of expense, if on no other, or in some centres patients are receiving more effective (or more dangerous) treatment than in others. Unfortunately no data confirm that carotid surgery definitely improves the outcome in these patients. The only randomised clinical trial was inconclusive since the apparent long-term benefit after successful surgery was balanced by the immediate risk of surgery.⁷

Our own differing attitudes towards carotid surgery and our concern that they are so very different have been highlighted by the overall risk of stroke and/or death in 24% of the 41 patients undergoing carotid surgery after randomisation. Our series is, however, small, the 95% confidence limits around the 24% risk estimate are quite large, and a population risk considerably below this figure remains plausible. Our results differ greatly from the risk reported in other papers, in which, with a few exceptions,^{8,9} the risk of stroke and/or death is under 10%. If our findings truly reflect the general situation in Britain it is unlikely that the perioperative risk of stroke and/or death is better than the risk in non-operated but similar patients over several years of follow-up, the natural history studies of transient ischaemic attacks giving an approximate annual risk of stroke and/or death of 10%.¹⁰ Possibly too few endarterectomies are being carried out in some centres to maintain an acceptable surgical mortality and morbidity and patients should be referred to other centres with more experience.

Some of us believe that if the surgical risk of stroke and/or death in our institution is less than about 5% patients with transient ischaemic attacks should be exposed to the risk of angiography with a view to carotid surgery. Others who have had unfortunate results with patients referred for surgery seldom recommend angiography. Still others hold views, more or less uncomfortably, between these two extremes. In fact, many in our group prefer randomly to allocate patients in the British and French carotid artery surgery trial which has recently begun in 16 institutions. We hope this trial will resolve some of the uncertainty surrounding the usefulness of the procedure, and we believe it is this very uncertainty which explains the

enormous variation in our individual referral policies for angiography and carotid surgery.

This trial is supported by a special project grant from the Medical Research Council.

Correspondence should be addressed to: UK-TIA Trial Office, Department of Clinical Neurology, Radcliffe Infirmary, Oxford OX2 6HE.

References

- 1 UK-TIA Study Group. Design and protocol of the UK-TIA aspirin study. In: Tognoni G, Garattini S, eds. *Drug treatment and prevention in cerebrovascular disorders*. Amsterdam: Elsevier, 1979:387-94.
- 2 Faught E, Trader SD, Hanna GR. Cerebral complications of angiography for transient ischemia and stroke: prediction of risk. *Neurology* 1979; **29**:4-15.
- 3 Eisenberg RL, Bank WO, Hedgcock MW. Neurologic complications of angiography for cerebrovascular disease. *Neurology* 1980;**30**:895-7.
- 4 Swanson PD, Calanchini PR, Dyken ML, et al. A co-operative study of hospital frequency and character of transient ischemic attacks. *JAMA* 1977;**237**:2202-6.
- 5 Haerer AF, Gotshall RA, Conneally PM, et al. Co-operative study of hospital frequency and character of transient ischemic attacks. *JAMA* 1977;**238**:142-6.
- 6 Dyken ML, Conneally PM, Haerer AF, et al. Co-operative study of hospital frequency and character of transient ischemic attacks. I. Background, organisation, and clinical survey. *JAMA* 1977;**237**:882-6.
- 7 Fields WS, Maslenikov V, Meyer JS, Hass WK, Remington RD, MacDonald M. Joint study of extracranial arterial occlusion. V. Progress report of prognosis following surgery or non-surgical treatment for transient cerebral ischaemic attacks and cervical carotid artery lesions. *JAMA* 1970;**211**:1993-2003.
- 8 Bouchier-Hayes D, De Costa A, MacGowan WAL. The morbidity of carotid endarterectomy. *Br J Surg* 1979;**66**:433-7.
- 9 Easton JD, Sherman DG. Stroke and mortality rate in carotid endarterectomy: 228 consecutive operations. *Stroke* 1977;**8**:565-8.
- 10 Warlow C. Transient ischaemic attacks. In: Matthews WB, Glaser GH, eds. *Recent advances in clinical neurology*. Edinburgh: Churchill Livingstone, 1982:191-214.

(Accepted 15 October 1982)

SHORT REPORTS

Meningoencephalitis associated with *Chlamydia trachomatis* infection

The recent increase in the incidence of non-specific genital infection has brought with it a growing awareness of *Chlamydia trachomatis* as a pathological agent at various sites. We report the first British case of meningoencephalitis associated with *C trachomatis* infection.

Case report

A 23 year old heterosexual man who had never been abroad was admitted with a one-week history of fever, headache, and myalgia. Two days before admission he had developed confusion, unsteadiness on his feet, and urinary frequency. He admitted to several recent sexual contacts but these were untraceable.

On examination he was feverish with rigors and sinus tachycardia of 120 beats/minute. Cardiovascular and respiratory examination disclosed no further abnormality. Abdominal examination showed the bladder to be distended to the umbilicus. On rectal examination the prostate felt enlarged with general tenderness. External genitalia were normal. He was completely disorientated in time and space but responded to commands. He had neck stiffness, and Kernig's sign was present. On fundoscopy he had bilateral papilloedema. There was no dysphasia or focal motor signs. His gait was ataxic.

Investigations showed normal haematology and biochemistry. Radiography of the chest, skull, and lumbar spine showed no abnormality. Urine obtained by catheterisation contained many threads and debris with 10×10^6 white cells/l. No organism was cultured. The table shows results obtained at

lumbar puncture. Prostatitis with septicaemia and secondary encephalopathy was diagnosed and cefotaxime begun.

A computed tomogram of the head showed small compressed ventricles, with the cisterns of the brain stem obliterated and the overall appearance compatible with generalised brain swelling with no localised lesion. Electroencephalography showed an 8-9 cycle/s symmetrical alpha rhythm with no focal abnormalities. Two further lumbar punctures were performed (see table). Urine specimens persistently showed a white cell count of $5-10 \times 10^6$ /l but no organism on routine culture. Six sets of blood cultures were sterile. Tests for Epstein-Barr virus, hepatitis B, mumps, measles, herpes simplex, herpes zoster, cytomegalovirus, enteroviruses, influenza A and B, mycoplasma, leptospirosis, and heavy metal poisoning gave negative results.

Microimmunofluorescence antibody tests for *Chlamydia* performed on paired sera showed a 1/256 titre of immunoglobulin G antibody to *C trachomatis* D-K rising to 1/1024. There was no detectable IgM antibody. Cerebrospinal fluid yielded IgG antibody to *C trachomatis* types D-K at a

Results in cerebrospinal fluid obtained at lumbar puncture

	Admission	At 10 days	At 20 days
Red blood cells ($\times 10^6$ /l)	270 (traumatic)	2	462 (traumatic)
White blood cells ($\times 10^6$ /l)	94 lymphocytes	208 lymphocytes	143 lymphocytes
Protein (g/l)	2.3	1.21	0.52
Glucose (mmol/l)	2.8	3.0	3.4
Culture	No growth	No growth	No growth
Ziehl-Neelsen stain	Negative	Negative	Negative
Fluorescence treponemal antibody test	Negative	Negative	Negative
Immunoglobulin: albumin ratio	Normal	Normal	Normal

Conversion: SI to traditional units—Glucose: 1 mmol/l $\approx$ 18 mg/100 ml.

titre of 1/8, which did not rise. Chlamydia were not isolated from cerebrospinal fluid or urethral swabs after cefotaxime was started.

Dexamethasone was given to reduce cerebral oedema, which improved over three weeks, his mental state returning to normal. He was unable to pass urine spontaneously, however, and remained slightly feverish. Cefotaxime was stopped after one week when there was no bacterial growth in blood, cerebrospinal fluid, or urine. After a two-week course of oxytetracycline by mouth his fever settled and he passed urine normally on removal of the catheter.

Comment

Microimmunofluorescence antibody tests for *Chlamydia* are replacing complement fixation tests in diagnosis as they are type specific. The tests are simplified by pooling the serotypes for trachoma, urethritis and inclusion conjunctivitis, lymphogranuloma venereum, and psittacosis.¹ Our patient's rising IgG titre was strong evidence of recent infection. IgM is short lived, and its absence does not exclude current infection.² IgG titres in genitourinary infection rarely exceed 1/64.³ Our patient's titre was 1/1024, which suggests systemic infection. Meningoencephalitis was shown by clinical findings and results of lumbar puncture and computed tomography. As other possible causes were excluded we would suggest that this was due to *C trachomatis*. Three similar cases have been reported from Sweden.^{4 5} We believe that *C trachomatis* merits serious consideration as an aetiological agent in encephalitis in sexually active adults.

We thank Dr J D Treharne, Institute of Ophthalmology, London, for performing the microimmunofluorescence tests and for helpful advice.

¹ Treharne JD, Darougar S, Jones BR. Modification of the micro immunofluorescence test to provide a routine serodiagnostic test for chlamydial infection. *J Clin Pathol* 1977;**30**:510-7.

² Philip RN, Casper EA, Gordon FB, *et al*. Fluorescent antibody response to chlamydia infection in patients with lymphogranuloma venereum and urethritis. *J Immunol* 1974;**112**:2126-34.

³ Treharne JD, Dines RJ, Darougar S. Serological responses to chlamydial ocular and genital infections in the United Kingdom and Middle East. In: Hobson D, Holmes KK, eds. Non-gonococcal urethritis and related infections. Washington, DC: American Society for Microbiology, 1977:249-58.

⁴ Myhre E, Mardh PA. Chlamydia trachomatis infection in a patient with meningoencephalitis. *N Engl J Med* 1981;**304**:910-1.

⁵ Myhre E, Mardh PA. Unusual manifestations of chlamydial infections. *Scand J Infect Dis (Suppl)* 1982;**32**:122-5.

(Accepted 2 November 1982)

Departments of Medicine and Microbiology, Queen Mary's Hospital, London SW15 5PN

J M GOLDMAN, MB, MRCP, registrar
C S MCINTOSH, MD, MRCP, consultant
G P CALVER, MB, CHB, senior registrar
R M PERINPANAYAGAM, MB, MRCPATH, consultant

Capillary flow velocity in leukaemia

With the growth of interest in the rheological properties of blood attention has centred on the deformability characteristics of the red cell. In-vitro measurements, however, suggest that white cells are considerably less deformable than red cells,¹ a concept supported by the leucocyte plugging temporarily blocking capillary flow that has

been described in several preparations, including human pedicle skin graft.² In disease states in which the number of white cells is increased or white cells are stiffer than usual, both conditions that may prevail in leukaemia, this phenomenon of leucocyte plugging may become clinically relevant.³ We therefore studied capillary flow characteristics in unselected patients with leukaemia with raised peripheral white counts using a non-invasive technique.

Patients, methods, and results

We studied six patients. Four had chronic granulocytic leukaemia, one had follicle centre cell lymphoma in leukaemic phase, and one had acute lymphoblastic leukaemia. All patients were studied on two occasions, the second study visit being after a spontaneous rise in white cell count (cases 3 and 5) or a fall in white cell count after chemotherapy (cases 1, 2, 4, and 6). Vascular studies were performed under identical conditions at both study visits after acclimatisation in a room at constant temperature. Capillary flow was assessed in the finger nailfold microcirculation by television videomicroscopy.⁴ Velocity of capillary blood flow was determined by frame to frame analysis of the video record by measuring the distance moved by plasma gaps or red cells in recorded time. A minimum of 20 such estimations from five different capillaries were used to derive the mean velocity. The proportion of stationary vessels was estimated by recording the number of capillaries visible in a stationary frame and assessing the proportion then observed to be flowing when the record was played. Total finger arterial inflow was determined by mercury strain gauge plethysmography (Periflow, Janssen Scientific Instruments).

The table gives the results. In all patients the velocity of capillary blood flow was lower in both arterial and venous limbs of the capillary loop at the time of highest count ($p < 0.05$, Wilcoxon's signed rank sum test). The absolute (mean $\pm$ SEM) capillary blood flow velocity at the time of highest count (0.20 ± 0.06 mm/s in the arterial limb, 0.11 ± 0.04 mm/s in the venous limb) was considerably lower than normal values recorded under these conditions in our laboratory (0.92 ± 0.42 mm/s in the arterial limb, 0.56 ± 0.28 mm/s in the venous limb). Five patients showed a greater proportion of stationary vessels at the time of highest count. In contrast, plethysmographic studies showed no consistent relation between total arterial inflow and white cell count. In five patients arterial inflow was normal or high at the time of highest count despite the coexistence of stationary capillaries.

Comment

Preston *et al* described the occurrence of neurological symptoms in patients with leukaemia attributable to cellular hyperviscosity.⁵ Our study is the first description of subclinical capillary hypoperfusion associated with leukaemia. Interestingly, conventional assessment of blood flow using plethysmography gave no indication of the limitation of the nutritional capillary circulation. The limitation of capillary flow probably represents partial plugging of capillaries with large numbers of poorly deformable white cells. Television videomicroscopy promises to be a valuable technique for the study of blood rheological behaviour in vivo as opposed to in a viscometer. Our results serve to emphasise that the white cell may be of greater rheological importance than has previously been appreciated.

We thank the British Heart Foundation for research grant number 80/58; and Dr J A Child and Dr B E Roberts for permission to study patients under their care.

¹ Bagge U, Skalak R, Attefors R. Granulocyte rheology. Experimental studies in an in vitro micro-flow system. *Advances in Microcirculation* 1977;**7**:29-48.

² Adell R, Skalak R, Branemark P-I. A preliminary study of rheology of granulocytes. *Blut* 1970;**21**:91-105.

³ Bagge U, Branemark P-I, Skalak R. Measurement and influence of white cell deformability. In: Lowe GDO, Barbenel JC, Forbes CD, eds. *Clinical aspects of blood viscosity and cell deformability*. Berlin, Heidelberg, New York: Springer-Verlag, 1981:27-36.

Capillary blood flow velocity (CBV) and results of plethysmography related to white cell count in six patients with leukaemia

Case No	Age (years) and sex	Diagnosis	Highest white cell count					Lowest white cell count				
			White cell count ($\times 10^9/l$)	CBV in arterial limb (mm/s)	CBV in venous limb (mm/s)	% of stationary vessels	Finger arterial inflow (ml/100 ml/min)	White cell count ($\times 10^9/l$)	CBV in arterial limb (mm/s)	CBV in venous limb (mm/s)	% of stationary vessels	Finger arterial inflow (ml/100 ml/min)
1	73 F	CGL	300	0.23	0.11	5	13.0	10	0.56	0.16	0	23.6
2	66 F	CGL	290	0.08	0.02	50	2.6	5	0.19	0.10	0	1.4
3	29 F	CGL	109	0.46	0.28	0	26.4	83	1.13	0.48	0	11.2
4	58 F	CGL	164	0.18	0.11	15	10.8	54	0.54	0.47	0	13.9
5	57 M	FCCL	900	0.03	0.03	25	17.8	766	0.19	0.12	12	5.4
6	19 F	ALL	325	0.20	0.12	50	11.7	90	0.44	0.30	0	6.8

CGL = Chronic granulocytic leukaemia. FCCL = Follicle centre cell lymphoma. ALL = Acute lymphoblastic leukaemia.