Artificial Vision for the Blind: Electrical Stimulation of Visual Cortex Offers Hope for a Functional Prosthesis

Abstract. *Electrical stimulation of the occipital cortex resulted in discrete photic sensations or "phosphenes" in two volunteers who had been totally blind for 7 and 28 years, respectively. Stimulation of multiple electrodes allowed one patient to recognize simple patterns, including letters. Both patients made an uneventful recovery, and the success of these experiments reinforces the hope that a functional visual prosthesis can be developed, although many problems remain to be solved.*

Approximately 110,000 people in Canada and the United States are totally without useful sight, and three times that number have sufficiently serious deficits to be classified as "legally blind." Fewer than 20 percent of those afflicted can read braille, and fewer than 10 percent have achieved mobility by using a cane or other device.

Numerous efforts (*1*) have been made to develop aids for the blind based on conversion of the optical image to auditory or tactile stimuli. However, the limited performance of such conversion devices and the difficulty in training patients to interpret the converted signals have seriously hampered practical applications.

Production of "artificial vision" by interfacing a television camera or similar sensor with the visual cortex might overcome these problems, and first elicited widespread interest because of the pioneering efforts of Brindley and Lewin (*2*). We confirmed and extended many of their results (*3*) in a series of sighted and hemianoptic volunteers undergoing surgical exploration of the occipital lobe for removal of tumors or arteriovenous malformations.

However, experiments (*4–6*) by Brindley and his associates on a second blind subject raised serious questions, including the possibility (*6*) that cortical prostheses might not work in patients who were sightless for more than a few years. Because of the possibility of changes in the visual cortex after deprivation of sight, we believe the feasibility of a visual prosthesis now depends on critical information that can only be obtained from blind subjects. Therefore, we extended our series to include blind volunteers.

Two totally blind subjects were chosen for initial experiments involving temporary implantation of electrode arrays. One patient, a 43-year-old electronics technician and piano tuner, was born with congenital cataracts. His right eye is microphthalmic but has limited motility. It afforded only minimal light perception, which faded until it was "mostly imagination" by late adolescence. The patient could see colors and read headline-size newsprint with the left eye until age 15, when glaucoma, retinal detachment, and hemorrhage necessitated its enucleation. The extraocular muscles were attached to each other at that time, providing a stump with good motility for his prosthesis. The other patient, a 28-year-old graduate student in social work, had normal vision until he lost both eyes at age 21 in a Vietnam landmine explosion. Bilateral corneoscleral nubbins, covered by conjunctiva but with intact extraocular muscles, provide excellent motility for scleral shell prostheses. Both patients experience the spontaneous photic sensations commonly reported by the totally blind, although this activity is more marked in the patient who had been blind for 28 years.

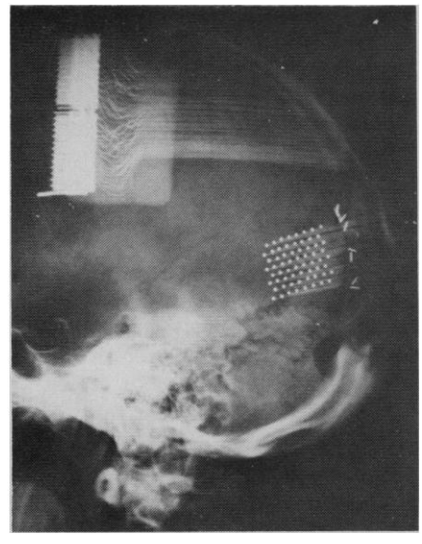

Fig. 1. Lateral skull film of the patient blind for 7 years. The electrode array can be seen in the subdural space, as well as the external portion of the ribbon cable and connector. The array is inserted at a slight angle to the falx, laterally displacing the right occipital lobe by pressure on the mesial surface. Electrodes are numbered in rows parallel to the axis of the ribbon cable. In this picture electrode No. 1 is in the upper right, No. 8 in the upper left, No. 57 in the lower right, and No. 64 in the lower left.

Both subjects had participated in the University of Utah's Artificial Eye Project for more than 4 years before surgery. The known surgical risks, including infection, were fully understood by the patients and their families. They also understood the possibility of unpredictable side effects of prolonged stimulation, although such risks were thought to be small because of experience with our series (*3, 7*) and particularly Brindley's subjects (*2, 4–6*). Both patients realized that such experiments might eventually lead to a useful device, but had no illusions that their participation would directly benefit them in the immediate future. After clearance was obtained from appropriate institutional authorities, formal informed consent was obtained from the patients and their wives in accord with all applicable standards of medical practice (*8*), including the Declaration of Helsinki and the Nuremberg Code.

Protocols, instrumentation, and other techniques were primarily based on a series (*3*) of 37 major neurosurgical procedures on volunteers undergoing operations for other reasons. This series includes work on other neuroprostheses, in particular an analogous auditory prosthesis for the deaf (*7*).

Both electrode arrays consisted of 64 platinum disk electrodes (1 mm^2 in area), arranged in an 8 by 8 hexagonal array on 3-mm centers in Teflon ribbon-cable matrix (*9*). These arrays were designed to be easily removed without reopening the incision. Each was placed in the subdural space, in contact with the mesial surface of the right occipital lobe, after modest parasagittal craniotomies under local anesthesia. To guide placement of the electrodes, stimulation experiments were conducted during the surgical procedure with conventional physiological instrumentation (*3*). The position of the array was similar in both patients (Fig. 1).

During the postoperative period, experiments were conducted with a special system (*10*) consisting of a 64-channel stimulator controlled by a PDP-8 computer. All stimulation and monitoring circuitry connected to the patient was battery-powered. Photoisolation was used to protect against electrical failures in the remainder of the system. The computer-controlled stimulator was connected by a two-way parallel data interface with a GT-40 graphics system, consisting of a modi-

fied PDP-11 computer and cathode-ray tube display. This second computer system was used in real time to display the relative position of responsive electrodes and their thresholds. More important, it was used to prepare a "best fit" map showing the relative position of each phosphene (photic impression) in the patient's visual field. By using a light pen, appropriate phosphenes could be selected by their position in the map displayed on the cathode-ray tube. This information was then transmitted from the graphics system to the computer-controlled stimulator, and the appropriate group of electrodes was automatically stimulated to present patterns.

At the discretion of the experimental team, the entire system could be operated by the patient, who had been trained to control the equipment by a Touch-Tone telephone keyboard interfaced with the computers. This keyboard was also used to transmit coded answers to experimental questions, and audible signals from the Touch-Tone were simultaneously amplified to provide feedback for the patient. The accuracy of their reports was periodically checked by both false stimulations and stimulations without warning, particularly when the system was under the patient's control.

All stimulus parameters and the patients' keyboard responses were automatically recorded by the computer system on a teletype and on magnetic tape. Supplementary data recording was accomplished with the video tape, photographs, and experimental notes.

The patient who had been blind for 28 years reported colorless, flickering phosphenes "about the size of a coin at arm's length" from all responsive electrodes. He had been briefed before surgery about the large elongated phosphenes reported by Brindley's second patient, and denied such phenomena. This is important, particularly if it can be confirmed with additional patients who have been blind for many years. As judged by electrode position (*11*), we believe these phosphenes were elicited from both striate and association areas, although phosphene appearance was uniform. Sixty-three electrodes exhibited electrical continuity, but the responsive area was of limited size, and only 43 adjacent electrodes produced phosphenes in the best position of the array.

The patient who had been blind for 7 years reported two distinct classes of phosphenes. The first group, which we believe were elicited from striate cortex on the basis of electrode position, were colorless. They ranged in size from "a grain of rice at arm's length" up to a "coin at arm's length," with the larger phosphenes appearing in the more peripheral portions of the visual field. Some flickered while others did not, but the patient felt the description of these phosphenes was otherwise similar to those reported by Brindley's first patient (*2*). The second group of phosphenes, which we believe were elicited from surrounding association areas, were all about the size of a coin at arm's length, had a characteristic orange hue, and all flickered. Sixty electrodes exhibited electrical continuity, and all produced phosphenes. Of these, 35 were thought to lie on area 17, and 25 were believed to be on surrounding association cortex.

These results and those on Brindley's first patient (*2*) differ from our experiments on sighted and recently hemianoptic patients (*3*). This suggests a number of cortical changes secondary to prolonged blindness. Blind subjects seem much more likely to experience phosphenes from stimulation of association areas, phosphenes seem more likely to flicker in blind patients, and chromatic effects seem less dramatic or absent after prolonged deprivation of sight.

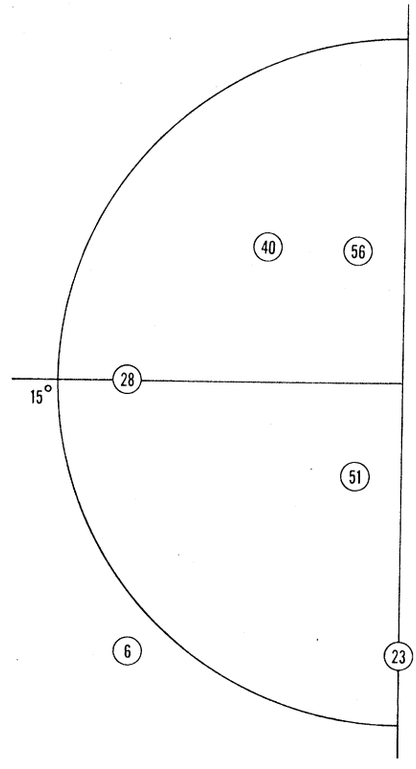

Fig. 2. Absolute map of selected phosphenes in the visual field of the patient blind for 7 years. This shows the general dimensions of the periphery of the map, but detailed interrelations are best determined from the computer graphics displays in Fig. 3.

Stimulation just above threshold produced only a single phosphene for each electrode in both subjects. Stimulation of some electrodes with higher amplitudes in both patients produced a conjugate additional phosphene inverted about the horizontal meridian, possibly due to excitation of one or more overlapping cortical maps. However, this phenomenon had previously been demonstrated (*2, 3*) and was not explored in detail during these experiments.

In both patients, brightness could readily be modulated by changes in pulse amplitude. However, the relation between pulse amplitude and brightness is nonlinear and will require further study. Both patients denied noticeably latency, although phosphene persistence for a minute or more could be caused by stimulation at two to three times threshold. Such persistence after high-amplitude stimulation was quite reproducible and represents another difference between blind and recently sighted patients (*3*). Both patients reported phosphene fading during stimulation with trains in excess of 10 to 15 seconds. This is similar to our previous experience (*3*) but differs from reports by Brindley's first patient (*2*). The patient blind for 7 years reported an initial increase in brightness during the first 3 to 5 seconds of such trains, but the other patient denied this.

In both patients, phosphenes moved with eye movements. This was further explored during stimulation of multiple electrodes in the patient blind for 7 years. He reported that all phosphenes moved proportionally and that their relative position did not change. He also reported that all phosphenes were coplanar, although of undetermined distance, and this was unaffected by eye movements. This agrees with all previous reports (*2, 3*).

Stimulation between each electrode and an indifferent electrode (bovie plate) on the patient's buttocks was alternated with connection of all surrounding electrodes in parallel as the indifferent electrode. This caused slight changes in threshold, but, as expected (*3*), the patients reported no change in subjective sensation.

Symmetrical, biphasic (+/−) pulses coupled through a 1.0-μf series capaci-

tor were routinely used for stimulation (*7*). As expected (*3*), reversal of polarity (−/+) had no effect on subjective sensation. Typical parameters were 0.5-msec duration for each phase (1.0-msec total) at 50 hertz.

Amplitude thresholds were studied by using a computer-controlled binary convergence technique to minimize the number of trials. In the patient who was blind for 7 years, these thresholds were remarkably low (*2*, *3*) and ranged from 0.6 to 2.8 ma (average, 1.3 ma) for the 46 points systematically tested. (Zero-to-peak values are given; multiply by 2 for peak-to-peak values.)

Thresholds for each electrode and the appearance of each phosphene remained constant during 48 hours of repeated testing, until the electrode array moved. Amplitude thresholds for the patient blind for 28 years ranged from 2.1 to 8.1 ma (average, 4.5 ma) for 32 electrodes systematically tested. This was partly due to poor electrode contact in this patient. However, this subject also had substantially higher

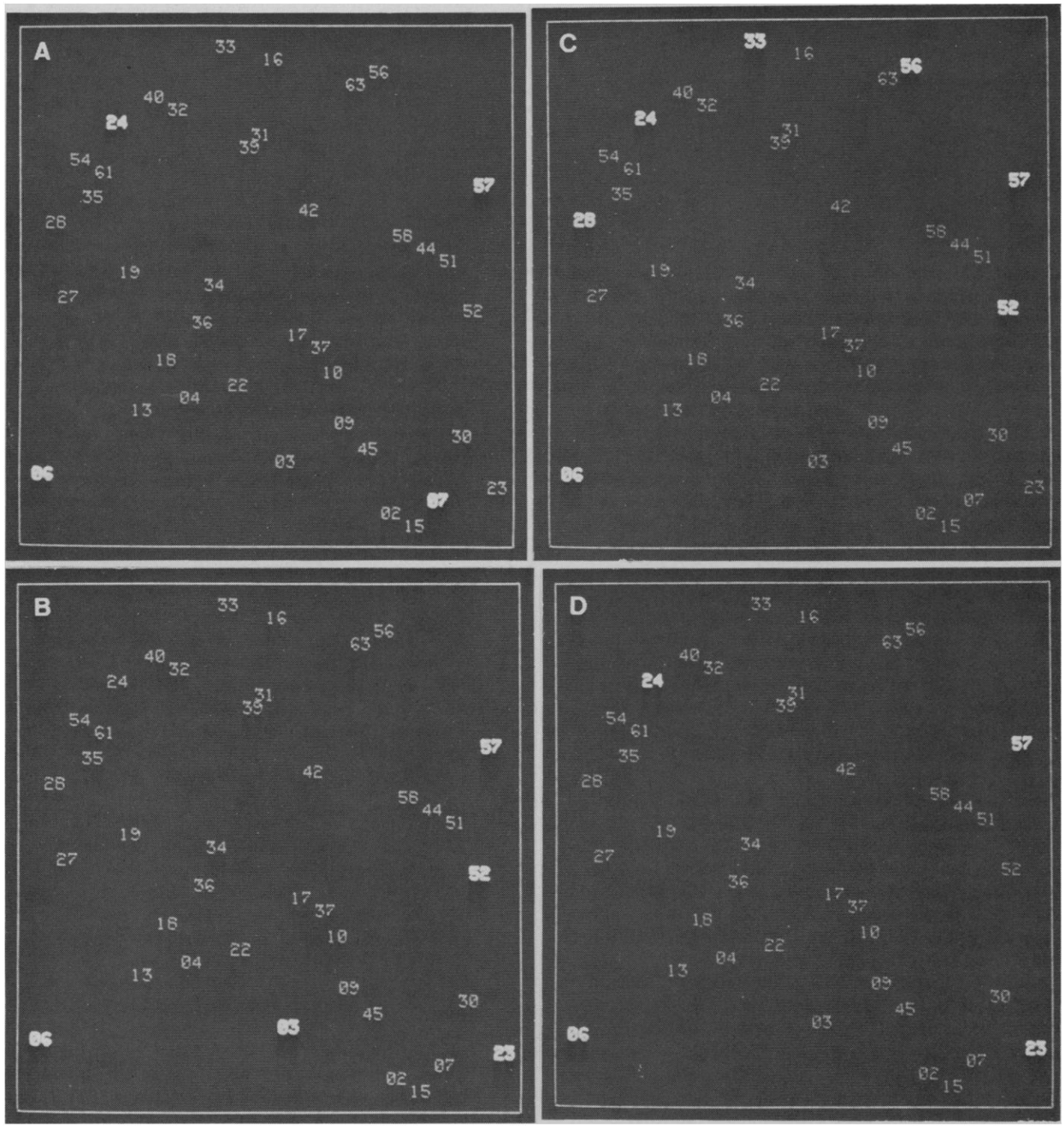

Fig. 3. Montage of four pictures of the cathode-ray tube display, repeating the detailed map of phosphenes produced by each of 39 electrodes, in the patient blind for 7 years. In each case, the brighter numbers indicate the electrodes selected in an attempt to present a particular pattern. The square in (A) and the backward L in (B) were readily recognized. The pattern in (C) was interpreted by the patient as "the letter A with the right leg dropped off." This was due to spurious additional phosphenes in the center of the field, which the patient thought were an attempt to present a crossbar. In (D), an attempt to present a square similar to that in (A) was unsuccessful because the patient was unable to perceive the phosphene we expected to be produced by electrode No. 23 (lower right).

thresholds than the other patient at surgery, when electrode apposition was similar in both cases. The patient with the higher thresholds also had a higher level of spontaneous photic activity than the subject blind for 7 years. Although no interaction between these spontaneous phosphenes and those resulting from stimulation was observed in either patient, the background photic activity may have contributed to the higher thresholds by masking dim, electrically produced phosphenes.

The effects of changing pulse duration, frequency, and train lengths were systematically explored in both patients. No changes in subjective sensation were observed, although thresholds varied as reported (*2, 3*). In the patient blind for 7 years, two electrodes with 0.6-ma thresholds were chosen for testing, and prosphenes were subsequently produced by single pulses at thresholds of 7.8 and 9.3 ma, respectively.

In the patient blind for 28 years, electrode apposition with the brain was poor, and the array did not remain in one position long enough to permit mapping or pattern presentation. In the patient blind for 7 years, the tip of the Teflon strip was wedged against the falx, and the occipital lobe was laterally displaced by the electrode array; excellent mechanical apposition was thus maintained. However, we believe that the difficulties with movement are restricted to our percutaneous electrode arrays. This should not be a problem in a permanent implant, as judged from the stability of the array (*12*) in Brindley's first patient for 6 years.

An absolute map for selected phosphenes was prepared by asking the patient blind for 7 years to place his right thumb on the fixation spot, and point with his left index finger to phosphene positions on a tangent screen (Fig. 2). The area of visual field subtended is very limited, despite the relatively anterior position of the electrodes. Phosphenes near the fixation point were produced by electrodes far from the tip of the occipital pole.

In this patient, detailed maps showing the relative position of each phosphene in the visual field were prepared under computer control by initially comparing the relative positions of selected pairs of phosphenes. Lists of phosphenes ordered in X and Y coordinates were then compiled and displayed on the cathode-ray tube, which provides an adequate approximation of the absolute map (*13*). The patient's responses were transmitted via the Touch-Tone keyboard interface, and the display was updated each time a new point was added to the sorted list. Of 64 electrodes, 4 had broken wires. The other 60 all produced phosphenes, although 21 of these were difficult to distinguish from other phosphenes. The relative map for the remaining 39 points is repeated in photographs of the graphics system display (Fig. 3).

We were then able to present recognizable simple patterns and letters to the patient (Fig. 3, A and B). Up to seven electrodes were used in these experiments, and the patient was repeatedly able to draw, without hesitation, the patterns he saw. Trains of 0.5 to 3.0 seconds were used in pattern presentations. Pulses delivered to each electrode were interlaced, and no two electrodes delivered current simultaneously. Changes in the interlacing interval had no effect on the appearance of the individual phosphenes or on pattern recognition.

Some attempts to present recognizable patterns were unsuccessful, and these failures fell into two classes. First (Fig. 3C), stimulation of multiple electrodes sometimes resulted in spurious additional phosphenes. However, the patient reported that these were generally dimmer than the primary phosphenes, and he felt that he could learn to ignore them. Second, stimulation of other electrode combinations resulted in failure to perceive one or more expected phosphenes (Fig. 3D). The patient indicated that the presence of bright phosphenes made it difficult to distinguish nearby dim ones. This suggests that the brightness of all phosphenes should be carefully equalized by adjustment of pulse amplitude, although this might adversely affect the future use of brightness modulation for presentation of more complex patterns. The patient also felt that we should have restricted presentations to either the colorless phosphenes or the orange ones, instead of intermingling the two groups. However, dynamic scanning of each pattern across the field of phosphenes (*13*) might permit pattern recognition even if all points could not be perceived simultaneously. Consequently, the practical significance of the failure to perceive expected phosphenes is uncertain.

Unfortunately, the electrode array had shifted when testing was resumed on the morning of the third postoperative day. Although the movement was only a few millimeters, a dramatically different detailed map resulted, subtending the same general area of visual field. Further pattern presentation experiments were similar to those reported above. However, it became clear that failures to perceive the expected number of phosphenes had a more complex explanation than just differential brightness, which suggested the possibility of lateral inhibition at the cortical level. Initial experiments suggest that the amount of inhibition may depend on phosphene position in the visual field rather than the geometric position of the electrodes.

After further electrode movement made experimentation difficult, both arrays were easily removed late on the third postoperative day. Both patients were released on leave of absence on the fifth day after surgery and made an uneventful recovery.

W. H. DOBELLE
M. G. MLADEJOVSKY
Neuroprostheses Program,
Institute for Biomedical Engineering,
University of Utah,
19 East Oakland Avenue,
Salt Lake City 84115

J. P. GIRVIN
Departments of Neurosurgery
and *Physiology,*
University of Western Ontario,
London, Ontario, Canada

References and Notes

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Dynamics of Expanding Inhibitory Fields

Glass (*1*) has discussed certain statistical properties of systems in which random initiation of structures is followed by the establishment around each structure of a growing zone of inhibition within which new structures cannot be formed. Glass found that the saturation density of structures (n^*) in systems in which the inhibitory field spreads at a uniform velocity is given by

$$n^* = k_d(F_d/\nu)^{d/(d+1)} \quad (1)$$

where d is the dimensionality of the system, F_d (units: $l^{-d}t^{-1}$) is the rate of random structure initiation, $\nu(lt^{-1})$ is the rate at which the inhibitory fields spread, and k_d is a dimensionless constant which depends on d. Although Glass was unable to find explicit expressions for the k_d, he did find by computer simulation that for $d = 1$, $k_1 \simeq 0.830$.

By attacking the problem in a different manner, I have obtained expressions for the fraction of area covered by inhibitory fields at time t and for the expected density of structures at time t. The value of k_d in Eq. 1 is then easily obtained.

Two cases must be examined. In the first, the fields may interpenetrate freely, and thus do not interfere with one another's growth. In the second, a field stops growing upon contact with the edge of another field: there is no further growth of either field along the arc where they intersect. Fortunately, in the situation in which the fields grow at a uniform rate, the solutions of these two cases are the same. The former case, in which the fields can interpenetrate freely, is mathematically simpler and will be explored first.

Consider first an infinite one-dimensional system. If a line segment of length L is marked off at random on a line containing randomly distributed structures of density λ_1, then the probability R that no structures will be found within the segment is given by the zero-category Poisson term $e^{-\lambda_1 L}$. Equivalently, if structures are being formed along a line at the rate $F_1(l^{-1}t^{-1})$, then the probability that no structures will be formed within the segment of length L in the time t will be $e^{-F_1 Lt}$. If L is not constant, but is a continuously varying function of time, this probability is given by

$$R = \exp\left[-F_1 \int_0^t L(\xi)\,d\xi\right] \quad (2)$$

The integral defines a space-time volume U; R is then the probability that no structure is located in U.

Consider now an infinite one-dimensional system free of structures at time $t = 0$. Note that if no structure is formed closer to a randomly selected point P than a distance $v\xi$ before time $(t - \xi)$, then no field can have reached P by time t. By use of the above arguments, it can be seen that a space-time volume U' can be defined for this problem such that if no structure is formed within U', then point P will be free of inhibitory fields at time t. The volume of U' is calculated in the present case by letting $L(\xi) = 2\nu(t - \xi)$. The probability that no structure will be formed within U' is then given by Eq. 2 provided that all of U' is available for structure formation (that is, that no part of U' is occupied by field associated with some structure outside U'). This condition will always be satisfied if the inhibitory field spreads at a constant rate.

But if the fields can freely interpenetrate, it is also true that the fields associated with any and all structures in U' will reach P by t. Therefore P will not be covered by inhibitory field at time t if and only if U' is free of structures. Thus we can calculate $\phi_1(t)$, the probability that a randomly chosen point is not covered by an inhibitory field at time t, and equivalently the expected fraction of points and hence length not covered by inhibitory fields at time t, by writing $L(\xi) = 2\nu(t - \xi)$ in Eq. 2, obtaining

$$\phi_1(t) = \exp\left[-F_1 \int_0^t 2\nu(t-\xi)\,d\xi\right] = e^{-F_1\nu t^2} \quad (3)$$

We can use Eq. 3 to calculate the density of structures $n_1(t)$ at time t. Since the rate of structure formation per unit length not covered by inhibitory fields per unit time is F_1, it follows that

$$\frac{dn_1}{dt} = F_1\phi_1 \quad (4)$$

so that the density of structures at time t is

$$n_1(t) = \int_0^t F_1\phi_1 dt \quad (5)$$

The saturation density n_1^* is simply the limit of n_1 as $t \to \infty$, so that

$$n_1^* = \int_0^\infty F_1\phi_1 dt = (\pi/4)^{1/2}(F_1/\nu)^{1/2} \quad (6)$$

Thus k_1 in Eq. 1 equals $(\pi/4)^{1/2} \simeq 0.886$, which is in good agreement with Glass's computer estimate (*2*).

This method is readily extended to higher dimensions by replacing $L(\xi)$ by an appropriate higher-dimensional form. For example, in two dimensions F_1 $(l^{-1}t^{-1})$ is replaced by $F_2(l^{-2}t^{-1})$ and $L(\xi)$ by a two-dimensional target area $A(\xi) = \pi\nu^2(t-\xi)^2$. Thus in two dimensions the time course of areal coverage by inhibitory fields is given by

$$\phi_2(t) = \exp(-\pi F_2\nu^2 t^3/3) \quad (7)$$

from which it follows that

$$n_2(t) = \int_0^t F_2\phi_2 dt \quad (8)$$

whence

$$n_2^* = (\pi/3)^{-1/3}(F_2/\nu)^{2/3} \int_0^\infty e^{-\eta^3}\,d\eta \quad (9)$$

where η is a dummy variable.