Visual Acuity and Contrast Sensitivity in Patients with Cerebral Lesions

Abstract. *Spatial contrast sensitivity as a function of spatial frequency was measured in patients with cerebral lesions. In most of these patients visual acuity, as measured by the Snellen chart, was 20/30 or better, yet marked departures from normal contrast sensitivity were found. The greatest loss in contrast sensitivity occurred at high frequencies, but in one patient the loss was greatest in the midfrequency range. This finding lends support to the channel hypothesis of spatial contrast discrimination.*

Evidence has been accumulated supporting the notion of spatial frequency channels in the human visual system (*1*). Visual acuity is usually tested by symbols, letters, or grating patterns presented at (nominally) 100 percent contrast. The smallest detectable image establishes the visual acuity of the observer but does not necessarily specify his sensitivity to larger targets of lower contrast.

O. H. Schade was the first to employ sinusoidal grating patterns as test targets to investigate the spatial contrast sensitivity of human observers (*2*). The graphic representation of this sensitivity as a function of the spatial frequency is called a contrast sensitivity curve. The last point of this curve is a measure of acuity; it represents the smallest resolvable image at 100 percent contrast.

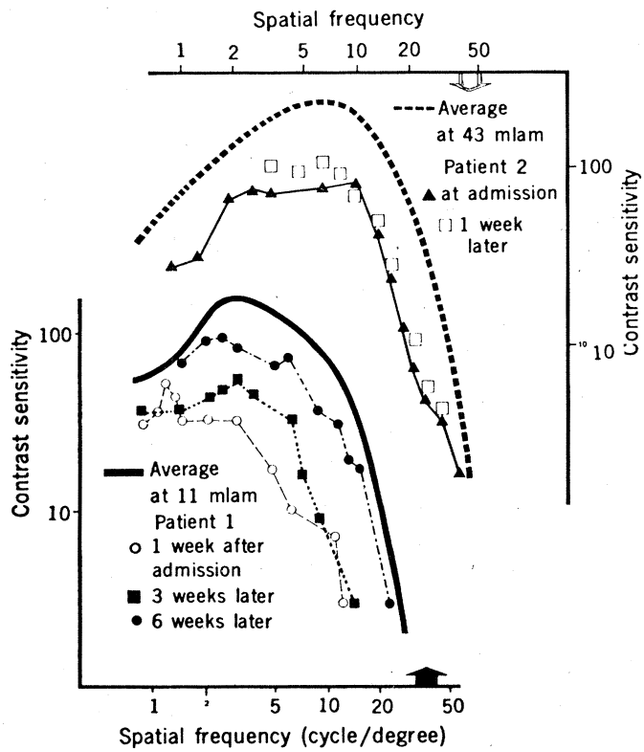
Campbell and Robson (*1*) found that the sensitivity of the human observer to complex gratings (that is, grating patterns of nonsinusoidal luminance distribution, for instance, a square wave grating) can be characterized by independent detecting mechanisms, with each responding to a frequency component of the complex grating. This finding led them to the conclusion that the human visual system consists of spatial frequency channels. Each channel responds to a narrow range of frequencies and each has its independent detecting mechanism.

Neurophysiological work indicates that separate anatomical structures may provide the spatial channels (*3*). This suggests that a nonuniform alteration might be found in the contrast sensitivity curve of observers who have lesions in the central nervous system.

I have investigated 16 patients who had clinically diagnosed lesions involving the visual pathways. This initial report presents the data of two of the patients. The findings for patient 1 are representative of those of most of the other patients. Although the findings for patient 2 are not representative, they seem to lend clinical support to the channel hypothesis.

Before each patient was tested, he had a complete eye examination. Only patients who had neither retinal pathology nor eye movement disorders were selected for testing. Cyclopentolate (1 percent solution) was used for cycloplegia, and a 3-mm artificial pupil was provided. Corrective lenses made the patient's eye emmetropic for each viewing distance.

The grating patterns were presented



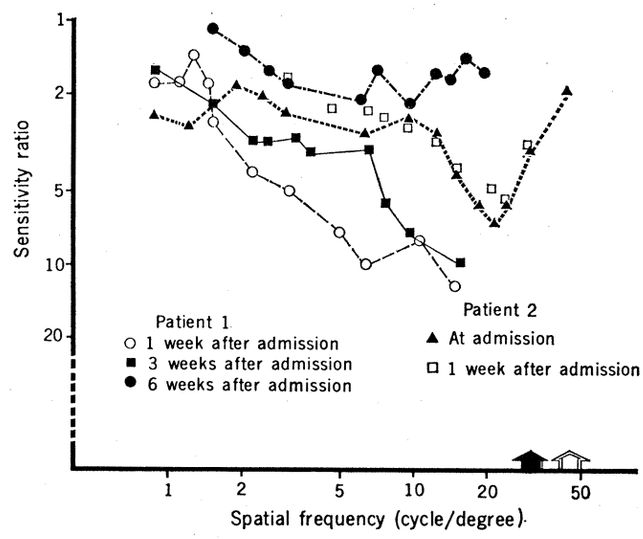


Fig. 1 (left). Spatial contrast sensitivity curves at a mean luminance of 11 mlam (A) and 43 mlam (B). The right eye was used throughout. Sensitivity (the inverse of threshold contrast) is plotted against the spatial frequency of the grating pattern on a log-log scale. The arrows represent the extrapolated cutoff frequencies (visual acuity) at 11 and 43 mlam for the normal (average of four individuals) observer. In (A), curves with interrupted lines were obtained from a patient with an infectious lesion of the left parieto-occipital area (patient 1). In (B) data points are from a patient with a meningioma pressing on the left occipital pole (patient 2). Fig. 2 (right). Visuogram of patients 1 and 2. On the ordinate the ratio of the normal observer's to the patient's sensitivity is plotted against the spatial frequency at the comparable mean luminance on a log-log scale. A uniform reduction in contrast sensitivity would yield a line parallel to the abscissa. The plot reveals some nonuniform reductions of contrast sensitivity. Note the negative "hump" in the visuogram of patient 2. The arrows represent the extrapolated cutoff frequency for the normal observer at 11 mlam (solid arrow) and at 43 mlam (open arrow).

vertically on the face of a Tektronix 561 oscilloscope with P11 (whitish) phosphor. The methods of Campbell and Green (*4*) were followed for the generation and presentation of the patterns. The contrast and the spatial frequency of the patterns varied without affecting the mean luminance of the screen. The grating was alternated at 0.5 cycle/sec with a blank screen of the same mean luminance. Two viewing distances were selected in order to cover a sufficient range of spatial frequencies without presenting too few bars at low frequencies. The oscilloscope screen was surrounded by another screen that was illuminated from behind the screen by a circularly shaped fluorescent tube at approximately the same color and luminance as the face of the oscilloscope. The patients used monocular vision. The observer or the experimenter could vary the contrast of the presented grating by means of a potentiometer. Threshold was determined as a mean of four independent decisions. The normal curve was established as a mean of four normal observers, aged 18 to 62.

Patient 1 was a 24-year-old female who was hospitalized because of a severe headache and blurred vision. It was found that at the time of admission she had a subacute bacterial endocarditis which caused a metastatic lesion in the left parieto-occipital region. She was treated with antibiotics. Within 8 weeks she improved subjectively and objectively.

At the time of admission before treatment was begun, standard eye examinations revealed no retinal pathology. Her visual acuity on the Snellen chart was 20/30 in both eyes. Perimetric examination revealed small, bilateral paracentral homonymous scotomas. These lesions gradually decreased in size and were not found at the time of her discharge from the hospital. Three contrast sensitivity curves were obtained at different times during recovery (Fig. 1A). They show a gradual improvement, although her optical conditions did not change. A plot of the ratio of the normal curve to the data of patient 1 is presented in Fig. 1B. By analogy to hearing tests, this plot could be called a "visuogram."

This patient had more loss in contrast sensitivity at higher than at lower spatial frequencies when tested 1 and 3 weeks after admission. At the time of her discharge from the hospital her contrast sensitivity curve had returned nearly to normal. At this time, in contrast to initial findings, electroencephalogram (EEG) studies indicated the absence of the abnormality in the right posterior region, angiographic studies revealed lack of the displacement of the parieto-occipital branch of the right posterior cerebral artery, and brain scan demonstrated a decrease in the size of the radioactively positive uptake region.

Before the improvement of this patient, she experienced difficulty in reading even though there was only a 20/30 reduction in her visual acuity. Such a small drop in acuity is generally considered a mild visual impairment. However, a reduction in contrast sensitivity over a wide range of spatial frequencies might affect visual competency rather markedly, because in everyday life there are very few objects encountered at 100 percent contrast.

If Campbell and Robson (*1*) are correct in their concept of the existence of size-tuned channels in the visual system, then there is another way of explaining a discrepancy between visual acuity and visual competency in a practical situation. Assuming the brain does use harmonic analysis for pattern recognition (*5*), then in suprathreshold stimulus conditions a proper balance of independent signals is required to account for an invariant neural representation of the retinal image. In this case a nonuniformly altered contrast sensitivity would pose a greater difficulty in pattern recognition than a simple drop in visual acuity. The visuogram of patient 2 suggests that selective frequency loss can occur in the presence of very slight change in the cutoff frequency.

Patient 2 was a 59-year-old male who noted episodic blurring of his vision, especially when attempting to read. He bumped into objects on his right side, and his writing had deteriorated. He developed severe headaches. At the time of his admission to the hospital, EEG, brain scan, and angiographic studies revealed an occipital lesion on the left side, located at the tip of the calcarine fissure. Also at this time, an eye examination showed his visual acuity to be 20/30 for the left eye and 20/40 for the right eye, and a perimetric examination revealed a dense right homonymous hemianopsia. There was no central scotoma. A tumor was diagnosed, and a meningioma (5 by 5 cm) was removed during subsequent surgery. This tumor pressed directly on, but did not infiltrate the brain tissue.

A sensitivity curve for this patient was obtained before steroid therapy was begun (Fig. 2). Additional data points were collected after 1 week of therapy but before surgical intervention. It is thought that steroid medication decreases the edema (swelling) of the tissues that were under pressure. Unfortunately, no test could be obtained after the tumor was removed. Figure 2 shows his visuogram. It is apparent that this patient had a greater loss in contrast sensitivity in the midfrequency region than at higher frequencies. Thus the cutoff point bore no relation to his loss in contrast sensitivity.

In most patients the visuogram demonstrated a general reduction in contrast sensitivity with relatively greater loss at high frequencies as represented by patient 1. Relatively greater loss at high frequencies can occur as an effect of optical blurring (*4*). However, all patients had been objectively refracted as the first step in this investigation. Also, there was an improvement in the contrast sensitivity curves of this patient during recovery, even though her optical conditions did not change during hospitalization (Fig. 1B). This type of visuogram (patient 1) cannot be explained by a relative difficulty of detecting high frequency gratings; the standard deviation is fairly constant at all frequencies. Thus if only attentional factors played a role, I would have expected a uniform reduction in contrast sensitivity. An explanation could be a loss of the neural "sharpening" in a single detector mechanism. Alternatively, it might be possible that the greater loss at high frequencies indicates relatively greater damage in channels tuned to these frequencies. This proposition demands an answer as to why the high frequency channels of the visual system should be more vulnerable to cerebral lesions.

The visuogram of patient 2 also shows some reduction in overall contrast sensitivity and relatively greater loss at midfrequencies than that at higher or lower frequencies. The finding of selective frequency loss, presumably due to the clinically verified damage in the occipital lobe area, supports the concept of spatial frequency channels in human vision. This is con-

sistent with current theories that harmonic analysis is one of the early stages of pattern recognition in humans (*5*). There are only six data points from one patient to support this theory. Nevertheless, in all cases it is clear that a given shift of the cutoff frequency is not a measure of the loss in the contrast sensitivity of other frequencies.

There are, however, difficulties before a rigorous theoretical interpretation of these findings can be attempted. The normal curve should be obtained from a larger population. Because in all patients there was some reduction in overall sensitivity, it would be worthwhile to investigate patients who have lesions not involving the visual pathways. Again it could be expected that lack of attention, or individually high threshold criteria, will produce a uniform reduction in contrast sensitivity (flat visuogram). The effect of cerebral lesions on spatial contrast sensitivity functions in humans requires further study. Besides their importance to pattern recognition theories, such studies would be revealing to the clinician who is confronted with patients that complain of difficulty with everyday visual tasks, yet have a seemingly adequate visual acuity.

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References and Notes

1. F. W. Campbell and J. G. Robson, *J. Physiol. London* **197**, 551 (1968); F. W. Campbell and L. Maffei, *ibid.* **203**, 237 (1969).
2. O. H. Schade, *J. Opt. Soc. Amer.* **46**, 721 (1956). The contrast of the grating pattern is customarily expressed as the ratio of the difference of maximum and minimum luminance over their sum. Sensitivity is measured by the inverse threshold contrast as a function of the spatial frequency of the grating. Spatial frequency is an equivalent measure of bar size in a grating pattern; that is, it is the number of alternating pairs of dark and bright bands of the pattern subtended in degree of visual angle at the observer's eye.
3. Cr. Enroth-Cugell and J. G. Robson, *J. Physiol. London* **187**, 517 (1966); F. W. Campbell, G. F. Cooper, Cr. Enroth-Cugell, *ibid.* **203**, 223 (1969).
4. F. W. Campbell and D. G. Green, *ibid.* **181**, 576 (1965).
5. C. Blakemore and F. W. Campbell, *ibid.* **203**, 237 (1969); D. A. Pollen, J. R. Lee, J. H. Taylor, *Science* **173**, 74 (1971).
6. Supported by grant US 5221-07. I thank Dr. S. P. Diamond for many suggestions and for the proposition of the name "visuogram," and Dr. J. Z. Levinson for our discussions.

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Regulation of Testis Function in Golden Hamsters: A Circadian Clock Measures Photoperiodic Time

Abstract. *The photoperiodic testicular response of adult golden hamsters was examined by the use of a 6-hour light period coupled with dark periods of 18, 30, 42, and 54 hours. Cycle lengths of 24 and 48 hours resulted in testicular regression, whereas testicular weight was maintained by cycle lengths of 36 and 60 hours. Our data demonstrate a circadian rhythm of sensitivity to the effects of light on the photoperiodic testicular response of the hamster. The position of light relative to the circadian system (as measured by the locomotor rhythm) is critical in the response.*

Synchronization of reproduction with environmental periodicities in the field has been observed in many mammalian species (*1*). However, the regulating role of light in timing annual reproductive cycles has been demonstrated experimentally in only a few mammals. The role of photoperiod in the timing of estrus has been examined (*1, 2*), whereas few studies have considered photoperiodic control of reproductive function in males. In autumn breeders (goat and ram), short days are necessary for induction and maintenance of spermatogenesis (*3*), whereas in spring breeders such as the vole, snowshoe hare, and ferret, testicular function is stimulated by long photoperiods (*1, 2, 4*).

Hoffman, Reiter, and colleagues (*5*) demonstrated that photoperiods of LD 16 : 8 (16 hours of light and 8 hours of dark per day) or LD 14 : 10 maintained testicular size and function in the golden hamster, whereas photoperiods of LD 2 : 22 or LD 1 : 23 induced testicular regression. Gaston and Menaker (*6*) subjected male hamsters to photoperiods with 0 to 24 hours of light per day; at least 12.5 hours of light per day were required to maintain spermatogenesis and prevent testicular regression. Although light has important effects on reproductive function in at least some mammals, the mechanism by which light synchronizes breeding cycles with the environment remains unknown. In the case of photoperiodic effects, such synchronization must surely depend upon the ability of the organism to distinguish one naturally occurring day length from another. This implies the participation of a biological time-measuring system in the reproductive response to light.

The hypothesis that an endogenous circadian clock mediates the measurement of photoperiodic time was advanced by Bünning to explain the mechanism of photoperiodic time measurement in plants (*7*). Bünning proposed that the organism possesses an endogenous circadian (approximately 24-hour) rhythm of sensitivity to photoperiodic induction by light. In Bünning's model, photoperiodic induction by long days occurs when light extends into the photoinducible phase of the rhythm. On short days, induction fails because light is restricted to the nonsensitive phase of the rhythm (*8*). A major difficulty in executing direct and critical tests of Bünning's hypothesis derives from the prediction that light will have a dual role in photoperiodic systems; in addition to its action as inducer of the photoperiodic response, light will also act as a "Zeitgeber" that entrains (synchronizes) the photoperiodic sensitivity rhythm through its action on the circadian clock to which that rhythm is coupled (*9*). Thus any critical test of the Bünning hypothesis must take into account the effect of light upon the phase of the rhythm as well as its more direct effect on the photoperiodic response (such as reproductive state). Ideally, one would like to assay the phase of the photoperiodic sensitivity rhythm during exposure to various inductive and noninductive light cycles, but the sensitivity rhythm by its nature cannot be assayed independently of induction. In testing the hypothesis it therefore becomes necessary to assume that the sensitivity rhythm is controlled by the same clock that controls another readily measurable overt circadian rhythm (such as locomotor activity) and to use that rhythm as an indicator. This requires the additional assumption that the measured rhythm bears a constant phase relation to the sensitivity rhythm. If these assumptions are made, the behavior of the overt rhythm can be taken to reflect the behavior of the hypothesized rhythm of photoperiodic sensitivity.

A circadian rhythm of sensitivity to