Creutzfeldt, 1967). If the first retinal spike resulting from an AP transition failed but the subsequent ones were transmitted, or if the first spike was delayed (Kaplan et al., 1993), the LGN relay would increase Δ_{AP}. A transient hyperpolarization occurs at light-off in ON LGN cells and light-on in OFF cells (McIlwain and Creutzfeldt, 1967; Coenen and Vendrik, 1972), and this might contribute to the failure of the first spike, and might account for the longer delay for shorter antipreferred epochs that we have observed.

Overall, we speculate that part of Δ_{AP} in the LGN is inherited from the retina, and the rest is caused by failure of the first EPSP to reach threshold because of a transient, fast-rising hyperpolarization (Coenen and Vendrik, 1972). Such a hyperpolarization could explain the observed increase in latency for shorter antipreferred pulses (schematized in Fig. 9*C*). There is evidence for inhibition in the geniculate (Singer and Creutzfeldt, 1970; Coenen and Vendrik, 1972; Sherman and Koch, 1986; Mastronarde 1987b), but significant opponent inhibition is absent because ON and OFF pathways remain separate through the geniculate (Casagrande and Norton, 1991; Schiller, 1992). This may account for Δ_{AP} being much smaller in LGN than in V1 simple cells. Also, we predict that retinal ganglion cells, having higher spontaneous and driven firing rates than LGN cells, should have smaller Δ_{AP} than LGN cells, in keeping with the observed anticorrelation between firing rate and Δ_{AP}.

Δ_{AP} in V1 simple cells

Δ_{AP} was significantly larger for simple cells than for other cell types. The mean value, 22 msec, was 12 msec longer than that for p-cells responding to similar stimuli. Thus, $<50\%$ of Δ_{AP} in most simple cells could be inherited from the LGN. Spontaneous rate is low in simple cells relative to retinal and geniculate cells, but it is also low in complex DS cells, which had Δ_{AP} values more similar to p-cells. Data from the cat indicates that the integration time for simple cells is $\leq$10 msec, on average. Volgushev et al. (1995) estimated the time from EPSP arrival to first spike in area 17 for flashed optimal bar stimuli to be 7.6 msec. Data of Hirsch et al. (1998) using less optimized stimuli also depict a fast rise in V_m before the first spike. This is supported by estimates of neuronal integration time from spike-triggered averages of V_m *in vivo* (Azouz and Gray, 1999) and *in vitro* (Nowak et al., 1997). Carandini et al. (1996) estimated integration time to range from 14 msec for sinusoidal current injection to 3 msec for broadband current injection. Furthermore, spike precision in striate and extrastriate areas is consistent with integration times of only a few milliseconds (Maunsell and Gibson, 1992; Bair and Koch, 1996; Marvšálek et al., 1997). It is therefore not possible to account for the large Δ_{AP} values of many simple cells on the basis of a combination of the Δ_{AP} from the geniculate and the neuronal integration time of the recorded cell.

Intracellular studies of cat simple cells have shown that hyperpolarization follows the removal of an excitatory stimulus (Creutzfeldt and Ito, 1968) and that this is likely to result from inhibition (Ferster, 1988; Borg-Graham et al., 1998; Hirsch et al., 1998; Ferster and Miller, 2000). When GABA-mediated inhibition on simple cells was blocked, strong transient responses appeared at the offset of the preferred stimulus (Sillito, 1975; Eysel et al., 1998), suggesting that potent inhibition normally occurs at this time. We therefore suspect that inhibition associated with the antipreferred stimulus delays the onset of the rise to threshold in simple cells. If so, there may be at least two mechanisms by which the inhibition arises to account for the variation of time dependence of the onset latency. An inhibition that adapts could account for cells that show increased delay for shorter A epochs, whereas an inhibition that integrates slowly could account for the increased delay for longer A epochs.

As mentioned above, it is possible that part of Δ_{AP} is inherited from the LGN, but a push–pull arrangement of inputs could prevent this. For instance, when a spot of light turns off and then on in an ON region of a V1 simple RF, the simple cell waits 10 msec longer for signals from an ON p-cell than it does for the loss of signal from an OFF p-cell. Thus, the first signal to the simple cell could be the removal of inhibition that could initiate the integration to threshold without delay. For this to be practical, inhibitory neurons would have to be fast, which they are (Agmon and Connors, 1992; Swadlow, 1995; Tamás et al., 1997; Porter et al., 2001), and must integrate inputs from many cells, which appears likely (Freund et al., 1985; Swadlow, 1995), so that real OFF signals are not confounded with long interspike intervals (Levick, 1973). Thus, one advantage to receiving inhibitory and excitatory inputs to the same receptive field location might be to cancel the Δ_{AP} timing asymmetry, which for suboptimal stimuli may be substantially longer than the 5–10 msec reported here.

Δ_{AP} in DS cells

The distributions of Δ_{AP} for complex DS cells and MT cells were similar (means were 10 and 11 msec, respectively) and resembled those for the LGN more than those for simple cells. If Δ_{AP} resulted purely from integration to threshold in the recorded cell, and if V1 cells drove MT cells in a direct manner (Movshon and Newsome, 1996), Δ_{AP} would be larger in MT than in V1 and response onset would occur later in MT. Neither of these conditions held for our data: MT average onset and offset were ~1–2 msec longer than V1 (Table 1). One possibility is that Δ_{AP} in MT is inherited completely from its V1 inputs. Rapid transmission time from V1 to MT (Movshon and Newsome, 1996) could explain why we observed no significant latency difference, and spatial convergence could account for an integration time in MT that was closer to one msec than ten. The large axon diameter from V1 to MT (Rockland, 1995) certainly suggests that rapid transmission is critical in this pathway. Alternatively, a push–pull circuit could account for the absence of increase in Δ_{AP} from V1 to MT, allowing cells in both areas to have similar neuronal integration times.

For DS cells, we observed an interesting trend in onset latency as a function of antipreferred duration. As A duration decreased, a reversal in latency occurred: responses after 10 msec of A came sooner than those after 40 msec of A (Fig. 10*C*). Perhaps a transient suppression is activated (as depicted in Fig. 9*C*) for longer A pulses, but the 10 msec pulse is simply not long enough to activate the suppressive mechanism (i.e., implying that the *thinnest line* in Fig. 9*C* would not make its downward deflection). If this trend is a signature of the interaction of opponent directional signals, it is surprising that it is similar in V1 and MT in light of evidence that opponency is stronger in MT than in V1 (Qian and Andersen, 1994; Heeger et al., 1999). We do not yet know the mechanisms behind this trend, or the trends for LGN and V1, but believe that studying them may reveal important differences in circuits and intrinsic neuronal properties across visual areas.

Response offset: the first sign of change

Response latency is usually calculated for increases in firing rate (Raiguel et al., 1989; Maunsell and Gibson, 1992; Nowak et al.,