The Δ_{AP} delay is derived from latency measurements and is therefore expected to be stimulus dependent. Response latency varies with stimulus parameters in the retina (Kuffler, 1953; Levick, 1973), in the visual areas studied here in monkey (Gawne et al., 1996; Lisberger and Movshon, 1999; Raiguel et al., 1999), and in human visual cortex (Parker and Salzen, 1977; Jones and Keck, 1978). In a separate study, we used slow movement for DS cells and high spatial frequencies for LGN cells and observed that Δ_{AP} may increase by $\geq$10 msec (our unpublished observations). Here, we have used optimal spatial frequencies, high temporal frequencies, and fast motion, which shorten neuronal integration time (Shapley and Victor, 1978; Sestokas and Lehmkuhle, 1986; Reid et al., 1992; Lisberger and Movshon, 1999), to allow comparison of the lower bounds for Δ_{AP} across areas. A full account of the variation of Δ_{AP} across multiple stimulus dimensions and multiple visual areas will be presented elsewhere.

The offset latencies that we found suggest that information from the photoreceptors can reach LGN within 15–20 msec and V1 and MT within 20–25 msec. We believe that the rapid, dynamic nature of our stimuli and the measurement of offset rather than onset latency was responsible for revealing these short latencies. Response decreases have been reported to have exceptionally short latencies in visual cortex (Bartlett and Doty, 1974, their Fig. 3).

Spontaneous rate

We found an inverse correlation between Δ_{AP} and spontaneous rate both across and within areas. For example, V1 simple cells had lower spontaneous rates and larger Δ_{AP} compared with LGN cells. Within the LGN and MT, cells with lower rates had higher Δ_{AP} values on average. Evidence that such a relationship could depend on resting potential comes from adaptation studies in area 17. Adaptation decreases spontaneous rate (Vautin and Berkley, 1977), increases onset latency (Saul, 1995), and hyperpolarizes cells (Carandini and Ferster, 1997; Sanchez-Vives et al., 2000). Further evidence comes from Azouz and Gray (1999), who reported a weak anticorrelation between latency to first evoked spike and the membrane potential (V_m) before stimulus onset for single trial data. If part of the variation in Δ_{AP} across cells reflects differences in V_m established before the stimulus begins, Δ_{AP} might be a useful extracellular indicator of changes in neuronal state, e.g., hyperpolarization or synaptic gain, caused by adaptation or other contextual manipulations. Δ_{AP} and onset latency are not equivalent in practice because, for example, stimulus contrast affects onset and offset latency, whereas adaptation affects only onset latency (Saul, 1995).

Several studies have shown that neurons with stronger inhibition or lower spontaneous rates have more specific trigger features, i.e., are more narrowly tuned (Pettigrew et al., 1968; Creutzfeldt and Sakmann, 1969; Snodderly and Gur, 1995; Carandini and Ferster, 2000). Similar observations were reported for the auditory nerve: cells with high intensity thresholds have less spontaneous activity (Kiang et al., 1965, 1976). We tested for a relationship between orientation tuning bandwidth and Δ_{AP} and found weak correlations for V1 simple cells ($r = -0.46$; $p = 0.02$; $n = 27$) and MT cells ($r = -0.32$; $p = 0.05$; $n = 34$) but not for complex DS cells ($r = -0.17$; $p = 0.33$; $n = 34$).

A versus N stimuli

Do antipreferred stimuli delay response onset? Lisberger and Movshon (1999) showed that brief pulses of antipreferred motion delayed responses in MT. Data from Celebrini et al. (1993, their Figs. 8 and 9) showed the same effect for flashed orthogonal gratings in awake macaque V1. We found that, when tested together in ternary sequences, A caused longer delays than N in LGN, V1, and MT and that firing rate for most cells was lower for A than for N just before the transition to P. There were exceptions, however, in which A produced a higher rate and a longer delay than N. In contrast, onset latency was never longer for NP than for AP transitions. Changes in timing and firing rate were only weakly correlated in our comparison of A and N stimuli across cells, and V1 simple cells showed no significant difference in firing rate but displayed significant timing differences. Therefore, differential effects on onset timing may provide useful information that is not available from firing rate for the quantitative evaluation and ranking of visual stimuli. To detect differences among candidate antipreferred and null stimuli, it might be critical to interleave them in rapid succession as we have done, because the visual system may adapt and conceal such differences if stimuli are displayed for a long time or are shown separately in a binary sequence that includes a potent preferred stimulus.

The origin of Δ_{AP}

We found similarities and significant differences in Δ_{AP} across cell classes and will consider below how they might arise. Several factors probably contribute to Δ_{AP}. First, spiking neurons share properties with integrate-and-fire devices and, as evident from intracellular current injection, their rise times to threshold can range from <1 msec to many tens of milliseconds. Cessation of spiking, however, is nearly immediate when the input is removed. If the Output in Figure 5*B* were interpreted as V_m, it would imply that Δ_{AP} was entirely neuronal integration time (the time from depolarization to spike, see Nowak and Bullier, 1997). As mentioned above, we have intentionally chosen stimulus parameters to minimize integration time. Second, because onset latency depended on the stimulus before the transition to preferred, perhaps inhibition driven by A delays the rise to threshold. Third, if signals are directly relayed by feedforward excitatory connections, Δ_{AP} should accumulate downstream. Thus, part of Δ_{AP} may be inherited from excitatory inputs.

Δ_{AP} in the LGN

For 30 msec antipreferred pulses, the average Δ_{AP} for m-cells driven via their center was significantly smaller than that for any other class of cells that we studied. m-Cells might have faster integration times than p-cells (5 msec vs ~10 msec) because of increased spatial convergence. Alternatively, achromatic stimuli may be suboptimal for p-cells, thereby increasing integration time. Both explanations are consistent with the lack of difference in Δ_{AP} when m- and p-cells were driven via their surround, under the assumption that their surround mechanisms are more similar than their center mechanisms (Lennie et al., 1991; Lennie, 2000). Slower retinal axon conduction speed for p-cells (Mitzdorf and Singer, 1979) should not increase Δ_{AP} because it would delay both onset and offset equally.

If LGN principle cells relay retinal spikes with near 1:1 transmission, as observed for some LGN X cells in cat (Cleland et al., 1971; Coenen and Vendrik, 1972; Mastronarde 1987b), then Δ_{AP} is simply inherited from the retina because retinogeniculate transmission delay is negligible (Wang et al., 1985; Mastronarde, 1987b). However, transmission ratios are often substantially <100%, and cat studies indicate that facilitation of consecutive EPSPs occurs (Singer et al., 1970; Mastronarde, 1987b; Usrey et al., 1998) possibly through simple summation (McIlwain and