity function.

Diamond et al. (1981) have investigated the possibility of rod and cone contributions to the steady-state VEP. Flickering stimulation was presented which varied on several dimensions: frequency, retinal locus and area, luminance, and wavelength. The manipulation of these dimensions was intended to differentially stimulate the rod and the cone systems. It was found that stimulus conditions of low frequency (9 flashes/sec), short wavelength (501 nm), extrafoveal stimulation, and low luminance all differentially increased the amplitude of one component of a double-component VEP. In contrast, a long wavelength stimulus presented at high luminance and repetition rates (25 flashes/sec) centered on the fovea differentially increased the other component. On the basis of these results Diamond proposed that the first component was derived from rods and the second from cones.

One puzzling result of this work was the presence of a large amplitude "rod" component under photopic conditions. This would suggest that both rod and cone signals were reaching the brain at photopic levels. Such an explanation has been proposed to account for the cancelation of flicker at mesopic levels

(MacLeod, 1972) and the desaturation of color during flicker (Ball, 1964; Walters, 1971). For example, MacLeod (1972) found that a 7.5 Hz flickering light which stimulated both rods and cones appeared as a steady field at mesopic levels of illumination. At higher and lower levels it appeared as a flickering light. Ball (1964) and Walters (1971) found that a blue-green light (510 nm) presented at photopic levels (475 trolands) desaturated when flickered at a rate of 10 Hz. Both these phenomena were explained by the difference in response times between rods and cones. MacLeod proposed that the cancelation of flicker was due to equal amplitude rod and cone signals ariving at a common analyzer 180 ° out of phase, producing the illusion of a steady light. Walters, on the other hand, thought that the 10Hz flicker resulted in a disinhibition of the rod activity at the ganglion cell level, producing the desaturation phenonmenon. Furthermore, Walters found that desaturation of the flickering blue-green light was accompanied by a marked increase in the scotopic b-wave.

Taken together, these experiments suggest that under conditions of flicker around 7-10 Hz and appropriate luminance levels photopic and scotopic activity can either interact or summate to produce changes in perception.

One method of distinguishing between scotopic and photopic activity at the cortical level is to measure the difference in arrival time of these signals at the brain. Both psychophysical and electrophysiological studies have shown that cone signals

arrive at the brain between 30-100 ms earlier than rod signals (Frumkes et al. 1973; Vaughan, 1966). In the present study the arrival of rod and cone signals at the brain was assessed by measuring the latency of the steady-state VEP. However, the determination of latency in the steady-state VEP has not been an easy task and has been approached differently by different investigators. A review of the nature of the steady-state VEP and methods devised to determine latency follows.

III. Frequency vs. time domain analysis of visual evoked potentials

Where evoked potential measures have been applied to the study of human vision, major emphasis has been placed on the use of transient VEPs and much less on steady-state VEPs. Perhaps one reason for this is that the transient VEP provides a simpler model for how the brain responds to stimulation. The model may be presented in the following way. If we are interested in examining the brain's response to a particular stimulus, then it makes sense to record brain activity just prior to, during, and for some time after stimuluation until the brain's response has subsided. Thus, one has a well defined time course of brain activity in response to a single stimulus which presumably encodes information about the stimulus. The technique of averaging improves the signal to noise ratio so that the response to the stimulus can be extracted from the backround EEG activity.

This simple case can be taken one step further by repeating the stimulus before the response to the first stimulus is completed. This obviously results in a more complex situation because responses to stimuli begin to overlap one another. When stimuli are regularly repeated, the brain reacts by producing a response which, over a fairly large range of stimulating frequencies, exactly follows the frequency. Thus, the

steady-state VEP provides information not only about the stimulus itself, but also on how the brain responds to the time relations among the stimuli. It can be concluded that the transient VEP and the steady-state VEP can be used to tap different aspects of human brain function. The most common method of analysis of the transient VEP has been in the time domain, that is, identifying different characteristics of the VEP (amplitude, slope, turning points) by referencing them to the onset of the stimulus. This method assumes that the stimulus activates different brain structures in a sequence and that the time-locked activity of the VEP components represent the sequential processing of the stimulus. Analysis of the transient VEP has largely become a matter of measuring the latencies and amplitudes of the components.

Regan (1977) has argued that this method is not appropriate for steady-state VEPs because of the difficulty of associating any particular cycle of the response with any particular stimulus. This problem will be taken up again when the latency of the steady-state VEP is discussed. Regan (1972, 1977) has favored the use of frequency domain analysis, in particular Fourier analysis, for steady-state VEPs. Fourier analysis analyzes the EEG signal into different frequency bands, taking advantage of the frequency following response of the brain to repetitive stimulation. It is possible to construct any complex waveform from a series of N harmonic sine and cosine waves using Fourier analysis. Each harmonic makes a certain weighted

contribution to the waveform, depending on how strongly a particular harmonic is present in the EP. Thus, the VEP may be adequately described by measuring the phase and amplitude characteristics of the harmonic components of the response.

The signal to noise ratio is enhanced by "filtering out" EEG frequencies not related to the stimulus frequency. This approach may result in a considerable loss of information about stimulus-response relationships because pure sinewave stimulation produces a brain response much more complex than a simple sinewave (Regan, 1968). In this case the stimulus-response relationship is said to be non-linear. If, however, the complex response is periodic, Regan (1966, 1968) claims that the response can be reduced into a number of different sinewaves, each harmonically related to the stimulus frequency.

One disadvantage of Fourier analysis is that it requires restrictive assumptions about the nature of the VEP generating mechanisms. Although Fourier analysis is a powerful analytic tool, there is no reason to believe that the brain acts as a "harmonic frequency generator". The following example illustrates this point. Suppose a brain response is produced such as that shown in Figure 1A. Fourier analysis of this response would yield two harmonics, a fundamental (1B) and a 2nd harmonic (1C). It is assumed that the fundamental and the second harmonic combine to form the original response. It also assumes the existence of two generators, one producing responses at the

fundamental frequency and the other at the second harmonic that are continually active over the entire stimulus cycle. However, it is equally possible that the response is produced by two independent generating mechanisms that produce waveforms shown in Figure 1D and 1E, which when recorded at the scalp by an "ignorant" electrode, summate to produce the recorded response. In fact, there may be an infinite number of ways in which the brain produced the response. However, the point is that there is no reason to accept the "harmonic frequency generator" hypothesis over any other. Frequency analysis requires such an assumption of frequency generators behaving in a certain way whereas time domain anaylsis posits no such assumption.

A second disadvantage of this method is a practical one. Very few investigators ever analyse the steady-state VEP beyond the second harmonic. This practice may result in a considerable loss of information if one considers that the steady-state VEP (as pointed out earlier) contains information about the stimulus (within a response cycle) which may be contained in higher harmonics. It is possible that the higher harmonics contained in the steady-state VEP are derived from primary cortex and the lower harmonics from non-specific cortex. Although this has been proposed for responses resulting from high and low frequency stimulation it may also be the case for the higher harmonics of low frequency stimulation.

Diamond (1977a, 1977b) has proposed using time domain analysis for the steady-state VEP as an alternative to frequency

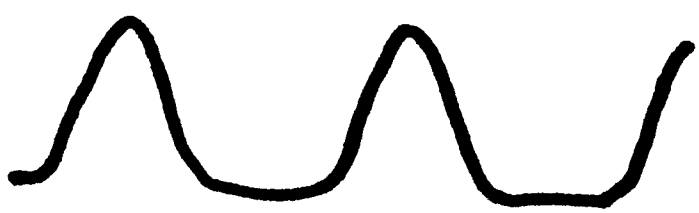
Figure 1. Two ways in which a sine wave can be decomposed. The original waveform is shown in A. B and C illustrate the fundamental and first harmonic of A, respectively. D and E show two arbitrary waveforms, which when added together equal the waveform in A.

A

B

C

D



E

analysis. This method does not require the assumptions of frequency analysis about the EEG response, and so may be applied to many situations in which frequency analysis is inappropriate. Some examples include when non-sinewave stimulation is used or when the stimulating frequency is not symmetric. In order to show that time domain analysis can yield meaningful information about the steady-state VEP Diamond (1977a, 1977b) devised a method of determining the latency of the steady-state VEP which is contrasted with the phase shift method of Regan in the next section.

Latency of the steady-state VEP

Regan (1972) has asserted that a measure of the latency of the steady-state VEP can only be considered a "true" measure of the response time of the visual cortex if it can be assumed that the nervous system responds to stimulation linearly and with minimum phase shift. This requirement results from the nature of the brain's response to repetitive stimulation. Regan (1972) maintains that when stimulus repetition frequencies are high enough, no individual brain response cycle can be associated with a particular stimulus cycle. Thus, unlike transient VEPs there is no way to determine the latency or response time delay of the brain to a particular stimulus, because the responses of the brain overlap in time.

Instead, Regan has proposed a measure of "apparent latency", T', which equals the true latency, T, if the

assumption of a linear, minimum phase-shift system holds and if EP amplitude changes little with stimulus frequency. In frequency regions where EP amplitude changes markedly with frequency a further transformation must be made in order to estimate the true latency. From investigations of amplitude/frequency relationships it is clear that amplitude does vary markedly over some parts of the frequency range (Regan, 1972; Spekreijse, 1966). These marked changes in amplitude also affect phase. If a correction is not made for variations in amplitude, then the apparent latency overestimates the true latency (Regan, 1972; Spekreijse et al. 1977).

However, the critical factor in estimating true latency from apparent latency is the assumption of a linear minimum phase-shift system. Unfortunately, it is clear that in many situations the stimulus-EP system is not linear (Regan, 1972). In fact, a considerable effort has been made by those researchers who use frequency analysis to either create stimulus conditions that linearize the system (Spekreijse, 1966) or to apply linear systems analysis to non-linear elements by using small stimulus signals where the distortion is thought to be minimal (Regan, 1972).

In spite of the uncertain nature of the stimulus-EP system latency measurements have been made for 3 different portions of the phase-frequency curve, the low frequency region (8-12 Hz), the medium frequency region (13-25 Hz), and the high frequency region (35-60 Hz) (Regan, 1972). The "apparent latency" is

calculated from the slope of the phase-frequency plot by the formula $T' = 1/360\ d\phi/df$ (where T' is in seconds, $d\phi$ is the change in phase in degrees, and df is the change in frequency in hertz; Regan, 1972). The apparent latency for the low frequency region has been calculated to be within 120-200 ms, between 110-140 ms for the medium region, and 48-62 ms for the high frequency region (Regan, 1966; Van der Tweel and Lunel, 1965; Spekreijse, 1966).

Spekreijse et al. (1977) have interpreted the shorter delay for the high frequency VEP as a reflection of different transfer processes along the visual pathway. They have further proposed on the basis of topological studies that these processes project to different cortical areas. The high frequency, short latency response appears to originate from the striate cortex, the medium frequency, medium latency response from areas 18 and 19, and the low frequency, long latency response from unspecified cortical areas.

The difficulty in meeting the assumptions of a linear minimum-phase shift VEP system requires validation of the above latency estimates. Spekreijse et al. (1977) provided validation for these estimates by comparing these latency values with those from the transient flash VEP and VEPs to Gaussian noise-modulated light. In noise-modulated light stimulation all frequencies are continuously present while the state of adaptation remains constant during the entire recording period. In this way problems of non-stationarity of the VEP system are

obviated. These researchers presented data from these two types of VEPs whose latencies corresponded with latencies determined from the phase-frequency plots from sinusoidal stimulation. VEPs generated from Gaussian noise modulation were found to have delays of 40-60 ms in the high frequency part of the spectrum. When corrected for the phase shift characteristics of the VEP system a "pure" latency estimate of 30 ms was found. This value corresponded to the latency of the first response of the transient VEP (Ciganek, 1961). It should be noted that the phase-frequency estimate of 40-68 ms overestimated the Gausian noise-modulation and the transient VEP estimates. A second "pure" latency estimate of 90-110 ms was found for the medium frequency range of the Gaussian noise-modulated VEP. This estimate corresponded to the latency of the second component of the transient VEP (Ciganek, 1961). Here again the phase-frequency estimate of 100-140 ms overestimated the time domain and the Gaussian noise-modulated estimates.

Diamond (1977a, 1977b) described an alternate method of determining the latency of the steady-state VEP which did not require the assumptions outlined by Regan. Diamond's method was based on a time domain analysis of the steady-state VEP which determined the difference in time between stimulus onset and some reference point on the VEP waveform, usually a peak or trough. In order to determine latency it was necessary to collect a set a VEPs over several different interstimulus intervals (ISIs). Latency values calculated in this way have

yielded latency estimates of 61 ms for short ISIs and 127 ms for long ISIs (Diamond, 1977b). These values were consistent with latencies estimated by frequency domain analysis for the high and medium frequency subsystems, respectively (Regan, 1966, 1972; Spekreijse et al. 1977).

This method assumed that the VEP latency was constant over the range of ISIs used and that the reference points chosen in the VEP cycles were consistently identifiable at the same time points within each cycle. However, since it has been shown tha latency does change as a function of ISI (Diamond, 1977b; Regan, 1972; Spekreijse, 1966), this assumption may not hold for the range of ISIs required to collect the information necessary to determine latency.

In the present experiment, asynchronous stimulus trains were used which employed alternating ISIs of two different durations. If a set of three different asynchronous stimulus trains were required to determine latency, then one of the intervals would be varied and the other kept constant. For example, three asynchronous trains could be produced in which the intervals (in ms) would alternate in an AB/BA/AB... etc., fashion. The first asynchronous train might be made up of intervals 100/110/100..., the second train of intervals of 117/110/117..., and the third of intervals, 128/110/128.... Thus, every train of stimuli would contain a constant interval, in this case 110 ms. If the latency was measured from a stimulus pulse which was always preceded by the constant interval, then

it would not be affected by the variable interval. This method, which avoids the possible confounding effects of different ISIs on latency, was originated by Van Dyke and Diamond (1982) and was found to measure steady-state VEP latencies over a wide range of ISIs (30-127 ms).

Luminance/latency relations of the steady-state VEP

Spekreijse (1966) examined the relationship between retinal illumination and latency by using a sinusoidal stimulus with a frequency of 30 Hz and a modulation depth of 20%. He found that latency decreased linearly with increasing retinal illumination, although slowly. Intensity, which was varied between 120-150,000 trolands, produced a change in latency from 62-48 ms. These latency changes reflected only photopic functions since rods do not follow frequencies above 20 Hz (Hecht et al. 1936).

For low frequency responses Spekreijse found that the latency of the response decreased roughly linearly with the logarithm of retinal illumination. This latency decrease was on the order of 100 ms between 3 and 9000 td. However, even the shortest latency of the low frequency response was still longer than the latency of responses to the high frequency stimulus. It should also be noted that the low frequency response had a far greater latency range for the range of retinal illumination used compared to the high frequency response.

Coupland (1978) also reported changes in VEP latency with different stimulus intensities and light adaptation levels.

Although no quantitative analysis was performed the author concluded that a 150 ms negative component decreased in latency over a range of 0.6 log units. A similar relationship was described when adapting luminance was varied over a range of 0.6 log units.

The above studies have described a relationship between intensity and a single response latency measure. However, since the steady-state VEP may be composed of more than one component per flash (Diamond et al. 1981; Kinney et al. 1973), the latency/intensity functions of multiple components may follow different courses. Some evidence has been collected to support the possibility of several different latency components of the steady-state VEP, however not as a function of intensity but as a function of ISI. Coupland (1978), using time domain analysis, found different latencies for positive and negative peaks. For slow frequency flicker in the range of 4.8-12.5 Hz the latency of response for negative peaks was 122.9 ms and for positive peaks 108.5 ms. For flicker in the range of 13.3-25 Hz the negative peak latency was 85.5 ms and the positive peak was 97.5 ms. The amount of latency change between low and high frequencies was also different for positive and negative components. For negative peaks the change was 38 ms, but only 11 ms for positive peaks. These data suggested that individual peaks of the steady-state VEP have different latency functions.

The present experiment investigated the relative contributions of the scotopic and photopic systems to the

steady-state VEP over a 5.5 log unit illuminance range spanning scotopic and photopic levels. It was expected that scotopic activity would be indicated by long latency VEP components and photopic activity by short latency components. Of particular interest was the possibility of scotopic activity in the photopic luminance range. If the double component VEP, such as that described by Diamond et al. (1981), was due to separate rod and cone components, then the long and short latency functions would overlap over some portion of the luminance range, probably in the mesopic range.

In addition, other tests of photopic and scotopic activity were carried out to compare with the luminance/latency functions. The photopic threshold was measured psychophysically under conditions similar to those in which the steady-state VEPs were collected. Secondly, VEPs were recorded to stimulation impinging on either the foveal or parafoveal areas. Foveal stimulation would result in a predominantly photopic VEP, while a predominantly scotopic VEP would result from parafoveal stimulation. VEPs in response to these two stimulus conditions were recorded as a function of stimulus luminance and provided an independent test of the luminance range over which the scotopic and photopic systems operate.

B. Method

Observers

Three observers participated in the experiment, a female, DVD, aged 44, and two males, WK and CA, ages 30 and 24, respectively.

Apparatus

The flickering light source was a bank of 6 Sylvania F875/D fluorescent tubes, spaced 7 mm apart with overall dimensions of 13 X 30 cm. The tubes were mounted in a wooden box covered by a clear plexiglass sheet on the front face. Apertures of various sizes could be placed over the plexiglass thereby controlling the total flux of emitted light in 0.5 log unit steps. Further attenuation of the light was accomplished by placing Kodak neutral density filters over the aperture, moving the light source away from a circular diffusing screen, or by varying the input voltage. The fluorescent tubes were never less than 5 cm behind the diffusing screen. Square wave pulses which drove the fluorescent bulbs were generated by a Wavetek model 116 function generator. Each flicker pulse had a rise and fall time of .1 ms and was 5 ms in duration. The duration of the pulse was measured from one of the tubes by a Mechanical Technology model KD-45A fotonic sensor. The luminance of a flicker pulse was measured

with a Pritchard Spectra Photometer.

Interstimulus intervals (ISI) were generated by interval timers, each capable of generating pulsed intervals between 1 usec and 999 sec. Wher connected in a series the timers could produce a continuous series of intervals, each of which could be independently manipulated. The interval timers triggered the Wavetek which generated the square wave trigger pulses. The ISI used for synchronous stimulation was 110 ms (9.1 Hz).

In addition to producing flicker, the fluorescent tubes also provided a steady adapting field between periods of stimulation. The adapting field was of the same visual area as the stimulus field. Its luminance was controlled by a grass SD5 DC source.

The fluorescent light source backlit a circular diffusing screen, the diameter of which was 9.6° in visual angle. A small red light, subtending a visual angle of 9' and -1.64 fL in luminance, was projected onto the center of the viewing screen and served as a fixation point.

Evoked response recording

Responses were recorded from a Beckman silver-silver chloride electrode located 2.5 cm above the inion referenced to a silver electrode clipped to the right ear. Another Beckman silver electrode clipped to the left ear served as ground. The EEG was recorded by a Schonander Mingograf 800 with a bandpass between 3.33 - 700 Hz. Averaging was done by a Fabri-tek 1072

signal averager. The VEP was digitized using 256 points over a 400 ms period. Veps were summated from 128 sweeps for stimuli at or below .4 log td. The latency of the response was not altered by the number of sweeps in the average.

The averager was triggered on the onset of a stimulus pulse. Successive alternating pulses were labelled "A" and "B" and were used to define two intervals, the AB and the BA interval. For synchronous stimulation the AB and BA intervals were both 100 ms. For asynchronous stimulation the computer triggered on the "A" pulse, which was always preceded by the constant 110 ms interval. The summated VEPs were stored on a Data General Nova 3D minicomputer.

Procedure

Observation of flicker took place in a light proof, electrically shielded booth. The observer was seated 76.5 cm away from the diffusing screen and viewed the flicker binocularly. Each session began with 20 min of dark adaptation. Prior to each stimulation period the observer viewed the adapting field for 36 sec. The luminance of the adapting light was previously judged by the observer to be equal in brightness to the flicker train. Each observer participated in several sessions in order to collect VEPs to synchronous ISI stimulation over the full range of luminances. Stimuli were presented from lowest to highest luminances within a particular session. VEPs were recorded in response to 30 luminances for WK, 26 for DVD,

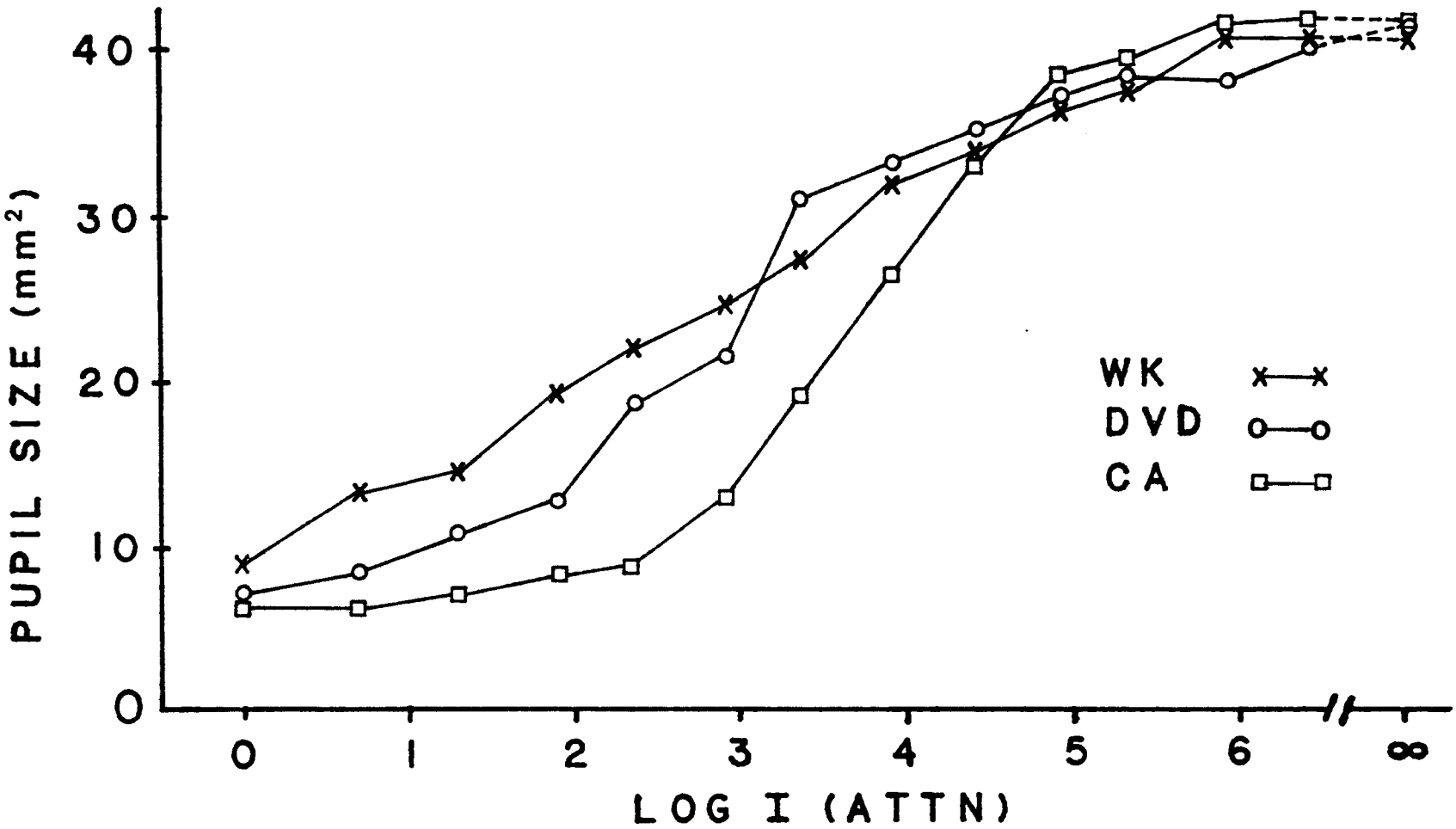
and 16 for CA.

In addition, other sessions were run to obtain VEPs in response to asynchronous stimulation for the determination of latency. Asynchronous stimulation employed two different intervals that alternated in an AB/BA/AB/BA... etc, fashion. The AB interval varied from 97 to 130 ms, while BA was constant at 110 ms. At a particular stimulus luminance four VEPs were collected in which the asynchrony was staggered around the synchronous 110/110 ms ISI. For example, AB/BA intervals of 100/110, 110/110, 117/110, 127/110 might be used. VEPs to asynchronous stimulation were recorded from 10 luminances for WK, 8 luminances for DVD, and 7 luminances for CA.

Pupil size

In order to determine the amount of light falling on the retina the pupil of each observer was measured as a function of flicker luminance. Twelve measurements were obtained from stimulus luminances that varied in approximately .5 log unit steps. The pupil was videotaped during stimulation with an infrared light source. Illumination of the eye by the infrared light did not affect pupil size. The function relating pupil area with stimulus luminance is shown for each observer in Figure 2. Pupil areas for stimulus luminance values which were not measured were interpolated from the graph. The luminance steps used in the collection of the data were converted into units of retinal illuminance (trolands) for each observer.

Figure 2. The relationship between the size of the pupil (in mm^2) and the luminance of the stimulus for each of the three observers. The three points at the far right indicate the pupil size under no stimulus conditions. Luminance is expressed in terms of log units of attenuation of a maximum luminance of 2.62 log fL.


PUPIL SIZE (mm²)
40
30
20
10
0
0
1
2
3
4
5
6
∞
LOG I (ATTN)
WK
DVD
CA

Photopic threshold

The photopic threshold was obtained for each subject under stimulus conditicns similar to those used to collect the VEP data. The stimulation of only ccnes was achieved by centrally fixating a stimulus field of 30' visual angle. A stimulus of this size is thought to be completely contained within the rod-free fovea (Rodieck, 1973).

A red fixation light was provided which was 2.4' in visual angle and approximately .2 log units above threshold. Fixation was aided by positioning the observer's head in a head restraint equipped with a chin rest. In previous work with the same observers under similar experimental conditions fixation was monitored with an infrared video system. It was found that less than 1% of the trials had to be rejected because of eye movements. Therefore, eye fixation was not monitored in the present experiment.

Between trials the observer viewed an adapting field of the same retinal area as the stimulus field. The luminance of the adapting field was maintained at the same level of brightness as the flicker train. Prior to the experiment each observer equated the brightness of the adapting light and the flicker train on a 9.6° field for each level of illuminance used in the experiment.

The method of forced choice was used. Pairs of trials were presented successively in which flicker appeared in either the first of second trial. The observer was first dark-adapted for

20 min. A trial consisted of a 5 sec warning after which the adapting light was turned off and either the flicker or no light was presented for a 2 sec period. After every second trial the observer reported whether the flicker was seen in the first or second trial. Twenty-five trials were run at each illuminance step.

Data analysis

Peaks were located in the VEPs by the computer algorithm, Peakfind1. The data were smoothed using two digital smoothing algorithms. The first was a non-linear smoother, 4253H,Twice, designed to extract the locally smooth pattern from data contaminated with spikey noise (Velleman, 1980). The second was a digital low pass filter with a high frequency cutoff of 19.1 Hz (-3dB). These smoothers had the advantage of producing little or no phase shifting of the data (Velleman, 1980).

Positive and negative peaks were identified by first locating all turning points on the filtered waveform which were defined as a change in the sign of the slope, including a change to zero slope. All turning points identified were subjected to two criteria which were applied to the data in two successive passes. The first was an amplitude criterion which rejected a turning point if its amplitude was less than four per cent of the difference between the minimum and maximum amplitude values of the waveform. The second criterion replaced two adjacent turning points of the same polarity with a single point which

was calculated as a weighted average of the original two points.[1] In a second pass a 12% difference amplitude criterion was applied to the data and if two adjacent points of like polarity were less than 40 ms apart they were replaced by a weighted average.

Since each waveform consisted of 3 cycles a criterion for the reliability of the occurrence of a peak was imposed on the data. A turning point was accepted as a peak if was repeated in at least 2 cycles. The process identified one or two components (defined as a negative-positive peak) per stimulus pulse. The peaks found by Peakfind1 for all data sets are shown in Appendix C.

Latency determination

The latencies of the peaks identified by the computer algorithm were determined by the asynchronous stimulation method at selected illuminance steps. At any particular illuminance, four VEPs were recorded, three to asynchronous ISIs and one to a synchronous ISI. Latency was measured by determining how a peak varied in time as a result of the stimulus asynchrony. Thus, it was necessary to identify peaks in the asynchronous VEPs that corresponded to the peaks in the synchronous records. However, because of the large/small effect (Diamond, 1979) asynchronous stimulation produced smaller peaks in the waveform than did synchronous stimulation, and, consequently, were not detected by

[1]The formula for finding the weighted average of two adjacent turning points is described in Appendix B.

the algorithm, Peakfind1. Therefore, a more sensitive algorithm, Peakfind2, was used which differed from Peakfind1 in its filtering characteristics and the criteria used to select potential peaks.

The data were smoothed with 4253H,Twice and then low pass filtered with a high frequency cutoff of 31.9 Hz (-3 dB). Turning points with amplitudes larger than 4% of the difference between the minimum and maximum amplitudes of the waveform were defined as peaks. Two adjacent peaks of the same polarity were replaced by a weighted average of the two peaks. This algorithm resulted in a larger number of peaks detected, however, only peaks in the synchronous VEPs found by Peakfind1 were analyzed for latency.

Peaks in the asynchronous data that were associated with the peaks in the synchronous data were found by the following method. Two peaks were associated with one another if the peak of a VEP recorded at one ISI, the test VEP, occurred within a critical time from a peak of another VEP, the reference VEP. A critical time range, which was dependent on the difference in ISI at which the two VEPs were recorded, was defined as $CR = t \pm 2.5\,\Delta I$, where 't' is the time of the reference peak and 'ΔI' is the difference between the variable intervals of the test and reference VEPs. The reference peaks were taken from the synchronous VEPs, and the critical ranges for these peaks were tested against the peaks of the asynchronous VEPs one step removed from the ISI of the synchronous VEP. For ISIs of

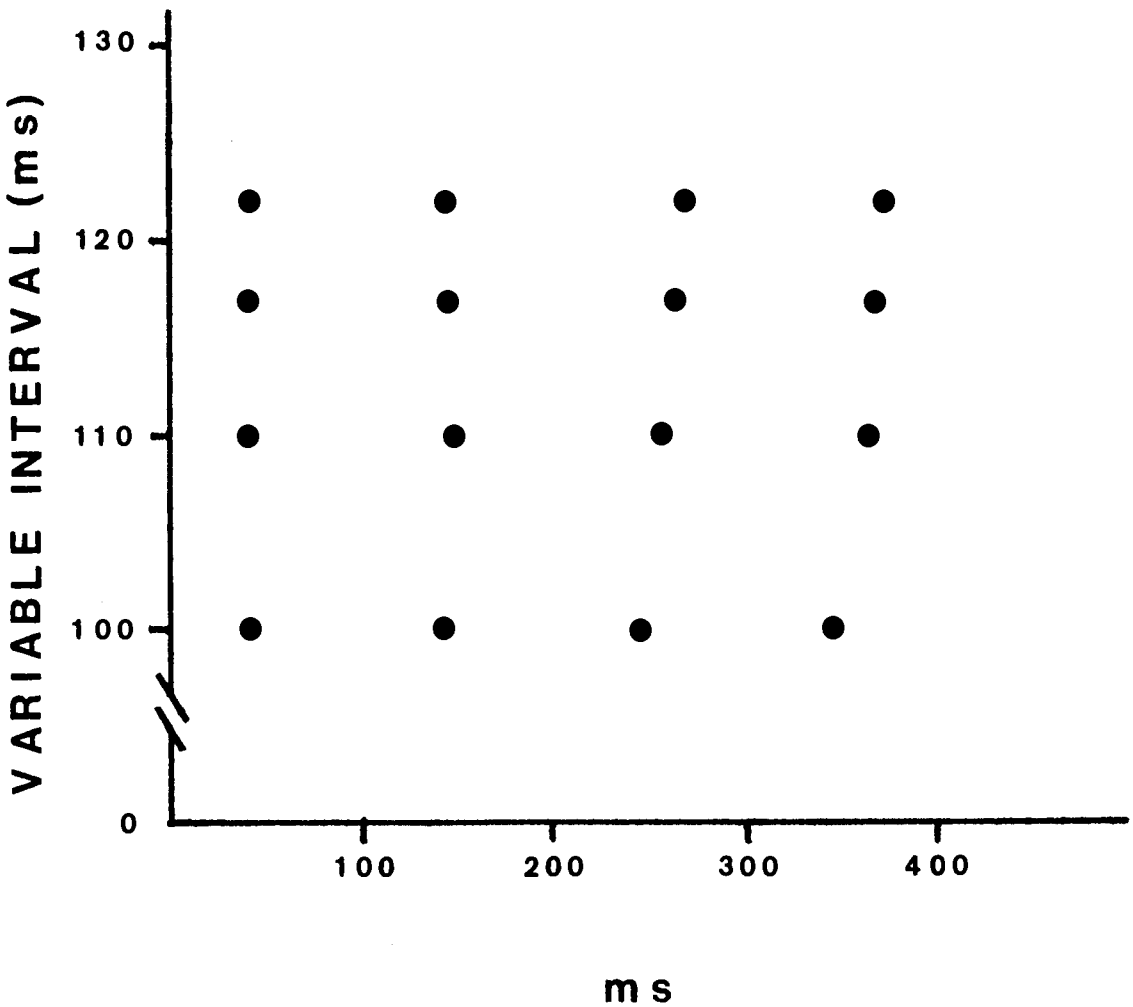
128/110 ms, 117/110 ms, 110/110 ms, and 100/110 ms, VEPs of ISIs 117/110 ms and 100/110 ms would be tested against the reference VEP of 110/110 ms ISI. Once the times of the peaks of these asynchronous VEPs were determined, they would be used as reference VEPs for asynchronous VEPs one step removed from them. Thus, the peaks of the asynchronous VEP of 117/110 ms ISI would be used as a reference for the peaks of the VEP of 128/110 ms ISI. If more than two peaks were found within the critical range the times of the peaks were averaged to yield a single peak. Positive and negative peaks were analyzed separately.

The times of all the peaks (either positive or negative) in a set of 4 VEPs made up a set of data points from which the latencies of the synchronous VEP peaks were calculated. Each data set was made up of a maximum of 16 data points. If fewer than 9 points were found for a data set the latency of that peak was not determined. The latencies of all peaks were calculated with respect to the first pulse of the sweep at time zero. If the times that a peak occurred in the set of VEPs were plotted as a function of the ISI at which the VEP was recorded a pattern of data points such as the one shown in Figure 3 would be produced. This data set may be described in matrix notation by the equation:

$$\mathbf{T} = \mathbf{AB} \times \mathbf{M} + \mathbf{BA} \times \mathbf{N} + \mathbf{R}$$

where **T** is the matrix of data points, **AB** is a vector of the variable intervals, **BA** is a vector of the constant intervals, **M** and **N** are vectors of integer multipliers, and **R** is the matrix of

Figure 3. A hypothetical graph of the time of occurrence of a VEP peak over a 400 ms period in response to various ISIs plotted against the variable ISI interval. The peak was repeated in four cycles of each VEP.


VARIABLE INTERVAL (ms)
130
120
110
100
0
100
200
300
400
ms

residuals. Expansion of the equation yields:

$$\begin{bmatrix} t_{11} & \cdots & t_{1j} \\ \vdots & & \vdots \\ t_{i1} & \cdots & t_{ij} \end{bmatrix} = \begin{bmatrix} ab_1 \\ \vdots \\ ab_i \end{bmatrix} \bullet \begin{bmatrix} m_k & m_{k+1} & \cdots & m_{k+j-1} \end{bmatrix} +$$

$$\begin{bmatrix} ba_1 \\ \vdots \\ ba_i \end{bmatrix} \bullet \begin{bmatrix} n_k & n_{k+1} & \cdots & n_{k+j-1} \end{bmatrix} + \begin{bmatrix} r_{11} & \cdots & r_{1j} \\ \vdots & & \vdots \\ r_{i1} & \cdots & r_{ij} \end{bmatrix} .$$

Subsets of M and N multipliers are inserted into the equation from the sets:

$M_K \in \{-2\ -2\ -1\ -1\ 0\ 0\ 1\ 1\ 2\}$, $N_K \in \{-3\ -2\ -2\ -1\ -1\ 0\ 0\ 1\ 1\}$ beginning with the first element in each set, that is, k = 1, and the equation is solved for R. Successive subsets of M and N are inserted into the equation by incrementing k until all subsets have been used. The variance of the elements in each of the residual matrices is calculated. The latency of the peak is defined as the mean of the elements of the R matrix with the least variance.

C. Results

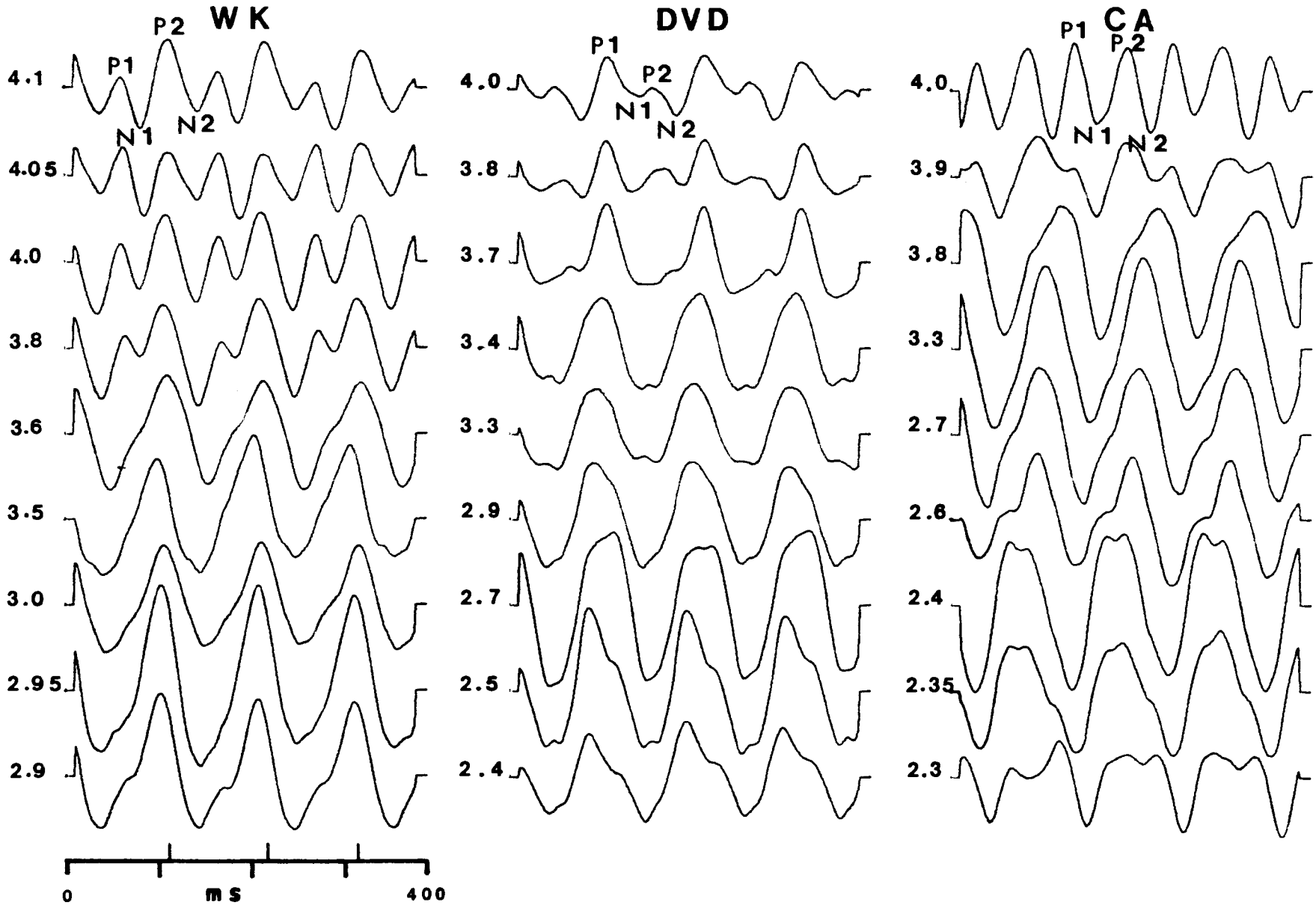
I. Experiment 1

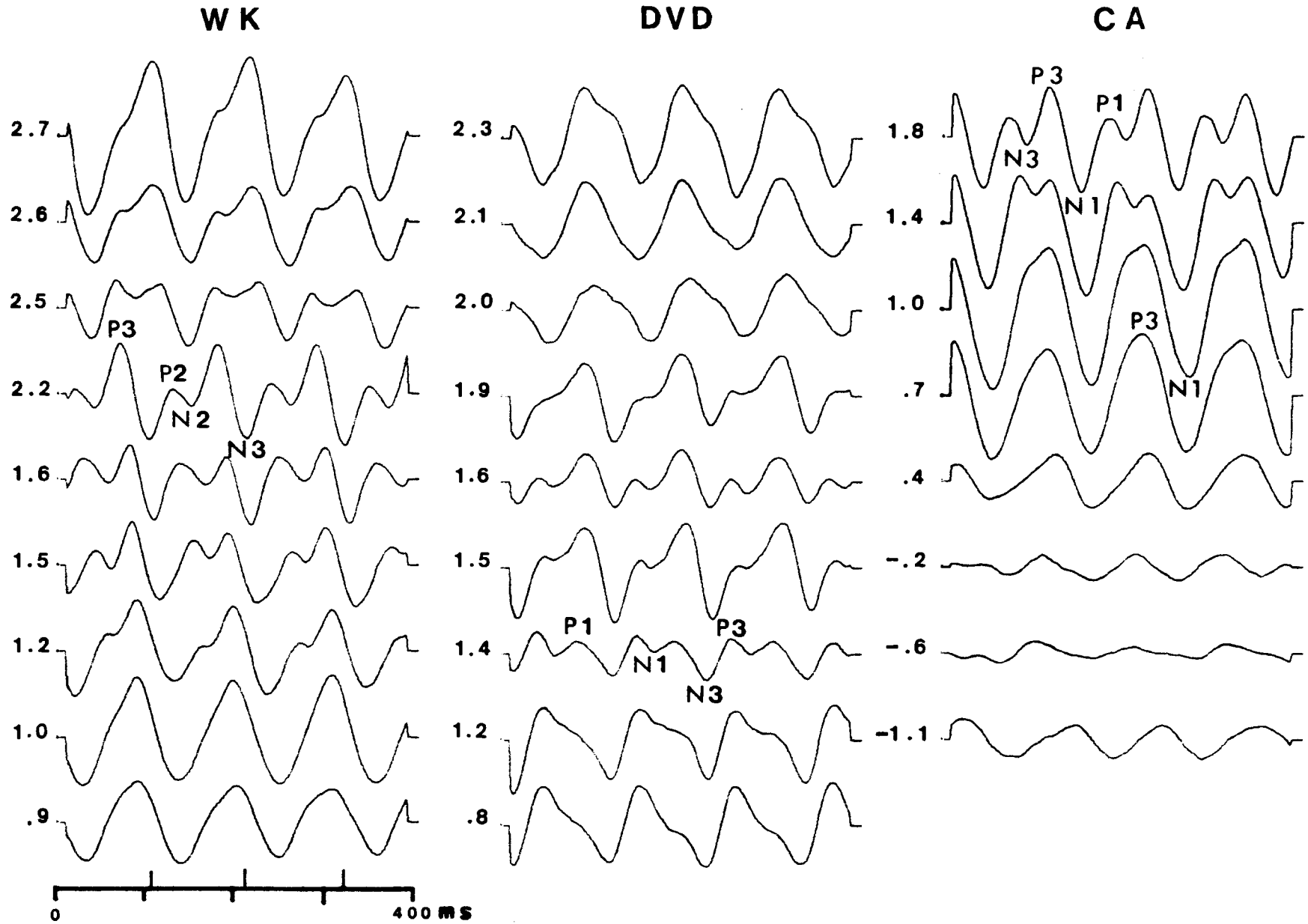
Figure 4 illustrates the changes in the VEP waveform as a function of retinal illumination. The data of all observers showed a similar pattern in the way in which waveform changed with retinal illuminance. At lowest levels of illuminance the VEP consisted of a single component per pulse. As illuminance increased the VEP alternated from one to two components. Over the 5.7 log unit range the transition from a single to a double component response occurred between 1.0 and 1.5 log td and between 3.6 and 3.9 log td while the transition from a double to a single component response occurred between 1.6 and 2.6 log td.

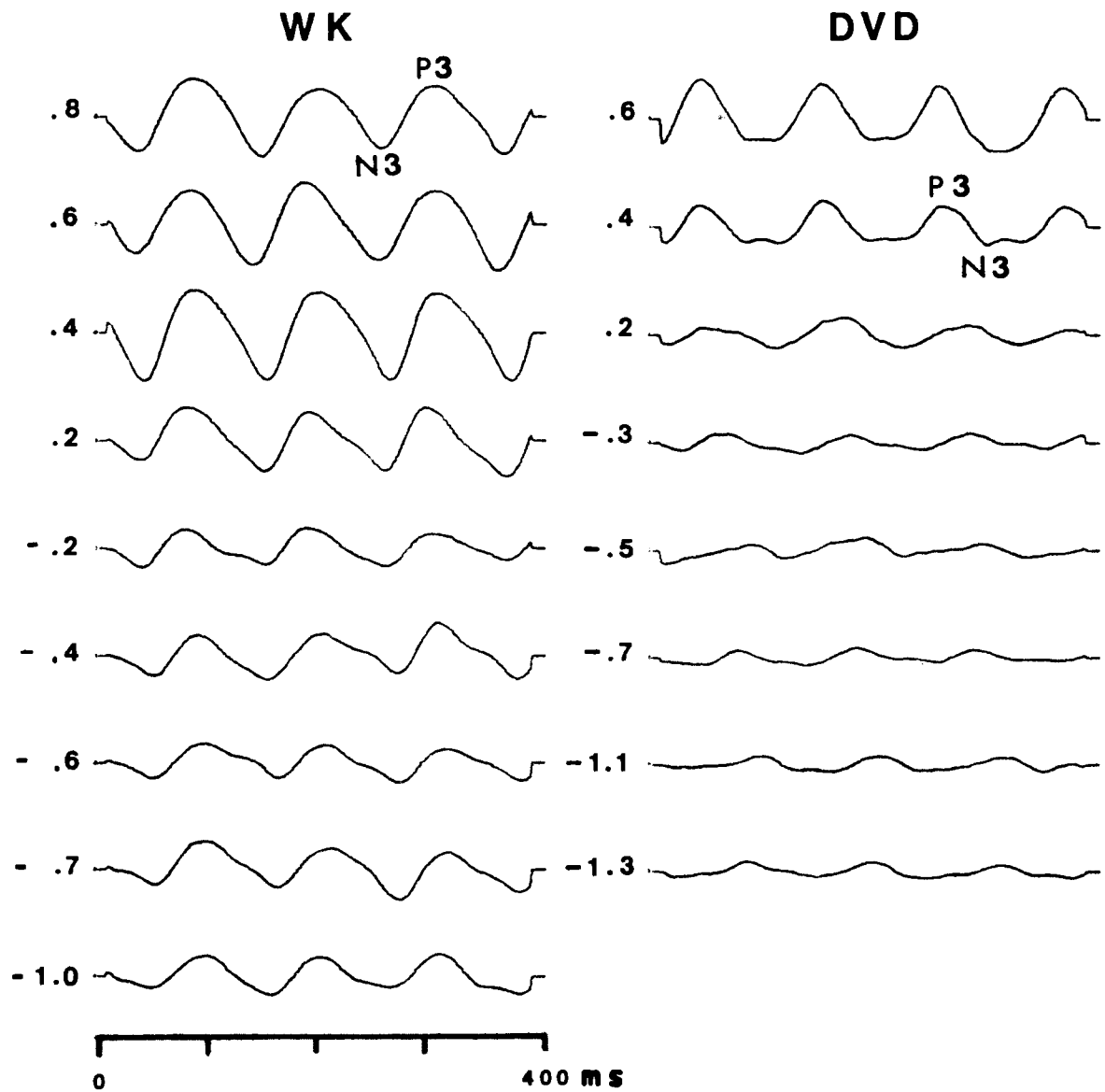
Peak latencies of the VEPs were determined at selected illuminance levels which sampled the full illuminance range except at the lowest levels. The increased variability of the VEP following response recorded to asynchronous stimulation at lower illuminances did not permit determination of latency.

VEP latencies are graphed in Figure 5 as the average of the positive and negative VEP peak latencies of each observer. Where the VEP consisted of a double component response the latency was the average of two positive and two negative peaks. It can be seen that the latency functions separated into two branches. The

Figure 4. Sets of VEPs obtained from each observer over the range of illuminances indicated. Sweep duration is 400 ms. Stimulus pulses are indicated at the bottom of each page. Responses consisted of either one or two components per pulse. Illuminances were measured in log trolands.


WK
P1
P2
N1
N2
4.1
4.05
4.0
3.8
3.6
3.5
3.0
2.95
2.9
DVD
P1
P2
N1
N2
4.0
3.8
3.7
3.4
3.3
2.9
2.7
2.5
2.4
CA
P1
P2
N1
N2
4.0
3.9
3.8
3.3
2.7
2.6
2.4
2.35
2.3
0
ms
400


WK
DVD
CA
2.7
2.6
2.5
P3
2.2
P2
N2
N3
1.6
1.5
1.2
1.0
.9
2.3
2.1
2.0
1.9
1.6
1.5
P1
P3
1.4
N1
N3
1.2
.8
1.8
P3
P1
N3
1.4
N1
1.0
P3
.7
N1
.4
-.2
-.6
-1.1
0
400 ms


WK
DVD
P3
N3
P3
N3
.8
.6
.4
.2
- .2
- .4
- .6
- .7
- 1.0
.6
.4
.2
- .3
- .5
- .7
-1.1
-1.3
0
400 ms

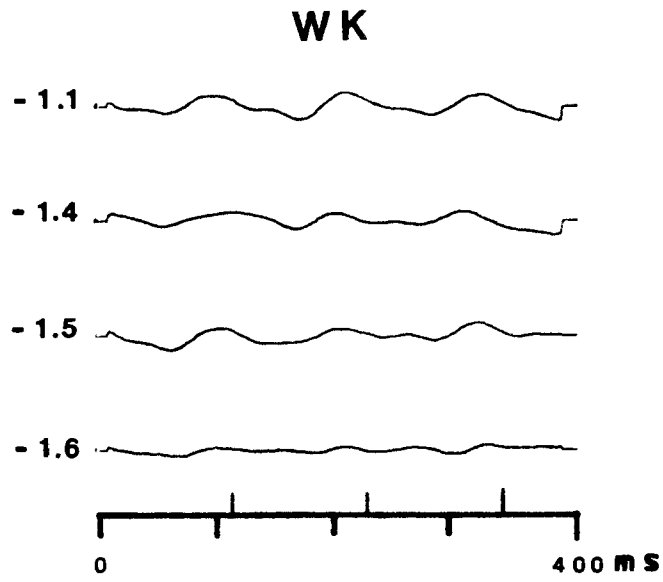
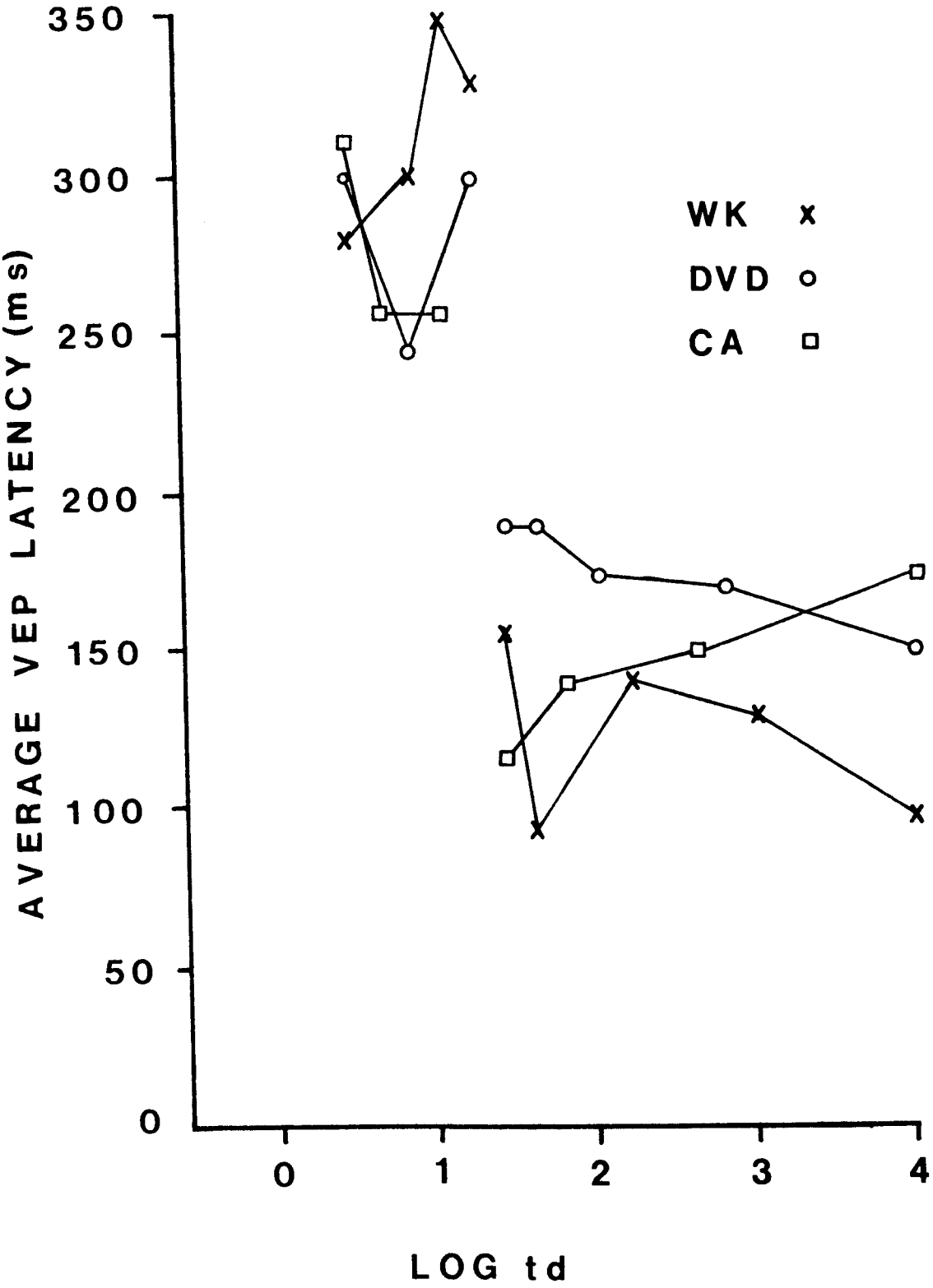

WK
- 1.1
- 1.4
- 1.5
- 1.6
0
400 ms

Figure 5. The average latency of the steady-state VEP is shown for each observer as a function of stimulus illuminance as determined by the asynchronous method. The data were plotted on the abscissa in increments of 0.2 log units.


350
300
250
200
150
100
50
0
AVERAGE VEP LATENCY (ms)
WK x
DVD o
CA □
0
1
2
3
4
LOG td

upper branch described a late latency function at the lower illuminances extending from .4-1.2 log td. The lower branch, representing an early latency function at higher illuminances, extended from 1.4-4.0 log td. The illuminance level at which the discontinuity in the curve occurred was similar for all observers. For WK and DVD the discontinuity occurred between 1.2 and 1.4 log td, and for CA it was between 1.0 and 1.4 log td. The change in average peak latency between the two branches of the curve was 173 ms for WK, 144 ms for CA, and 110 ms for DVD. This was a mean latency change of 142 ms (SD = 31.5 ms) over an average illuminance range of .27 log td (SD = .12).

Peaks were labelled on the basis of their latency and polarity starting from the high end of the illuminance scale. As the waveforms variously changed from double to single component responses some peaks dropped out and new peaks emerged. Thus, successive positive peaks were labelled P1, P2, and P3, and negative peaks were labelled N1, N2, and N3 as shown in Figure 4.

The latencies of individual peaks are graphed for observers WK, DVD, and CA in Figures 6, 7, and 8, respectively. The standard errors of the latency means were all less than 3.0 ms. It was apparent that the low illuminace single component VEPs were of long latency. When the VEP changed from a single to a double component at intermediate illuminances the latencies of the peaks shifted from long to short. As illuminance increased the latencies of all peaks continued to be short.

Figure 6. A plot of the latencies of the individual peaks of the steady-state VEP as a function of illuminance for observer WK. Data on the abscissa were plotted in increments of 0.2 log units. The arrow indicates the least upper bound of the psychophysical photopic threshold.

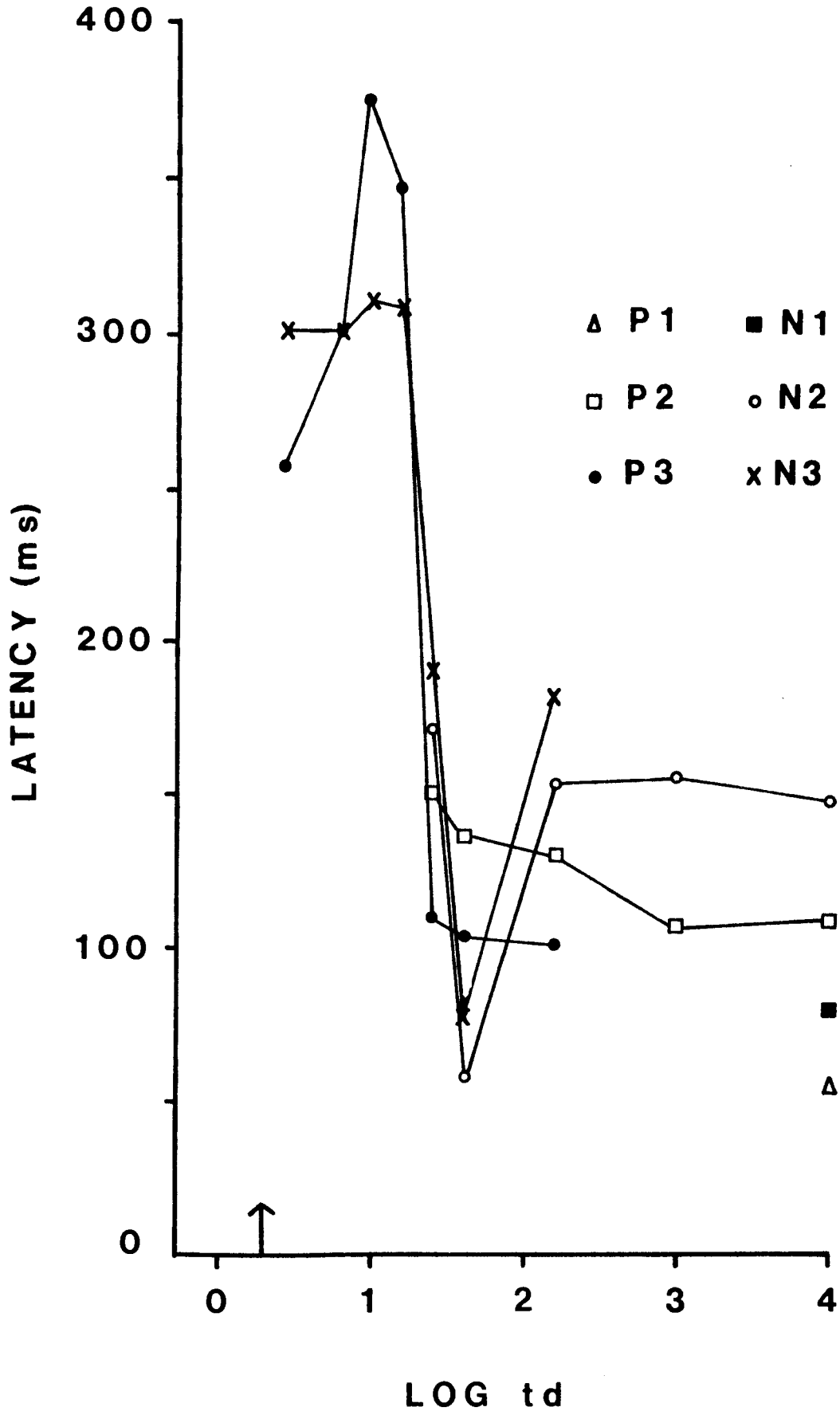

400
300
200
100
0
LATENCY (ms)
P1
N1
P2
N2
P3
N3
0
1
2
3
4
LOG td

Figure 7. The relationship between individual VEP peak latencies and illuminance for observer DVD. Data were plotted on the abscissa in increments of 0.2 log units. The arrow indicates the least upper bound of the psychophysical photopic threshold.

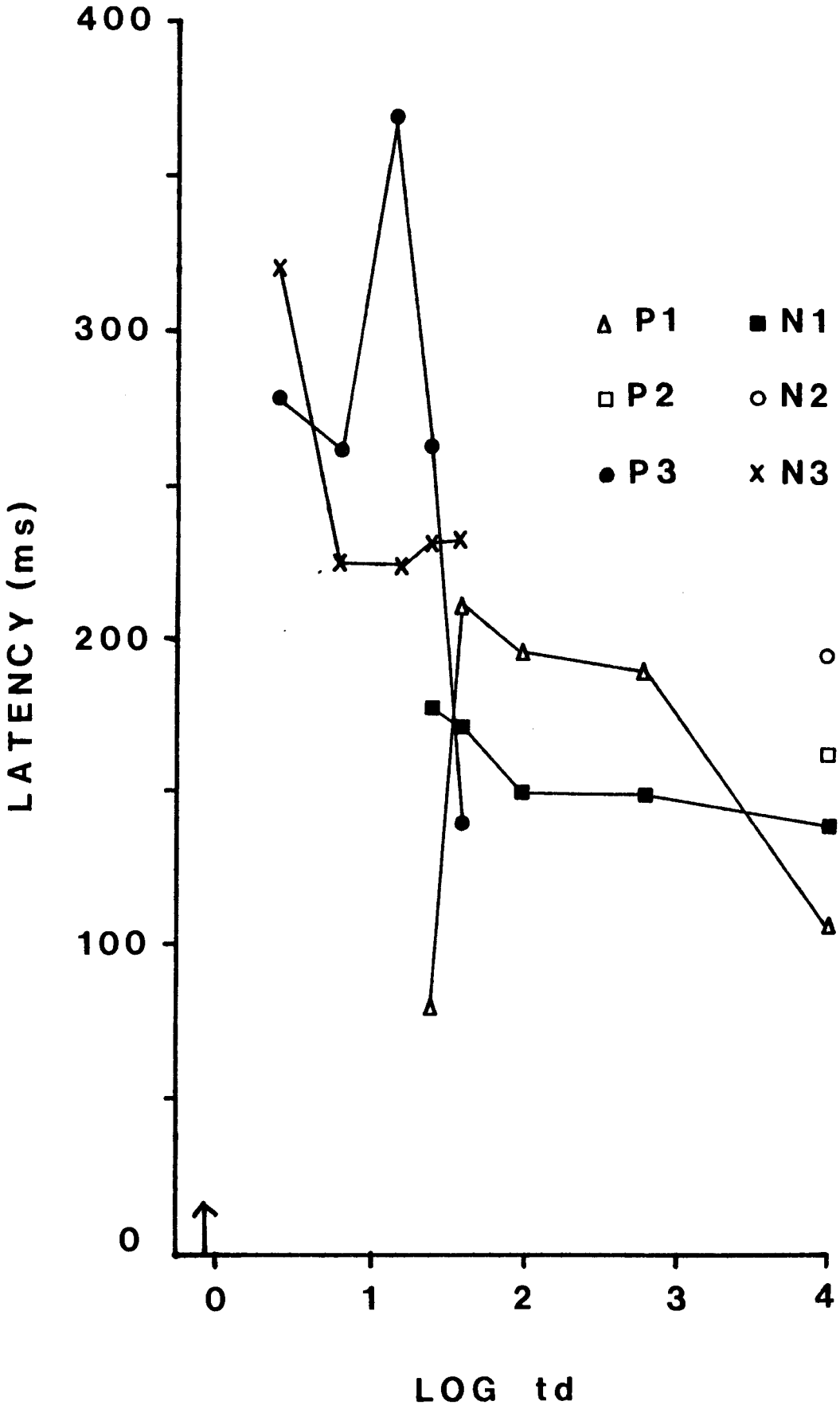

400
300
200
100
0
LATENCY (ms)
P1
N1
P2
N2
P3
N3
0
1
2
3
4
LOG td

Figure 8. The relationship between the individual peak latencies and stimulus illuminance for observer CA. Data were plotted on the abscissa in increments of 0.2 log units. The arrow indicates the least upper bound of the psychophysical photopic threshold.

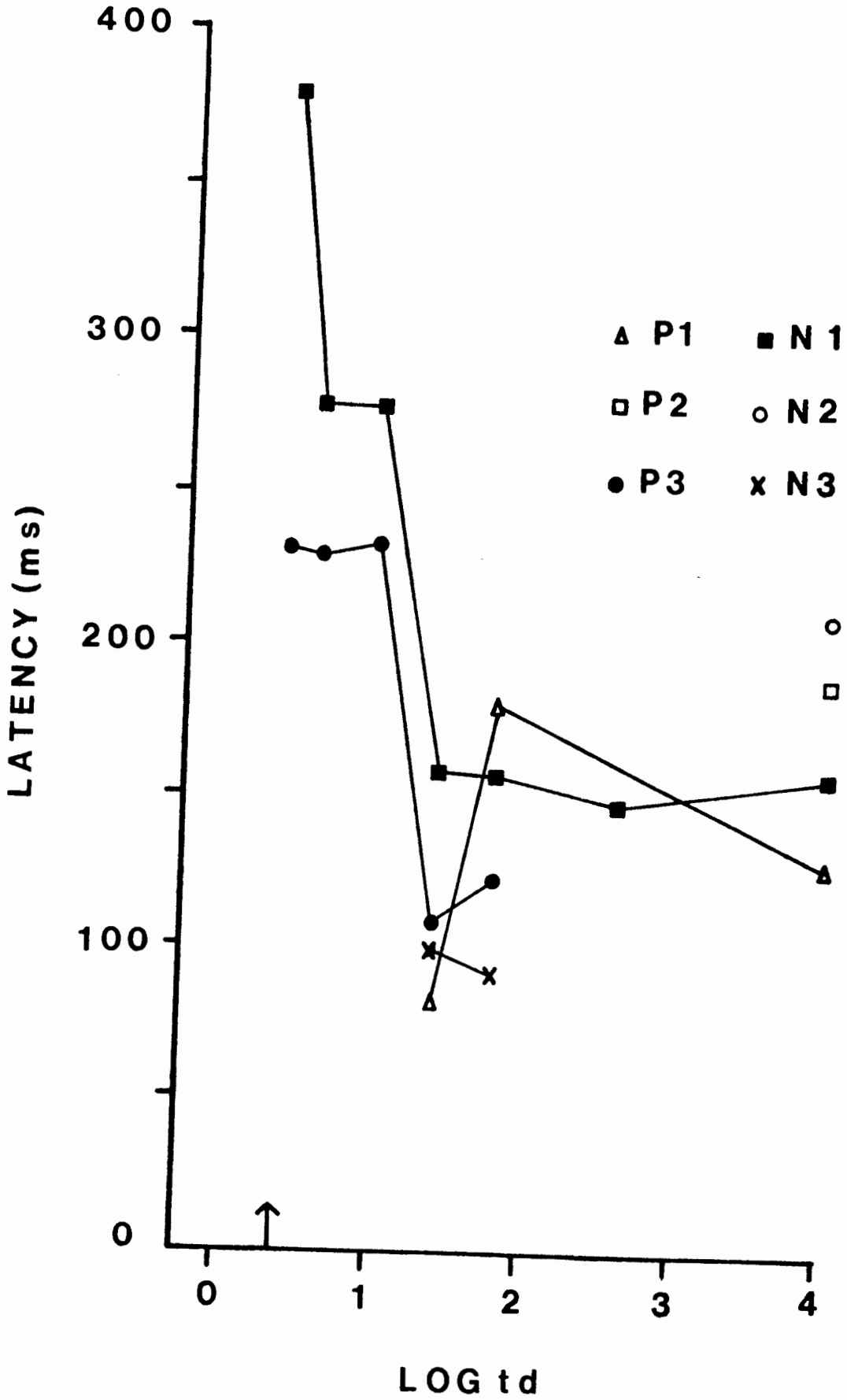
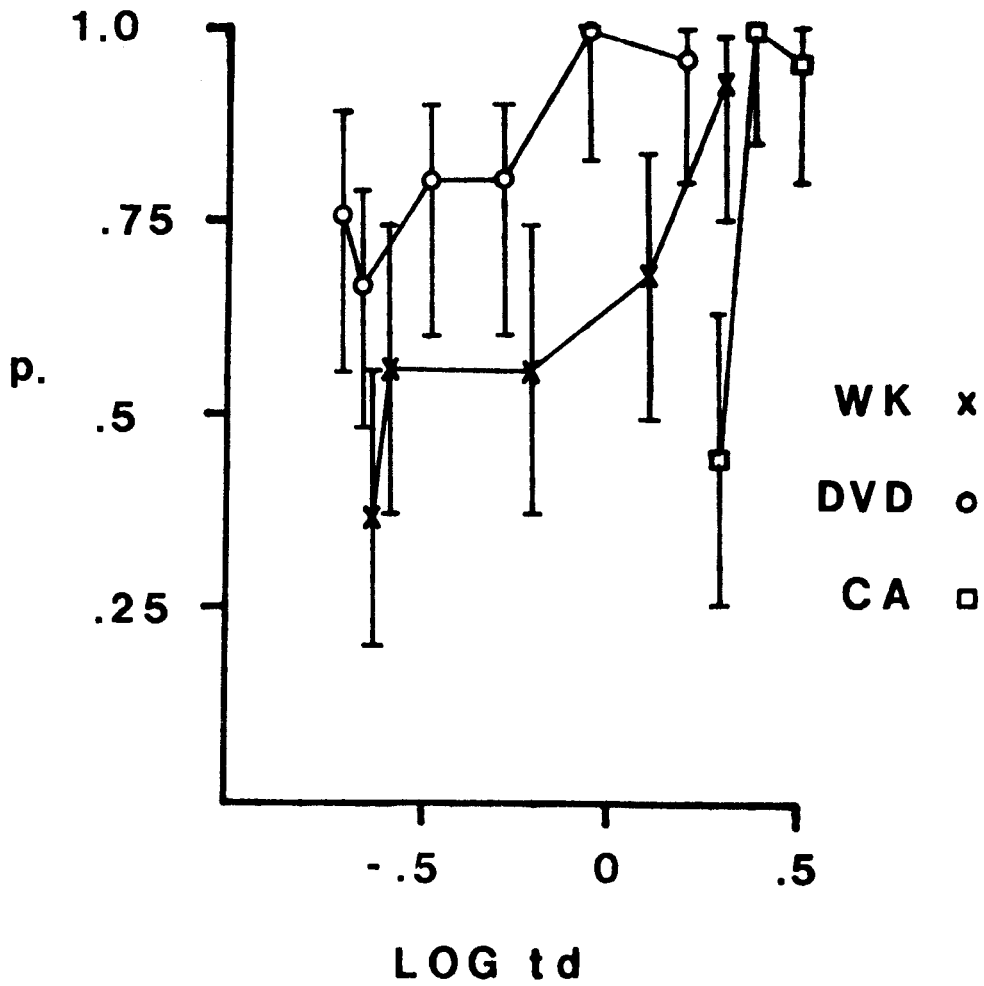

400
300
200
100
0
LATENCY (ms)
P1
N1
P2
N2
P3
N3
0
1
2
3
4
LOG td

Figure 9. The proportion of correct responses in the forced-choice threshold detection of a brief flickering light. The vertical bars at each point indicate 95% confidence intervals. Twenty-five responses were collected for each data point.

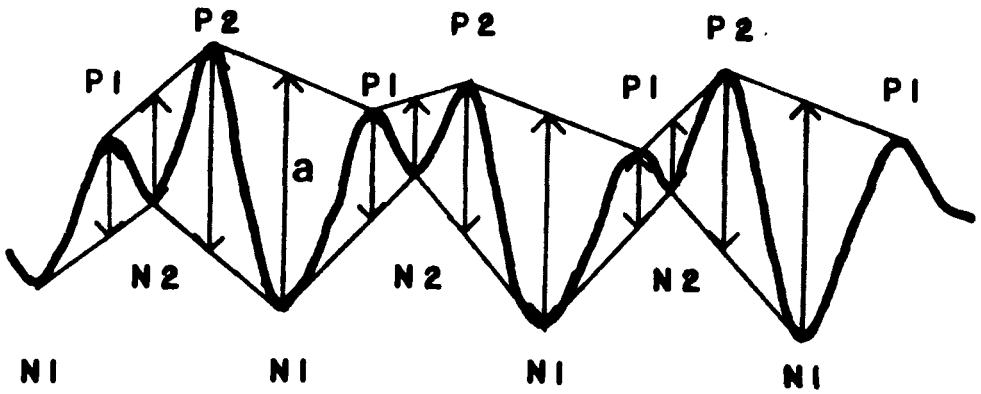

1.0
.75
.5
.25
p.
-.5
0
.5
LOG td
WK x
DVD o
CA □

The results of the psychophysical photopic threshold experiment are shown in Figure 9 as the proportion of correct responses from each observer. The vertical bars represent 95% confidence intervals. For each observer the lower and upper bounds of the photopic threshold were estimated from the graph. The upper bound was defined was the lowest illuminance level for which the 95% confidence interval did not extend below the 75% correct level. Thus, the upper bound for WK was .3 log td, -.05 log td for DVD, and .4 log td for CA. The lower bound was defined as the highest illuminance level for which the 95% confidence interval did not extend above the 75% correct level. The lower bound for WK was -.2 log td, and .28 log td for CA. A lower bound was not determined for DVD, however, as shown by the graph it appeared to be below -.7 log td.

The difference between the upper bound of the photopic threshold and the illuminance at which the VEP shift to the short latency function occurred was 1.1 log td for WK, 1.45 log td for DVD, and 1.0 log td for CA. That is, the long latency VEP persisted for an average of 1.2 log td (SD = .24) above the upper bound of the photopic threshold.

Amplitudes of the negative and positive peaks were measured as shown in Figure 10. Since negative and positive peaks described similar functions their amplitudes were averaged. The combined positive and negative amplitudes are shown for observers WK, DVD, and CA in Figures 11, 12, and 13, respectively. The functions were similar for all observers. The

Figure 10. Illustration of how the amplitude of a peak was measured. The amplitude was measured as in (a) and was averaged from several repetitions of the peak within the waveform.



amplitude (a)

low illuminance component, P3N3 (P3N1 for CA), reached a maximum amplitude between .8-1.0 log td, and disappeared between 1.6 and 2.4 log td. The intermediate intensity component, P2N2 (P1N1 for CA and DVD) first appeared at 1.4 log td for all observers and reached a maximum amplitude between 2.6 and 3.2 log td and diminished thereafter. At the highest illuminances another component appeared between 3.6-4.0 log td.

Also shown in each of the three figures is an arrow which illustrated the point at which short latencies were found. It can be seen that the low illuminance component peaked prior to this point and was declining at the time the intermediate component first appeared.

To summarize, a late latency component showed an increasing amplitude function in the low to intermediate illuminance range. This component decreased in amplitude as the latency of the response shifted from long to short. The second component, which was coincident with the first component in the intermediate illuminance range, was an early latency component which initially increased in amplitude and then decreased as illuminance increased.

Figure 11. Amplitudes of three VEP components (each consisting of an average of a positive and a negative peak) for observer WK as a function of illuminance. Amplitudes were plotted in increments of 0.2 log units of illumination. The arrow shows the illuminance level at which the VEP shifted to a short latency response.

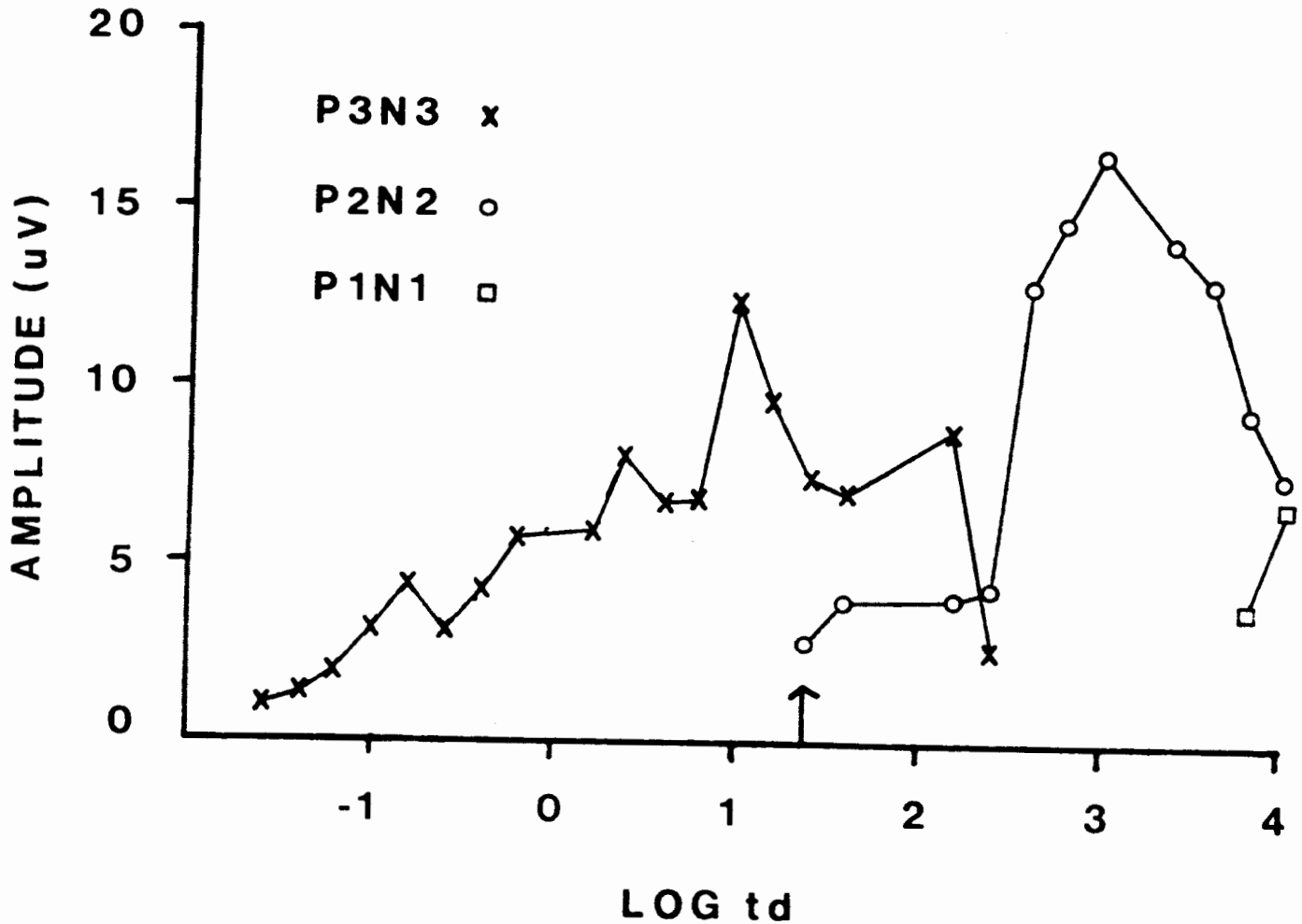

20
15
10
5
0
AMPLITUDE (uV)
P3N3 x
P2N2 o
P1N1 □
-1
0
1
2
3
4
LOG td

Figure 12. Amplitudes of three VEP components (each consisting of an average of a positive and a negative peak) for observer DVD as a function of illuminance. Amplitudes were plotted in increments of 0.2 log units of illumination. The arrow shows the illuminance level at which the VEP shifted to a short latency response.

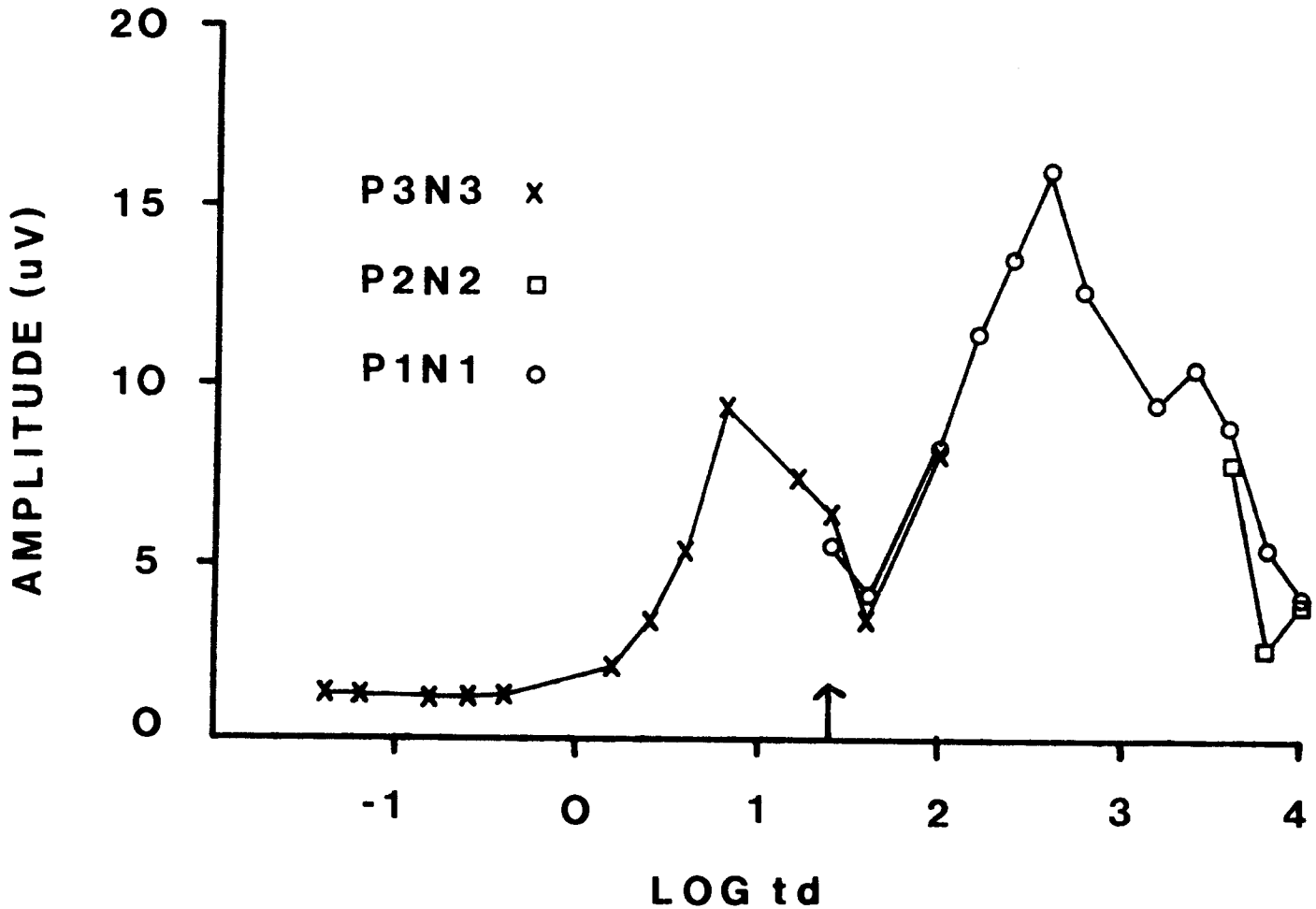
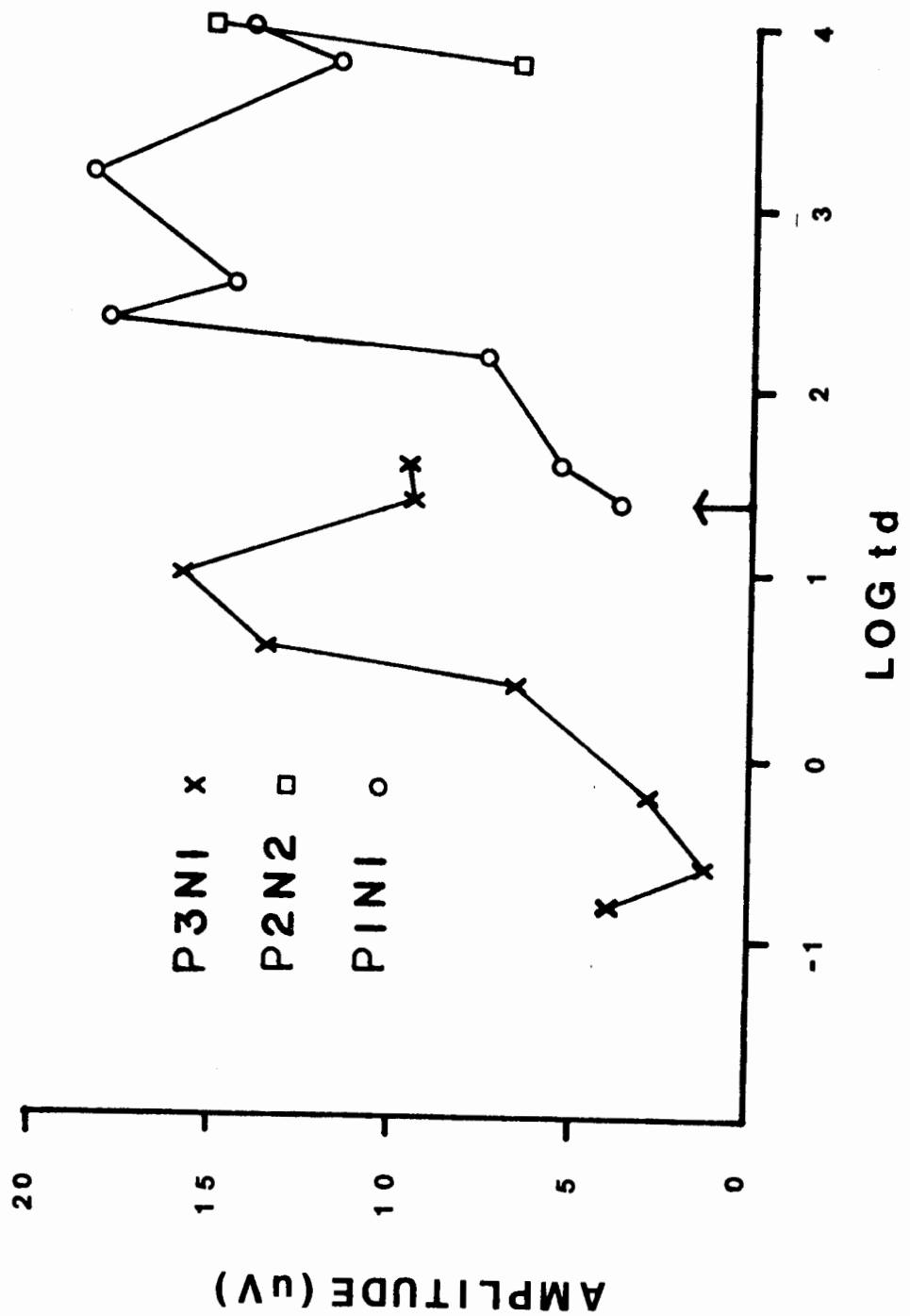

20
15
10
5
0
AMPLITUDE (uV)
P3N3 x
P2N2 □
P1N1 o
-1
0
1
2
3
4
LOG td

Figure 13. Amplitudes of three VEP components (each consisting of an average of a positive and a negative peak) for observer CA as a function of illuminance. Amplitudes were plotted in increments of 0.2 log units of illumination. The arrow shows the illuminance level at which the VEP shifted to a short latency response.


AMPLITUDE (uV)
20
15
10
5
0
-1
0
1
2
3
4
LOG td
P3N1 x
P2N2 □
P1N1 o

II. Experiment 2

The relative contributions of the rods and cones to the steady-state VEP were investigated at levels of illuminance immediately above the photopic threshold by selectively stimulating retinal areas containing either rods or cones.

Method

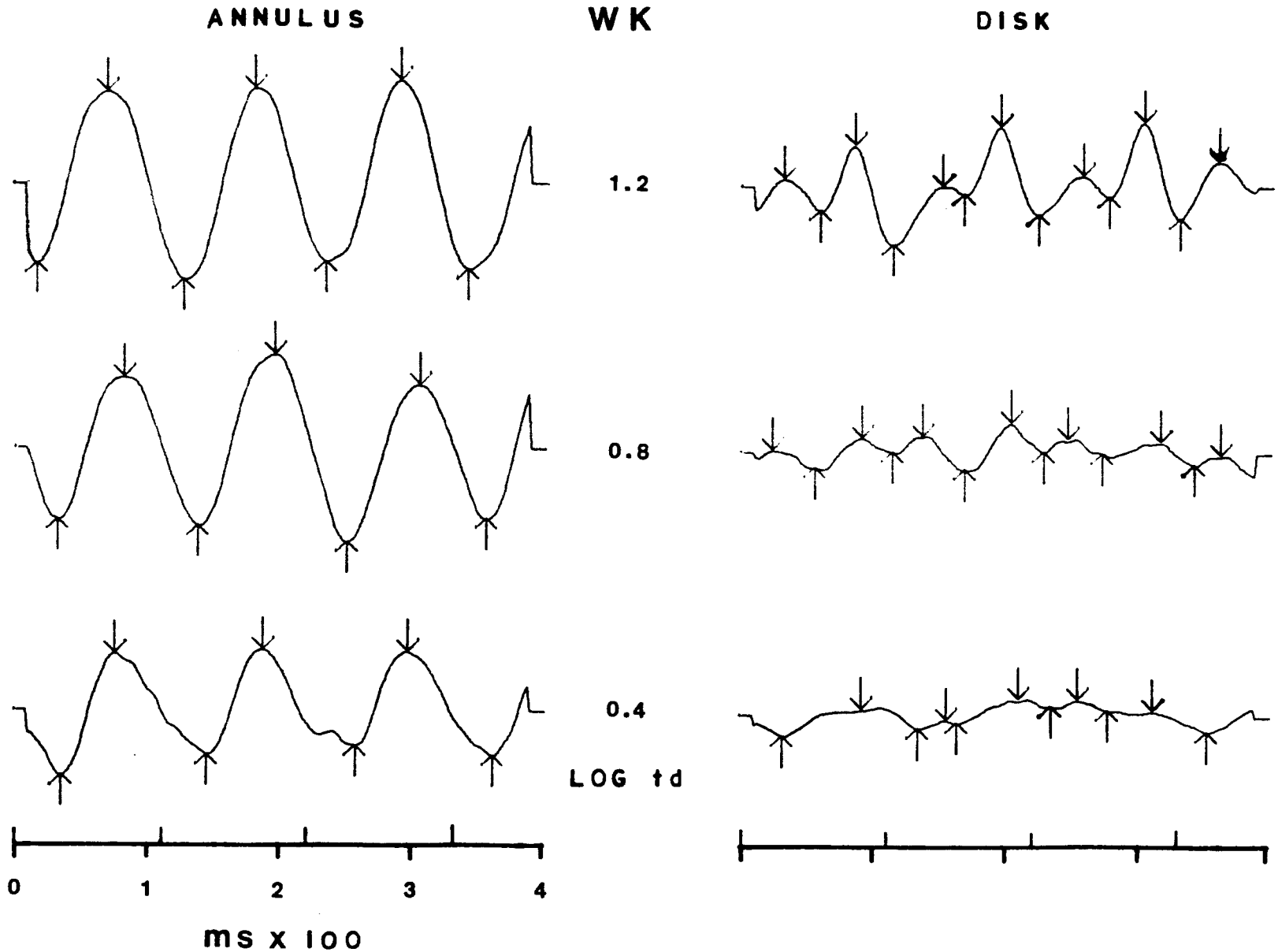
The 9.6° stimulus was partitioned into a centrally viewed 3° disk and a 9.6° annulus with an interior diameter of 3°. This partition did not completely separate rods from cones. However, the ratios of rods to cones within these stimulus fields were estimated from the data of Østerberg (1935). The ratio of rods to cones in the area circumscribed by the annulus was about 6:1. The ratio of cones to rods in the central 3° area was about 5:1.

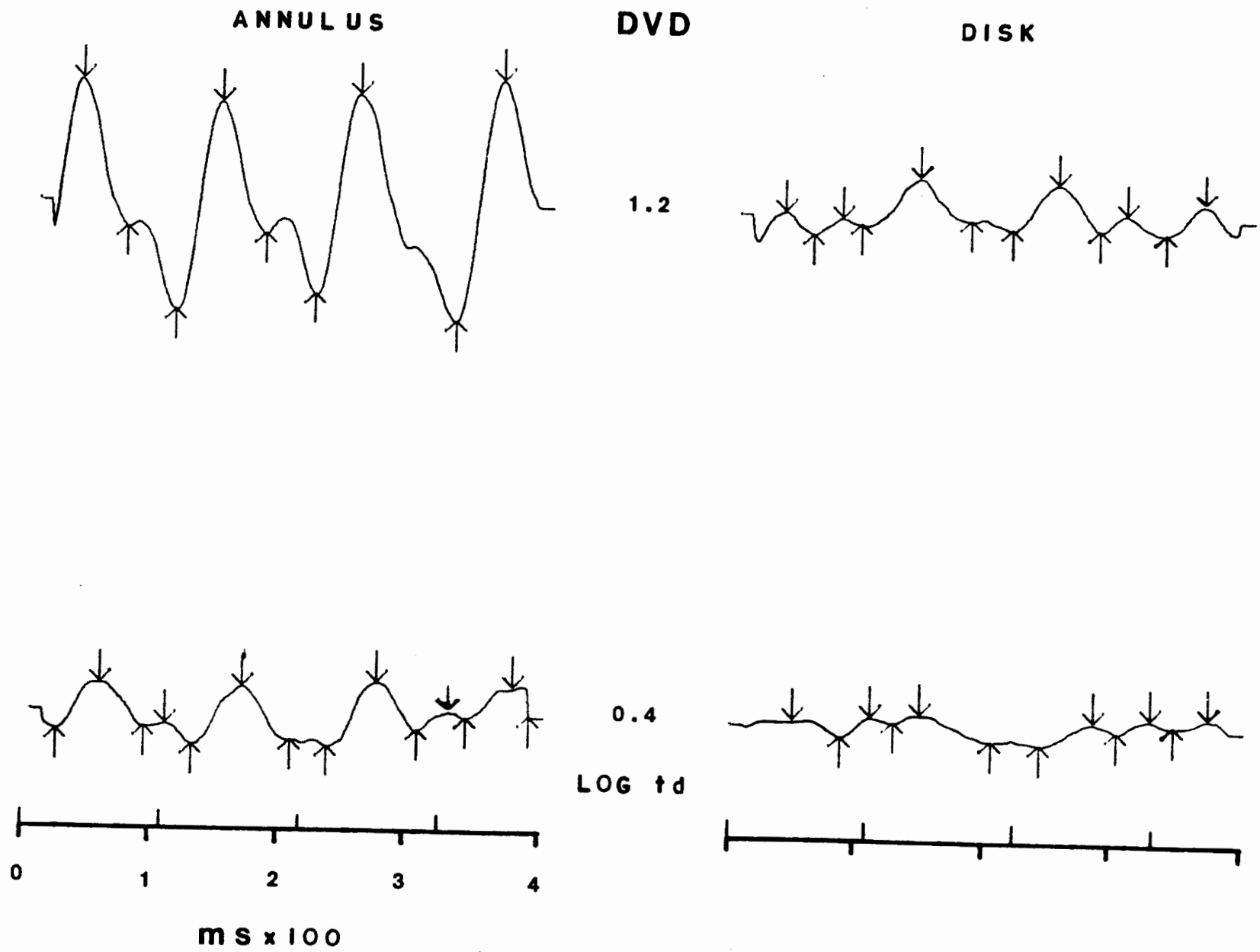
The effect of scattered light was controlled by an adapting light which illuminated the complementary part of the stimulus field. The brightness of the adapting light was adjusted at each stimulus illuminance such that no flicker was seen by the observer in the unstimulated portion of the field. It was found that the adapting light had to be brighter than the flicker in order for the flicker to be seen only within the boundaries of the stimulus field. The luminances of the annulus and disk adapting fields were -1.1, -.8, and -.2 log fL. for stimulus luminances of -1.7, -1.3, and -.8 log fL., respectively.

Results

VEPs obtained from stimulation of the annulus and disk are shown for all observers in Figure 14. Positive and negative peaks were found by applying the algorithm, Peakfind1, to the data. Positive and negative amplitudes were combined to yield an average amplitude measurement for each VEP. The average amplitudes of the disk and annulus VEPs are shown as a function of illuminance in Figure 15. The annulus and disk VEP amplitudes each displayed a monotonic increase with illuminance. The annulus VEP had a much larger amplitude at each illuminance compared to the disk VEP. The ratios of the amplitudes of the annulus to the disk VEPs are shown in Figure 16 in terms of raw amplitude and per unit area of retina. The average amplitude of the annulus VEP of the three observers across stimulus intensity was 6.0 (SD=1.5) times that of the disk VEP. On the other hand, the average ratio of the disk to the annulus VEP amplitude per unit retinal area was 1.8:1 (SD=.32).

Figure 14. VEPs recorded in three observers at three illuminances in response to a centrally fixated circular stimulus subtending 3° in visual angle and a 9.6° annulus with an interior diameter of 3°. The arrows show the peaks found by Peakfind1 from which amplitudes were measured. Stimulus intensity is in log trolands; sweep duration is 400 ms.


ANNULUS
WK
DISK
1.2
0.8
0.4
LOG td
0
1
2
3
4
ms x 100


ANNULUS
DVD
DISK
1.2
0.4
LOG td
0
1
2
3
4
ms x 100

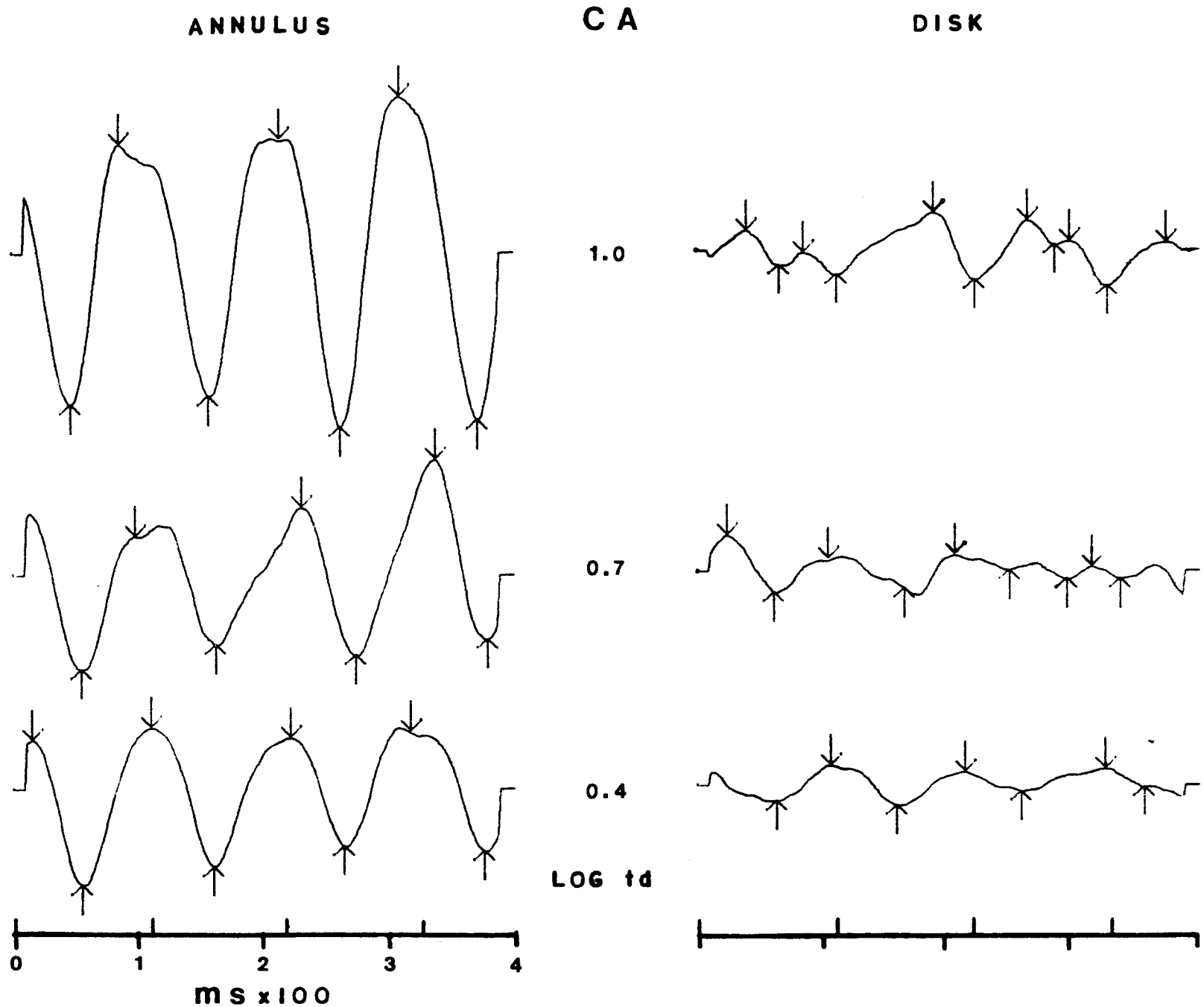

ANNULUS
CA
DISK
1.0
0.7
0.4
LOG td
0
1
2
3
4
ms x100

Figure 15. Average amplitudes of the steady-state VEP for the 3° disk and the annulus (3° and 9.6° diameters) recorded from each observer at three illuminances between the psychophysical photopic threshold and the shift in the latency function.

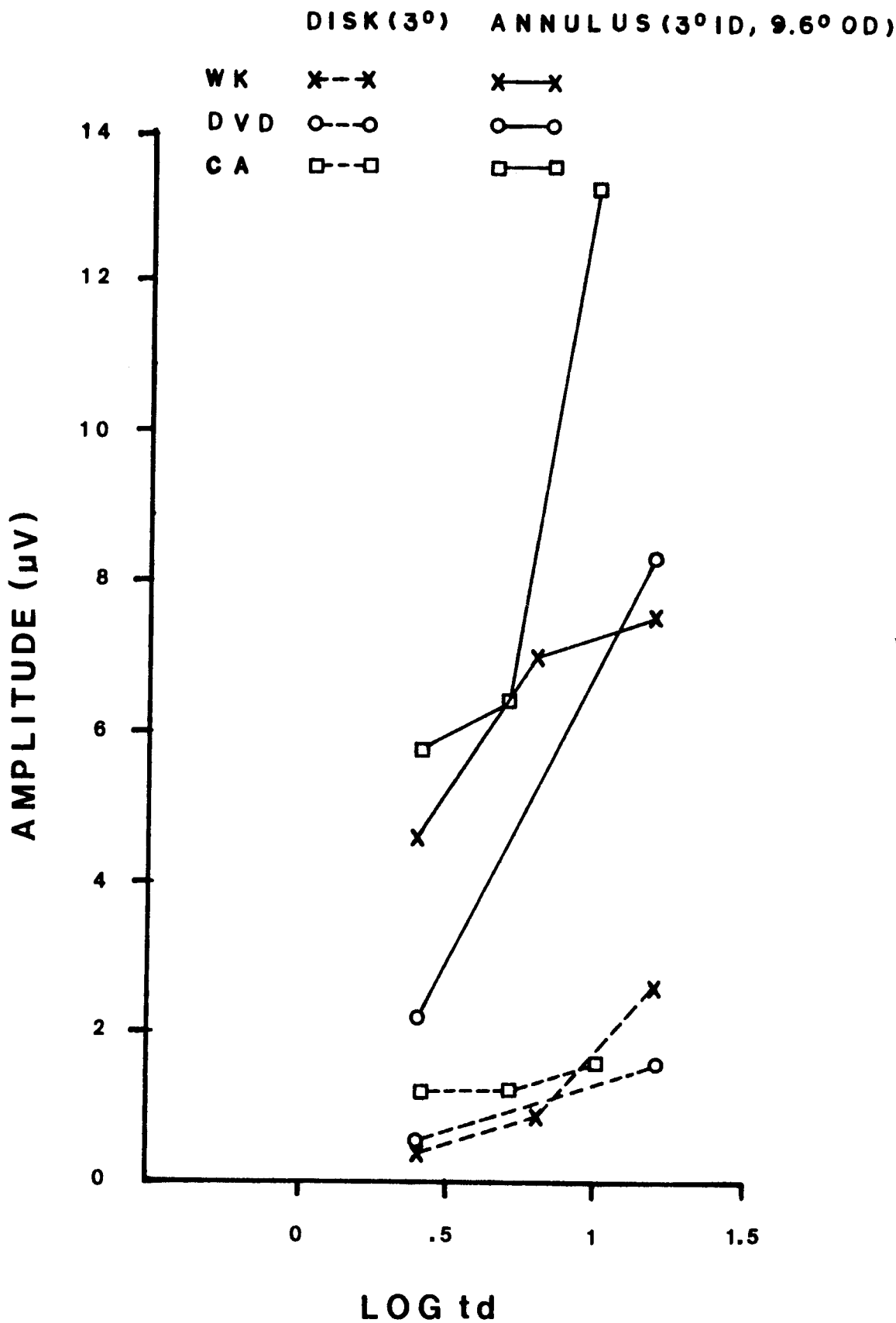
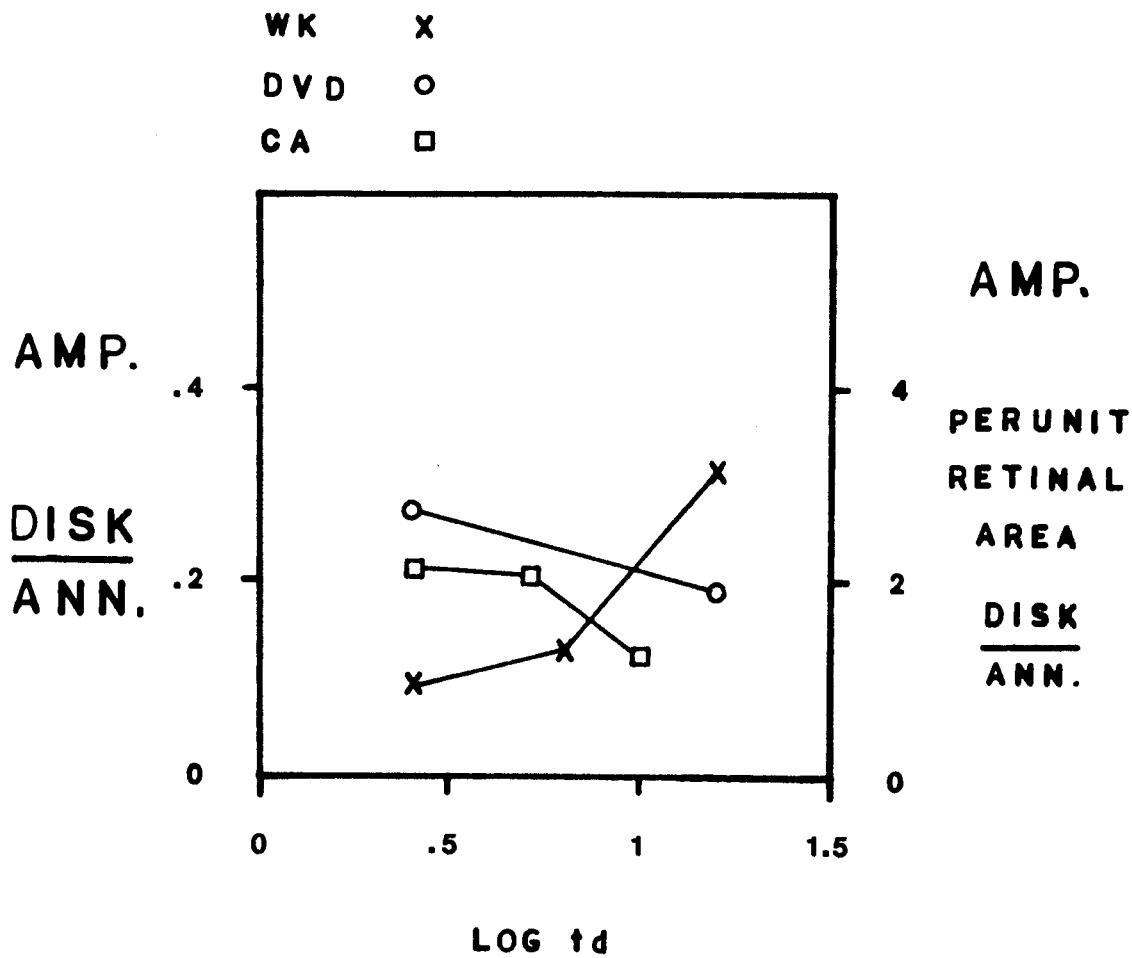

DISK (3°)
ANNULUS (3° ID, 9.6° OD)
WK
DVD
CA
14
12
10
8
6
4
2
0
AMPLITUDE (μV)
0
.5
1
1.5
LOG td

Figure 16. The ratio of the disk to annulus VEP amplitudes of each observer at three stimulus illuminances. The left ordinate shows the ratio in terms of raw amplitude and the right ordinate in terms of amplitude per unit retinal area stimulated.


WK X
DVD O
CA □
AMP.
DISK
ANN.
.4
.2
0
0
.5
1
1.5
LOG td
AMP.
PERUNIT
RETINAL
AREA
DISK
ANN.
4
2
0

D. Discussion

The duplicity theory of vision states that the scotopic system mediates vision at low luminances while the photopic system mediates vision at high levels of luminance. The present study investigated the possibility that a range of illuminances exists in which the two systems overlap. Thishypothesis was tested by measuring several variables indicative of photopic and scotopic function.

The results showed that for each observer the average latency functions of the steady-state VEPs separated into two branches, one describing a late latency function at low illuminances and the other an early latency function at higher illuminances. This is the first report of such a discontinuity in the latency/intensity function of the steady-state VEP, although a similar function has been described for the transient VEP by several investigators (Adachi-Usami, 1974; Klingaman, 1979; Van Balen and Henkes, 1960; Vaughan, 1966; Vaughan and Hull, 1965). These investigators proposed that the break in the latency function of the transient VEP was due to the transition from a scotopically to a photopically based response. This conclusion was supported with various other data collected by the investigators. Klingaman (1979) compared the latency functions of a normal and a rod monochromat and found no break in the curve of the rod monochromat. Van Balen and Henkes (1960)

found the break in the latency function was related to the bend in the dark adaptation curves, while Vaughan and Hull (1965) found that the break in the latency function occurred at a slightly lower luminance level than the psychophysically obtained photopic threshold. Therefore, the break in the steady-state VEP latency function was taken as the point at which the the photopic system contributed to the VEP. In the present experiment this level was about 1.43 log td.

The results of the photopic threshold experiment, however, showed that the photopic system first became active at an average illuminance of .22 log td. The discrepancy between the the VEP latency data and the psychophysical data was 1.2 log td (1.43 - .22 log td). was 1.2 log td. The difference may be due to the differences in sensitivity between the two measures. At lower illuminances the photopic system, although active, may not have provided sufficient input to drive the cortex to the extent that that would be required to be picked up by a scalp electrode. Such a difference in the sensitivity between the psychophysical and the electrophysiological indices of photopic function at threshold has been noted to be between .8 and 2 log units in increment threshold experiments (Adachi-Usami, 1974; Klingaman, 1979). However, at least part of this difference may be methodological rather than physiological. Klingaman (1979) found that a closer agreement between the two measures was obtained when the effects of light adaptation were minimized by accumulating many VEPs of small sample size, rather than

collecting a large sample VEP which would increasingly light adapt the eye over time. He attributed the closer agreement of his VEP and psychophysical functions compared to the data of Adachi-Usami (1974) to this technique and speculated that even smaller sample sizes would result in further agreement between the two functions. Stimulation of the 3° disk in the present experiment produced a small but recordable following response suggesting that the photopic system was indeed active at these low illuminances,although it was not strong enough to show up in the latency function which was dominated by the scotopic VEP response to the 9.6° field.

In contrast to the discrepancy between the psychophysical and VEP estimates found here, Vaughan and Hull (1965) reported that their measure of the photopic threshold was at a luminance level slightly higher than the rod-cone break. The reason for the difference between the two studies is difficult to ascertain, since Vaughan and Hull reported few details of their measurement of the photopic threshold. They used a 1.5° foveal target illuminated by retangular flashes of 10 ms duration which were delivered every 750 ms. The investigators found a correspondence between the disappearance of the foveal VEP and the report of the same by the subjects. One of the differences between the two studies was the rate of stimulus delivery, 750 vs. 110 ms. It is possible that the photopic threshold for stimuli delivered at a faster rate might be lower. In addition, the present experiment used a forced-choice method which

eliminated response bias. It is possible that the subjects of Vaughan and Hull's experiment, if response bias was not controlled, overestimated the threshold for photopic vision.

On the other hand it may be that the photopic system, though active, was suppressed or masked by the larger scotopic response when the two receptor systems were jointly stimulated at mesopic luminances. However, this intrepretation is in apparent conflict with those of Gouras (1965, 1967) and Gouras and Link (1966) who studied the response latency of perifoveal ganglion cells in the monkey retina that received input from both rods and cones. They reported that, once cone threshold was reached, the pattern of firing of the ganglion cell was entirely determined by the faster cone signals and that the rod signals were suppressed.

There are two possible mechanisms that can be invoked to resolve this apparent conflict. First, it is important to note that either rod or cone signals may be suppressed depending on which signal follows in the refractory wake of the other (Gouras & Link, 1966). Thus, when rods and cones are simultaneously stimulated the rod signal always arrives after the cone signal, due to the slower responsiveness of the rods (Gouras, 1967). However, in accordance with a model proposed by Walters (1971), if the rate of stimulation were such that the succeeding cone signal arrived at the ganglion cell within the refractory period generated by the preceding rod signal, then the rod signals would be transmitted instead of the cone signal.

Gouras (1965, 1967) determined the difference in response latency of the monkey ERG b-wave from incoming rod and cone signals. He estimated that the cone signals preceded rod signals by about 100 ms for stimulus energies capable of stimulating rods and cones simultaneously. A similar estimate of 100 ms was found for the difference in arrival times of rod and cone signals at the perifoveal retinal ganglion cell of the monkey (Gouras and Link, 1966). Frumkes et al. (1973) found differences in response latencies between rods and cones to be between 30-75 ms. Thus, repetitive stimulation of the appropriate frequency could result in cone signals following rod signals. In the present experiment in which an ISI of 110 ms was used, a lag time of 55 - 110 ms would be necessary in order for the interval between the rod signal and the succeeding cone signal to be shorter than the interval between the rod signal and the preceeding cone signal. The lag times reported by Gouras (1967) and Frumkes et al. (1973) of 110, and 30-75 ms, respectively, correspond to such an interval. Therefore, given an ISI of 110 ms, it is more likely that rod signals alone were transmitted in the present experiment, rather than cones. This might explain the lack of a short latency branch just above the photopic threshold. The site of such an interaction would likely be the inner plexiform layer of the retina where signals from separate rod and cone channels converge on ganglion cells, as proposed by Gouras (1966).

This mechanism does not, however, explain why cone signals from the fovea did not appear in the VEP record. Since foveal cones do not share their connections to the ganglion cells with rods (Polyak, 1941), there would not seem to be an cpportunity for an interaction with the rod system. Thus, it appears that no one explanation adequately accounts for the lack of evidence of photopic activity in the steady-state VEP at illuminances immediately above the photopic threshold.

The critical importance of flicker rate on rod-cone interaction has been noted in other studies (MacLeod, 1972; van den Berg and Spekreijse, 1977; Von Grunau, 1976; Walters, 1971). Rather than antagonistic interactions some of these studies (MacLeod, 1972; van den Berg and Spekreijse, 1977) have suggested that summation of rod and cone signals can occur. Thus, stimulation rate appears to be a critical factor in the antagonistic and additive interactions between rods and cones.

Thus there are several lines of evidence to suggest that the scotopic system continued to transmit signals to the brain at photopic illuminance levels. The presence of a long latency response up to 1.2 log td into the photopic range suggested a scotopically mediated response. The amplitude of a peak which reached a maximum within this range as well as the larger amplitude response from the parafoveal region of the retina implicated the scotopic system as the source of the steady-state VEP at illuminance levels below 1.4 log td.

The primarily photopic nature of the VEP has been stressed in many research papers (Armington, 1966; DeVoe et al. 1968; Rietveld et al. 1965; Wooten, 1972). In studies in which characterizable evoked responses have been obtained in the periphery (e.g. Eason, Oden, and White, 1967; Eason and White, 1967; Ohba, 1967; Osaka and Yamamoto, 1978) it is not clear that the photopic system did not also contribute to the response either by stimulation of peripheral cones or by stimulation of foveal cones through scattered light. The apparently 'pure' scotopic steady-state response under the stimulus conditions reported here may make the steady-state VEP an effective tool in the clinical study of the scotopic system at the cortical level, where such measures are in need of development (Adams et al. 1969).

Another explanation for the presence of scotopic activity at photopic levels does not assume a rod-cone interaction, but rather is contingent on the differences in the relative sensitivities of the foveal and parafoveal cones. Sperling and Hsia (1957) found that the foveal cones were relatively more sensitive in the red end of the spectrum and relatively less sensitive in the blue end of the spectrum than cones located 10° in the periphery. If the foveal cones were more sensitive to white light then it is possible that low luminances capable of activating foveal cones would not be strong enough to activate peripheral cones. Thus, the only signals to be transmitted from the peripheral retina would be from rods, whole cond signals

would be transmitted from the fovea. Both rod and cone signals could then be sent to the brain via independent channels avioding any antagonistic interaction due to convergence at the retinal ganglion cell. If this situation prevailed then one would expect to find evidence of both scotopic and photopic activity in the range of luminances where the two systems were thought to overlap.

Although no evidence of joint photopic and scotopic activity was found below the break in the latency function, it remains possible that joint activity was present above the latency break. It has been reported that the scotopic system does not saturate until about 3.0 log td (Aguilar and Stiles, 1954; Klingaman, 1979; Sakitt, 1976), which is about 1.4 log units above the latency break in the present experiment, so it is reasonable to pursue the question of scotopic activity at these levels. Other studies have claimed that the VEP is entirely photopic once the photopic threshold is exceeded (Vaughan and Hull, 1965; Wooten, 1972). The results of the present experiment give some support to the contention that the VEP is entirely photopic once the break in the latency function is crossed. As shown in Figures 11, 12, and 13 the positive amplitude of the scotopic VEP declines as the shift in the latency curve is approached and it reaches a minimum at the point where early latencies are first recorded. A similar decline in the VEP amplitude of a rod monochromat has been reported by Klingaman (1979) and suggests the onset of

saturation of the rod system. The rebound in the amplitude of this peak seen in all three subjects between 1.4 and 2.5 log td is probably due to a photopic contribution, since at this point the latency of this peak was early.

The latency functions also indicated that for two of the three observers the changeover from rod to cone function was complete. Observers WK and CA showed a complete shift from long to short latency responses. DVD, on the other hand, showed evidence of simultaneous long and short latency peaks at 1.4 log td. Studies that have investigated both scotopic and photopic VEP latency curves have generally found that scotopic latencies did not fall below 200 ms, nor did photopic latencies go very much higher than 200 ms (Klingaman, 1979; Vaughan, 1966; Wooten, 1972). Klingaman (1979) also reported that the latency function of a rod monochromat did not go below 210 ms even at high stimulus intensities. The latency functions of WK and CA also separated at the 200 ms mark, indicating a complete separation of rod and cone activity. On the contrary, DVD showed response latencies overlapping the 200 ms dividing line at a single stimulus intensity, suggesting joint photopic and scotopic activity. The absence of scotopic activity at higher stimulus intensities appeared to be the case for some, but not all observers. Thus, the double component VEP, found in the intensity range just above the break in the latency curve, seemed to represent either an entirely photopic response or individual photopic and scotopic components. These results lend

partial support the to report of Diamond et al. (1981) of separate photopic and scotopic VEP components in the photopic range. Further research is needed to substantiate this interpretation, however.

It has been implicit in this discussion that long latency responses at low illuminances indicated scotopic activity rather than the result of photopic activity. Although the amplitude of the foveal VEP was only about 16% of the annulus VEP amplitude, this might be expected due to the small size of the retinal area stimulated. When receptor density is considered the contribution of the fovea to the full field response per unit retinal area was 1.8 times that of the parafovea. The remaining proportion of the response may have been generated by peripheral cones, not rods. However, it is unlikely that parafoveal cones could be responsible for the large amplitude of the annulus VEP. First, despite the ten-fold increase in the retinal area stimulated by the annulus, the density of the parafoveal cones is far less than foveal cones (Østerberg, 1935) and therefore, the actual number of cones in the parafovea was estimated to be only 2.5 times that in the central 3° of the retina. It is unlikely that the parafoveal cones could account for a response 6 times larger than the foveal response. Second, other studies have found a much higher per unit retinal area contribution of the fovea than that reported here. Rietveld et al. (1965) asserted that over 99% of the amplitude of a transient VEP recorded in response to stimulation of a 15° 16' diameter disk was due to the central 2°

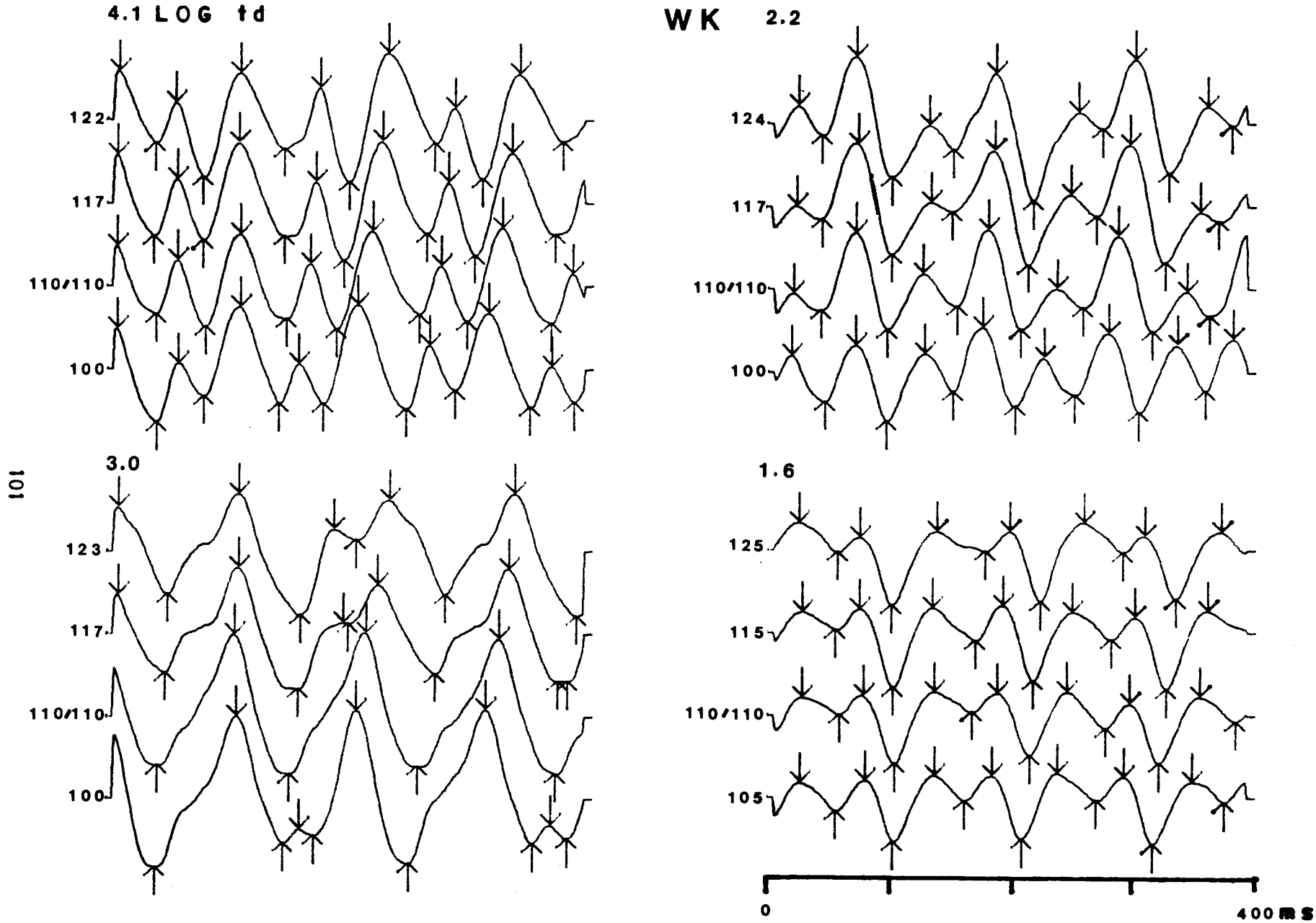
of the fovea. For pattern stimulation, Rietveld et al. (1967; Rietveld, 1966) estimated that 70% of the response was due to the central 3° of retina. Regan (1966), using stimulus parameters similar to those of the present experiment (a 2.2° foveal field and an annulus with dimensions of 4° and 11°) found that the amplitude per unit retinal area of a steady state VEP was about 6.5 times greater for foveal compared to extrafoveal stimulation for blue stimuli of mean retinal illumination between 3.0 and 4.0 log td.

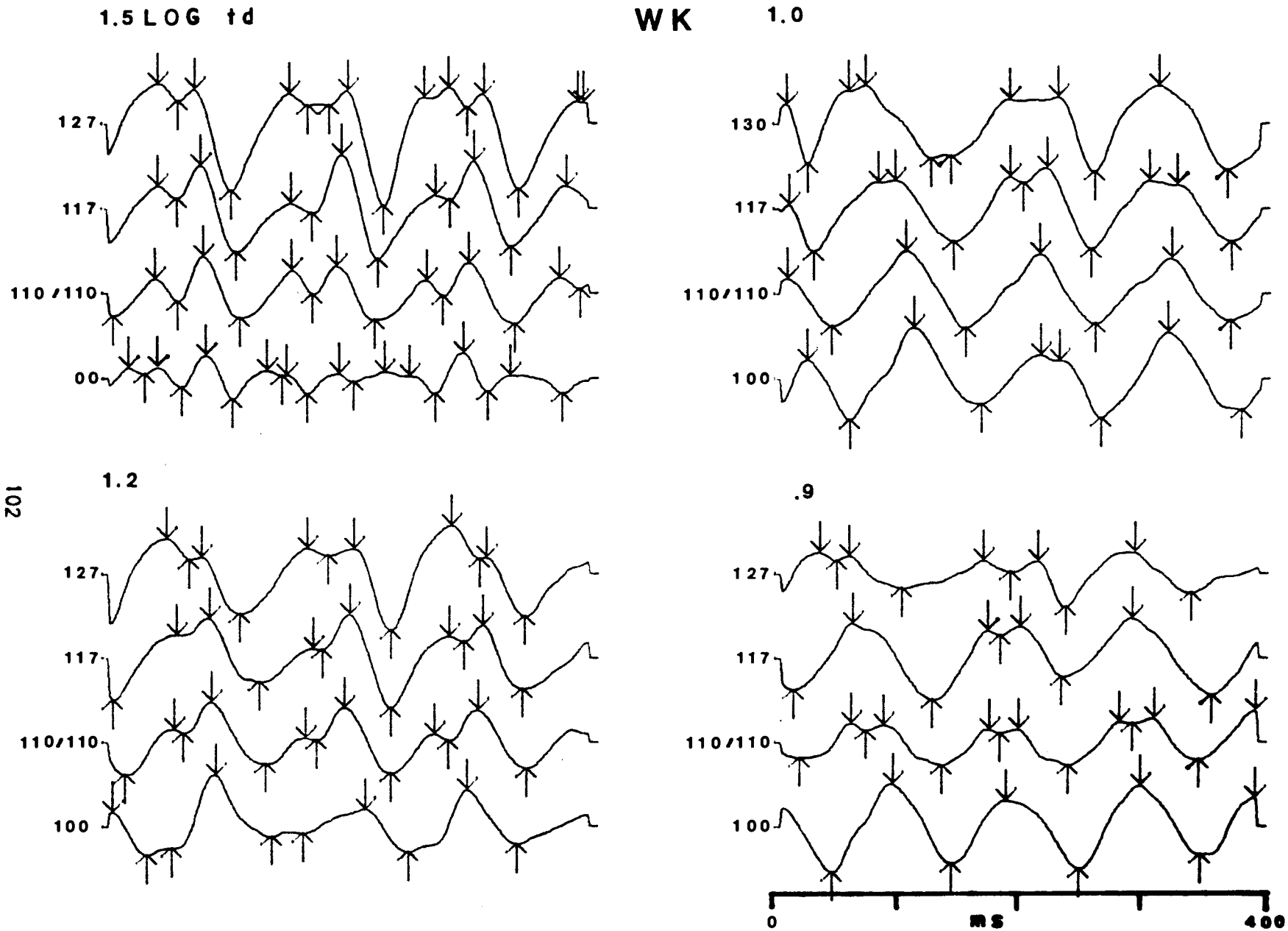
The factors that might account for the different unit area estimates of the two studies (i.e. Regan's and the present one) are the stimulus colors, white vs. blue, and the stimulus intensity, .4-1.2 log td vs. 3.0-4.0 log td. Since Sperling and Hsia (1957) and Weale (1951) have shown that the parafoveal area is relatively more sensitive to blue light than the fovea, stimulus color would not account for the relatively smaller contribution of the parafovea in Regan's study. On the other hand, lower stimulus intensity would account for a relatively greater contribution of the parafovea in the present study through stimulation of the rods. The results of Eason et al. (1967) who reported a relatively greater contribution of the peripheral retina to the VEP amplitude when low intensity blue light was used as opposed to high intensity red light, also support such an interpretation. These findings further supported the hypothesis that the long latency full field responses between .4 and 1.2 log td were due to stimulation of the rods.

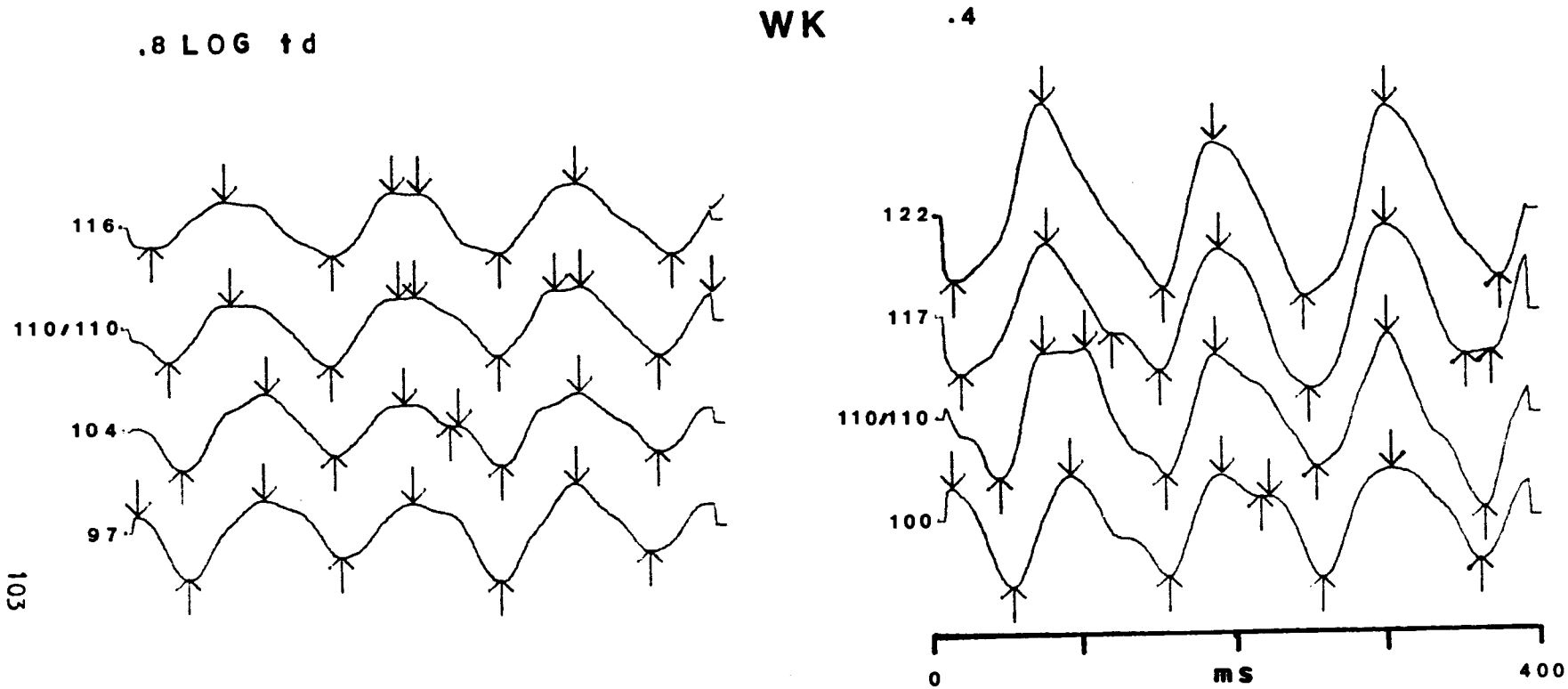
In summary, the evidence obtained from latency and amplitude measurements of the steady-state VEP obtained, (a) over a range of retinal illumination of 5.75 log td, (b) from selective stimulation of retinal locations reflecting primarily rod or cone activity, and (c) from the measurement of the photopic threshold suggested that the scotopic system is capable of sending signals to the brain at photopic levels. The photopic system, although shown to produce a small but recordable VEP at levels immediately above the photopic threshold, appeared to be suppressed or masked by the scotopic system when both were simultaneously stimulated. Finally, there was some evidence for joint photopic and scotopic activity in the mesopic range near the break in the latency function, however, this seemed to be subject to individual differences, with other observers showing exclusively photopic activity at these levels.

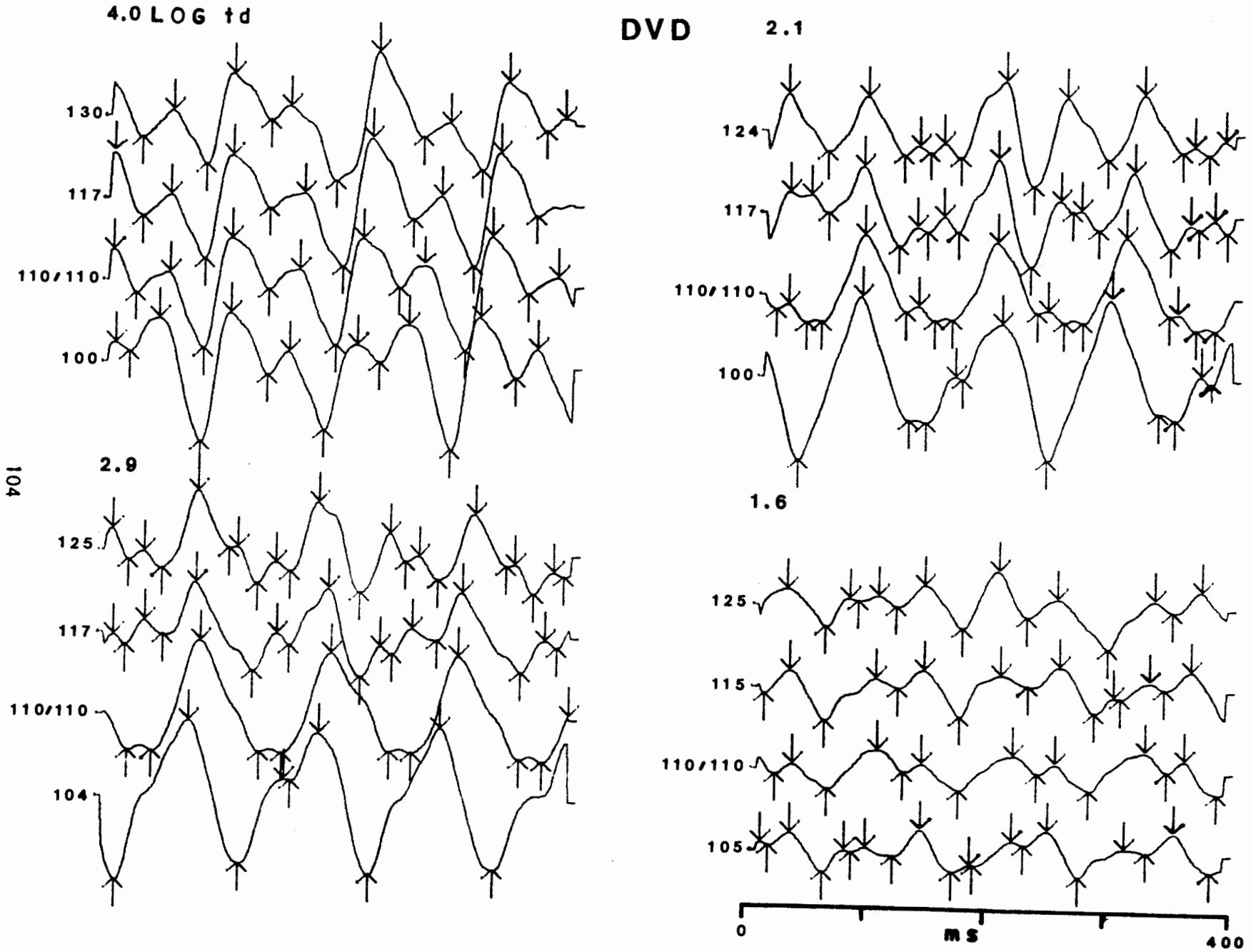
E. Appendix A

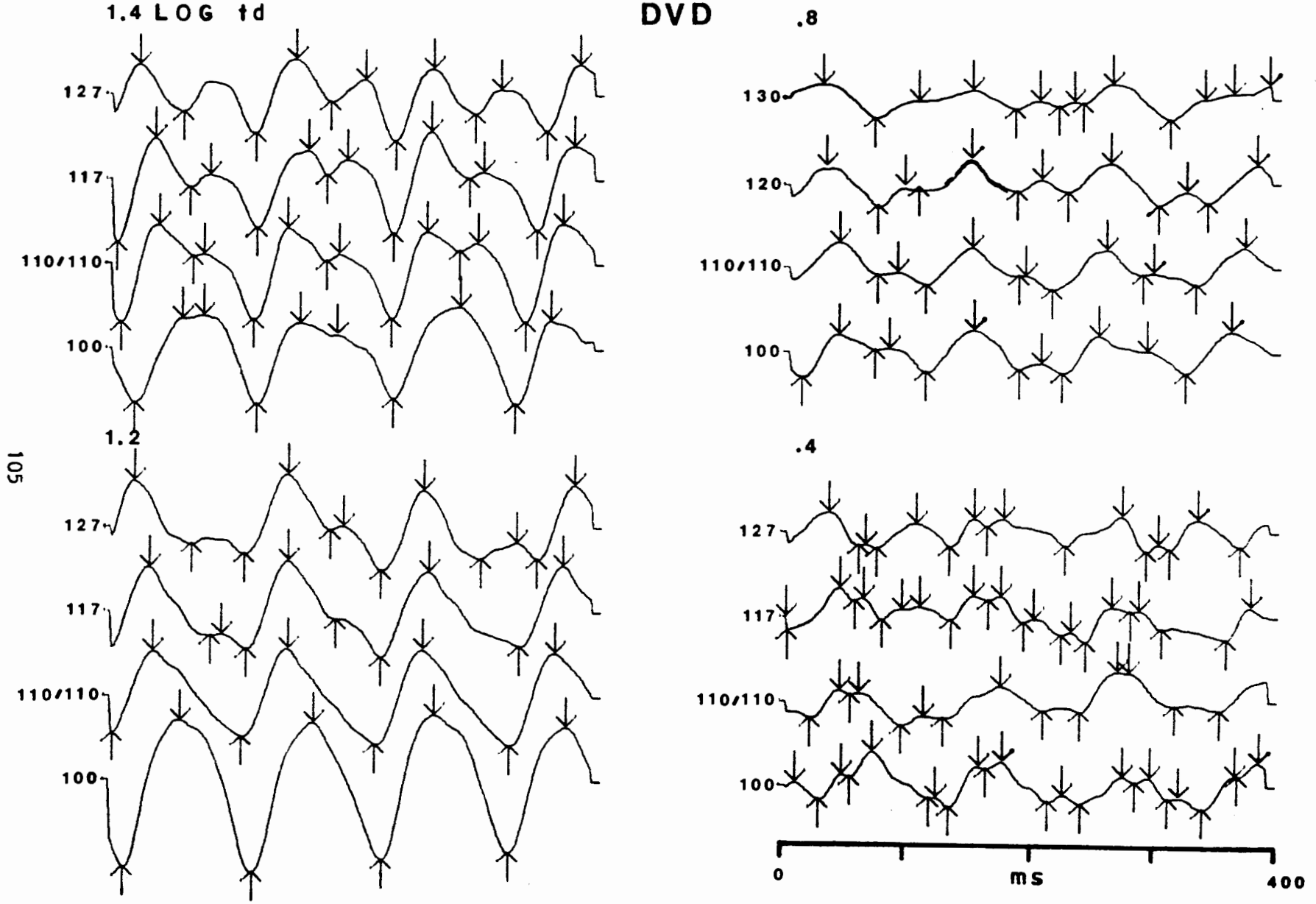
Sets of VEPs recorded to synchronous and asynchronous stimulation from which latencies were calculated. The upper left corner of each set of VEPs shows the stimulus intensity at which the responses were recorded. The ISI is shown to the left of each waveform. For the asynchronous ISIs only the variable interval is noted. The arrows show the peaks detected bythe algorithm, Peakfind2, from which asynchronous VEP peaks that were associated with peaks of the synchronous VEPs were determined.

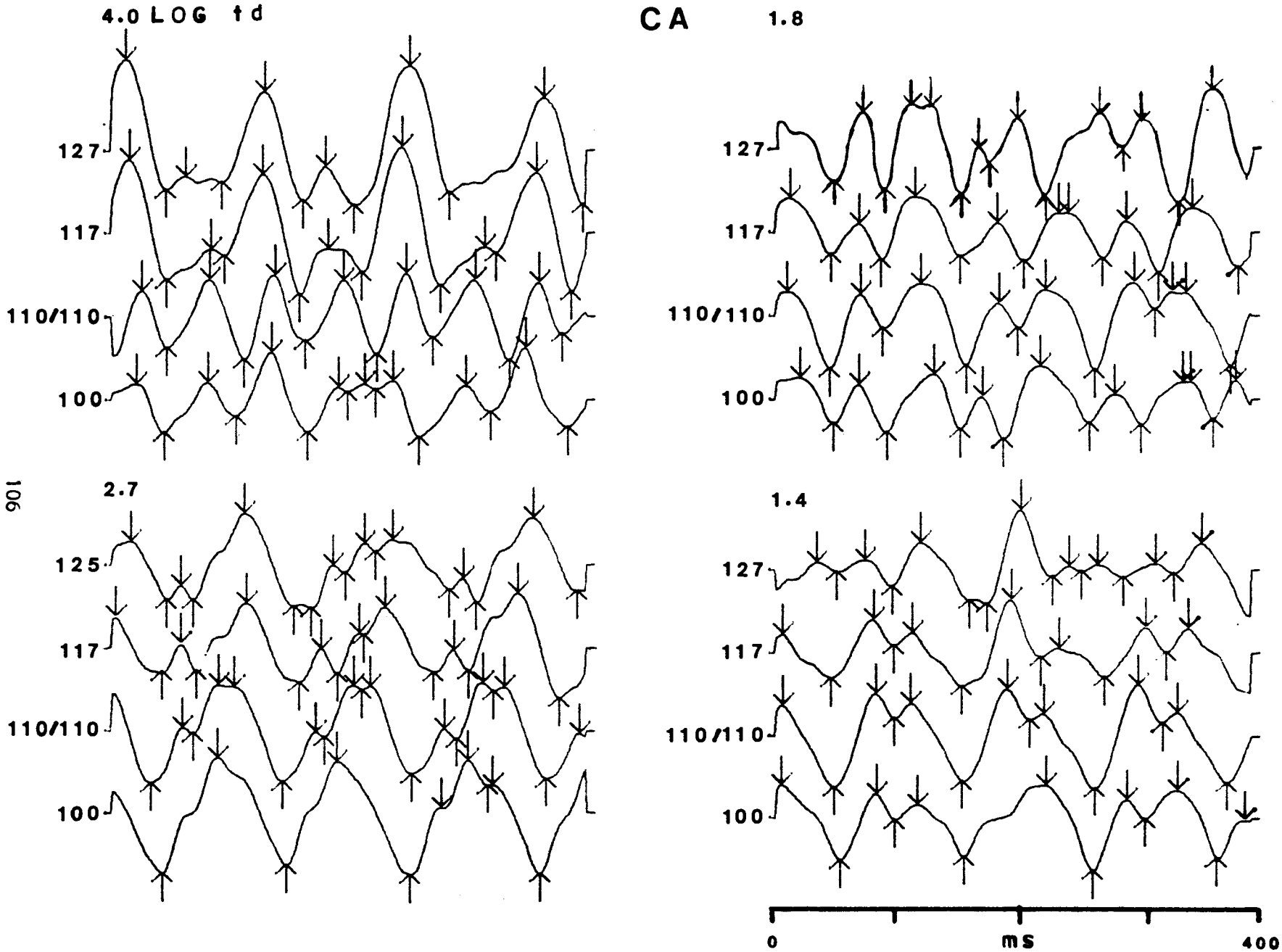

4.1 LOG fd
122
117
110/110
100
WK 2.2
124
117
110/110
100
3.0
123
117
110/110
100
101
1.6
125
115
110/110
105
0
400 ms

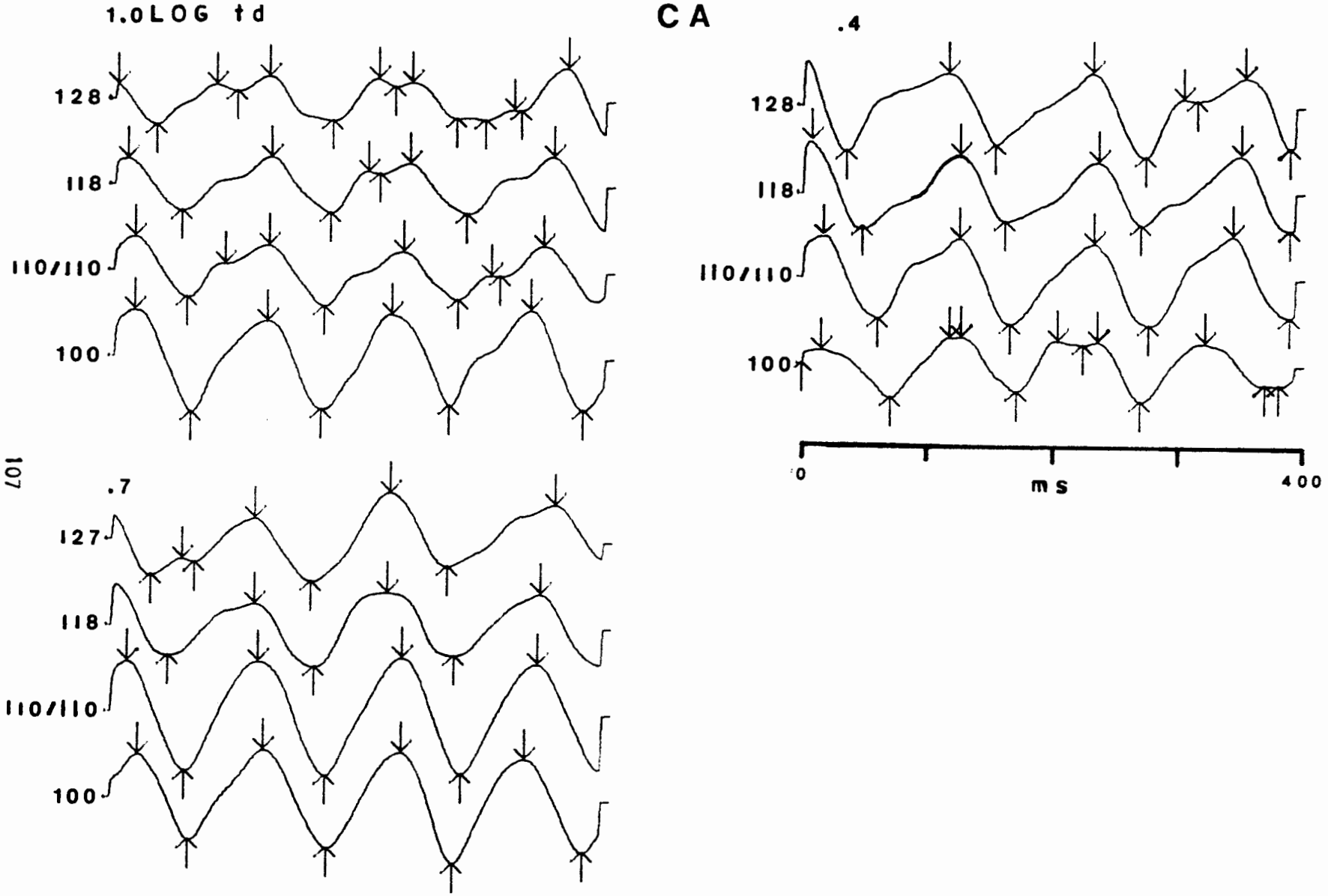

1.5 LOG td
WK
1.0
127
117
110/110
00
130
117
110/110
100
1.2
.9
127
117
110/110
100
127
117
110/110
100
0
ms
400


.8 LOG td
116
110/110
104
97
103
WK
.4
122
117
110/110
100
0
ms
400


4.0 LOG td
130
117
110/110
100
2.9
125
117
110/110
104
104
DVD
2.1
124
117
110/110
100
1.6
125
115
110/110
105
0
ms
400


1.4 LOG td
127
117
110/110
100
1.2
127
117
110/110
100
105
DVD
.8
130
120
110/110
100
.4
127
117
110/110
100
0
ms
400


4.0 LOG td
127
117
110/110
100
2.7
125
117
110/110
100
106
CA
1.8
127
117
110/110
100
1.4
127
117
110/110
100
0
ms
400


1.0LOG td
128
118
110/110
100
.7
127
118
110/110
100
107
CA
.4
128
118
110/110
100
0
ms
400

Appendix B

The figure on the following page describes the method for determining the amplitude and the time of a turning point y*, a weighted average of the two points y_1 and y_2. Four turning points are shown in the figure: y_0, y_1, y_2, and y_3 at times t_0, t_1, t_2, and t_3. The amplitudes of y_1 and y_2, a_1 and a_2, respectively, were measured from a line, L, drawn through y_0 and y_3 which has a slope, S, where

$$S = \frac{y_3 - y_0}{t_3 - t_0} .$$

The amplitudes of a_1 and a_2 are defined as

$$a_1 = y_1 - b_1 \qquad \text{and} \qquad a_2 = y_2 - b_2$$

where b_1 and b_2 are the Y-intercept values on line, L, at times t_1 and t_2 or,

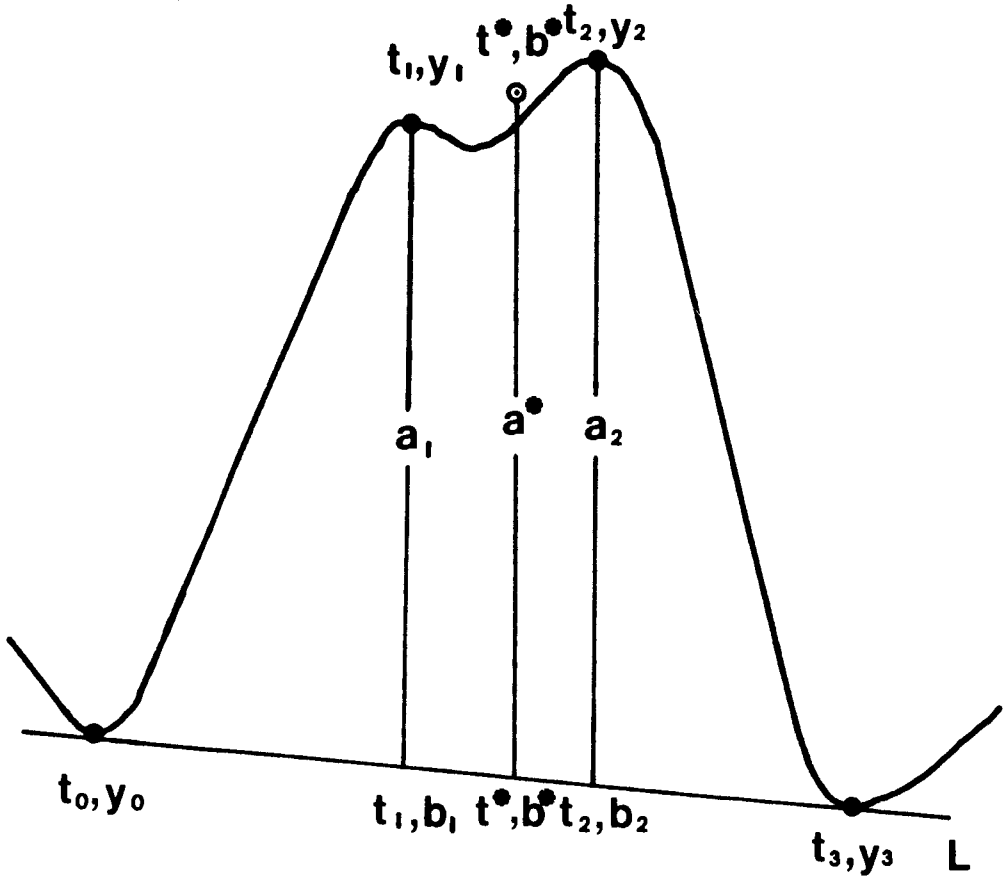
$$b_1 = y_0 + S(t - t_0) \quad \text{and} \quad b_2 = y_0 + S(t_2 - t_0).$$

Thus, the time of occurrence of y* is

$$t^* = \frac{a_1 t_1 + a_2 t_2}{a_1 + a_2}$$

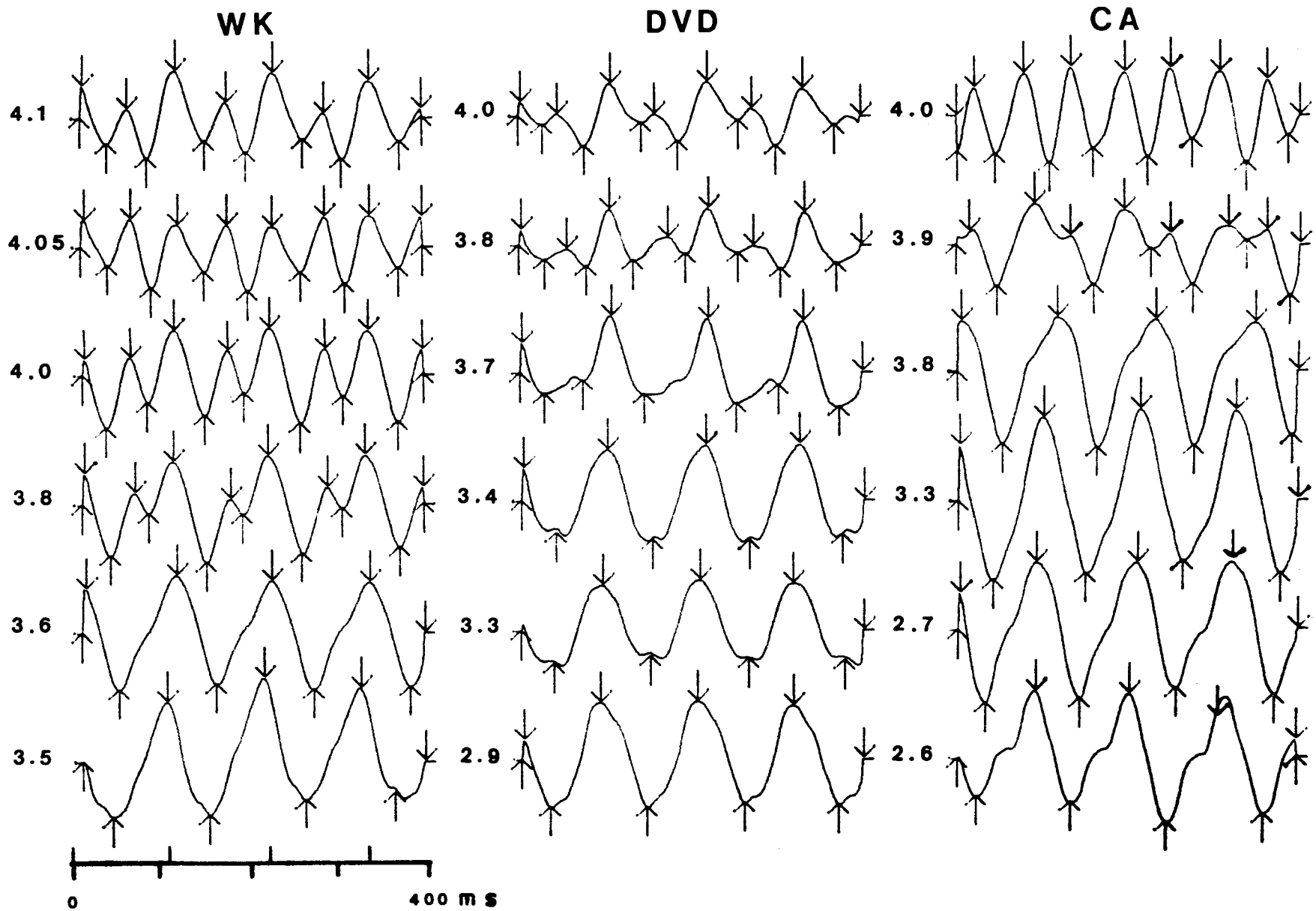
and the amplitude of y* is

$$a^* = \frac{a_1 + a_2}{a_1 + a_2} .$$

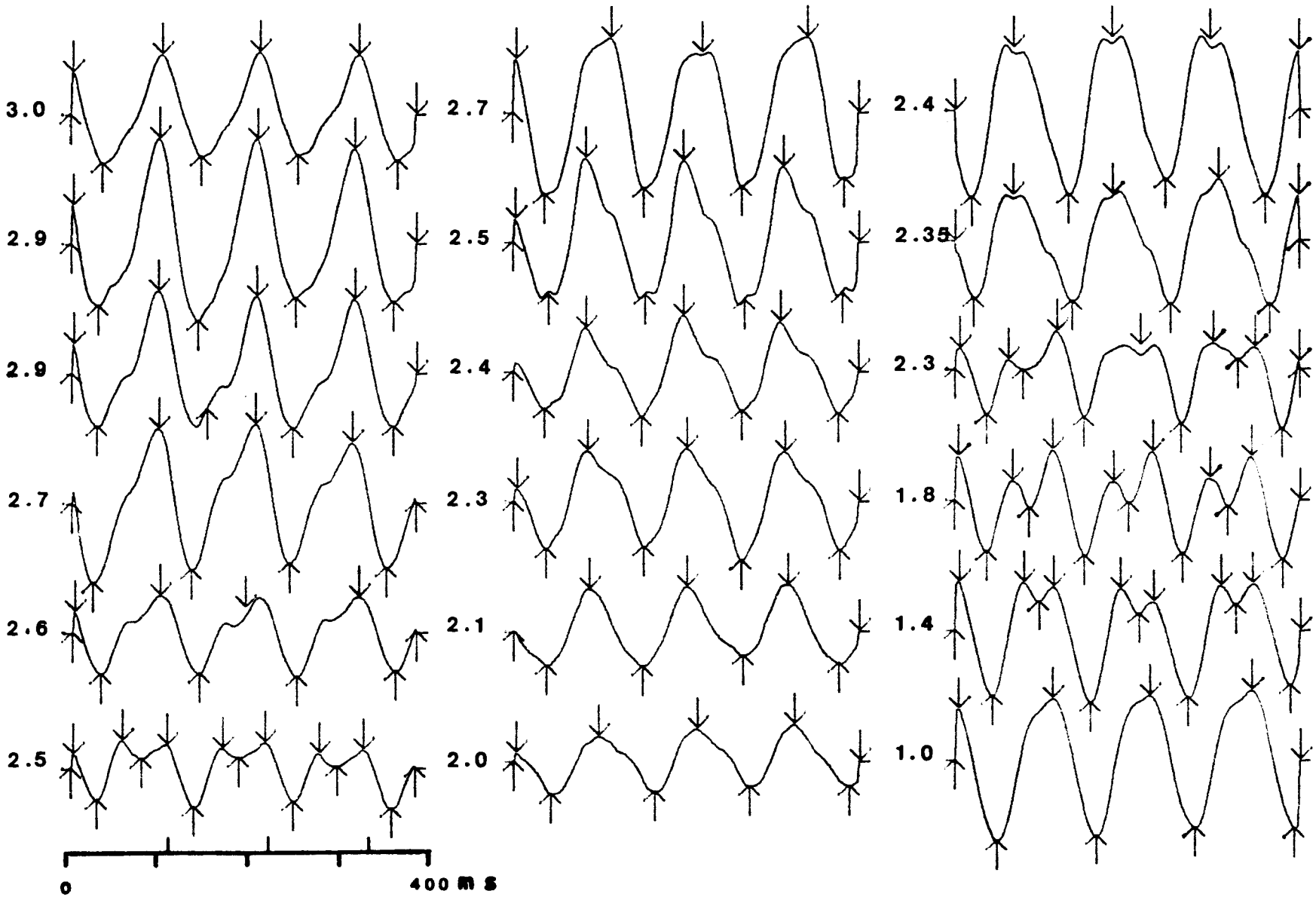

t₁,y₁
t*,b*
t₂,y₂
a₁
a*
a₂
t₀,y₀
t₁,b₁
t*,b*
t₂,b₂
t₃,y₃
L

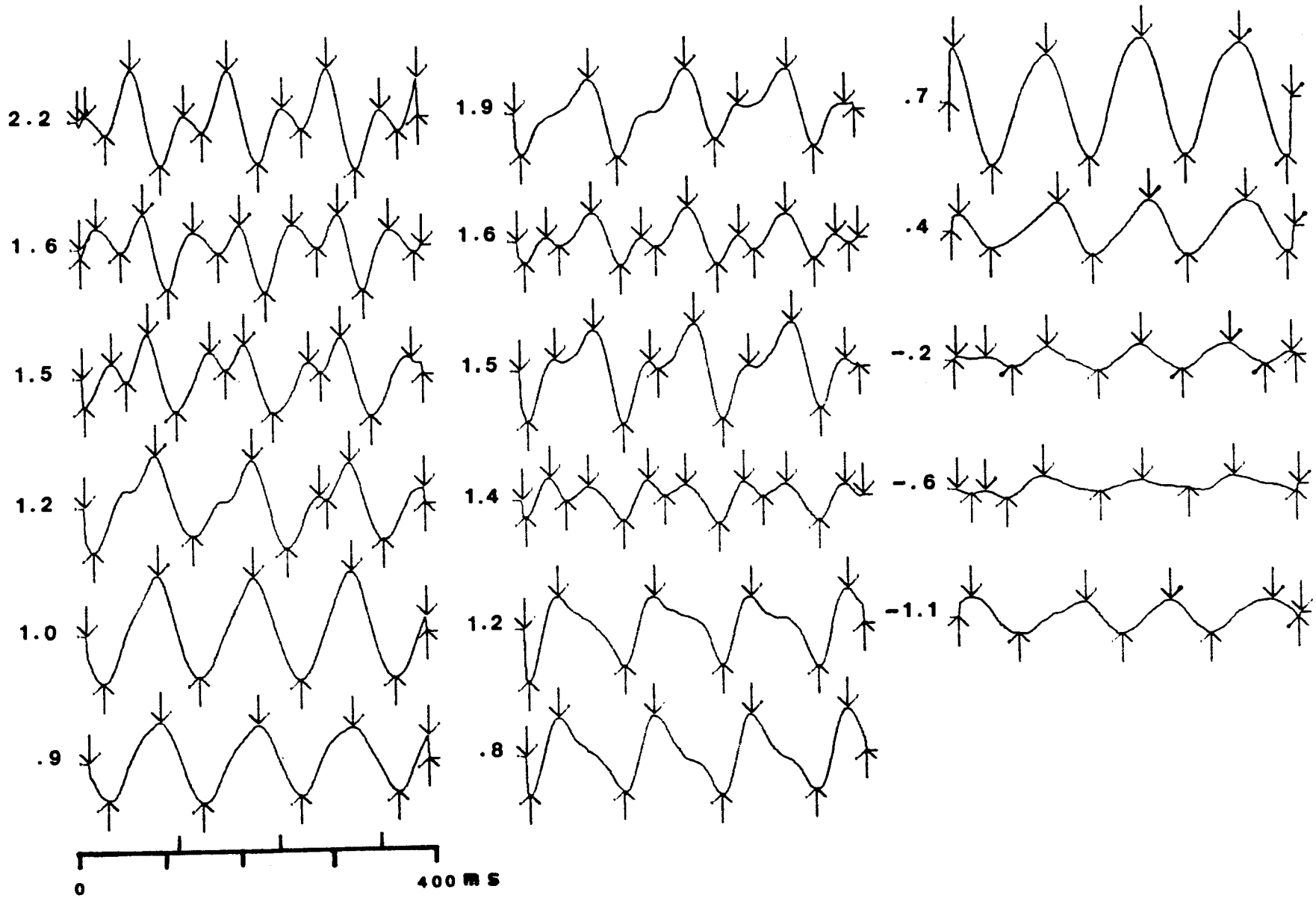
G. Appendix C

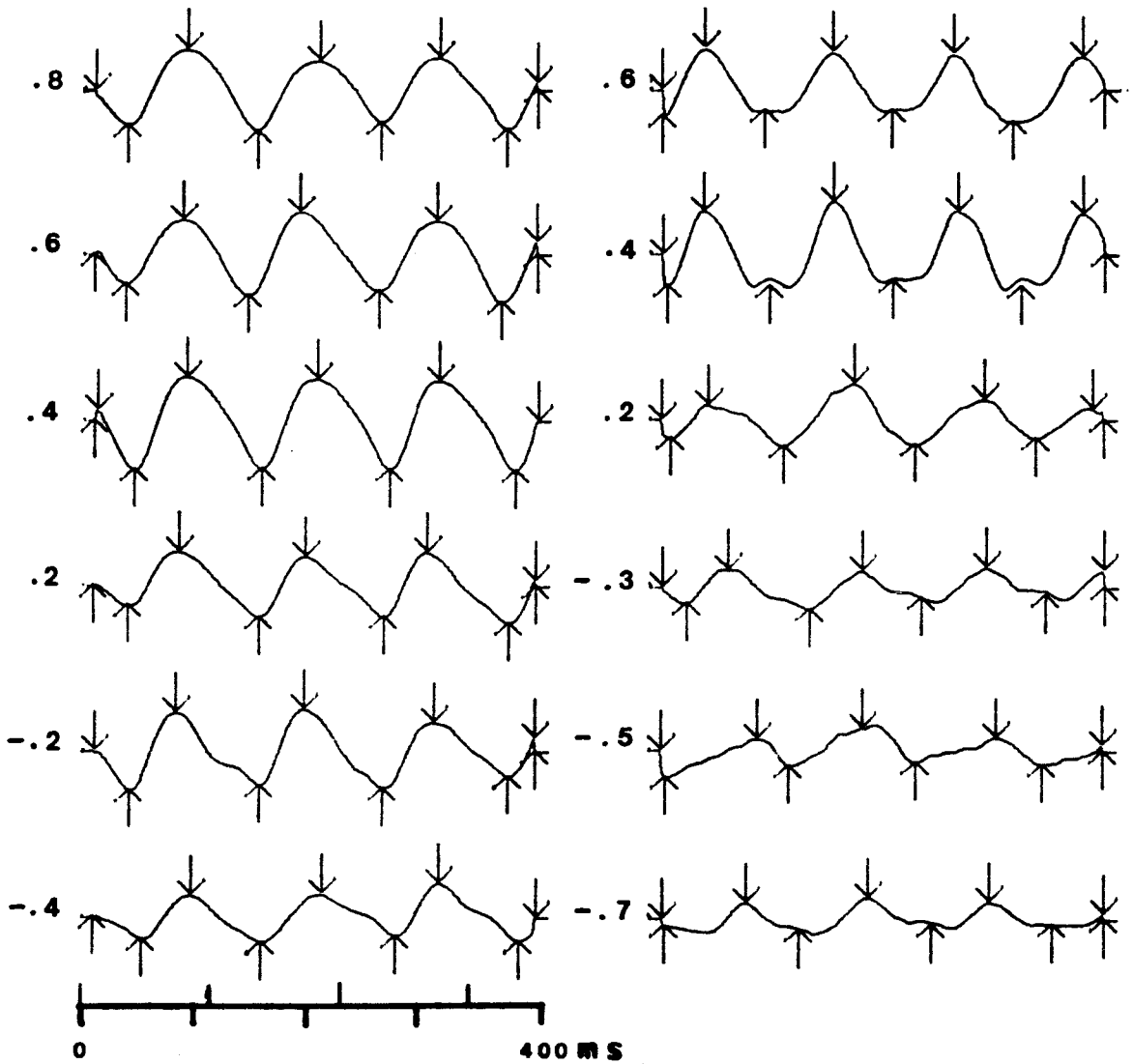
The peaks found by the algorithm, Peakfind1, are shown for all VEPs recorded to synchronous stimulation for all observers. Stimulus illuminance is shown to the left of each waveform and was recorded in log trolands. Relative amplitude is arbitrary. The time base is shown at the bottom of the page with marks showing the location of each stimulus pulse. Each response consisted of appproximately 3 cycles with one or two negative and positive peaks per cycle.

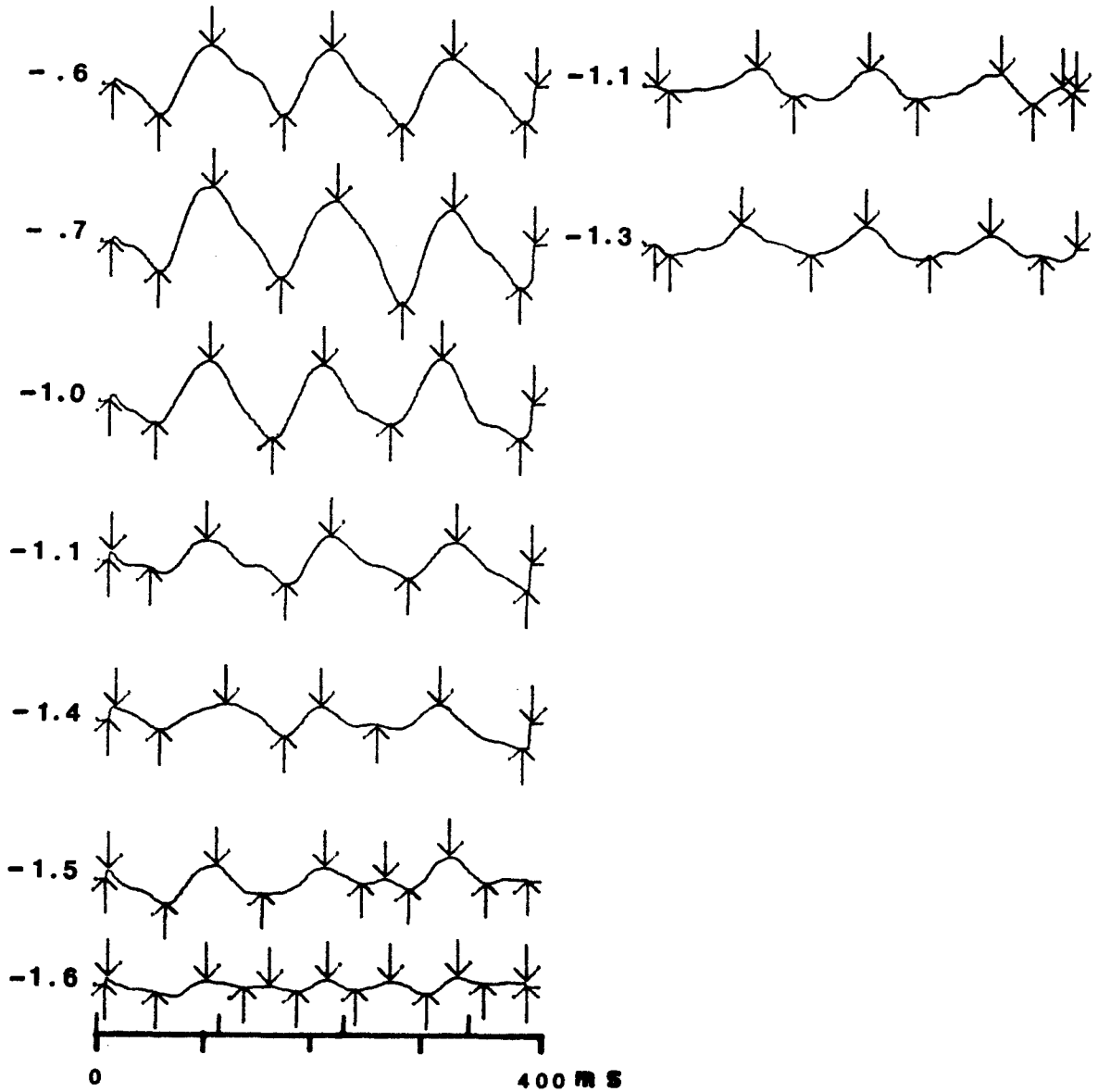

WK
DVD
CA
4.1
4.05
4.0
3.8
3.6
3.5
4.0
3.8
3.7
3.4
3.3
2.9
4.0
3.9
3.8
3.3
2.7
2.6
0
400 ms

111


3.0
2.9
2.9
2.7
2.6
2.5
2.7
2.5
2.4
2.3
2.1
2.0
2.4
2.35
2.3
1.8
1.4
1.0
0
400 m s


2.2
1.6
1.5
1.2
1.0
.9
1.9
1.6
1.5
1.4
1.2
.8
.7
.4
-.2
-.6
-1.1
0
400 ms


.8
.6
.6
.4
.4
.2
.2
-.3
-.2
-.5
-.4
-.7
0
400 MS


-.6
-.7
-1.0
-1.1
-1.4
-1.5
-1.6
-1.1
-1.3
0
400 MS

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