W. Collins presented a review of the use of South American monkeys in the testing of antimalarial vaccines. Different Aotus and Saimiri monkeys have been shown to be susceptible to infection with the human malarial Plasmodium falciparum, P. vivax, and P. malariae. Antisporozoite vaccine trials are being conducted in Bolivian Saimiri; Aotus lemurinus griseimembra from Colombia and A. vociferans from Peru are candidates for P. falciparum sporozoite vaccine trials. Blood-stage vaccines are being tested in A. nancymai from Peru, A. lemurinus griseimembra from Colombia, A. lemurinus lemurinus from Panama and Saimiri sp. from different geographic origins. Studies with these monkeys allow for the testing of immunogenicity and efficacy using different antigens and adjuvants to provide data not obtainable from other animal and human studies.

J. Gysin reported on the protective immunity against the asexual forms of P. falciparum in Saimiri monkeys. Passive transfer of intact IgG anti-P. falciparum antibodies isolated from protected monkeys protected naive non-immune monkeys against P. falciparum infection. Antibodies are species specific but not strain restricted and opsonize infected erythrocytes via the FcRIII receptors expressed on circulating monocytes. Non-protective anti-P. falciparum antibodies are not opsonizing and compete with the protective opsonizing antibody at the target level. Protection of the host depends not only on recognizing antigens exposed on the surface of infected erythrocytes but also on the balance between the two antibody populations. Oposnizing antibodies are capable of conferring protection to P. falciparum in non-immune and splenectomized Saimiri monkeys.

B. Enders described the protection of Aotus monkeys immunized with recombinant single and combined antigens of P. falciparum. Aotus monkeys were immunized with E. coli derived fused proteins coding for partial sequence of the merozoite surface antigen (MSA-1), serum-stretch protein (SERP), and the histidine ala-nine rich protein II (HRPII), as well as a group of recombinant antigens obtained by antisera raised against a protective 41 kD protein band. Intact monkeys were immunized three times and then splenectomized prior to challenge. HRPII, a combination of three different fusion proteins of the 41 kD group and a mixture of two sequences of SERP with AL(OH)3, adjuvant conferred significant protection against challenge with P. falciparum. Two hybrid proteins expressed in E. coli administered in a new oil-based adjuvant protected monkeys from severe experimental P. falciparum infection.

S. Herrera assessed different P. falciparum vaccine candidates in Aotus monkeys in Colombia. Monkeys were immunized with the recombinant protein 190L, a fragment of the MSA-1 antigen, and a construct containing 190L and the T helper epitope CSTD, of the CSP. Two out of five of the 190L group spontaneously cured the parasitemia and three out of four immunized with the construct were protected. Monkeys were immunized with the synthetic protein SPF (66)30 containing fragments of different blood stages and the NANP peptide from the P. falciparum CS protein. Monkeys received six immunizations with the peptide in Freund's adjuvant or alum. This protein failed to significantly protected the animals.

L. Monjour reported on the humoral and cellular responses to six different antigenic fractions prepared from the blood stages of P. falciparum. Homogenized parasitized erythrocytes were electrophoresed on SDS-polyacrylamide gells. Antigens were prepared by electrophoresis of six strips by referring to molecular markers run is parallel. Mice, rabbits
and *Saimiri* monkeys were immunized three times at four week intervals using Freund's complete and incomplete adjuvants. All fractions led to the formation of antibodies reactive with the asexual blood stages or the surface membrane of parasitized erythrocytes as detected by IFA. As measured by Western blot analysis, each of the six fractions recognized only one or two major proteins. Maximal inhibition of parasite growth in *in vitro* cultures was obtained with antiserum induced by the 94 to 67 kDa band.

M. Aikawa presented *P. coatneyi*-infected rhesus monkeys as a model for human cerebral malaria. Studies were made of the brains of rhesus monkeys infected with *P. coatneyi*. Sequestration and cytoadherence of knobs of parasitized erythrocytes (PRCB) was demonstrated in the cerebral microvessels of these monkeys. Cerebral microvessels with sequestered PRCB were shown by immunohistochemistry to possess CD36, thrombospondin and intracellular adhesion protein-1. These proteins were not evident in the cerebral microvessels of uninfected control monkeys.

R. Weller reported on the detection of impaired renal function in 16 *Aotus nancymai* monkeys 25 and 37 months following infection with *P. falciparum*. Impaired renal function was suggested by significant decreases in endogenous creatinine clearance, creatinin excretion, and urine volume, and increases in blood urea nitrogen, urine protein, and fractional excretion of phosphorus, potassium, and glucose. Serum concentrations of calcium and glucose were also significantly decreased. The results suggest a subclinical pathologic process, characterized by chronic progression, persisted in the kidneys of these monkeys following resolution of their parasitemias.