Electrophoretic (A) and Southern blot (B) analysis of RT-PCR products from AIDS-KS and normal skin tissues.

Lanes 1–5 and 10 = AIDS-KS lesions, lanes 6–9 = normal skin. Samples for lanes 5, 6, 9, and 10 obtained from the same AIDS-KS patient. M = φX174 RF DNA/HaeIII fragments (5'-GAGGAGACCCGCTGCTGAGA-3' and 5'-CTAGAAGCATTGGAGGAGGCCC-3') and FGFD (5'-CTCTACTGAGCACTGGGGG-3' and 5'-TGAACGATGGGTCGTTACAATCT-3'), in a PCR was done in buffer containing 10 mmol/l tris-HCl (pH 8.3), 50 mmol/l KCl, 1.5 mmol/l MgCl2, 70 µg/µl gelatin, 2.5 units of Taq polymerase and 200 µmol of each dNTP. Samples were subjected to 35 cycles of denaturation (94°C, 1 min), annealing (55°C, 1 min) and extension (72°C, 1 min).

Southern blot hybridisation was with both 32P end-labelled probes for β-actin (5'-GAAACCTGCGTGACATTAAGGAGAAG-3') and for FGFD (5'-TTGGAAGCTGGGCGTTGCGTACAATCT-3'), respectively. Autoradiogram was obtained after exposure of Kodak X-ray film at −70°C for 1–4 days.

Acute transverse myelitis after tetanus toxoid vaccination

SIR—A case of optic neuritis and myelitis following a booster dose of tetanus toxoid was reported by Dr Topaloglu and colleagues (Jan 18, p 178). We recently managed a patient with acute transverse myelitis that also followed tetanus toxoid booster administration.

A 50-year-old man with a penetrating wound to the foot was given tetanus toxoid and imunoglobulin, single intravenous doses of benzylpenicillin and flucloxacillin, and a course of oral benzylpenicillin. 6 days later he returned with a widespread erythematous, maculopapular eruption, which was attributed to tetanus toxoid vaccination. 12 days after his initial presentation he was admitted after the onset, over several hours, of flaccid, areflexic paralysis of the legs, associated with sensory loss to T6, moderately severe midthoracic back pain, and urinary retention requiring an indwelling catheter. His temperature was 38.6°C but no focus of infection was evident. Cerebrospinal fluid (CSF) examination revealed neutrophil pleocytosis (white cells 16,920 x 106/µl; red cells 885 x 106/µl), raised protein (3900 mg/l), and a low glucose (1.6 mmol/l). Gram stain, bacterial antigen tests for Streptococcus
Embolism Study Group showed that HT was commonly patients treated with conventional dose heparin. The Cerebral haemorrhage on the initial scan done 1-6 days after stroke. The peak incidence was between day 7 and 14. Half of Laureno and developed within a month of cerebral embolism showed 21 after stroke found that 24 of 28 scans had HT after day 3 and the heparin or aspirin was used, have shown a high frequency of HT a quarter of HT occur late. As these workers indicate, there is evidence that about (March 7, p 589) advises that intravenous heparin should probably place by then. As these workers indicate, there is evidence that about the myelopathy in our patient occurred independently may implicate tetanus toxoid. The possible that the myelopathy in our patient occurred independently to include brachial plexus neuropathy, polyradiculoneuritis, and for triple antigen and diphtheria, tetanus, and poliomyelitis, but Topaloglu et al were the first to report myelitis after tetanus toxoid vaccination alone. Reported neurological sequelae of tetanus toxoid include brachial plexus neuropathy, polyradiculoneuropathy, and relapsing demyelinating polyneuropathy. Although it is possible that the myelopathy in our patient occurred independently implicate tetanus toxoid.

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Stroke

SIR—In your recent Stroke Octet series, the use of anticoagulants after cardioembolic stroke was discussed. Dr Hart (March 7, p 589) advises that intravenous heparin should probably not be given until at least 48 h after stroke because the peak rise in haemorrhagic transformation (HT) takes place before this time. Dr Oppenheimer and Professor Hachinski (March 21, p 721) advise waiting 4 days from stroke onset because most HT's will have taken place by then. As these workers indicate, there is evidence that about a quarter of HT occur late.

Two studies of haemorrhagic infarction in which sequential computed tomographic (CT) scans were done and only low-dose heparin or aspirin was used have shown a high frequency of HT after 3 days. Morning et al using sequential scans on days 3, 7, 14, and 21 after stroke found that 24 of 28 scans had HT after day 3 and the peak incidence was between days 7 and 14. Half of Laureno and co-workers' sequentially scanned cases had HT after day 3. Okada et al found that only 10 (15%) of 65 patients in whom HT developed within a month of cerebral embolism showed haemorrhage on the initial scan done 1-6 days after stroke.

Large cerebral infarcts are generally agreed to be a risk factor for HT, and HT increases the risk of haemorrhage into the infarct in patients treated with conventional dose heparin. The Cerebral Embolism Study Group showed that HT was commonly associated with large infarcts, and less commonly with smaller infarcts. The only study that has established statistically that a specific size of infarct is associated with a low risk of HT is that of Okada et al. These workers showed that infarcts of less than 10% of the ipsilateral hemisphere area had a very low frequency of HT (3%). Medium size infarcts had a high frequency of HT (50%), similar to that of large infarcts (51%).

The small size of an infarct on CT seems to be a better predictor of a low frequency of future HT than does the absence of haemorrhage on a 48 h or a 4 day scan. In deciding which patients to treat with anticoagulants after embolic stroke, the main purpose of an early CT scan should thus be to assess infarct size; the secondary aim should be to exclude patients in whom HT has already developed. Patients with infarcts larger than about 10% of the ipsilateral hemisphere area should be given anticoagulants only with caution because of the increased risk of future HT. A scan to assess infarct size should probably be done about 48 h after stroke since most infarcts are visible on CT by this time.

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SIR—Dr Hart's article (March 7, p 589) emphasising the importance of atraumatic fibrillation (AF) in cardiogenic embolism to the brain, affords the opportunity to address the regular absence of mural thrombi from the left atrium and appendage, at necropsy and on echocardiography, in non-valvular AF associated with embolism. In 1953, I briefly reported that in such cases special microscopic examination always disclosed a mural thrombus. The details were not included and this information has not become generally known, although it forms the pathological rationale for prophylactic anticoagulation.

This study involved the post-mortem examination of the left atrial appendage in 20 cases of non-valvular AF associated with systemic embolism, in which the left atrium and appendage showed no grossly visible thrombus and no other source of an embolus. Each appendage was divided longitudinally after which the two halves were embedded in paraffin and sectioned serially at 8 μm, and every 50th section was stained with haematoyxin and eosin. Each appendage was divided into 20 sections. In every case, using routine post-mortem cases without AF, and mural thrombus was absent.

Longitudinal section of left atrial appendage showing small mural thrombus (dark spot at top).

Haematoxylin and eosin stain (x 1).


Haematoxylin and eosin stain (x 1).