Burst Firing and Modulation of Functional Connectivity in Cat Striate Cortex


Department of Electrical and Computer Engineering, Vanderbilt University, Nashville Tennessee 37235

Snider, R. K., J. F. Kabara, B. R. Roig, and A. B. Bonds. Burst firing and modulation of functional connectivity in cat striate cortex. J. Neurophysiol. 80: 730–744, 1998. We studied the influences of the temporal firing patterns of presynaptic cat visual cortical cells on spike generation by postsynaptic cells. Multunit recordings were dissected into the activity of individual neurons within the recorded group. Cross-correlation analysis was then used to identify directly coupled neuron pairs. The 22 multunit groups recorded typically showed activity from two to six neurons, each containing between 1 and 15 neuron pairs. From a total of 241 neuron pairs, 91 (38%) had a shifted cross-correlation peak, which indicated a possible direct connection. Only two multunit groups contained no shifted peaks. Burst activity, defined by groups of two or more spikes with intervals of \(\leq 8\) ms from any single neuron, was analyzed in terms of its effectiveness in eliciting a spike from a second, driven neuron. We defined effectiveness as the percentage of spikes from the driving neuron that are time related to spikes of the driven neuron. The effectiveness of bursts (of any length) in eliciting a time-related response spike averaged 18.53% across all measurements as compared with the effectiveness of single spikes, which averaged 9.53%. Longer bursts were more effective than shorter ones. Effectiveness was reduced with spatially nonoptimal, as opposed to optimal, stimuli. The effectiveness of both bursts and single spikes decreased by the same amount across all measurements with nonoptimal orientations, spatial frequencies and contrasts. At similar firing rates and burst lengths, the decrease was more pronounced for nonoptimal orientations than for lower contrasts, suggesting the existence of a mechanism that reduces effectiveness at nonoptimal orientations. These results support the hypothesis that neural information can be emphasized via instantaneous rate coding that is not preserved over long intervals or over trials. This is consistent with the integrate and fire model, where bursts participate in temporal integration.

INTRODUCTION

Current concepts of the receptive field organization and behavior of visual cortical cells have been developed almost entirely from single-unit recordings. In general, these recordings have been interpreted on the basis of average firing rate as the primary response indicator. The historical roots of this approach lie in the demonstration by Adrian and Zotterman (1926) that the amplitude of a sensory signal is well represented by the firing frequency (rate code) of the neuron. This viewpoint has had considerable influence on the interpretation of neural responses and has provided the philosophical framework for the wide acceptance of averaged poststimulus time (PST) histograms as indicators of neural responsiveness. However, the sequential averaging of histograms eliminates any information about the temporal relationships of individual spikes.

Another view suggests that discrete structures within the spike train form a specific code that is relevant to the decision making processes of postsynaptic neurons. The exact means by which information is encoded in these sequences (or even if it is encoded) are being debated (Shadlen and Newsome 1994). Information theory predicts that the more random a signal, the more information it contains, provided the proper decoding mechanism exists (Shannon 1948). Thus the more random the intervals between the spikes and the more precisely these intervals can be detected, the more information a neuron could convey to other neurons. This general principle has stimulated research into the representation of information by specific temporal patterns within spike trains (e.g., Dayhoff and Gerstein 1983a; Lestienne and Strehler 1987; Richmond et al. 1990).

The challenge in the study of pattern-based representation of information lies not only in finding representation schemes but also in elucidating mechanisms by which these schemes can be decoded. Even though more information could be encoded in random patterns that are measured very precisely, the time constants of pyramidal cortical neurons (e.g., 7.3 ± 2.9 ms for dendrites and 16 ± 5.3 ms for the soma; means ± SD) (Kim and Connors 1993) tend to constrain interval-based coding schemes involving very short or extremely long intervals, at least in single neurons. Although longer time constants have been measured, the number of synapse traversals required for cognitive processing tasks (Thorpe and Imbert 1989) puts a practical limit on the integration time allowable at each stage (see DISCUSSION). The fact that transmitter release is a probabilistic function (del Castillo and Katz 1954) adds to the uncertainty that a highly precise mechanism can be used in decoding.

Most previous studies of neural coding (Abeles and Gerstein 1988; Abeles et al. 1993; Dayhoff and Gerstein 1983a,b; Frostig et al. 1990a,b; Lestienne and Strehler 1987, 1988; Stein 1967) have been based on an empiric search for patterns in the output of single neurons, the spike train. A second method for analyzing the neural code is to start at the other end and examine what information a target neuron could reasonably be expected to extract from a single spike train (Bialek and Rieke 1992; Bialek et al. 1991). We have taken this approach to shed some light on the operational transfer of information between two cortical cells. In earlier work (Debusk et al. 1997), we have described how the generation of bursts can be affected by the form of the visual stimulus. Here we demonstrate that burst structures play a significant role in mediating the effectiveness of neural coupling in the visual cortex.

Bursts in the visual cortex were first described by Hubel...
(1959), who remarked on the irregularity of single unit spike trains in awake, behaving cats. Cattaneo et al. (1981a) subsequently found a relationship between clustered spikes (bursts) and stimulus orientation. They showed that with stimuli at optimal orientations, proportionally more spikes were contained in bursts than were found with stimuli at nonoptimal orientations. Debsk et al. (1997) confirmed and extended these results. The reduction of spikes in bursts with nonoptimal stimuli arises from a decrease in average burst length, even after correction for dependence on firing rate. If bursts are important to the transmission of information in the cortex, this result would imply that nonoptimal stimuli result in a less efficient neural code, thereby enhancing the filter characteristics of cortical cells. This has been shown to be the case in the primary auditory cortex, where bursts act to sharpen frequency tuning (Eggermont and Smith 1996).

To test the hypothesis that bursts play a significant role in the transmission of information between cortical cells, we recorded the activity of several neurons simultaneously. Neural pairs that showed coupled activity, as determined via cross-correlation, were analyzed to test the effectiveness of bursts in causing the driven neuron to fire. We found that bursts of as few as two spikes were more effective in eliciting a response in the driven neuron than isolated single spikes and that this efficiency rose monotonically as burst length increased. It is the aim of this article to demonstrate that bursts are an important element in the landscape of neural codes.

**METHODS**

**Preparation**

Six adult cats (2.5–4.0 kg) were prepared for electrophysiological studies following guidelines established by the American Psychological Society and Vanderbilt University’s Animal Care and Use Committee. Each cat was injected intramuscularly with 0.5 ml acepromazine maleate (TechAmerica, Elwood, KS) and 0.5 ml atropine sulfate (Elkins-Sinn, Cherry Hill, NJ). After ~45 min, the cats were anesthetized with 5% halothane (Fluothane, Ayerst, Philadelphia, PA) in O2. After cannulating two forelimb veins, the halothane was discontinued and anesthesia was maintained with intravenous injection of 0.4 mg·kg⁻¹·h⁻¹ of Propofol (Stuart Pharmaceuticals, Wilmington, DE) through one of the canulae. The trachea then was cannulated, and the head was mounted in a stereotaxic device. A small craniotomy (3 × 5 mm) was performed over the area centralis representation in Area 17 (H-C coordinates P4-L2). The dura was incised, and after positioning the electrode over an area free of surface vessels, the hole was covered with agar mixed in mammalian Ringer solution. Melted Tackiwax (Cenco, Chicago, IL) then was poured over the agar to provide an effective hydraulic seal.

For recording, paralysis was induced via intravenous injection of gallamine triethiodide (Flaxedil, American Cyanamid, Pearl River, NY; 10 mg · kg⁻¹ · h⁻¹) through the second cannu. Propofol anesthesia was continued at 0.3 mg · kg⁻¹ · h⁻¹ while the cat was respired at 30 breaths/min with a mixture of N2O:O2:CO2 (70:28.5:1.5%), and pCO2 was held at 3.9%. The heart rate and brain activity were monitored, via electrocardiograms and electroencephalograms, respectively, to ensure anesthetic stability. Accelerated heart rate or lack of occasional slow spindles were treated with bolus intravenous injections of Propofol. Rectal temperature was maintained at 37.5°C with a servo-controlled heat pad. The pupils were dilated with 1% atropine sulfate, and the nictitating membranes were retracted with 10% phenylephrine hydrochloride. Contact lenses with 4-mm artificial pupils were fitted to the nearest 0.25 mm base curve radius, and auxiliary lenses were added as dictated by direct ophthalmoscopy to render the retina conjugate with the viewing screen 57 cm distant. Retinal landmarks (optic disk and area centralis) were projected onto the plotting screen with a reversible ophthalmoscope.

**Stimulation**

Stimuli were presented on a monochromatic CRT display (Tektronix 608; mean luminance 30 cd/m², P31 phosphor) with a 10° circular field and 256-Hz frame rate. Initially in these experiments a Video Monitors 20 inch gray-scale monitor with a 60-Hz frame rate was used, but this was discontinued because the 60-Hz frame rate had the undesirable effect of introducing periodic peaks in the cross-correlation histograms. The periodic peaks resulted from synchronous entrainment of cortical neurons by the screen refresh rate (Snider et al. 1996; Wollman and Palmer 1995). Single-wave gratings were generated with a microprocessor-based pattern generator similar in concept to that described by Milikman et al. (1978). Spatial frequency, contrast, orientation, and drift rate were systematically varied to characterize the spatial filter properties of groups of neurons. Quantitative curves of group activity were based on amplitude threshold triggering. This characterization was aimed at finding stimulus conditions that maximized the firing rate of a group of neurons as opposed to a single isolated neuron.

The receptive field size of the multiunit groups did not appear to be much larger than the receptive fields of single neurons that we have recorded in Area 17. Receptive field dimensions were approximated by rectangular areas determined by manual plotting. The centers of the aggregate response fields were all within seven degrees of area centralis. The areas ranged from a minimum of 3.0 deg² to a maximum of 13.0 deg², with an average of 7.1 ± 3.21 (SD) deg². This is comparable with the receptive fields of single cells measured by Hubel and Wiesel (1959) that ranged in size from 4 to 10 deg².

**Data acquisition**

Multiunit action potentials were recorded with single tungsten-in-glass microelectrodes (Levick 1972) with uninsulated tips 20–30 μm long and 2 μm wide at the base. The neural signal coming from the microelectrode was amplified by 1,000, bandlimited between 100 and 10,000 Hz, and sampled at 30 kHz by a digital signal processing (DSP) board (AT&T DSP32C). The DSP board continuously monitored the incoming signal and when a sampled voltage exceeded >5 SD above (or below) the mean noise level, the sample was presumed to represent an action potential and was time marked for storage. Chebyshev’s Theorem (Walpole and Myers 1985) defines the probability that any random variable X will assume a value within k standard deviations of the mean as

\[
P(u - ks < X < u + ks) \approx 1 - \frac{1}{k^2} \quad (1)
\]

where \( u = \text{mean}, s = \text{standard deviation}, X = \text{random variable}, \) and \( k = \text{number of standard deviations from mean}. \) This holds for any distribution of observations and is a lower bound only. By using a criterion of 5 SD for acceptance, the stored sample had no more than a 4% chance of being noise. On this basis, we feel that all action potentials were recorded in addition to a small amount of noise. To capture the action potential, we saved the waveform from one msec before the trigger point to 3 ms after the trigger point. The sample window thus totalled 4 ms and contained a total of 120 sampled points. The trigger was not reset until the sampled values no longer exceeded 5 SD from the mean.
Classification of action potentials

The data associated with each stored sample window represented the action potential of one of several neurons that were firing during the recording. To analyze the interaction between the neurons contributing to the group activity, the waveform contained within each window was assigned to a specific class, which was associated with the activity of a specific neuron. We used a spike sorting method (Snider and Bonds, unpublished data) to classify the recorded waveforms. The activity of up to six neurons could be recorded simultaneously and subsequently classified but most groups discussed here consisted of from two to four neurons.

The basic strategy of the classification algorithm was first to project each waveform as a 120-dimensional vector, each dimension representing the magnitude of one of the samples. These projected vectors then were grouped into many small clusters, where each point in a cluster represented an action potential. The final step was to resolve which of these clusters could be combined to represent the activity of a given neuron.

We started the classification procedure by partitioning the waveform space into many small clusters using the method of binary tree bisection (Linde et al. 1980). We defined an initial mean vector (the average of all vectors in a cluster) based on all of the waveform vectors. A second mean then was created by adding small random values to the first mean. The vectors were grouped to the closest mean and the two means were updated as the average of the grouped vectors. This process was repeated until convergence. These means then were split into two and the process was continued. Once a given waveform vector was assigned to a particular mean, it stayed with that particular mean’s descendants. In practice we typically split the data six times for a total of 64 cluster means representing ∼50,000 action potentials.

Cluster consolidation was based on the underlying assumption that if an action potential changes shape, it does so gradually and should leave a continuous trail of points as it moves through the 120-dimensional waveform space. Membership of two small clusters in a larger smeared cluster would be indicated if the density of points falling between these two clusters changes in a linear fashion from the density of the first cluster to the density of the second cluster. We first determined local densities by calculating how many points fall in an area that is one standard deviation away from the mean of each cluster. The points between the clusters were assumed to be random variables with probability \( P \) of falling between the clusters and probability \( q \) of falling within the clusters. This is a binomial distribution, which can be approximated by the standard normal distribution. The \( z \) score specifies the area under the standard normal distribution and thus can be interpreted as a confidence interval. By using the \( z \) score of the standard normal distribution, we calculated the difference between the actual number of points falling between the two clusters and what we expected if the clusters are linked. If the number of points on the trajectory between clusters resulted in a value that is equal to or above a set threshold \( z \) value, we then concluded that the two clusters belonged to a single distribution and combined them. By plotting the number of clusters versus \( z \) thresholds, we typically found a plateau in the number of clusters that spanned a range of threshold values. In practice this plateau represented a robust threshold representing reasonably separated clusters even though the clusters could have arbitrary boundaries.

If more than one action potential peak was detectable within a 4-ms window, each waveform was projected as a separate event, which permitted independent identification of some events within the window. A small number of sample windows were not unambiguously classifiable, presumably due to overlap of spike waveforms or excessive noise. These windows typically represented only 1–3% of the data and were discarded. Discarding overlapping spikes had minimal effect on our results. We were primarily interested in action potentials that had a time-delayed coincidence of 3–12 ms and overlapping spikes had a time-delayed coincidence of ∼0–2 ms.

Cross-correlation

We used the cross-correlation histogram (Perkel et al. 1967a,b) to determine putative connectivity between spike train pairs. The cross-correlation histogram was constructed by selecting each spike from neuron A and making a histogram of the intervals to all spikes (within, say, ±100 ms) from neuron B. If the spike trains from neurons A and B are independent, then the resulting histogram should equal the constant value of 1/\( \mu_b \) where \( \mu_b \) is the mean interval between spikes from neuron B. Peaks (valleys) in the cross-correlation histogram indicate temporal relationships that are more (less) probable than independent behavior indicated by a flat value of 1/\( \mu_b \). If both neurons are excited from a common source, a peak occurs centered at a time lag of zero. If neuron A directly excites neuron B, then a shifted peak occurs and the shift corresponds to the time it takes for neuron A to affect B (Moore et al. 1970).

It is possible that, in addition to direct excitation between neurons, common excitation of two neurons can occur as a result of both responding to a particular stimulus. This response should not be misinterpreted as indicating direct connectivity between the neurons. Common excitation usually is synchronized with the stimulus over a period of time that is relatively long compared with the simultaneous excitation due to connectivity, which is found over the order of milliseconds. The difference in time scales permits the effects of common excitation to be removed. This usually is accomplished by subtracting the shift predictor from the original cross-correlation histogram. Here we corrected for common excitation using the method of Aertsen et al. (1989). We first created a “raw” joint poststimulus time histogram (JPSTH). This is a two-dimensional histogram in which each bin represents a delayed coincidence of the two spike trains averaged over all stimulus presentations, and is represented by \( \langle x(s)y(t) \rangle \).

As in the cross-correlation histogram, this JPSTH will contain contributions from both common stimulation and coincident firing due to coupling. The contributions that come from stimulus-related modulation of the joint histogram can be predicted. We begin by making PSTHs representing responses of individual cells averaged over \( K \) stimulus presentations

\[
\langle x(t) \rangle = \frac{1}{K} \sum_{k=1}^{K} x_k(t)
\]

where \( x_k(t) \) denotes the response of neuron \( x \) at bin \( t \) of sweep \( k \) and \( K \) represents the set of all sweeps. The only restriction is on the binwidth, which is chosen such that in each trial we collect at most one spike per bin. The JPSTH predictor is created by taking the cross product matrix of the individual PSTHs representing the responses from two cells, \( \langle x(s)y(t) \rangle \), and represents the null hypothesis of no interaction between the neurons. To compare the difference between the raw JPSTH and the JPSTH predictor, we just subtract the two

\[
D_{x,y}(s,t) = \langle x(s)y(t) \rangle - \langle x(s) \rangle \langle y(t) \rangle
\]

The quantity \( D(s,t) \) is the cross-covariance matrix of the spike trains \( x \) and \( y \), each collected over all presentations. In terms of neural responses (Aertsen et al. 1989), the first term on the right represents the joint PST histogram that was developed directly from the data and the second term represents the JPSTH that would be predicted if neurons \( x \) and \( y \) were firing independently even though responding to a common source. The values of \( D \) cannot, however, be a good indication of the connectivity of the neurons \( x \) and \( y \) because they depend on the firing rate. To compensate for
this, Aertsen et al. (1989) propose the use of the normalized PST histogram

\[ C_{x,y}(s,t) = \frac{D_{x,y}(s,t)}{\sqrt{D_{x,x}(s,s)D_{y,y}(t,t)}} \]  

(4)

The denominator can be shown to be the standard deviation of the predicted PSTH. If the normalized cross-covariance matrix now is integrated along the principal diagonal \((s = t)\), the result is the normalized cross-correlation histogram, which is the measure of correlated firing that we used in this study. This approach of constructing the cross-correlation histogram is equivalent to using the standard shift predictor histogram which has been averaged over the set of all possible shift predictors, i.e., all different orders of shift including zero, which gives a more robust statistical result than when using the ordinary shift predictor.

RESULTS

Temporally related spike pairs

We examined the corrected cross-correlation histograms for peaks that were shifted from time lag zero. This shifted peak strongly indicated a direct causal relationship between two neurons, i.e., excitation from one cell contributing to the action potential from a second cell. There were 241 neuron pairs in the 22 multunit groups recorded. These groups yielded collectively 56 individual neurons. A multunit group typically showed activity from two to six neurons and thus contained between 1 and 15 neuron pairs. Of the 241 neuron pairs, 91 of these pairs (38%) had a shifted cross-correlation peak, which indicated a possible direct causal connection. Only two multunit groups contained no shifted cross-correlation peaks. Of the 91 pairs that showed a causal connection, 82 (90.1%) showed complex-to-complex interactions, where a complex cell participated in the firing of a complex cell, 2 (2.2%) showed simple-to-simple interactions, 2 (2.2%) showed simple-to-complex interactions, and 5 (5.5%) showed complex-to-simple interactions.

Figure 1 shows examples of typical cross-correlations that we found under different stimulus conditions. The cross-correlation peak is not restricted to one specific latency, but rather is spread out between 3 and 12 ms. Miles and Wong (1986) found a latency to peak of excitatory postsynaptic potentials (EPSPs) of 7–12 ms. Their measurement was done in slices from guinea pig hippocampus, where activation stemmed from a single presynaptic neuron or antidromic activation that activated only part of the CA3 population. We believe that our latency range encompasses shorter values because spikes were presumably generated not only by temporal summation but also by synchronous spatial summation. With an intact preparation and optimal stimulation conditions, the postsynaptic cell receives input from many active neurons in addition to the one we identified as presynaptic. Activity from these other cells also contributes to the depolarization of the postsynaptic cell, thus increasing the probability that the rising phase of a single EPSP will trigger a spike.

Figure 1A shows the responses to presentation of nine orientations. There is a pronounced peak at both 10 and 20°, but at 40 or 350° this peak has disappeared even though there remains some presynaptic activity. The average firing rates of the two analyzed neurons are shown in the figures as \(X:Y\) where \(X\) is the firing rate of the driving neuron and \(Y\) is the firing rate of the driven neuron. Although the coupling is to some extent dependent on the absolute firing rates (averaged across the presentation), it should be noted that these cross-correlation histograms have been normalized against firing rate (Eq. 4) using the method of Aertsen et al. (1989) to enable direct comparisons of synaptic efficiency independent of firing rate. The decrease in coupling is thus not just due to a decrease in the number of spikes available for analysis, rather the effectiveness of the driving neuron in causing the driven neuron to fire also has decreased. Local dependence of coupling on changes of firing rate within a given response period was possible, but it is likely that any such changes were not organized. Most recorded cells were complex, and the response was simply a generalized elevation of firing rate, with no temporal features associated with stimulation.

Figure 1B is another example of the responses to different orientations though with lower firing rates for both driving and driven neurons. The coupling between the neurons as a function of orientation is similar to that seen in Fig. 1A. The reduction in coupling at nonoptimal orientations is likely due in part to the reduction of aggregateafferent signals at nonoptimal orientations. There are other factors that can influence this coupling, including reduced burst length and inhibition, that will be investigated later in the paper.

Figure 1C shows the coupling as a function of spatial frequency. The dependence of coupling on spatial frequency is typically not as pronounced as the coupling change with varying orientation. Figure 1D shows how the coupling changes as a function of contrast that is presented in a stepped sequence of rising then falling values. This stimulation paradigm is to show the dynamic nature of contrast gain control (Bonds 1991). Here again, the degree of coupling seems less dependent on firing rate than was found with variation of orientation. The relationship between particular kinds of stimulation and coupling will be considered in more detail later.

Burst analysis

Analysis of spike intervals from visual cortical cells does not yield a uniform or even Poisson distribution of the interval values. Most cells produce a bimodal distribution with a prominent peak at short intervals and a lower, broader peak or plateau at longer intervals (Debusk et al. 1997; Gray and McCormick 1996). Figure 2 shows an example of this behavior in three cells. The large peak at ~3–5 ms drops off steeply, and frequently there is a noticeable dip in the region of 10–12 ms that precedes the broader peak at 15–30 ms. As proposed by Cattaneo et al. (1981a,b) we define bursts to be any group of two or more spikes that have intervals of ≤8 ms, corresponding to spikes contained in the peaks near the origin. This criterion is consistent with the falloff seen in the histograms and also has been used by Mandl (1993) in cat superior colliculus and by Bair et al. (1994) in monkey medio-temporal (MT) cortex.

We found that across our entire sample (56 cells, 2,792,593 spikes) an average of 60.5% of the spikes were contained in bursts. This value is higher than the figure of...
FIG. 1. Cross-correlations as a function of parametric variations of the stimulus. In all cases, the magnitude of the cross-correlation function is normalized to the number of spikes and is therefore independent of firing rate. Total measurement duration for each stimulus condition was 100 s (10 10-s sweeps). Average firing rate of the pre- and postsynaptic cells is shown (at left of each histogram) for each example by pre:post. Degree of correlation is more sensitive to stimulus change when orientation is varied (A and B) than when spatial frequency (C) or contrast (D) is varied.

50% of 507 cells having ≥ 42% of their spikes in bursts found by Debusk et al. (1997). However, the multunit activity in the experiment reported here was recorded mainly from the supragranular layers. Gray and McCormick (1996) reported a tendency for bursting cells to be located in the upper laminae. The lower figure cited by Debusk et al. (1997) is probably the result of averaging activity over all layers. Similar levels of burst activity also are found in other cortical areas. Eggermont and Smith (1996), defining bursting as spikes that could not be predicted by a modulated Poisson process, found that in the auditory cortex 54 ± 11% of spikes were contained in bursts.

Neural coupling

The delayed peak found in the cross-correlograms of 91 neuron pairs suggested strongly that spikes from the first neuron contributed to the decision to fire by the second neuron. These pairs were analyzed further to see how bursts in the spike train affected the transmission of a spike from one neuron to the next. To quantify the strength of the coupling between a pair of related cells, Levick et al. (1972) defined effectiveness (or efficacy) (Aertsen et al. 1989), which is the percentage of spikes from the presynaptic neuron (of all of its spikes) that are time related with spikes of the postsynaptic neuron. It is important to note that this definition is based purely on the statistics of the linkages between two cells and does not directly address the actual mechanisms involved in spike transmission. Levick et al. (1972) defined effectiveness for spikes that were related by a precise time delay. In most cases, generation of a spike in pyramidal cells requires integration of a number of input events over several milliseconds (Miles and Wong 1986). We therefore modified the previous definition of effectiveness. Instead of a precise time delay, we used a time-delayed window, where the width of the window was determined from the width of the peak in the cross-correlation histogram. For example, if the peak in the cross-correlation histogram started at 5 ms and ended at 8 ms, then any spike from the second neuron that had a time lag of 5–8 ms after the first neuron’s spike was considered to be time related to the first neuron’s spike. The criterion for correlation in the 91 pairs judged to be coupled required a positive peak that remained above the noise floor for at least four 1-ms bins.

Effectiveness of bursts

Figure 3 diagrams the populations of bursts of varying length as well as their effectiveness. In our sample, the number of single spikes approached one million (1st column).
The dark gray bars represent the number of bursts of a particular length and the superimposed light gray bars represent the number of those bursts that were effective in eliciting a spike from the driven neuron. The effectiveness of each burst length is represented by the percentage above the dark gray bars. The longer bursts are much more likely to cause a spike in a postsynaptic cell than shorter bursts, but their overall impact is much less because there are fewer long bursts. In general, bursts were short. Figure 3, inset, plots the cumulative probability of events, where an event is either a single spike or a burst, as a function of event length. About 62% of the total events were single spikes with two-spike bursts.
bursts adding another 31%. Bursts of five spikes or less (including single spikes) comprised 97.7% of the data and bursts of \( \geq 10 \) spikes (including single spikes) account for nearly all the data (99.7%).

Figure 4 is a direct comparison of the effectiveness of bursts in transmitting a postsynaptic spike as a function of burst length. This distribution was calculated from our entire database, consisting of 588,434 bursts of various lengths together with 976,424 single spikes. The effectiveness of single spikes averaged 9.5%. This means that across 91 cell pairs 9.5% of the presynaptic single spikes from a given cell contributed to the firing of the particular postsynaptic cell under study. A burst was considered effective if any of the spikes in the burst were time related to a spike of the driven neuron. For bursts of two or more spikes, effectiveness increased markedly. The relationship is very nearly linear \((r = 0.9982)\) from an effectiveness of 15.0% for 2-spike bursts to 46.6% for 10-spike bursts. This is comparable with records from hippocampal pyramidal cells, where single spikes were 5% effective (Miles and Wong 1986) and presynaptic bursts ranged in effectiveness from 30 to 50% (Traub and Miles 1991). We found that the average effectiveness of all bursts was 18.5%, about twice the effectiveness of single spikes. This is less than the effectiveness averaged between 2- and 10-spike bursts because of the far greater numbers of bursts with lower spike counts. Altogether, \(~54\%\) of the correlated postsynaptic spikes were associated temporally with bursts.

One might question whether the increase in effectiveness found with longer bursts results simply from the fact that longer bursts have more spikes. This would lead to an increase in the probability of classifying any spike from the postsynaptic neuron as time related with a presynaptic spike. If this was the case, then any spike in a burst would have equal probability of correlating with a postsynaptic spike. To test this possibility, we examined how the order of spikes within a burst related to excitation of a postsynaptic spike. We first identified the bursts associated with at least one postsynaptic spike and sorted them with respect to burst length (number of spikes). We then summed the total number of effective presynaptic spikes for each burst length. Figure 5 shows the fraction of the total effective spikes represented by each spike in a burst sequence of a given length. Several of the spikes within a presynaptic burst could be time related to the same postsynaptic spike because the time windows (3–12 ms) were longer than the average time between spikes in a burst (4–12 ms). Because shorter bursts last only 4–8 ms, in many cases, we could not unambiguously resolve whether any particular spike within those bursts was more effective than the others; this resulted in equal probability with respect to their contribution toward inducing a postsynaptic spike. For the longer bursts, the later spikes in the burst were more likely to result in a postsynaptic spike. For example, the last spike in a five-spike burst is almost twice as effective (28%) as the second spike (15%).

The data in the figure are very linearly correlated \((r = 0.9999)\), with a range from 4.3 ms for a 2-spike burst to 36.8 ms for a 10-spike burst. Across all bursts, the average time between spikes is 4.1 ms, which is well within the time constants of pyramidal neurons (e.g., 7.3 ± 2.9 ms for dendrites and 16 ± 5.3 ms for soma) (Kim and Connors 1993) and thus is suited ideally for burst summation at the postsynaptic site.

We also looked to see if bursts were not only effective in eliciting a single post synaptic spike but also caused the target neuron to burst as well. We built cross-correlation histograms that treated bursts as single events. No consistent pattern of bursts begetting bursts was found.

**Burst effectiveness for optimal versus nonoptimal stimulus conditions**

The above measurements, showing that bursts enhance coupling effectiveness, were based on responses to all stimuli. Because Cattaneo et al. (1981a) showed that more spikes were contained in bursts for optimal stimulus conditions than for nonoptimal conditions, we expected to see a dependence between coupling effectiveness and the stimulus configuration. We examined this question by first driving neurons with optimal (or nearly so) stimuli with optimality being defined by stimuli that yielded the greatest firing rate within the ensemble. We were recording from groups of cells with receptive fields that were concentrated within an area averag-
ing 7.1 deg², and independent stimulus optimization for each cell was not possible. Although spatial selectivity within a given group differed in detail, most of the cells detected at a single recording site tended to have similar spatial tuning properties (Gawne et al. 1996). This presumably occurred because we were recording group activity from a single microelectrode with a region of sensitivity that did not spread significantly beyond a given organizational column. Figure 7A shows the orientation tuning curves for three multiunit groups each consisting of two or three cells. The neurons in each group show peak firing rates at about the same orientation, although these firing rates are different. Figure 7B shows the spatial frequency tuning curves for three multiunit groups. Within each group of from two to four cells, the peak firing rates for each cell within the group result from spatial frequencies within 0.1 cycle/deg of one another.

Using the preceding definition of optimality, we compared the effectiveness of bursts at the optimal stimulus condition to the nonoptimal conditions for orientation, spatial frequency, contrast presented randomly, and contrast presented sequentially. The nonoptimal response was averaged over all the nonoptimal stimulus conditions and the optimal response was averaged across all of the optimal stimuli. The result can be seen in Fig. 8. The effectiveness of bursts in responses to optimal and nonoptimal stimuli was similar in that for both effectiveness rose monotonically with burst length. However, for nonoptimal stimuli, the effectiveness was lower at all burst lengths by a nearly constant amount averaging 5.84% (absolute level of effectiveness). This shift fell outside the 95% confidence level for the standard error of the mean.

The decrease in effectiveness for nonoptimal stimuli could simply result from a decrease in overall firing rate. We consider it highly unlikely that a postsynaptic spike is the result of one or even several spikes from a single presynaptic neuron. Rather, it is the consequence of the aggregate activity of both the presynaptic cells that we can see (via recording) and those we cannot see. A general decrease in firing rate lowers the statistical expectation that other cells will be firing in concert with the presynaptic cells that we can observe, which leads to a decrease

**FIG. 5.** Distribution of effectiveness within bursts. This figure describes the likelihood that a particular spike in a burst will result in a postsynaptic spike in comparison with other spikes in the same burst (total probability = 1). Duration of 2-spike bursts is so short that either spike could be related causally with a postsynaptic spike, so probabilities are equal. As the duration of bursts grow, it becomes apparent that the later spikes in a burst are more effective in eliciting a postsynaptic spike, presumably due to temporal summation.

**FIG. 6.** Burst duration as a function of the number of spikes in a burst. Average interval within a burst is 4.08 ms, and the relationship is highly correlated ($r = 0.9998$) for bursts of ≤10 spikes.
in the overall probability of postsynaptic spikes occurring. To examine whether the change in effectiveness with different stimuli was related solely to firing rate, we constructed plots of average effectiveness for responses to optimal versus nonoptimal stimuli across different stimulus classes, including variations in orientation, spatial frequency, contrast presented randomly, and contrast presented in sequential order (rising, then falling) to measure contrast gain control (Bonds 1991). The comparison across different stimulus modalities was made so that we could differentiate the changes in effectiveness resulting from modulation of firing rate via different mechanisms.

Figure 9 shows the change in effectiveness (in absolute percent) as a function of the change in firing rate of the presynaptic cell, averaged across optimal and nonoptimal stimulating conditions, for each stimulus class. The points representing both sequenced and randomized contrast presentations show moderate absolute losses in effectiveness across a broad range of response reduction. We believe that the greater response reduction seen with randomized contrast (across the same set of contrasts used for sequential contrast presentations) results from cells undergoing less contrast adaptation on average for sequentially presented stimuli than for randomly presented stimuli, especially at lower contrasts. Nonoptimal spatial frequencies yield slightly greater losses of effectiveness than low contrasts, but the difference is not significant.

When compared with the results from variation of contrast and spatial frequency, the variation of orientation causes a clearly different impact. Across all nonoptimal orientations, the firing rate decreased an average of 30.8 spikes/s and the effectiveness dropped by 6.9% (in absolute effectiveness, not a relative change). This is almost double the average decrease in effectiveness (a drop of 3.6%) seen with, e.g., randomized contrast, even though the decrease in firing rate for lower contrasts, at 36.7 spikes/s, was greater.

These results suggest that the reduction in transmission efficiency resulting from changing orientation involves
For variation of orientation, the driving neuron is consistently most effective at the greatest firing rate and when the burst length is the longest. The effectiveness drops off quite rapidly with a decrease in firing rate, which in this case is the result of stimulation with orientations to which the cell responds less robustly. Longer bursts are generally more effective than shorter bursts irrespective of firing rate, although at low firing rates all bursts are ineffective. The increase in effectiveness is much more pronounced for longer bursts than for shorter bursts as the firing rate is increased. This is most likely due to the fact that neighboring neurons are much easier to recruit (or synchronize) when they are excited with their preferred orientation, represented by the highest firing rates and longest bursts.

This characteristic behavior changes dramatically when the neural pairs are being excited by their preferred orientation but with their firing rate being modified by variation in the contrast. In this case, the driving neuron is much more effective at low firing rates (regardless of burst length) than at these same firing rates when they result from nonoptimal orientation. From a functional standpoint, effectiveness is required at low contrasts because the visual cortex needs to recruit (or synchronize) neighboring cells to process the visual scene on the basis of sparse information. This requirement would be less desirable with firing rates that were low due to a nonpreferred orientation because the cortex then would be linking or recruiting cells that were carrying less relevant visual information. As the firing rate increases, the effectiveness also increases but only to a certain point. At moderate contrasts, contrast gain control (Bonds 1991; Ohzawa et al. 1985) is activated and drives down the overall effectiveness, presumably to prevent overdriving the postsynaptic neuron. This gain suppression is seen in the contrast plots as a clear dip beyond the peak found at moderate firing rates. This peak is not evident in the plots parametric on orientation; the plots were measured at a fixed contrast.

A more detailed way of showing the difference between the influences of orientation and contrast on effectiveness is to compare slices through the two surfaces at similar firing rates. Taking one of the cells found in Fig. 10 (2nd row), we chose a nonoptimal orientation that caused the cell to fire at \( \sim 45 \) spikes/s. We then found the closest contrast value that caused the same cell to fire at nearly that same value (44 spikes/s). At this slice in firing rate, we plotted the effectiveness as a function of burst length for both orientation and contrast. This was done also for the cell in the bottom row of Fig. 10 but at a lower firing rate. Results are shown in Fig. 11, which displays the actual data points instead of the fitted surfaces found in Fig. 10. There is a general tendency for effectiveness to rise with burst length. The decrease for long bursts in Fig. 11B is a result of a low count of bursts for these lengths. In both cases, the effectiveness of nonoptimal orientations ( - - - -) is less than that of low contrasts (——) at all burst lengths even though the curves for orientation were taken at a greater firing rate than for the contrast curves. The reduction of effectiveness for nonoptimal orientations most likely reflects a stimulus-related variation in the postsynaptic membrane potential that

![Image](http://jn.physiology.org/)

**FIG. 9.** Change in effectiveness vs. change in firing rate of the presynaptic cell resulting from variation of different stimulus parameters. Each point represents the average reduction of both firing rate and effectiveness for stimulation with nonoptimal orientations, spatial frequencies and contrasts (both randomized and sequenced presentations), as labeled. Dependence of effectiveness on firing rate can vary markedly depending on which stimulus property is varied.

some process beyond that which reduces effectiveness when contrast is reduced but orientation is optimal. Effectiveness clearly depends on burst length, and burst length has been shown to be dependent on at least two factors. Within the range of moderate contrasts, burst length is more or less proportional to firing rate when contrast is varied at the optimal orientation. When the firing rate is varied by driving the cell with nonoptimal orientations, average burst length is shorter than that found at the same firing rate (but lower contrasts) at the optimal orientation (Debusk et al. 1997). We thus would expect to see effectiveness reduced more at nonoptimal orientations as a result of shorter average burst lengths. It is also possible that reduced effectiveness at nonoptimal orientations could result from a systematic modification of the response waveform, which could disproportionately reduce transmission efficiency without changes in overall mean firing rate.

To isolate the dependency of effectiveness on burst length at optimal and nonoptimal orientations, we created three-dimensional plots for variation of both orientation and contrast. Orientation was systematically varied across 10 different values centered on the optimal orientation with 10 presentations at each specific orientation in a random sequence. Thus there were 100 presentations, each 10 s long. For each presentation, we calculated effectiveness, average firing rate and average burst length. This resulted in 100 points to plot, each point represented in three dimensions. To clarify the trends in this three-dimensional space, we fitted a two-dimensional surface representing a polynomial of order five in both dimensions to these points. Figure 10, left, shows the surface plots of effectiveness in three neural pairs as a function of both burst length and firing rate resulting from varying orientation. Plots from the same cells resulting from randomized variation of contrast are shown in Fig. 10, right.
FIG. 10. Effectiveness as a function of burst length and firing rate of the presynaptic neuron. Left: firing rate is varied by varying stimulus orientation and contrast is held constant. Right: firing rate is varied by varying contrast and the orientation is optimal and constant. Meshes are 5th-order polynomials fit to the actual data to clarify general trends. In general, low firing rates resulting from nonoptimal orientations are much less effective than those same rates resulting from low contrasts.

is in addition to dependences on presynaptic firing rate and burst length but the actual mechanism remains unknown. We suggest that this additional mechanism is likely to involve inhibition (Berman et al. 1991; Bonds 1989; Bush and Sejnowski 1994; Douglas and Martin 1991; Li et al. 1960; Morrone et al. 1982; Sillito 1975). If there was not some type of inhibitory mechanisms controlling the difference between contrast and orientation, then the curves based on variation of orientation, measured at greater firing rates, should show greater effectiveness not less.

FIG. 11. Detail of effectiveness as a function of burst length with variation of orientation and contrast. These 2 examples represent slices taken from the 3-D plots of Fig. 10, 2nd (A) and 3rd (B) rows. Even though for each example the firing rate from variation of contrast (○) is lower than that from variation of orientation (□), the effectiveness at the optimal orientation (varied contrast; ○) is higher at all burst lengths.
Contrast gain control

Bonds (1991) demonstrated the dynamic nature of contrast gain control in cortical neurons of cats by presenting contrast values sequentially in first increasing then decreasing logarithmic steps. All cortical cells show response hysteresis, where the response to a given contrast is lower when it is preceded by a higher contrast than when it is preceded by a lower contrast. The adaptive effects of higher contrasts are seen nearly instantaneously but take several seconds to subside. The active gain control mechanism observed in Fig. 10, right, can be more clearly seen in Fig. 12 where the responses and effectiveness found with sequentially varying contrast are superimposed. Each contrast was presented for 10 s, and these curves have been normalized to facilitate comparison. The response plot peaks at 40% contrast and drops slightly at 56%, indicating some supersaturation (Li and Creutzfeldt 1984). Presentation of 40% contrast immediately thereafter results in a response amplitude that is only 64.7% of the peak value, and responses to successive presentations are also attenuated down to 3% contrast. On the other hand, effectiveness peaks at only 20% contrast, falling to 79.5% of its peak value at the response peak at 40% contrast, and is only 68.3% of its peak at the highest contrast of 56%. One should note that the peak effectiveness is seen where the slope of the response versus contrast curve is highest, and the deceleration of this curve is reflected by a decrease in effectiveness. As the state of adaptation recovers on presentation of lower contrast levels, effectiveness also recovers, with some restoration of both response and effectiveness at 14% contrast, and preadaptation levels appearing at ~7% contrast. We thus conclude that contrast adaptation in cortical cells is dependent on modulation of the effectiveness with which presynaptic spikes evoke postsynaptic spikes, although this may not be the only factor. The physiological mechanism underlying this modulation remains unclear, but tonic hyperpolarization in the postsynaptic cell may be involved (Carandini and Ferster 1997).

Discussion

Modulation of effective coupling

We have shown that the more spikes a burst contains, the more effective this burst is in eliciting a time-related spike from a driven neuron. Since the effectiveness of a multipike burst is slightly less than the summed probability of effectiveness of the individual component spikes taken in isolation, one might question whether bursts actually provide any real advantage in the propagation of information. The key issue is timing. When looking at neural coding from the perspective of the target neuron (Bialek et al. 1991), bursts can be viewed as “packets” of information that are instantaneously more meaningful than isolated spikes. Our results show that bursts are about twice as effective as single spikes. Longer bursts, although fewer in number, can support event-based transfer ratios approaching 0.5. With this mechanism, a single neuron can markedly amplify the probability of its immediate influence on postsynaptic cells. This is done in a deterministic fashion that is not equivalent to summation of the probability of propagation of individual spikes over some arbitrary time interval because the message is specific not general. One consequence is an effective enhancement of orientation tuning. Stimulation with optimal orientations yields a higher proportion of spikes contained in bursts, as well as longer bursts (even at low firing rates) (Debusk et al. 1997), compared with stimulation at nonoptimal orientations. At optimal orientations, intercellular linkage thus is amplified, leading to an increase of orientation selectivity (Cattaneo et al. 1982) across a wide range of contrasts. Bursts similarly have been found to sharpen the tuning of neurons in auditory cortex (Eggermont and Smith 1996).

We have found that there are at least three factors governing the effectiveness of presynaptic spikes in eliciting a postsynaptic spike. The first two are firing rate and burst length. Although related, these two are not wholly dependent. Decreasing burst length at a given firing rate, which occurs with stimuli of nonoptimal orientations, will decrease the effectiveness. Similarly, decreasing the firing rate with the burst length remaining constant will decrease the effectiveness. This is presumably due to reduced spatial summation as a consequence of decreased activity contributed from other cells. However, even when burst length and firing rate are accounted for, there is yet another stimulus-dependent influence on effectiveness, which we attribute to suppression mediated by network interactions.

Bursting is likely an intrinsic property of the neuron membrane and is mediated via entry of Ca$^{2+}$ ions (Pumain et al. 1983; Schwartzkroin and Wyler 1980). It can be modulated by the surrounding circuitry, which synchronizes, suppresses, or possibly terminates the bursts (Bush and Sejnowski 1996; Gray and McCormick 1996). We believe that bursting reflects a natural tendency of cortical cells that is necessary to overcome the high threshold of the postsynaptic cortical cell (Creutzfeldt and Ito 1968). The transfer of information then is tempered by a network “moderator” that
is engaged actively in determining the relevance of this information. \(\gamma\)-Aminobutyric acid-A (GABA\(_a\))-mediated inhibition is a possible basis for this mechanism because it has been shown to shorten bursts (Debusk et al. 1997; Dykes et al. 1984) and is likely to reduce firing of other cells that contribute to spatial summation. It is thus difficult to disengage network interactions from bursting.

One can postulate that these bursts form the basis of a combinatorial space-time representation (Wehr and Laurent 1996) of information in cell assemblies (Braitenberg 1978; Gerstein et al. 1989; Hebb 1949; Palm 1982). An important function of the network would be to synchronize these bursts. Synchronization would allow the next cortical stage to bind together cortical neurons that are temporally dependent. One consequence of functional linkage is a support for learning by grouping neurons that usually become active together to cause a postsynaptic neuron to fire (Hebb 1949). MacLeod and Laurent (1996) have shown that fast GABA-mediated inhibition underlies neuronal synchronization in the olfactory system. Kim et al. (1995) also have shown that it is possible to phase lag and lead EPSPs in the dendritic branches by inhibitory control. It is therefore reasonable to expect that the network can adjust and synchronize bursts. The effects of such synchronized bursting would be profound. Braitenberg (1978) proposes the existence of an “amplification” with respect to the number of active synapses needed to raise a neuron above its threshold. He notes that an epileptic focus on one side of the brain may excite the contralateral cortex producing epileptic activity. Because the callosal fibers are only a few percent of the total cortico-cortical fibers, he concludes that synchronous activation of only a few percent of the synapses is sufficient for exceeding threshold. Thus with synchronous bursts, not only does temporal summation occur, but spatial summation as well, a symbiotic interaction reinforcing the activity of the target neuron.

Recently, there has been interest in the irregularity of spike timing (Softky and Koch 1993), which has heightened interest in the issues of neural coding (Shadlen and Newsome 1994). One of the reasons proposed for this irregularity is the balance between excitation and inhibition in order for the neuron to avoid saturation and yet be near the firing threshold (Bell et al. 1995). Shadlen and Newsome (1994) raise the question of whether neurons should be viewed as coincidence detectors or integrate-and-fire devices and that bursts of two to five spikes, having intraburst firing rates as high as 800 spikes/s, and having burst repetition at intervals of 15–50 ms. The interspike interval (ISI) histograms of these cells are similar to those described here as well as in Debusk et al. (1997) and in Mandl (1993). Gray and McCormick (1996) have identified the chattering or bursting cells as layer 2/3 pyramidal neurons. These neurons have axon collaterals which project into layers 2/3 and 5, and the axon itself projects into white matter and to higher cortical areas. Because layer 2/3 pyramidal cells are the major output cells of the striate cortex, bursting might play a significant role in conveying information to, and perhaps synchronizing, the higher cortical centers.

Decoding of bursts is biologically plausible

Any mechanism for information encoding and decoding in spike trains must be consistent with the duration required for viewing the coincidence at the burst level, the precise timing of single spikes, which in any organic environment would be difficult to attain, becomes relatively unimportant. More important is whether successive spikes fall within the time constant of the postsynaptic membrane, thus allowing temporal integration to occur. The integrate-and-fire model would be sufficient for the detection of bursts that occur synchronously and still allow for some random nature of individual spikes on the order of several milliseconds (Bialek et al. 1991).
to complete a visual task. An example of a severe constraint for temporal codes is the 100–150 ms it takes to excite inferotemporal cortex (IT) neurons, which are selective for facial features (Perrett et al. 1982). A more detailed argument is presented in Thorpe and Imbert (1989), but the main points will be given here to argue that burst coding is consistent with these constraints whereas other proposed schemes for temporal coding challenge these constraints.

The minimum number of synaptic stages that visual information must traverse for activation of IT appears to be ≈10. One synaptic stage is between the photoreceptors and bipolar cells, one between the bipolar cells and ganglion cells, one between ganglion cells and cells in the lateral geniculate nucleus (LGN), one between LGN cells and layer 4 cells in V1, one between layer 4 and layer 2/3 cells in V1, two in V2, two in V4, and one in IT. Because neurons in the temporal lobe have visual response latencies on the order of 100–150 ms and there are ≈10 synapses to negotiate, this yields a limit on average of 10–15 ms per synapse in which to process visual information. About 10 ms total would be required to account for delay through all of the 10 synapses. Given a conduction speed of intracortical axons of ~1 m/s (Bullier et al. 1988), one can estimate ≈70 ms for spike transit from retina to V1 to IT. With these adjustments, the average processing time per synapse is reduced to <10 ms. This figure is reasonably consistent even if we ignore the processing before V1. The latency to V1 is 50–70 ms (Celebrini et al. 1993), leaving 50–80 ms to go from V1 through five synaptic stages to neurons in the temporal lobe.

This model is particularly troublesome for the coding theory of Lestienne and Strehler (1988) because their proposed symbols (spike triplets) are spread in time ≈100 ms. It would take 100 ms for single recipient neurons to decode these symbols, yet this is the time actually required for visual information to go through 10 synapses. A similar problem exists for Richmond and Optican (1987) who propose methods of information transfer requiring integration over periods of ≈100 ms, although a more recent examination of their data (Heller et al. 1995) suggests that most of the information is contained within the first 75 ms. The model of Von der Malsburg and Bienenstock (1986) proposes that information is carried in the degree of correlation between neurons and that 100 ms is likewise needed for the determination of this correlation. For all of these models, if each neuron has to wait 100 or even 75 ms to determine the presence of relevant information, the time to receive and decode visual information in an area such as IT could take upwards of 1 s, which is clearly not the case. One could argue that the particular temporal structures of a given model are preserved through several stages of processing and are not decoded at every synapse, but there is thus far no clear evidence that this degree of coherence is maintained from one cell to the next.

How does burst coding fit into this time constraint? If we looked only at the average firing rate of neurons, which is typically <100 spikes/s sustained, this would yield on average <1 spike within a 10-ms window. This is hardly enough information on which to base a decision, but bursts can easily generate three to five spikes in 10 ms. Not only is this very informative in terms of an instantaneous rate code (similar to a screaming infant in a room of noisy children), this information can be decoded readily by the postsynaptic neuron using simple temporal summation as opposed to complicated and sensitive anatomic/physiological decoding mechanisms. It should be noted that the mean of the burst lengths (in time—up to 5 spikes) are on average <20 ms, which is well within the practical summation capabilities of pyramidal neurons. We would note that because we have not explored the temporal relationship between stimulus onset and burst genesis, we cannot be assured that bursting plays a role in rapid identification tasks. Moreover by no means does our argument rule out the existence of coding schemes that require longer integration times for some visual purposes. We do suggest that by their very nature such schemes are less useful for visual judgments that must be made rapidly.

We thank V. Casagrande for thoughtful comments on the manuscript.

This study was supported by National Eye Institute Grant R01EY-03771-14 and Core Grant R30EY-08126. J. F. Kabara was supported by National Institutes of Health Training Grant T32-07135.

Address for reprint requests: A. B. Bonds, Dept. of Electrical and Computer Engineering, Box 1824 Sta. B, Vanderbilt University, Nashville, TN 37235.

Received 11 August 1997; accepted in final form 28 April 1998.

REFERENCES


Celebrini, S., Thorpe, S., Trotter, Y., and Imbert, M. Dynamics of