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 years with different group relationships to the isoantigenic systems of A, B, O and Rh. Various infectious processes occurred in sick children: pseudotuberculosis in the form of the Far-Eastern scarlatiniform fever, infectious and serum hepatitis, cholecystitis, salmonellosis, rheumatoid arthritis etc. Both in healthy and sick children rather distinct regularities in the genetic patterns of the immunoglobulin potential were detected in all the classes studied irrespective of the etiology and pathogenetic features of the infection. A particularly distinct dependence was revealed in the level of IgG, one of the functions of which is the close relation to the organism's predisposition to allergy. The obtained data allow to reveal the groups of the special risk and choose the exact individual 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 which the larger group had Ig G values inferior to the normal mean value. Ig A values were clearly decreased, whereas 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 variations 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 develop GVH reaction after administration of immunologically competent cells (i.e. bone marrow or organ 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 erythema, 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 periadnexal cuffing consisting of lymphocytes and plasmacytoid type of cells. These findings, especially single cell dyskeratosis and necrobiosis were consistent with GVH reaction as previously 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 monilia of 10 days duration. The thymus was dysplastic in both. Lymphoid tissues (spleen, lymph nodes, intestines) showed marked depletion of lymphocytes with proliferation of plasmacytoid cells. A striking degree of plasmacytosis and histiocytosis was present in the hypoplastic bone marrows. The skin of the first case revealed epidermal necrolysis with intra epithelial stallite cells. Histiocytosis 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 exchanged 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 apart 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 immunoglobulin 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.