there is no standard doctor. Might not reluctance to discuss the problem represent an understandable failing in the *physician* to come to terms with the illness?

In our experience there is no substitute for the truth when communicating in medicine and it should very rarely be necessary to tell lies to patients. What is required is the ability to explain the nature of the problem clearly and sympathetically in terms that the patient and the relatives can understand and accept. A series of interviews is necessary and it is particularly helpful when husband and wife (or a close relative) can share in some of the discussion.

We all know of the pain and suffering caused by acute leukaemia and these can extend far beyond the patient to affect relatives, loved ones, doctors, nurses, medical students, and anyone with whom the patient has contact. When communication has been successful the mututal atmosphere of fear, suspicion, and mistrust which ignorance produces can be dispelled and everyone concerned can concentrate on looking after the patient.

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Pneumonia during treatment of acute leukaemia

SIR,—We read with interest the comments of Professor F G J Hayhoe and Dr J K N Rees (6 December, p 1566) on your leading article entitled "Pneumonia during the treatment of acute leukaemia" (8 November, p 1235).

While we wholeheartedly agree with them that an intimate knowledge of the pathogens in the hospital environment in question is essential and at least as important as knowing the site of colonisation, our experience with the site of infection is in complete contrast with theirs. At St Bartholomew's Hospital, pneumonia is both the most frequently documented manifestation of bacterial infection and the commonest cause of death in patients receiving remission induction therapy for acute myelogenous leukaemia (AML). Tobias *et al*[1] found 105 episodes of pulmonary infection, of which 32 were fatal, compared with 55 documented septicaemias (of unknown source), of which 21 were fatal, in an analysis of 200 patients with AML treated at St Bartholomew's Hospital from 1971 to 1975. A subsequent analysis (not yet published) confirms these results. Three hundred and ninety febrile episodes occurred in 168 patients with AML treated from November 1974 to May 1980. Eighty-five of these episodes were caused by pneumonia and 23 were fatal, whereas septicaemia was recorded in 42 patients and was fatal in 12.

The frequency with which chest infection occurs and our belief (shared by Professor Hayhoe and Dr Rees) that it is essential to know the infecting organism as early as possible prompted us to introduce transtracheal aspiration into the investigation of suspected pneumonia in 1979.[2] Experience with this safe and simple technique has shown that even in the absence of abnormalities on chest radiograph a pathogen can often be isolated when either sputum is unobtainable or culture is negative. It is hoped that this technique, by providing an early bacteriological diagnosis, will lead to a reduction in the incidence of fatal pulmonary infection.

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[1] Tobias JS, Wrigley PFM, O'Grady F. *Europ J Cancer* 1978;14:383-91.
[2] Slevin ML, Lowes JA, Bell R, *et al. Leukaemia Research* (in press).

Paracetamol—induced hepatic failure

SIR,—The paper from the King's College Hospital liver unit by Dr J Canalese and others (17 January, p 199) highlights the risk of severe hepatic damage and death from paracetamol in those patients who present to hospital at a time when specific antidotes such as *N*-acetylcysteine (NAC) or methionine may be ineffective. We have recently reviewed the outcome of 67 patients, referred to the poisons unit of Guy's Hospital from 1975 to 1979, who had been admitted to hospital later than 10 hours following an overdose of paracetamol and had received supportive treatment only.

Plasma paracetamol concentrations were measured in each case and all but two patients were in the so-called "high-risk" category defined by Prescott *et al*.[1] Fifty-one patients (76%) developed severe hepatic damage (maximum serum aspartate transaminase (AST) > 1000 IU/l), of whom 10 (15%) died. As a result of this high incidence of severe or fatal hepatic damage we have since carried out charcoal haemoperfusion on eight patients presenting to hospital later than 10 hours following a large paracetamol overdose, in the hope that the removal of further quantities of the unchanged drug, even at a late stage, might confer some therapeutic benefit. Details of some of these patients have recently been reported[2]; the toxicological and haemoperfusion data and the maximum recorded serum AST levels are shown in the table. In all cases there was a rapid fall in the plasma paracetamol concentrations during haemoperfusion and no complications resulted from this procedure. In some instances significant amounts of the drug were removed (cases 4, 5, 7, and 8). Severe hepatic damage occurred in one patient and another died despite further haemoperfusion for acute hepatic failure; the remainder developed only minor disturbances of liver function.

Since the efficacy of NAC in preventing paracetamol-induced liver damage diminishes after eight hours and is completely absent after 15 hours we consider that charcoal haemoperfusion was justifiable in this situation. With supportive treatment alone Dr Canalese and his colleague have shown that mortality in "high-risk" patients may be as great as 68%, and accordingly we are planning a prospective clinical trial of charcoal haemoperfusion versus supportive therapy in patients who present late (after 15 hours) after ingesting potentially hepatotoxic quantities of paracetamol.

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[1] Prescott LF, Illingworth RN, Critchley JAH, Stewart MJ, Adam RD, Proudfoot AT. *Br Med J* 1979; ii:1097-100.
[2] Winchester JF, Gelfand MC, Helliwell M, Vale JA, Goulding R, Schreiner GE. *Arch Int Med* (in press).

SIR,—I read with interest the recent article by Dr J Canalese and colleagues (17 January, p 199) discussing factors contributing to mortality in paracetamol-induced hepatic failure. The authors warn that delay in administration of hepatoprotective agents in cases of suspected severe paracetamol overdose while awaiting the results of plasma paracetamol concentrations may adversely affect the ultimate prognosis. They advocate the immediate administration of protective drugs until the plasma paracetamol concentration is known. Since initial suspicion of "severe" paracetamol poisoning is relatively common in clinical practice such a course of action will inevitably result in a large number of patients receiving inappropriate therapy. However, the toxicity of paracetamol in overdosage in the adult population is such that their advice is probably justified.

I would caution against the same clinical approach in the paediatric age group. Paracetamol ingestion is common in children, who accounted for 18% of inquiries regarding paracetamol poisoning received by the London centre of the British National Poison Information Service during 1975.[1] There is a significant lack of correlation between the reported amount ingested and subsequent plasma levels.[2] In addition, hepatic toxicity is usually mild even with plasma paracetamol levels commonly lethal in adults. Elimination kinetic studies of paracetamol metabolism in children[3] indicate relatively more sulphate than glucuronide formation, but how this relates to the apparent resilience of the liver in children is unclear.

The adverse effects of these antidotes can be significant and experience of their use in children is limited. In view of the difference in tolerance, these drugs should be withheld in children until the plasma paracetamol level

Details of eight patients treated with haemoperfusion after paracetamol overdose

Patient No	Age (y)	Dose (g)	Plasma paracetamol concentration (mg/l) (hours after ingestion)	Duration of haemoperfusion (h)	Amount of paracetamol removed (mg)	Maximum serum AST (IU/l)
1	42	30	262 (14)	5	837	349
2	69	?	245 (16)	5	380	327
3	24	24	178 (11·5)	4·5	365	30
4	69	?	150 (13)	6	4747	393
5	30*	50	250 (18)	10	6699	2920
6	49	75	149 (24)	5	742	401
7	27	30	174 (24·5)	4	3129	343
8	21	50	169 (16·5)	2·5	1966	>1000†

AST = aspartate transaminase.
*Patient died of fulminant hepatic failure.
†Dilution of serum not performed.