the cells of Lymphoma AKRL1 in hosts of the above mentioned sorts, and it likewise failed to inhibit growth of cells of 2 additional lymphomas (AKR13 and AKR17) in C3H mice. The findings make it plain that guinea pig serum does not act against the 3 types of AKR lymphoma cells even when these are implanted in alien hosts; indeed in some of the experiments guinea pig serum seemed to enhance the growth of AKRL1 cells in alien hosts. Considered together the observations weigh heavily against the supposition that the mouse host provides natural or induced isoantibodies which act in conjunction with guinea pig serum in killing the cells of Lymphoma 6C3HED *in vivo*, and they support the inference that cells of Lymphoma 6C3HED differ intrinsically from those of Lymphoma AKRL1, the former being susceptible to the effects of guinea pig serum *in vivo* while the latter are insusceptible.

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Effect of Tumors on Level of Plasma Amino Acids of Eviscerate Rats.* (22201)

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It was expected that the presence of a growing tumor would suppress the rise in plasma amino acids which occurs following evisceration. The contrary is true. The rise is more rapid in eviscerate rats which are hosts to a Walker carcinoma.

Methods. Male rats of the Sprague-Dawley strain were fed Rockland rat diet. Suspensions of tumor were implanted intramuscularly into each thigh near the trunk and permitted to grow for periods of 4 to 14 days. At a weight of approximately 250 g non-fasted rats were anesthetized with cyclopal sodium and functionally eviscerated. Some of the animals were kept without infusions and others were given continuous intravenous infusions of glucose with and without insulin for periods of either 3 or 6 hours. Heparinized blood was collected by cannula from the abdominal aorta and the level of plasma amino acids determined by the method of Hamilton and Van Slyke(1).

Results. The conditions of individual experiments and the results are shown in Table I. The values are grouped according to the combined weights of tumors of each rat at autopsy. The level of plasma amino acids is not elevated in non-eviscerated tumor hosts. The average values for plasma amino acids increased more rapidly in tumor-bearing rats, the extent of rise showing a positive relationship to the weight of the tumors. Although insulin suppresses the rise in plasma amino acids in all eviscerate animals(2) it does not prevent a more rapid rise in tumor hosts. Whether the extra plasma amino acids arise from the tumor itself, or from normal tissues which are catabolized more rapidly in the presence of a tumor, is not known to us. We have no information about changes in tumor metabolism following evisceration.

Summary. Following evisceration the level of plasma amino acids rises more rapidly in rats which are hosts to a Walker carcinoma

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TABLE I. Level of Plasma Amino Acids in Rats Bearing Tumors. Averages and standard errors.

Glucose load, mg/100 g/rat/hr	Insulin, units/24 hr	Hr	No. rats	Tumor wt, g	Plasma amino acids, mg %
0	0	0	12	36.94 ± 5.46	3.97 ± .28
0	0	3	12	0.0	9.35 ± .33
0	0	3	12	2.34 ± .39	11.55 ± .45
0	0	3	8	8.98 ± 1.12	14.26 ± .88
0	0	3	10	22.63 ± .79	15.09 ± .79
0	0	3	10	37.72 ± 2.73	15.83 ± .60
0	0	6	8	0.0	15.00 ± .82
0	0	6	8	1.68 ± .27	17.91 ± 1.13
0	0	6	6	8.15 ± 1.36	20.77 ± .76
0	0	6	6	25.36 ± 1.50	20.26 ± 1.13
0	0	6	10	53.78 ± 6.21	21.64 ± 1.11
70	0	3	17	0.0	8.82 ± .09
70	0	3	17	19.1 ± 5.46	10.59 ± .25
70	16	3	17	0.0	4.48 ± .11
70	16	3	17	19.76 ± 3.41	7.30 ± .44

than in similar rats without tumors. The extent of rise shows a positive relationship to the size of the tumors at autopsy. The differences are evident in the presence and absence of insulin.

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Studies on Agammaglobulinemia VI. Hemostasis in Patients with Agammaglobulinemia.* (22202)

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The discovery of agammaglobulinemia(1) has opened a new field of investigation in immunology and protein metabolism. Appreciable information concerning these patients has accumulated, especially in the field of immunology(2). In all conditions with a congenital or acquired defect of protein synthesis the problem arises whether one is dealing with an isolated error of metabolism or with a group of deficiencies. Typical examples of isolated defects of protein synthesis are the congenital disturbances of coagulation: *e.g.* afibrinogenemia, the various subgroups of hemophilia, hypoprothrombinemia, etc. Examples of combined defects of protein synthesis are found with severe liver damage, where hypoprothrombinemia is usually associated with deficiencies of prothrombin conversion factors, hypofibrinogenemia and hypoalbuminemia. The association of a number of defects can usually be traced to a dysfunction of the common organ of synthesis.

Because of the existing controversy about the site of synthesis of gamma globulins, it was considered of interest to determine the concentration of the presently known clotting factors and wherever possible to evaluate the adjustment of these factors in response to stressful situations in patients with demonstrated inability to synthesize gamma globulin. Complete tests of hemostasis were performed in 4 children with congenital agamma-

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