saccade amplitudes was 1.10 (Figure 7d). These differences were small but statistically reliable ($p = 0.01$). Although primarily hypometria was seen using 300Hz stimulation, hypermetria was also evident across the sample. Thus we conclude that qualitatively similar results are obtained with frequencies ranging from 75, 125 and 300Hz (cf., Figures 5c,d and 7a,b). In light of the suggestion that low frequency stimulation for DBS often makes symptoms worse (Benabid 2003; Moro et al. 2002), it will be interesting to compare even lower frequencies of stimulation such as 20Hz in the future.

Saccade Latency. The influence of SNr stimulation on saccade latency is shown in Figure 8. Electrical stimulation of the SNr influenced the latency of both memory-guided and visually-guided saccades. The same effects on saccade latency were obtained for all three stimulation parameters although the effects of 75 and 125Hz were less profound (Figure 8a-d, 300Hz; e-h 75 and 125Hz). Furthermore, the stimulation influenced saccade latency bilaterally. For memory-guided saccades, latency was reduced on stimulated trials compared to no stimulation trials (Figure 8a,e). By ~200ms, saccade latencies were increased for contralaterally directed memory-guided saccades (Figure 8a,e). A similar trend was evident for ipsilateral saccades (Figure 8b,f). Ipsilateral saccades had a reduced latency ~<200ms and a prolonged saccade latency beyond 200ms. We confirmed this observation statistically using the Kruskal Wallis ANOVA and determined that saccade latency was significantly different in stimulation trials compared to controls for contralateral ($p<0.001$) and ipsilateral ($p<0.02$) memory-