(3) How often do you drink medium strength beer/strong beer?†	
More or less daily	(7·0)
Some time during week	(1·5)
More seldom	(0·5)
Never	
(4) How much do you drink when you drink beer?	
3 Cans (450 ml) or more	(135·0)
2 Cans	(90·0)
1 Can	(45·0)
A glass	(25·0)
Never drink beer	
(5) How often do you drink wine/strong wine?‡	
More or less daily	(7·0)
Some time during week	(1·0)
More seldom	(0·5)
Never	
(6) How much do you drink when you drink wine?	
1 Bottle (750 ml) or more	(75·0)
½-1 Bottle	(58·0)
½ Bottle	(38·0)
A glass	(20·0)
Never drink wine	

*Mean alcohol content 39·9% (by volume).
†Mean alcohol content 4·3% (by volume).
‡Mean alcohol content 13·5% (by volume).

References

1 Ledermann S. *Alcohol, alcoolisme alcoolisation. Donnes scientifiques de caractere physiologique, economique et social.* Paris: Presses Universitaires de France, 1956.
2 Blackwelder WC, Katsuhiko Y, Rhoads GG, Kegan A, Gordon T, Palesch Y. Alcohol and mortality: the Honolulu heart study. *Am J Med* 1980;**68**:164-9.
3 Gordon T, Kannel WB. Drinking and mortality: the Framingham study. *Am J Epidemiol* 1984; **120**:97-107.
4 Marmot MG. Alcohol and coronary heart disease. *Int J Epidemiol* 1984;**13**:160-7.
5 Sundby P. *Alcoholism and mortality*. Oslo: Universitetsforlaget, 1967.
6 Peterson B, Kristenson H, Ternby NH, *et al.* Alcohol consumption and premature death in middle aged men. *Br Med J* 1980;**280**:1403-6.
7 Cullen K, Stenhouse NS, Wearne KL. Alcohol and mortality in the Busselton study. *Int J Epidemiol* 1982;**11**:67-70.
8 Kozarevic D, Vojvodic N, Gordon T, *et al.* Drinking habits and death: the Yugoslavia cardiovascular disease study. *Int J Epidemiol* 1983;**12**:145-55.
9 Eckhardt MJ, Harford TC, Kaelber CT, *et al.* Health hazards associated with alcohol consumption. *JAMA* 1981;**246**:648-66.
10 Marmot MG, Rose G, Shipley MJ, Thomas BJ. Alcohol and mortality: a U-shaped curve. *Lancet* 1981;i:580-3.
11 Tomacho TC, Kaplan GA, Cohen RD. Alcohol consumption and mortality in Alameda county. *J Chronic Dis* 1987;**40**:229-36.
12 Agrell J. *Missbruk av narkotika, thinner och läkemedel bland inskrivningsskyldiga 1967-1970/71.* Stockholm: Militärpsykologiska Institutet, 1972. (A-rapport nr 15.)
13 Hollstedt C, Rydberg U. Hazardous alcohol consumption and early diagnosis of alcohol linked diseases. *Lakartidningen* 1981;**78**:795-9. (English summary.)
14 Miettinen OS. *Theoretical epidemiology: principles of occurrence research in medicine.* New York: John Wiley and Sons, 1985.
15 Orrego H, Bellendis LM, Blake JE, Kapur BM, Israel Y. Reliability of assessment of alcohol intake based on personal interviews in a liver clinic. *Lancet* 1979;ii:1354-6.
16 Poikoeainen K, Kärkkäinen P, Pikkarainen J. Correlations between biological markers and alcohol intake as measured by diary and questionnaire in men. *J Stud Alcohol* 1985;**46**:383-7.
17 Watson CG, Tilleskjor C, Hoodesheck-Schow BA, Pucel J, Jacobs L. Do alcoholics give valid self-reports? *J Stud Alcohol* 1984;**45**:344-8.
18 Popham RE, Schmidt W. Words and deeds: the validity of self-report data on alcohol consumption. *J Stud Alcohol* 1981;**42**:355-9.
19 Benson G, Holmberg MB. Validity of questionnaires in population studies on drug use. *Acta Psychiatr Scand* 1985;**70**:9-18.
20 Rydelius PA. Alcohol abusing teenage boys. *Acta Psychiatr Scand* 1983;**68**:368-80.
21 Rajs J, Jakobsson SV. Cause of death in persons aged between 15 and 50 years in the community of Stockholm. A forensic-pathologic and statistical study. *Forensic Sci Int* 1985;**29**:213-26.
22 Abel EL, Zeidenberg P. Age, alcohol and violent death: a postmortem study. *J Stud Alcohol* 1985;**46**:228-31.
23 Wilhelmsen L, Elmfeldt D, Wedel H. Cause of death in relation to social and alcoholic problems among Swedish men aged 35-44 years. *Acta Med Scand* 1983;**213**:263-8.
24 Wall S, Rosen M, Nyström L. The Swedish mortality pattern: a basis for health planning? *Int J Epidemiol* 1985;**14**:285-92.
25 Romeder I-M, McWhinnie JR. Potential years of life lost between ages 1 and 70; an indicator of premature mortality for health planning. *Int J Epidemiol* 1977;**6**:143-51.
26 Brook DW, Brook JS. Adolescent alcohol use. *Alcohol and Alcoholism* 1985;**20**:259-62.
27 Schuckit MA. Genetics and the risk for alcoholism. *JAMA* 1985;**254**:2614-7.
28 Goodwin DW. Alcoholism and genetics. *Arch Gen Psychiatry* 1985;**42**:171-4.
29 Vaillant GE. *The natural history of alcoholism: causes, patterns and paths to recovery.* Cambridge, Mass: Harvard University Press, 1983.

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Prognosis of patients receiving intensive care for lifethreatening medical complications of haematological malignancy

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Abstract

The mortality of patients admitted to intensive care units with haematological malignancy is high. A humane approach to the management of the critically ill as well as efficient use of limited resources requires careful selection of those patients who are most likely to benefit from intensive care. To delineate more accurately the factors influencing outcome in these patients the records of 60 consecutive admissions to the intensive care unit (37 male, 23 female) with haematological malignancy were reviewed retrospectively. Fifty patients were in acute respiratory failure, most commonly (34 patients) with a combination of pneumonia and septicaemic shock. The severity of the acute illness was assessed by the APACHE II (acute physiology and chronic health evaluation II) score and number of organ systems affected. Thirteen patients survived to leave hospital. The mortality of patients with haematological malignancy was consistently higher than predicted from a large validation study of APACHE II in a mixed population of critically ill patients. Moreover, no patient with an APACHE II score of greater than 26 survived. Mortality among the 22 patients with relapsed malignancy (21 deaths), was significantly higher than among the 35 patients at first presentation (26 deaths). On discharge from the intensive care unit all survivors had responded well to chemotherapy and had normal or raised peripheral white cell counts. They included seven patients who had recovered from leucopenia (white cell count $<0{\cdot}5\times10^9$/l). In contrast, 36 of the 47 patients who died were leucopenic at the time of death.

The overall mortality of critically ill patients with haematological malignancy is higher than equivalently ill patients without cancer. The dysfunction of an increasing number of organ systems, an APACHE II score of greater than 30, failure of the malignancy to respond to chemotherapy, and persistent leucopenia all point to a poor outcome.

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Introduction

Recent developments in the use of very myelosuppressive cytotoxic chemotherapy, as well as advances in supportive care, have considerably improved the prognosis of many patients with haematological malignancy, and a large proportion can now be cured.[1] These

patients, however, are highly susceptible to overwhelming infection (usually pneumonia or septicaemia, or both), particularly during the period of profound leucopenia induced by aggressive chemotherapy.[2] If infection persists further deterioration may often be accompanied by multiple organ failure. Occasionally an acute lifethreatening illness may be due to a direct effect of the malignant process itself, or drug toxicity, on vital organ function, while in others associated thrombocytopenia may precipitate dangerous haemorrhage. Usually such acute complications are potentially reversible, and in view of the improved long term outlook for these patients it is appropriate to consider intensive care for selected cases.

TABLE I—*Patient population*

Disease	No of patients	Age (years)		APACHE II score		No of systems affected	
		Median	Range	Mean	Range	Median	Range
Acute myeloid leukaemia	22	51·0	15-69	31·5	14-48	5	2-7
Acute lymphatic leukaemia	15	38·0	20-61	25·2	14–36	3	2-7
Chronic myeloid leukaemia	2	—	30, 65	—	24, 30	—	2, 5
Multiple myeloma	2	—	52, 70	—	21, 37	—	5, 5
Hodgkin's lymphoma	5	30·0	19-48	26·6	24-35	4	3-6
Non-Hodgkin's lymphoma	14	48·5	28-70	27·7	12-44	4	3-6

TABLE II—*Numbers (percentages) of patients in each stage of treatment of malignancy*

Disease	First presentation	In relapse	In remission
Acute lymphatic leukaemia	8 (53)	5 (33)	2 (13)
Acute myeloid leukaemia	17 (77)	4 (18)	1 (5)
Chronic myeloid leukaemia	2 (100)	0	0
Multiple myeloma	1 (50)	1 (50)	0
Hodgkin's lymphoma	1 (20)	4 (80)	0
Non-Hodgkin's lymphoma	6 (43)	8 (57)	0
Total	35 (58)	22 (37)	3 (5)

As might be anticipated, however, mortality is high among patients with leukaemia or lymphoma who develop an acute illness severe enough to warrant admission to an intensive care unit, particularly those with respiratory failure.[3-8] Moreover, intensive care is expensive, particularly for non-survivors,[9] and may be distressing for patient and relatives. Both for a humane approach to the management of malignant disease and to ensure that limited resources are used appropriately it is important to select those patients most likely to benefit from intensive care and to limit further aggressive treatment when the outlook is clearly hopeless.[10] These decisions must be based on the best possible understanding of the factors determining both the immediate and long term outcome in the various categories of patient.

A preliminary report confirmed that hospital mortality was high in these patients (82% in our series[11]) and suggested that dysfunction of an increasing number of organ systems, failure to recover bone marrow function after chemotherapy, and unresponsive malignancy all indicated a very poor prognosis. We have now analysed a larger number of patients to delineate more clearly those factors that most influence outcome and in particular to establish the relation between the severity of the acute illness (as assessed by the acute physiology and chronic health evaluation II (APACHE II) score[12]) and survival in this category of patient.

Patients and methods

Patients referred to the intensive care team by the oncologists were assessed jointly on the ward before admission. Those with a lifethreatening acute illness which had failed to respond to conventional measures (supplemental oxygen, chest physiotherapy, antibiotics, and expansion of circulating volume) were considered for admission provided that there was a reasonable prospect of cure or worthwhile palliation. Patients with malignant disease admitted for routine postoperative care, children, and those with complications related to solid tumours were excluded from this survey.

ANALYSIS

The records of all eligible patients admitted to intensive care during a five year period were reviewed retrospectively. The severity of the acute illness was assessed by the APACHE II score, calculated from the most abnormal variables recorded during the first 24 hours of admission.[12] The number of major systems affected was also noted. Patients were categorised as non-survivors (death in the intensive care unit or after discharge to the general ward) and survivors (discharged from hospital). Parametric statistics were employed in the analysis of APACHE II scores and non-parametric methods used for other comparisons. Results were considered to be significant when $p<0·05$.

TABLE III—*APACHE II scores, numbers of systems affected, and outcome*

	APACHE II score			No of systems affected	
	Mean	Range	95% Confidence limits	Median	Range
Intensive care unit deaths	32·5*	20-48	30, 35	5	3-7
Ward deaths	23·0*	14-40	17, 29	3	2-4
Total hospital deaths	30·6**	14-48	28, 33	5***	2-7
Hospital survivors	20·2**	12-26	17, 23	3***	1-4

*$p<0·01$; **$p<0·001$ (t test); ***$p<0·001$ (Mann-Whitney U test).

PATIENTS

Sixty patients (37 male, 23 female) representing six disease categories were admitted during the five years (table I). There was a large range of both APACHE II scores (12-48) and number of organ systems affected (two to seven). Fifty patients were in acute respiratory failure, defined as either an A-aPO_2 of >47 kPa or a $PaCO_2$ of >8 kPa or a combination of both. Of these 50 patients, nine had pneumonia alone, three pneumonia and pneumothoraces which had occurred after endoscopic transbronchial biopsy, 34—that is, more than half of all patients in the series—both pneumonia and

septicaemic shock (defined as systolic blood pressure <80 mm Hg, oliguria, altered mentation, and clinical evidence of sepsis), three upper respiratory tract obstruction, and one both pneumonia and a myocardial infarction. Respiratory failure was not present in 10 patients at the time of admission to intensive care. Of these patients, five had septicaemic shock, one had pneumonia, one was admitted after a cardiorespiratory arrest, one had uncontrollable haemorrhage, one was in acute renal failure, and one had both impaired consciousness due to cerebral lymphoma and pneumonia.

Fifty eight patients had received cytotoxic chemotherapy before admission. The remaining two, who were critically ill on arrival, were given chemotherapy in the intensive care unit. Thirty five patients were receiving their induction course of chemotherapy, and 22 had relapsed and were receiving further induction chemotherapy. Only three patients were in established remission (table II).

Thirty eight patients were admitted between 10 and 25 days after the last course of chemotherapy (median 16 days, range 0-51). This corresponds with the time of most severe marrow suppression.

Results

Thirteen patients left hospital alive (long term survivors). Forty seven patients died in hospital, of whom 38 died in the intensive care unit and nine died shortly after discharge to the general ward. In this second group the longest period of survival was 20 days, eight patients having died within a week of discharge from the intensive care unit. Ten patients died within 24 hours of admission to intensive care.

Survival and APACHE II score—None of the 28 patients with an APACHE II score greater than 26 survived to leave hospital (table III). The mean score of patients who died in hospital (30·6; SD 8·2) was significantly higher than that of patients who were discharged alive (20·2; SD 5·2) ($p<0·001$). The subgroup of nine patients who died shortly after discharge from the intensive care unit had a mean APACHE II score significantly lower than that of patients who died in the unit but not significantly different from that of survivors ($p<0·01$ and $p>0·05$ respectively). Within each of six score bands (fig 1) the mortality in this series of patients with haematological malignancy was consistently higher than that predicted from a large validation study of the APACHE II system in a mixed population of critically ill patients.[12]

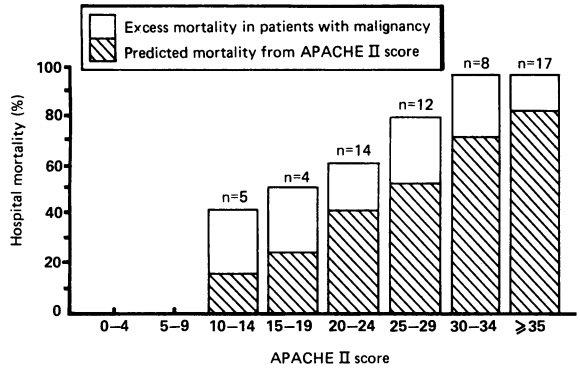

FIG 1—Hospital mortality with increasing APACHE II score from validation study by Knaus *et al*[12] and mortality of patients with malignancy investigated in this study. Number of patients in each score band shown.

Survival and numbers of systems affected—Patients who died in hospital had significantly more systems affected (median five; range two to seven) compared with those who were discharged alive (median three; range one to four) (Mann-Whitney U test, $p<0·001$; table III). There was, however, no significant difference in numbers of systems affected between the subgroup of patients who died after discharge from the intensive care unit and the long term survivors.

Survival and underlying malignancy—Only four of the 22 patients with acute myeloid leukaemia and two of the 14 with non-Hodgkin's lymphoma survived their acute illness to be discharged from hospital, whereas five of the 15 patients with acute lymphatic leukaemia survived. This difference, however, was not significant. There was no significant difference in severity of the acute physiological disturbance, as measured by the APACHE II score, between those patients who had relapsed (mean 28·0; SD 7·8) and those who had presented for initial chemotherapy (mean 29·0; SD 9·5). Nevertheless, nine of the 35 patients admitted during their first presentation became long term survivors, whereas only one of the 22 patients who had relapsed survived to leave hospital. This difference was significant (χ^2 test with Yates's correction, $p<0·05$). All three patients who were in remission at the time of their acute illness survived to be discharged from hospital.

Survival and acute illness—Nine of the 50 patients who were in respiratory failure at the time of admission survived to leave hospital. In contrast, four of the 10 patients who were not in respiratory failure were discharged alive. This second group included the three patients who were in remission but receiving maintenance chemotherapy. One patient in remission had uncontrollable bleeding due to thrombocytopenia; of the other two, who had comparatively low white cell counts, one developed pneumonia and the other septicaemic shock. The combination of pneumonia and septicaemic shock was associated with a very high mortality (32 deaths among the 34 patients). Pneumonia with or without other complications but excluding septicaemia had a much lower mortality (nine of 14 patients died). This difference was highly significant (χ^2 test with Yates's correction, $p<0·01$). Positive microbiological identification of an organism did not improve the outcome compared with those patients who had clinical evidence of sepsis but in whom no positive cultures were obtained. Forty five patients required intermittent positive pressure ventilation. Of the 47 patients who died in hospital, 39 required mechanical ventilation, whereas of the 13 survivors, only six did so. This difference was significant (χ^2 test with Yates's correction, $p<0·01$). Two survivors required lengthy periods of intermittent positive pressure ventilation (nine and 13 days respectively). The 39 patients who required inotropic agents had a high mortality (35 deaths); only three of the 30 patients who received inotropic doses of either dopamine or dobutamine were discharged from hospital alive. There were no survivors in the group given adrenaline.

Survival and white cell count—Thirty nine patients were leucopenic on admission to the intensive care unit, with a total white cell count of less than $1·0\times10^9$/l and an absolute neutrophil count of less than $0·5\times10^9$/l. A further seven patients became leucopenic during their stay in the unit (table IV). At the time of death in the intensive care unit 31 out of 38 patients either remained or had become leucopenic. Two of the seven who were not leucopenic when they died had recovered their bone marrow function after a period of leucopenia, while the others had reasonable white cell counts throughout. Of the nine patients who died in the general ward shortly after discharge from the intensive care unit, five were leucopenic at the time of death; only one of the nine had recovered a reasonable peripheral white cell count, the remaining three patients having had good marrow function throughout their acute illness. Thus altogether 36 of the 47 patients who died in hospital were leucopenic at the time of death. One of the three patients who regained marrow function died unexpectedly some time after discharge from intensive care as a result of a pulmonary embolus; the other two died from irreversible multisystem failure which developed during their period of leucopenia (17 and 21 days). By contrast, all patients who survived to leave hospital had normal white cell counts at the time of discharge from the intensive care unit. They included seven of the 13 who recovered from leucopenia to have normal counts.

TABLE IV—*Survival and white cell count*

	Hospital deaths	Hospital survivors
Total	47	13
Leucopenic on admission to intensive care unit	33	6
Leucopenic at death in or discharge from intensive care unit	36	0
Normal white cell count at death	11	—
Recovered white cell count	3	7

Discussion

In this series of 60 consecutive adult admissions to intensive care for lifethreatening complications of haematological malignancy, only 13 patients (22%) were discharged from hospital alive. This is similar to the 18% survival reported in our previous study[11] and comparable to that reported by other workers in patients with various malignancies who required admission to intensive care.[3-6] The proportion of patients who died shortly after discharge from intensive care, however, had fallen from 27%[11] to 15%. Most of these patients had further aggressive supportive treatment withdrawn when it became clear that the underlying malignancy had failed to respond to cytotoxic chemotherapy. The fall in the

proportion of these cases in our present series may reflect a more selective admission policy.

In this group of immunocompromised patients respiratory failure, usually due to pneumonia, remained the most common acute illness precipitating admission to intensive care and was associated with a very high mortality (41 deaths among 50 patients; 82%), similar to that (80-96%) reported in other series.[5 7] Johnson and coworkers found a mortality of 93·5% in granulocytopenic patients who developed respiratory failure. In our series the mortality in leucopenic patients who developed respiratory failure was 86% (29 deaths among 34 patients). Pneumonia with septicaemic shock was associated with a particularly poor outcome (32 of 34 patients died). The mortality of patients who required inotropic support was high and there were no survivors in the group given adrenaline. None of the patients with pneumonia and septicaemia who received inotropes survived. Nevertheless, in those with respiratory failure due to pneumonia with or without other complications but excluding septicaemia the mortality was 64% (nine deaths among 14 patients).

Schuster and Marion reported that the four patients (8%) who survived a period of intermittent positive pressure ventilation in their series had "predictably reversible conditions," whereas all their patients with bilateral pulmonary infiltrates died.[5] In contrast, the pulmonary abnormality in the six patients who survived a period of mechanical ventilation in this series could not be distinguished from that in patients who died—namely, pneumonia and diffuse pulmonary infiltrates in *x* ray films. In the survivors, however, respiratory failure was not associated with extensive multisystem failure. Thus in the 14 patients who had acute respiratory failure requiring mechanical ventilation but in whom three or fewer organ systems were affected the mortality was 57% (eight deaths). Conversely, when four or more systems were affected none of the patients with respiratory failure needing artificial ventilation survived. Indeed, those who survived a period of intermittent positive pressure ventilation had at most three system disease and consequently a lower mean APACHE II score (20·8; SD 5·6) than those who died (32·2; SD 7·5) ($p<0·001$). The need for artificial ventilation does not of itself imply a poor prognosis. The progress of the underlying malignancy and severity of the acute illness associated with acute respiratory failure are the determinants of outcome.

It has also been suggested that failure to respond rapidly (within five days) to intermittent positive pressure ventilation is indicative of a poor outcome.[5] Survival, however, is not precluded by a requirement for lengthy periods of mechanical ventilation. Two patients (one with acute lymphatic leukaemia, one with acute myeloid leukaemia) required ventilation for nine and 13 days respectively because of respiratory failure associated with diffuse bilateral pulmonary infiltrates in the chest *x* ray film. During prolonged mechanical ventilation there was no deterioration in the function of their other organ systems. Both were recovering from the nadir of effective chemotherapy and were therefore able to mount a satisfactory granulocyte response to their infection.

Johnson *et al* found that overall mortality in a population of granulocytopenic patients with haematological malignancy receiving intensive care or ward based care did not correlate with the duration of leucopenia.[8] Some of their patients did not have lifethreatening illness, and comparison with our series is therefore difficult. In contrast, all long term survivors in our study either had adequate neutrophil counts throughout or showed an appreciable recovery of bone marrow function during their stay in the intensive care unit. Six of the seven patients who survived after recovering from leucopenia had pneumonia (one pneumonia and septicaemia) and five were in respiratory failure. In two patients delayed recovery (17 and 21 days) of the peripheral neutrophil count allowed sepsis to persist with consequent multiple organ failure and death. The striking absence of marrow recovery in patients who died and recovery of the white cell count in seven of the 13 patients who survived despite similar underlying acute illness suggests that in patients who develop lifethreatening infection persistent granulocytopenia will result in an adverse outcome.

Knaus and colleagues have shown the value of the APACHE II scoring system in the prognostic stratification of groups of acutely ill patients.[12] The death rate within any given score band, however, may be influenced by other factors—for example, the nature of the underlying disease process.[13 14] In this series the mortality in patients with haematological malignancy was higher in every score band by a factor of not less than 20%, rising to 100% mortality in patients with scores greater than 30 (fig 1). No patient with an APACHE II score greater than 26 survived to leave hospital, and in the study of Johnson *et al* all patients with scores of more than 23 on admission to the ward or intensive care unit died. This is despite weighting the score in patients with haematological malignancy, who because of immunosuppression automatically score five additional points. Diseases which have poorly understood pathophysiology, such as respiratory failure from infection and septic shock, carry a higher than average mortality, even in patients without cancer.[13] Most of our patients fell into this disease category, yet, as illustrated by figure 2, their mortality was still higher than that of a general intensive care unit population with similar acute illness.

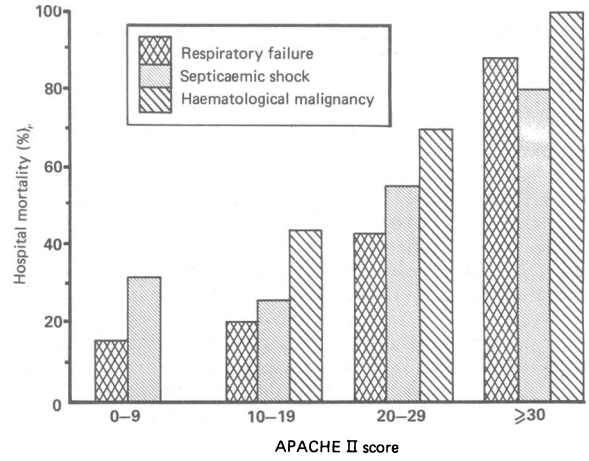

FIG 2—Hospital mortality for different disease groups with increasing APACHE II score from validation study by Knaus *et al*[12] and mortality of patients with haematological malignancy investigated in this study.

The interaction and coordination of staff in an intensive care unit influences the outcome in critically ill patients.[14] APACHE II scoring is not used routinely in this unit and an assessment of our performance in this respect is not possible. Nevertheless, our organisation and policies conform to those identified as associated with the optimisation of care.[14] Until larger multiunit surveys are undertaken we must assume that the increased mortality shown by our series is related to the underlying disease process. This assumption is supported by the similar overall mortality figures reported by other workers who have studied patients with haematological malignancy.[3-8]

The progress of the underlying malignancy is of considerable importance in determining the outcome of an acute lifethreatening illness. The prospects for cure are poor in patients who have relapsed or failed to achieve complete remission after an induction course of chemotherapy.[15 16] In this series the mortality of patients who had relapsed was higher than of those on first presentation (21 of 22 patients dead compared with 26 of 35), though the severity of their acute illness was similar. This difference was just significant at the 5% level. The survival of patients with acute lymphatic leukaemia was better than of those with acute myeloid leukaemia or non-Hodgkin's lymphoma. Because of small numbers this difference did not reach significance. All three patients in remission survived. Similarly, the APACHE II scores and numbers of systems affected in patients who died in the ward were not significantly different from those in patients who left hospital alive, confirming the importance of the underlying disease in determining ultimate outcome.

The overall mortality of critically ill patients with haematological malignancy is consistently higher than that of an equivalent population of patients without cancer. Nevertheless, for some

patients prolonged periods of artificial ventilation and lengthy stays in the intensive care unit may be life saving, most patients still being alive and in remission several years later. We suggest that an APACHE II score of greater than 30, the dysfunction of an increasing number of organ systems, failure to recover marrow function after chemotherapy, and unresponsive malignant disease, particularly in patients who have relapsed, are all indicative of a poor prognosis. Further aggressive supportive treatment in these patients may be inappropriate.

We thank Dr W A Knaus for helplful advice.

References

1 Bell R, Rohatiner AZS, Slevin ML, *et al.* Short term treatment for acute myelogenous leukaemia. *Br Med J* 1982;**284**:1221-30.
2 Schimpff SC. Infection in the leukemic patient: diagnosis, therapy and prevention. In: Gunz FW, Henderson ES, eds. *Leukemia*. New York: Grune and Stratton, 1983:799-822.
3 Goldiner P, Pinilla J, Turnbull A. Acute respiratory failure in patients with advanced lymphoma. In: Lacher MJ, ed. *Hodgkin's disease*. New York: John Wiley, 1976:371-6.
4 Hauser MJ, Tabak J, Baier H. Survival of patients with cancer in a medical critical care unit. *Arch Intern Med* 1982;**142**:527-9.
5 Schuster DP, Marion JM. Precedents for a meaningful recovery during treatment in a medical intensive care unit. *Am J Med* 1983;**75**:402-8.
6 Snow RM, Miller WC, Rice DL, Ali MK. Respiratory failure in cancer patients. *JAMA* 1979;**241**:2039-42.
7 Estopa R, Torres-Marti A, Kastanos N, Rives A, Agusti-Vidal A, Rozman C. Acute respiratory failure in severe hematologic disorders. *Crit Care Med* 1984;**12**:26-8.
8 Johnson MH, Gordon PW, Fitzgerald FT. Stratification of prognosis in granulocytopenic patients with hematological malignancies using the APACHE II severity of illness score. *Crit Care Med* 1986;**14**:693-7.
9 Turnbull AD, Graziano C, Baron R, Sichel W, Young C, Howland W. The inverse relationship between cost and survival in the critically ill cancer patient. *Crit Care Med* 1979;**7**:20-3.
10 Jennett B. Inappropriate use of intensive care. *Br Med J* 1984;**289**:1709.
11 Lloyd-Thomas AR, Dhaliwal HS, Lister TA, Hinds CJ. Intensive therapy for life threatening medical complications of hematological malignancy. *Intensive Care Med* 1986;**12**:317-24.
12 Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. *Crit Care Med* 1985;**13**:818-29.
13 Knaaus WA, Wagner DP, Draper EA. Relationship between acute physiologic derangement and risk of death. *J Chronic Dis* 1985;**38**:295-300.
14 Knaus WA, Draper EA, Wagner DP, Zimmerman JE. An evaluation of outcome from intensive care in major medical centres. *Ann Intern Med* 1986;**104**:410-8.
15 Lister TA, Rohatiner AZS. The treatment of acute myelogenous leukemia in adults. *Semin Hematol* 1982;**19**:172-5.
16 Horwich A, Peckham M. "Bad risk" non-Hodgkin's lymphomas. *Semin Hematol* 1983;**20**:35-56.

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Non-mydriatic Polaroid photography in screening for diabetic retinopathy: evaluation in a clinical setting

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Abstract

Because of fears that Polaroid colour prints produced with a non-mydriatic fundus camera may not detect important sight threatening lesions in diabetes a study was conducted comparing retinal images obtained on Polaroid prints taken in "field" conditions with those on 35 mm transparencies and fluorescein angiograms. Almost one in five (22/127) Polaroid prints could not be assessed owing to poor quality compared with 3 (2·4%) 35 mm transparencies and 2 (1·6%) fluorescein angiograms. The pick up rate of microaneurysms, haemorrhages, and hard and soft (cotton wool spots) exudates was equivalent for Polaroid prints and 35 mm transparencies of equivalent quality. In two cases with disc new vessels, however, these were not seen on the Polaroid prints.

The widespread use of Polaroid colour prints obtained with a non-mydriatic camera without the necessary operative and interpretive skills further limits the usefulness of the technique.

Introduction

Diabetes remains the leading cause of blindness in people of working age.[1] It is cheaper to detect and treat a patient with diabetic retinopathy than to look after a blind person.[2] Thus the argument for a cost effective screening programme is a strong one.[3] Polaroid non-mydriatic fundus photography has been advocated as a possible cheap and easy to use screening method in diabetes[4] but its value is controversial.[5-7] Nevertheless, it is increasingly being used clinically, with assessment of the photographs being performed primarily by non-ophthalmologists.

We have assessed the Polaroid prints of 127 eyes retrospectively in an attempt to gauge their usefulness in screening. The prints were compared with fundus photographs taken by using Ektachrome 64 transparencies through a dilated pupil of the same 127 eyes and fluorescein angiograms.

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Patients and methods

Patients were selected at random from the diabetic clinic or were newly diagnosed non-insulin-dependent diabetics. All photography was carried out in the medical illustration department at this hospital. Initially patients were photographed by any one of six medical photographers using the Canon CR3 NM (non-mydriatic) retinal camera without mydriasis and with Polaroid 779 instant print material. At no time were any of the photographers aware that their results would later be assessed in the study.

Patients were reviewed a week later, when retinal photographs with use of Kodak Professional Ektachrome 64 film were taken through pupils dilated with 0·5% tropicamide by using the Canon CR3 non-mydriatic camera. Before the colour films all patients had undergone fluorescein angiography after an intravenous injection of 5 ml 20% sodium fluorescein, Ilford FP4 film and a Carl Zeiss (Oberkochen) retinal camera being used. In all angiograms the posterior pole plus a five point survey of one eye were recorded, and a late transit photograph of the other eye was also taken. All the angiograms and Ektachrome transparencies were taken by the same medical photographer.

All assessments were conducted double blind by an experienced ophthalmologist (DJ), the Polaroid prints, 35 mm transparencies, and fluorescein angiograms being presented in a masked fashion. Images were examined and reported according to quality, pathological lesions present, and grade of retinopathy. The quality of the image was graded 1-5 as (1) excellent clarity, (2) definition of most retinal details (defocused/flare), (3) limited definition, (4) gross detail only visible, and (5) no detail visible. Images of grade 3 or worse usually resulted in a negative pathological grading owing to insufficient information.