Timing in alert macaque LGN

P and M cells
Marrocco (1976), Dreher et al. (1976), Sherman et al. (1976), Schiller and Malpeli (1978), Kremers et al. (1997), DeValois et al. (2000), Reid and Shapley (2002)
Sustained and transient M

Kaplan and Shapley (1982), Blakemore and Vital-Durand (1986), Spear et al. (1994), Levitt et al. (2001), Xu et al. (2001)

Sustained P
Timing in magnocellular and parvocellular LGN

- Recorded in awake fixating monkey

- Trials had 5 s durations, with compensation for eye position

- Measured timing in several ways, with single spots modulated as square or sine waves, and with various types of noise stimuli

- Used chronic implants, enabling laminar localization

- Applied chromatic modulation to identify cone input types
Importance of LGN timing for V1
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- Comedy
Implant

designed by Max Snodderly and Elsie Wong

Reitboeck (platinum-tungsten) electrodes in polyimide sleeves that slip over guide tubes

33-gauge guide tubes extend 15 mm below cortical surface
Delineating layers from many penetrations
4-part flashing spot stimulus

Percent area = mean/max = 0.48

Half-rise = 46 ms

Half-fall = 34 ms
Population results from flashing spot stimulus
Sinusoidally modulated spot
Population results from sinusoidally modulated spots
Lagged P cell

Latencies: (ON)
Half-rise = 78ms
Half-fall = 62ms
Percent Area = 0.74
Summary and implications

• Parvocellular LGN appears to have relatively uniform timing, almost always sustained. Magnocellular LGN, however, has diverse timing, including not only transient responses, but often sustained responses. Lagged cells exist in both groups. Latencies vary little across the population.

• Why do so many labs report large differences in timing? Electrode sampling bias may be the main factor. Large cells seem to be more transient. Magnocellular LGN contains a soma size distribution that largely overlaps that of parvocellular LGN (Montero and Zempel, 1986; Liu and Wong-Riley, 1990; Ahmad and Spear, 1993; Weber et al., 2000), but most electrodes probably miss the many small cells in the magno layers.

• The magnocellular pathway is often mentioned as the locus of the neural deficit in specific learning impairment (e.g., Stein and Talcott, 1999). This is based partly on arguments about timing. More likely, a subdivision of the magno system is key, and the bulk of the magno pathway may remain intact in these diseases.