

This allows the varices to prolapse into the lumen of the instrument. It is then a simple matter to advance the needle directly into the varix.

The injection is made as near to the cardia as possible (see fig. (b)). After injecting 5 ml of ethanolamine oleate the needle is withdrawn, and the oesophagoscope advanced to compress the site of the injection for one to two minutes (see fig. (c)). The oesophagoscope is then withdrawn to allow the widened proximal end which carries the lighting to emerge from the mouth so that the instrument can be rotated. It is then advanced again. Five or six sites equally spaced around the circumference of the lumen are usually injected at the first sitting; up to 30 ml of sclerosant may be used. A Sengstaken-Blakemore tube may be passed if there is any significant bleeding; it is usually removed after six to 12 hours.

¹ Rothwell-Jackson, R. L., and Hunt, A. H., *British Journal of Surgery*, 1971, 58, 205.

² George, P., et al., *British Journal of Surgery*, 1973, 60, 635.

³ Pugh, R. N. H., et al., *British Journal of Surgery*, 1973, 60, 646.

⁴ Orloff, M. J., et al., *Archives of Surgery*, 1974, 108, 293.

⁵ Johnston, G. W., and Rodgers, H. W., *British Journal of Surgery*, 1973, 60, 797.

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Bone Marrow Suppression by Antilymphocytic Globulin

The problem of suppressing bone marrow function by preparations of antilymphocytic globulin (A.L.G.) was reported at a recent conference on A.L.G. at the Royal College of Physicians. We report some preliminary studies which show that A.L.G. inhibits human bone marrow colony forming cells.

Case Histories

Burroughs Wellcome A.L.G. was studied as our group has used this preparation in marrow transplantation in two patients where acute graft-versus-host disease followed marrow transplantation from a family donor judged compatible by H-LA typing and the mixed lymphocyte reaction. The first patient, a 6-month-old boy with severe combined immune deficiency disease, was given a single dose of A.L.G., 8 mg/kg. This improved the graft-versus-host disease without impairing marrow function. The second patient, a 27-year-old man with severe aplastic anaemia, received A.L.G., 10 mg/kg, which abolished the graft-versus-host disease. No engraftment occurred and the patient died three days after a second graft was given.

To study bone marrow colony growth bone marrow in heparin was taken from haematologically normal patients. The buffy coat obtained by sedimentation was washed three times in culture medium and the cell count adjusted to 10^6 cells/ml. Marrow was incubated for three hours in the presence of A.L.G. in concentrations varying from 0 µg/ml-100 µg/ml with or without the addition of autologous serum as a source of complement. The marrow was then plated in soft agar on previously prepared peripheral blood leucocyte feeder layers as a source of colony stimulating factor, using the method described by Pike and Robinson.¹ After 10-14 days colony counts were performed on triplicate cultures and the results of two such experiments are shown in the table.

Bone Marrow Colony Growth after Incubation with A.L.G. or Horse Globulin

Marrow	Autologous Serum	Colonies/ 3×10^4 Cells Plated Mean of Three Culture Plates					
		Control No Feeder	Control	A.L.G. µg/ml			
				0.1	1.0	10	100
1	None	0	75	67.6	56.3	31.6	16.0
2	None	1.0	141	—	158	94.6	73.0
1	0.1 ml	0.6	76	52.3	33.3	25.3	5.3
2	0.1 ml	2.0	145	78	71	23	5.5
				Horse Globulin µg/ml			
				0.1	1.0	10	100
2	0.1 ml	2.0	145	97.6	118	163	159

Discussion

The results show that A.L.G. significantly inhibits marrow colony formation at doses as low as 0.1 µg/ml ($P < 0.05$) and that fresh serum significantly enhances this effect ($P = 0.005$) while horse globulin causes no inhibition. A.L.G. causes depression of rat stem cells² but no direct measure of human stem cells is available. The action of A.L.G. on human colony forming cells may be relevant to its harmful effect after marrow transplantation. Our preliminary experience shows that the dose of A.L.G. used may be critical in allowing marrow to take. We hope to use this technique to test a variety of A.L.G.s for marrow toxicity, and to investigate the possibility of removing the anti-stem-cell activity while preserving its anti-lymphocyte action by absorption with myeloid cells in the way Marmont described.³

¹ Pike, B. L., and Robinson, W. A., *Journal of Cellular Physiology*, 1970, 76, 77.

² Field, E. O., and Gibbs, J. E., *Nature*, 1968, 217, 561.

³ Marmont, A. M., in *Proceedings of the Symposium on Anti-lymphocyte Globulin in Clinical Practice*, ed. R. Rousell. In press.

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Postpartum Rubella Vaccination and Anti-D Prophylaxis

Rubella vaccination of recently delivered women for preventing rubella and congenital malformations in future pregnancies is now routine. Simultaneously some patients run a risk of Rh immunization. As anti-D immunoglobulin preparations from pooled human sera, which should be administered post partum, also contain rubella antibodies, Alderman and Charles¹ have recommended the postponement of rubella vaccination in these cases. This practice has several disadvantages; particularly if conception occurs during the viraemia. We have used an anti-D immunoglobulin preparation with a 1:1024 rubella HI-titre and studied its influence on the development of immunity after rubella vaccination.

Case Histories

We screened 587 antenatal patients for the presence or absence of rubella antibodies by a haemagglutination-inhibition method. No rubella antibodies were detectable (seronegative—that is, titre <8) in 6% of these patients. Twenty one and 25 were investigated in detail three and six weeks after vaccination, respectively. Previous experience had shown that seropositive patients with titres as low as 1:8 did not show an appreciable antibody titre rise after rubella vaccination. Patients in group A received the rubella vaccine—0.5 ml of live, attenuated rubella virus vaccine, duck embryo cell-adapted HPV-77 strain, containing not less than 1000 TCID₅₀ of rubella virus (Meruvax, MSD)—on the fifth day of the puerperium. Patients in group B (both rhesus-negative and -positive) were given 250 µg anti-D immunoglobulin in 2 ml of a 16% gammaglobulin preparation intramuscularly (immunoglobulin anti-D SRK) within 48 hours of delivery, followed three days later by the rubella vaccine. Several patients had no alteration of rubella antibody titre three days after administration of this anti-D preparation. Three weeks and six weeks after the rubella vaccination, rubella HI-titres were found in both groups (see table). The conversion rate in both (admittedly small) groups was 100%. Two patients responded poorly, one in group A with a titre of 1:8 six weeks after vaccination, another in group B with a titre of 1:16. The latter had received 3 units of blood at delivery.

Results

We conclude that the rubella antibodies administered with the anti-D immunoglobulin do not delay or prevent active immunity against the rubella virus when the vaccine is given at about the same time. Our