opsonic activity of neonatal sera occurred only when C5 was added; (3) 5-day-old stored, ACD bank plasma was markedly deficient in re-constituting opsonic activity of neonatal serum. Fresh (<24 hrs) ACD plasma, however, effected significant reconstitution. The deficiency of 5-day-old plasma in opsonic reconstitution of neonatal sera was *completely corrected* by the addition of C5 containing mouse serum, or by purified human C5, but *not* by C5 deficient mouse serum.

These studies demonstrate that the opsonic deficiency of neonatal serum involves a functional deficiency of C5 and that restoration of opsonic activity can be brought about by infusion of *fresh* plasma, which contains functionally active C5. A rationale is, therefore, presented for the use of fresh plasma in therapy of neonatal septicemia.

Immunological abnormalities in Job's syndrome. HENRY F. PABST, BEULAH HOLMES, PAUL G. QUIE, HENRY GEWURZ, GLENN RODEY, and ROBERT A. GOOD. *Variety Club Heart Hospital, Univ. of Minnesota, Minneapolis, Minn.*

A 9-year-old girl was investigated with Job's syndrome to establish the underlying pathogenesis. Recurrent large but "cold" staphylococcal abscesses in skin, kidney and abdomen were unaccompanied by fever, pain and signs of acute inflammation. Normal parameters of the immunological investigation were: cellular, delayed hypersensitivity type of immunity as demonstrated by: 1) skin test response to staphylococcal protein A, SKSD, Candida and challenge with 2,4-DNFB; 2) transformation of lymphocytes with phytohemagglutinin and with staphylococci; 3) skin homograft rejection in 10 days; immunoglobulin levels other than IgE; complement component levels and function; heat stable and labile opsonin activity; in vitro phagocytosis by polymorphonuclear leukocytes of several types of staphylococci including the patient's (P.S.); respiratory enzyme activity of these cells during phagocytosis; in vitro chemotaxis of her leukocytes as generated by P.S. in the Boyden chamber; endogenous pyrogen production by her granulocytes in presence of P.S. The striking abnormalities were: 1) marked scarcity of neutrophilic granulocytes in staphylococcal abscesses; 2) heavy eosinophilic infiltration of the surrounding tissue, 3) basophilic granulocyte accumulation in inflammatory cycle studies using P.S., and 4) extremely elevated serum IgE level. It is concluded that in vivo chemotaxis for neutrophilic granulocytes is defective in this syndrome.

Acrodermatitis enteropathica with dysgammaglobulinemia. O. RENNERT, R. JULIUS, M. SCHULKIND, and T. SPRINKLE. *Univ. Fla., Coll. Med., Gainesville, Fla.*

In 1942 Danbolt and Closs described the association of diarrhea, eczematoid rash and alopecia. These physical findings constitute the triad essential for the diagnosis of this autosomal recessive disease. The demonstration of the efficacy of Diodoquin therapy in this otherwise fatal disease was by Dallaha and Lorincz in 1953. Reports since then indicated partial success utilizing this agent, breast milk or a combination of the two. The major cause of death has been overwhelming infection. This sequellous has been ascribed to the marasmic status of the children. The case described is an 8-month-old boy who demonstrated the classic triad of this syndrome in association with total absence of humoral IgA and IgG, and normal levels of IgM. Autopsy revealed sparse lymphoid elements and supported the diagnosis of dysgammaglobulinemia. Arachidonic acid was detectable at extremely low levels in serum. Metabolism of tryptophan was normal. The association of acrodermatitis and dysgammaglobulinemia may represent the occurrence of two distinct disease entities or suggest a pathogenetic relationship. Our experience with this patient supports the view that halogen-substituted-8-hydroxyquinolins specifically affect the skin manifestations in this syndrome but may be ineffective against the malabsorptive component. Both these features were well controlled by therapy with Diodoquin and breast milk.

Altered reactivity to respiratory syncytial virus: Antibody response to RSV antigens and studies of the pathogenesis of atypical RSV illness in recipients of inactivated vaccine. JERRY J. ELLER, VINCENT A. FULGINITI, ARTHUR A. ROBINSON, HITOSHI NAGAHAMA, JAMES SHIRA, and DANIEL C. PLUNKET. *U.S. Army Med. Res. and Nutrition Lab., and Univ. of Colo. Med. Ctr., Denver, Colo.*

The vaccine, in contrast to natural infection, failed to produce significant serum IgA antibody. Kinetic experiments revealed no defect in the quality of IgG antibody produced by the vaccine. Arthus and precipitating antibodies were not demonstrable in post-vaccine sera. The mean percent blastoid formation and uptake of radiouridine were greater in vaccinees than in controls in vitro lymphocyte stimulation tests. A delayed dermal hypersensitivity reaction could be demonstrated in guinea pigs and African Green monkeys after multiple injections of adjuvanted RSV vaccine. Immediate wheal and flare reactions were not detected. A positive delayed skin test was still present in the monkeys 12 months after immunization. An African Green monkey previously immunized developed a temperature elevation above 106° and rales in the lungs, but no wheezing, lasting about 48 hours, beginning 9 days after intranasal challenge with live 11th MK passage RSV. The vaccine was shown to contain a disproportionate amount of soluble, CF antigens compared to surface, viral antigens. Evidence indicates that the vaccine overstimulated delayed hypersensitivity and IgE in young atopic children. Enhanced delayed hypersensitivity may account for the high fever, rash and some pulmonary infiltration, and the IgE-mediated response for the marked wheezing seen in atypical illness.

A new test of delayed hypersensitivity to measles virus *in vitro*. RICHARD J. LABOWSKIE, ROBERT EDELMAN, MARY C. BIUNDO, and JOSEPH A. BELLANTI. *Georgetown Univ. Med. Ctr., Washington, D. C.*

The purpose of these studies was to develop an in vitro method for the demonstration of delayed hypersensitivity to measles virus. Using cell lines chronically infected with measles virus, a lymphocyte-target cell interaction was studied, using morphology or $Cr^{51}$ release as an end point. Preliminary morphologic studies employing control and chronically infected HeLa cells indicated a greater cytotoxic effect with lymphocytes from children previously immunized with live measles vaccine than in unimmunized controls. All subsequent studies employed two human fibroblast lines, one infected (WI-M1) and one normal (WI-38). The WI-M1 carries measles antigen at the cell surface in steady state replication. Modifying the cytotoxic method of Holm and Perlmann, lymphocytes purified by glass bead adsorption were added to $Cr^{51}$ labelled target cells in a ratio of 100:1. The release of $Cr^{51}$ in excess of background was interpreted as a measure of lymphocytotoxic factor. The percentage release of $Cr^{51}$ from WI-M1