CORRESPONDENCE

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- *No letter should be more than 400 words.*
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- *Because we receive many more letters than we can publish we may shorten those we do print, particularly when we receive several on the same subject.*

Use of microaggregate filters: need for audit

SIR,—We have recently discovered that the use of red cell filters of pore size 40 μm in our hospital is haphazard, relying more on ward folklore and the date of the last visit by the company representative than on a formal policy. This has resulted from the fact that wards order blood cell filters directly from the supplies department. Furthermore, the same occurs in many of the hospitals within the west midlands: only four out of 24 acute hospitals lay claim to a formal written policy for using these filters, and in two of these four units this policy is ignored in practice.

Indications for the use of microaggregate filters are debatable, but many claim that they are of potential value in treating patients undergoing massive transfusion, those in intensive care units, and those with thrombocytopenia.

Last year the West Midlands region purchased around 26 500 filters at a total cost of £113 500, and during this time around 169 000 units of blood were transfused. Assuming that one filter lasts for two units, this implies that over 30% of transfused blood is filtered. Experience suggests that this is unnecessary in many instances, and we suggest that hospitals critically evaluate their use of such filters. A hospital transfusion committee may be the most appropriate group to formulate written criteria, but implementation of such a policy will depend on restricting the issue of filters to intensive treatment units and blood banks. This state of affairs also emphasises the importance of clinical budgeting at a ward level.

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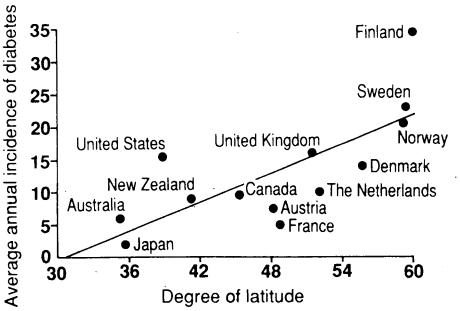
Coffee consumption as trigger for diabetes in childhood

SIR,—Professor Jaakko Tuomilehto and colleagues used regression analysis as a means of showing "association between annual average coffee consumption per person and the age standardised incidence of insulin dependent diabetes."[1] They rightly point out that correlation analyses must be used with caution, and we concur with this view. To highlight the problem with this method of hypothesis generation we have taken the same countries and plotted the latitude of their capital cities against the average annual incidence of diabetes mellitus (figure). These data correlate slightly better than the coffee consumption data ($R^2=0{\cdot}54$ *v* $R^2=0{\cdot}53$) and imply that the incidence of diabetes increases with distance from the



Average annual incidence of insulin dependent diabetes mellitus in patients aged 0-14 adjusted for age per 100 000 population[1] by latitude of capital cities. Incidence= 0·74×latitude −22·7; $R^2=0{\cdot}54$

equator. This may help us to generate another hypothesis related to hours of sunlight, but we need to be aware of the fact that there are many other differences between Scandinavia and Japan that may explain the altered incidence of diabetes.[2] Coffee consumption may be a trivial marker of such a difference.

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1 Tuomilehto J, Tuomilehto-Wolf E, Virtala E, LaPorte R. Coffee consumption as trigger for insulin dependent diabetes mellitus in childhood. *Br Med J* 1990;**300**:642-3. (10 March.)
2 Mann JI, Pyörälä K, Teuscher A. *Diabetes in epidemiological perspective.* London: Churchill Livingstone, 1983.

SIR,—Professor Jaakko Tuomilehto and colleagues report a correlation between the incidence of insulin dependent diabetes in subjects aged 0-14 years and average coffee consumption in some developed countries.[1] They suggest that exposure of the fetus to high concentrations of caffeine may be the environmental factor which triggers β cell destruction in susceptible people. We question whether the correlation can be taken at face value.

The effect of coffee intake on serum cholesterol concentration has recently been shown to depend heavily on the mode of preparation.[2] A randomised study over 12 weeks of 107 young adults found that drinking filtered coffee had no effect on serum lipid concentrations whereas consumption of boiled coffee produced a mean increase of 10% in serum cholesterol concentration. The practice of boiling coffee as described in that paper is common in Scandinavia (where the consumption of coffee and the incidence of insulin dependent diabetes are highest). If this group of countries is excluded from the analysis of Professor Tuomilehto and colleagues on the grounds that their population is inbibing a different preparation then the correlation between coffee intake and incidence of insulin dependent diabetes loses significance ($r^2=0{\cdot}0154$). This is despite there being a fourfold difference in coffee intake and a sevenfold difference in annual incidence of insulin dependent diabetes in the remaining nine countries.

Even if the correlation remained, generation of a hypothesis specifically implicating caffeine is not justified. The annual intake of caffeine depends not only on the amount of coffee consumed but also on the consumption of other beverages, especially tea. In the United Kingdom, for example, the average consumption of tea each year is 2·81 kg/person.[3] The mean caffeine content in each cup varies between 15 μg and 75 μg depending on whether loose tea or tea bags are used. This compares with 61-70 μg of caffeine in each cup of instant coffee and 97-125 μg in each cup of filtered coffee.[4]

Professor Tuomilehto and colleagues state that they intend to test their hypothesis in animals. We suggest that they direct their attention to the by-products of coffee prepared by boiling. The correlation between the intake of other forms of coffee and development of insulin dependent diabetes seems to be weak and may not exist at all.

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1 Tuomilehto J, Tuomilehto-Wolf E, Virtala E, LaPorte R. Coffee consumption as trigger for insulin dependent diabetes in childhood. *Br Med J* 1990;**300**:642-3. (10 March.)
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3 International Tea Committee. Apparent consumption of tea. *Retail Business* 1990;**383**:56.
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Monitoring cardiopulmonary resuscitation by end tidal carbon dioxide concentration

SIR,—We congratulate Dr D Higgins and colleagues on the simplicity of their approach to end tidal carbon dioxide monitoring during cardiopulmonary resuscitation.[1] Perhaps they should have mentioned that changes in expired carbon dioxide concentration reflect pulmonary perfusion—that is, cardiac output—only if alveolar ventilation and carbon dioxide production stay constant.[2,3] If manual rather than mechanical ventilation is used or if sodium bicarbonate is given then these conditions may not apply.

Our own work confirms the value of monitoring end tidal carbon dioxide concentration in these

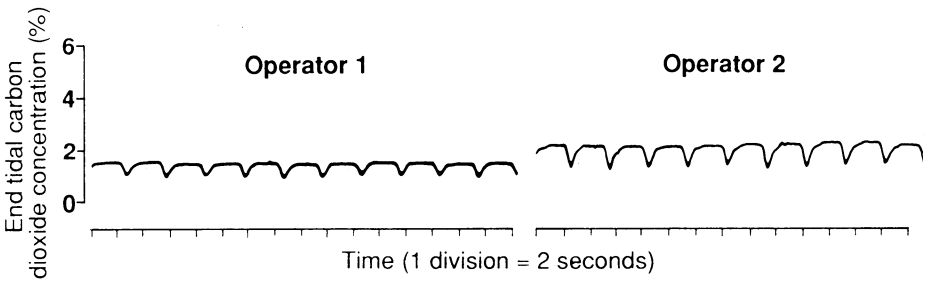

FIG 1—*Infrared analyser trace showing end tidal carbon dioxide concentration with two different physicians giving external chest compression*

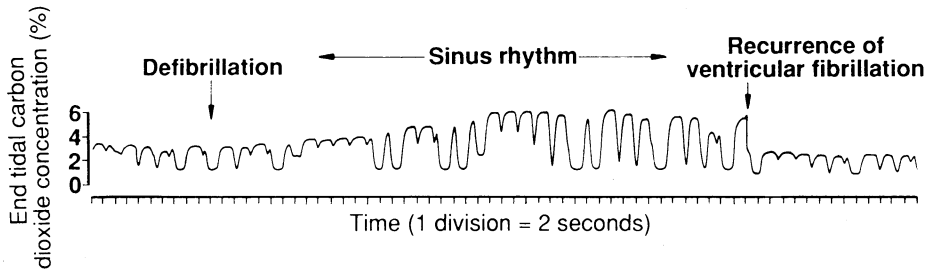

FIG 2—*Infrared analyser trace showing changes in end tidal carbon dioxide concentration with successful defibrillation and with recurrence of ventricular fibrillation*

circumstances. We have studied 13 adults who had suffered cardiac arrest and required endotracheal intubation and cardiopulmonary resuscitation. The lungs were ventilated with 100% oxygen by using a Motivus 2 portable ventilator delivering a constant minute volume, and end tidal carbon dioxide concentration was measured with a Godart Model BE infrared analyser.

During external chest compression the mean (SD) end tidal carbon dioxide concentration was 2·23 (0·92)%. When chest compression was stopped to check for a spontaneous circulation it fell to 1·57 (0·86)%. During each phase the end tidal concentration was calculated for each patient by taking a mean value from five consecutive breaths. This difference was significant (paired Student's t test, $p<0·01$). We also noted the rapid and sustained rise in end tidal concentration associated with the return of a spontaneous circulation, when it reached a mean of 5·08 (1·48)%.

The technique provides an instantaneous and continuous guide to the efficacy of external chest compression and, as Dr Higgins and colleagues noted, will indicate when this is inefficient. Figure 1 shows the rise in end tidal concentration that occurred (from 1·53% to 2·39%) in one patient when chest compression was taken over by a more experienced and vigorous physician.

Equally helpful was the monitoring of the abrupt fall in carbon dioxide concentration that was seen with any serious failure of the circulation once it had been restored. Figure 2 shows the increase in end tidal concentration after successfully defibrillating a patient in ventricular fibrillation and then the rapid fall as ventricular fibrillation recurred.

Although giving sodium bicarbonate produced no consistent discernible change in end tidal concentration, in some cases it was injected into a peripheral vein and the lack of effect may have reflected the considerable delay in its reaching the central circulation and the tissues thereafter. Others have found the rise in end tidal concentration after giving bicarbonate to be short lived and far less than that seen when spontaneous circulation is restored.[2]

Previous workers have studied patients either arriving in the accident and emergency department[2] or already in intensive care units.[3] Our study and that of Dr Higgins and colleagues show that this technique can be applied equally well in general wards. The studies confirm that monitoring end tidal carbon dioxide concentration provides a practical and non-invasive means of assessing the circulation during cardiopulmonary resuscitation. As such it forms a valuable guide for the resuscitation team and a useful teaching aid for those learning these techniques.

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Dangers of thrombolysis

SIR,—With reference to the leading article by Mr M C Petch concerning the early use of thrombolytic agents in acute myocardial infarction[1] I would like to report the following case history.

I am a general practitioner in an isolated rural practice, which is 40 km from the nearest district general hospital and 11 km from the nearest ambulance station. I was called to see a patient—a 51 year old businessman (who was normotensive, overweight, and a former smoker)—at 3 am. He had suffered three days of epigastric discomfort, which had suddenly worsened and become a crushing low chest pain with breathlessness, which was not helped by sublingual glyceryl trinitrate. He did not describe any other symptoms.

His medical history included the occurrence of an anteroseptal infarct in June 1986. Investigations at that time showed a fasting cholesterol concentration of 8·9 mmol/l and triglyceride concentration of 2·2 mmol/l, and he weighed 96 kg (body mass index 31·4 kg/m^2). He was discharged on a low cholesterol and weight reducing diet and taking metroprolol 50 mg daily. He stopped smoking but continued to suffer occasional episodes of chest pain, although it was not clear whether these were due to angina. On review in April 1987 his fasting cholesterol concentration was 7·5 mmol/l, so he was given bezafibrate 400 mg daily, which reduced his cholesterol concentration to 4·86 mmol/l. His alcohol intake was in excess of 20 units per week. He had no history of peptic ulceration.

On examination he was apyrexial, dyspnoeic, distressed, pale, and sweaty. His pulse was regular at 84 beats/min and blood pressure was stable at 120/70 mm Hg. There were no signs of cardiac failure, and I found no evidence of acute abdominal disease. Being unsure of the diagnosis I withheld anistreplase and performed electrocardiography, which showed no acute changes. I gave him morphine 15 mg and oxygen. Thankfully, despite the dispute, the local ambulance crews were providing an emergency service, and a call direct to Maldon ambulance station achieved an immediate response.

After eight minutes my patient's condition deteriorated rapidly; he had severe crushing retrosternal chest pain and epigastric pain. I therefore gave him diamorphine 5 mg and anistreplase 30 units intravenously as I had little doubt that he was having a myocardial infarction.

I travelled with him to hospital (a journey taking 40 minutes) but thankfully he remained stable and his pain gradually subsided. Further electrocardiography performed after admission to the coronary care unit showed no acute changes, and his enzyme activities were later found to have been normal. As he had clearly not had an infarction and as he subsequently developed a fever he was treated with intravenous cefuroxime for presumed cholecystitis and his care was transferred to the members of the surgical team who were on call; they could find no evidence of a surgically amenable cause. An ultrasound scan and oral cholecystogram failed to show any gall bladder abnormality, and he has remained well since discharge.

I think that the fever was probably an adverse reaction to the anistreplase, and both the patient and I are convinced that the anistreplase prevented an impending infarction. If fibrinolysis had been withheld until admission the outcome might have been quite different. I advocate that all rural general practitioners should keep thrombolytic agents and be prepared to use them, but the present arrangements for paying general practitioners for the cost of these expensive drugs make this unlikely.

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1 Petch MC. Dangers of thrombolysis. *Br Med J* 1990;**300**:483-4. (24 February.)

Genital warts

SIR,—I was interested in the lesson of the week by Dr C A Carne and Mr G Dockerty,[1] having recently published a similar study.[2] Our study was carried out prospectively at the department of genitourinary medicine at Guy's Hospital during the latter part of 1988. Unlike Dr Carne and Mr Dockerty we considered only female patients. We too felt that it was appropriate to reassess the observations of Kinghorn[3] in the light of the availability of testing for *Chlamydia trachomatis* infection.

Whereas Dr Carne and Mr Dockerty found that 18·5% of women had coincident infection with *C trachomatis* we found by a similar technique that only 9% were positive. One of these women positive for chlamydia was also infected with *Trichomonas vaginalis*; there were no other cases of sexually transmitted infection among our patients.

A similar rate of *C trachomatis* infection (10·7%) has been found in a general practice population.[4] Fish *et al* found that 9·5% of women aged 16-25 attending a gynaecology clinic were infected with chlamydia.[5] We therefore concluded that among our female patients with genital warts the incidence of other sexually transmitted diseases was no higher than among women of similar age within the community. Clearly this was not so among the Cambridge patients. There must have been some intrinsic difference between the two groups of