On resuming ventilation an immediate and irreversible colour change from mauve to yellow occurred in the carbon dioxide detector. Consequently, the device no longer predicted either correct tube placement or efficacy of resuscitation efforts.

We have further investigated this phenomenon in vitro by instilling drugs into the device. An identical and permanent change to yellow occurs with the other drugs—lignocaine, adrenaline, and atropine—used intratracheally in patients suffering a cardiac arrest and with gastric aspirate. No significant colour change occurred with sterile water, physiological saline, or pulmonary oedema fluid.

Our findings suggest that the FEF device would not necessarily detect oesophageal intubation if the colour indicator became contaminated with gastric acid and remained permanently yellow. During cardiopulmonary resuscitation the device could no longer be used as an indicator for the effectiveness of resuscitation manoeuvres once lignocaine, adrenaline, or atropine had been instilled into the endotracheal tube and an irreversible colour change had occurred. Additionally, if the device is used in a breathing system that allows rebreathing—for example, a Bain or Mapleson B system—the usual breath to breath colour change of the carbon dioxide detector is not seen.

In conclusion, it is clear that the FEF end tidal carbon dioxide detector is not specific for carbon dioxide and its use must be tempered with caution. We have informed the device's manufacturers of our findings.

J D MUIR
P B RANDALLS
G B SMITH

Intensive Therapy Unit,
Queen Alexandra Hospital,
Portsmouth PO6 3LY

1 Higgins D, Hayes M, Denman W, Wilkinson DJ. Effectiveness of using end tidal carbon dioxide concentration to monitor cardiopulmonary resuscitation. *Br Med J* 1990;**300**:581. (3 March.)
2 Goldberg JS, Rawle PR, Zehnder JL, Sladen RN. Colorimetric end-tidal carbon dioxide monitoring for tracheal intubation. *Anesth Analg* 1990;**70**:191-4.
3 Feinstein R, White PF, Westerfield SZ. Intraoperative evaluation of a disposable end-tidal Co_2 detector. *Anesthesiology* 1989; **71**:A461.
4 Chamberlain DA. Advanced life support. Revised recommendations of the Resuscitation Council (UK). *Br Med J* 1989; **299**:446-8.

AUTHORS' REPLY,—We wish to add our comments to those of Dr J D Muir and colleagues. Our experience of using the FEF carbon dioxide detector[1] during cardiopulmonary resuscitation had alerted us to the problems of gastric acid contamination and the effects of rebreathing when inappropriate circuitry is used.

Soiling with gastric acid produces a deep orange colour on the dome of the device, which is permanent and quite different from the yellow colouration produced by carbon dioxide. The use of an inappropriate gas delivery system during resuscitation attempts, which results in rebreathing to the extent that the detector does not return to the expected purple colour during inspiration, is an event that causes concern. There is an urgent need for junior medical staff to be aware of the basic principles of all gas delivery systems so that they are used appropriately during cardiopulmonary resuscitation. The FEF detector does have a useful role here in that it will indicate rebreathing.

The effect of intratracheal instillation of drugs on the detector during cardiopulmonary resuscitation has been highlighted by Dr Muir and colleagues and is further cause for concern. We investigated the effects of both nebulised drugs and the same drugs in direct contact with the detector. The table gives the results.

The yellow colouration produced by a 1:10 000 solution of adrenaline was identical with that produced by carbon dioxide and that shown on the dome of the detector. This colour was permanent and occurred despite the presence of a heat and moisture exchanger filter in the circuit between the nebuliser and the detector. We believe that the manufacturers of this device should issue a warning about this effect (we have advised them of our findings). Interestingly, in our study of 100 intubations during which the larynx and trachea were sprayed with 4% lignocaine the detector remained responsive to carbon dioxide.[2]

In conclusion, those who use this device during cardiopulmonary resuscitation must look for a cyclical colour change from purple to yellow during respiration to indicate effective ventilation and perfusion of the lungs. A fixed orange colour indicates contamination with acid. A fixed yellow colour indicates either an inspired carbon dioxide concentration above 2% or contamination of the device with tracheally administered drugs. A fixed purple colour indicates either inadequate perfusion or failed intubation. We still believe that this simple apparatus provides considerable information during cardiopulmonary resuscitation which is of great value.

M HAYES
D HIGGINS
E H S YAU
W DENMAN
D J WILKINSON

Department of Anaesthesia,
St Bartholomew's Hospital,
London EC1A 7BE

1 Higgins D, Hayes M, Denman W, Wilkinson DJ. Effectiveness of using end tidal carbon dioxide concentration to monitor cardiopulmonary resuscitation. *Br Med J* 1990;**300**:581. (3 March.)
2 Denman W, Hayes M, Higgins D, Wilkinson DJ. The Fenem carbon dioxide device. *Anaesthesia* 1990;**45**:465-7.

Effect of drugs on colour change shown by FEF carbon dioxide detector when nebulised and when in direct contact with detector

Drugs	pH	Permanent colour change with nebulised drugs	Permanent colour change with drugs in direct contact
Adrenaline (1:10 000)	3·33	Yellow	Yellow
Atropine	4·80	No effect*	Light purple
Lignocaine (4%)	1·72	Yellow discolouration at the edges*	Orange/yellow
Salbutamol (5 mg/ml)	3·28	No effect*	Light purple
Physiological saline	5·80	No effect*	No effect

*The detector remained responsive to carbon dioxide.

Vitamin B-12 and folate deficiency presenting as leukaemia

SIR,—I was surprised by the content of the Lesson of the Week by Dr I S Dokal and colleagues, who report on two patients with megaloblastic anaemia who were initially mistakenly diagnosed as having acute myeloblastic leukaemia. They state that the haematological findings were not typical of the correct diagnosis. With the exception of the raised white cell count in case 1 all of the features described are characteristic of patients with megaloblastic anaemia.

It is unusual not to be able to aspirate bone marrow from these patients, but if this is impossible a good trephine biopsy sample will give adequate cellular morphology. The typical appearances in megaloblastic anaemia may, as Dr Dokal and colleagues show, be frightening to those unfamiliar with them. It is standard practice in the Northern region for photographs of such trephine biopsy samples to be used in training sessions for young haematologists, and good photographs are available in current atlases of haematology.[2]

The bone marrow appearances in case 2 were more unusual, but, rarely, precursors of red cells are lost almost completely from the marrow and confusion with acute myeloid leukaemia can occur.[3]

I find it disturbing that chemotherapy was avoided in case 1 only because technical difficulties led to delays in getting treatment started. I think that there are lessons to be learnt from this paper but, unfortunately, I do not think that they are those that the authors intended.

ANNE LENNARD

Department of Haematology,
Royal Victoria Infirmary,
Newcastle upon Tyne NE1 4LP

1 Dokal IS, Cox TM, Galton DAG. Vitamin B-12 and folate deficiency presenting as leukaemia. *Br Med J* 1990;**330**:1263-4. (12 May.)
2 Hoffbrand AV, Pettit JE. *Sandoz atlas of clinical haematology*. London: Gower Medical Publishing, 1988.
3 Weatherall DJ, Ledingham JGG, Warrell DA. *Oxford textbook of medicine*. Oxford: Oxford University Press, 1987.

AUTHORS' REPLY,—We agree with Dr Anne Lennard that in case 1 there were some features that are typical of megaloblastic anaemia. As she herself points out, however, this patient had a greatly increased white cell count ($24{\cdot}4\times10^9$/l), she had a normal mean corpuscular volume (89 fl), and aspiration of bone marrow proved to be impossible. On the basis of the bone marrow trephine biopsy a diagnosis of acute myeloid leukaemia was made by experienced haematological morphologists (including DAGG). It seems likely that the coexistent chest infection contributed to the atypical morphology, and we are not convinced that examination of a trephine roll would have given the correct diagnosis.

In case 2 acute myeloid leukaemia was considered as one of the possible diagnoses because of the presence of numerous promyelocytes showing heavy granulation. Megaloblastic anaemia was considered from the outset and was substantiated by the low serum folate concentration and response to treatment. In this case again the presence of infection may have been responsible for the unusual morphology.

In this Lesson of the Week we intended to emphasise the diagnostic difficulties posed by these two patients. We have seen several similar cases over the years but were not able to include them in our account because some essential information could not be found. We are pleased that Dr Lennard is confident that she can recognise these cases, but we have not found clear descriptions of their atypical clinical and laboratory features and fear that mistakes will continue to be made.

I S DOKAL
T M COX
D A G GALTON

Department of Haematology,
Royal Postgraduate Medical School
and Hammersmith Hospital,
London W12 0NN

Antihypertensive and adverse biochemical effects of bendrofluazide

SIR,—Perhaps Professor Peter Sever could explain the apparent contradiction between the views he expressed as a member of the British Hypertension Society working party: "First line treatment with a diuretic or β-blocker is equally acceptable: other agents may be necessary when these drugs are contraindicated or ineffective,"[1] and the view expressed in his letter that "the use of diuretics as