Children infected with HIV, in contrast of infected adults, often have higher virus loads and progress to AIDS faster. It is widely accepted that destruction of CD4+ T cells is the primary cause of immunodeficiency in HIV-1 infected adults and children. Macrophages, important cell components of the innate immune system and link between innate and adaptive immunity, are also important targets of HIV/SIV infection. Recently, we have reported that a massive turnover of peripheral monocytes associated with death of tissue macrophages correlated with SIV infection and AIDS in adult macaques. More importantly, the onset of high monocyte turnover was demonstrated to be a better predictive value of AIDS progression than level of immune activation, viral load or CD4 count (Hasegawa et al. Blood 2009). In this study, we thought to determine if high destruction of tissue macrophages demonstrated by high monocyte turnover may also explain why pediatric AIDS progress more rapidly compared to HIV infected adults. We first compared the monosyte turnover among groups of SIV-infected newborn macaques and control uninfected newborn macaques. Interestingly, the base line monocyte turnover from control newborn macaques was much higher compared to adult normal macaques. This high monocyte turnover of the control uninfected newborn macaques was comparable to the SIV-infected group of adult macaques previously described. In contrast to adult SIV-infected macaques, a very homogeneous high monocyte turnover was observed in all SIV-infected newborn macaques soon following infection equivalent to the turnover rate observed in the adult SIV-infected macaques in the terminal stages of AIDS. This data strongly suggest that homologous massive destruction of tissue macrophages soon after SIV infection may be involved in the mechanism of rapid AIDS progression in SIV-infected newborn macaques.