and WI-38 cells was determined in a group of 8 seropositive adults with no history of clinical measles. The results showed that lymphocytes from the adults with measles effected as much as a 13% increase in Cr^{51} release from WI-M1 as compared to WI-38 cells. The lymphocytes from the two individuals with negative histories showed a much diminished release. These results indicate that the measurement of lymphocytotoxic response may be a useful in vitro method for studying delayed hypersensitivity to measles virus.

Absence of serum immunoglobulin E in newborns: Heterogeneity in time of synthesis in infants. MICHAEL BAZARAL, H. ALICE ORGEL, and ROBERT N. HAMBURGER. *Univ. of Calif., San Diego Sch. of Med., Ped. Immunol. and Allergy Div., La Jolla, Calif.*

Immunoglobulin E (IgE) in serum was measured by an improved quantitative test (an assay with competitive inhibition of the binding of I^{125} Sha-IgE to bromacetylcellulose-anti-ND-IgE).

Group	Number	Median ng/ml	Mean ng/ml	Range ng/ml IgE
Postpartum mothers	35	119	205.5	19.4–810
Newborns (cord)	33	<1	2.3	<1–5.4
6 week infants	23	1.9	5.5	<1–31.6
6 month infants	17	37	88.6	4.1–458

In 31 maternal-infant pairs we confirm the absence of transplacental passage of IgE. Cord serum levels reported here are significantly lower than those from other laboratories; in our study half of the newborns had less than 1 ng/ml of serum IgE.

No synthesis of IgE was apparent in the serum of one-third (8) of the 6-week-old infants, whereas the mean of the remainder of this group was 8.3 ng/ml.

All 6-month infants had measurable IgE levels. Those 3 with clinical allergy had the highest serum IgE levels (60, 68 & 458 ng/ml).

In the adult group there is a suggestive fit to the Hardy-Weinberg distribution, consistent with simple Mendelian heredity of basal IgE levels.

A zone common to IgG globulins in the Fd region. WALTER L. HENLEY, and SIRJE OKAS (Intr. by Horace L. Hodes). *Mount Sinai Sch. of Med. of the City Univ. of N. Y., New York, N. Y.*

The carboxy terminal portion of Fd of human IgG has been shown to be antigenically similar in all IgG globulins studied. An antibody was made in rabbits against a polypeptide from a myeloma globulin comprising this zone. This antibody was shown to give a reaction of identity with the four subclasses of IgG and other IgG myeloma globulins by precipitation after immunodiffusion. At the same time an antibody made in rabbits against a polypeptide comprising most of Fc as well as commercial anti-IgG showed these IgG globulins to be similarly related. No precipitation with either antibody occurred when several pure alpha and mu chains or either the kappa or the lambda light chains were used as antigens. There was no interaction between the antibody to the Fd and the antibody to the Fc polypeptides. A region in the carboxy half of Fd of IgG antigenically related, and paralleling the constant part of the light chains has therefore been demonstrated by serologic means. This additional constant zone in IgG had been postulated by other investigators.

Secretory defense system (SDS) in health and disease. E. R. STIEHM, A. MILLER, P. M. ZELTZER, R. M. KATZ, and A. T. SAPSE. *Univ. of Calif., Los Angeles, Calif.*

The secretory defense system (SDS) provides antimicrobial activity to mucous surfaces. Tears were used as the representative secretion of the SDS because of a high protein content and ease in obtaining reproducible samples. Tears were obtained with crying or after stimulation with salt; serum was also obtained. The components of the SDS were evaluated in 25 normals, 9 newborns, and 25 patients with increased susceptibility to infection and included assay for: 1) immunoglobulins, 2) protein, 3) isoagglutinins (anti-A, anti-B), 4) lysozyme and 5) complement (C). In normal adults, mean IgG level was 17 mg%, IgA 11 mg%, IgM and C absent, total Ig 28 ± 7 mg%. Mean protein was 11.8 mg/ml, lysozyme 2.0 mg/ml, isoagglutinin titers 1:8. Tear IgG was correlated with serum IgG; tear IgA was not correlated with serum IgA. SDS abnormalities were noted in newborns, in patients with serum antibody deficiencies, and in 4 patients with increased susceptibility to infection and normal serum immunoglobulins. Newborn tears are low in protein, IgA, isoagglutinins, and lysozyme (mean .62 mg/ml) but have normal IgG. Other patients with SDS abnormalities had low total Ig in the tears (<15.0 mg/100 ml), low isoagglutinins, or normal or elevated tear IgM; lysozyme was normal. We conclude that: a) newborns have an SDS deficiency with low lysozyme, b) transport of serum IgG to tears may occur, and c) low tear immunoglobulins, absent tear isoagglutinins or the presence of tear IgM indicate SDS abnormality.

Prevention of immune complex disease (Serum Sickness) by antagonists of vasoactive amines. W. T. KNIKER, F. A. GUERRA, and S. E. M. RICHARDS. *Univ. of Texas Sch. of Med., San Antonio, Texas.*

Antagonists of vasocative amines prevent localization of circulating immune complexes in rabbits, thereby preventing acute or chronic serum sickness (S.S.) and causing amelioration of chronic complex-induced glomerulonephritis. The opportunity to apply these observations to the human has been afforded by a recent epidemic of diphtheria. We have studied 119 hospitalized individuals who each received 20–100,000 u of equine diphtheria antitoxin. Every patient was randomly selected to receive either placebo or one of two antagonists of histamine and serotonin, cyproheptadine (Periactin) or hydroxyzine HCl (Atarax). The oral medications were taken from the 4th through 16th day after antitoxin, in doses adequate to maintain continuous therapeutic blood levels of drug.

Data on 100 cases are complete. None of 15 placebo or 27 drug-treated children under age 10 had S.S. Six of 23 (26.1%) older placebo-treated patients developed S.S., lasting 2–5 days. Of the 29 Periactin and 6 Atarax patients over age 9, only 2 (5.7%) manifested definite S.S. Serum hemolytic complement activity was lowered in most of the cases with S.S., but was low in many individuals without S.S. in the second week after antitoxin. Studies of horse globulin elimination and serum immune complexes should clarify the pathogenetic mechanisms. These preliminary findings suggest that S.S. in humans can be prevented by antagonists of permeability factors required for the passive deposition of circulating immune complexes, and that this approach deserves evaluation in the management of chronic immune complex diseases.