

## **The Incidence and Prevalance of Elevated Blood Pressures in HIV Infected Persons on Antiretroviral Therapy**

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**Background:** Highly active antiretroviral therapy (HAART) has prolonged the lives of persons with HIV infection. The use of antiretroviral agents has been associated with the development of hypertension (HTN). It is not clear whether elevations in blood pressure are induced by specific antiretroviral medications, associated with immune reconstitution or unrelated to HIV specific factors. No prospective study of blood pressure collection in persons with HIV has been published. **Hypothesis:** Potent antiretroviral therapy containing a protease inhibitor results in a significant increase in blood pressure within the first four to six weeks of initiating treatment. **Methods:** We performed a prospective study of blood pressure measurement. Subjects receiving no HIV therapy (controls) were compared to those changing antiretroviral therapy or beginning new therapy. Subjects were enrolled from the Infectious Diseases Center at the University of Cincinnati. Demographic information included age, race, height, weight, use of alcohol and drugs, current and past medications, family history, and hip and waist circumference measurements. A standardized blood pressure reading was taken twice in each arm at baseline. Participants returned four to six weeks later for their final readings. **Results:** To date, number of participants enrolled=35. Follow up visits completed with 22. There is median age of 36 years (range=26-64 years). There are 7 (20%) African-Americans, 28 (80%) whites, 27 (77%) men, and 8 (23%) women enrolled. Twenty-two (63%) subjects reported smoking cigarettes. Twenty-two (63%) reported a first degree relative with HTN. There was a baseline median CD4 lymphocyte count = 391 (range, 0-1354) cells/cu mm, baseline median HIV RNA level = 31, 352 copies/mL (range, 88- >750,000), and 14 (40%) reported a history of prior AIDS-defining OI. BP measurements recorded in mmHg. Controls (n=11) had baseline medians of SBP=113, DBP=76, and MAP=90. At follow up, SBP=117, DBP=77, and MAP=91. Patients enrolled in the PI arm showed baseline medians of SBP=114, DBP=81, and MAP=93. Follow up SBP=111, DBP=71, and MAP=83. Patients using no protease inhibitor (NPI) showed median baseline SBP=116, DBP=76, and MAP=87. Follow up SBP=114, DBP=73, and MAP=85. **Conclusions:** Preliminary analysis shows no statistically significant change in MAP after 4 weeks of treatment with either PI based therapy or Non-PI based therapy. Patients will be enrolled until a final number of 15 treatment-naïve subjects, 20 PI treated subjects, and 20 non-PI treated subjects is reached.