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British Journal of Haematology, 1977, 36, 447.

Correspondence

HAEMOPHILIA CENTRE DIRECTORS' ANNUAL STATISTICS FOR 1975

Since 1969 the Directors of Haemophilia Centres in the United Kingdom have collected information about the numbers of patients having haemophilia A and B, about the treatment received by these patients and about the complications of treatment. A report on the statistics for the 6 years 1969–74 has appeared in *British Journal of Haematology* (Biggs & Spooner, 1977). The purpose of the present letter is to bring the statistics up to date by adding the main figures for 1975. It is intended to carry out this updating process annually until the accumulation of data warrants a more complete report. The data now presented will enable those who wish to do so to fill in one further year's figures for Tables III, VI, IX, X, XI, and XIV of the prior publication (Biggs & Spooner, 1977).

TABLE III. Deaths during 1975

Haemophilia A	6 deaths (1 cancer, 2 cerebral haemorrhage, 1 cardiovascular disease, 1 no information, 1 hepatitis B, 1 post-operative bronchopneumonia)
Haemophilia B	2 deaths (both come under the heading of 'Other types of bleeding')

TABLE VI. Factor VIII preparations used during 1975 to treat haemophilia A

Type of material	*Factor VIII (units)*	*% total*
Plasma	355400	1.43
Cryoprecipitate	16286998	65.45
NHS VIII concentrate	3085465	12.40
Commercial VIII concentrate	5551935	20.70*
Other	6420	0.02
Total	24886218	100.00

Number of Haemophilia Centres	53
Number of patients treated*	1670
Average amount of factor VIII units used per patient	14902

* Excluding those not transfused and adjusted for duplicates.

TABLE IX. Material used to treat haemophilia B (Christmas disease) patients during 1975

Type of material	*Factor IX(units)*	*% Total*
Plasma	23450	0.48
NHS factor IX concentrate	4832393	98.33
Commercial factor IX concentrate	58800	1.19
Total	4914643	100.00

Number of Centres with Christmas disease patients	43
Number of patients treated*	275
Average amount of factor IX units used per patient	17871

* Excluding those not transfused and adjusted for duplicates.

TABLE X. The incidence of jaundice in haemophilia A patients during 1975

Patient-treatment-years	*No. of incidents of jaundice*		*%*
1670	45*	(56)	2.69

* One patient had two attacks of jaundice. The figure in parentheses includes patients who had raised LFTs but were not ill.

TABLE XI. The incidence of jaundice in Christmas disease patients during 1975

No. of patient-treatment-years	*No. of incidents of jaundice*	*%*
275	2	0.73

TABLE XIV. Incidence of factor VIII or factor IX antibodies in patients having haemophilia A or B

Haemophilia A				*Haemophilia B*			
Cumulative total number of patients in survey	*Cumulative number with factor VIII antibody*	*%*	*New cases detected in 1975*	*Cumulative total number of patients in survey*	*Cumulative number with factor IX antibody*	*%*	*New cases detected in 1975*
2854	184	6.45	13	446	5	1.12	0

Points which may be emphasized in the figures for 1975 are:

(1) The total number of haemophilia A patients who have been treated at the Centres is now nearly 3000, which was the number originally estimated to be the total for the United Kingdom.

(2) The amount of commercial factor VIII used has increased from 13% in 1974 to 20% in 1975.

(3) The incidence of jaundice in 1975 was substantially less than that in 1974.

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REFERENCE

BIGGS, R. & SPOONER, R. (1977) Haemophilia treatment in the United Kingdom from 1969 to 1974. *British Journal of Haematology*, 35, 487.

PLATELET SIZE AND THROMBOCYTOPOIETIC STIMULUS IN MAN

Recent correspondence in the Journal (1977, 35, 473) prompts me to publish relevant results in man. Fig 1 shows circulating macrothrombocytes, bone marrow megakaryocyte differential, and plasma thrombopoietic activity in 20 patients: 18 with severe thrombocytopenic and two with thrombocytosis plus carcinomatosis. Patients in the positive (+) mouse test group include one varicella-induced thrombocytopenia, one acute ITP, two chronic ITP (one splenectomized), one Moschcowitz's syndrome, and one familial thrombocytopenia thrombocytopathy. Patients with negative (−) mouse test: nine chronic ITP (two splenectomized), one subacute ITP (a child), one drug-induced thrombocytopenia, and one liver cirrhosis. In the mouse test, 5 or 6 d after starting (a single dose of 0.2 ml plasma i.v. or 0.4 ml i.p.) increases in circulating platelet counts of 30% or more in all, or all but one of the animals (usually five) were considered as positive. Platelet size was measured by ocular micrometry in EDTA smears.

Our mouse test is too insensitive to detect moderate changes in plasma thrombocytopoietic activity. Nevertheless, even stimuli which do not cause positivity in this test induce significant macrothrombocytosis and macrothrombocytosis which is associated with a shift in the megakaryocyte differential to the left. We observed that the frequent failure to demonstrate higher levels of plasma thrombocytopoietic activity in our mouse test, injecting the animals with chronic ITP sera, needs to be explained (Kelemen *et al*, 1960). Paulus (1974) demonstrated that macrothrombocytosis may be present even in conditions with normal platelet survival, e.g. hypersplenism. Plasma of hypersplenic patients, however, may be active in the mouse test (Kelemen, 1969: Table 106). Thus, although shortened platelet survival alone may not be the only cause of macrothrombocytosis, the basic observation of Karpatkin remains valid. May I refer to my monograph 'As in the case of the other systems a so-called shift to