aware of the possibility of this injury, we believe that patients should be strongly advised to wear seat belts, properly fitted in compliance with the manufacturer's instructions. This case report in no way detracts from the value of the seat belt but tends to support the view that major injury will be minimised in this type of dangerous accident.

We present this case to indicate another variant of the seat belt injury syndrome and in accordance with the suggestion that such injuries should be recorded to assess the magnitude of the problem.

We thank Mr J H Balmer for permission to report this case and Miss Hilary Cromie for typing the manuscript.

ADDENDUM—Since this report was written this patient was seen again at another hospital with total avulsion of his ileostomy as the result of direct trauma during a brawl. The ileostomy was satisfactorily revised, and his postoperative course was uncomplicated. There was no history or evidence to suggest that either of these injuries were self-inflicted.

[1] *British Medical Journal*, 1973, **1**, 195.
[2] MacLeod, J H, and Nickolson, D M, *Canadian Journal of Surgery*, 1969, **12**, 202.
[3] Fish, J, and Wright, R H, *Journal of Trauma*, 1965, **5**, 746.
[4] Williams, J S, and Kirkpatrick, J R, *Journal of Trauma*, 1971, **11**, 207.

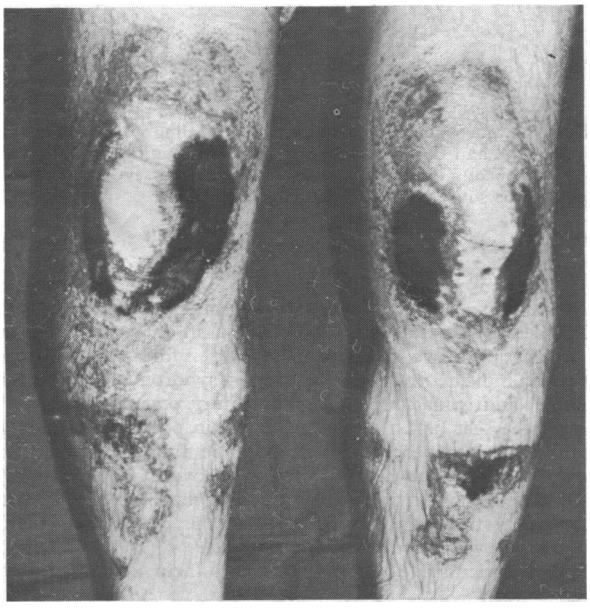
(*Accepted 20 January 1978*)

Craigavon Area Hospital, Craigavon, Co Armagh, N Ireland
A J WILKINSON, MB, DOBSTRCOG, senior house officer, general surgery
W G HUMPHREYS, MD, FRCS, senior registrar, general surgery

Burn hazard with cement

Warnings about the danger of exposure of the skin to prolonged contact with builder's cement are not normally given to workers in the construction industry, and are not displayed on the containers used by "do-it-yourself" enthusiasts. We have seen several cases of chemical burns caused by cement, and think that more attention should be brought to the risks in its use and to the need for proper diagnosis and treatment.

Most of the patients we see were not aware that prolonged skin contact with cement can produce burns. The usual story is of a building site worker who has allowed cement to spill over the top of or to leak through rents in his boots. Because there is no thermal burn the patient does not feel immediate pain and allows contact to persist. Burns of the hands are more common, but are normally mild and are often accepted as "allergy" to the cement and treated by doctors as "contact dermatitis," often with steroids. Here we describe an unusual presentation.



Case report

A 29-year-old man was seen in our unit two days after spending a period of about one hour on his knees in a shallow layer of wet Readymix concrete while laying a floor in his house. The burns were full thickness in patches around both knees and on both shins, with the areas of pressure spared (see figure). He noticed no pain at the time but was aware of slowly increasing burning and reddening of the skin over the 48 hours before seeking advice. After skin grafting the wounds had healed fully in four weeks. He was completely unaware that cement was corrosive.

Comment

An analysis[1] of typical Portland cement gives the following results: CaO 60-67%; SiO_2 17-25%; Al_2O_3 3-8%; Fe_2O_3 0·5-6%; MgO 0·4-4%; alkalis 0·4-1·3%; and SO_3 1-3%. Thus the corrosive agent, calcium oxide (quicklime), forms 65% of the total weight[2] of this amount; only a small proportion (1·5%) is present as free lime, and the rest is present in the form of compounds, mainly as tricalcium silicate. Hydration of cement is exothermic, but the rise of temperature is slow (about 10 C in five hours) and this is not responsible for any of the burns we have seen. When the concrete is hard, a typical sample analysed shows a content of 37% by weight of free calcium oxide.[3]

It would seem justifiable, therefore, to warn user and employer of the risks of exposure to cement. Adequate protection for the hands and feet, and the avoidance of prolonged skin contact, as well as clearly marked warnings on all containers in which cement is supplied, should be the minimum requirement. Much cement is used where the user does not see the container, and the general public needs to be made aware of the dangers.

Awareness of this hazard on the part of medical personnel would ensure correct diagnosis, rapid treatment, and proper supervision.

[1] Neville, A M, *Properties of Concrete*. London, Pitman, 1975.
[2] Rugby Portland Cement Co Ltd, personal communication.
[3] Messrs Sanberg (Testing Engineers), personal communication.

(*Accepted 20 January 1978*)

The General Infirmary at Leeds, Leeds LS1 3EX
M W FLOWERS, FRCSED, consultant surgeon, accident and emergency department

HLA phenotypes in long-term survivors treated with BCG immunotherapy for childhood ALL

In recent years evidence has been produced to show the association of numerous diseases with individual HLA antigens.[1] Theoretically, acute lymphoblastic leukaemia (ALL) was initially supposed to be likely to show such an association, since in mice the susceptibility to virus-induced leukaemia is genetically linked with the major histocompatibility complex (MHC).[2] Nevertheless, Kourilsky *et al* were unsuccessful in their search for an association between ALL and any of 10 HLA antigens tested.[3] Since this pioneer work many reports have shown conflicting results. If susceptibility to ALL is not clearly associated with HLA antigens, another method of study would be to look for differences in HLA phenotypes between long-term and short-term survivors. We report here the HLA phenotypes of children with ALL who are surviving without relapse for over six years and being treated with immunotherapy.

Patients, methods, and results

A total of 13 patients were studied, all Caucasians under 20 at diagnosis. Remission induction and maintenance chemotherapy were given from six to 15 months* and then stopped. At this time immunotherapy was started with BCG (Institut Pasteur) applied weekly on scarifications, together with injections of allogeneic irradiated lymphoblasts. This immunotherapy lasted from four to 10 years. No relapse has been observed after three years of

*Except for patients No 6 and 8, who received chemotherapy for 30 months.

lasting remission with immunotherapy. All patients are alive without any relapse six to 14 years after beginning immunotherapy.

HLA phenotypes were determined by microlymphocytotoxicity assay in microtest plates. Selected monospecific or bispecific antisera enabled us to test for 31 HLA antigens. Frozen rabbit serum was used as the source of complement. Antigen frequencies of these patients were compared with those of 591 healthy individuals and of 14 ALL long-term survivors from the initial study by Kourilsky *et al*, which had used chemotherapy exclusively.

The HLA phenotypes are listed in the table. When antigen frequencies among patients treated with BCG immunotherapy were compared with those obtained in normal individuals, a significant increase (Fisher's exact test and χ^2 comparison with Yates's correction) in antigens HLA-BW17 (46·1% *v* 7·3%) and HLA-AW33 (30·8% *v* 1·2%) was observed ($P < 0.001$). These increases remained statistically significant when corrected for the number of specificities tested. Nine patients had at least one of these two antigens (69·2% *v* 8% in controls: $P < 0.001$) but only one patient (No 5) had both antigens. No patient possessed HLA-B5 antigen (*v* 13·2% in controls). Deviations in other HLA antigen frequencies were not apparent.

Long-term survivors treated with chemotherapy alone showed no alteration in HLA-BW17, AW33, or B5 frequencies (0/14 BW17 in the "chemotherapy" group *v* 6/13 in the "immunotherapy" group. χ^2 with Yates's correction: 7·63 ($P < 0.01$)).

Discussion

HLA antigens HLA-BW17 and HLA-AW33 were significantly increased in 13 patients with ALL who were surviving in their first remission for over six years after immunotherapy with BCG. Previous reports have not shown that the frequencies of these antigens were different in patients with ALL, the only alteration in some series being a high incidence of HLA-A2 (or HLA-A2-B12 haplotype), which was not apparent in our patients. Some have suggested that the HLA phenotype may interfere with the clinical course of the disease and prognosis. Nevertheless, in patients treated only with chemotherapy, we found no statistically significant difference between short-term and long-term survivors—though in some prospective series, a better survival time has been found to be associated with HLA-A2[4] and HLA-A9.[5] These latter two reports dealt with patients treated by chemotherapy exclusively. In our group of BCG-treated patients, HLA-A2 and B9 were slightly (but not significantly) decreased.

Our patients differ from other series of those with ALL reported in only one obvious point: their treatment. We suggest that the observed difference in HLA phenotypes is in some way related to the fact that these patients were good responders to BCG immunotherapy. Thus our results suggest the existence in man of HLA-linked genes which are concerned with the response to immunotherapy in ALL. Whether these genes govern a specific immune response to some leukaemia antigen, or a non-specific response to BCG immunotherapy cannot be assessed.

Beside its theoretical interest, the finding that a good response to BCG immunotherapy in ALL is at least in part genetically determined and HLA linked could possibly lead to the use of HLA phenotyping to choose those patients with a good chance of cure with immunotherapy (as BW17 and AW33?), or conversely of patients with a "poor chance" (as B5?) who require long-term chemotherapy without immunotherapy. A prospective study is needed to obtain a clear definition of these groups of good and bad responders to BCG immunotherapy. Moreover, similar studies should be done in patients treated by immunotherapy for malignancies other than ALL.

We thank Professor J Dausset for helpful discussions and advice, and Mrs G Boudard, M Y Guillard, M Duteil, and Y Thies for technical help. This work was supported by grants from Institut Gustave-Roussy and Inserm-France.

[1] Dausset, J, and Hors, J, *Transplantation Review*, 1975, **22**, 44.
[2] Lilly, F, *Transplantation Proceedings*, 1971, **3**, 1239.
[3] Kourilsky, F M, *et al*, *Journal of the National Cancer Institute*, 1968, **41**, 81.
[4] Rogentine, G N, *et al*, *Tissue Antigens*, 1973, **3**, 470.
[5] Lawler, S D, *et al*, *British Medical Journal*, 1974, **1**, 547.

(*Accepted 26 January 1978*)

Service de Médecine A et Laboratoire d'Immuno-Hématologie, Institut Gustave-Roussy, 94800, Villejuif, France, and Laboratoire de Génétique de la Transplantation Humaine (INSERM U 93), Institut de Recherche sur les Maladies du Sang, Hôpital Saint-Louis, 75010, Paris, France

THOMAS TURSZ, MD, assistant in oncology and immunology
JACQUES HORS, MD, associate professor of immunology
MARC LIPINSKI, MD, research fellow
JEAN-LOUIS AAMIEL, MD, professor of oncology and chief of department of medicine

HLA phenotypes of long-term survivors with ALL

Case No	Sex	Age at diagnosis (years)	Date of diagnosis	Length of chemotherapy	Length of immunotherapy (years)	HLA phenotype	
						A locus	B locus
				Patients treated with immunotherapy			
				(months)			
1	F	4	1966	8	4	1	14, *W17*
2	M	7	1971	8	5	3, *W33*	7, W27
3	M	2½	1969	10	6 y 6 m	1, 11	*W17*, W18
4	F	19	1971	6	5	2, *W33*	7, W15
5	F	10	1968	8	7	9, *W33*	8, *W17*
6	F	6	1963	30	10	3	7
7	F	12	1971	6	5	2, W32	*W17*, W40
8	M	18	1963	30	10	2, 3	8, *W17*
9	F	8	1971	6	5	2, *W33*	12
10	M	5½	1966	8	5 y 6 m	9, 28	12, 14
11	F	4	1967	8	5	1, 29	12, *W17*
12	F	13	1962	15	9 y 2 m	2, 28	W32, W35
13	M	3	1968	12	8	2, W23	12
				Patients treated exclusively with chemotherapy			
				(years)			
14	M	4	1966	6	0	2, 3	7, W38
15	F	4½	1965	7	0	11, 29	W35
16	F	6	1968	5	0	2, 11	7
17	M	16	1966	5½	0	2, 11	5
18	M	15	1966	5	0	1, 9	13, W40
19	F	5¼	1965	6	0	1, 2	8, 12
20	F	4¼	1962	11	0	2	12, W41
21	F	4	1966	¾	0	2, 19–2	5, W21
22	M	2½	1967	5	0	1, 19–2	7, W40
23	M	3	1964	7	0	9, 28	12, W35
24	F	2⅓	1968	7	0	1, 9	8, W15
25	F	4¼	1968	5	0	3, W32	8, W35
26	F	17	1966	5	0	2, W28	7, 12
27	M	11½	1966	4	0	1, 2	W37, W40

Spurious polycythaemia developing during observation

Whether spurious polycythaemia, defined as an increased packed cell volume with a normal red cell mass,[1 2] is a disease or a variant of normal is not clear. Recent studies have suggested two distinct forms[2 3]—one characterised by a reduced plasma volume, normal red cell mass, hypertension, vascular disease, and increased mortality; the other by a high-normal red cell mass, low-normal plasma volume, no specific clinical features, and a normal prognosis. The second form was thought to represent the extreme of the normal range.[2 3] I describe