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Discussion

Dr. Basil A. Pruitt, Jr. (San Antonio): We at the Army Burn Unit have been interested in these same changes, because sepsis continues to be the most common cause of death in our burn patients. We have followed the changes in circulating granulocytes in a laboratory rat burn model, both infected and uninfected; and following early demargination in the uninfected burn, total circulating granulocytes return to approximately normal levels. However, with the infected model there is a persistent granulocytic defect.

If one further looks, as shown on the following slide (slide), at both the dividing and the nondividing compartments, one can see that in the uninfected animal the subsequent restitution of granulocytes is due to a marked increase in the dividing compartment, whereas in the infected animals there is a persistent decrease in both the dividing and the nondividing compartments.

I ask Dr. Alexander: Can you separate out a specific granulocytic defect from the effect of a progressively older average aged leukocyte?

President Moore: I would like to ask Dr. Alexander a question. Has he examined the effect of antibiotics themselves on this particular leukocytic activity?

Dr. J. Wesley Alexander (Closing): To answer Dr. Pruitt's question first, we have not been able to relate the apparent age of neutrophils to their function, although we do not feel that we have been able to study this with sufficient clarity to really define whether or not abnormalities may be associated with immature granulocytes.

There have been some studies reported in the past in patients with myelocytic leukemias in which there is evidence that stages before the metamyelocyte are inefficient in bacterial killing, but once the stab form is reached, they can kill efficiently.

In our studies on either burn patients or controls we have not been able to establish a relationship between segmented and nonsegmented ratios and their efficiency of bacterial killing.

To Dr. Moore's question about the effect of antibiotics on this important mechanism, we have done several studies in experimental animals which would indicate that antibiotics, in themselves, do not cause an inefficiency of killing by the neutrophils. However, they do cause many other problems, and we feel that the use of prophylactic antibiotics in patients who are susceptible to opportunistic infection should be rigidly avoided.