become identifiable, their transfer from person to person may be explained by simple wafting, perhaps from victim to doctor to accused. Awareness of this hazard should lead to the taking of proper precautions.

In the course of his survey of fibre evidence **Dr Nigel Watson** (Forensic Science Unit, University of Strathclyde) referred to the Wayne Williams multiple child murder case in the USA. After much closely argued cross examination, the court accepted that green acrylic fibres of unusual appearance originating from a carpet linked several of the bodies. The methods of characterizing and identifying fibres were explained, but there remains the problem of putting this evidence in a statistical frame acceptable to a court.

The final contribution was by **Dr Norman Wallace** (Police Surgeon, Lothian and Borders) who had compiled a training video film on the diagnosis of intoxication in drivers. The requirement to perform clinical examination of drunk drivers has become much rarer since the introduction of evidential breath machines, with a corresponding decrease in the level of experience in dealing with such cases.

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Letters to the Editor

Preference is given to letters commenting on contributions published recently in the JRSM. *They should not exceed 300 words and should be* **typed double-spaced.**

Value of emergency cardiac enzymes: audit in a coronary care unit

We read with interest the audit of emergency cardiac enzymes in the coronary care unit by Lewis *et al.* (July 1991 *JRSM*, p 398), and agree that urgent creatine kinase (CK) measurements are required in the management of acute myocardial infarction (AMI).

It has been recognized that in acute myocardial infarction, electrocardiography (ECG) provides the most convenient and reliable method for diagnosis[1]. Over 80% of patients with acute myocardial infarction have abnormal ECG changes on presentation[2]. Twenty to 25% of patients with AMI will present with non specific or normal ECG's[3], and these patients require biochemical tests to substantiate the diagnosis. We conducted a study on a group of patients admitted with chest pain into our coronary care unit, to determine how to provide a better cardiac enzyme service. It included 41 patients who had blood taken on admission, 2, 4, 6, 8, 12 and 24 h. Total creatine kinase (CK) was assayed the next day on the Cobas Bio, using the Randox CK NAC-activated kit, serial electrocardiographs (ECG) were done daily. Twelve of these patients were diagnosed as unstable angina with no ECG changes and their serial CK results remained within the reference range. Twenty-nine of these patients were diagnosed as acute myocardial infarction based on serial CK results, ECG changes and clinical history, but only 19 of them had raised CK above the reference range on admission. Of the 10 patients with 'normal' CK results on admission, four patients demonstrated an elevated CK in the 2 h sample, and the remaining six had elevated levels by the 4 h sample (Table 1).

Table 1. Timed sequential creatine kinase measurements

	Time from admission (h)	*Elevated CK patients*
Unstable angina	0	0
(n=12)	2	0
	4	0
	6	0
Myocardial infarction	0	19(67%)
(n=29)	2	23(79%)
	4	29(100%)

We agree with the guidelines given by Lewis *et al.* about requesting urgent CK, but we would like to stress in addition, that the group of patients who present with non specific ECG changes and 'normal' admission CK, require sequential CK estimations to confirm the diagnosis of AMI, supporting the suggestion in a previous study[4]. This suggestion should be taken into account when providing a cardiac enzyme service.

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The history of Cushing's disease

We read with interest the account by Medvei on the history of Cushing's disease (June 1991 *JRSM*, p 363). This review is accurate and covers the topic from the origin to the discovery of ectopic ACTH syndrome in the early sixties. However, some additional information is required to make the story complete. An important omission was the failure to quote the report by Itsenko. This Russian physician reported in an obscure journal in 1924 the case of an obese and hirsute patient with pituitary enlargement and

presumed hypothalamic involvement[1]. In the Soviet Union the disorder is called Itsenko-Cushing's syndrome. A partial English translation of Itsenko's paper has been reported by Christy[2]. The hypothesis of hypothalamic involvement was revived more recently, particularly in the decade between 1968 and 1977, when endocrinologists had to explain the results of new endocrine tests in Cushing's disease, in particular the dichotomy between absent ACTH response to hypoglycaemia and normal or exaggerated responses to metyrapone and vasopressin. During that time, the theory of a fundamental defect of supra-pituitary regulation of ACTH secretion in Cushing's disease gained wide, although not universal, acceptance[3,4]. It is probably not by chance that Itsenko's paper was so extensively quoted in Christy's historical note published in 1971. The situation changed suddenly in 1978 when it was demonstrated that virtually all patients with Cushing's disease have a pituitary adenoma and that successful adenoma removal is followed by transient secondary hypo-adrenalism, a situation that excludes continuing hypothalamic stimulation of the pituitary cortico-trophs, and suggests instead chronic suppression of corticocotrophin releasing hormone (CRH)[5]. It is notable that this result was obtained by a group that had been critical of the hypothalamic theory[6]. Further clarification was provided by the demonstration that CRH is suppressed in Cushing's disease and that it remains suppressed after hypoglycaemia and metyrapone[7]. These data elucidated the mechanism of ACTH unresponsiveness to hypoglycaemia and suggested that the responses to metyrapone, vasopressin and exogenous gluco-corticoids are probably mediated by an action on the pituitary adenomatous cells. So, 60 years after the last Cushing's papers the mechanism of his disease appears almost completely clarified; the pituitary tumour is the primary event and hypothalamic involvement is secondary to chronic hypercortisolism.

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Recurrent pancreatitis

I cannot but agree with the conclusions reached by Davies *et al.* (April 1991 *JRSM*, p 235) that at any operation on a patient with pancreatitis of whatever aetiology, cholecystectomy should be performed, the author has made this a policy for the last 20 or 30 years. Their case adequately illustrates the difficulty in palpating very small stones through the wall of the gallbladder at operation. But even without stones the gallbladder may be the culprit as indicated in the following history.

A 40-year-old woman had a laparotomy by a surgical registrar for acute abdomen and was found to have acute pancreatitis. The gallbladder felt normal and was not removed. The patient continued to have mild attacks of recurrent pancreatitis; cholecystogram (before ultrasound was available) proved normal. Subsequently in another hospital the gallbladder was removed; no stones were found therein, but the patient's attacks ceased. Presumably some patients produce a generation of tiny stones which passing through the ampulla, cause spasm and pancreatitis. Investigation immediately afterwards will show the gallbladder to be normal, but when another generation of stones is produced, the attack recurs.

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Immunology of the tonsils

I was grateful for the replies (July 1991 *JRSM*, p 447) to my letter (January 1991 *JRSM*, p 58).

My first introduction to tonsillitis deficiency was in 1955 when an ENT Consultant in Rhodesia commented on how few African children had tonsillectomies. Fresh from England I assumed this meant that African children were neglected.

In Rhodesia, now Zimbabwe, GPs learned from other GPs how to do their own tonsillectomies as did GPs in the UK prior to the NHS. I had to learn on European children, though my main task was African women and children at Shabanie Mine. A position of trust ensued, but it was two years before I did a T & A on an African child. It was in the course of this operation that I remembered my righteousness at the ENT surgeon's remark.

My only two later African Ts & As over 3 years were for children of school teachers who had learnt European ways. The knowledge they acquired is now extensive at the expense of traditional understandings described by Dr Campbell, who may tell us if the modern consequences also include African appendicectomies. Burkitt used to say. 'Do not diagnose appendicitis in an African unless he speaks English'. This was certainly true when Burkitt said it.

Dr Fry's comment on dried milk at Tobruk is valid. It has the same sequels as liquid milk. Deaths following WHO assistance have been commonly attributed to lactase deficiency, but there may be other considerations.

In the former Colonial areas there is a great silent pool of knowledge which, if awakened, may question many commonly held beliefs including, perhaps, the one about calcium in milk. Rare primary cancers of the liver, found only in Africans, used to be medical mysteries. I have not found a reference to this in discussions on AIDS.

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