29 THE GENETIC PATTERN OF THE IMMUNOGLOBULIN POTENTIAL IN CHILDREN.

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After the Mancini's et al (1965) method we investigated the levels of the main immunoglobulin classes - ,M,G - in healthy and sick children in age of 1 - 14 ears with different group relationships to the isoantigenic systems of A.B.O and Rh.Various infectious rocesses occured in sick children: pseudotuberculosis n the form of the Far-Eastern scarlatiniform fever, nfectious and serum hepatitis, cholecystitis, salmoollosises, rheumatoid arthritis etc. Both in healthy nd sick children rather distinct regularities in the enetic patterns of the immunoglobulin potential were letected in all the classes studied irrespective of he etiology and pathogenetic features of the infeion. A particularly distinct dependence was revealed n the level of IgG, one of the functions of which is he close relation to the organism's predisposition o allergy. The obtained data allow to reveal the roups of the special risk and choose the exact indiidual approach to the patient's treatment.

30 SERUM IMMUNOGLOBULIN VARIATIONS DURING THE EVOLUTION OF ACUTE LYMPHOBLASTIC LEUKAEMIA (A L L) IN CHILDHOOD.

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Sérum immunoglobulin titers using continuous flow liquid immunonephelometry in 62 patients with A L L gave the following results :

1. Before therapy, IgM values were elevated whereas those of Ig A and Ig G were within normal limits.

2. During induction therapy, all three Ig classes were temporarily decreased.

3. During the remission period, Ig G levels were normal, however the distribution curve showed the patient populations in whic the larger group had Ig G values inferior the normal mean value. Ig A values were clearly decreased, where as those of Ig M were normal.

4. During reinduction therapy there was a 10 to 30 p. 100 decrease in Ig A + G values.

5. Localised or systemic relapse did not show any varia - tions in serum immunoglobulin levels.

31 GRAFT VERSUS HOST (GVH) REACTION MANIFESTED AS TOXIC EPIDERMAL NECROLYSIS (TEN) IN A CHILD WITH ACUTE LEUKEMIA

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Patients with deficient cell mediated immunity levelop GVH reaction after administration of immuologically competent cells (i.e.bone marrow or rgan grafts or blood transfusions).

A fatal GVH disease was observed in a 5 year old male with acute leukemia who has been treated with corticosteroids, 6 mercaptopurine, and blood transfusions. A typical picture of TEN (intense erytaema,bullous lesions, a positive Nikolsky's sign) was seen 7 weeks after the diagnosis of leukemia. Multiple skin biopsies revealed acute epidermal necrolysis with areas of cleavage in the malpighian zone and focal single cell necrobiosis with intra epithelial satellite lymphocytes. There was also mild perivascular and pediadnexal cuffing consisting of lymphocytes and plasmacytoid type of cells. These findings, especially single cell dyskeratosis and necrobiosis were consistent with GVH reaction as mostly described in patients after allogeneic bone marrow transplantation.

32 GRAFT-VERSUS-HOST (gvh) REACTION OCCURRING IN TWO NEONATES WITH THYMIC DYSPLASIA DUE TO POSSIBLE MATERNO-FETAL TRANSFUSION

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Typical findings of gvh disease were detected at autopsy in two infants, 2 and 22 day olds, products of related parents (first cousins). The first case presented with jaundice, erythema, peeling of the skin mimicking Ritter's disease. The second case came with history of cyanosis, cough, diarrhea, and moniliasis of 10 days duration. The thymus was dysplastic in both. Lymphoid tissues (spleen, lymph nodes, intestines) showed marked depletion of lymphocytes with proliferation of plasmatoid cells. A striking degree of plasmacytosis and histocytosis was present in the hypoplastic bone marrows. The skin of the first case revealed epidermal necrolysis with intraepithelial stallite cells. Histocytosis was the main finding in addition to bronchopneumonia in the second case.

Since neither of the immunoincompetent infants were given plasma or blood transfusions, possible materno-fetal transfusion was suspected to be the cause of the gvh reaction.

33 EXCHANGE TRANSFUSION AND ROSETTE-FORMING CELLS.

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Cellular immunity was studied by the spontaneous rosette forming lymphocyte technique in the peripheral blood of fourteen jaundiced newborns, who were exchange transfused. Following exchange transfusion a gradual increase of both per cent and absolute values of rosette-forming cells was observed. On the 7th post exchange transfusion day per cent rosettes had almost doubled ($t=11.2$, $P<0.001$) whereas absolute numbers had increased by more than two fold ($t=6.37$, $P<0.001$). These results suggest that the therapeutic benefit of exchange transfusion in severely ill newborns may be due appart from other factors to its stimulating effect on cellular immunity.

34 DEFICIENCY OF LATE COMPLEMENT FACTORS WITH RECURRENT MENINGOCOCCAL SEPTICAEMIA.

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In a 2 years old boy two episodes of meningococcal septicemia gave rise to investigate the immune defense mechanisms. No abnormalities were found in respect to serum immunglobulin levels, lymphocyte subpopulations, response of lymphocytes to PHA and granulocyte functions (NBT reduction, phagocytic and bactericidal activities). The patient' s serum, however, totally lacked whole complement haemolytic activity. Detailed analysis of the complement system revealed a marked diminution of the functional activities of the complement factors C6 through C9. However, no single complement factor was completely absent.

Bactericidal and opsonic activities of the patient's serum on various bacterial strains were investigated in microcultures containing bacteria, serum of the patient and - when opsonic activity was studied - normal granulocytes. Inhibition of H^3-thymidin uptake by growing bacteria was used as a parameter of serum-associated anti-bacterial activities. The results indicate that impaired function of late complement factors may predispose affected individuals for septicemia.