Acknowledgment

The authors are grateful for the cooperation and assistance given by Dr. Frank Fales of the Biochemistry Department of Emory University School of Medicine.

References

1. Klatskin, G.: Bile Pigment Metabolism. Ann. Rev. Med., **12**:211, 1961.
2. Larson, D. L.: Relationship of Dark Urine to Septicemia. U. S. A. Surgical Res. Unit Ann. Rep., Ft. Sam Houston, Texas, 1960.
3. Liu, P. V.: Survey of Hemolysin Production Among Species of Pseudomonads. J. Bact., **74**:718, 1957.
4. Thomsen, O.: Ein vermehrungsfähiges Agens als Veränderer des isoagglutinatorischen Verhaltens der roten Blutkörperchen, eine bisher unbekannte Quelle der Fehlbestimmung. Z. Immunitätsforschung, **52**:85, 1927.

Discussion

Dr. H. Harlan Stone (closing): When I first saw this urine, I was astounded by its color. Considerable time was required to determine exactly what was producing the color change.

(Slide) This slide shows our antibody studies. As you can see, using the patient's serum obtained when the patient was first admitted, there was a zero titer to red cells which had been coated with Pseudomonas antigen. Terminally this titer was quite high. In fact it was greater than 1:2,048.

On testing the patient's red cells with Coomb's serum, there was no reaction, even terminally. Therefore, we concluded that the hemolytic anemia was not on the basis of an autoimmune process, but rather secondary to Pseudomonas hemolysins. (Slide) Verdoglobin is fluorescent in uultraviolet light, and you will note the chalky green color. (Slide) This slide is merely a summary of the differential fluoresence that one can obtain. The fluorescent pigment can be detected in the urine up to three weeks prior to the time the patient develops clinical Gram negative septicemia.