cardial infarction.[1] Some evidence suggests that this is to be explained by smokers' leukocytosis,[2] though other studies have shown an effect independent of smoking status.[3] The mechanism is debated but leukocytes affect flow in the microcirculation, and influence platelet aggregation,[4] and Light[5] has suggested their proteinases might affect the development of atheroma.

To investigate the possibility that the leukocyte count predicts clinical events in the case of cerebrovascular disease, we have reviewed a group of patients with transient ischaemic attacks (TIAs) and related their leukocyte count at presentation to their subsequent course. The clinical outcome of some of these patients has been reported previously,[6 7] many being from the study of strokes in young people.[7]

The case records of 68 patients (56 males and 12 females of average age 48 years) presenting to one of us (JM) with a history of recent TIAs were reviewed. Patients with a recent completed stroke were excluded as a leukocytosis may be a response to a recent infarct or haemorrhage. Patients with polycythaemia rubra vera were also excluded. Note was taken of conventional risk factors, age, sex, blood pressure and smoking status. The white blood cell count recorded at their first clinic or hospital visit was extracted from the notes. The presence or absence of subsequent TIAs or strokes during a follow up period of an average of 5 years was also noted.

Twenty three patients had had further TIAs or strokes. The age, sex, blood pressure, smoking status, duration of follow up and leukocyte counts at presentation of those with and without further events is shown in the table. There was no difference in sex distribution, the prevalence of a blood pressure over 150/90 mm Hg, the proportion currently smoking or in the length of follow up achieved, between the two groups. Those with subsequent cerebrovascular events were slightly older (51·8 ± 6·9 years cf 46·0 ± 9·5 years, $t = 2{\cdot}5$, $p < 0{\cdot}02$), and had higher leukocyte counts (9·74 ± 1·1 × 10^9/l cf 7·774 ± 2·5 × 10^9/l, $t = 3{\cdot}25$, $p < 0{\cdot}002$).

Fifty three per cent of patients with leukocyte count over 8·0 × 10^9/l had had further events whilst only 12·5% of those with a count of 8·0 × 10^9/l or less had (Chi Square 10·5, $p < 0{\cdot}002$). Amongst the smokers there was still an apparent association between leukocyte count and risk. Thus the mean leukocyte count in smokers with subsequent events was 10·55 ± 1·9 × 10^9/l, that in smokers with no further TIAs or strokes was 8·05 ± 2·36 × 10^9/l ($t = 3{\cdot}72$, $p < 0{\cdot}001$). The chance of further events for a smoker with a leukocyte count over 8·0 × 10^9/l was 61% with a count of 8·0 × 10^9/l or less (n = 19), it was zero (Chi Square 15·5, $p < 0{\cdot}001$).

Relationship of risk factors including the leukocyte count to recurrence of TIAs or strokes

Risk factors	Patients with further events n = 23	Patients with no further events n = 45
Mean age (years)	51·8 ± 6·9	46·0 ± 9·5*
Sex	20 M 3 F	36 M 9 F
BP ⩾ 150/90 mm Hg	11 (48%)	19 (42%)
Smokers	17 (74%)	30 (67%)
Mean follow up (y)	5·2 ± 3·3	6·0 ± 4·7
Leukocyte count	9·74 ± 2·2	7·74 ± 2·5†

*$p < 0{\cdot}02$ Student's t test.
†$p < 0{\cdot}002$ Student's t test.

This small retrospective study of young patients with early evidence of cerebrovascular disease presenting with one or more TIAs suggests that the leukocyte count in the peripheral blood may be predictive of the risk of further cerebrovascular events. A similar trend was obvious in a study from Hiroshima though these were asymptomatic patients not at such a high risk,[8] as those with TIAs.

The data permit of no conclusion as to mechanism though the difference in apparent risk is clear within smokers as shown for myocardial infarction by Zalokar *et al.*[2] It is possible that the degree of smokers leukocytosis is a reflection in some way of the biological impact of smoking in the individual. Other risk factors may be involved since leukocyte counts are higher in women on an oral contraceptive.[9]

The evidence suggests that more investigation of the role of leukocytes in thrombosis,[4] on blood rheology,[10] and in atherogenesis is warranted.

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MJG HARRISON
JOHN MARSHALL
Institute of Neurology,
National Hospital for Nervous Diseases,
Queen Square,
London WC1N 3BG, UK

References

1 Friedman GD, Klatsky AL, Siegelaub AB. The leukocyte count as predictor of myocardial infarction. *N Engl J Med* 1974;**290**:1275–8.
2 Zalokar JB, Richard JL, Claude JR. Leukocyte count, smoking, and myocardial infarction. *N Engl J Med* 1981;**304**:465–8.
3 Grimm RM, Neaton JD, Ludwig W. Prognostic importance of white blood cell count for coronary, cancer, and all cause mortality. *JAMA* 1985;**254**:1932–7.
4 Harrison MJG, Emmons PR, Mitchell JR. The effect of white cells on platelet aggregation. *Thromb Diath Haem* 1966;**16**:105–21.
5 Light RT. The white blood cell count as a risk factor. *JAMA* 1986;**225**:1877.
6 Corston RN, Kendall BE, Marshall J. Prognosis in middle cerebral artery stenosis. *Stroke* 1984;**15**:237–41.
7 Marshall J. The cause and prognosis of strokes in young people under 50 years. *J Neurol Sci* 1982;**53**:473–88.
8 Prentice RL, Szatrowski TP, Kato H, Mason MW. Leukocyte counts and cerebrovascular disease. *J Chron Dis* 1982;**35**:703–14.
9 Bain B, Seed M, Godsland I. Normal values for peripheral blood white cell counts in women of four different ethnic origins. *J Clin Pathol* 1984;**37**:188–93.
10 Lowe G. Blood rheology in arterial disease. *Clin Sci* 1986;**71**:137–46.

Correction

Cysticercosis in the UK (*J Neurol Neurosurg Psychiatry* 1987;**50**:1080). The title of this letter should have been Cysticercosis in Birmingham and the authors C Shieff, ER Hitchcock, SP Valsangkar.