Risk of AIDS for Recipients of Blood Components From Donors Who Subsequently Developed AIDS

By Herbert A. Perkins, Susan Samson, Jane Garner, Dean Echenberg, James R. Allen, Morton Cowan, and Jay A. Levy

Reported cases of acquired immunodeficiency syndrome (AIDS) in San Francisco as of March 31, 1986, include 92 individuals who had donated blood subsequent to 1978. Their donated blood components had been transfused into 406 different recipients. The current status of 336 of these recipients was ascertained as of April 1, 1986. Of these, 223 had died at the time of our first contact, almost all as a result of the condition for which they were transfused. Seven had developed AIDS; five of these died, two before entry into the study and three subsequently. Forty-six additional living recipients were interviewed and evaluated. Seven had the AIDS-related complex, 18 had antibody to the human immunodeficiency virus (HIV) but were otherwise healthy, and 19 had no detectable anti-HIV. Two had risk factors other than transfusion. The frequency of infection of the recipient decreased as the time interval between transfusion and the diagnosis of AIDS in the donor increased. This information should be useful when counseling patients who have been transfused with blood components from donors later found to be infected with HIV.

ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS) is caused by a virus or a group of related viruses designated lymphadenopathy-associated virus[1] human T cell lymphotropic virus, type III,[2] or AIDS-associated retrovirus.[3] Recently an international committee has recommended that these viruses be named human immunodeficiency viruses (HIV).[4] HIV can be transmitted to patients by transfusion of blood components from donors who are carriers of the virus. Although most of these carrier donors appear healthy when subsequently examined, virus can usually be cultured from their peripheral blood lymphocytes.[5] Other HIV-infected donors have later developed symptoms, signs, and laboratory test results consistent with AIDS-related complex (ARC),[6] and a small proportion subsequently meet the criteria for a diagnosis of AIDS as defined by the Centers for Disease Control (CDC).[7] Before 1983 it was not recognized that persons from groups at high risk for AIDS should be excluded as blood donors, whereas it is now evident that substantial numbers of cases of AIDS occurred in this country as early as 1979, with a few cases occurring even earlier.

The three national blood bank organizations (American Association of Blood Banks, American Red Cross, and Council of Community Blood Centers) have endorsed a statement that recipients should be notified when a donor of a blood component that they have received subsequently develops AIDS.[8] Interviewers of patients with AIDS should always ask whether they had donated blood for transfusion. If so, the center where the blood was donated should be notified. Often this procedure is done through the local health department.

Ultimately the recipient is informed of the potential exposure, and the patient's first concern will be about the likelihood that he or she may develop AIDS. This report presents information on the risk of becoming infected with HIV and of developing AIDS for recipients of blood components whose donors subsequently develop AIDS. It is likely that the findings apply equally well when the donor remains healthy but anti-HIV is detected. The majority of donors with Western blot–confirmed anti-HIV (over 90% in our experience) have cells in their blood from which HIV can be cultured.

PATIENT SELECTION AND METHODS

The Irwin Memorial Blood Bank collects blood from volunteer donors in San Francisco and seven other counties of northern California. More than 100,000 donations have been given yearly for the past 7 years. As of March 31, 1986, 1,870 cases of AIDS had been reported to the San Francisco Department of Public Health, which has resulted in the highest per capita incidence of any major city in this country. The first case of AIDS associated with receipt of a blood transfusion was reported from San Francisco in December 1982.[9,10] The blood donor developed AIDS 9 months after the blood had been given. In December 1982, the Irwin Memorial Blood Bank and the San Francisco Department of Public Health agreed to share information so that they could identify all possible AIDS patients who had donated blood at an Irwin facility subsequent to 1978.

The list of AIDS patients reported to the San Francisco Department of Public Health was compared at regular intervals with the blood bank files to identify the dates of donation, the blood components made from each donation, and the hospitals to which the components were dispensed. The hospital contacted the physician who ordered the blood or the current personal physician of the recipient. The physician informed the patient that he or she may have been exposed to the AIDS agent through transfusion. The physician was also requested to ask his or her patient to participate in this current investigation. With permission of the personal physician, the recipient was contacted, provided with information about the study, and asked for written consent to be included in the study after the nature of the procedures had been fully explained. The protocol and the consent forms were approved by the University of California San Francisco Committee on Human Research.

Each consenting recipient was interveiwed by an experienced member of the blood bank epidemiology staff to determine whether there was any evidence that he or she had a risk factor for AIDS

From the Irwin Memorial Blood Bank, San Francisco; the San Francisco Department of Public Health; the Centers for Disease Control, Atlanta; and the University of California School of Medicine, San Francisco.

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Address reprint requests to Herbert A. Perkins, MD, Irwin Memorial Blood Bank, 270 Masonic Ave, San Francisco, CA 94118.

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apart from transfusion. Such patients were eliminated from the final analysis. The interview, which included a brief physical examination, also searched for evidence of AIDS or ARC. Blood samples were taken for laboratory tests. T cell subset analysis was done in the first 13 cases by fluorescent microscopy using OKT4 and OKT8 monoclonal antibodies (Ortho Pharmaceutical Corp, Raritan, NJ) and in the remainder by flow cytometry using Leu-3 and Leu-2 monoclonal antibodies (Becton Dickinson, Mountain View, CA) in a Becton Dickinson FACS Analyzer. Mitogen stimulation of lymphocytes with phytohemagglutinin (PHA) was done in most cases. Mixed lymphocyte cultures (MLC) were attempted in the first 13 cases and stimulation with pokeweed mitogen (PWM) in the remainder. β_2-Microglobulin levels were measured by an enzyme immunoassay microtiter technique using reagents supplied by Pharmacia Fine Chemicals (Piscataway, NJ). Commercially available Abbott kits (Abbott Laboratories, Abbott Park, IL) were used for the hepatitis tests, and anticytomegalovirus (CMV) antibody was detected by reverse passive latex agglutination (BBL Microbiology Systems, Cockeysville, MD). Serological tests for antibody to HIV were performed by licensed enzyme immunoassay (EIA) methods at the blood bank. The Abbott EIA method was used in all cases; ENI (Electronucleonics, Columbia, MD) and Genetic Systems (Seattle, WA) methods also were used when there were discrepancies with other tests. Other tests for anti-HIV in all cases were the immunofluorescent antiglobulin slide test (IFA) at the University of California, San Francisco (UCSF)[3] and the immunoblot (IB) (Western blot) technique at CDC and UCSF.[11] Antibody was considered to be present if both IFA and IB were positive and to be absent if both IFA and IB were negative.

Although the majority of recipients had consistent results in their multiple tests for antibody to HIV, discrepancies between IFA and IB reports were initially noted in six of the 53 cases (11%). Criteria for the IB were not standardized initially, but later a gp41 band was required for a positive reaction, and with multiple blinded replicates, the two laboratories ultimately reported identical results.

RESULTS

Clinical status of the recipients. As of March 31, 1986, the San Francisco Department of Public Health list of 1,870 reported cases of AIDS meeting requirements of the CDC definition included 90 previous donors from the Irwin Memorial Blood Bank. Two additional donors from a neighboring blood bank were included early in the investigation; subsequently, only Irwin donors were followed. Most donations had been separated into components, each of which had been given to a different recipient, and most of the donors had given blood multiple times since 1978. The total number of patients who had received the components under investigation was 406. Of these, 223 (54.9%) were deceased at the time of our first contact with their physician. According to the records, almost all of these died as a result of the illness for which they were transfused, but two cases of AIDS had been recognized. In the rest, no evidence for HIV infection had been noted clinically or (when done) at autopsy, but it had rarely been considered in the differential diagnosis. The most common indications for transfusion of the deceased patients were gastrointestinal bleeding, leukemia, and cardiac surgery. The most common cause of death was leukemia, but death of unknown cause was equally common. The average interval between transfusion and death was 10 months. Of the remaining recipients, 113 (27.8%) were alive, and the status of 70 (17.2%) has not yet been determined. Information on the clinical status of the 223 dead and 113 living recipients was obtained from their personal physicians.

Seven of these recipients had developed AIDS, two died before initiation of this study and three between entry into this study and preparation of this report. Two were still alive at the time of this report. We interviewed and evaluated 46 additional recipients who were alive at the time of our initial contact. These were all of the living recipients who lived within several hundred miles of us and for whom permission had been obtained to participate in the study. Table 1 summarizes the data obtained concerning these 53 recipients. Eight of the recipients had signs, symptoms, and laboratory test results consistent with ARC.[6] The remaining 38 recipients appeared to be healthy, but 19 of them had antibody to HIV in their sera, and in the other 19 no antibody could be detected. The percentage of potentially exposed recipients for whom evidence of infection was obtained was thus 62% (33/53). Two of the living recipients (one with ARC and one with anti-HIV only) acknowledged having other risk factors for AIDS; therefore, they will be eliminated from further analysis. Case no. 14 had had repeated sexual contacts with a bisexual male whose antibody status is unknown. Case no. 33 gave a history of using drugs by injection.

Although there was a wide distribution of recipient ages in all four groups, of the four recipients aged 10 or less, one had AIDS, and three had ARC. Of the five recipients aged 80 or older, two had AIDS, and two had ARC. There was no clear effect of the sex of the recipient on susceptibility to infection or disease. Although 13 of the 31 infected individuals were female and only four of 20 noninfected individuals were female, the differences are not statistically significant. The most common indications for transfusion were cardiac surgery (20 cases), orthopedic surgery, (7 cases) and gastrointestinal bleeding (7 cases), all conditions that tend to require large amounts of blood. There was no obvious correlation between the condition prompting transfusion and the occurrence of infection or AIDS in the recipient, although the numbers are too small for meaningful statistical analysis. Diseases commonly associated with immunologic depression were not encountered among the living patients we investigated. It should be noted that they would not have been reported to us as AIDS cases because the CDC definition excluded them.

Among those patients who were alive when first seen, the death rate was highest in those with AIDS, was much lower in those with ARC and with antibody only, and was zero in those without antibody. These statements are based on the patient status when we first attempted to contact them. The numbers of RBC, platelet, and fresh frozen plasma components were all significantly greater for recipients who developed AIDS, but not if patient no. 3 is omitted. Patient no. 3 received many platelet transfusions and repeated therapeutic plasmapheresis for idiopathic thrombocytopenic purpura. HIV infection was not suspected in this case before the transfusions. There were no differences in the numbers of components transfused between those patients with ARC, those with antibody only, and those without detectable

Table 1. Recipients of Blood Components From Donors With Aids

Case No.	Age	Sex	Diagnosis	Status	AIDS	Antibody to HIV			Transfused			Component From AIDS Donor	WBC	Lymph	H/S	PHA	PWM	MLC	Beta 2-M	HBsAg	Anti-HBc	Anti-HBs	Anti-CMV
						EIA	IFA	IB	RBC	PLT	FFP												
1	1	M	Hem Dis NB	Dead	AIDS			0	22	8	0	PLT	7.7						5.9				0
2	80	M	Cardiac Surg	Dead	AIDS	+	+	+	5	0	2	RBC	4.1	0.4	0.3	10,473			16.5	0	0	0	+
3	52	M	Thromb Purp	Alive	AIDS	+	+	+	31	128	362	PLT	3.8		0.1	1,253	560		5.1	0	0	0	0
4	56	F	Cardiac Surg	Alive	AIDS	+	+	+	17	18	12	PLT	7.7	3.3	0.4	3,380	3,800		10.0	0	0	0	+
5	81	F	Orthop Surg	Dead	AIDS	+	0	0	2	0	0	RBC			0.1	34,095	4,588		6.6	0	0	+	+
6	23	M	Trauma	Dead	AIDS		+		2	0	0	RBC			0.5								
7	50	F	Orthop Surg	Dead	AIDS	+	+	+	13	21	2	PLT	1.8	0.3		16,600		2,270	4.9				
8	10	M	Orthop Surg	Alive	ARC	+	+	+	10	7	0	PLT	6.4	2.6	0.5	13,290		7,040	1.8	0	0	0	+
9	1	F	Plastic Surg	Alive	ARC	+	+	+	1	0	0	RBC	5.7	4.1	1.7	28,890			2.1	0	0	0	+
10	89	F	CA kidney	Dead	ARC	+	+	+	7	0	0	RBC	4.8	1.5	0.3	87	0		15.0	0	0	0	+
11	87	F	GI bleed	Dead	ARC	+	+	+	7	0	0	RBC	3.6	1.0			4,477		13.5	+	0	0	+
12	68	M	Cardiac Surg	Alive	ARC	+	+	+	7	8	4	FFP	3.8	0.8	0.1	295	240		2.5	0	0	0	0
13	4	M	Resp distress	Alive	ARC	+	+	+	12	0	1	FFP			0.9	951	1,398		4.8	0	0	0	0
15	67	F	Orthop Surg	Alive	ARC	+	+	+	2	0	0	RBC			0.4				4.7	0	0	0	+
16	29	F	Eclampsia	Alive	Ab+	+	+	+	19	9	0	PLT				31,262	20,482		3.0	0	0	0	+
17	40	F	Miscarriage	Alive	Ab+	+	+	+	2	0	0	RBC	6.1	1.5	0.7	18,748	15,829		2.1	0	+	0	+
18	80	F	Orthop Surg	Alive	Ab+	+	+	+	3	0	0	RBC			1.6	44,590		21,789	2.4				
19	42	F	Gyn Surg	Alive	Ab+	+	+	+	1	0	0	RBC			0.7				3.6	0	0	0	+
20	51	M	Cardiac Surg	Alive	Ab+	+	+	+	9	10	6	PLT	4.3	1.6	0.7	8,374	447		3.0	0	0	0	+
21	50	M	GI bleed	Dead	Ab+	+	+	+	19	0	0	RBC	3.6	1.4		25,010		7,860	5.3				
22	61	M	Cardiac surg	Alive	Ab+	+	+	+	12	20	6	RBC			0.6	758	3,611		5.3	0	+	0	+
23	42	M	Cardiac surg	Alive	Ab+	+	+	+	10	10	3	RBC			0.7	7,529	3,518		2.8	0	0	0	0
24	61	M	GI bleed	Alive	Ab+	+	?	+	3	0	0	RBC	6.7	1.4	0.8				5.5	0	0	0	+
25	46	F	GI bleed	Alive	Ab+	+	+	+	8	10	5	PLT	5.7	1.9	0.3	2,014	375		4.8	0	0	0	+
26	38	M	Gen Surg	Alive	Ab+	+	+	+	2	0	0	RBC	7.7	2.5	0.5	2,141	285		3.6	0	0	0	+
27	62	M	Cardiac Surg	Alive	Ab+	+	+	+	9	0	0	RBC	3.0	1.0	0.7	9,655	15,009		2.2	0	0	0	+

28	72	F	Arthritis	Alive	Ab+	+	+	+	2	0	0	RBC			2.5	13,449	13,386		5.7	0	0	0	+
29	77	M	Cardiac Surg	Dead	Ab+	+	+	+	8	0	0	RBC	4.6	2.8					5.6	0	0	0	+
30	27	M	GI bleed	Alive	Ab+	+	+	+	10	0	0	RBC	4.8	1.6	0.7	6,805	885		3.8	0	+	+	+
31	60	M	Cardiac Surg	Alive	Ab+	+	+	+	8	0	0	RBC	5.5	1.9	0.4	11,130		10,800	2.9				
32	59	M	Cardiac Surg	Alive	Ab+	+	+	+	4	10	2	RBC			0.2	1,088	312		3.0				
34	70	F	Gen Surg	Alive	Neg	0	0	0	14	5	1	PLT			1.3	10,690		9,490	6.9				
35	50	M	Cardiac Surg	Alive	Neg	0	0	0	3	0	0	RBC	7.6	1.7	2.0	32,400		1,665	2.2	0	0	0	+
36	29	M	Polycyst Kid	Alive	Neg	+	0	0	5	0	0	RBC	3.9	1.1	0.1	144	149		6.1	0	0	0	+
37	65	F	Vasc Surg	Alive	Neg	0	0	0	10	10	0	R/P			0.5	21,611	4,343		2.5	0	0	0	+
38	52	M	Cardiac Surg	Alive	Neg	0	0	0	6	0	1	RBC	4.3	1.5	1.1		3,685		1.2	0	0	0	+
39	66	F	Orthop Surg	Alive	Neg	0	0	0	3	0	0	RBC	3.6	1.4	2.8		2,379		4.0	0	0	0	+
40	70	M	Vasc Surg	Alive	Neg	0	0	0	25	6	4	PLT	7.4	1.5	1.4				5.5	0	+	+	+
41	71	M	Cardiac Surg	Alive	Neg	0	0	0	3	0	0	RBC			1.6				1.5	0	+	0	+
42	60	M	Cardiac Surg	Alive	Neg	0	0	0	7	10	4	RBC	8.1	2.4	2.0	32,930		2,440	2.8				
43	76	F	Orthop Surg	Alive	Neg	0	0	0	2	0	0	RBC			0.9	16,846	7,622		9.2	0	0	+	+
44	74	M	Cardiac Surg	Alive	Neg	0	0	0	12	20	5	PLT	6.6	1.9	1.0	11,618	7,890		2.3	0	0	0	+
45	70	M	Cardiac Surg	Alive	Neg	0	0	0	10	10	8	FFP	8.8	2.9	1.6				1.4	0	+	+	+
46	27	M	Fracture Fem	Alive	Neg	0	0	0	14	0	0	RBC	5.6	2.3	0.5	6,006	2,204		1.3	0	0	0	+
47	70	M	Cardiac Surg	Alive	Neg	0	0	0	10	10	4	PLT	4.9	1.0	2.4	28,900		23,400	2.0				
48	72	M	Cardiac Surg	Alive	Neg	0	0	0	8	0	0	RBC	8.1	2.0	1.7	22,142	29,358		3.4	0	0	0	+
49	68	M	Trauma	Alive	Neg	0	0	0	8	0	0	RBC	8.6	2.3	3.2	11,132	14,179		1.5	0	0	0	0
50	40	M	Cardiac Surg	Alive	Neg	0	0	0	6	5	0	PLT	4.5	2.6	2.1	21,500		15,360	1.0				
51	50	M	GI surgery	Alive	Neg	0	0	0	6	0	0	RBC	6.7	2.4	1.3	25,725		2,414	1.2				
52	77	M	GI bleed	Alive	Neg	0	0	0	3	0	0	RBC	6.7	2.5	0.8		1,217		2.9	0	0	0	+
53	64	M	GI bleed	Alive	Neg	0	0	0	13	0	2	FFP	6.9	3.1	2.1				2.4	0	0	0	+

Abbreviations: RBC, red blood cells including whole blood; PLT, platelets; FFP, fresh frozen plasma; R/P, RBC and platelets; WBC, white blood cell count per microliter × 1,000 (4.0 to 10.0)*; Lymph, lymphocyte count per microliter × 1,000 (1.5 to 4.0)*; H/S, helper/suppressor ratio (>1.0)*; PHA, phytohemagglutinin stimulation (>11,500 cpm)*; PWM, pokeweed mitogen stimulation (>3,000 cpm)*; MLC, mixed lymphocyte culture (>5,000 cpm)*; Beta 2-M, β_2-microglobulin (<2.6 mg/dL)*; Hem Dis NB, hemolytic disease of the newborn; Thromb Purp, thrombocytopenic purpura; Surg, surgery; Orthop, orthopedic; CA, cancer; GI, gastrointestinal; Resp, respiratory; Vasc, vascular; Fem, femoral bone.

*Normal range.

antiviral antibody. All three types of components had been provided by the donors who later developed AIDS. RBC had been given to 21 infected recipients and 13 not infected; platelets to eight and seven, respectively; and fresh frozen plasma to two each.

Table 2 relates the recipient's chance of infection or disease to the time interval between the transfusion and the diagnosis of AIDS in the donor. In six of the seven cases where the recipient developed AIDS, the donor was recognized to have AIDS within 22 months. Infection of the recipient was apparent in 90% of the cases where the donor developed AIDS within a year, in 62% when the donor's AIDS was recognized between 1 and 4 years, in 20% of those with an interval between 4 and 5 years, and in neither of the two cases where the interval was over 5 years. Although it seems obvious that the longer the interval between transfusion and the diagnosis of AIDS in the donor, the less likely the donor was infected when the blood was taken, it should be noted that the differences in Table 2 lack statistical significance.

Laboratory data. Of the seven cases of AIDS, two had no detectable antibody by IB. Case no. 1 (our original case of transfusion-associated AIDS)[10] was tested by IB alone. Case no. 5 was positive in the Abbott EIA, negative by Genetic Systems EIA, and negative by IFA and IB and was therefore considered to be a false-positive by the Abbott method. All tests on the ARC cases were consistently positive. In case no. 36 three EIA tests were reactive (Abbott, ENI, and Genetic Systems), but both IFA and IB were nonreactive. Based on the latter two test results and the known propensity of EIA tests to result in false-positives, this result is assumed also to be an EIA false-positive.

A low WBC count was more common in the AIDS and ARC groups. The absolute lymphocyte count was progressively lower from the antibody-negative to the antibody-positive groups to those with ARC to those with AIDS, but it is noteworthy that three of the 15 measured in the antibody-negative group (20%) had levels below 1,500/μL. Helper/suppressor ratios were below 1.0 in all of the AIDS cases and in more than 80% of those with ARC or with anti-HIV antibody alone. They were also low in 25% of those who were negative for anti-HIV antibody. Stimulation indexes with PHA, PWM, and MLC did not correlate with the category into which the patient fell. β_2-Microglobulin levels were high in all of the AIDS cases, but they were abnormal in more recipients who had anti-HIV antibody alone than in those with ARC. Moreover, 40% of those negative for antiviral antibody had an elevated β_2-microglobulin level.

The laboratory tests were performed at a mean interval of 26 months after transfusion (range, 10 to 43) in the patients with AIDS, 38 months (range, 12 to 71) with ARC, 32 months (range, 9 to 60) with anti-HIV only, and 43 months (23 to 78) in the patients with no antibody to the virus detected.

Table 2. Effect of the Time Between Blood Donation and the Development of AIDS in the Donor on the Risk of Infection in the Transfused Recipient

Months	AIDS	ARC	Asymptomatic With Anti-HIV	No Anti-HIV
0-12	4	2	3	1
13-24	2	1	4	7
25-36	0	1	6	4
37-48	1	3	3	2
49-60	0	0	2	3
>60	0	0	0	2

DISCUSSION

Our observations on previous recipients of blood components from donors who later developed AIDS demonstrate that these recipients are at relatively high risk of developing AIDS themselves or of being infected by HIV. Of 336 recipients whose current status is known, seven (2%) have developed AIDS. Of 46 living recipients who do not have AIDS, 24 have antibody to HIV (52%), and seven of these meet the criteria for ARC.

The blood components transfused from the donors who developed AIDS included all routine components. This fact confirms the prevous CDC report that AIDS can be transmitted by whole blood, RBC and platelet concentrates, and fresh frozen plasma.[12] There is no evidence in this small series that any of these components is more likely to transmit AIDS than the others.

Infection occurred after transfusion of even a single unit (case nos. 9 and 19), but in general our patients received relatively large amounts of blood components. Recipients who developed AIDS received more components on the average than those with ARC, with anti-HIV only, or with no evidence for infection; but the difference is not significant if the atypical case 3 is omitted. Curran et al[12] have already shown that recipients with AIDS tended to receive large numbers of blood components and have reasoned that the more donors to whom a recipient is exposed, the greater the possibility of receiving an infected unit. This is undoubtedly true, but in contrast to the study of Curran et al where the recipients were selected because they had developed AIDS, the recipients in our study were selected because they had received a component from a donor who developed AIDS. Moreover, we have to explain why antibody-negative recipients had received an equally large number of components. The most likely explanation for our results is that a single specified component, no matter how selected, is more likely to have been given to a patient who received large numbers of components than to a recipient of one or two components. Whatever the explanation, the relatively large number of components given to these patients could act as a cofactor for the development of AIDS in view of the well-established immunosuppressive effects of blood transfusion.[13]

That two thirds of the recipients we identified were already dead when we first attempted to contact them can also be explained by our case selection method and should not be interpreted to be the typical fate of all transfusion recipients. Patients who receive large amounts of blood are more likely to have conditions that result in early demise. There was no evidence in the medical records that this excess

of early deaths was related to AIDS. Our assumption that they were an artifact of the method of case selection requires comparison with a control group of recipients selected by identifying a donor who was not infected with HIV. Such control studies are underway.

The absolute lymphocyte count and, to a lesser degree, the total WBC count were lowest in AIDS cases compared with those with ARC, were less often low in antibody-only cases, and were most often normal in the antibody-negative group. Helper-suppressor ratios were low in all AIDS cases, in 80% of antibody-positive cases (whether or not ARC was present), but also in 25% of antibody-negative cases. Obviously, in this group of subjects ill enough to require previous transfusions, a low helper-suppressor ratio is even less indicative of AIDS or HIV infection than it is in a healthier population. Functional lymphocyte stimulation tests showed a very poor correlation with the presence or absence of HIV infection or the stage of disease and would appear to be of no help in this type of patient who has other reasons for abnormal results in these procedures. β_2-Microglobulin, which has been suggested as a test that might indicate the likelihood of overt AIDS occurring,[14] also appears to be of little help in this situation because of the high frequency of abnormal results in patients with no evidence of infection by the AIDS virus, presumably a result of coexisting illnesses.

Hepatitis B tests were usually negative in these patients, in contrast to their high rate of positive results among the more common high-risk groups for AIDS. Homosexually active males and intravenous drug users are very likely to have been exposed to hepatitis B virus. Blood transfusion recipients are exposed far less often because all donor blood is tested for hepatitis B antigen, and positive units are discarded. In contrast to the results with hepatitis B tests, our blood recipents had a very high frequency of antibody to CMV (34/40, or 85%) compared with the 51% rate of positive results among our blood donors. This finding suggests that exposure to CMV is very likely to occur during blood transfusions, but the numbers are small, and the recipients were generally older than most blood donors. The frequency of anti-CMV increases with the age of the subject. It is noteworthy that the frequency of anti-CMV antibody did not correlate with the presence or extent of HIV infection in view of the common hypothesis that infection with CMV may be a cofactor that increases susceptibility to the AIDS virus.[15]

The most useful prognostic indicators for these recipients were the time interval from transfusion until the donor was diagnosed as having AIDS and the age of the recipient. Recipients below age 11 or over age 79 were much more likely to develop overt disease than those of intermediate ages. This latter finding may reflect the state of the immune system at these ages. The shorter the time interval between the transfusion and the onset of AIDS in the donor, the more likely the donor was to be infected already at the time the blood was given, and the more likely, therefore, that the recipient would have antibody as a sign of infection. Moreover, the occurrence of disease as well as infection appears to be more likely the shorter the interval (Table 2).

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