● 1021 RESPIRATORY SYNCYTIAL VIRUS(RSV)IN IMMUNOCOMPROMISED CHILDREN(IC).

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We prospectively studied in'74-'80 winters 353 children 3-5 yr old hospitalized with culture proven RSV to compare IC to normals (N). 30 were potentially IC: 13(group M) were on chemotherapy for malignancy or had immunodeficiency disease(ImD)(3); 17 in Group S were on steroids for 2mo-3 yr for non-malignant conditions. Their RSV illness is compared to 323 N(Table). In M most were nosocomial with lower respiratory disease(LRD) in all, even 3-5 yr olds (RSV from lung in 1), vs only <2yr olds in S and N. RSV shedding patterns(Table) from sequential nasal washes of M and S were compared to N, matched for age and disease. M had significantly greater titers & longer shedding. S also shed more virus than N. These findings suggest that immunosuppression for malignancy or ImD predisposes to more severe RSV illness and greater viral replication. Steroid treatment alone in other diseases may have allowed greater shedding initially but did not appear to result in more severe RSV illness.

RSV Groups (#):	M(13)		S(17)	N(323)
Age mos/range	16(6-63)		13(3-42)	6(.5-53)
% Nosocomial Dis	69*		53†•	23
% Lower Resp.Dis.	100		71•	88
% ICU Case due to	62*+		0	16
% Deaths {RSV	23*		0	0
RSV SHEDDING:	M-13 vs N-33		S-13 vs N-26	
Titer/Logs	5.6*	4.0	4.6†	2.9
Days Shed-av.	21*+	6	7	4
Range	4-47	1-19	3-20	1-21
% Shed ≥20 d	69*+	0	8	4

p<.01 for*M-N, +M-S, †S-N; p< .05 for •S-N

1022 EFFECT OF BLOOD TRANSFUSIONS ON GROUP B STREPTOCOCCAL ANTIBODY LEVELS.

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The morbidity and mortality rate from group B streptococcal infection continues to be exceedingly high even with the use of potent antibiotics and optimal support measures. In previous studies we suggested that transfusion of fresh, whole blood containing opsonic antibody might alter mortality in this fulminant neonatal disease (Lancet I: 636, 1978). In the present studies we have examined type specific streptococcal antibody levels and opsonic activity in 38 infants who received transfusions of fresh, whole blood. Thirty-three infants received donor blood containing higher levels of opsonic activity than in their own pre-transfusion specimen. Twenty of these infants had increased opsonic activity in their post-transfusion specimen. Among the 20 with increased levels, 18 received partial or complete exchanges amounting to 50% or more of their blood volume (mean 65 ± 7.2 SE%). The mean transfusion volume in the patients whose opsonic level did not increase was only 33 ± 7.5 SE%. Five patients received blood with lower opsonic activity than their own and all had decreased levels in post-transfusion specimens. The mortality rate in the 21 patients studied who had group B disease and received transfusion of fresh whole blood (14%) was exceedingly low. Thus, transfusion may significantly affect the outcome of group B disease.

1023 CHLAMYDIA TRACHOMATIS SEROPOSITIVITY IN SUDDEN INFANT DEATH SYNDROME CASES (SIDS) AND CONTROLS.

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Because of similarities in the epidemiology of Chlamydia trachomatis (C.t.) infant pneumonia and SIDS, antichlamydial antibodies were assayed in postmortem serum from 44 SIDS, age 13.0±6 wks and serum from 42 hospitalized living infants (C), age 13.3±6 wks. Cord blood (CB) from 35 lower socioeconomic black and 13 higher socioeconomic white infants were examined to establish representative cord serologies. A single-antigen (serotype L-2) indirect immunofluorescent test was used. The incidence of seropositivity (≥1:64) was higher in black than white infants but was higher in SIDS than controls in both black and white infants (*p< .05. These findings could represent either a higher incidence and/or degree of seropositivity in mothers of SIDS or C.t. infection of the infants. Although C.t. seropositivity may be merely a marker for the socioeconomic group known to be at high risk for SIDS, the data may suggest that factors associated with maternal C.t. infection, with or without transmission to the infant, are related to SIDS. Titers ≥1:2048 at 13.3±6wks of age in 14% of SIDS suggest that infant infection may be a factor in some SIDS.

Percent Chlamydia trachomatis Seropositive by Titer

	Total		White			Black		
Titer	C(42)	SIDS(44)	C(24)	SIDS(24)	CB(13)	C(18)	SIDS(20)	CB(35)
≥1:64	29%	61%*	21%	50%*	39%	39%	75%*	80%
≥1:1024	9.5%	25%*	4%	13%	8%	17%	35%	66%
≥1:2048	4.8%	14%	0%	15%	8%	11%	15%	43%

1024 MONOCLONAL ANTIBODIES PROTECT AGAINST FATAL GROUP B STREPTOCOCCAL (GBS) INFECTION IN MICE.

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Despite the use of broad spectrum antimicrobial agents, GBS sepsis remains a fulminant, often fatal disease. Using the technique of somatic cell hybridization, we have developed hybridomas which secrete monoclonal antibodies that react with each of the 5 serotypes of GBS. The purpose of this investigation was: 1) to determine whether type-specific monoclonal antibody conferred protection against fatal GBS infection in mice, and 2) to study the functional properties of the antibodies (agglutination, complement fixation). BALB/c mice were given a subscapular injection of 5 X 10^6 hybridoma cells that produced antibody with one of the following specificities: anti-GBS IA/IC, IB, or II. (Type III GBS does not infect adult BALB/c mice). After 14 days, tumor mice and an equal number of controls were challenged with an I.P. injection of 10^9 live GBS of identical specificity to the tumor. 100% of mice with tumors survived; controls were dead within 24 hours. Surviving tumor mice had anti-GBS titers of > 1/10,000 against the respective bacterial serotypes. Each monoclonal antibody fixed complement and agglutinated bacteria of the same specificity. GBS monoclonal antibodies protect mice against fatal infection in vivo, fix complement and agglutinate GBS in vitro and may have potential use in the therapy of life-threatening disease in infants.

1025 INDUCTION OF UTERINE CARCINOMA BY HERPES SIMPLEX VIRUSES TYPES 1 AND 2 (HSV-1 AND HSV-2) IN THE MOUSE.

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Epidemiological studies support the hypothesis that genital infection by HSV-2 is a cause of cervical (cerv) carcinoma (ca). Presumably a long latent period occurs between infection and onset of malignant changes. These studies also indicate that cerv ca has the epidemiological characteristics of a venereal disease, including sexual activity at a young age and with multiple partners. This suggests that genital HSV-2 infection acquired by sexually active adolescents may be the initiating event that results in cerv ca in adult life. There is no conclusive evidence in humans or animals, however, that cerv ca is the direct effect of genital exposure to HSV-2. The objective of this study, therefore, was to determine if exposure of the mouse cervix to HSV-1 or 2 induces cerv ca. C57 mice were exposed to formalin or ultraviolet-inactivated HSV-1 or 2 by insertion of virus-saturated cotton pledgets into the vagina 5 times a week for 80 weeks. This was felt to simulate the conditions of latent infection. Control mice were exposed to cell culture fluids without virus. Pap smears for cerv cytology were obtained biweekly. Cerv dysplasia progressing to invasive ca occurred in 24-60% of virus-exposed mice. Endometrial ca was detected in 7-28% of exposed mice. All controls remained normal. It was concluded that in the mouse prolonged exposure of the female genital tract to inactivated HSV-1 or 2 induces cerv dysplasia and ca and, less frequently, endometrial ca. Histologically these lesions were the same as those in humans.

1026 VENTRICULITIS WITH NEONATAL MENINGITIS: IDENTIFICATION BY REAL-TIME ULTRASOUND (US).

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Ventriculitis is reported to occur in 65-90% of neonatal bacterial meningitis and contributes to its recalcitrance to antibiotic therapy and to the occurrence of hydrocephalus and other neurological sequelae. The diagnosis of ventriculitis necessitates the invasive procedure of ventricular puncture, and identification of the structural changes associated with inflammation of the ventricular lining is made only at autopsy.

We have used the noninvasive technique of portable US to identify structural changes of the ventricular lining, presumably the consequence of ventriculitis, in two infants with neonatal bacterial menigitis. US scans demonstrated ventricular dilation and abnormal intraventricular echoes which were maximal in the first 2 weeks of the illness and which disappeared by 4 weeks. Ventriculitis was confirmed by analysis of ventricular CSF. The temporal profile of changes in intraventricular structures identified by US correlates with published neuropathological findings in neonatal meningitis, e.g. purulent ependymal exudate and organization into layers and fibrin strands in the first week of the illness with resolution by one month.

Our data demonstrate that US can identify structural correlates of ventricular inflammation and thus, provides a convenient, safe and informative means for assessing the complications of ventriculitis, e.g. loculated infection and hydrocephalus, in neonatal bacterial meningitis.