

the Brighton Rotary Club, from many other local organisations, from patients, and from their relatives and friends.

## References

- <sup>1</sup> Pantridge, J F, and Geddes, J S, *Lancet*, 1967, **2**, 271.
- <sup>2</sup> Adgey, A A J, *et al*, *Lancet*, 1971, **2**, 501.
- <sup>3</sup> Pantridge, J F, *Quarterly Journal of Medicine*, 1970, **39**, 621.
- <sup>4</sup> Cobb, L A, *et al*, *Circulation*, 1975, **52**, suppl No 3, p 223.
- <sup>5</sup> Rose, L B, and Press, E, *Journal of the American Medical Association*, 1972, **219**, 63.
- <sup>6</sup> Nagel, E L, *et al*, *Journal of the American Medical Association*, 1970, **214**, 332.
- <sup>7</sup> Crampton, R S, *et al*, *American Journal of Medicine*, 1975, **58**, 151.
- <sup>8</sup> Lewis, R P, and Warren, J V, *American Journal of Cardiology*, 1974, **33**, 152.
- <sup>9</sup> Working Party of the Royal College of Physicians of London and the British Cardiac Society, *Journal of the Royal College of Physicians of London*, 1975, **10**, 5.
- <sup>10</sup> White, N M, *et al*, *British Medical Journal*, 1973, **3**, 618.
- <sup>11</sup> Sloman, G, *Medical Journal of Australia*, 1975, **1**, 612.
- <sup>12</sup> Gearty, G F, *British Medical Journal*, 1971, **3**, 33.
- <sup>13</sup> Pozen, M W, *et al*, paper presented at 47th Scientific Sessions, American Heart Association Meeting, Dallas, 1974.
- <sup>14</sup> Barber, J M, *et al*, *Lancet*, 1970, **2**, 133.
- <sup>15</sup> Sandler, G, and Pistevo, A, *British Heart Journal*, 1972, **34**, 1283.
- <sup>16</sup> Registrar General's Statistical Review of England and Wales. London, HMSO, 1971.
- <sup>17</sup> Spiekeman, R E, *et al*, *Circulation*, 1962, **25**, 57.
- <sup>18</sup> Mather, H G, *et al*, *British Medical Journal*, 1976, **1**, 925.
- <sup>19</sup> *British Medical Journal*, 1975, **2**, 5.
- <sup>20</sup> McNeilly, R H, and Pemberton, J, *British Medical Journal*, 1968, **3**, 139.
- <sup>21</sup> Nixon, P G F, in *Acute Myocardial Infarction*, ed D G Julian and M F Oliver, p 318. Edinburgh, Livingstone, 1968.
- <sup>22</sup> Pantridge, J F, in *Lidocaine in the Treatment of Ventricular Arrhythmias*, ed D B Scott and D G Julian. Edinburgh, Livingstone, 1971.
- <sup>23</sup> Chopra, M P, *et al*, *British Medical Journal*, 1971, **3**, 668.
- <sup>24</sup> Hampton, J R, *British Medical Journal*, 1976, **1**, 201.
- <sup>25</sup> Chaturvedi, N C, *et al*, *British Heart Journal*, 1974, **36**, 533.
- <sup>26</sup> Green, K G, *et al*, *British Medical Journal*, 1975, **3**, 735.

# Association of HLA-A9 and HLA-B5 with Buerger's disease

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## Summary

**Eighteen patients who satisfied stringent criteria for the diagnosis of Buerger's disease, healthy controls, and patients with atherosclerosis were tested for various HLA antigens. The incidence of HLA-A9 and HLA-B5 was significantly greater among those with Buerger's disease. This finding supports the concept that Buerger's disease is a distinct clinicopathological condition.**

## Introduction

Since Buerger originally described the clinicopathological disorder that bears his name,<sup>1</sup> considerable doubt has been cast on its existence as a disease separate from early-onset atherosclerosis of a peripheral distribution.<sup>2-3</sup> Support for its existence as a separate disease has been given, however, by clinical, epidemiological, and arteriographic studies.<sup>4-8</sup> As the disease is more common in certain ethnic groups<sup>9,10</sup> and within families,<sup>11-14</sup> we investigated the histocompatibility types of all patients reported to us as having Buerger's disease on Merseyside.

## Patients and methods

In response to a letter written to consultants with an interest in peripheral vascular disease 28 patients with Buerger's disease were

referred for study. Their case records and arteriograms were reviewed independently. The clinical criteria of Mozes *et al*<sup>8</sup> were applied. These require that in addition to ischaemic symptoms in the leg, the patient must show at least two "systemic manifestations"—migrating phlebitis, Raynaud-like phenomena in the hands or feet, or involvement of the hands. The radiographic criteria of McKusick *et al*<sup>4</sup> were used to evaluate the arteriograms. Routine investigations included full blood count, measurement of erythrocyte sedimentation rate, urea and electrolyte estimation, liver function tests, Rose-Waaler test, antinuclear antibody test, and cryoglobulin and cryofibrinogen estimation. Tissue typing was performed according to the method of Dausset<sup>15</sup> using fresh lymphocytes and 26 standard antisera (National Tissue Typing Reference Laboratory, Bristol). To compare HLA frequencies 616 healthy blood donors and 91 patients with atherosclerotic disease of the leg attending the same vascular clinic were

*HLA types in patients with Buerger's disease, patients with atherosclerotic disease, and controls*

HLA antigens	No of patients with Buerger's disease (n=18)	No of patients with atherosclerotic disease (n=91)	No of controls (n=616)
<i>1st segregant series</i>			
A1	5	26	195
A2	7	36	266
A3	3	20	148
A9	9*	11	83
A10	1	7	40
A11	2	7	56
A28	1	5	46
AW29	0	0	NT
AW32	0	1	NT
19 Cap	0	0	NT
<i>2nd segregant series</i>			
B5	15†	3	37
B7	3	20	151
B8	4	22	157
B12	0‡	27	185
B13	1	3	18
B14	1	4	46
B17	1	6	46
B27	1	4	40
BW5	1	8	49
BW10	1	6	56
BW15	0	5	40
BW16	0	0	NT
BW18	0	0	NT
BW21	0	0	NT
BW22	0	1	NT
TY	0	0	NT

\* $\chi^2 = 15.98$ ;  $P < 0.001$ . † $\chi^2 = 128.81$ ,  $P < 0.001$ . ‡ $\chi^2 = 6.25$ ;  $P < 0.09$ . NT = Not tested.

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studied. Findings in the patients with Buerger's disease and in the control groups were compared with Fisher's exact test, correcting the level of significance for the total number of antigens considered.

## Results

Ten of the 28 patients referred with presumed Buerger's disease were excluded because they did not fulfill the necessary diagnostic criteria. Of the remaining 18 patients 14 had confirmatory findings on arteriography. All were Caucasian unrelated men, and the mean age of onset of the disease was  $33.2 \pm 10.8$  years. All were cigarette smokers, smoking on average  $36 \pm 10$  cigarettes a day. The distribution of histocompatibility antigens in this group and in the two control groups is shown in the table. The incidence of HLA-A9 and HLA-B5 was much greater among those with Buerger's disease than in the controls ( $P < 0.001$ ). HLA-A9 and HLA-B5 occurred together in seven (39%) of the 18 patients with Buerger's disease but in only nine (1.5%) of the 616 healthy controls.

## Discussion

Several diseases have now been reported to show HLA associations.<sup>16</sup> Ohtawa *et al*<sup>17</sup> first noted an increase in the incidence of HLA-A9, BW10, and a Japanese specific antigen (J-1-1) together with an absence of HLA-B12 in Japanese patients thought to be suffering from Buerger's disease. HLA-B5 was found in 43% of their patients with the disease, but the difference between this and the 30% incidence found in their relatively small control group was not significant. In contrast, HLA-B5 in the Merseyside control population was rare (6%). We also found that HLA-B12 was absent from our patients compared with a 30% incidence among controls.

We have shown a significantly increased incidence of HLA-A9 and HLA-B5 antigens in patients who fulfilled strict clinical and arteriographic criteria for the diagnosis of Buerger's disease.

This finding supports the concept that Buerger's disease is a distinct clinicopathological condition separate from early-onset atherosclerosis. While the aetiology of Buerger's disease remains obscure, the published reports indicate that patients are inveterate cigarette smokers.<sup>18</sup> As the relative risk of patients with HLA-B5 antigen developing the disease is 78:2, one might speculate the exposure of this phenotype to tobacco is the precipitating factor in the pathogenesis of this disorder.

We thank the surgeons and physicians of the Merseyside region who referred their patients to be studied, Professor R Shields for his advice and constructive criticism, and the National Tissue Typing Reference Laboratory, Bristol, for supplying the antisera.

## References

- Buerger, L, *American Journal of Medical Science*, 1908, **136**, 567.
- Wessler, S, *et al*, *New England Journal of Medicine*, 1960, **262**, 1149.
- Fisher, C V, *Medicine*, 1957, **36**, 196.
- McKusick, V A, *et al*, *Bulletin of the Johns Hopkins Hospital*, 1961, **109**, 241.
- Ishikawa, K, *et al*, *Angiology*, 1962, **13**, 398.
- Szilagyi, D C, *et al*, *Archives of Surgery*, 1964, **88**, 824.
- Inada, K, *et al*, *Archives of Surgery*, 1964, **88**, 454.
- Mozes, M, *et al*, *Journal of Cardiovascular Surgery*, 1970, **11**, 52.
- Goodman, R M, *et al*, *American Journal of Medicine*, 1965, **39**, 601.
- Kjeldsen, K, and Mozes, M, *Acta Chirurgica Scandinavica*, 1969, **135**, 495.
- Samuels, S S, *American Journal of Medical Science*, 1932, **183**, 465.
- Martorell, F, *Angiology*, 1952, **3**, 271.
- Weber, F P, *Lancet*, 1937, **2**, 72.
- Biddlestone, W R, and Lefevre, F A, *Cleveland Clinic Quarterly Journal*, 1954, **21**, 226.
- Dausset, J, in *Manual of Tissue Typing Techniques*, ed J G Ray, D B Hare, and D C Kayhoe, p 28. Bethesda, Maryland, Department of Health, Education and Welfare, 1973.
- Dausset, J, *et al*, *Clinical Immunology and Immunopathology*, 1974, **3**, 127.
- Ohtawa, T, *et al*, *Journal of the American Medical Association*, 1974, **230**, 1128.
- Larson, P S, *et al*, *Tobacco. Experimental and Clinical Studies*. Baltimore, Williams and Wilkins, 1961.

# Preoperative skin preparation: clinical evaluation of depilatory cream

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## Summary

**Preoperative hair removal by a depilatory cream was compared with routine shaving. Although the incidence of wound infection was similar in both groups, cream depilation was found to be better. It was effective, atraumatic, non-toxic, and could be self-administered. Furthermore, it could be used safely on granulating**

**wounds and did not support bacterial growth. Depilation was associated with a significant reduction in skin surface bacteria and proved to be cheaper than shaving.**

## Introduction

Removing hair from the operative site is a time-honoured preoperative exercise. But if shaving is performed more than a few hours before operation the minor abrasions produced provide a good culture medium, with the subsequent risk of wound infection.<sup>1-3</sup> It has therefore been suggested that immediate preoperative shaving—that is, after anaesthesia has been induced—should be performed. Although this may be surgically desirable it is obviously not possible in busy surgical units. An alternative to shaving is the use of a depilatory agent. Such substances are widely available for cosmetic use but are not commonly used in surgical practice.

We evaluated one preparation (Ipsso) and compared it with conventional shaving. After establishing that the depilatory cream was non-irritant and did not support bacterial growth,

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