The dazzle reflex: electrophysiological signals from ocular muscles reveal strong binocular summation effects

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Abstract
Under dark adapted or dim conditions the mammalian visual system is carefully programmed to respond rapidly to the sudden onset of bright lights. This response, called the dazzle reflex, is controlled from sub-cortical structures of the brain. It is known anecdotally that exposure to a bright light when dark adapted induces an instinctive closure of one eye to reduce the pain associated with dazzle. This binocular summation of the dazzle response has not previously been reported. The dazzle reflex can be measured in human subjects by recording the electrical activity from surface electrodes located near the muscles around the eye. In this paper we report an investigation of the apparent binocular summation of the dazzle reflex using this technique. The data reveal a clear difference between monocular and binocular stimulation, with the binocular response being much larger than the monocular response. Furthermore this monocular/binocular difference arises only if the stimulus duration is longer than approximately 1 s. These observations are interpreted in terms of the known physiology of blink mechanisms.

Keywords: binocular summation, blink mechanisms, dazzle reflex, discomfort glare, electrical signals, night driving, orbicularis muscle

Introduction
Sudden exposure to a bright light under dim, or dark-adapted, conditions induces a strong spasm of the extraocular muscles in all mammalian eyes. This primitive ‘dazzle reflex’ is likely designed to reduce the intensity of light incident on the retina and is thought to arise in the sub-cortical structures of the brain (Grant, 1945; Ongerboer de Visser and Kuypers, 1978; Hackley and Johnson, 1996; Burke and Hackley, 1997). The response is accompanied by discomfort of varying degrees of intensity, ranging from a mild sensation of ocular stress to extreme pain. It is now established that the presence or absence of this pain depends on the intensity of the glare source and the level of adaptation of the eye (Hopkinson, 1956; Howarth et al., 1993; Berman et al., 1994; Kim and Koga, 2004). When fully light-adapted, little or no dazzle occurs, but in the dark or partially dark-adapted state, a strong dazzle reflex is generated by lights of only moderate brightness. The effects are most commonly experienced when driving at night, when the headlamps of oncoming vehicles induce strong and frequent spasms of the muscles around the eye and face (Murray et al., 2002).

The precise physiological origin of the pain remains obscure. Hopkinson (1956) speculated that it might be related to the opposing (antagonistic) actions of the dilator and sphincter muscles, attempting to simultaneously constrict and dilate the pupil. This idea was further explored by Fry and King (1975) who thought that the presence of glare induces changes in the power spectrum of ‘hippus’, which is the rhythmic involuntary fluctuation of pupil size under steady lighting conditions. However, Howarth et al. (1993) recorded no differences in the power spectrum of hippus with and
without glare and therefore ruled out the role of the pupil.

Although the iris is well invested with pain receptors, it seems more likely, as argued below, that the extra-ocular muscles contribute to glare-induced pain. Eye closure is primarily the responsibility of the orbicularis oculi (OO), a substantial sphincter-like muscle, which surrounds the eye. Many studies (e.g., Manning and Evinger, 1986) have shown that a blink is a complicated two-stage process, involving the co-ordinated action of the orbicularis and the levator palpebrae, in the upper eyelid, which is responsible for maintaining the eye open. Immediately prior to a blink, the activity of the levator is reduced and this is accompanied by a burst of action potentials in the underlying muscle fibres and the peak in the amplitude reflects high frequency motor-neuron activity (Evinger et al., 1991). Moreover, recent studies (Berman et al., 1994; Murray, 1999; Murray et al., 2002) have linked EMG activity of the OO or other nearby muscles, e.g. the corrugator supercilli, to discomfort glare. Specifically, Murray (1999) suggested that the EMG activity induced by a bright light is approximately linearly related to the logarithm of the illuminance incident on the cornea. Murray et al. (2002) showed the amplitude of the EMG to a light stimulus of particular intensity to be closely related to the subjective response to the same stimulus. In other words, the EMG not only provides an index of the extra-ocular muscle activity, but it may also give an objective estimate of the severity of discomfort due to a bright light.

It is well known anecdotally that the discomfort due to a bright light is easily suppressed by instinctive closure of one eye. Moreover, it has been reported that binocular brightness summation occurs in the presence of large targets of high intensity (Bourassa and Rule, 1994). To investigate why lights might appear brighter under binocular than monocular viewing, we have monitored the change in electrical activity of the extra-ocular muscles induced by a glare source. We find that the binocular stimulus generates a much larger signal than that obtained under monocular conditions. From pilot studies we have established that this summation effect depends primarily on three stimulus parameters: stimulus intensity, stimulus duration and background luminance. Here we present data on the effects of stimulus intensity and duration.

Methods

Subjects

EMG responses were elicited in six subjects, aged 24–49. All subjects had normal vision and no ocular or neurological pathology. No glasses or contact lenses were used. Three of the subjects (SP, IJM, LG) had participated previously in eyelid recording experiments.

Procedure

The glare source was a projector fitted with an 150 W tungsten lamp. A diffuser was placed in front of the lamp. The size of the stimulus was 3 cm in width by 2 cm in height, located at a distance of 1 m directly in front of the subject’s eyes, providing a field size of $1.7^\circ \times 1.15^\circ$. The intensity of the stimulus was varied with the use of neutral density filters. The stimulus onset and the start of EMG data sampling were electronically timed to be simultaneous. Glare source intensity was expressed in terms of horizontal corneal illuminances (measured with a lux metre) ranging from 22 to 5850 lux. The duration of the stimulus was varied but for most experiments was 2 s.

The subjects were seated on an adjustable chair with the head maintained in a constant position with the aid of a chin rest. They looked straight toward the glare source. They were dark adapted for 3 min prior to each recording session. All room lights were extinguished during the experiment. The glare stimulus was viewed through natural pupils. For monocular trials an eye patch was placed over the left eye. Each subject participated in a single session lasting approximately 75 min.

Recording the EMG

The EMG responses from the peri-orbital muscles were recorded bipolarly from the right eye using two 9 mm surface Ag–AgCl electrodes, positioned one below the lower eyelid and the other lateral to the eye at the temporal margin, as close as possible to the lateral canthus. A third electrode placed on the forehead served as a ground. Before the electrode application, the skin was cleaned with alcohol. Electrodes were attached with conductive paste; electrode impedance was maintained below 6 kΩ at all times. The techniques for analysing the EMG have been described previously (Murray et al., 2002). Briefly, signals from the electrodes were amplified simultaneously using either a Medelec Sensor amplifier.

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or a purpose built amplifier and transmitter (the Ocular Stress Monitor, OSM), designed for remotely monitoring EMG activity. The Medelec amplifier was used to test the accuracy and signal/noise characteristics of the purpose-built amplifier, which was much more convenient to use. All measurements were made with this instrument when it was shown to be performing satisfactorily. The Medelec is a commercially available, conventional physiological amplifier designed for obtaining evoked potentials. Its filters were set to 10 and 250 Hz (6 dB/octave) and common mode rejection was 60 dB. The gain could be adjusted between 5 K and 20 K. The purpose built amplifier/transmitter was developed in the Visual Sciences Lab. It had a gain of 5 K. Signals were filtered with a narrow band (20 Hz at half height) time-active filter tuned to 200 Hz. The rationale for choosing this frequency range is fully discussed by Murray et al. (2002).

The EMG records from both amplifiers were digitised with an A/D converter (CED 1401: Cambridge Electronic Design Ltd., Cambridge, UK), which in turn was interfaced with a PC. The EMG activity was sampled at 512 Hz over a 4 s epoch (2048 data points), which was triggered by the stimulus onset. A recovery period of at least 3 min was allowed between each trial. Three measurements were obtained for each level of stimulus intensity. Stimulus intensity was tested in turn starting from the lowest and ending with the highest values. At the beginning and the end of each session the spontaneous EMG activity, i.e. the ‘noise’, was sampled for 4 s with the subject in a relaxed state, but with eyes open.

**Data analysis**

A PC and the software from the CED 1401 were used to store the data for later off-line analysis. The recorded EMG from both amplifiers (see left panel, Figure 1) was rectified and 5-point smoothed. In preliminary experiments, the integral of the rectified signal was calculated as an index of EMG activity. Note that these signals were recorded simultaneously from the same electrode.

Using this time domain analysis, the precise quantitative contribution of low-frequency artefacts, such as blinks, to the surface EMG signal can be difficult to assess. A further disadvantage is that duration effects are difficult to evaluate. For example, visualisation of the change with time of the response after stimulus onset can be important under some conditions. In preliminary studies, we have found the response to be sensitive to the duration of the stimulus in a complex manner and this could not be studied using time domain analysis. Hence in order to provide flexibility, the data were also analysed in the frequency domain.

Figure 1 shows the data plotted in the time domain (left panel) and frequency domain (right panel). The upper plots are data obtained with the purpose built OSM and the lower plots data obtained with the Medelec amplifier. EMG amplitude spectra were sampled in the 0–256 Hz frequency range with a resolution bandwidth of 0.25 Hz/bin (see right panels). The area (A) under the frequency spectrum [restricted to a range between 180 and 220 Hz, see Equation (1)] was then calculated and used as an index of evoked EMG:

![Figure 1](image-url)

**Figure 1.** (Left) Raw EMG samples of the extra-ocular muscles for a condition of binocular stimulation (corneal illuminance: 2000 lux) as measured with the broad-band Medelec amplifier (lower) and the narrow-band OSM (upper). The glare stimulus (indicated by a horizontal dashed line) had a 300 ms slow onset presentation and was removed after 2 s. (Right) FFT amplitude spectrum derived from the 4 s of the rectified EMG records with the two amplifiers. The integration of the FFT spectrum (grey area) can give an index of discomfort glare. The area calculated was restricted to between 180 and 220 Hz, according to the frequency characteristics of the OSM.
In order to take into account baseline EMG noise, signal-to-noise (S/N) ratios were calculated using the following expression;

\[
S/N = \frac{\sum_{i=180}^{219} S}{\sqrt{\sum_{i=180}^{219} N}},
\]

where \( S \) is the area under the amplitude spectra for binocular or monocular stimulation and \( N \) is the area under the amplitude spectra for spontaneous EMG activity.

Figure 2 compares data obtained from one subject for a range of intensities of the glare source, as calculated using both amplifiers. It is clear that the narrow-band OSM and the broad-band Medelec amplifiers produce comparable results. In the following figures, only data from the OSM amplifier are presented.

Results

Figure 3a illustrates raw EMG samples (each composed of three sets of superimposed responses) recorded for binocular stimulation for subjects SP (left panel) and LG (right panel) for a range of corneal illuminances from 32 to 5850 lux for SP and from 22 to 4200 lux for LG. The corresponding data for monocular stimulation are illustrated in Figure 3b. The stimulus timing, onset for 2 s – offset for 2 s, is indicated by a dashed line at the top of each panel. These time-domain plots enable us to characterise the manner in which the response builds as a function of time after the stimulus onset. They reveal a dramatic increase in the level of electrical activity generated by the muscles as a result of the presentation of the stimulus. It is evident that the EMG undergoes a series of spasms of around 0.2 s duration, with the first spasm appearing about 250 ms after stimulus onset. After the stimulus has been turned off, there is a gradual decrease in muscle activity. It is also apparent that the ‘recovery’ time is slower for higher intensities of the glare source. Another crucial observation is that for binocular stimulation (Figure 3a) there is a systematic increase in the magnitude of the response as corneal illuminance is increased. Surprisingly, there is no such increase in response for monocular stimulation (Figure 3b). Note that all subjects reported feeling little discomfort for monocular stimulation even at the highest illuminances. The subjective rating was more thoroughly examined in a previous study (Murray et al., 2002).

The dramatic difference in the EMG response between monocular and binocular stimulation can be generalised to all subjects, although there is a significant inter-subject difference in the absolute magnitude of EMG response. Figure 4 presents individual S/N ratios for the two conditions as a function of corneal illuminance. A value of one indicates a signal with no difference to the baseline measure. Two points should be noted from the data: first, for some subjects (LG, SP in particular), a linear relationship between S/N ratios and the logarithm of corneal illuminance is obvious. In others the response is a more complex function of retinal illuminance, but in all cases there is a significant increase. Second, there are signs of a saturation of the response at high levels of stimulus intensity under monocular, but not under binocular stimulation. We have not investigated the mathematical link between stimulus intensity and response in this figure because the main point is to highlight the difference between the monocular and binocular responses.

A two-factor ANCOVA was performed on these data (dependent variable: area of the frequency spectrum of EMG activity) using corneal illuminance as a co-variante (this is justified because it is known, a priori, that glare sensitivity increases with target luminance), with method of stimulation (MS, binocular vs monocular) and subjects (S) as factors. Significant effects of interactions between all factors were found \( F (5, 155) = 2.513, p < 0.032 \). Moreover, when a one-way ANOVA was performed with method of stimulation as the factor, the difference between monocular and binocular stimulation was highly significant \( F (1, 35) = 51.49, p < 0.001 \).

Figure 5 displays average monocular (filled symbols) and binocular (open symbols) data for all six subjects in
the form of S/N. The error bars reveal ±1 S.D. Again, it is apparent that the EMG increases for higher corneal illuminances with the effect being more pronounced for the binocular stimulation. Note that the stimulus appears much less intense monocularly: the difference in responses for the two conditions is such that 200 lux of binocular stimulation is equivalent to 5000 lux of monocular stimulation. This means that the light intensity to produce glare under monocular conditions has to be an order of magnitude greater than under binocular conditions.

As indicated above, a critical factor in the link between EMG activity and glare source intensity, is the duration of the glare source. Figure 6 depicts S/N for a range of stimulus durations at different corneal illuminances for one subject. The left-hand panel shows responses to monocular conditions and the right-panel responses for binocular conditions. It is evident that in

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**Figure 3.** Superimposed raw EMG samples (three sets in each case) of the extra-ocular muscles for a range of corneal illuminances under (a) binocular and (b) monocular (ipsilateral) stimulation. The glare stimulus has a 300 ms slow onset presentation and is removed after 2 s. The left panels present data from subject SP, while the right panels data from subject LG. The top traces show the noise obtained (mean of three trials).
both cases there is an increase in EMG for the longer presentation time. For the 3 s duration stimulus the slope is much steeper for binocular than for monocular viewing as would be expected from the data in Figure 5. The main point here is that for the monocular stimulation there is no difference in S/N between the 1.0 and the 2.0 s stimulus duration for the whole range of intensities, suggesting that the 2.0 s duration is the ‘threshold’ for ocular stress for this particular subject and the experimental conditions used here. We did observe this pattern in other subjects, but we have insufficient data for including them in the figure. On the other hand, in binocular viewing, the ‘threshold’ for duration increases as S/N increases markedly as duration increases.

Discussion

Excessive brightness in the field of view, when dark-adapted, can induce considerable discomfort, ocular stress, and pain, depending on a variety of factors, such as the intensity and duration of the stimulus and the

Figure 4. Individual plots of variation of the signal-to-noise ratios (S/N) with corneal illuminance for binocular (open symbols) and monocular (filled symbols) stimulation. Data from six subjects are shown. Each data point corresponds to the mean of three responses. The index of discomfort glare is calculated as shown in Figure 1b.

Figure 5. Average S/N (six subjects) as a function of corneal illuminance under binocular (open symbols) and monocular (filled symbols) stimulation. The bars indicate ±1 S.D.
adaptation state of the observer. In previous papers we have described an electrophysiological method of characterising this ‘dazzle reflex’, based on the EMG generated by the orbicularis muscle. In this paper, we describe the application of this technique to investigate dazzle when it is induced under either monocular or binocular conditions. The main finding is that binocular stimulation generates a much larger response than monocular stimulation. Although it may be well-known anecdotally, this summation effect has not to our knowledge been reported previously. Effectively, our data confirm the subjective experience; the light intensity to produce extreme dazzle under monocular conditions has to be an order of magnitude greater than under binocular conditions. This effect has been repeated on six subjects and corresponds to everyday experience; when dark adapted (or in extreme conditions such as looking toward the sun) a bright light induces the instinctive closure of one eye, thus immediately suppressing the sensation of dazzle.

One possible explanation for the pain and discomfort associated with dazzle, is that it may originate in the pain detectors in the extra-ocular muscles. It is known that in order for a blink or an ocular flinch to occur, the activity in the levator muscle (which raises the upper lid) must be momentarily suppressed (Evinger et al., 1991; Manning and Evinger, 1986). The presence of high-intensity lights, especially of prolonged duration, may reduce the ability of the two muscles to co-ordinate, thus forcing them to contract simultaneously and induce pain.

**Different components of eyelid response**

The binocular summation effect occurs only if the stimulus is present for more than 1 s. Studies of the Photic Blink Reflex (PBR) have shown it to have two different components, an initial burst of action potentials, the R50, that begins at 40–90 ms (depending on the intensity of the light flash), followed by a second burst, the R80, that begins at 70–130 ms and persists for at least 100 ms (Mukuno et al., 1983; Anthony, 1985; Hackley and Johnson, 1996). The first has a short duration and a fast rise time, while the second shows a slower rise time and a considerably longer duration. These different temporal characteristics suggest they may represent the activity of fast (transient) and slow (sustained) populations of fibres in different regions of the orbicularis muscle (McLoon and Wirtschafter, 1991). Manning and Evinger (1986) investigated the neural basis of blinks evoked by light stimuli by recording the EMG in the OO and levator palpebrae (LP) of rabbit. They also described blinks as a two-step process. First, in response to initial stimulation, a burst of OO activity of a preset frequency and duration is programmed in advance of the lid movement. The reflex arc mediating this early component originates in the cerebellum and the motoneurons of the third nerve nuclei. The initial volley of activity is based on the intensity of the initial portion of the stimulus. Increments in stimulus duration increase the intensity of this activity, but not its duration, causing it to increase in size. When the stimulus reaches a critical duration, the blink-generating neurons either switch their mode of action or a second population of fibres is recruited. Either way, rather than increasing the frequency of OO motorneuron activity and generating a larger peak amplitude, the duration of activity increases. This results in a regular lengthening of blink duration while increasing only slightly the amplitude of the signal.

Burke and Hackley (1997) suggested that R50 triggers the start of a blink when an intense onset transient is detected, and then R90 maximises protection by
sustaining lid closure for the duration of the luminance increment. They proposed the startle-dazzle theory, according to which the R50 component is functionally related to startle, whereas the R80 component contributes more substantially to a generalised, sustained, dazzle reflex. The function of the startle reflex, according to the widely accepted theory of Graham (1975), is to interrupt ongoing perceptual and motor processing, automatically ensuring that the organism can better evaluate and respond to the intensity of the evoking stimulus. The R80 component, presumably, is to maintain eyelid closure until adequate pupillary constriction can develop (which would minimise retinal bleaching and temporary blindness), a process requiring several hundred milliseconds (e.g., Beatty, 1986).

Unilateral and bilateral pathways?

The circuitry underlying R50 and R80 has not yet been delineated, but recent evidence (Hackley and Johnson, 1996; Burke and Hackley, 1997) suggests that both components are based entirely on sub-cortical mechanisms. Whether they are mediated by parallel central pathways, or by two serial volleys within a single reflex arc, is not yet known. However, Burke and Hackley (1997) claimed that the fact that the sudden onset of a bright light can excite the OO muscle and trigger a pause in tonic LP contraction suggests the involvement of multiple branching and converging central pathways. How might this explain the binocular summation of the dazzle reflex? It seems feasible that a continuous sustained burst of activity from both cerebellar hemispheres, due to binocular stimulation, could be amalgamated into several components somewhere along the output pathway, giving rise to a strong, sustained dazzle reflex, based perhaps on the slow fibres. The more rapid startle reflex, which would require more precise timing, would give rise to the transient startle reflex and be derived only from a single cerebellar hemisphere and therefore be driven only from one eye. In this way, the sustained activity would be associated with binocular stimulation and the short duration, transient activity, associated with monocular stimulation.

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