How patterns of bleached rods and cones become visual perceptual experiences: A proposal

Robert Galambos* and Gábor Juhász

University of California, San Diego, CA 92093; and Eötvös Loránd University, 1088 Múzeum krt, 4/A, Budapest, Hungary

Contributed by Robert Galambos, August 6, 2001

In an attempt to increase information about how mammalian visual systems create a perceptual experience out of a retinal photochemical bleach pattern, this article brings together recent rat physiological data acquired with large electrodes, an old cat behavioral experiment, and two complex human behaviors: reading and the reversible blindness people experience when the scene being viewed is stabilized on the retinal surface. The outcome suggests this juxtaposition of disparate data sets has been logical, reasonable, and informative. The link between rats and reading is the fact that both rat and human retinas convert bleach patterns into ganglion cell volleys 3 times a second. The probable trigger for these episodic retinal volleys is a more or less abrupt change in the pattern of bleached rods and cones, and we claim the absence of this trigger when the image is stabilized is responsible for the blindness. The cat behavioral experiment correlates performance on visual discrimination tasks with the number of nerve fibers remaining after lesions of the optic tract. The analysis of the result, which shows that as few as 2% of the normal number of nerve fibers supports perfect performance of such tasks, prompts the concept of a second dynamic visual system, operating in parallel with the anatomical nervous system pictured in the textbooks. The dynamic visual system model, which brings into the foreground important old facts that have been neglected and integrates them with new data, offers a synthesis that may be useful in interpreting classical visual behavioral phenomena.

retinal ganglion cell volleys | stabilized image | reading physiology | optic tract lesion

Most human visual behavioral responses still lack convincing physiological explanations. A remarkable example of these is reading, that visual behavior children everywhere learn early and practice throughout a lifetime. But certainly the most mysterious example is the reversible blindness people report when a clever arrangement of mirrors negates the effect of eye movements, and the image in view is artificially maintained in exactly the same retinal location. Such images disappear within a second, and if the room in which the measurement takes place is lighted, the subject may ask who turned off the lights. This is the so-called stabilized image experiment, a sensory experience as surprising and improbable as any ever described.

That a half-century of microelectrode measurements has so far failed to supply a satisfactory explanation for either of these visual perceptual phenomena suggests that crucial facts necessary for understanding them may lie beyond the reach of that technique. In an effort to do better with such problems, we have for the past decade been studying rats implanted with both a stimulus-producing device [remotely controlled light-emitting diodes (LED)] and relatively large electrodes that record the retinal and cortical activity the LED stimulation evokes (1, 2). The rats are free to move within a small testing box and are studied daily, often for many weeks, when awake and asleep. We believe the measurements reveal facts pertinent to the reading and stabilized image problems, and to other visual perceptual experiences whose physiological origins are still debated.

What follows is divided into five parts. The first deduces from an examination of reading behavior what must be true about the visual system that makes reading possible. The second section examines what happens when saccades like those used in reading are not permitted to perform their natural function, which is to place new scenes on the retinal surface. The third section presents an example, in the cat, where the eyes move normally but an optic tract lesion has reduced the number of nerve fibers that transfer to the cortex the information extracted from the retinal bleach patterns. The fourth part, a selection from our experiments on normal rats pertinent to the interpretation of the other three, is introduced where appropriate throughout the text. Finally, the fifth part summarizes the findings and shows ways the pioneering information can be applied.

Two Neglected Visual Behavioral Responses. *Reading: Basic facts and some inferences from them.* For over 100 years, reading behavior has been studied (3, 4); yet with rare exceptions (5), modern textbooks do not even mention the topic. The basic facts, which have not changed materially for decades, can be summarized briefly.

(*i*) The eyes move together across a page of text and stop abruptly 2 to 4 times each second for fixations lasting about 200 ms.

(*ii*) What happens during a fixation is complex, but the general outline of what must take place is obvious. Light bleaches the rods and cones in a pattern corresponding to the words in focus on the retinal surface. The intrinsic retinal neurons then translate the information in the photochemical bleach pattern into the exact ganglion cell equivalent and deliver it into the optic nerve. The information encoded at the retinal level then reaches the visual cortex after a synaptic relay in the lateral geniculate nucleus. Simply summarized, the retina prepares about three analyses of the reading material every second, and each of them, on reaching the cortex, is added to the earlier arrivals to produce the continuing visual perceptual experience. The evidence for these assertions is sometimes indirect but given the existing facts, a reasonable alternative is almost inconceivable.

(*iii*) Converting photochemical information into neuronal information is only part of the task of the retina; it must also restore itself to the biological equivalent of unexposed photographic film, fully prepared to deal with the new bleach pattern the next fixation will produce. Because both parts of the retinal respond/recover cycle are completed within a third of a second, it is likely the two processes at least partly overlap in time.

Logical conclusions emerge automatically from the facts just enumerated. We offer here a list of these as predictions, or hypotheses, or propositions that suitable experiments have already tested, or should test.

(*i*) When an eye is open in a lighted environment, its optic nerve transports an endless procession of discrete about 300-ms ganglion cell volleys. Although what has just been said must be

Abbreviation: LED, light-emitting diode.

*To whom reprint requests should be addressed at: 8826 La Jolla Scenic Drive, La Jolla, CA 92037. E-mail: rgalambos@ucsd.edu.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. §1734 solely to indicate this fact.

 www.pnas.org/cgi/doi/10.1073/pnas.201420798

true for people during reading, it is possible that episodic retinal output is, in fact, the rule for all vertebrate retinas.

(*ii*) Every ganglion cell volley is unique because it is the neuronal version of the information contained in a particular pattern of bleached rods and cones, no two of which can ever be identical. At the rate of 3 Hz, a reader's two retinas create about 400 such unique volleys every minute, each of which represents activity in a substantial fraction of the more than 1 million axons each human optic nerve is estimated to contain.

(*iii*) Conclusions reached about the retinal output during reading can safely be generalized to other visual behaviors. For example, a decade ago, an automobile driver instrumented with an eye movement-recording device changed the direction of his gaze an average 2.5 times per second while rolling over London streets (6). Reading behavior would seem to be only the most obvious demonstration that mammalian retinas regularly analyze patterns of bleached rods and cones at rates as high 3 or 4 Hz (the rate varies with interest in the task, among other reasons).

(*iv*) The intraretinal events that organize the about 300-ms ganglion cell volleys have never, to our knowledge, been intentionally modeled despite what must be tens of thousands of macro- and microelectrode experiments thus far reported on vertebrate retinas.

(*v*) Similarly, no detailed model exists for the ≈300-ms recovery events that convert an activated retina into one where photoreceptors and intrinsic neuronal network are ready to deal with the information the next fixation will deliver. However, it is known that in the macaque retina both the magno- and parvocellular ganglion cell adaptations are "rapid and largely complete in 100 msec or less" (7); similar recovery time constants must also characterize the human ganglion cells activated during reading.

Reversible Blindness: The Stabilized Image Experiment. Like the reading experiments, stabilized image experiments find no place in the modern textbook. They began a half-century ago when the question, "What does a person see when eye movements are prevented from moving the scene across the retinal surface?" was asked almost simultaneously in Ireland (8), Rhode Island (9), and Russia (10). Each group attached a small mirror to a contact lens, like the one Riggs (11) first described in 1941, and devised an external optical system that counteracted eye movements and maintained the image in a fixed position on the retinal surface. What follows quotes the pioneers who share the discovery and gives their opinions about its physiological origin.

The Rhode Island group (L. Riggs, F. Ratliff, J. C. Cornsweet, and T. N. Cornsweet) stated, ". . . with an essentially motionless retinal image, prolonged fixation results in the disappearance of objects from the field of view," and suggested light adaptation of the rods and cones is responsible (ref. 9, p. 500).

The Russian scientist Yarbus (ref. 10, p. 100) wrote, ". . . if a strictly stationary and unchanging retinal image is created artificially, the eye ceases to see," and suggested that "constancy and immobility of the retinal image will banish impulses entering the optic nerve from the eye or will sharply reduce their number. In these circumstances, absence of signals from a certain part of the retina gives the visual system information that this area corresponds to a uniform surface."

Ditchburn (12) recognized four types of stabilized images, describing Type III as "total loss of visual perception leaving a black field" (p. 132). His first explanation (13) was followed 20 years later by one that summarizes the 1973 version of the Kuffler–Hubel–Wiesel microelectrode experiments, and says, ". . . in all of the situations in which a sharp image is seen, suitable signals are generated by processes which have been extensively investigated by electrophysiologists" (ref. 12, p. 194). Ditchburn than rejects light adaptation as a possibility and says, "When the hazy field occurs [this is his Type II], most of the specific pattern detectors are not giving clear signals . . . The signals associated with meaningful patterns are confused. This leads to a reduction of those signals that control visual attention, possibly through the reticular formation. This loss of awareness leads to a further loss of intelligible visual information . . . By this kind of 'vicious circle,' or positive feedback, the whole visual perceptual system becomes inoperative and the field goes black."

Neither the Yarbus "banished impulse" suggestion nor the "confused meaningful patterns" of Ditchburn has become a useful working hypothesis. More recently suggested mechanisms include retinal adaptation only (14, 15), cortical processing only (16), and both retinal and cortical processing (17). A combination of cell assembly and Gestalt theories has been proposed (18), and two groups claim stabilization requires binocular interaction and is not a retinal phenomenon (19, 20). Evidently, the universally acceptable working hypothesis is as evasive as the phenomenon is hard to believe.

The New Facts Pertinent to Visual Perceptual Experiences. *The rat 300-ms ganglion cell volley.* Our experiments, aimed at understanding the physiological basis of perceptual phenomena, used white rats with implanted corneal, chiasm, and cortical electrodes. Information collected at these three sites—the beginning, middle, and end of the anatomical visual pathway—provides a broad sample of the activity visual stimuli produce. Remotely controlled LEDs attached permanently to the skull deliver the stimuli. Fig. 1 shows typical responses time-locked to 1-ms LED flashes varied in luminance through about 3 log units. Two constant features of the chiasm records are their similar duration—about 300 ms—and that they resemble each other in being approximately triphasic waveshapes. Our publications show these responses appear in waking, sleeping, and anesthetized animals when dark- or light-adapted and after stimuli that vary in luminance, duration, and rate (1, 2).

We emphasize that these ganglion cell volleys are uniform and replicable. In thousands of recordings collected over several years on more than 40 rats, the response at the chiasm electrode has been a recognizable variation of the polyphasic waveshape seen in Fig. 1 in latency, waveshape, and duration whenever the rat delivered any response at all. The optic tract response has been described often in the past but no one, so far as we are aware, has examined its properties by using a wide range of full-field stimuli delivered to a normal mammal, and then inferred from the data what is constant about the ganglion cell output of an activated retinal neuropile.

Ganglion Cell Volleys During Reading Extrapolated. We believe it is not a coincidence that both rats and readers deliver about 300-ms ganglion cell volleys, and extrapolate the fact to mean the obligatory output of a normally activated mammalian retina is not a continuous stream of individual ganglion cell spikes; it is a compact collection of tens of thousands of them packed in an orderly way into an interval of about 300 ms. We sometimes call such a volley an A/B/C/ganglion cell sequence (GCS) and sometimes a histogram.[†] Whatever the name, each one is to be viewed, we believe, as a kind of biological constant, the retinal functional unit. The histograms in Fig. 1 visualize these functional units in the optic tract of a rat, and we presume similar invisible ones are being created endlessly by the reader of this sentence.

Do the about 300-ms volleys produced by human saccades reach

[†]*Webster's Third New International Dictionary* (21) defines histogram as "a graphical representation of a frequency distribution by means of rectangles whose widths represent the class intervals and whose heights represent the class magnitudes." Microelectrode physiologists use the term to cover single-unit interval and latency distributions, whereas others plot phase histograms, two-dimensional histograms, etc. We use the term here for the poststimulus time distribution, at the computer sampling rate, of the ion current amplitude axons produce as they sweep past a fixed electrode.

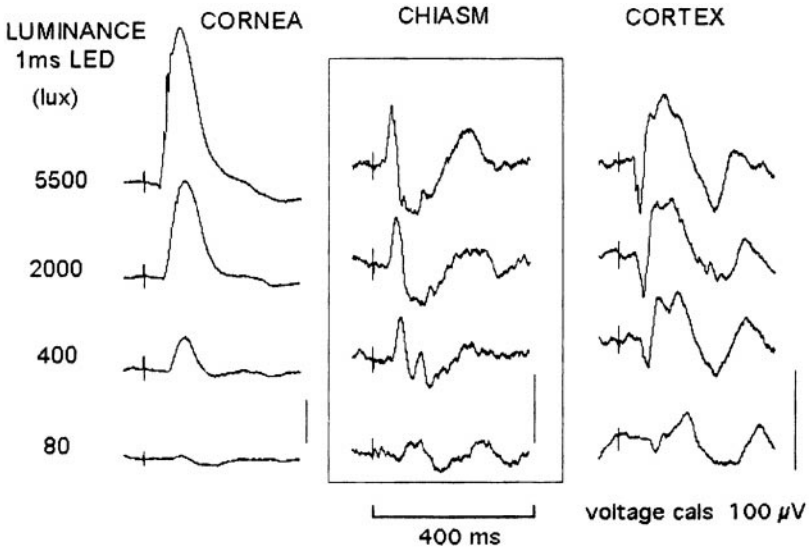

Fig. 1. Activity evoked at three levels of a dark-adapted rat visual system by 1-ms red LED flashes (n = 50) graded in luminance. At the retinal level (left column), the near-threshold flashes (bottom trace) evoke a barely visible electroretinogram (b-wave) response that increases systematically in amplitude as the luminance rises through about 3 log units. The second retinal output, the ganglion cell activity recorded at the chiasm level (center column), behaves in an entirely different manner. Its near-threshold triphasic waveshape is apparent in every suprathreshold response, and, except for minor latency and waveshape changes, they are all similar. Evidently, two different processes proceed concurrently in a stimulated retina, each time-locked to stimulus onset. The cortical activity (right column) closely resembles a mirror image of the corresponding chiasm response, which can happen only if the retinal ganglion cell volley passes through the lateral geniculate synapses without major change. The penetration throughout the entire visual system of the neuronal representation of the scene created in the retina is an important conclusion to be drawn from these facts.

the cortex and produce cortical evoked potentials? Yes. The lambda wave-evoked cortical potential is activity time-locked to the optic nerve volleys voluntary saccades produce, as Armington (22) first demonstrated in 1972 (see also ref. 23, figure 7.35).

The Stabilized Image Experiment. If human vision does begin in the form of ganglion cell volleys lasting a third of a second, then a person with normal vision should see nothing if the retina fails to produce them. In our view, the bulk of the stabilized image blindness phenomena can be explained by assuming first, that changes in light distribution on the retinal surface initiate ganglion cell volleys, and second, that this does not happen when the image is stabilized. These hypotheses predict there will be a threshold value for the hypothetical retinal illumination change that destabilizes stabilized images.

There are in fact two thresholds, one for the minimum successful movement, the other for the minimum successful change in luminance, and the Ditchburn (24) group measured both with the following results. An abrupt displacement measuring 1 min of arc destabilizes an image 50% of the time, and 2.5 arcmin displacements always succeed. (For readers not familiar with visual measurement details, on a newspaper held 50 cm from the eye 1 arcmin equals about 0.3 mm or, on the retina, a few cone diameters.) As for the minimum ambient luminance change, which can be either a decrease or increase, Ditchburn says the 1 arcmin change in the peak to peak amplitude of a 10-Hz oscillation will destabilize an image 80% of the time.

Ditchburn's numbers are interesting because they predict the retina will deliver a 300-ms ganglion cell volley after very small movements of eye, head, or body, as well as after changes in locus or luminance in the scene itself. Measurements show that even when the eyes are deliberately held in one place, the involuntary saccades vary up to 10 arcmin in amplitude as often as several times each second (ref. 12, table 4.2; see also ref. 5, p. 306ff.). Obviously, the body has redundant ways to prevent image stabilization and it continually forestalls blindness by using one or another of them to trigger a ganglion cell volley.

The Role of the Retina in Complex Behavioral States. *Sleep.* The second of our recent publications reports that the retinal output systematically changes during sleep (2). The change, a significantly enhanced amplitude of the chiasm response during slow-wave sleep, may be mediated by the fibers of the serotonin system known to reach the retina from the midbrain. This finding is direct evidence the retina does more than passively convert photochemical events into ganglion cell volleys. The retina is a brain-like structure, separated during embryology from the brain itself but in adulthood still connected reciprocally with it. The endless creation of ganglion cell volleys during reading is testimony to its brain-like abilities—it is no small feat to create, 3 times a second, a unique sample of human ganglion cells, each sample a precise neuronal transcription of the information in a rod/cone bleach pattern. (Parenthetically, these remarkably high retinal response/recovery rates challenge conventional explanations of retinal function.) What does the brain do with these discrete packages of information the retina delivers? We suggest the following cat experiment gives a useful answer.

Optic Tract Lesions Do Not Eliminate Pattern Discrimination. More than 30 years ago, R.G. and colleagues reported on the visually guided behavior of cats with bilateral lesions of the optic tract (25–27). The experiment is the visual analog of an earlier auditory experiment in which cutting the cochlear inputs to the thalamus failed to prevent normal cortical electrophysiological responses to sounds (28).

A thirsty cat inside a black box learns to press one of two lighted panels; pressing the "correct" panel causes a few drops of milk to be delivered into a small cup. The panels display either different patterns (9 vs. 6) or different luminances (the flux task). After extensive training, each cat (n = 8) received what was intended to be bilateral destruction of both optic tracts beyond the chiasm level by using a stereotaxic device. Postmortem microscopic examination estimated the lesion to be complete in three cases; these cats failed both the 9 vs. 6 and the flux tasks, and did not relearn them. Three cats with an estimate of no more than 1% remaining performed perfectly in the flux task but failed, and did not relearn, the 9 vs. 6 task. For two cats, an estimated 1.5 and 2.0% remained. One performed perfectly on the flux task and relearned the pattern-discrimination task. The other animal had no preoperative training; with only 2% of its ganglion cell output, it learned both tasks after about the same number of training trials the normal cats required. However, the animal failed the tasks when a neutral filter lowered the contrast of the test displays to a level where they were still readily discriminated by the human eye. The optic nerve of a cat is estimated to contain 190,000 fibers (29), thus this animal used only about 8,000 of its normal complement of 380,000 ganglion cells while learning two difficult visual discrimination tasks.

When these experiments were performed in 1967, there was no reasonable explanation for the findings. Visual systems were

then known to be topographic: a point on the retina can be traced to a point in the lateral geniculate and to a point in the cortex. The universal expectation was that a postoperative cat with only 2% of those point to point projections intact would perform poorly; at best, the animal would have limited tunnel vision. For this reason, many agreed that our claim, that such cats behave as though there had been no operation, was preposterous; obviously, mistakes had been made, and the report should be tossed into the dustbin of history.

However, a rational data analysis would conclude that the information cats require for discriminating flux and patterns is distributed throughout the cross section of the optic tract and radiation, and that a small random sample of optic tract fibers—1 or 2%—contains enough of that information to support nearly normal performance on difficult visual tasks. The evidence we have presented here, showing how the retinas of both rats and readers convert visual input information into 300-ms ganglion cell volleys, supports this conclusion. It is certain that every normal 300-ms volley contains all of the information that can be extracted from a particular scene, and, as Fig. 1 demonstrates for the rat, that information traverses the entire pathway and reaches the cortex in its original compacted form, inverted but otherwise mostly unchanged during its synaptic transfer in the lateral geniculate body. Assuming cat and rat visual systems handle the analysis of scenes similarly, a random destruction of cat optic fibers anywhere would interrupt a random fraction of the fibers carrying the information, but the remaining intact fraction would still carry the information.

The behavioral results support the conclusion that the point to point projection information is included in every such fraction. When 2% remain, the topographic information delivered to the cortex enables both the flux- and the pattern-discrimination tasks. When 1% or so remain, it enables flux differences but not the presumably more difficult pattern differences. When no intact fibers remain, the animal is blind. We also compared pre- and postoperative electrophysiological recordings from the cats, and the results provide additional support for the main conclusion: the pre- and postoperative-evoked potential amplitude and waveshape differences recorded at the chiasm, thalamus, cortex, and other sites, were all appropriate for the lesion size and the behavioral discrepancy (27).

The Proposal. This cat experiment supplies the evidence for a second conceptual model of the visual system. Cajal is the icon of the first, or anatomical, model—the wiring diagram, the textbook display of possible synaptic connections, and the logical basis for a cell by cell analysis using microelectrodes. The reading saccade is the icon of the second, or dynamic, model, the one with a substantially different collection of concepts, properties, rules, and activities. The dynamic model is retinocentric; animals with a retina like ours analyze scenes as humans do, whether or not they have complex forebrains. Fish, frogs, snakes, birds, and mammals begin their analysis of the visual world in the same way, with an endless stream of brief ganglion cell volleys containing all of the information recently stored transiently in photoreceptors, or some recognizable variant on this scheme (the frog retina seems to do nothing until a fly-like object moving across the scene begins triggering histograms). When the dynamic system is not operating, as in the stabilized image situation, there can be no visual perceptual experience. On the other hand, when Cajal's system is badly damaged, as in our cats with optic tract lesions, the dynamic system continues to function perfectly, making use of even small surviving fragments of the anatomical system to produce the cat equivalent of our visual perceptual experiences.

Summary and Conclusions

Three Questions Are Answered.

(*i*) What evidence supports the claim that the normal retinal product is a ganglion cell volley lasting about 300 ms?

- Human reading saccades end in fixations about 3 times each second.
- Human saccades, in commonplace situations like driving an automobile, also take place about 3 times each second.
- Normal rat ganglion cell volleys all last about 300 ms.

(*ii*) What evidence supports the claim that human ganglion cell volleys are triggered by changes in the pattern of bleached rods and cones?

- The stabilized image experiment in which an arrangement of mirrors makes it impossible for eye movements to cause an image to move across the retinal surface provides such evidence. The first result is that the scene disappears immediately and the viewer reports a black field. The second result is the viewer instantly sees again if a mirror is moved or if the overall luminance of the scene is changed, both of which change the retinal bleach pattern.

(*iii*) What evidence supports the claim that the visual perceptual experience depends more on the order in which ganglion cell axons leave the retina than on their number?

- Rat and human retinas encode, in about 300-ms ganglion cell volleys, both where and when luminance changes take place on their surfaces, and this information is transferred to the cortex monosynaptically at the lateral geniculate nucleus.
- Cats perform perfectly on difficult visual discrimination tasks with as little as 2% of the optic tract intact.

The Vertebrate Visual System. Vertebrate visual systems perform two major analyses on the scenes its bleached rods and cones display. The first analysis takes place in the retina, the second in central structures such as the tectum, the Wulst in birds, and the visual cortex in mammals.

The most important feature of our dual-analysis model is the major role assigned to the retina. The information in the photochemical portrayal of each scene is converted into the about 300-ms ganglion cell volleys (in mammals) that summarize what, where, and when things just happened in the real world, including movement and the ambient light level. The big difference between the dynamic and anatomical models comes at this point; the dynamic model assigns to the vertebrate retinal neuropile much activity that is conventionally allotted to the mammalian neocortex.

Direct evidence supporting this view comes from the Bullock group (30). In their definitive experiments, a fish optic nerve *in situ* responds to a brief series of flashes delivered at the rate of several Hz; when the flashes stop, a response appears with a latency correct for the stimulus that would have been delivered next but was not. Remarkably, fish eyeballs produce these omitted stimulus potentials (OSP) after the optic nerve is cut, which identifies the retina as a place where the history of a brief stimulus epoch is stored, delivered in the form of the OSP response, and then "forgotten." Bullock *et al.* (31) describe similar human retinal OSPs. Psychologists call such event sequences temporal conditioning, and attribute the phenomenon to activity taking place within the central nervous system.

The remaining evidence for the primacy of the retina is indirect. Thus, although birds lack neocortex, the analysis their retinas produce enables the natural hunting behaviors of eagles, hawks, and falcons, as well as the exquisite visual discriminations pigeons make in psychology laboratories (32). As for fish, one investigator concludes the mechanisms that mediate contrast sensitivity in goldfish are like those other vertebrates use (33), and another shows the goldfish learns conditioned visual behavioral responses even after the optic tectum has been removed (34). The comparison of salamander and rabbit ganglion cell volleys reported by Berry (35) also illustrate the point being

made here; both of those retinas produce similar ganglion cell volleys at about 3 Hz. It seems fair to conclude, even from this limited survey of the evidence, that throughout the vertebrate phylum, the genes guarantee the possibility of a remarkably comprehensive retinal analysis of scenes, and that the mammalian neocortical analysis is a unique elaboration many keen-eyed vertebrates neither have nor need.

In mammals, each retinal analysis initiates a cortical analysis on arrival. An estimate of the information a single retinal synthesis can contain is given by what people report about scenes illuminated briefly; tachistoscopic experiments dating back at least a century reveal that viewers correctly describe at least a few details even when the single volley represents a scene illuminated by a microsecond flash. When, as in reading, the volleys organized after 200-ms exposures arrive at 3 Hz, the retina is successively delivering the maximum information a single ganglion cell volley can contain. The cortex then sequences each new arrival with the earlier ones and creates the running series we know as the visual perceptual experience. In vertebrates lacking cortex, the central structure(s) presumably use comparable mechanisms when acting on the abundant information the retina supplies.

Our dynamic model of the active visual system, which begins with the information-rich ganglion cell volley, has provided a single plausible explanatory hypothesis for the three perceptual experiences this article first singled out: the human reading and stabilized image events and the cat optic tract lesion results. The anatomical model, by contrast, has not been similarly heuristic despite many decades of research using microelectrode evidence. Furthermore, we have probably identified the three features that make the visual systems of both predator and prey so successful: mammalian retinas create a complete orderly analysis of the scene in as little as a third of a second; the sampling rate at which these analyses are delivered to the brain, 3 Hz, is high; and, in the rat at least, each retinal analysis is transferred to the cortex, largely unchanged, at the lateral geniculate synapse. Analogous events in the visual systems of nonmammalian vertebrates remain to be worked out.

Some Practical Consequences. Replacing the wiring diagram model by the dynamic model makes it possible to entertain new explanations for classical human behavioral experiments with ambiguous, multiple, or otherwise unsatisfactory interpretations, and to examine assumptions now commonly made. For instance, psychologists usually begin their experiment assuming the subject is in some kind of visual neutral state, like the runner waiting for the starter's pistol to sound. However, voluntary saccades normally sample the visual environment at rates around 3 Hz, and the retina creates a ganglion cell volley when each one terminates in the fixation; there is every reason to believe this behavior will continue in the laboratory if the subject's eyes are open and the lights are on. Another uncontrolled experimental variable: involuntary saccades frequently move the eyes through excursions large enough to trigger a ganglion cell volley, which means the investigator does not know this is happening if saccades between 1 and 10 arcmin in amplitude are not being measured. Finally, a volley is almost surely triggered when the subject's eye leaves a fixation point, and another one is certainly triggered when the moving eye finds its target. These examples illustrate some of the hazards in supposing the visual system is ever at rest, and suggest it will be prudent to remember, when interpreting data, that unexpected and unwanted ganglion cell volleys can be created at many points during an experimental procedure.

A different example comes from Ditchburn's (12) statement, noted above, that when a stabilized image disappears the entire field "becomes black." One naturally assumes, incorrectly according to Ditchburn's account, that perception of ambient illumination automatically follows receipt by the cortex of the ganglion cell activity continuous illumination produces. That this is not the case raises the likelihood that only the information arriving in organized 300-ms epochs engages the central mechanisms involved in creating perceptual experiences. If this is true, assigning behavioral significance to long stimulus-driven spike trains in microelectrode experiments needs to be reconsidered.

Finally, in experiments where two stimuli are delivered, the advent of the second ganglion cell volley will interrupt the development of the first one whenever the interstimulus interval (ISI) is shorter than 300 ms or so. This inevitable interaction must certainly be noted when sifting through possible physiological explanations for the reports subjects give in ISI experiments.

Cracking the Retinal Code. Retinas encode the information in photoreceptor bleach patterns by the temporal sequence in which excited ganglion cell axons leave the eyeball. What is that code? Tens of thousands of active axons (up to 1 million or more in humans) make up every volley, and no one knows yet which leave first, which last, and whether some respond throughout the entire 300-ms epoch. Our initial speculative answer (1) was based partly on classical morphological facts: the large-diameter optic nerve axons belong to the magnocellular group that, logically, should leave early. We also guessed that the ones leaving late are small axons of the parvocellular group, carrying hue, pattern, and movement information. However, the classical fiber-diameter distinction is unlikely to be highly relevant when the entire complement leaves as a group throughout 300 ms.

Systematic cataloguing of single axon behavior in the optic nerve, tract, or radiation could answer questions like the following. What ganglion cells are involved in the redistribution measured during slow-wave sleep (2)? Movement within a scene will cause minor "smears" on the retinal surface during a 200-ms fixation. Which ganglion cells monitor the size, direction, and velocity of the image motion these smears define? Peck and Lindsley (36) showed "off" units cluster in the second half of a full-field flash response and progressively disappear, the earliest first, as luminance rises. Is this finding the general rule, and what can be said of the on-off fibers?

Actually, the cataloguing is already under way. For instance, Berry's (35) observations on the episodic retinal output of rabbit and tiger salamander retinas is an exemplary first step. Also, measurements by the Schmolesky group (37) on lateral geniculate units indicate that some ganglion cells respond throughout the entire 300 ms, whereas others have sharply restricted temporal niches. Finally, Mehta *et al.* (38, 39) have published many excellent examples of what happens to monkey ganglion cell volleys when, as we claim, they reach their cortical destination.

Remaining Problems

Single-cell responses to stimuli that illuminate only part of the retinal surface (e.g., spots, annuli, bars) do not, in general, respect the 300-ms rule rats and readers obey. This fact raises major problems to solve. The huge literature correlating a small perturbation at one anatomical location with a small reaction elsewhere has developed endless details about the interactions taking place within the anatomical visual system. These details presumably belong somewhere within the dynamic description that originates at the retinal level and spreads wave-like through the morphological road map the microelectrodes explore. The next step toward a satisfactory understanding of the visual perceptual experience should bring the two seemingly conflicting data sets into a single rational scheme.

We thank O. Szabó-Salfay, P. Barabás, J. Pálhalmi, and N. Szilágyi for help in collecting and analyzing the rat data; and G. P. Frommer, S. A. Hillyard, T. Norton, and G. Sperling for helpful comments on the text. This work was supported in part by Hungarian Science Foundation Grant OTKA 628.

1. Galambos, R., Szabó-Salfay, O., Barabás, P., Pálhalmi, J., Szilágyi, N. & Juhász, G. (2000) *Proc. Natl. Acad. Sci. USA* **97,** 13454–13459. (First Published November 14, 2000; 10.1073/pnas.240448697)
2. Galambos, R., Szabó-Salfay, O., Szatmári, E. & Juhász., G. (2001) *Proc. Natl. Acad. Sci. USA* **98,** 2083–2088.
3. Kolers, P. A. (1976) in *Eye Movements and Psychological Processes*, ed. Monty, R. A. & Senders, J. W. (Erlbaum, Hillsdale, NJ), pp. 373–395.
4. Rayner, K. (1998) *Psych. Bull.* **124,** 372–422.
5. Rodieck, R. W. (1998) *The First Steps in Seeing* (Sinauer, Sunderland, MA), p. 562.
6. Land, M. F. (1992) *Nature (London)* **359,** 318–320.
7. Yeh, T., Lee, B. B. & Kremers, J. (1996) *Vision Res.* **36,** 913–931.
8. Ditchburn, R. W. & Ginsborg, B. L. (1952) *Nature (London)* **170,** 36.
9. Riggs, L. A., Ratliff, F., Cornsweet, J. C. & Cornsweet, T. N. (1953) *J. Opt. Soc. Am.* **43,** 495–501.
10. Yarbus, A. L. (1967) E*ye Movements and Vision* (English translation) (Plenum, New York), p. 222.
11. Riggs, L. A. (1941) *Proc. Soc. Exp. Biol. Med.* **48,** 204–297.
12. Ditchburn, R. W. (1973) *Eye Movements and Visual Perception* (Clarendon, Oxford), p. 421.
13. Ditchburn, R. W. (1955) *Opt. Acta (London)* **1,** 171–176.
14. Burbeck, C. A. & Kelly, D. H. (1984) *J. Opt. Soc. Am. A* **1,** 216–220.
15. Olson, J. D., Tulunay-Keesey, U. & Saleh, B. E. (1994) *Vision Res.* **34,** 2907–2915.
16. Brown, D. R., Schmidt, M. J., Cosgrove, M. P. & Zuber, J. J. (1972) *Psychon. Sci.* **29,** 106–108.
17. Blakemore, C., Muncey, J. P. & Ridley, R. M. (1971) *Nature (London)* **233,** 204–205.
18. Pritchard, R. M. (1961) *Sci. Am.* **204,** 72–78.
19. Rozhkova, G. I., Nickolayev, P. P. & Shchadrin, V. E. (1982) *Vision Res.* **22,** 293–302.
20. Bolanowski, S. J., Jr., & Doty, R. W. (1987) *Vision Res.* **27,** 967–982.
21. Gove, P. B., ed. (1993) *Webster's Third New International Dictionary* (Merriam–Webster, Springfield, MA).
22. Armington, J. C. (1972) *Behav. Res. Methods Instrum.* **4,** 61–63.
23. Armington, J. C. (1974) *The Electroretinogram* (Academic, New York), p. 478.
24. Ditchburn, R. W. (1987) *J. Opt. Soc. Am. A* **4,** 405–406.
25. Galambos, R., Norton, T. & Frommer, G. (1967) *Exp. Neurol.* **18,** 8–25.
26. Norton, T., Galambos, R. & Frommer, G. (1967) *Exp. Neurol.* **18,** 26–37.
27. Frommer, G. P., Galambos, R. & Norton, T. (1968) *Exp. Neurol.* **21,** 346–363.
28. Galambos, R., Myers, R. E. & Sheatz, G. C. (1961) *Am. J. Physiol.* **200,** 23–28.
29. Wässle, H., Levick, W. R. & Cleland, B. G. (1975) *J. Comp. Neurol.* **159,** 419–438.
30. Bullock, T. H., Hofmann, M. H., Nahm, F. K., New, J. G. & Prechtl, J. C. (1990) *J. Neurophysiol.* **64,** 903–914.
31. Bullock, T. H., Karamürsel, S., Achimowicz, J. Z., McClune, M. C. & Basar-Eroglu, C. (1994) *Electroencephalogr. Clin. Neurophysiol.* **91,** 42–53.
32. Zeigler, H. P. & Bischof, H.-J., eds. (1992) *Vision, Brain, and Behavior in Birds* (MIT Press, Cambridge, MA), p. 415.
33. Bilotta, J. & Powers, M. K. (1991) *Vision Res.* **31,** 577–585.
34. Schlumpf, B. E. & Davis, R. E. (1985) *Behav. Brain Res.* **18,** 183–199.
35. Berry, M. J., Warland, D. K. & Meister, M. (1997) *Proc. Natl. Acad. Sci. USA* **94,** 5411–5416.
36. Peck, C. K. & Lindsley, D. B. (1973) *Exp. Brain Res.* **16,** 371–382.
37. Schmolesky, M. T., Wang, Y., Hanes, D. P., Thompson, K. G., Leutgeb, S., Schall, J. D. & Leventhal, A. G. (1998) *J. Neurophysiol.* **79,** 3272–3278.
38. Mehta, A. D., Ulbert, I. & Schroeder, C. E. (2000) *Cereb. Cortex* **10,** 343–358.
39. Mehta, A. D., Ulbert, I. & Schroeder, C. E. (2000) *Cereb. Cortex* **10,** 359–370.