LIGHT ADAPTATION
IN THE HUMAN STEADY-STATE VISUAL EVOKED RESPONSE

BY

LYN RICHARDS WOOL

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APPROVAL

Name: Marilyn A. Wool
Degree: Master of Arts
Title of Thesis: Light Adaptation in the Human Steady-State Visual Evoked Response

Examining Committee:

Chairman: V. Modigliani

A. L. Diamond
Senior Supervisor

B. L. Beyerstein

A. H. Burr
External Examiner
Assistant Professor
Simon Fraser University

Date Approved: 11 May 11, 1978

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ABSTRACT

Light adaptation is the decline in visual sensitivity which occurs following any increase in ambient luminance levels. The effects of light adaptation on the steady-state visual evoked response, that is the cerebral response to repetitive visual stimulation, have not yet been investigated. The present study used a new measure of the latency of the steady-state visual evoked response to follow the course of light adaptation.

Two experiments were conducted on the effects of light adaptation on the visual evoked response. In Experiment I, responses from 10 second periods were averaged together. In the first phase of this experiment, the first four of these 10 second periods were studied in seven subjects; in the second part of the experiment, latency changes over 6 minutes of light adaptation were observed in four subjects. Results indicated an increase in evoked response latency during the first 30 to 40 seconds of light adaptation, followed by a slight latency decrease. The poor quality of the data obtained in this experiment was interpreted as indicating that the sample period from which responses were averaged was too long, and should be decreased.

Experiment II used shorter sample periods. In the first phase of this experiment, responses from 3-second periods were averaged together, and a marked latency decrease was observed during the course of the first 15 seconds of light adaptation for

three of five subjects. The improved quality of this data supported the hypothesis that recording periods shorter than those used in Experiment I were appropriate for this study. In the second part of Experiment II, very short recording periods were studied with two subjects. The results of this study indicated that the quality of the data could still be improved by further decreasing the sample period.

Following the above results, a technique for collecting data on light adaptation in the steady-state visual evoked response is described which should obviate most of the problems encountered in this study.

This thesis is dedicated to the people who gave me so much, and without whose support it could not have been completed: my son, Ezekiel; my mother, Anne; and my friends, Mike and Kie.

I am truly grateful to Len Diamond, who gave me the opportunity to work in his lab, encouraged me to continue doing so, and offered most generous assistance throughout this work.

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SECTION I: INTRODUCTION.

Light adaptation is a fundamental visual process of considerable intrinsic interest. Study of this process using visual evoked potentials in human subjects permits comparison of brain responses in light adaptation with psychophysical adaptation data. An important outcome of such comparison will be the bridging of the present gap between psychophysical and physiological lines of research into light adaptation. Study of adaptation in the visual evoked response is also of methodological interest for the use of averaged responses.

Each of these three aspects of adaptation in the visual evoked response will be discussed in some detail below, since the intrinsic importance of light adaptation, the implications of a visual evoked response study for our full understanding of the process, and the methodological significance of any visual adaptation study for the use of averaged evoked response measures constitute the basis for the experimentation to be reported here.

The term 'light adaptation' refers to a decline in sensitivity to a visual stimulus, which follows any increase in the intensity of that stimulus. This process has been studied with a wide variety of measures. Changes in visual sensitivity have been related to alterations in the concentration of unbleached visual pigment in the retina (Hecht, 1937; Wald, 1954), and intracellular and field potential recordings document

the electrical activity in retina and visual cortex which is associated with light adaptation (Granit, 1963; Dowling, 1967; Armington, 1964). Brightness matching and absolute and difference threshold studies have provided the psychophysical data on light adaptation. All of these measures will be reviewed to provide a broad context for visual evoked response studies of the process.

Unfortunately, light adaptation data from the studies reviewed in the following two sections are not directly comparable to those in the experiments to be described in the final section of this paper, because the measures involved are quite different. In the psychophysical, and electroretinogram and intracellular recording studies, responses to a stimulus superimposed upon, or following the extinction of, an adapting light are measured. Adapting and test stimuli are identical in the steady-state visual evoked response experiments here, and this is not usually the case in light adaptation studies. Hartline's recordings from the visual cells of Limulus (1938, 1940) are exceptions to this generalization.

A second difference between the studies reviewed below and the experiments described thereafter concerns the measures used. In both psychophysical and electrophysiclogical research on light adaptation, response magnitude and amplitude, respectively, and threshold for some criterion response have been of primary interest, as is apparent from the preponderant use of those measures.

The review will emphasize technical problems in the study of light adaptation, especially where subjects of our own species are employed, since the primary purpose of the experiments was to discover, and to solve, problems associated with the study of visual adaptation using the human steady-state visual evoked response.

The experiments reported here consist of pilot studies of light adaptation in the human steady-state visual evoked response. The steady-state visual evoked response is a relatively new measure which employs high-frequency (3 Hz or greater), repetitive stimulation. The experimental work explored some of the problems inherent in the use of averaged responses to repetitive stimulation in the study of light adaptation.

In response to those problems, a research program was adumbrated, incorporating a method of obtaining averaged responses to repetitive stimulation which is especially appropriate for the study of evoked response adaptation with such stimulation. This program will be described in the concluding section.

SECTION II: PSYCHOPHYSICAL EXPERIMENTS.

Psychophysical measures have historical priority over physiological approaches to the study of light adaptation, since the experience of adaptation was accessible to systematic observation and experimental manipulation long before the instrumentation for electrical recording and chemical assay of visual processes became available. Psychophysical measures have, therefore, formed the basis for the elaboration of a powerful theoretical metastructure for explanation of the response of eye and brain to light (Rodieck, 1973).

The techniques of absolute and difference threshold measurement, and brightness matching have been employed to determine the rate, extent and pattern of changes in visual sensitivity and in perceived brightness characterizing the course of adaptation. Each method has both informational advantages and deficiencies. The functions derived by means of the various approaches differ, especially in their temporal characteristics; they cannot be expected to reproduce one another exactly, however, since each technique is designed to provide answers to a slightly different question, and may well tap a different set of sensory subprocesses.

Some of the problems associated with the definition of light adaptation arise from the fact that there are several changes effected by light on the visual system; and as we shall see, the available evidence does not strongly support the notion

that a unitary process underlies all the observed phenomena associated with sensitivity control.

Unlike measures employing absolute threshold, studies of difference threshold and brightness matching involve stimuli more closely resembling those of the natural environment, where small temporal and spatial differences in intensity constitute the most common visual stimuli. Davson (1950) has suggested that absolute threshold can be seen as a special case of difference threshold, in which the luminance value of the adapting background happens to be zero. Studies of light adaptation using the absolute threshold have generally sought the stimulus intensity at which fifty to sixty percent of stimuli were detected (Murch, 1973; Schiffman, 1976).

A. BRIGHTNESS MATCHING.

Von Kries first employed brightness matching in the study of light adaptation in 1877, and the measure has since proved to have a minimum of associated technical problems, though interpretational difficulties abound (Geldard, 1928a & b, 1972). Either all or part of the retina of one eye is exposed to a constant adapting light. At regular intervals a test light, set at the initial intensity of the adapting light, is projected onto the other dark-adapted eye or upon an unilluminated portion of the same retina. The test light is adjusted to achieve a

perceptual match with the adapting light, and the difference between the two luminances is plotted as a function of time after adapting-light onset.

Von Kries' experiments involved adapting the central portion of the visual field, with a concentric surround as match. This arrangement almost certainly produced some contamination of results through retinal interactions and considerable scattering of light in the retina (Cobb, 1916). Geldard's early experiments, although designed to avoid some of the difficulties associated with Von Kries' work, employed a bisected, circular stimulus field, of which one half served as adapting stimulus, the other as test (Geldard, 1928). Geldard engaged in further experimentation to reveal the extent of "interdependence" of response between retinal areas. He carried out studies with binocular, or haploscopic, brightness matching, in which one eye was illuminated with the adapting light and the other, with the test light. The manipulation clearly revealed evidence of interaction within the retina in monocular brightness matching (1928b). This finding will receive further consideration below.

The pattern of light adaptation response emerging from Geldard's work showed a brightness curve which decreases tc an asymptote about the end of the two minute period over which response change was followed. That is, intensity of the equated match field fell to forty percent of the initial intensity (69 apparent candle metres) of the dimmest adapting field at the end of 120 seconds. A 200-fcld increase in adapting luminance

produced a more rapid drop in brightness to a lower final value (Geldard, 1928b). Wallace (1937) subsequently confirmed Geldard's findings. He, too, used haploscopic stimulation, and obtained very similar results. His 66 ml adapting light produced asymptotic values within about one minute.

Another methodological improvement in Geldard's technique was restriction to one second of the time allowed the observer to effect a match. Previously ten seconds had been allowed for this procedure, by design, in order to bypass the initial rapid increase in match stimulus brightness immediately following its onset. This rapid initial phase of light adaptation was construed as part of the "action time of the retina" (approximately equivalent in meaning to "rise time for sensation"), and unrelated to the "fatiguing" process of interest in the study (Geldard, 1928a). In further explorations employing a one second match time, however, the entire response curve was observed to shift even further than would be predicted from calculation of nine seconds' adaptation, suggesting that an important part of the light adaptation process had been ignored (Geldard, 1928b).

Schouten and Ornstein (1939) took this difference seriously, and explored the early portion of light adaptation in further brightness matching experiments on direct and indirect adaptation effects. They concluded that there were two, possibly distinct processes at work, for which they coined the terms 'alpha- and beta-adaptation'. The characteristics of

beta-adaptation fit the then-accepted facts of photochemical changes in adaptation, as expounded by Hecht (1937).

Beta-adaptation was local to illuminated portions of the retina, and was relatively slow (3 to 5 minutes to completion), whereas alpha-adaptation exhibited some surprising features. Within a fraction of a second (0.1 sec or less), sensitivity across the entire retina dropped to a constant, low level after weak illumination of a small area (Schouten and Ornstein, 1939). The rate at which this effect spread across the retina was considerably greater than that considered possible for the diffusion of photoproducts (Bartley, 1963).

It seems not unlikely that alpha-adaptation is related to the "interdependence" between retinal areas previously described, since both involve action at a distance and, apparently occur over the same time course. Boynton, Bush and Enoch (1954) and Fry and Alpern (1953) criticised Schouten and Ornstein's pioneering studies on the grounds that the observed rise in threshold in areas distant from the site of direct illumination was produced by scattered light within the eye.

A number of investigators have since pursued the question of remote adaptation effects, employing experimental conditions which obviate the problem of scattered light (Lipetz, 1961; Rushton & Westheimer, 1962; Rushton, 1963,1965; Westheimer, 1967; Easter, 1968; Tong & Green, 1977; Green, Tong & Cicerone, 1977). These studies have uniformly found evidence of rapid changes of threshold in unstimulated portions of the retina.

The direction of this remote effect is less clear. A number of early studies demonstrated a decrease in threshold level (Geldard, 1931; Beitel, 1934). Geldard remarked that, at the moderate luminances employed in his study, the comparison light for a monocular brightness match after light adaptation required approximately half the stimulus energy necessary for a binocular match. This suggested to him that an increase in the sensitivity of the monocular match area had taken place. Fry and Bartley (1936), the other hand, believed remote adaptation effects were to be explained in terms of inhibition.

At least two early studies demonstrated an increase in absolute and difference threshold in regions adjacent to the site of illumination (Fry & Bartley, 1936; Beitel, 1936, respectively). Even before Schouten and Ornstein's findings became known, Wallace had directed attention to the weight of evidence supporting the "fact" that interdependence effects in monocular stimulation studies reflected events in a true nervous centre (i. e., the retina), and constituted the results of neural interaction in the eye (Wallace, 1937).

Granit and Wright went further, not only claiming that alpha-adaptation represented retinal inhibition of neural origin, but also demonstrating the similarity of time course in alpha-adaptation and in the PIII component of the electroretinogram. This last Granit construed as an inhibitory component of the retinal field potential resulting from light stimulation (Granit, 1963; Wright, 1938).

Schouten, somewhat presciently I believe, suggested that alpha-adaptation was associated with, or related to, the mechanisms effecting simultaneous contrast (Schouten, 1937, cited in Wright, 1938). This phenomenon is now generally believed to arise through lateral inhibition by neural elements in the retina (Diamond, 1953, 1960), although cortical processes may also play a role (De Valois & Pease, 1971). It was not thought then that that an effect as complex as simultaneous contrast could have an origin below the cortical level.

In addition to approximating the sort of task normally handled by the visual system, brightness matching enjoys the added advantages of obviating the threshold definition problems mentioned above and, in the case of haploscopic stimulation, of convenience.

B. ABSOLUTE THRESHOLD STUDIES.

While experiments with brightness matching address the question, "How bright will a stimulus of a particular intensity appear after some given duration of viewing?", absolute threshold studies query whether a stimulus of a specific low intensity will be detected at all after light adaptation at various levels of intensity and duration. The difficulties surrounding the definition of threshold are among a multitude of methodological problems plaguing the study of light adaptation using the

recovery method, which employs absolute threshold as a measure of visual sensitivity. Here the eye is exposed to an adapting light of constant luminance. The adapting light is extinguished after a systematically varied exposure time, and the eye presented with test flashes of very low luminance. These flashes are gradually raised in intensity until just perceived.

The problems associated with this technique are twofold. First, some period of darkness must intervene between adapting light extinction and the presentation of the test flash. No matter how brief this dark interval, much of the rapid, early phase of dark adaptation will have been completed by the time the test light is turned on, since only milliseconds are required for the early part of dark adaptation (Geldard, 1972; Dowling, 1967). This difficulty may, in principle, be remedied by computation of the extent to which the resulting function would be shifted in compensation for the amount of dark adaptation which would have taken place in the interval between adapting and test lights.

But such a remedy relies upon detailed knowledge of the course of dark adaptation. We do know that the initial phase of dark adaptation proceeds very rapidly; its rate and extent are determined by the duration and intensity of the adapting light. This aspect of the process has been relatively well documented (Mote & Riopelle, 1953).

Were we confident that some fully and immediately reversible, unitary photochemical or neural process underlies light adaptation, and that light and dark adaptation are

functionally independent, the procedure would appear straightforward. We cannct be certain, however, that there are nct complex interactions between the complementary processes of light and dark adaptation which would introduce nonlinearlities into the values obtained here. The available evidence does not support these hopes. Even within Mote and Riopelle's data, there appear to be two photochemical processes at work in dark adaptation. Moreover, if inhibitory neural interactions are involved in effecting adaptation in both directions, one may expect that states of inhibition may both build up and decay in some nonlinear fashion.

In effect, the dark adaptation functions which have been obtained depend upon the intensity of the preadapting light. With an intense adapting light, the rise in sensitivity following the offset of illumination may be considerable. Variations between investigators in choice of adapting luminance and duration of dark interval (between adapting light offset and test flash) may be expected tc produce varied experimental results.

Lchman (1906 & 1907, cited in Bartlett, 1965) used a dark interval of ten seconds in his study of the absolute threshold changes produced by light adaptation. He found a rapid rise in threshold during the first few seconds, followed by a slower rise to an asymptotic level. The rate of change in threshold as measured by this method depends upon adapting light intensity. Thus, the time at which a final value is achieved may be as long as half an hour for moderate intensities, and as short as a few minutes with high adapting luminances. While this measure would

perhaps describe the time course of threshold changes adequately, it would undoubtedly be necessary to extrapolate from known dark adaptation functions (with the provisos observed above) in order to determine the extent of those changes.

Similarly, Muller's 1931 study demonstrated that, while most of the threshold change was completed within the first ten minutes, a slow rise continued for yet another thirty minutes (cited in Graham, 1965). In this study, measurements were obtained a full minute after the adapting light had been extinguished, which no doubt shifted the curve along both axes considerably.

A second difficulty with the use of an absolute threshold measure in the study of light adaptation involves the probe stimuli used to achieve threshold stimulation following adapting light offset. Commonly, threshold is determined by the method of limits, using only the ascending series begun well below the expected liminal value. Wagman, Hartline and Milne have demonstrated that a subliminal flash may affect markedly the threshold level of response to subsequent supraliminal stimulation (Wagman et al, 1949). They report that the recovery time from a subliminal flash may be as long as five seconds. Obviously, such effects may well result in artificially elevated threshold values. It is not surprising, considering the multiplicity of methodological problems associated with the techniques, that the recovery method and the absolute threshold measure have seen little use in the study of light adaptation.

C. DIFFERENCE THRESHOLDS.

A third psychophysical measure of light adaptation effects which has been widely used employs the difference threshold as an indicator of visual sensitivity. The just discriminable intensity increment upon an adapting field (jnd) is a measure of visual sensitivity which gives evidence of change in a direction apparently opposite to that observed in absolute threshold and brightness matching experiments. Baker (1949) found the size of the incremental jnd to decrease at the beginning of light adaptation, which suggests that there is improved sensitivity immediately following a rise in luminance. Sensitivity then decreased rapidly over two to three minutes, and declined slowly thereafter to asymptote within ten to fifteen minutes. The recovery phase proceeded most rapidly after adaptation to dim backgrounds (e.g., 5 T), but reached its greatest extent with a 5000 T adapting field. Crawford (1947) and Boynton and Triedman (1953) observed changes in foveal (cone) and peripheral (rod) thresholds, respectively, during light adaptation. Crawford found a rapid initial drop in sensitivity during the first few seconds of adaptation, a period not observed in Baker's study. Otherwise, Crawford's results and those of Boynton and Triedman were generally in good accord with those reported by Baker, indicating that rod and cone responses to light exposure are

similar enough to produce similar activity in succeeding neural processing stages.

Difference threshold studies are free of some, and not others, of the methodological problems associated with other psychophysical measures. Since the test stimuli may be presented in series, beginning with subthreshold values, the test intensity which is finally detected may well be artificially raised by the effect of the unsuccessful probes which preceded it (Wagman, et al., 1949). Evidence vindicating this concern was presented above. Spatial aspects of remote adaptation effects need not be expected to distort our view of the rate and extent of response changes, since adapting and test stimuli are presented to the same portion of the retina. And, indeed, it is the relationship between the effect of the adapting, or background, light and that of the test stimulus which is of interest in a difference threshold study.

SECTION III: THE SITES AND MECHANISMS OF ADAPTATION -- PHYSIOLOGICAL STUDIES.

There are two kinds of questions one can ask in pursuing knowledge of light adaptation beyond description of the results of psychophysical experiments. It is of interest to know where the process takes place, and how it is effected.

The intial question about the site of adaptation must be whether the effect is entirely peripheral in origin or has some cortical components. Uttal (1969) contributes the general observation that the response compression observed in psychophysical studies is also evident in the response patterns of peripheral sensory neurons.

But, similarities cf response pattern are insufficient to persuade us entirely that no further processing of visual information related to light adaptation occurs between retina and cortex. Uttal further observes that orthodromic stimulation of peripheral axons (cf sensory neurons) produces rapid saturation cf cortical evoked potentials, demonstrating a very narrow dynamic range of post-peripheral processing. Since evoked potentials in most sensory modalities usually respond over a wide dynamic range, it appears that peripheral visual preprocessing must contribute significantly to the extension of response range.

This information accords well with the early intuitions of researchers like Barlow, who remarked that even the histological appearance of the retina is incompatible with the notion that its

elements do no more than pass on to the brain unaltered the mosaic of stimulation incident upon the receptors (Barlow, 1953a). The results of his experiments with optic nerve activity confirmed that the spatictemporal pattern of receptor illumination has been transformed by the time that information exits the retina. Admittedly, such transformation need not represent any significant modification of the signal by the peripheral visual processing apparatus, and may only constitute another dialect change in the language of the nervous system.

The pressure-blinding studies of Craik and Vernon provide the sort of definitive information that is required to localize convincingly the level at which light adaptation takes place (Craik & Vernon, 1963). Subjects were pressure-blinded during exposure of their retinae to adapting light. This procedure, which involves the application of pressure to the far temporal portion of the eyeball, temporarily obviates normal visual experience (including that of light adaptation) by preventing the passage of signals exiting (and entering) the retina via the optic nerve. Information about light incident upon the eye does not then reach the brain. Nonetheless, light adaptation proceeded normally, as demonstrated by the subjects' psychophysical responses at an adapted level when pressure was released.

A second approach, which effectively denies the possibility that centrifugal efferents play a role in light adaptation, involves electroretinograms from excised eyes. Here,

too, light adaptation follows its usual course (Barlow, 1953a; Donner & Reuter, 1971).

The obvious course of inquiry, now, is to seek the specific retinal loci involved in light adaptation. We are looking for mechanisms which can effect a dynamic range of response extending over as much as 5 to 6 log units. In their 1969 review of light adaptation, Creutzfeldt and Sakmann concluded that we may reasonably expect to find that adaptation results from the activity of a "series of filter processes along the vertical signal transfer chain."

The retinal sites and mechanisms of adaptation will be considered in this section, from the most peripheral -- effects at the site of quantum catch, photopigment bleaching -- to the more central interactions in the neural network of the retina. Evidence of activity in the lateral geniculate nucleus and visual cortex associated with light adaptation, and problems in the measurement of such activity, will be described in the final section of this paper, as part of the more specific introduction to the experiments to be reported there.

The first level of visual function to be investigated with respect to light adaptation was visual pigment bleaching. Although the intuitively obvious notion, that visual sensitivity should correlate with rhodopsin concentration, has not received impressive experimental support, its history will be reviewed briefly below. Evidence for an intra-photoreceptor adaptation

mechanism which is independent of rhodopsin concentration, and for important contributions to light adaptation from inhibitory interactions within the neural network of the retina, will subsequently be considered.

A. PHOTOPIGMENTS AND VISION.

Hecht published his thecry of the photochemical basis of vision in 1937. His thesis was that light adaptation occurs when the visual pigment concentration of the photoreceptors is depleted by the bleaching action of light, decreasing the amount available for futher activation of the rod. Dark adaptation, or the recovery of sensitivity, was presumed to be a direct result of the regeneration of photopigment, which would permit a new cycle of excitation and adaptation.

In the past four decades, the vision literature has been regularly enriched by a steady stream of experimental and theoretical contributions which attempt to deny that Hecht's thesis was entirely correct, and which disagree with his assertion that the decay and regeneration of photosensitive pigments have overriding importance for visual adaptation. Naturally, Hecht's disciples have countered such criticism with sophisticated studies and theoretical reformulations designed to support the original thesis, at least in principle, and the most important of these will be considered below.

The issue is not whether the photochemical reactions described initially by Hecht are necessary for vision; for clearly the light energy incident upon the retina must be transduced into the electrochemical currency of the nervous system -- graded and spike potentials activating neural membranes. And there is nc longer any disagreement among vision scientists that the cis-trans-photoisomerisation of retinal (a component of the rhodopsin in receptor outer segment membranes) is the necessary event for the initiation of the visual response. There is argument, however, as tc whether the facts of photopigment decay and regeneration are, or could be, sufficient to explain all the facts of light and dark adaptation. The scientific consensus is increasingly that they are not. Let us examine briefly the pertinent events, and theories, of retinal photochemistry in order to clarify the grounds for disagreement.

Hecht's ideas were not new. Kuhne, in 1879, had reported his observation that, just as light adaptation induces a decrease in perceived brightness, which recovers during dark adaptation, so the retinal concentration of rhodopsin decreases during light exposure and is restored in the dark. It was not unreasonable to speculate about possible causal relations between the two sets of facts: a light-induced decrease in visual pigment concentration producing directly a decrease in visual sensitivity; and, improvement of sensitivity during dark-adaptation resulting from photopigment regeneration. Unfortunately for the model, systematic observation of the phenomena associated with light and

dark adaptation have demonstrated that many aspects of adaptation have no direct relation to the amount of visual pigment bleaching or regeneration that has taken place, as described below.

Lythgoe, in 1940, reported that his brightness matching studies showed evidence of considerable loss of sensitivity in response to an adapting field too dim to effect significant photochemical reaction. Investigations using almost every other measure of light adaptation have similarly found considerable change in sensitivity without significant alteration of retinal photopigment concentration.

The rate of threshold or response alteration observed in light adaptation, too, is inconsistent with any photochemical explanation of the rapid early phase of the process (Dowling, 1967). The discussion in the preceding section of remote adaptation effects included much of the evidence pertinent to this incongruity between the observed course of Schouten and Ornstein's alpha-adaptation and predictions from Hecht's equations.

What, then, given an extensive list of important light adaptation phenomena which are anomalous to photochemical theory, is the nature of the relationship between visual pigments and light adaptation? For, such a relationship must exist.

One may seek a more complex photochemical basis for light adaptation than that proposed by Selig Hecht in 1937. This has been Wald's enterprise. He has proposed a "compartment theory", in which individual rhodopsin-containing compartments within

photoreceptors (corresponding to rod discs, or saccules, presumably) are separately inactivated by the absorption of even a single quantum of light (Wald, 1954). This arrangement should produce a temporal shift in light adaptation function maxima and minima corresponding to different intensity and duration values of the adapting light.

Light adaptation curves derived from naturally- (Troxler effect) and artificially-stabilized image experiments, and from electroretinogram (erg) and retinal ganglion cell discharge recording, fail to show time course changes consistent with Wald's predictions. This lack of experimental support for Wald's model casts some doubt upon the explanatory value of such a compartment theory (Pirenne, 1962).

Nonetheless, Marimont (1965), in analysing data used by Fuortes and Hodgkin (1964) in developing their feedback model of adaptation (which will be discussed below), concluded that the data were better described by a model with many compartments than by the feedback model originally fitted to the data.

More successful is the observation that the fall in rhodopsin concentration is paralleled by the rise in log threshold intensity (Dowling & Wald, 1963). This relation holds for a variety of measurements on several species: frog erg; human scotopic thresholds; rat and skate erg and ganglion cell recordings (Ripps & Weale, 1976). Dowling's equation describing the relationship between photopigment concentration and log threshold apparently enjoys fewer weaknesses than previous,

similar attempts (Ripps & Weale, 1976; Dowling, 1967). The physiological basis for this rhodopsin concentration - log threshold intensity relationship remains unelucidated, however. Some mechanism of response compression must intervene to effect that log transformation.

The shortcomings which do exist in this formulation comprise the basis of Dowling's enthusiasm for a theory of adaptation which focuses upon inhibitory feedback mechanisms in the neural networks of the retina, rather than on photochemical decay and regeneration processes (Dowling, 1967). This search for alternative sites of adaptation constitutes a more exciting, and probably more viable, response to the deficiencies of Hecht's model than reformulations thereof. If one accepts the evidence cited by Dowling (1967), visual chemistry contributes even less to sensitivity control than does the pupillary mechanism -- about 0.3 log units compared to approximately 1.0 log unit -- although it should be evident that these contributions are different in kind. Pupillary constriction reduces the amount of light incident upon the retina, whereas photochemical bleaching alters the responsivity of that surface.

As will be described later, the data demonstrating extensive intra-receptor adaptation are more consistent with active, negative feedback within the receptor, apparently an inhibition by photoproducts, than with loss of photosensitive substance.

B. ADAPTATION IN THE ELECTRORETINOGRAM.

Electrical phenomena associated with light adaptation can be detected in extracellular recordings of the activity of retinal neurons. The electroretinogram reflects extracellular, or field, potentials generated across the entire retina (or across some local region of the retina -- the local electroretinogram, or lerg). This potential consists of a sequence of positive- and negative-going components, each of which responds in a characteristic way to changes in visual stimulation.

Light adaptation can be observed to produce a decrease in the overall amplitude of the erg, due primarily to disappearance, or decrement, of some positive "on-response" components, and to increased prominence of negative phases: the cornea-positive c-wave and the slow component of the b-wave disappear; the negative a-wave becomes larger; and, the d-wave, or off-response, of negative polarity increases in amplitude (Granit & Riddell, 1934; Brown, 1968).

Armington (1964) has recently reported results which are not entirely in accord with those from Granit's laboratory. Armington observed amplitude of the erg b-wave (to a 170 ms flash at 0.5 Hz) to increase as a function of stimulus intensity for a given light adaptation time, whereas Granit and Riddell (1934) reported a decrease in erg amplitude with increasing intensity.

In Armington's study, psychophysical sensitivity decreased

during the first minute of light adaptation, remaining at a low level thereafter. This was the case at all stimulus intensities. Study of the erg b-waves tc the first five individual flashes (at 0, 0.5, 1.0, 1.5 and 2.0 s after the beginning of the 2 Hz stimuluse train) reveals that the second response is of much lower amplitude than the first, the third and succeeding responses recovering slightly to achieve a stable level which was still below that cf the first response.

Such apparently paradoxical results can also be seen in psychophysical difference threshold measures of light adaptation. While the liminal intensity increment is larger for very bright backgrounds, the size of the just-discriminable increment decreases over the course of light adaptation (Baker, 1949).

While some of this effect may be due, in the case of psychophysical studies, to peculiarities of the relative values of adapting- and match-field intensity values, there is another important aspect to these data which ought not be ignored. The opposite effects produced in both erg and psychophysical difference threshold studies by adaptation time and by adaptation level suggest that at least two distinct processes may be involved in coding stimulus intensity and in extending the range of brightness response in the visual system; and that, although opposed, the effects of these mechanisms do not entirely cancel each other. That is, erg amplitudes and psychophysical thresholds are generally higher at high than at low background stimulus intensities, and these response levels decrease in

parallel over the temporal course of light adaptation. This is consistent with the existence at least two separate loci for the fast and slow aspects, or processes, of light adaptation. And, Werblin's extensive research has yielded data which is strongly supportive of his hypothesis that light adaptation is primarily executed at a site peripheral to the inner nuclear layer, while spatiotemporal brightness discriminations depend upon the synaptic apparatus of the inner plexiform layer (Werblin, 1974).

Wright thought that he had proved this same distinctness of adaptation and intensity coding as long ago as 1935, using a psychophysical threshold measure. His experimental controls were inadequate by current standards, however, and his reasoning rather obscure.

There continues to be considerable disagreement over the retinal elements generating, or contributing to, the various components of the erg and over the correct interpretation of the electrical activity measured by the erg, even where the origin of a component has been reasonably well identified. That is, erg studies alone do not much further our understanding of the mechanisms effecting light adaptation.

Microelectrode recordings of the activity of single retinal units cannot help but increase our information about their behavior during light adaptation. No greater understanding of the mechanisms of light adaptation will necessarily result from such studies, however, unless the research is pursued in the context of biochemical and/or

biophysical models of light adaptation.

The following section of this paper will describe briefly the results of some recent studies of single retinal unit activity, and the relationship of these findings to current models of photoreceptor adaptation and of neural interaction in the retina.

C. ADAPTATION OF INDIVIDUAL RETINAL ELEMENTS.

Intracellular recordings of each type of retinal unit show adaptation effects (Werblin, 1974). Photoreceptors and horizontal, bipolar, amacrine, Muller and ganglion cells each have a characteristic pattern of response during adaptation. Each cell type will be considered in turn in this section.

A variety of experiments have explored photoreceptor light adaptation. These studies have sought to discover the mechanisms by which photoreceptor adaptation is effected.

Grabowski et al., (1972) recorded intracellularly from rods in isolated retinae of the salamander, Ambystoma mexicanum. They observed rapid decline in the amplitude of photoreceptor hyperpolarization, which recovered in several minutes to a level below that of the pre-adaptation potential. Recovery followed closely the decay of metarhodopsin II, rather than visual pigment regeneration (not possible in the isolated retina). The fact that recovery was not completed by the time that metarhodopsin II

had fully decayed suggests that some other photoproduct may influence the recovery level achieved (Rodieck, 1973).

Normann and Werblin (1974) observed in recordings from mudpuppy photoreceptors that the rod response simply saturated in the presence of a 5.5 log unit background, whereas the peak responses of cones (with a 3.5 log unit response range) shift as a function of background light intensity, in accord with Weber's Law. This range shifting effect begins 2 log units before measurable bleaching occurs. Their data represent responses characteristic of the steady adapted state, rather than demonstrating whether this shift in peak response follows a progressive, unidirectional course. Evidence from studies of light adaptation using other measures, such as the psychophysical difference threshold experiments described above, indicates that it is not safe to assume monotonicity of response over time. Normann and Werblin suggest that the adjustment of response range must involve a mechanism located after the site of quantum catch, since sensitivity does not always correlate well with visual pigment concentration.

Recent studies of intra-photoreceptor adaptation have been directed toward testing a new model of rod excitation and its implications for rod adaptation (Yoshikami and Hagins, 1971). The model describes the intracellular communication from rod disc to plasma membrane of information about photon absorption by rhodopsin molecules. Whereas in the dark a current is carried across the rod outer segment (ROS) plasma membrane by inflowing

sodium ions (the dark current), the absorption of just one photon in a rod can substantially reduce this sodium conductance. The effect of photon absorption by the rhodopsin of the intracellular disc must be communicated to the plasma membrane, in order to effect this decrease in sodium conductance and, thereby, the hyperpolarization of the rod cell characteristic of rod excitation.

Numerous experiments have now been conducted to adduce support for Yoshikami and Hagin's suggestion that calcium may serve as that intracellular messenger for excitation in vertebrate rods (for example, Brown, Coles & Pinto, 1977). Lisman and Brown (1972) proposed a similar role for calcium ions in adaptation of ventral photoreceptors in the invertebrate, Limulus. This latter hypothesis has also received experimental support from other laboratories (Fein & Charlton, 1977a & b).

In the models mentioned above, calcium ions are released from the disc following photon absorption, and bind rapidly and reversibly to sodium channels in the plasma membrane, effecting hyperpolarization of the rod cell. According to Hemila's model of ROS adaptation (for which he has adduced supporting experimental results), a continuous release of internal transmitter is required to maintain the hyperpolarized state. Kuhn and Bader (1976) have proposed that the light- induced phosphorylation of rhodopsin may effect a change in the permeability of the disc membrane to calcium ions, which decreases the transmitter release rate and, hence, the plasma

membrane potential of the cell. Presumably, an hypothetical Ca sequestering mechanism then becomes the dominant process in the regulation of Ca movement, returning the transmitter to the disc. Kuhn and Bader's suggestion that rhodopsin phosphorylation may play this role in adaptation is based upon the similar time courses of the two processes, the former having a half-time of about 2 minutes, and the latter being complete within 6 minutes or less on most psychophysical and physiological measures. Like the other models of photoreceptor excitation and intracellular light adaptation, this hypothesis has not yet been proven.

Werblin (1974) has recorded sensitivity changes intracellularly during light adaptation in each type of retinal unit. Bipolar cells exhibit an extensive dynamic range of response, which shifts as a function of background intensity. Horizontal cells demonstrate some extension of response range, but this saturates quickly -- about 3.5 log units above threshold. Amacrine cells behave rather more like vertically-signalling units, with operating point alterations extending their response range over about 6 log units.

Whereas photoreceptors and horizontal, bipolar and amacrine cells all respond to stimulation with graded signals, for which amplitude measures are appropriate, a different sort of measure is required in the observation of ganglion cell response. This latter cell type produces spike, or action potentials along its axon, and response level is reflected in discharge rate, or spike frequency.

Recordings of retinal ganglion cells indicate that the discharge rate for on-center cells is an inverse function of sensitivity and of ERG b-wave amplitude (Barlow, 1953a). ("On-centre" ganglion cells respond with a burst of spikes to illumination of the central portion of the 2-dimensional matrix, or field, of bipolar cells from which they receive input. Stimulation of the peripheral portion of such a cell's receptive field suppresses its low-level spontaneous discharge. "Off-centre" ganglion cells respond to stimulation of the central and peripheral, or surround, portions of their receptive fields with suppression and enhancement, respectively, of their spontaneous discharge rates.) The on-centre ganglion cell discharge frequency shows an initial burst at light onset, then decreases rapidly to an asymptotic level, (Granit, 1962 b; Hartline & Graham, 1932). As well, inhibitory surrounds appear in ganglion cell receptive fields only in the light-adapted state, interfering with spatial summation in those receptive fields, but undoubtedly contributing to visual acuity Barlow et al., 1957; Kuffler, et al., 1957). Barlow interpreted his results as indicating: first, that these inhibitory surround effects are related to lateral inhibition in the retina; and, further, that this lateral inhibition must occur at a stage more peripheral than, and hence prior to, spatial summation.

Werblin has recently demonstrated that bipolar cells behave very similarly to ganglion cells, with antagonistic centre-surround organization (Werblin, 1971). Bipolars do not

generate spike potentials. Rather, they show graded hyperpolarization or depolarization, which increases abruptly at light onset. Response amplitude can subsequently be seen to decline somewhat during adaptation. Unfortunately, the time course of this response was not a feature of interest in Werblin's study; hence, one can only obtain a rough approximation of the course of light adaptation during the two second period of stimulation. It appears that adaptation is complete within one half to one second at most stimulus intensities. A similar constancy in the time course and extent of oscillations in ganglion cell spike frequency has been reported (Barlow, 1965).

D. NEURAL FEEDBACK MECHANISMS IN LIGHT ADAPTATION.

The first mechanism of adaptation to be suggested as an alternative to Hecht's photochemical model of the process was active inhibition within the neural plexes of the retina (Granit, 1933, cited in Granit, 1963; Wright, 1935; Wallace, 1937). Granit, especially, was confident that active inhibition plays an important role in retinal information processing.

The subsequent history of light adaptation studies appears to support Granit's view, although positive experimental evidence was not immediately forthcoming. For, most investigators in this area now assume that both intra-receptor sensitivity declines and inhibitory interactions between retinal neurons contribute to

light adaptation. Hemila (1977), for example, introduced his new model of ROS adaptation under the explicit assumption that such a model of intra-receptor adaptation must provide for the possibility of cooperation between neural network and intra-photoreceptor adaptation processes.

Barlow, too, apparently viewed the effects observed in adaptation as the result of active inhibition. In an early paper, for instance, he postulated that a rise in ganglion cell threshold in adaptation reflected an increase in retinal inhibition (1953b). That claim appeared to have been arrived at through interpretation of the results of experiments on retinal ganglion cell receptive fields, rather than through direct evidence of inhibitory processes.

In the past decade, or so, research has been more ambitiously directed toward garnering direct evidence of inhibition between specific retinal elements. Fuortes and Hodgkin (1964) proposed a feedback model for an automatic gain control mechanism in the eye of Limulus. Research by Pinter (1966) and by DeVoe (1966) on the eye of the wolf spider produced considerable support for that model. A subsequent analysis by Marimont (1967) found that a compartmental model better described Fuortes and Hodgkin's original data, although there is little difference between the models and their fit to the data. Naka and Kishida (1966) also succeeded in finding support for their assumption that there is presynaptic inhibition in the compound eyes of insects.

More pertinent to our interests here, however, are studies on vertebrate retinae. In their electron microscope studies of primate retina, Dowling and Boycott (1969) observed reciprocal synapses between bipolar and amacrine cells. On the basis of such information, Dowling postulated a mechanism of adaptation involving inhibitory feedback from the amacrine cells to effect bipolar cell response decrement. Easter (1968), in his study of adaptation in the eye of the goldfish, also favored the amacrine cell-modulated synapse between bipolars and ganglion cells as an important adaptation site.

More recently, however, Miller and Dowling (1972) have observed in intracellular recordings that the only cell type which produces a response with the same time course and extent as the erg b-wave is the Muller cell. Rodieck (1973) has explained the response of the Muller cell as typical of glia (although it is an epithelial rather than a glial cell), depolarizing in the presence of extracellular potassium. Extracellular potassium concentration can be expected to change in the immediate vicinity of hyperpolarizing and depolarizing neural elements. Muller cells may be responding to extracellular cation concentration shifts induced by graded and/or spike activity of local neural elements.

This explanation would make sense of the otherwise puzzling fact that no other single unit shows a light-evoked response identical in form to the b-wave, while the presumably nonsignalling Muller cell mimics the behavior of the b-wave. For

the b-wave may not be a unitary response. Instead, two or more retinal cell types may contribute differently to its positive- and negative-going components, while the Muller cell simply responds to all local activity. This is consistent with Rodieck's statement that the components of the erg are but "electrical epiphenomena" of retinal function which reflect, rather than constitute, functional activity in the retina (Rodieck, 1973, p. 525).

The Muller cell is a relatively large cell, which spans most retinal layers, extending from the receptors through the ganglion cell layer, with its cell body in the inner nuclear layer. Thus, it may be subject to influences from widely distributed points in the retina. Miller and Dowling (1972) suggest, on the basis of b-wave polarity through these layers, that the primary influence must be from regions distal to the inner nuclear layer.

Miller and Dowling's conclusion that the Muller cell generates the b-wave is not altogether warranted. For, it is not unreasonable to suppose that the field potential component (b-wave), like the response of the epithelial cell (Muller cell), reflects the activity of several retinal elements. This is consistent with the tradition begun several decades ago, by Granit (1963), of analyzing positive and negative deflections in the erg in terms of underlying components which are only partially reflected in the whole potential.

Data have also become available which provide support for the notion that the outer plexiform layer (OPL) is most importantly involved in the adaptation process. There is still disagreement as to whether direct axo-axonal junctions between visual receptors at this level, such as those observed by Sjostrand, (1958, cited in Tamar, 1972) do in fact mediate lateral interactions, and whether such interactions are mutually inhibitory.

Other research appears to support Rodieck's conclusion that "the main path for lateral interaction . . . is via the horizontal cell layer" (Rodieck, 1973, p. 456). This layer is thought to effect, through inhibitory feedback, the adjustment of the response range of the bipolar cell to a level appropriate to the luminous environment. In Necturus, this has the effect of extending the response range of the bipolar -- very necessary considering that the input to a bipolar is a multiple of the dynamic range of each contributing receptor by the number of receptors contacting the bipolar (Rodieck, 1973).

The outer plexiform layer is apparently the major site for the neural feedback component of the light adaptation process, forming the third or fourth level of control, after visual pigment bleaching, adaptation processes within photoreceptors, and, possibly, inhibitory interaction between the receptors.

It appears now that there are several stages of adaptation, reflected in measures which reveal the responses of

different retinal units. There is, in addition to visual pigment bleaching, feedback inhibition within receptors produced by photoproducts. Both of these processes were discussed in this section. The synaptic machinery for interactions between receptors is present, as well (Sjostrand, 1965, cited in Tamar, 1972). And the synaptic ccntacts in the OPL are appropriate to a variety ofinhibitory interactions between photoreceptor, bipolar and horizontal cells, which may effect response decrements in, and adjust the response ranges of, all three types of retinal unit.

Some of the more important sites and mechanisms which may be responsible for effecting adaptation in the retina have been discussed above. There is some evidence to support the claims made for each of these, but none appears to have been proved as yet. Neither have the relative contributions of each stage of adaptation in the retina been fully determined.

SECTION IV: THE EXPERIMENT.

A. VISUAL EVOKED RESPONSES AND LIGHT ADAPTATION.

The development of computer averaging techniques for the extraction of evoked, or event-related, potentials from the the ongoing encephalographic record has opened new avenues for the exploration of central nervous system activity in intact, living organisms. Such techniques are required by the small amplitudes of event-related cerebral potentials, which may be 20 to 100 times smaller than the ongoing "biological noise" of activity which is not directly related to experimental stimuli (Regan, 1972). There is also special value in using human subjects in evoked response studies, as psychophysical measures can be obtained under conditions very similar to those use for the psychophysiological measure, permitting comparison of responses on experiential and neural measures.

In obtaining an averaged evoked response (AER), a series of identical stimuli are presented successively to the subject, presumably evoking thereby a series of identical responses. The averaging process is, then, intended to "average out" brain electrical activity which is unrelated to the stimuli, but not variability between individual responses, since no such variability is anticipated.

If there is variation among the individual responses in a series of responses which are being averaged together, however, it is methodologically interesting and important to determine the source and nature of that variation. Adaptation is one possible source of systematic change in evoked response latency and/or amplitude. Random variations resulting from movement or attentional fluctuations may be of less interest. The phenomenon of light adaptation in the visual evoked response is, therefore, of methodological, as well as intrinsic, interest.

Scalp-recorded average evoked potentials may be of two sorts: "transient" responses, in which interstimulus interval is 333 or more milliseconds; and "steady-state" responses evoked by repetitive stimuli at approximately 3 to 50 Hertz (Regan, 1972). Transient visual evoked responses (VER's) consist of a series of "components", positive and negative deflections of the record. Response amplitude is usually measured as the the extent (in mV) of these deflections, from a positive to a negative peak, or vice versa. Components are often labelled according to the polarity and the latency of the peak from the stimulus (e.g., P200 would refer to a positive peak of 200 ms latency). Although there are a number of other systems of nomenclature in common use as well, this format will be employed where transient VERs are described here. Steady-state VERs consist of a series of responses at the frequency of the stimulus, or some harmonic thereof, and are often termed "photic driving" or "following responses". These responses are often Fourier-analysed, and described in terms

appropriate to such analysis. Recently, two measures of steady-state VER latency have been developed, one involving Fourier analysis (Regan, 1972), and another which does not require such treatment of the data (Diamond, 1977a). These will be described after some review of the transient literature on light adaptation, below.

There is an important limitation to the use of transient VERs, which has also been a problem with the use of steady-state VERs until recently. Since we lack information about the precise cerebral (or subcerebral) origins of particular VER components, we cannot progress in our search for correlations between the activity of specific neural subsystems and visual experience. An effective correlation between sensation and neural function should be specific about the locus of that neural activity.

Few AER studies have explicitly, or successfully, investigated the time course of light adaptation. Some of the experiments that have been done will be reviewed below. A major deficit in these studies is their excessive concern with the contribution of pupillary change to the observed effects, which demonstrates some indifference to the considerable body of research by psychophysicists and sensory physiologists which was already available. Hence, although pupillary change is an obvious candidate for some contribution to light adaptation effects, the rate and extent of that contribution was already known and was distinguishable from the retinal events of adaptation. Given the information available even two decades

ago, pupillary effects ought to have been controlled out of the studies, or else investigated for their intrinsic interest, rather than belabored as a causal feature of the major portion of light adaptation (see, for example, Regan's discussion of VER habituation, 1972, p. 135).

The reason for this unfortunate emphasis probably resides in another of the inadequacies which characterize these studies. For the most part, the experiments sampled data points which were widely separated in time, relative to the rapid course of light adaptation. It was not possible, therefore, to observe in detail any rapidly changing response, whereas the slow time course of pupillary constriction produced an observable change on the measure. The physiological studies discussed in the preceding section indicate that considerable change in visual response may take place in the first 0.5 to 1.0 second of adaptation; and transient VER studies, by definition, use stimuli at frequencies below 3 Hz (Regan, 1972).

Another problem with such studies is that they usually average across time. This creates a further difficulty in the detection of effects on response latency. This problem will be discussed in some detail in the concluding remarks on the experiment. Here, discussion will be restricted to a review of transient VER studies pertinent to light adaptation.

Armington's 1964 paper described a successful study of light adaptation in the occipital VER. His experimental results were most interesting. At all intensities of flash, the

amplitude of a component of latency N100-P200 decreased markedly on the second and third responses (to a train of 170 ms flashes at 0.5 Hz), then recovered on the fourth flash, to asymptote at a level well below that of the first response. The comparable erg record shows recovery of b-wave amplitude on the third flash. This temporal difference between peripheral (erg) and central (VER) response raises the question whether the delayed recovery of the occipital potential represents further adaptation, at either the retinal, the thalamic or the cortical level.

Callaway has reviewed a number of studies of visual evoked response "habituation" which are relatively informative, consonant with the psychophysical and microelectrode literature on adaptation, and reasonably well executed (Callaway, 1973). He describes four kinds of response decrement, of which "slow" and "fast" habituation are of some interest here. Much as in Armington's results, components between 100 and 300 ms latency show the two kinds of decrement. But the proportional amplitude reduction differs: less than ten percent in slow habituation; eighty percent or more in rapid habituation. The rate and extent of these response changes compare favorably with findings from psychophysics: rapid, alpha-adaptation producing most of the observed effect; slow, beta-adaptation effecting a shift over a longer time course (six tc twelve minutes), but only covering about 0.3 log unit intensity change (Granit, 1962a, b). Indeed, Armington's data fit well the values for beta-adaptation -- 0.3 log unit change with a mean time to asymptote of about 6 minutes.

Since beta-adaptation has been associated traditionally with the slow time course and limited effect attributed to the achievement of photochemical-reaction equilibrium during light adaptation, it is reasonable to suppose that Armington's results reflect primarily photochemical adaptation and fail to show the contribution of rapid, presumably neurally-based, alpha-adaptation.

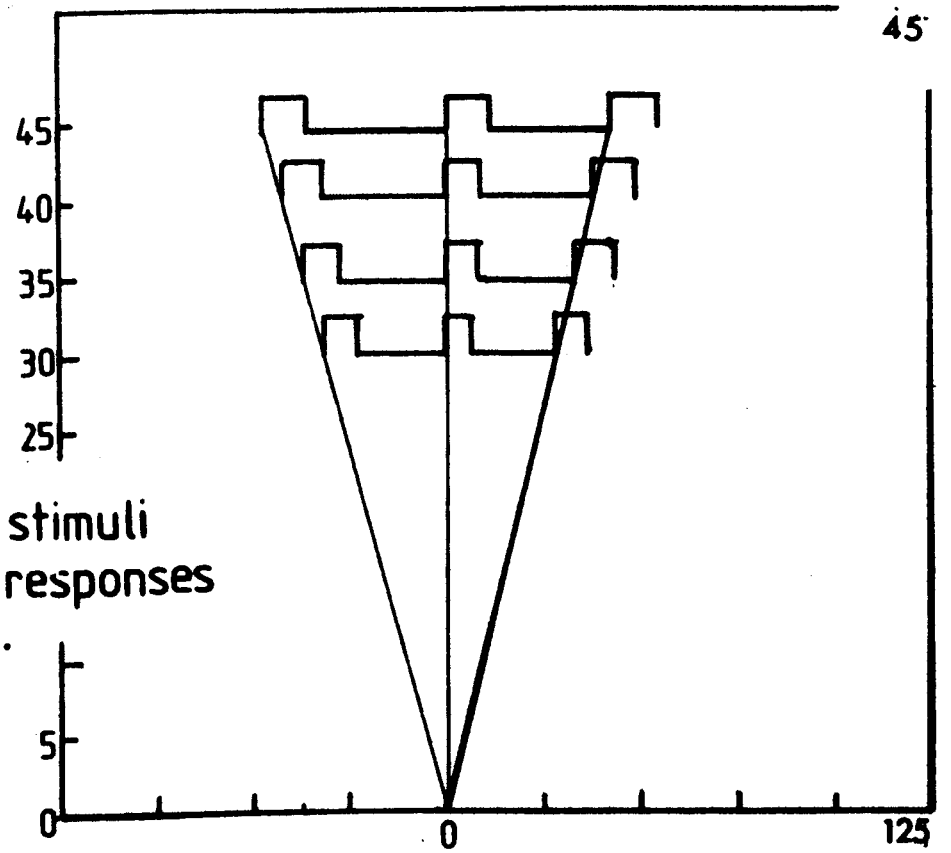
Until recently, the steady-state VER has been plagued, not only by the problem of determining the specific brain origin for its apparently unitary, biphasic response, but also with the difficulty of determining which in a train of similar responses is related to any particular stimulus. A new technique developed by Diamond appears to resolve both difficulties simultaneously, rendering possible the determination of response latency at a cerebral locus directly below the recording electrode on the scalp, when several electrode locations are used (Diamond, 1977a). The technique uses recordings obtained at four or more stimulus frequencies between about 22 and 35 Hz, or between 10 and 15 Hz, from which the positive and negative peak values in the 100 or 200 millisecond records of averaged responses are entered into a regression program. The y-intercept value produced by that program is thought to represent the latency of the response in question. On visual inspection of the recordings, this value is observable as an alignment of positive or negative waves at one latency across all frequencies of of the stimulus-response cycle. The alignment (or response latency) may

fall between a positive and a negative component, as well (on the shoulder of a positive wave, for example). Figure 1 shows the temporal relationship between stimuli and responses, and includes the regression lines which converge at the sweep trigger point (0 ms) in the case of the stimuli, and at the response latency in the case of the VERs, at the minimal ISI value of 0 ms.

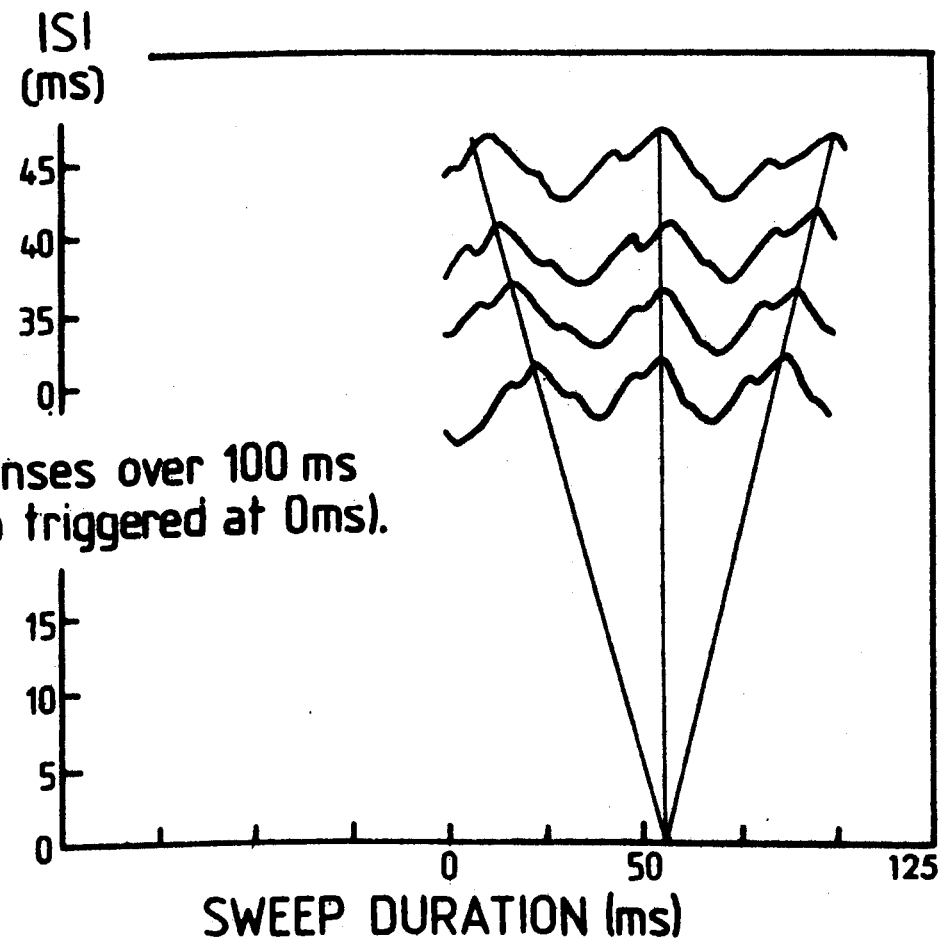
Regan has developed a similar measure in which several stimulus frequencies are presented simultaneously to the subject and the raw data are Fourier analysed (Regan, 1972). This procedure yields latency values very close to those obtained with Diamond's method, when comparable electrode placements are used. Diamond's technique appears better suited for studying the rapid changes in light adaptation than does Regan's, since fewer assumptions about the nature of the response and its relationship to the stimulus are required by the former measure, which also enhances its appeal. Regan's technique presupposes that the evoked responses will be sinusoidal in form, and that the stimulus-response relationship will be linear (see Regan, 1972, for explanation of these assumptions). The use of Diamond's measure does not depend on such assumptions (Diamond, 1977b).

FIGURE 1.

Steady-state EP latency determination



a. Square-wave stimuli which evoke responses shown in (b).



b. Evoked responses over 100 ms sweep (sweep triggered at 0ms).

B. THE EXPERIMENTAL QUESTION.

The experiments reported below were designed to study the effect of light adaptation on the latency of the human steady-state VER, using Diamond's latency determination technique. The rapid, initial phase of the process, observed in the retinal response, was of particular interest. It was not possible to predict from the available VER literature whether an increase, a decrease or a non-monotonic change in latency should be expected, since that literature consists exclusively of amplitude measures. Moreover, previous studies employed transient VER's. No experiments in light adaptation using a steady-state evoked response have been reported.

C. EQUIPMENT & PROCEDURE.

Subjects were dark-adapted for 30 minutes prior to commencement of each experimental session. For the first 20 minutes of the dark-adaptation period, the subject wore snugly-fitted goggles with red filter material set into the eye-pieces. The final 10 minutes were spent in the darkened experimental chamber, wearing a pair of goggles prepared for the experimental session, which were fitted with artificial pupils of 2 mm diameter.

Each subject sat in a comfortable chair, so positioned that the stimulus field was 1 m from her, or his, eyes. The stimulus field was then 2 degrees in diameter. Subjects were instructed to fixate without blinking upon the center of that field during stimulation, and were required to fixate upon the black matte surface surrounding the unilluminated stimulus field during the final portion of the initial dark adaptation and between trials. The intertrial dark adaptation time (DAT) varied as a function of the preceding stimulus train's duration as follows: below 1 minute of stimulation, 2 minutes DAT; between 1 and 2.5 minutes of stimulation, 5 minutes DAT; above 2.5 minutes of stimulation, 10 minutes DAT.

The stimulus intensity was regulated by interposing neutral density filters between the light source and the translucent, circular stimulus field through which that light was transmitted to the subject. In Experiment I, the effective stimulation at the eye was 2.99 log ml (0.4 neutral density filter), 2.66 log ml (0.7 filter), 2.38 log ml (1.0 filter), or 1.97 log ml (1.4 filter). Only the 1.4 filter was used in the the second experiment. Ideally, all recordings would have have been obtained using a single intensity of stimulation. As the experiments progressed, however, subjects were employed who found the stimulus intensity used with previous subjects uncomfortably bright. Downward adjustments of the intensity were made to meet their objections, resulting in in the use of four different intensities. This problem was obviated in Experiment II by using

only the lowest of those intensity values -- that produced by the 1.4 neutral density filter.

Stimulus trains were produced by an episcotister with a light-dark ratio of 1:4, which chopped the light emitted by a Kodak Carousel 800h projector into 100 percent-modulated square waves. Stimulus frequency, or its inverse, interstimulus interval (ISI), which varied as a function of episcotister rotation speed, was calibrated on a Tektronix 5031 dual-beam storage oscilloscope. In Experiment I, the ISIs used were 30, 35, 40 and 45 ms; in Experiment II, these were 28, 31, 34 and 37 ms in the first phase, and 31 ms only in the second phase. The stimulus train duration was varied from 0.3 seconds to 6 minutes, depending upon the stage of adaptation being recorded.

Steady-state VERs were picked up by a monopolar scalp electrode placed 2 cm above the inion, which was referred to an electrode on the right earlobe. An electrode on the left ear led to ground. The Ag-AgCl active electrode was affixed securely with collodion, and gold electrodes were attached to the earlobes with clips. Good electrical contact was secured with a saline gel electrode paste, and impedance between each pair of electrodes was always 2 kOhms or less.

The electrical signal from the brain was filtered and amplified by a Schonander Electroencephalograph, with high and low frequency inputs attenuated above 3000 Hz and below 0.3 Hz, respectively. The filtered, amplified signal was stored and averaged by a Fabritek 1072 averaging computer.

Altogether, eight different sampling periods were used. A sampling period consists of a series of successive sweeps of the ongoing electroencephalograph (EEG), which are averaged together into an AER record. (The term 'sweep' refers to a segment of the EEG record, the start of which is time-locked to a stimulus. In this study each sweep is 100 ms in duration, and the term is used synonymously with 'repetition'.) The sampling periods were 10 s in Experiment I, and 3 s in the first phase of Experiment II. In the second phase of Experiment II, data were obtained using several other sample times in order to elucidate further the influence of sample duration upon the quality of the data obtained in previous experiments. This last set of recording, or sample, durations was as follows: 0.3 s; 0.5 s; 0.75 s; 1.0 s; 1.5 s; and 2.0 s. The interval between temporally-successive samples was 1 s in all experiments. For example, in Experiment I, the first 10 s of responses to repetitive flashes (the adapting stimulus) was recorded, and the eleventh second omitted; the following 10 s (the twelfth through twenty-first seconds of adaptation) were recorded, and the twenty-second, omitted, and so on.

The number of repetitions included in an AER varied between 72 and 145 as a function of ISI and and sample time. In Experiment I, 74 to 95 repetitions were used in the averages; Exeriment II required 72 to 112 responses for an average. A sample period of 10 s or 3 s only provides 74 to 95, or 24 to 29

repetitions per average, respectively, the exact number depending upon stimulus frequency. When the 3 s sample period was used, especially, there were often insufficient repetitions included in an average to produce a clear record. For this reason, the trial was repeated one to four times, until the average was satisfactory.

These averaged responses were converted to digital form, and stored on disc by a Hewlett-Packard model 2116B general purpose laboratory computer. All of the stimulation and recording apparatus was controlled by Grason-Stadler logic circuitry.

Evoked response latencies were determined according to Diamond's technique, as described above, from data sets consisting of averaged steady-state VER records for each of 4 stimulus frequencies. Regressions were calculated for each set of peaks appearing across all of the ISI's used, according to the method illustrated in Figure 1, producing a response latency for each time sample. Latency values in temporally-successive samples were then plotted against time.

D. RESULTS & DISCUSSION.

EXPERIMENT I

The records for all subjects showed one or more of three problems in some degree. First, the data was often very "noisy", containing high amplitude activity at frequencies much above those of the following responses, which were the activity of interest. Where present, this noise partially or completely obscured the following responses.

A further complication was the appearance of "double" positive or negative components through some or all ISI's of many records, and the presence of poorly-defined, broad peaks of low amplitude. The presence of these double peaks was not remedied by including more repetitions in an average. This tactic merely decreased the level of noise in the records, to reveal both sub-components more clearly without reducing the amplitude of, or favoring, either. That is, the duplicity of those waves appeared to reflect some real process inherent in the subject's response to the stimulation. The record for 3 - 5 seconds after the beginning of light adaptation, shown in Figure 7b, is typical of these responses. The double-peaked waves were interesting in themselves, and were investigated in an experiment previous to the ones reported here. The phenomenon will not receive further attention here, however, since it does not appear to be directly pertinent to these experiments on light adaptation.

Unfortunately, there was no consistent set of rules for deciding which of a pair of peaks, or which point on a broad, low wave, to use for peak latency determination that could be applied uniformly to all data sets without producing some unsatisfactory results. For example, the decision rule for determining the latency of a broad, low component, or wave, ought to be applied equally to all other positive and negative waves. Yet, the employment of such a rule often yields unacceptable peak determinations for waves of triangular shape which do have well-defined peaks, locating the rule-consistent "peak" on the shoulder of the wave, rather than at its highest point. Similarly, a rule indicating the selection of the single highest point on a wave as the peak latency was inappropriate where that point reflected only random, noise activity in no relation to the stimulus frequency. Figure 2 illustrates some of these difficulties. A set of rules was selected and applied, however, which least distorted the data. These decision criteria are listed in Appendix I.

The third problem involved an apparent incongruity between records for ISI's around 40 ms, or longer, and those of 35 ms or less. Since the ISI's used in Experiment I were 30, 35, 40 and 45 ms, many data sets appeared to reflect two quite different latencies -- one for the 40 and 45 ms records, and another for responses to the faster rates of stimulus presentation. An example of such a data set is shown in Figure 3. These data, which could not be used here, index an interesting problem of

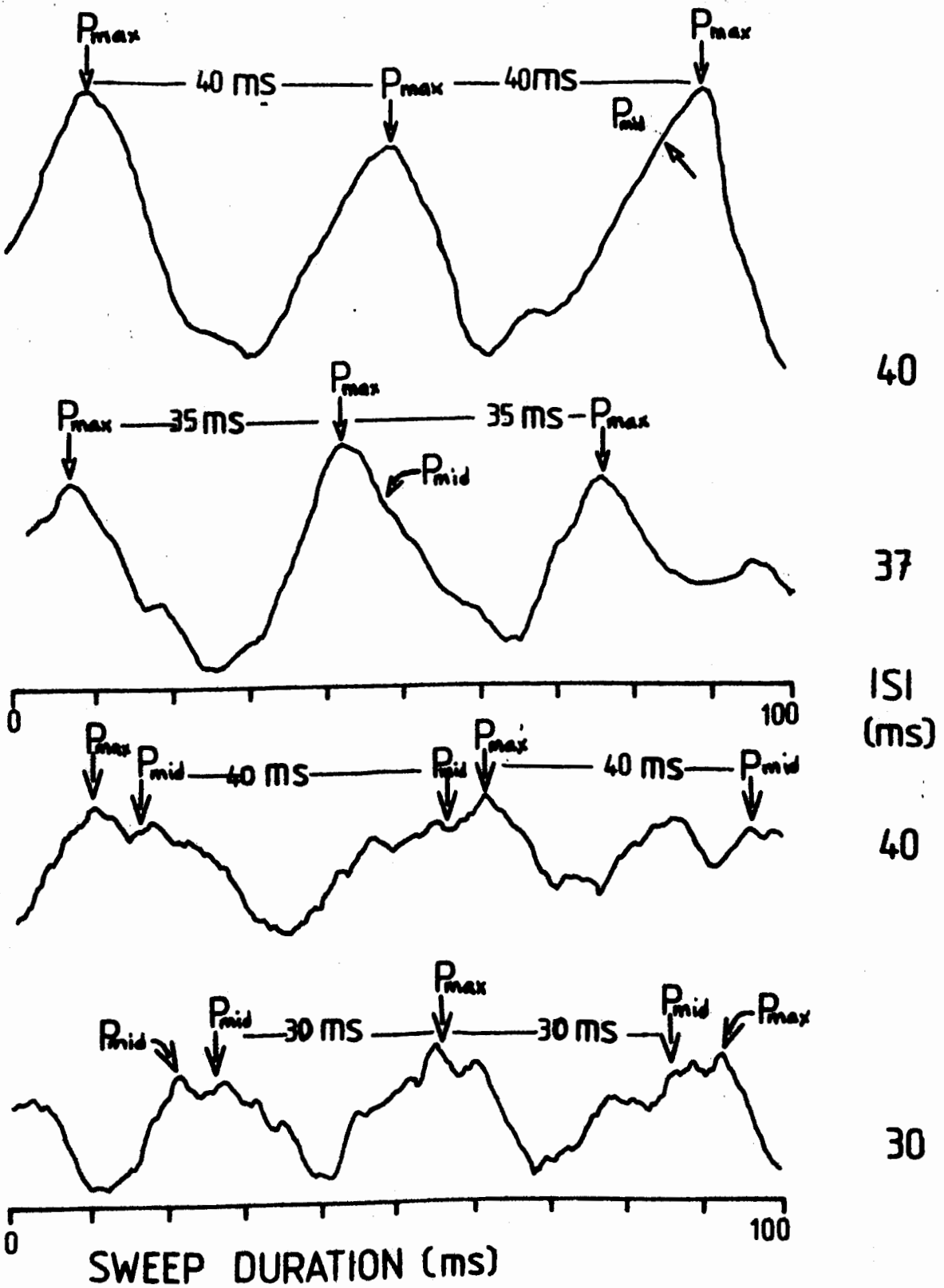


FIGURE 2. LATENCY DETERMINATION PROBLEMS.
P_{max}: highest point on wave, P_{mid}: midpoint of wave.

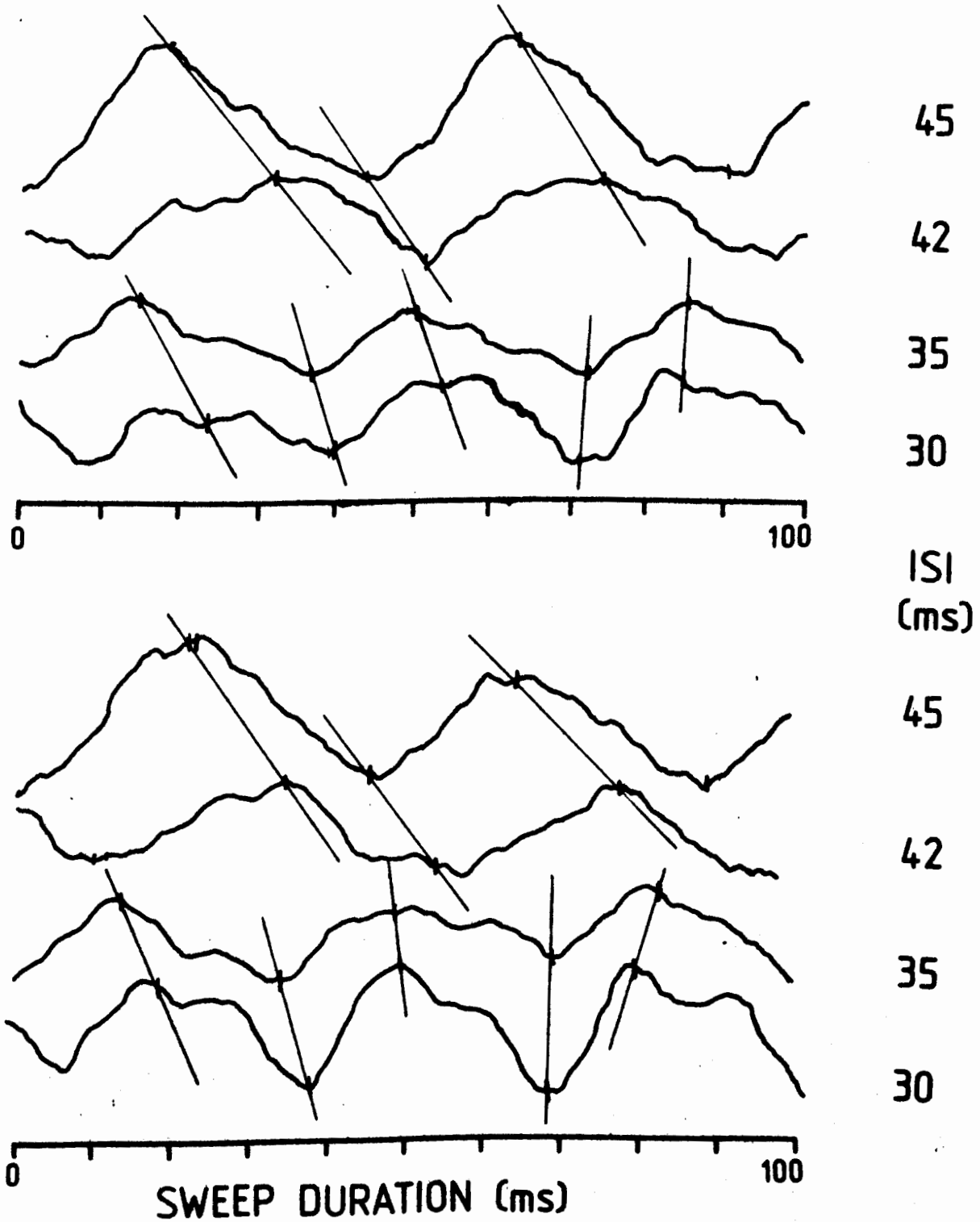


FIGURE 3. Two samples of data in which evoked responses appear to reflect different latencies at short (30-35 ms) and long (42-45ms) ISI's.

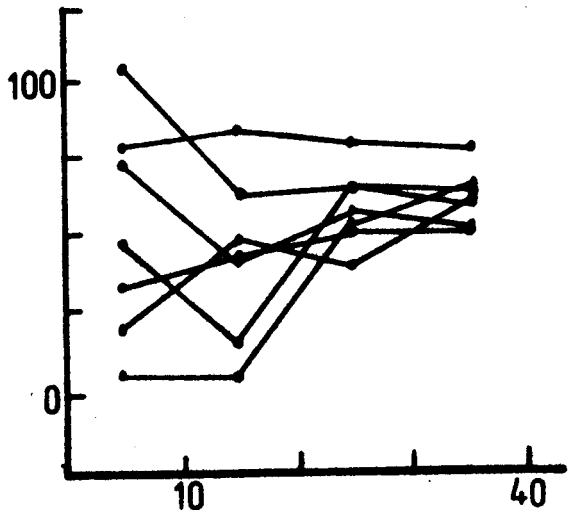
undetermined importance for the experimental questions of this study.

Data were obtained from 7 subjects in the first phase of this experiment (first 43 s of light adaptation), and from 3 subjects in the second phase (which recorded 6 minutes of light adaptation). In the first part of Experiment I, four of those subjects contributed one data set each; 2 sets were obtained from 2 other subjects; and the sixth subject (the experimenter) produced 5 usable records. One of the subjects in the second phase of Experiment I participated in two of the long sessions required for the 6 min light adaptation data. Six of the thirteen data sets for the first 43 s of light adaptation had to be discarded for one or more of the reasons indicated above; all four of the 6 minute data sets were usable.

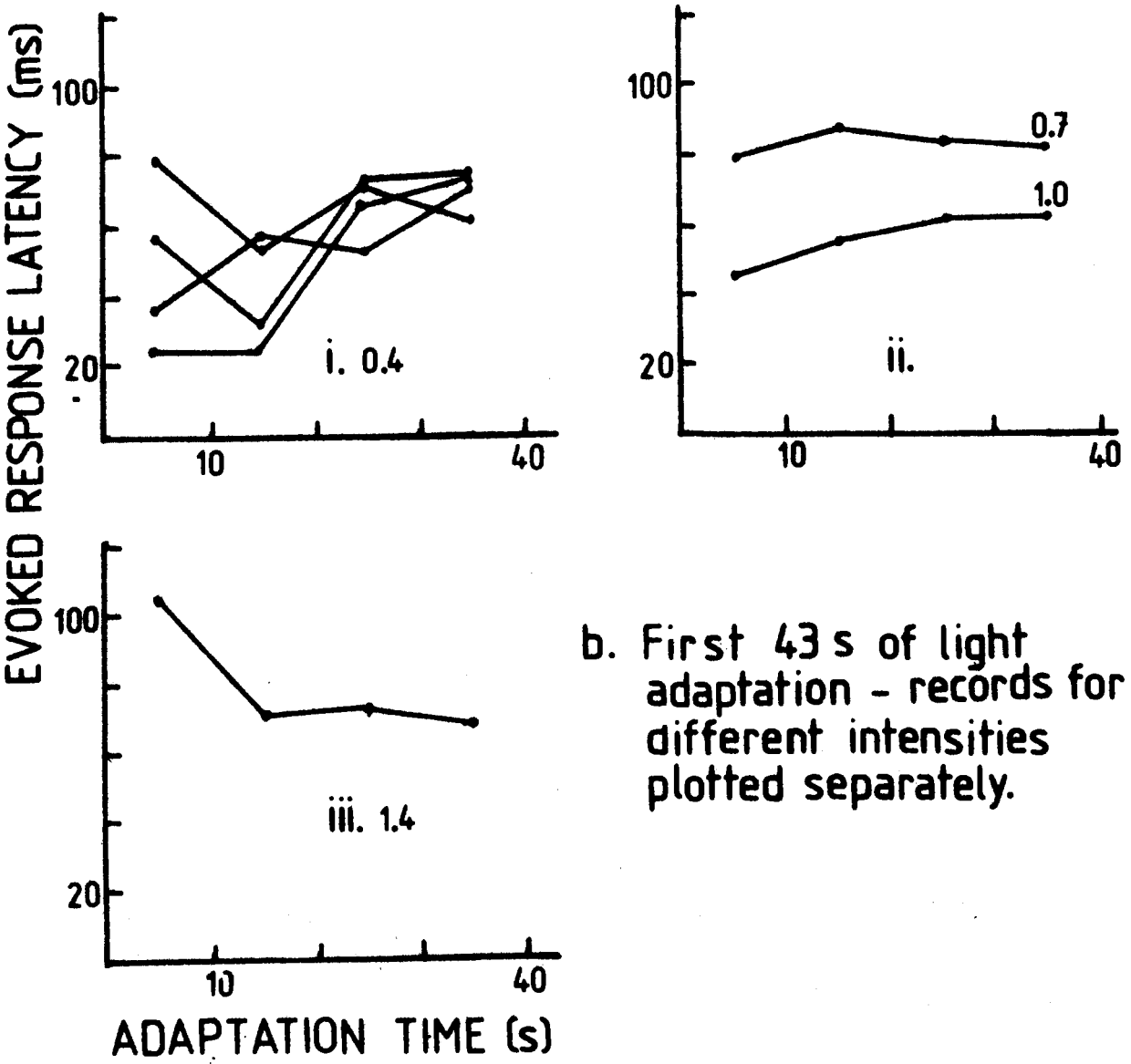
The results of the first phase of this experiment (first 43 s) are shown in Figure 4a & b, and the latency values are presented in tabular form in Table I of Appendix II. As can be seen, there is little evidence of any consistent rate or direction of latency change. While there is a slight increase in the average latency of response across adaptation time, the range of values observed at any single sample time far exceeds the average latency difference between sample times.

In the second part of this experiment, in which adaptation is followed for 6 minutes, there is again an increase in latency which can be seen to peak around 40 s, and to recover almost completely within 1 to 2.5 minutes. Here, too, is a greater

FIGURE 4.



a. First 43 s of light - adaptation - records for all stimulus intensities.



b. First 43 s of light adaptation - records for different intensities plotted separately.

range of values observed within sample periods than between time samples (Figure 5). Table II of Appendix II shows these results.

It was often difficult to derive a latency for the first 10-second sample, which almost invariably exhibited one or more of the above-mentioned problems characteristic of this data. The averaged responses for this portion of the data usually failed to show well-defined positive and negative peaks, while the latencies that were derived therefrom showed the greatest range in values of any sampling period, extending over almost 80 ms from 25 ms to 104 ms.

An hypothesis which could account for both of these effects is that a rapid shift in peak latencies occurs in the first 10 to 20 s of light adaptation. The way in which such a pronounced latency shift would affect this data is described below.

Steady-state AERs are usually obtained by averaging responses across time. That is, successive stimulus-locked portions (sweeps) of an ongoing electroencephalographic recording are averaged together to produce a record which is well-resolved, or relatively free of EEG noise (EEG activity which is random with respect to the stimuli, and which therefore sums to zero during the averaging process). This averaging method is most satisfactory for any response of constant latency with respect to the stimulus. It is here termed "averaging across time".

In techniques which average across time, however, any significant peak latency "jitter", oscillation, or shift produces

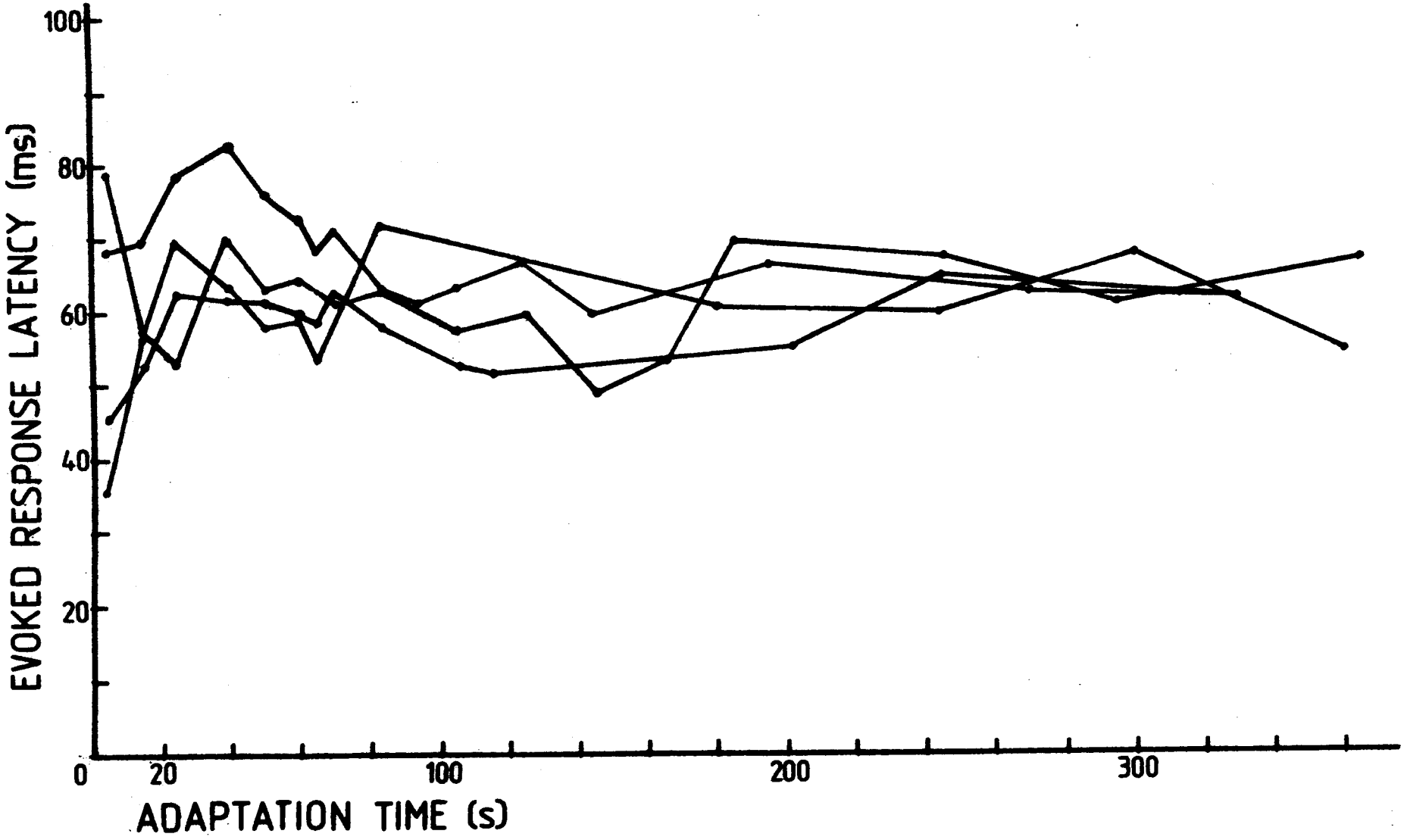


FIGURE 5. VER latency change during 6 minutes of light adaptation.

"mushy" data with low amplitude, indistinct components rather than well-defined peaks with readily determinable latency values. Less important for the latency study here, but of interest in any extension of this inquiry, is the fact that response amplitude of a steady-state VER can be expected to decrease more or less in proportion to the extent of latency shift. The extreme case of 180 degree phase shift of a set of positive-negative deflections within the averaged series would yield zero amplitude, and no peaks for latency determinations in the averaged record if the constituent individual responses maintained constant voltage throught the duration cf the sample period.

In a steady-state VER study of light adaptation, where a latency shift may reflect the visual system's response, averaging across time can be expected to yield poor results. This must be especially true of the first one or two seconds of adaptation, where other physiological approaches have demonstrated rapid changes in response.

In order to investigate this hypothesis of rapid VER latency shift in the early part of light adaptation, a second experiment was performed in which the sampling period was of only 3 s duration. The same interval of 1 s between samples was retained, so that in Experiment II only 15 seconds was covered by the first 4 successive records. A data set again consisted of 4 averaged records at different ISI's. The ISI's used (28, 31, 34 and 37 ms) were selected sc as to obviate the third problem in latency determinatien encountered in Experiment I, by avoiding lower stimulus frequencies.

EXPERIMENT II

The data from this experiment did not yield evidence of a consistent functional light-adaptation effect on VER latency. Records from 2 subjects showed a latency decrease during the 15 s period of observation. The records for 2 other subjects showed no decrease, and even some increase in VER latency (see Figure 6a).

Eight sets of data were obtained from a fifth subject over as many recording sessions to reveal the extent to which within-subject variability of response from one experimental session to another might contribute to the observed lack of consistency in that data. As can be seen in Figure 6b, these records displayed considerable variability both in the latencies at any one time during the observed portion of the light adaptation process, and in the functions obtained for the entire recording period. The range of values obtained within these sampling periods was considerably less, however, than was found in Experiment I. This improvement suggests that the modifications embodied in this second experiment are of the right sort, and that further changes of experimental procedure in the direction of decreasing sample time may produce cleaner and more consistent data.

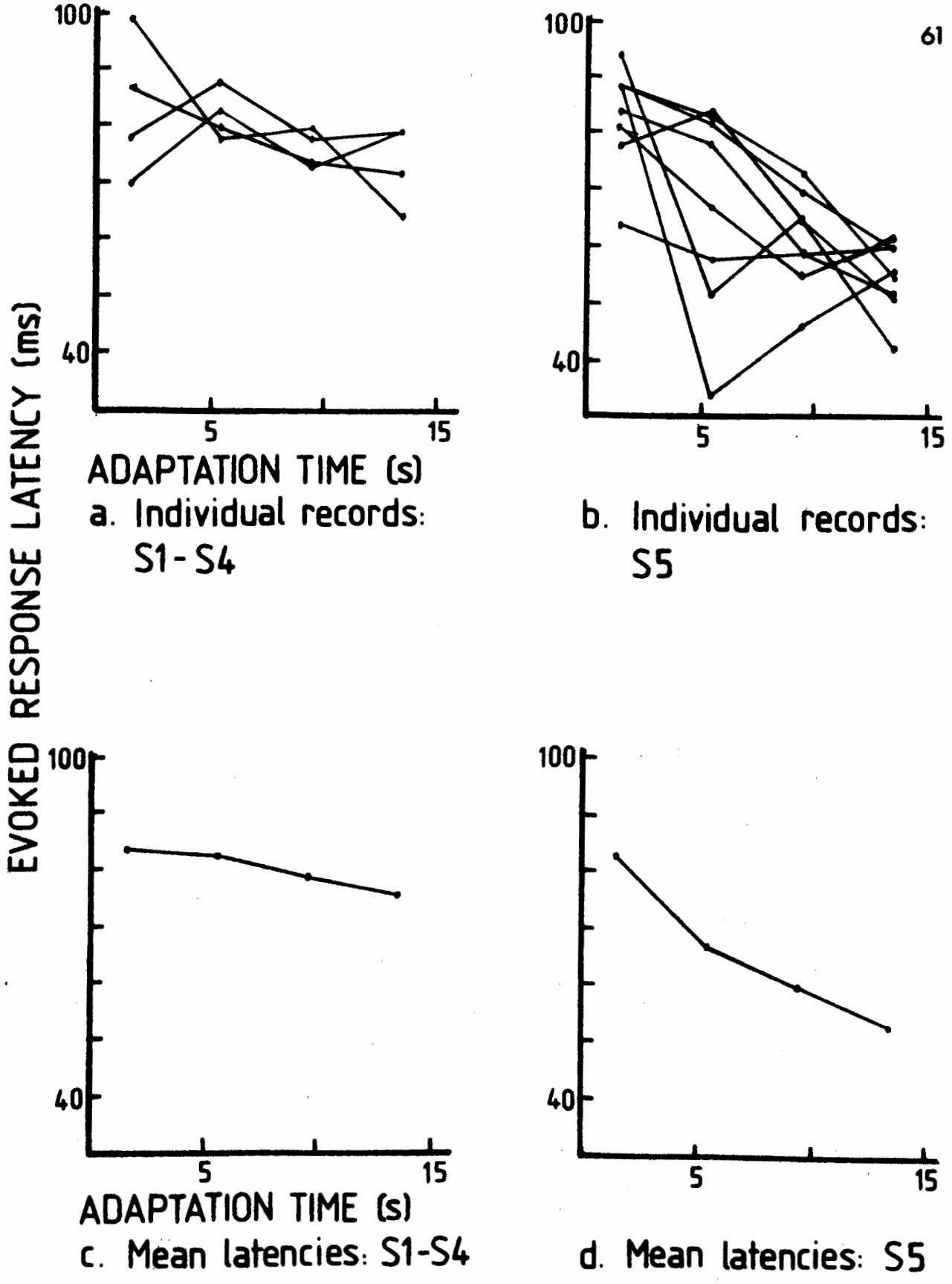


FIGURE 6. First fifteen seconds of light adaptation.

Moreover, if average latencies are constructed for each 3 s recording period, either for the data set for Subject 5, or for the data from the first 4 subjects, there is in both cases a decrease in VER latency as a function of light adaptation time (Figures 6c & 6d). The still-considerable range of latency values within any single recording period and of functions across this initial 15 s of adaptation mitigates, however, the formulation of any strong statement about the observed effect for average values. (These data are tabulated in Table III.)

Some further data was obtained as part of Experiment II, employing still shorter sampling periods with 2 subjects whose VER's in this experiment had been characterized by, in one case, by double peaks and, in the other, very low amplitude components lacking definite peaks. Data at a single ISI (31 ms) are shown for the first of these 2 subjects in Figure 7. Clearly, as the sample duration increases from 1.5 through 2 to 3 s, there is increasing duplicity in the responses.

Records from the second subject showed no measurable peaks when a sampling period of 3 s was used. Limiting recording time for each sample to 2 s and, in a second session, to 1.5 s produced data sets from which latencies could easily be derived (Figure 8). In these records a marked phase shift (and, hence, a latency change) occurs from the first to the second sample in the 1.5 s data. The time encompassed by these two successive samples and the interval between them is 4 s, slightly longer than the

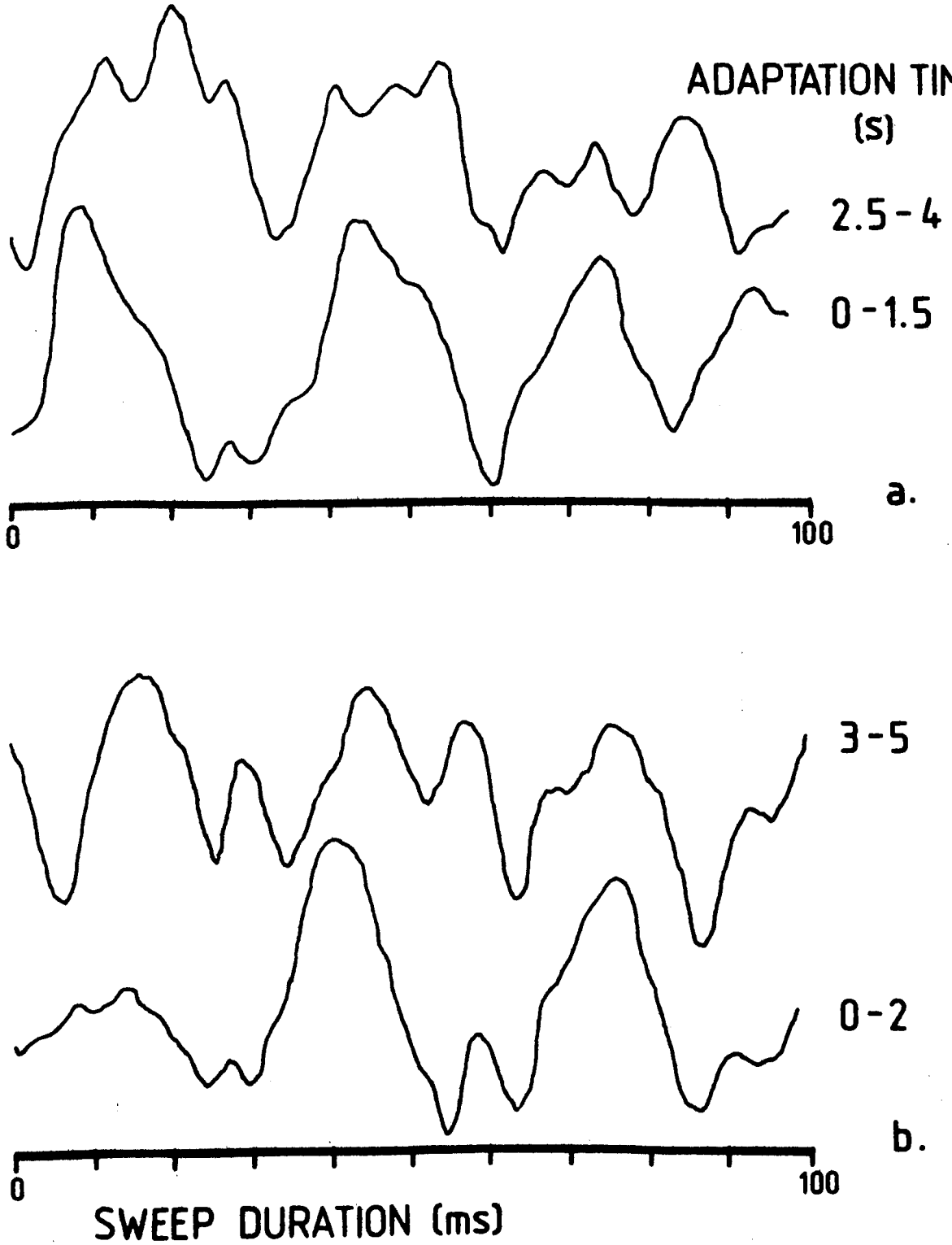


FIGURE 7. Emergence of "double" peaks during the course of light adaptation: a. 1.5 s sample,
b. 2 s sample.

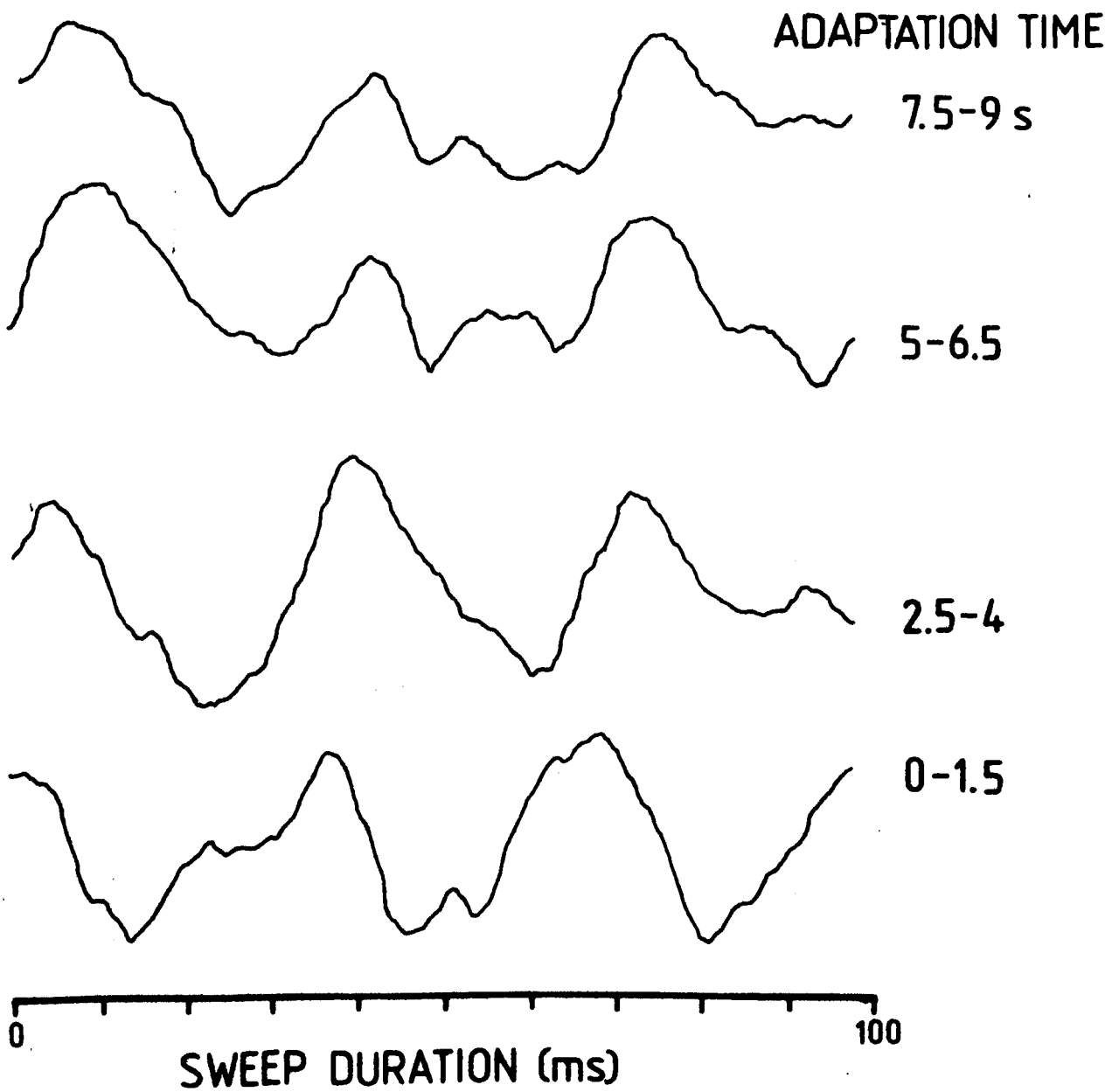


FIGURE 8. Marked phase shift of evoked response components from first (0-1.5 s) to second (2.5-4 s) sample during light adaptation (ISI = 34 ms).

duration of a 3 s recording period. Such a phase shift might well account for the poor quality of this subject's data when the longer sample time of 3 s was used.

E. CONCLUSIONS.

These experiments yielded no conclusive results. While there was some evidence of an initial large decrease in VER latency during the early phase of light adaptation (here, 43 s), followed by a slight recovery, this effect accounted for too little of the variability that was present in the data to afford confidence in the representativeness of the values obtained, or in any interpretation of these results.

Our hypothesis, that some of this variability was due to rapid VER latency shift, received some support from the results of Experiment II. This latter experiment, which investigated the first 15 s of light adaptation in more detail, provided stronger evidence of an initial latency decrease.

The information provided by Experiment II was not sufficient to permit a strong statement about light adaptation on the steady-state VER. It does indicate one reason for the paucity of good studies of light adaptation using VERs (transient or steady-state). The phenomenon is not especially amenable to measurement with the conventinal, on-line averaging systems which are appropriate for most other VER measures. The results of the

experiments reported here suggest an appropriate modification of research strategy for the successful study of light adaptation with the steady-state VER. An averaging technique which conveniently permits the sampling of very brief periods, very close together, especially during the first 0.5 to 1 minute of adaptation, will better reveal the changes in steady-state evoked response latency, if any, that occur during light adaptation.

I propose that an averaging technique similar in some respects to that used for transient VER's should be used with steady-state stimulation frequencies to provide averaged VER records, from which response latency can then be determined using Diamond's technique, as described above. Rather than averaging successive responses within a continuous EEG record, data would be stored in raw form on FM tape. Any number of continuous records of evoked responses throughout a light adaptation period (of whatever duration is considered interesting by the experimenter) could be collected. Our pilot work suggests that as few as thirty records need be obtained for clear, noise-free data when the response of interest has a reasonably stable latency. One can then use an appropriate computer program to recall temporally identical portions of the records, and to average those sweeps together. Put another way, an average would be constructed using the first sweep (for example, the first 100 millisecond segment) from each record; then, a second average from the second sweep of each record, and so on throughout the entire recording.

This approach would provide averaged VERs at steady-state frequencies, which could then be used in Diamond's latency determination program to follow changes in VER latency at a known cerebral locus in considerable temporal detail throughout the course of light adaptation. The use of Diamond's technique allows this study to be extended to compare activity at several brain loci with multiple monopolar electrode placements over occipital and other cortex, as well as the obvious experimental manipulations of test flash intensity and background luminance.

The more important methodological and technical considerations involved in studying light adaptation using visual steady-state evoked potentials have been discussed, and an averaging technique has been described which should obviate some problems associated with the usual averaging method used in obtaining evoked potentials.

A computer program has yet to be written which will carry out this averaging procedure satisfactorily. The extensive DECUS program library for DIGITAL's PDP line of computers contains no programs which will compute averages according to the specifications indicated above. In particular, it is important in this study to obtain a set of averaged evoked responses which represent the temporal course of light adaptation in maximal detail, in order to make valid comparisons with other electrophysiological measures of peripheral processing and with psychophysical reports of the course of sensory change.

The experimentation procedures recommended will, no doubt, be tedious; but, should yield important new information about neural events at the cortical level during the course of light adaptation. Such information should help bridge the present gap between electrophysiological and psychophysical studies of the process of light adaptation.

APPENDIX I: PEAK IDENTIFICATION PROCEDURE.

1. If a set of waves in one polarity is, on the average, the distance of the ISI between peaks, and if the distance between any two peaks is neither more than 130% nor less than 70% of the ISI, determine latency values for that set of peaks according to Rule 3, below.

2. If a wave is "double" (e. g., has two positive subpeaks), determine latencies for peaks in that polarity only if the wave of opposite polarity (i. e., small negative wave) between the two subpeaks is less than 35% of the maximum amplitude from highest point on (positive) wave to lowest point on adjacent (negative) wave.

3. a. Determine full amplitude of wave from its highest point to the lowest point on the nearest adjacent wave of opposite polarity.
   b. Determine 30% of that amplitude.
   c. Peak latency is the mid-point of that wave, 30% of the distance from its highest point.

APPENDIX II: TABLES OF VER LATENCY VALUES.

TABLE I: VER LATENCIES (ms) FOR FIRST 43 SECONDS OF ADAPTATION

LIGHT ADAPTATION TIME (s)	STIMULUS INTENSITY (neutral density filter)						
	0.4	0.4	0.4	0.4	0.7	1.0	1.4
0 - 10	58	78	35	25	80	46	104
11 - 21	32	53	58	25	88	55	71
22 - 32	74	70	51	76	84	62	73
33 - 43	75	61	68	67	82	63	68

TABLE II: VER LATENCIES (ms) DURING 6 MINUTES OF ADAPTATION

LIGHT ADAPTATION TIME (s)	SUBJECT			
	1	2	3	4
0 - 10	79	69	46	36
11 - 21	57	70	53	57
22 - 32	53	78	63	70
33 - 43	60	83	62	64
44 - 54	63	77	62	58
55 - 65	65	74	59	59
60 - 70	61	68	58	53
66 - 76	65	72	64	60
77 - 87	61	64	57	72
90 - 100	--	62	--	--
100 - 110	58	64	53	--
110 - 120	--	--	52	--
120 - 130	59	67	--	--
140 - 150	48	--	--	--
120 - 170	--	60	--	--
160 - 170	51	--	--	--
160 - 210	68	67	--	--
170 - 220	--	--	--	61
180 - 230	--	--	55	--
220 - 270	67	--	65	60
245 - 295	--	63	--	--
280 - 330	60	--	--	68
305 - 355	--	62	62	--
340 - 380	64	--	--	54

TABLE III: VER LATENCIES FOR FIRST 15 SECONDS OF ADAPTATION

LIGHT ADAPTATION TIME (s)	SUBJECT: INDIVIDUAL RECORDS												MEANS	
	1	2	3	4	5a	5b	5c	5d	5e	5f	5g	5h	1-4	5
0 - 3	78	98	70	86	94	88	88	88	83	81	78	63	83	83
4 - 7	87	67	82	79	51	83	81	33	78	67	85	57	82	67
8 - 11	77	69	73	73	64	73	69	46	58	55	64	58	78	60
12 - 15	78	53	78	71	42	54	60	56	60	62	51	52	76	53

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