Study of the pathology of malaria has contributed to understanding of its pathogenesis. I will summarize my work of 30 years in the fields of malaria pathology as follows. The main pathologic changes appear to be caused by the response of the reticuloendothelial system to the parasita, by the destruction of erythrocytes which together with their defective production causes anemia and by the obstruction of capillary lumens in the brain and other organs by parasitized erythrocytes (PRBC). Most pathologic studies of malaria are of \textit{P. falciparum} which causes severe malaria. Cerebral malaria is recognized by the blockage of the cerebral capillaries by PRBC and associated pathologic lesions. Based on these findings, the development of malaria vaccines against cerebral malaria is now in progress. Splenomegaly and hepatomegaly are common findings among malaria patients. Renal disease also occurs in malaria. In falciparum malaria, mesangiopathic glomerulonephritis causes abnormal urinary sediment including proteinuria and cylindruria. In contrast, with quartan malaria membrano-proliferative glomerulonephritis leads to a nephrotic syndrome. In pregnancy, falciparum malaria severely affects the placenta, especially in primiparous women, often resulting in abortion. Microscopic fields show intervillous spaces with many PRBC and macrophages. Recently adhesion molecules such as hyaluronic acid and CSA have been identified. These molecules assist PRBC to cytoadhere to trophoblasts and PRBC sequestration results.

Despite the fact that both the anatomic and microscopic alterations associated with malaria are well recognized, the pathophysiologic changes in malaria infection are not well characterized. A better understanding of the mechanisms of malaria infection may contribute to more efficacious treatment with pharmacological, vaccines and molecular approaches.