thing occurring in the lung? Animals made tolerant to endotoxin often demonstrate increased numbers of various cell types. Would this partly explain your results?

Dr. Rylander: That’s probably what we see: the exposure levels in workers are above those required to cause mill fever. A similar thing happens to sewage workers.

Dr. Kreutzer: Have you used a variety of endotoxin preparations? Might there be chemotactic factors in your preparation?

Dr. Rylander: We have not done this.

Dr. Ward: Your lavage neutrophils seem very low in number. What’s the reproducibility of your counts?

Dr. Rylander: The figures given need to be multiplied by $10^4$ to give the true value. The reproducibility is good as long as we do not work with lungs that are infected.

Question: Since housedust is made up of a large amount of cotton, do you have any feelings about endotoxin playing a role in housedust allergy?

Dr. Rylander: Yes, I do. An article has been published which suggests that housedust contains significant amounts of endotoxin.

Dr. Brooks: Since prevalence of byssinosis varies from plant to plant and also with the quality of the cotton, have you looked at the different kinds of cotton and their endotoxin content?

Dr. Rylander: Yes, and the variation in content of bacteria parallels the variation in byssinosis.

Dr. Stechaschulte: Is IgA monomeric or dimeric in serum or secretions?

Dr. Rylander: Bronchial secretions of cotton workers are 50 percent secretory of IgA. We don’t know if it is monomeric or dimeric.

Dr. Karr: I’d like to ask Dr. Rylander about his feelings regarding the possible role of complement in the response to inhaled endotoxin?

Dr. Rylander: Maybe complement is not the important factor in the production of inflammation in the airways.

Dr. Ward: I think the story is very complex and depends upon the type of endotoxin preparation.

**Enumeration of Pulmonary Immunoglobulin Secreting Cells in Human Bronchoalveolar Lavage**

E. Clinton Lawrence, M.D.; R. Michael Blaese, M.D.; R. Russell Martin, M.D.; R. Keith Wilson, M.D.; William J. Deaton, M.D.; and Paul M. Steoens, M.D., F.C.C.P.

Our previous studies in animals suggested that the lung is rich in immunoglobulin-secreting cells (IgSC). We have now enumerated pulmonary IgSC obtained by bronchoalveolar lavage, as compared to IgSC found in peripheral blood, in human volunteers.

IgSC were enumerated using a "reverse-plaque" assay which detects cells secreting IgG, IgA, IgM, or IgE by the lysis of staph protein A-coated indicator erythrocytes in the presence of developing antisera specific for each immunoglobulin (Ig) class. IgSC for all four classes of Ig were found in the lung, usually in greater numbers than found in the blood (approximately 1800 total IgSC/10^6 lymphocytes in the lung, vs approximately 550 total IgSC/10^6 lymphocytes in the blood). The pulmonary IgSC were probably lymphocytes since maneuvers to deplete alveolar macrophages or to remove cytophilic antibody had no discernible effect on the numbers of IgSC. The lung was particularly enriched for IgA secreting cells, underscoring the importance of IgA in pulmonary secretions. The findings of IgSC for IgE in five of ten normal lavage samples but not in peripheral blood may be important in defining the susceptibility of the lung to hypersensitivity reactions.

The demonstration of IgSC in normal lavage samples emphasizes the role of local lung immune responses in defense against pulmonary infections. An example of the potential clinical utility of this assay was the finding of ten times the normal numbers of IgSC for IgG and IgA in the lavage from a patient with lymphomatoid granulomatosis of the lung. It should be feasible similarly to define pulmonary immunoregulatory mechanisms in interstitial and inflammatory lung diseases, using this technique. Moreover, the enumeration of IgE secreting cells could be an important tool in the study of hypersensitivity lung diseases, such as asthma.

**DISCUSSION**

Dr. Karr: If lymphoid granulomatosis is a local immunologic process, how do you explain findings of systemic involvement?

Dr. Lawrence: Yes, it is systemic, but we have only studied peripheral blood and lung tissue.

Dr. Karr: Were study subjects atopic?

Dr. Lawrence: No history was available.

Dr. Karr: Was IgG, A, or M in the lavage fluid?

Dr. Lawrence: All three were up.

Dr. Shastry: Did you look at monoclonal (IgA)?

Dr. Lawrence: No, we haven’t done that.