Comment

In this study a simple comparison between patients who had and had not received transfusions suggested that transfusion has a significant ($p=0.04$) adverse effect on survival time. When adjustments were made for other prognostic factors, notably the tumour pT stage, however, it became obvious that transfusion was not a significant predictor of survival in patients with renal cell carcinoma.

These results do not confirm the results found in patients with colorectal cancer. We suggest that other cancers should be assessed along similar lines.

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Department of Urology, Leicester General Hospital, Leicester LE5 4PW

I T MANYONDA, BSC, MB, senior house officer
D E SHAW, BSC, MSC, medical statistician
A FOULKES, MB, FRCS, senior house officer
D E OSBORN, MS, FRCS, consultant urological surgeon

Correspondence to: Mr Osborn.

Industrial exposure to hydrogen cyanide: implications for treatment

We had the opportunity of studying nine patients exposed to hydrogen cyanide in a single incident at an industrial plant and three other patients exposed in separate incidents. We believe that our observations have important implications for treatment of such patients.

Patients and outcome

A suspected leak from a valve allowed hydrogen cyanide to escape. Nine men developed symptoms compatible with cyanide toxicity, of whom three lost consciousness. None of the men had definitely identified the "bitter almonds" smell said to be characteristic of hydrogen cyanide. The symptoms experienced included lightheadedness (eight men), breathlessness (eight), feeling shaky (six), headache (four), and nausea (four). The three unconscious men rapidly recovered consciousness after being moved from the area where they had been working. All nine men were attended by the works medical officer on site and then taken to hospital. All patients either showered or bathed, and oxygen was administered. Arterial blood was sampled and whole blood samples analysed for cyanide¹ (table).

Mean pulse on admission was 104 (SEM 4) beats/min, mean systolic blood pressure 140 (8) mm Hg, and mean diastolic blood pressure 84 (5) mm Hg. These figures, however, include the values found in three patients who showed relative hypotension—that is, systolic blood pressure 20-30 mm Hg below its normal value (nadir values 90, 100, and 110 mm Hg)—for up to three hours after admission. These three men included two of those who had been unconscious. Four patients had headache that persisted for up to eight hours after admission. There was no clearcut relation between blood cyanide concentrations on

Biochemical findings in patients exposed to hydrogen cyanide

Case No	pH	Carbon dioxide tension (kPa)	Bicarbonate (mmol/l)	Oxygen tension (kPa)	Cyanide ($\mu\text{mol/l}$)	
					On admission to hospital	One month after incident
1*†‡	7.40	3.2	19	34.3	115	11
2*	7.44	3.7	23	16.3	130	6
3*†‡	7.34	3.2	24	49.1	104	5
4	7.35	5.1	22	12.3	35	5
5	7.41	4.0	22	11.1	57	5
6	7.42	4.7	24	11.2	96	7
7†	7.40	4.1	22	11.2	50	9
8	7.37	5.1	23	10.4	67	3
9	7.38	4.7	23	9.3	41	3

*Patients initially unconscious.

†Patients with relative hypotension.

‡Patients receiving high flow oxygen when blood was drawn.

Conversion: SI to traditional units—Oxygen and carbon dioxide tensions: 1 kPa $\approx$ 7.5 mm Hg. Bicarbonate: 1 mmol/l = 1 mEq/l. Cyanide: 1 $\mu\text{mol/l}$ $\approx$ 2.7 $\mu\text{g/100 ml}$.

admission and symptoms, although the three patients who had lost consciousness had the highest concentrations (table).

Three other men were also treated in separate incidents between 1970 and 1984 after exposure to hydrogen cyanide. Two were unconscious, although both regained consciousness rapidly. Cyanide concentrations in blood taken about 30 minutes after exposure were 284 and 173 $\mu\text{mol/l}$ (768 and 467 $\mu\text{g/100 ml}$) in the patients who lost consciousness and 59 $\mu\text{mol/l}$ (160 $\mu\text{g/100 ml}$) in the other patient.

Comment

All of our patients had absorbed appreciable amounts of cyanide, as shown by their blood cyanide concentrations. One of the problems in interpreting previous reports is that blood concentrations were measured in only a few patients after short term exposure to hydrogen cyanide, experience at Billingham being the exception (D D Bryson, personal communication). It has been suggested that cyanide concentrations above 111 $\mu\text{mol/l}$ (300 $\mu\text{g/100 ml}$) are potentially lethal,² yet four of our patients had values above this. Thus blood cyanide concentrations are not accurate in predicting severity of toxicity and should be used only to confirm that appreciable cyanide absorption has occurred, not to determine treatment.

The most important lesson to be learnt is that patients can recover spontaneously and quickly from apparently severe poisoning with hydrogen cyanide, as shown by the five patients who lost consciousness: their conscious level had largely returned even in the short time before the arrival of the works medical officer (probably less than 10 minutes). This emphasises the importance of making the decision to give antidotes to cyanide on clinical grounds and on the basis of the patient's changing condition, particularly his conscious level.³ Our experience suggests that if the patient has been removed from the area of exposure and further absorption of hydrogen cyanide prevented then even if he is unconscious an antidote does not have to be administered immediately unless the vital signs are deteriorating. Caution in giving antidotes to cyanide, particularly dicobalt edetate, the most popular antidote in the United Kingdom, is advisable because of their potential toxicity.^{3,4} Clearly, appropriate supportive care must be begun immediately: several severely poisoned patients who have ingested potassium cyanide have recovered after being given full supportive measures but no antidote.^{2,5} Most probably any patient exposed to hydrogen cyanide who reaches hospital fully conscious will require merely observation and reassurance.

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Medical Unit, Falkirk and District Royal Infirmary, Falkirk FK1 5QE

N R PEDEN, MRCP, consultant physician
A TAHA, MRCP, medical registrar
P D MCSORLEY, MRCP, consultant physician

BP Chemicals Ltd, Grangemouth

G T BRYDEN, MB, CHB, works medical officer
I B MURDOCH, MRCP, works medical officer
J M ANDERSON, PHD, chief works chemist

Correspondence to: Dr Peden.

Haem arginate in the treatment of acute hepatic porphyrias

Haematin has been used for 15 years to treat acute porphyric attacks. Its disadvantages are instability in solution, harmful effects on coagulation, and thrombophlebitis.^{1,2} For these reasons safer, more stable preparations have been developed. We report the effects of haem arginate, a new haem preparation, in patients with acute intermittent porphyria and variegate porphyria.