The fluid-blood level in intracranial haematoma due to anticoagulant medication

Sir: The occurrence of intracranial haemorrhage in patients receiving anticoagulant medication is a well-documented complication.¹ This complication most commonly occurs in patients with poorly controlled haemostasis. Intracranial haemorrhage may also occur in anticoagulated patients with carefully controlled haemostasis as manifested by coagulation results (bleeding time, prothrombin time, partial thromboplastin time) which are within the accepted therapeutic range. I wish to describe the fluid-blood level in five anticoagulated patients with intracranial haemorrhages.

A typical case history is that of a 57 year old normotensive woman with a history of two prior myocardial infarctions and was admitted to hospital because of substernal chest pain. Electrocardiogram was consistent with new acute ischaemic changes. Her blood pressure was 130/70 mm Hg. She was neurologically normal. She was treated with a loading dose of intravenously administered streptokinase and heparin. Her prothrombin time (180 seconds with normal of 20 seconds) and partial thromboplastin time (180 seconds with normal of 50 seconds) became markedly elevated. The dose of streptokinase was reduced and the heparin was discontinued. Despite this, 4 hours later she suddenly became unresponsive, right hemiplegic, apnoeic with no response to noxious stimulation, negative dolls head and negative caloric response, fixed and dilated pupils. CT was immediately performed and showed a large left hemisphere intracerebral haemorrhage with a fluid-blood level (fig). The patient died 4 hours later and no necropsy was performed.

In patients with normal haemostasis who develop an intracranial haemorrhage, CT shows a homogeneously nonenhancing hyperdense lesion in the acute stage. In certain anticoagulated patients, the appearance of the acute haematoma may be different, with the finding of a fluid-blood interface which is unique for this condition.² It is believed that the fluid seen above the haematoma represents plasma; the layering is a sedimentation effect. This appearance of layering within the haematoma was seen in all our patients. This fluid-blood interface occurred when there was not adequate hemostasis. When anticoagulant medication was discontinued and coagulation studies returned to normal, CT showed hyperdense hematoma without the fluid-blood interface. This change was seen in two of these patients.

Fig CT shows a left hemispheric hyperdense lesion with a sharply demarcated fluid-blood level.

The finding of intracranial fluid-blood interface has been seen in patients with peritumoural haemorrhage³ and ruptured arteriovenous malformations.⁴ In patients with angiomas, the origin of the fluid-blood level is believed to be haemorrhage into cystic spaces. The fluid-blood level has been reported with primary (glioblastoma multiforme) and metastatic neoplasms (melanoma, lung). In neoplastic conditions in which a fluid-blood interface is seen, there is frequently enhancement at the lesion periphery. In one report, fluid-blood level was seen in patients with spontaneous intracerebral haematoma without any identified aetiology or abnormal haemostasis.⁵ In these cases several potential mechanisms for fluid-blood level were postulated; (1) haemorrhage was acute and clotting of extravasated blood had not been completed when CT performed, (2) large haematoma occurs within the brain using up all clotting factors to result in two components and create fluid-blood interface. If either of these mechanisms were responsible for the fluid-blood interface, we would have expected this finding of fluid-blood interface to be common in other intracerebral haematomas. We have not seen this finding in nontumorous intracerebral haemorrhage unless there was impaired haemostasis, as seen in these five patients.

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Reversible movement disorder in a patient with post traumatic basal ganglia haematoma

Sir: The eponyms Brueghel's syndrome¹ and Meige syndrome or disease² have been coined for an idiopathic disorder consisting of blephrospasm and/or oromandibular dystonia of adult onset. The symptoms are progressive or static, though improvement in a few cases has been reported.^{3,4} The cause of Meige disease is not known and evidences for its organic nature are indirect. A single report on the brain pathology did not reveal any abnormality.⁵ Fornio⁶ described nerve cell loss and other reactions of the substantia nigra in a brain with an old basal ganglia infarct. We describe a patient who developed transient oromandibular dystonia with blephrospasm following basal ganglia hematoma presumably of post-traumatic origin.

A 64 year old man had an accidental fall and sustained an abrasion over left temporal region and a left periorbital haematoma. He was unconscious for 15 minutes and thereafter complained of a dull continuous headache with inability to concentrate. Sensorium deteriorated again over next days. He was reported to be irritable, confused and drowsy. He was admitted to a provincial hospital where involuntary eye closure, clenching of teeth, forceful jaw opening and jerking of limbs were noted bilaterally, but more on right side. He was unable to walk owing to unsteadiness, involuntary posturing and stiffness of body. A left carotid angiogram was performed and