NO plays important roles in signal transduction and immune defense. A crucial element in understanding the molecular mechanism of these physiological functions of NO is structural characterization of Fe(II)NO heme complexes, because the known physiological receptor for NO is the heme protein guanylate cyclase; also, T-state Fe(II)NO hemoglobin has been proposed to be the physiological carrier of NO. Because Fe(II)NO heme complexes are paramagnetic (S=1/2), they have been studied extensively by EPR techniques. The EPR spectra of Fe(II)NO heme complexes contain two species, a more rhombic species I and a more axial species II. Model compounds studies have shown that increased solvent polarity and hydrogen bonding to the bound NO increase the ratio of species I to species II. Thus it was surprising that the EPR of the H64L mutant of Fe(II)NO myoglobin (Mb) exhibits a higher species I to species II ratio, because His64 has been shown to be a hydrogen bond donor to the bound NO in Fe(II)NO Mb, and replacing His 64 with a Leu, that is, eliminating the hydrogen bond as well as decreasing polarity of the protein environment of the bound NO, should decrease, rather than increase, species I to species II ratio in the EPR spectrum. Because species I is a more rhombic EPR signal, it can represent a Fe(II)NO heme complex with a smaller (more bent) Fe-NO bond angle. The increased ratio of species I to species II in the EPR of Fe(II)NO Mb(H64L) may suggest that the presence of the bulky Leu residue exerts a large steric effect on the bound NO, resulting in a smaller Fe-NO bond angle in the mutant. To test this hypothesis, EXAFS of Fe(II)NO Mb and Mb(H64L) has been carried out. Small differences in amplitude and frequency are observed for the two proteins. \textit{Ab initio} FEFF calculations and multiple scattering EXAFS analyses are in progress in order to compare the Fe-NO bond angles in Mb and its H64L mutant.