379 BLOOD PRESSURE RANGE IN NORMAL CHILDREN:MATURATIONAL DIFFERENCES BETWEEN MALES AND FEMALES.

L. George, T. A. Riemenschneider, G. Lee, S.J. Woerner, D.L. Meryash and D.T. Mason, Univ. Cal., Davis, CA. 95616.

Limited recent data are available on the range of blood pressure (BP) in normal children at different ages. Thus we studied 239 normal school children from 5 to 14 years (Y) of age: 136 boys and 103 girls. BP was obtained by the same examiner by auscultation; systolic pressure (SP) at the appearance of sound, and diastolic (DP) at its muffling. Pulse pressure (PP) and percentage of PP/SP were computed. Results were as follows for mean and standard deviations:

	BOYS				GIRLS			
Age	SP	DP	PP	PP%	SP	DP	PP	PP%
5Y	97±6	60±4	37±7	38±5	93±7	59±2	34±7	36±5
6Y	99±5	57±5	42±8	42±6	102±11	63±7	39±7	38±5
7Y	98±7	61±5	37±7	38±5	97±7	61±5	37±7	38±6
8Y	101±6	64±5	37±6	36±5	100±7	60±3	41±6	40±4
9Y	111±10	69±9	41±10	37±7	111±13	72±8	40±9	35±6
10Y	105±9	67±8	38±8	36±7	112±10	73±10	39±7	35±6
11Y	106±8	69±7	37±8	35±6	122±14	73±9	49±12	40±6
13Y	114±11	66±5	49±9	42±5	117±12	74±9	43±12	36±8
14Y	118±11	68±8	50±11	42±7	116±10	75±7	41±8	35±5

Our data show that after 8Y of age SP and DP increase rapidly. An earlier peak SP is achieved in girls by 11Y, while a slower rise in DP in boys results in a wider PP in the early teen years. All these changes may be related to the pubertal growth period.

(Sponsor: Eli Gold)

380 TRANSMISSIBILITY OF TYPE B HEPATITIS IN A SPECIAL CARE NURSERY;

Michael A. Gerber, Edward B. Lewin, Robert J. Gerety, Chinh T. Le, (Spon. by Martin R. Klemperer) University of Rochester School of Medicine and Dentistry, Strong Memorial Hospital, Department of Pediatrics, Rochester, N.Y.

The opportunity to investigate nurse-infant hepatitis B virus (HBV) transmissibility arose when a nurse in the special care nursery (SCN) developed acute Type B hepatitis. This nurse had close contact with all 31 neonates in the SCN during the month prior to the onset of her clinical hepatitis. Six months later sera were obtained from 24 of the 31 infants and from 22 of their mothers. Sera were analyzed for HBsAg, anti-HBc and anti-HBs. The nurse's serum and saliva were strongly positive for HBsAg; her serum anti-HBc = 1:32. Neither HBeAg nor anti-HBe was detectable in her serum. No infant or mother demonstrated evidence of HBV infection. These data show that a nurse caring for infants in a SCN did not transmit HBV to any of the neonates with whom she had direct contact despite the fact that she had acute icteric hepatitis, HBsAg in her serum and saliva, and ongoing viral replication. The fact that the nurse was HBeAg negative may have influenced the results.

381 HAEMOPHILUS INFLUENZAE TYPE B (HIb) DISEASE IN A DAY CARE CENTER.

C.M. Ginsburg, G.H. McCracken, Jr., and J.C. Parke, Jr. University of Texas Southwestern Medical School and Charlotte Memorial Hospital, Departments of Pediatrics, Dallas, Texas and Charlotte, North Carolina.

During a 14 month period, 7 of 48 children attending a day care center developed HIb disease. Five infants developed meningitis, 1 septic arthritis and the 7th infant had pneumonia and septicemia. Surveillance studies showed that at least 58% of infants carried HIb in their nasopharynx on 1 occasion.

Specific measures were instituted to control the outbreak; these included antimicrobial prophylaxis and immunization with HIb polysaccharide vaccine. Ampicillin (100 mg/kg/d) was administered to 6 asymptomatic carriers of ampicillin-susceptible HIb. At 1 and 7 days after therapy, HIb was isolated from 3 infants and at 14 days, 4 of 5 infants had positive cultures. HIb vaccine was administered subcutaneously to 34 children. Sera obtained prior to immunization showed detectable antibody in all infants. Only 9 (26%) infants had two-fold or greater rises in serum HIb antibody titers after vaccination. Antibody response was independent of age, pre-immunization antibody concentration and HIb carrier status. Vaccine was not only ineffective in reducing the carrier rate, it failed to prevent acquisition of the organism by non-carriers. An 18 month old infant previously identified as a carrier, developed meningitis 4 months after receiving and responding to the vaccine.

HIb is a highly contageous agent capable of causing disease in closed populations of susceptible infants.

382 GENETIC FACTORS IN THE DETERMINATION OF HUMAN ADIPOSE TISSUE MASS.

Fredda V. Ginsberg-Fellner, Andrew Davis, Gary J. Bergman, Jacqueline S. Schenkein-Stern and Jerome L. Knittle. Mount Sinai School of Medicine, N.Y.,N.Y. Department of Pediatrics and Columbia University, School of Dental and Oral Surgery, N.Y., N.Y.

The determinants of human adipose tissue mass are not yet elucidated. In order to characterize the role of genetic factors, total fat cell number was measured in 12 sets of identical and 6 sets of fraternal twins, ages 1 to 16, and one set of 10 year old identical female triplets. 8 of 10 sets of normal weight identical twins were concordant for total adipose cell number, current weight and height and birthweight while the other two sets of identical twins had significantly dissimilar birthweights and total cell numbers. In these latter children, cell number was lower in the twin with the smaller birthweight. In addition, the triplets were identical for all the parameters measured. Three sets of identical obese twins with similar birthweights all displayed significant and concordant adipose tissue hypercellularity for age. Differences in the degree of their adiposity were reflected primarily by alterations in fat cell size. The 6 sets of fraternal pairs had both similar and disparate fat cell numbers independent of their current weights and birthweights.

The data indicate an important genetic component in the development of the adipose tissue depot in man with in utero factors modulating the final outcome.

383 BLACK-WHITE SIMILARITIES IN CORD BLOOD LIPIDS AND LIPOPROTEINS.

C.J.Glueck, P.S.Gartside, R.C.Tsang, M.J. Mellies, P.M.Steiner. Univ.Cincinnati,Coll.Med., GCRC.

Cord blood cholesterol (C), triglyceride (TG), high density C (C-HDL), low density C(C-LDL) and C-HDL/C-LDL were quantitated in 117 neonates (58 white,59 black) to assess for neonatal expression of racial lipid differences. The 117 neonates were studied in their consecutive birth order in a lipoprotein survey of 3000 births. Comparisons of cord blood lipoproteins were made using the following groups: all black(B) vs all white(W), all male(M) vs all female(F), and sex-race interaction. Tests of difference were made using the general linear hypothesis method, allowing analyses of variance on data composed of unequal numbers of observations:

Lipids (X±SD)	White M,n=32	White F,n=26	Black M,n=41	Black F,n=18
TG	28±13	29±16	34±17	38±21
C	71±15	74±13	72±17	71±15
C-HDL	34± 8	38± 9	35±12	35±11
C-LDL	33±11	30±11	35±12	32±10
C-HDL/C-LDL	1.2±.5	1.4±.8	1.2±.6	1.2±.5

There were no black-white or male-female differences in cord blood C, C-HDL, C-LDL, or C-HDL/C-LDL. Cord blood TG was slightly higher in black neonates, $p<.02$. For all 117 neonates, C-HDL correlated with total C ($r=.63, p<.001$), but not with C-LDL ($r=.002$). At comparable total C levels there were no neonatal black-white differences in C-HDL in contrast to older children and adults, where blacks have higher mean C-HDL. Within the limitations of "genicity" as expressed by cord blood lipoproteins, black-white C-HDL differences are not apparent at birth and may be acquired later in childhood.

384

Abstract withdrawn