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VENOM IMMUNOTHERAPY FOR HONEYBEE STING-SENSITIVE PERSONS. John W. Yunginger, Barry R. Paull, and Gerald J. Gleich (Spon. by G. S. Gilchrist), Depts. of Ped., Int. Med., and Immunol., Mayo Clinic and Foundation, Rochester, MN.

Preliminary studies have suggested that venom immunotherapy for Hymenoptera-sensitive persons may be superior to conventional immunotherapy with whole insect extracts. The immunological and clinical effects of honeybee venom (HBV) "rush" hyposensitization were tested in 18 patients, who received an intensive two-day course of HBV injections once monthly. Initial doses of 0.5 µg were raised as rapidly as possible to 200 µg; one to nine treatment courses were required to reach 200 µg. Anaphylactic reactions were experienced by 11/18 patients during injection therapy. Patients then returned for deliberate honeybee sting challenges. Initial sting challenges were tolerated well by 10/17 patients completing therapy; 7 other patients required epinephrine. With subsequent treatment 11/17 patients achieved complete clinical protection; partial protection was achieved by 3 others. Protection was maintained by periodic deliberate stings or by HBV injections at 2-8 week intervals. Serum IgE antibodies to HBV and to venom phospholipase-A (PLA) rose dramatically ($\bar{x}$ =350%) after therapy and remained elevated over baseline levels in 14 patients. Serum IgG antibodies to PLA rose steadily during therapy and approximated those seen in hyperimmune beekeepers in 8 patients. There was no level of IgG antibody which was protective for all patients. The results indicate that under controlled conditions HBV immunotherapy provides clinical protection in the majority of patients.

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IMMUNOCOMPETENCE AS MEASURED BY RESPONSE TO PHYTOHEMAGGLUTININ (PHA) IN T-CELL AND NON-T-CELL ACUTE LYMPHOBLASTIC LEUKEMIA (ALL). Jaime Zusman, John

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70 pts with ALL were studied with *in vitro* lymphocyte stimulation to PHA. Dose response curves (DRCs) and time response curves (TRCs) were performed and correlated with the % of lymphocytes (L), blasts (BL), T-cells (T) in peripheral blood and the clinical course (followup 8-175 weeks). The 48 pts with WBC < 20,000/mm³ (L 20%, BL < 80%, T 29±5%) had normal DRCs and TRCs. The maximal response was achieved on day 4 (78,500±5,800 cpm). All pts had Null cell leukemia; 7/48 (14%) pts have relapsed in the marrow (BM). The remaining 22 pts with WBC > 20,000/mm³ (L < 20%, BL > 80%, T 17.5±4%) all responded abnormally. 13/22 pts had a definite but decreased response on day 4 (18,846±5,400 cpm) with a 2- to 4-fold greater response on day 6 (WBC 100,000 ± 8,000, % L 9.1±3, %BL 88±8, %T 7.3±3). All pts had Null cell leukemia; 4/13 (30%) have relapsed in the BM. The remaining 9 pts were unresponsive to PHA on day 4 and failed to show a delayed peak on day 6 (224,000±10,500, % L 4.3±2, %BL 93±7, %T 39±9). 4/9 pts had T-cell leukemia; 6/9 (66%) have relapsed in the BM. Three response patterns to PHA were found in pts with ALL: (a) normal (b) delayed peak indicating lymphocyte dilution by non-PHA responsive blasts and (c) flat response indicating dilution and possible immunodeficiency. A flat DRC and TRC to PHA further characterizes pts with T-cell leukemia in addition to predicting a poorer prognosis for other high WBC pts with ALL.

INFECTIOUS DISEASE

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THE INFLUENCE OF LEUKOPENIA ON SURVIVAL IN EARLY ONSET GROUP B STREPTOCOCCAL SEPSIS. Robert D. Bacsik, Larry N. Cook, Roger J. Shott, Billy F. Andrews,

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To determine the prognostic significance of leukopenia in early onset neonatal group B streptococcal sepsis, Nursery ICU admissions were reviewed from 1969-1976. 40 cases of blood culture proven Group B streptococcal sepsis were found in infants under 5 days of age. 55% were premature infants. The majority of patients (78%) presented with respiratory distress often associated with apnea. Leukopenia (total WBC < 5000) was found in 48% of the septic infants. Of 21 patients who had lumbar punctures, 12 had Group B streptococcus isolated from the CSF as well as blood.

The overall survival of combined term and preterm infants was 70%. Term infants showed no correlation between leukopenia and survival (83% (10/12) of infants with normal TWBC survived versus 83% (5/6) for infants with TWBC < 5000). In premature infants leukopenia seemed to be associated with a decreased survival (78% (7/9) of infants with normal TWBC survived versus 46% (6/13) of infants with TWBC < 5000), however, this difference was found to be insignificant (p=0.15 by the Fisher Exact Test).

Currently more cases are being sought to determine if the trend of decreased survival in leukopenic prematures persists but, at this time, it would appear that there is no significant correlation between the level of TWBC and survival in early onset neonatal group B streptococcal sepsis.

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THE EFFECT OF 3 CORD CARE REGIMENS ON BACTERIAL COLONIZATION OF NORMAL NEONATES. Fred F. Barrett, Edward O. Mason, Darrel Fleming, Baylor College of Medicine, Dept. of Pediatrics, and C. T. Parker Laboratory, Texas Children's Hospital, Houston.

One hundred infants were randomly assigned to each of three treatment groups. All infants received a castile soap (CS) bath at 6 hrs of age followed by no further cord care in the control group and either a single application of silver Sulfadiazine (SS) or triple dye (TD) in the 2 study groups. Semiquantitative umbilical and nasopharyngeal cultures were obtained at 48 hrs of age. SS significantly reduced umbilical group B streptococcal colonization at 48 hrs (27%) as compared to CS (40%) and TD (40%). SS also significantly reduced umbilical gram negative rod colonization (52%) as compared to CS (90%) and TD (90%). TD was more effective in reducing *Staph. aureus* colonization (15%) than SS (54%) but both were more effective than CS (72%). *Staph. epi.* colonization was significantly higher in the SS group (61%) than in the TD and CS groups (48% and 41% respectively). There were no significant differences in group D streptococcal colonization. No adverse reactions or significant side effects were encountered in the study population.

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CHRONIC EOSINOPHILIC MENINGITIS DUE TO LYMPHOCYTIC CHORIOMENINGITIS VIRUS (LCMV). P. Joan Chesney, Murray L. Katcher, Donald B. Nelson, Sheldon D.

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LCMV infection of adult mice is the classic example of an immunopathologic disease, in that the cell mediated immune response of the host causes the disease.

We have seen a 2 1/2-year-old female with chronic meningitis of 80 days' duration manifested by fatigue, lethargy, CSF pleocytosis and high spiking fevers. A peripheral eosinophilia (1,472-2,688 cells/mm³) and CSF eosinophilia (3-25%) were found during a one-month hospitalization at the height of her illness. Fourfold rises in CF, Neut. and Fluorescent antibody titers established LCMV as the cause. Extensive evaluations for parasitic or other CNS or systemic disease were unrevealing.

T & B cell function and macrophage studies were normal during her illness. During a documented intercurrent influenza A/Victoria infection the following values were found:

	2/26/76	3/5/76
Total Lymphocyte Count	2,639/mm ³	4,182/mm ³
T Cells: Total E rosettes	264/mm ³	1,924/mm ³
(590-3090/mm ³)		
Active E rosettes	0/mm ³	1,004/mm ³
(≥225/mm ³)		
B Cells:	343/mm ³	627/mm ³
(≥225/mm ³)		

This is the first known report of prolonged fever, eosinophilia and chronic eosinophilic meningitis due to LCMV.

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MODULATION OF HERPESVIRUS HOMINIS INFECTION IN NEWBORNS BY MATERNALLY TRANSFERRED ANTIBODIES.

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The protective role of maternally transferred antibodies on resistance to H. hominis infection was examined by epidemiologic survey of 211 human sera for antibodies and by experimental infection in newborn mice.

Twenty five cord blood and 186 sera from persons of varying ages were tested for antibodies against H. hominis by indirect immunofluorescent staining method. The antibody reached the highest levels in the cord blood (mean titers = 1:1477) and the lowest levels between 6 months and one year of age (mean titers = 1:156). The antibody approached the adult levels (mean titers = 1:1193) by the age of 17 years. The apparent inverse relationship between the antibody levels in the population and the incidence of the disease indirectly suggests the protective role of the antibodies.

Exogenous administration of antibodies directed against H. hominis to pregnant mice resulted in an enhanced resistance to H. hominis infection in the offsprings. The mortality was 75% for the controls (35 mice) and 15% (P < 0.005) for the offsprings (39 mice) of mothers receiving antibodies.

These data suggest that maternally transferred antibodies play a significant role in modulating the pathogenesis of H. hominis infection in the newborn.