

10 years, and a large scale study did not find any case in the first five years of the disease.¹ There have been a few reports of proliferative retinopathy developing at an early stage of type I diabetes,² and proliferative retinopathy is occasionally seen as a presenting feature of type II diabetes, usually in patients aged 40 or older.³ In case 1 the patient had typical type I diabetes with islet cell antibodies and no residual β cell function. The other patient was less typical of patients with type I diabetes as he had residual β cell function and had been obese some years previously. Both patients, however, had the HLA-DR tissue type typical of type I diabetes.⁴ It has been suggested that this predisposes to proliferative diabetic retinopathy, though a recent study could not confirm this.⁵

As duration of diabetes is so powerful a predictor of retinopathy, both patients had probably had undetected diabetes for many years. They may also

have had some other underlying factor facilitating the rapid development of their complications.⁵

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Childhood leukaemia around the La Hague nuclear waste reprocessing plant

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The incidence of childhood leukaemia around nuclear facilities has been a topic of much public attention and epidemiological investigation.^{1,2} The Sellafield and Dounreay nuclear waste reprocessing plants have been particularly investigated.^{3,4} A similar reprocessing plant has been operating in La Hague, Normandy, since 1966, several years after Sellafield was commissioned. The amount of radioactive effluent discharged has been much lower than that from Sellafield (the maximum annual discharge in the years to 1980 was 1 592 887 GBq in 1975). The main radionuclides released are ruthenium-106, cerium-144 (up to 1980), caesium-137, and antimony-125. Discharges from Sellafield and La Hague differ in their isotope contents: Sellafield releases 10 to 160 times more ¹³⁷Cs and La Hague four times more ¹²⁵Sb.³ We report preliminary results on mortality from childhood leukaemia observed around the plant at La Hague.

Methods and results

Mortality for the periods 1968-78 and 1979-86 was provided by the Institut National de la Santé et de la Recherche Médicale, which records all the medical causes of deaths in France centrally. Three age groups were examined: 0-4 years, 5-14 years, and 15-24 years. We studied all electoral wards (around 10 000 inhabitants) that had half or more of their area within a specified radius of the nuclear plant. Radiuses of 10 km, 20 km, and 35 km were chosen before the

analyses, so that 10 wards were included in the study. The expected numbers of cases of leukaemia (International Classification of Diseases (8th revision) codes 204-207; 9th revision codes 204-208) were estimated by applying the age specific rates for the département de la Manche for 1968-78 and 1979-86 to the 1975 and 1982 census populations of the predefined areas. Results were analysed by two tailed tests based on a Poisson distribution.

Only one death occurred in the area closest to the nuclear installation between 1968 and 1986 (table). Only one standardised mortality ratio was significantly different from one: the ratio for the age group 5-14 living 10-20 km from the plant during 1968-78 showed a decreased risk (observed number of deaths=0, expected=3.935). Because of the number of tests carried out (table) one of the tests would be expected to yield a significant result ($p < 0.05$) on the basis of chance.

The standardised mortality ratio for all the age groups, periods, and areas was 89% (observed number of deaths=21, expected=23.6); and was not significant (95% confidence interval 0.55 to 1.36). Furthermore, no significant trend between the two periods was found.

Comment

Assessing the risk of disease around a source of environmental pollution is subject to many statistical problems, including the selection of the boundaries of the area to be studied, the choice of a reference rate, and the use of the Poisson distribution. It is difficult to attempt any other kind of statistical analysis with the available data. A registry of all cases of leukaemia and their exact location needs to be established, especially as improvements in treating childhood leukaemia are being reflected in a falling mortality. The Commission d'Information de la Hague instigated a comprehensive

Mortality from leukaemia around La Hague nuclear reprocessing plant according to age, period of study, and distance from plant

Period of study and distance from plant	0-4 Years			5-14 Years			15-24 Years		
	Observed deaths	Expected deaths	Standardised mortality ratio (%)	Observed deaths	Expected deaths	Standardised mortality ratio (%)	Observed deaths	Expected deaths	Standardised mortality ratio (%)
1968-78:									
<10 km	0	0.143	0	0	0.340	0	0	0.136	0
10-<20 km	2	1.831	109	0	3.935	0*	5	2.002	250
20-<35 km	0	1.038	0	3	2.697	111	0	1.199	0
1979-86:									
<10 km	0	0.096	0	0	0.220	0	1	0.214	467
10-<20 km	2	0.986	203	2	2.126	94	1	2.579	38
20-<35 km	1	0.669	149	2	1.617	123	2	1.746	115

* $p < 0.05$.

survey of cancer in the area, but detailed geographical results have not to our knowledge been published or made publicly available. A retrospective study of hospital or pathology records, or both, is required but might be difficult to carry out in view of the quality of record keeping in the past.

Our report highlights the need to establish a permanent cancer registry for this area of France available to researchers. This is all the more important as a nuclear power station began to operate 16 km away in December 1985.

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Effectiveness of using end tidal carbon dioxide concentration to monitor cardiopulmonary resuscitation

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End tidal carbon dioxide concentration indicates both correct tracheal intubation¹ and effective cardiac compressions during cardiopulmonary resuscitation.² Its application has been limited by the cost and practicality of providing infrared analysers or mass spectrometers at all sites where resuscitation may be attempted. We report the use of a cheap pocket sized detector that allows immediate recognition and correction of oesophageal intubation and ineffective cardiac compression.

Methods and results

The end tidal carbon dioxide detector (FEF; Fenem) is a portable device that does not require a power supply. It has an internal volume of 38 ml, a flow resistance <3 cm water at 60 l/min, and weighs <30 g. An easily visible colour change shows the variation in carbon dioxide concentration during inspiration and expiration.

We used the detector in 30 varied resuscitations at this hospital. At the site of resuscitation the patient's trachea was intubated and the detector inserted between the tracheal tube and the breathing system. Each patient was then ventilated with six breaths of 100% oxygen and the colour of the indicator during full expiration was matched to one of the three colour ranges illustrated on the detector (table).

The detector indicated a concentration of <0.3% in

Colour shown by end tidal carbon dioxide detector after six breaths of pure oxygen in 30 patients being resuscitated

Colour	End tidal carbon dioxide indicated (%)	No of patients	Events noted
Purple	<0.3	3	Endotracheal tube misplaced in two patients (oesophageal intubation). Ineffective precordial compression in third case
Light purple	0.5-1	8	Two patients successfully resuscitated; in both cases return of spontaneous circulation was associated with change in colour to yellow
Yellow	>2	19	Spontaneous circulation had returned at time of assessment in six patients; resuscitation attempts failed in 13 patients

three patients. In two of these the tube had been placed in the oesophagus; when the tube was correctly positioned under direct vision the detector changed colour to yellow, indicating a concentration of >2%. In the third patient correct placement of the tube was confirmed by direct vision and auscultation. The cardiac compression was immediately altered and the detector changed colour to light purple, indicating a more effective circulation.

In two other patients the end tidal carbon dioxide concentration spontaneously increased without any apparent alteration in the efficiency of cardiac compression or ventilation. The increase was associated with a return of spontaneous circulation; during inspiration the detector returned to a purple colour (<0.3% carbon dioxide), indicating that the effect was not due to the patient rebreathing expired gas.

Comment

The detector successfully differentiated between oesophageal and tracheal intubation in every case. It is important to note that making a clinical decision before administering at least six breaths through the detector can yield false results. If the stomach is distended with air before intubation carbon dioxide concentrations may be as high as 4.5%. Ventilation with six breaths of pure oxygen will, however, reduce the carbon dioxide concentration in the oesophagus to nearly zero.³

We identified one case in which ineffective cardiac compression produced inadequate pulmonary blood flow. Smalhout and Kalenda reported a similar case, which they attributed to chest compressions being less vigorous than necessary because the person performing resuscitation was tired.⁴ A spontaneous increase in end tidal carbon dioxide concentration was first linked with the return of a patient's circulation by Garnett *et al*.⁵ Our study confirmed this finding and showed that precordial compression need not be interrupted to confirm that spontaneous circulation has been restored. We believe that the end tidal carbon dioxide detector provides a valuable aid in managing cardiopulmonary resuscitation and should be available at all sites where such resuscitations are attempted.

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