injury. As an ambulance carries only one scoop stretcher, another has to be obtained. In a busy accident department a second ambulance may often be about from which a scoop stretcher could be borrowed. We, however, believe that a second stretcher should be standard equipment in an accident and emergency department. It could then also be used to move the severely injured from trolley to x-ray table or bed. This is much easier and safer, as well as more comfortable for the casualty, than the traditional straight-lift or poles-and-canvas method.

This stretcher has proved popular with the ambulance service and with our general-practitioner accident unit on many occasions. We have found its versatility of great value, even on wet and uneven ground.¹ We have successfully used it to remove a casualty with spinal injuries lying face down at the bottom of a narrow, 2-m-deep trench and on many occasions at the road-side and on building sites in all weathers.

We thank Mr R Drucker and his colleagues, of the London Ambulance Service, for their help and co-operation, and F W Equipment and Co, Bradford, for the loan of the stretchers.

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¹ Robertson, B, *Journal of the Royal Army Medical Corps*, 1976, **122**, 21.

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Chromosome 1 heteromorphism in patients with malignant disease: a constitutional marker for a high-risk group?

Polymorphism for the heterochromatic regions of the No 1 chromosomes is common and has no apparent phenotypic effect.^{1,2} My attention was drawn, however, to a possible association with malignancy by the observation that the normal No 1 chromosomes in the aneuploid tumour cells of two ovarian carcinomas and a seminoma were heteromorphic for their C-band-positive regions. I could not ascertain whether the heteromorphism was present in the patients' normal cells but decided to investigate the incidence of heteromorphism in a series of patients with malignant disease at these and other sites.

Methods and results

Normal cells from 58 patients with malignant disease were studied, either in blood cultures (47 cases) or in direct preparations or cultures of lymphoid or tumour tissue. Seventeen patients had received chemotherapy or radiotherapy. Peripheral blood from 12 normal people and from 36 patients sampled at random from among those referred to the haematology laboratory for blood tests was studied. Cultured lymph nodes from five patients, spleen from one patient, and a fibroblast culture from a normal fetus were also studied. All these control patients had non-malignant conditions. Chromosome preparations made by standard procedures were stained by a C-banding technique.³ Heteromorphism was considered to be present when the areas of the heterochromatic regions of the No 1 chromosomes differed on average by at least 25% (measurements were made on photographs at a final magnification of $\times 4500$; in most cases, however, the presence or absence of heteromorphism could easily be assessed by direct microscopic observation). All assessments and measurements were made "blind."

Thirty-two of the 58 patients with malignant disease showed the heteromorphism compared with 17 of the 55 controls (see table). This difference was statistically significant ($\chi^2 = 6.74$ with Yates's correction; $P < 0.01$). Nine of the 32 positive patients had received treatment. No difference was discovered between the "malignant" and control groups in factors that might

Incidence of heteromorphism in 58 patients with malignant disease and 55 controls

	Heteromorphism present	Heteromorphism absent
<i>Patients with malignant disease</i>		
Tumour site or type:		
Ovary	12	13
Testis	4	3
Hodgkin's disease	4	4
Other lymphomas	6	2
Various carcinomas	5	4
Melanoma	1	0
Total	32 (15 men)	26 (7 men)
Mean ratio of areas of greater:lesser heterochromatic regions	1.50 (1.27-2.01)*	
Mean age (years)	51.5	51.1
<i>Controls</i>		
Healthy people:		
Men	1	3
Women	3	6**
Patients with non-malignant conditions:		
Men	6	8
Women	7	21
Total	17	38
Mean ratio of areas of greater:lesser heterochromatic regions	1.50 (1.30-1.63)†	
Mean age (years)	44.5	43.2‡

*Based on 207 metaphases. †Based on 61 metaphases. **Fetus included. ‡Fetus not included.

have caused an artefactual difference in C-band areas—for example, a difference in the lengths of the two No 1 chromosomes due to unequal spreading on the slide.

Comment

It seems reasonable to assume that there is a relation between the amounts of heterochromatin on the No 1 chromosomes and susceptibility to at least some forms of malignant disease. A common heritable chromosomal variant—a marker whose presence can be determined by a simple technique—can serve as a guide to the risk of cancer. (Although usually traceable to one or other parent, variant chromosomes may sometimes arise de novo during a lifetime, as suggested by the occurrence of mosaicism for the variant⁴; however, mosaicism was clearly present in only one normal man in this present study.)

The nature of the association with cancer is unclear. The heteromorphism might be related to a general instability of the chromosomes or more directly to the No 1 chromosomes' susceptibility to undergo structural changes. Such changes are common in the aneuploid tumour cells of ovarian carcinomas and lymphomas, at least (N B Atkin and M C Baker, unpublished data). It is interesting that the frequency of the association of the heterochromatic regions of the No 1 chromosomes with acrocentric chromosomes is positively correlated with the extent of the regions on the No 1 chromosomes.⁵

Studies are in progress to try to determine whether the association with malignancy is limited to variation affecting the No 1 chromosome or also encompasses the other chromosomes that are subject to similar variation, such as Nos 9 and 16.

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¹ Craig-Holmes, A P, Moore, F B, and Shaw, M W, *American Journal of Human Genetics*, 1973, **25**, 181.

² McKenzie, W H, and Lubs, H A, *Cytogenetics and Cell Genetics*, 1975, **14**, 97.

³ Sumner, A T, *Experimental Cell Research*, 1972, **75**, 304.

⁴ Craig-Holmes, A P, Moore, F B, and Shaw, M W, *American Journal of Human Genetics*, 1975, **27**, 178.

⁵ Schmid, M, Vogel, W, and Krone, W, *Cytogenetics and Cell Genetics*, 1975, **15**, 66.

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