Contrast gain control in the cat visual cortex

I. Ohzawa, G. Sclar & R. D. Freeman

School of Optometry, University of California, Berkeley, California 94720, USA

The eye functions effectively over an enormous range of ambient illumination, because retinal sensitivity can be adapted to prevailing light levels\(^1\). Higher order neurones in the visual pathway are presumably more concerned with relative changes in illumination, that is, contrast, because a great deal of information concerning absolute light level is processed at the retinal level\(^2\). It would therefore be of considerable functional value if cells in the visual cortex could adapt their response levels to a steady-state ambient contrast, in a manner analogous to the sensitivity control mechanism of the retina. We have examined here the idea that adaptation of neurones in the visual cortex to ambient contrast is similar to adaptation in the retina to ambient illumination. The experiments were performed by measuring contrast response functions (response amplitude as a function of contrast) of striate neurones, while systematically adapting them to different contrast levels. Our results show that, for the majority of cortical neurones, response-contrast curves are laterally shifted along a log-contrast axis so that the effective domains of neurones are adjusted to match prevailing contrast levels. This contrast gain control mechanism, which was not observed for lateral geniculate (LGN) fibres, must be of prime importance to visual function.

Cats were prepared for single unit recording from the striate cortex by standard procedures described in detail elsewhere\(^3\). Anaesthesia was induced with halothane. A radial vein was cannulated and anaesthesia was continued with sodium thiopental while a tracheal tube and an electrode were positioned. Animals were then paralysed with gallamine triethiodide and artificially respirated using 70% N\(_2\)O and 30% O\(_2\). Sodium thiopental was added as required to maintain anaesthesia. The electroencephalogram, electrocardiogram, body temperature and expired CO\(_2\) were monitored. Isolated spikes from single neurones were amplified and fed into a computer for storage and analysis. Receptive fields were mapped for each unit with light bars and spots, and their ocular dominance and cell type determined using standard criteria\(^4\). The receptive field of the dominant eye was then aligned with the centre of a cathode ray tube screen (substance, 30"x22"; mean luminance, 250 cd m\(^{-2}\)) on which drifting sinusoidal gratings were displayed at optimal orientation, spatial frequency and drift rate.

Because of the limited time available for recording from a single neurone, we used a procedure which enabled us to adapt to a given ambient contrast level while at the same time testing to obtain a contrast response function. To do this, we varied the contrast of the test grating (defined as \((I_{\text{max}} - I_{\text{min}})/(I_{\text{max}} + I_{\text{min}})\) where \(I_{\text{max}}\) and \(I_{\text{min}}\) are, respectively, maximum and minimum intensity levels in the grating pattern) by small amounts above and below a fixed ambient level of adaptation contrast. The testing contrast levels were restricted to within \(\pm 1\) octave of the ambient contrast in order to maintain a reasonably fixed contrast level. Each contrast was presented for 4 s, a total of 10 times, and these presentations were randomly interleaved with 4 other contrasts within the small range indicated. This procedure generated a contrast response function for a particular level of contrast adaptation. The same procedure was repeated to obtain functions for other adaptation levels using different ambient contrasts. The resulting histograms were harmonically analysed to obtain d.c. and first

![Fig. 1](image-url)
harmonic components as response measures. Because simple cells respond with modulated discharge to drifting gratings, the first harmonic was used for these cells, while the d.c. component was used for complex cells since they show elevated average discharge rates in response to drifting gratings. For LGN fibres, the d.c. component was used.

A total of 91 cells were studied in detail, of which 44 were classified as simple, 39 as complex and 8 as LGN fibres. Figure 1a and b show the results of measurements for a simple and complex cell, respectively. Curves in solid lines represent contrast response functions measured under different levels of contrast adaptation (from left to right, 3, 1, 3, 6, 12, 25, 50%). Four features of these curves are evident in both a and b. First, the curves shift along the log-contrast axis to the right as contrast increases. Second, slopes of the curves are very steep and the first four curves are approximately parallel to each other. Only at the highest contrast (>30%) is some reduction of slope found. Third, the curves span approximately the same response range (spikes per s) despite the fact that they cover considerably different input contrast domains. Fourth, even a relatively low contrast (6–10%) seems to exert a significant adapting effect, as illustrated by the first three curves from the left. These features are remarkably similar to the behaviour of retinal neurones with respect to variations of ambient light intensity. However, the operating ranges of the cortical cells are quite narrow compared with those for retinal neurones.

One other feature of our data should be noted. We measured the steady-state response at the ambient contrast for each contrast response function to see if the curves are centred. This is a measurement of the response level of a neurone after a sufficiently long delay following an onset of a stimulus or a step change in input levels. In other words, it is the level to which the neurone's response finally settles down when the adaptation process to a constant contrast is completed. If this steady-state response level falls in the middle of the corresponding contrast response function, one may conclude that the curve is actually centred to match the prevailing contrast level. This measurement was made before that of the corresponding contrast response function to ensure adaptation, and the results are shown as dashed lines in Fig. 1a and b. Note that these lines approximately go through the mid-points of the other contrast response functions (solid lines), establishing that the curves are, in fact, nearly centred to match the ambient contrast.

The analogy we draw between the retinal sensitivity control mechanism for light intensity and contrast adaptation in the striate cortex is limited because all cortical cells do not show clear adapting behaviour of the type illustrated in Fig. 1a and b, while presumably, retinal neurones invariably adapt to prevailing light intensity levels. The adaptability of cortical cells to contrast varies from none to extensive. Figure 1c shows a complex cell which apparently did not adapt. All contrast response functions (solid lines) measured with different adapting contrasts and the steady-state response curve fall very close to each other, indicating fixed contrast–response properties for this unit. We found other cells, of both complex and simple types, which displayed no adaptation. One other type of unit that we recorded is of interest here. Of eight LGN afferents recorded in the striate cortex, none showed substantial adaptation. Figure 1d illustrates the results from an on-centre Y-cell from this group. As in the previous example (Fig. 1c), the contrast–response function of this LGN fibre was rigid, that is, it was not modified by adaptation. This suggests that contrast adaptation is primarily cortical in origin rather than occurring at the retinal or geniculate levels.

To facilitate comparisons, we computed an index of adaptability for each cell based on the slopes of the contrast–response functions. The index was computed by first normalizing the response and contrast ranges. By definition, the index is the mean of the contrast response functions (solid lines in Fig. 1) minus the mean of the slopes of the curve connecting the mid-points of the functions at the adapting contrasts. For the cells we studied, the distributions of indices are unimodal and similar for simple and complex cells, with means and standard deviations of 0.52 ± 0.39 and 0.58 ± 0.41, respectively. Although this quantification is somewhat arbitrary, it suggests that contrast adaptation is a rule rather than an exception in the visual cortex, because most cells exhibit some degree of adjustment. On the other hand, indices for LGN fibres were very low (0.17 ± 0.22), reflecting their rigid contrast–response functions.

Considered together, these results have intrinsic functional significance, but they further suggest that caution must be used in trying to specify the absolute contrast threshold of cortical neurones. Our results demonstrate that the response amplitude and contrast threshold of a striate neurone can be strongly influenced by the contrast levels the cell has experienced in the recent past, even for contrasts as low as 6–10%. Previous investigations in which absolute contrast thresholds of cortical cells have been estimated have not included controls for the effects of adaptation when measuring contrast–response functions. To illustrate this point, we used a procedure which was similar to that used in these other studies (all contrasts were presented in one randomized session) to generate a contrast–response function spanning the entire range. The result of this measurement is shown by dotted lines in Fig. 1a and b. Comparison of threshold estimates by extrapolation to the contrast response curves clearly that these determinations tend to overestimate, or even possibly exceed, the contrast threshold, because of an overall adapting effect of the test stimuli.

We have shown that for most striate neurones, contrast adaptation behaves in a notably similar way to the retinal sensitivity control mechanism. Response–intensity curves in the retina and response–contrast curves in striate neurones shift laterally along log-intensity and log-contrast axes, respectively, so that they appropriately match prevailing input levels. We describe this mechanism in the cortex as contrast gain control, because of the lateral displacement of the central portion of the neurone's response curve along a logarithmic axis. This is equivalent to multiplicative or divisive scaling, although we cannot specify the exact underlying mechanism. Shapley and Victor have proposed a contrast gain control mechanism for cat retinal ganglion cells. The mechanism we have investigated is probably quite different because we have found that LGN fibres show little contrast adaptation. Presumably, retinal neurones would behave similarly in this respect.

One may speculate on possible functions of contrast gain control in the cortex. For example, it may provide an advantage for the maintenance of a relatively high differential contrast sensitivity. Perhaps the visual system can cope with a wide range of contrasts because of this auto-ranging capability made available by contrast gain control. Alternatively, the outputs of neurones equipped with contrast gain control might provide inputs to possible succeeding pattern processors which must be capable of extracting pattern information regardless of the contrast of images. This can be realized by matching the input domain of pattern processors with that of image contrast through contrast gain control.

This work was supported by grant EY01175 and Research Career Development Award EY00092 from the US National Eye Institute to R.D.F. G.S. was supported by training grant EY07043.

Note added in proof: None of 27 cells recorded directly in the LGN showed significant adaptation.

Received 17 February; accepted 10 March 1982.