These risk factors may be used in this way to identify people at high risk so that intervention may be directed towards them. In population terms this may be a profitable approach as the top 15% of the risk distribution may provide 32% of the cases of myocardial infarction over the subsequent five years. For individual people, however, the accuracy of the prediction is low: among those without initial evidence of coronary heart disease only 7% characterised as being at high risk will actually develop a myocardial infarction in the subsequent five years. Prevention has been suggested to be more effective in those with initial ischaemia.[12 13] Even in this group, however, only one fifth of those characterised as being at high risk will develop a myocardial infarction in the subsequent five years. This discrepancy between the importance to the population as a whole and that to a single patient may explain some of the difficulties of persuading people of the potential benefits of reducing risks.[8]

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Analysis and management of renal failure in fourth MRC myelomatosis trial

MRC WORKING PARTY ON LEUKAEMIA IN ADULTS

Abstract

During March 1980 to February 1982, 73 out of 80 patients in renal failure admitted to the fourth MRC myelomatosis trial were managed by a planned policy of high fluid intake (≥3 l/24 h) in addition to receiving one of the two chemotherapeutic regimens being tested in the main trial. Patients were also randomised to receive either sodium bicarbonate to render their urine neutral or no supplement. Follow up continued till death or to April 1983.

Of 49 patients who survived more than 100 days, 39 achieved reversal of their renal failure (18 complete, 21 partial). Recovery of renal function, as assessed by a fall in the serum creatinine concentration, was achieved even when light chain proteinuria persisted. Partial recovery of renal function was associated with prolonged useful life in several patients. In only 14 of the 80 patients studied was death directly attributable to renal failure. Survival of patients in the study was appreciably better than in equivalent groups of patients in other MRC trials in which less stringent policies of fluid intake were used. Patients randomised to receive alkali fared marginally better than the others, but the difference was not significant.

These results show that in many cases patients with myelomatosis who develop renal failure may have this complication reversed by taking a high fluid intake. Furthermore, though light chain is an essential component of renal disease in these patients, other factors are also important and are accessible to treatment.

This report was prepared by: I C M MacLennan, professor of immunology, University of Birmingham; J F Falconer Smith, consultant biochemist, Leicester Royal Infirmary; R A Crockson, senior chief technician, University of Birmingham; E H Cooper, professor of cancer research, University of Leeds; F R Knight, chief technician, University of Birmingham; J Cuzick, head of department of mathematics, statistics, and epidemiology, Imperial Cancer Research Fund, London; and J Hardwicke, professor of experimental pathology, University of Birmingham.

Members of the working party were: N C Allan, K D Bagshawe, P Barkhan, A J Bellingham, B J Boughton, C Bunch, S Callendar, D Catovsky, H Cuckle, J Cuzick, I W Delamore, J Durrant, I Fraser, D A G Galton, P Hamilton, F G J Hayhoe, J Hobbs, R M Hutchinson, H E M Kay, G A McDonald, I C M MacLennan, G W Marsh, E E Mayne, R Peto, R Powles, A G Prentice, F E Preston, J K H Rees, E G Rees, O S Roath, B E Roberts, I Temperley, R B Thompson, G Wetherley-Mein, J A Whittaker, and D A Winfield.

Correspondence to: Professor I C M MacLennan, Department of Immunology, University of Birmingham, Medical School, Birmingham B15 2TJ.

Introduction

Several reports have emphasised the poor clinical outlook for patients with myelomatosis who develop renal failure.[1-8] It is not clear, however, that this is due entirely to death from renal failure itself. The incidence of renal failure is considerably higher than the death rate attributable to this cause.[7]

The single factor most commonly implicated in the induction of renal lesions in myelomatosis is free light chain.[9 10] Nevertheless, only a proportion of patients with light chain proteinuria develop renal failure. This has led to the widely held concept of

the existence of nephrotoxic and non-nephrotoxic light chains.[9] Although the nephrotoxic potential of different light chains may vary,[11 12] this may not be the major reason why many patients with light chain proteinuria escape renal failure. An alternative hypothesis is that most if not all light chains cause tubular dysfunction but that this does not by itself reduce the glomerular filtration rate. Rather, it facilitates the precipitation of renal failure by other factors. If correct, this implies that measures apart from those to reduce the amount of light chain reaching the kidney may be of therapeutic value.

In planning the fourth MRC myelomatosis trial two measures were assessed for treating patients presenting in renal failure—(*a*) the maintenance of a high fluid intake, and (*b*) the administration of bicarbonate in sufficient quantity to render the urine neutral. This report analyses the clinical course of 80 patients who presented in the trial with renal failure and who were managed by these two methods. It also examines factors that may be associated with the response of renal failure to treatment and gives a detailed study of the causes of death in patients presenting with myelomatosis in renal failure.

Patients and methods

Between 1 March 1980 and 28 February 1982, 522 patients with myelomatosis were admitted to the fourth MRC myelomatosis trial. All patients were followed up till death or to 1 April 1983. Criteria for entry were as for the previous MRC trial.[5] Only patients aged under 75 were included.

ENTRY TO RENAL STUDY

All patients entering the main trial whose blood urea concentration exceeded the normal range received an initial 48 hours of hydration. Serum creatinine and blood urea concentrations were then reassessed, and patients whose blood urea concentration was still $\geqslant$15 mmol/l ($\geqslant$90 mg/100 ml)—that is, those whose raised blood urea value was not attributable to haemoconcentration alone—were admitted to the renal study. After 1 June 1981 patients whose serum creatinine concentration was $\geqslant$200 μmol/l ($\geqslant$2·3 mg/100 ml) but whose blood urea value was $<$15 mmol/l were also included, as in the first part of the trial there had been a high early mortality in this group.

RENAL STUDY MANAGEMENT

The 80 patients entered into the study were, with seven exceptions, treated with a continued oral fluid intake of at least 3 l/24 h. The exceptions were three patients with oliguric renal failure not reversed by dialysis and four patients with poorly controlled congestive cardiac failure. A further four patients presented in oliguric renal failure but managed to tolerate a high fluid intake after an initial period of rehydration and dialysis. In addition to a high fluid intake, patients were randomised to receive either sufficient oral sodium bicarbonate to render the urine neutral or no supplement. Irrespective of randomisation, any patient whose plasma bicarbonate concentration was below 20 mmol (mEq)/l was encouraged to take alkali until the concentration had returned to this value or above.

CHEMOTHERAPY

Patients in the renal study were randomised to one of the two chemotherapy arms being tested in the main trial.

Arm (*a*) comprised courses of melphalan 10 mg daily by mouth for seven days with prednisone 40 mg daily by mouth for seven days. If the blood urea concentration exceeded 10 mmol/l (60 mg/100 ml) melphalan was given as four day courses. Patients with oliguria received melphalan in a dose of 5 mg daily for four days. Courses were repeated at intervals of four weeks between the first day of one course and the first day of the next. In the event of myelotoxicity reduction of courses by one or more days was permitted. If intervals between courses had to extend to six weeks or more for three successive courses because of myelotoxocity the treatment was changed to cyclophosphamide 600 mg/m^2 intravenously every three weeks.

Arm (*b*) comprised the same regimen as (*a*) but with the addition of vincristine 1 mg intravenously on the first day of each course.

INVESTIGATIONS AND FOLLOW UP

Clinical details were recorded, routine blood counts and bone marrow evaluation carried out, and blood urea, uncorrected serum calcium, and plasma bicarbonate concentrations measured at the centres where the patients were being treated. The results were sent to the trials office in the department of immunology, University of Birmingham. Blood and an aliquot of a 24 hour urine specimen were sent to the trials laboratory by first class mail. Generally samples arrived within 24 hours of dispatch.

A centrifugal analyser (Instrumentation Laboratories) was used for assays of serum and urinary creatinine (Jaffe reaction) and total serum protein (biuret reaction). Serum albumin and serum and urinary α_1 microglobulin concentrations were measured by single radial immunodiffusion using monospecific polyclonal antisera.[13]

Sodium dodecyl sulphate polyacrylamide gel electrophoresis was performed using a discontinuous system based on that of Laemmli[14] incorporating 0·1% (wt/vol) sodium lauryl sulphate in a vertical slab apparatus. The running gels contained 12% wt/vol acrylamide. Unconcentrated urine or diluted urine samples were used so that roughly 5 μg protein was applied to the gel. The gels were stained with polyacrylamide gel electrophoresis blue 83 (BDH).

Total trichloroacetic acid precipitable protein was assessed in urine samples by the biuret reaction.

Urinary free κ and λ light chains were assessed using specific sheep antisera which did not react with light chain bound to heavy chain. They were used in radial immunodiffusion assays with 3% (wt/vol) polyethylene glycol 6000 daltons in agarose. Standards of κ and λ were prepared from the urine of several patients with myelomatosis without high molecular weight proteinuria by pooling equal amounts (as assessed by optical density) of light chains purified individually. Purification was by both ion exchange chromatography and gel filtration. The weight of protein in the κ and λ standards was assessed by the Kjeldahl method. Results are expressed as units, 1·0 U being equivalent to 1·0 g of standard. Units are used because light chains from individual patients vary in their degree of polymerisation and antigenicity.

Results

Figure 1 shows the overall prospects of survival among the 80 patients. The median survival of the group was 380 days and survival

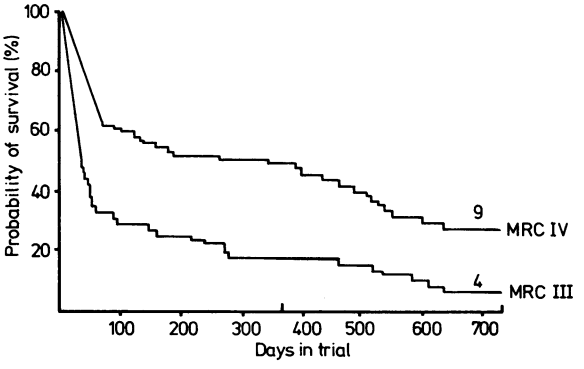

FIG 1—Survival prospects of all 80 patients in fourth MRC myelomatosis trial renal study. Survival of patients in third MRC myelomatosis trial who had blood urea concentration $\geqslant$15 mmol/l ($\geqslant$90 mg/100 ml) at presentation but after correction of dehydration shown for comparison. Numbers on survival curves refer to patients who had survived >2 years at time of analysis on 1 April 1983.

at two years 28%. The survival prospects for all 522 patients entered into the trial were 76% at one year and 52% at two years. Figure 1 also shows the survival rates among the 54 patients entered into the third myelomatosis trial whose blood urea concentration was 15$\geqslant$ mmol/l ($\geqslant$90 mg/100 ml) before chemotherapy but after the initial 48 hours of hydration. Both groups had a high early death rate. The

chemotherapy and management policy in the third trial differed from that in the fourth trial. Details of this have been published.[5] In the third trial 37 (69%) of the 54 uraemic patients had died within 100 days of entry; in the fourth trial 31 (39%) of the renal study patients had died by that time. After 100 days the death rate in both trials slowed appreciably. The effect of presentation blood urea values on survival in all 522 patients in the fourth trial was dramatic—χ^2 for trend 42·6; $p<0.0001$. When presentation blood urea concentration was assessed for survival prospects among the 422 patients surviving 100 days from entry its prognostic significance was considerably less (fig 2; χ^2 for trend 6·6; $p=0.01$).

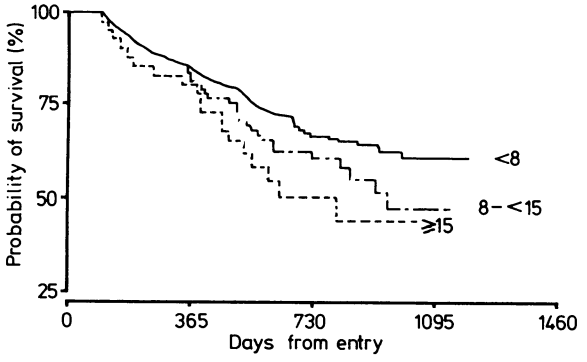

FIG 2—Effect of presentation blood urea value on survival prospects of patients in fourth MRC myelomatosis trial who survived 100 days from entry. Numbers on survival curves refer to range of blood urea concentrations in mmol/l at presentation but after correction of dehydration (1·0 mmol/l ≈ 6·0 mg/100 ml).

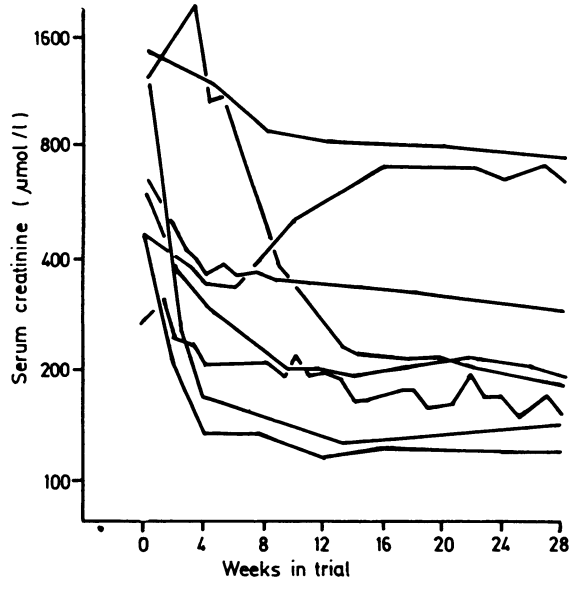

FIG 3—Patterns of change in serum creatinine concentrations with time in renal study patients receiving high fluid intake. Chart shows examples from patients in each of three groups defined under Results. (Creatinine: 1·0 μmol/l ≈ 0·01 mg/100 ml.)

CHANGE IN SERUM CREATININE VALUE IN PATIENTS SURVIVING 100 DAYS FROM ENTRY

Of the 80 renal study patients, 49 survived more than 100 days. In all but two of these, serial estimations of serum creatinine values were obtained. Figure 3 illustrates the three types of changes in serum creatinine concentrations seen with time in different patients—namely, (*a*) patients who showed rapid and complete resolution of their raised values, (*b*) those who showed improvement that was not complete, and (*c*) patients who showed a slow, progressive rise in serum creatinine concentrations, usually after an initial modest improvement. The group with *complete resolution* comprised 18 patients whose serum creatinine concentration returned to ≤130 μmol/l (≤1·5 mg/100 ml); the group with *partial resolution* comprised 21 patients with a sustained reduction in serum creatinine concentration, though at a value above 130 μmol/l (range 845-148 (median 241) μmol/l; 9·6-1·7 (median 2·7) mg/100 ml)); and the *no resolution* group comprised eight patients, two of whom were in oliguric renal failure and were maintained by dialysis. Figure 4 shows the relevance of these three categories to survival.

Factors that might affect renal state in these groups and the 31 patients who died within 100 days of entry to the study are analysed below.

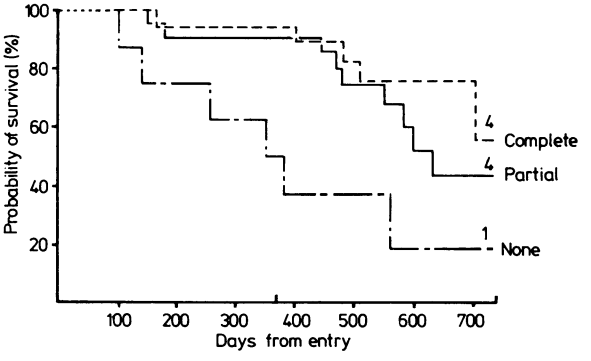

FIG 4—Survival prospects in complete resolution, partial resolution, and no resolution groups in renal study. (Groups defined under Results.) Numbers on survival curves as in fig 1.

TABLE I—*Serial serum creatinine concentrations and 24 hour urinary light chain excretion in two patients. (These patients exemplify those showing reduction in serum creatinine without major fall in light chain output)*

Patient		Months from entry to trial						
		0	1	2	3	6	9	12
A	Serum creatinine (μmol/l)	1460	1046	896	845	885	778	665
	Urinary λ chain (U/24 h)	13·6	13·8	9·8	4·0	10·1	10·8	17·2
B	Serum creatinine (μmol/l)	418	554	505	340	260		240
	Urinary κ chain (U/24 h)	9·5	13·2	8·9	4·5	6·8		13·3

Conversion: SI to traditional units—Creatinine: 1·0 μmol/l ≈ 0·01 mg/100 ml.

URINARY LIGHT CHAIN OUTPUT AND FALL IN SERUM CREATININE

Almost all patients entering the renal study had substantial free light chain production at presentation. Sixty had urinary free light chain values exceeding 1·0 U/l and 16 of the remaining 20 values between 0·1 and 1·0 U/l. Fifty patients had κ producing tumours and 30 λ producing tumours.

There was no obvious difference in the distribution of urinary light chain type or concentration between patients dying within 100 days and any of the three groups defined by change in serum creatinine value. Thirty nine patients surviving 100 days showed complete or partial resolution of renal failure as assessed by serum creatinine concentration. The fall in serum creatinine concentration in these patients was compared with their change in urinary light chain output. This analysis was made before starting chemotherapy but after the patients had been fully rehydrated. Substantial falls in serum creatinine were achieved before a reduction in urinary light chain output occurred in 11 of the 18 patients showing a return to normal serum creatinine values and 11 of the 21 patients showing partial resolution of values. Table I exemplifies this for two patients who failed to achieve a significant reduction in light chain output with chemotherapy. Some patients stopped producing light chains after chemotherapy but had residual renal impairment.

SERUM CALCIUM AND CREATININE, GLOMERULAR AND TUBULAR PROTEINURIA, AND OUTCOME

Serum calcium concentration was raised (>2·6 mmol/l (>10·6 mg/100 ml) after correction for serum albumin value by adding 0·1 mmol/l (0·4 mg/100 ml) to calcium for every 6 g/l by which serum

albumin was below 40 g/l and vice versa) in 39 (49%) of the renal study patients after the initial 48 hours of hydration but before chemotherapy. Of the 31 patients dying within 100 days, 16 (52%) had raised serum calcium concentrations. In the groups showing complete, partial, and no resolution of renal failure raised serum calcium concentrations were seen in 9 (50%), 11 (52%), and 3 (38%) respectively. The prevalence of hypercalcaemia at presentation in the 442 non-renal study patients (23%) was lower than that in any of the renal study groups.

Serum creatinine concentrations—Figure 5 shows the serum creatinine concentrations at presentation in the four groups defined above. More patients achieving complete resolution of renal failure had serum creatinine concentrations of 200-300 μmol/l (2·3-3·4 mg/100 ml) than in any other group. Three patients with concentrations

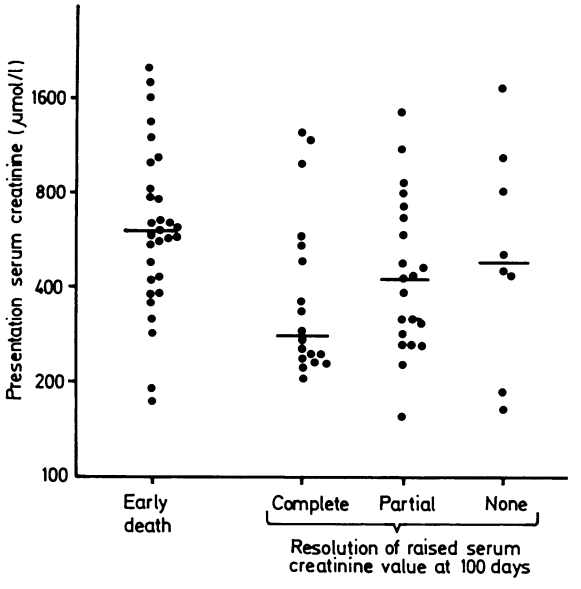

FIG 5—Distribution of serum creatinine concentrations in renal study patients analysed in groups defined under Results. Horizontal bars represent median values. (Creatinine: 1·0 μmol/l ≈0·01 mg/100 ml.)

above 1000 μmol/l (11·3 mg/100 ml) at presentation, however, achieved normal values subsequently. Two of these presented in oliguric renal failure and underwent a short period of dialysis before achieving a good diuresis. They were then maintained with a high fluid intake.

Non-selective glomerular proteinuria (defined as more than 1 g trichloroacetic acid precipitable protein per litre of urine, of which a substantial proportion was of higher molecular weight than albumin and not simply paraprotein as shown by sodium dodecyl sulphate polyacrylamide gel electrophoresis) was present in 54 (10·3%) of the 522 patients entered into the trial, including 10 of the 80 patients in the renal study. Four patients had selective albuminuria. In the renal study group non-selective glomerular proteinuria was found in 10 of the 80 patients—in six of the 31 patients dying before 100 days, in two of the complete resolution group, and in two of the partial resolution group; it was not found in any patient of the no resolution group.

Tubular function will be reported in detail elsewhere. Analysis showed that around two thirds of patients entered into the fourth myelomatosis trial had selective tubular proteinuria which correlated strongly with urinary light chain output. This correlation was seen both in patients with normal and in those with raised serum creatinine concentrations. Selective in this context means failure to reabsorb α_1 microglobulin and α_1 acid glycoprotein but with retained capacity to reabsorb retinol binding protein, β_2 microglobulin, and glucose. This is illustrated in figure 6, which correlates α_1 microglobulin concentrations in the urine in relation to urinary light chain output and renal failure. α_1 Microglobulin is a 27 kilodalton protein which is normally reabsorbed in the proximal renal tubules. All patients in each of the renal study groups had tubular proteinuria.

Pyogenic infection at presentation—Pyogenic infection was a common feature in the trial patients as a whole. Of all 522 entrants, 100 (19·2%) had a pyogenic infection requiring medical attention in the year up to diagnosis of myelomatosis. The same proportion of renal study patients had pyogenic infection in this period.

CAUSES OF DEATH IN RENAL STUDY

Careful analysis of cause of death was made in all patients dying in the renal study. It was difficult to assign a single cause of death in some cases. The principle followed was to identify the main factor precipitating the final episode—for example, someone with uncontrolled growth of tumour who died with terminal bronchopneumonia was recorded as dying from tumour growth. Someone whose tumour seemed to be in control, who was not immobilised by bone pain, and who died from lobar pneumonia, however, was recorded as dying from pyogenic infection. Table II summarises the causes of death.

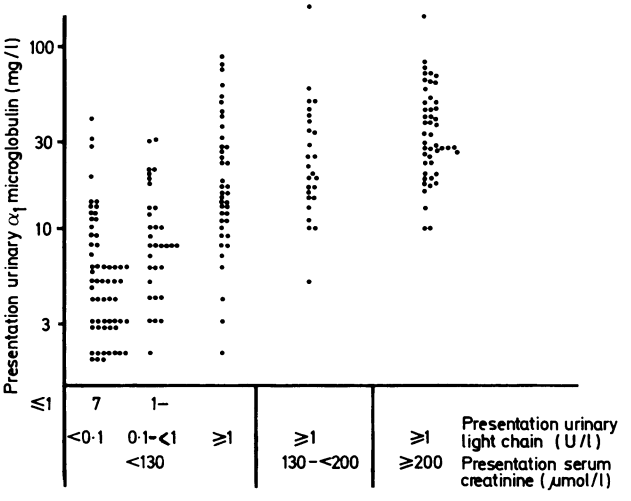

FIG 6—Relation between urinary light chain, urinary α_1 microglobulin, and serum creatinine concentrations. All values taken after correction of initial dehydration but before chemotherapy. (Creatinine: 1·0 μmol/l ≈0·01 mg/100 ml.)

TABLE II—*Summary of causes of death*

Principal cause of death	All patients	Death <100 days	Death >100 days*		
			Resolution of raised serum creatinine: Complete	Partial	None
Overwhelming tumour	15	5	5	4	1
Renal failure: High fluid intake tolerated	7	5			2
Renal failure: Fluid tolerance impaired	7	6			1
Pyogenic infection; other factors not immediately life threatening	13	7		3	3
Myocardial infarction	4	3		1	
Cerebrovascular accident	4	2		1	
Gastrointestinal haemorrhage	2	2			
Other	2	1			

*In two patients living more than 100 days insufficient follow up information was available to allow allocation to a group.

Renal failure was the principal cause of death in 14 of the 54 patients dying in the study. Seven of these were unable to tolerate a high fluid intake—in four owing to congestive cardiac failure and in three because of unreversed oliguric renal failure. Seven patients died of renal failure despite a high fluid intake. None of the patients showing improved renal function at 100 days subsequently died from renal failure. Progressive myelomatosis was the main cause of death in these patients.

Pyogenic infection in patients whose tumour load and renal state at the time of onset of infection were not thought to be life threatening was a major cause of death. The incidence of death from pyogenic infection in the renal study was a little higher than that in other patients in the trial—that is, 24% of 54 deaths as opposed to 19% of 174 deaths.

EFFECT OF ALKALI ADMINISTRATION

Treatment with alkali was randomly allocated to 40 patients. The remaining 40 were randomised to receive high fluid intake alone. The group allocated alkali administration fared slightly better in terms of survival than the control group (fig 7). This difference, however, was not significant (log rank test; $p = 0{\cdot}26$).

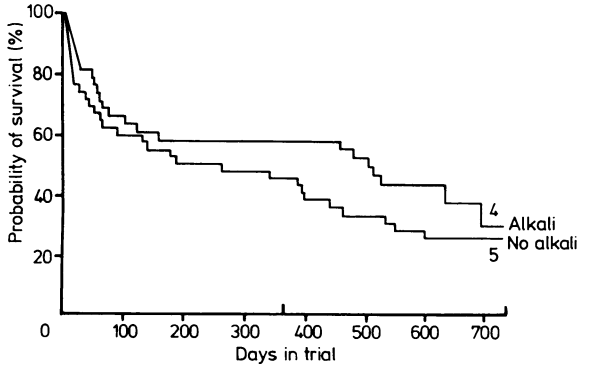

FIG 7—Effect of alkali administration on survival prospects of renal study patients. Numbers on survival curves as in fig 1.

Discussion

Analysis of renal failure in myelomatosis is complicated by the number of pathological processes that may affect the kidney in this disease.[10 15] It is also difficult to relate particular dysfunctions to the onset of renal failure—for example, both tubular and glomerular proteinuria may be present without raised concentrations of serum creatinine. In a second report on all 522 patients in the fourth MRC myelomatosis trial it will be shown that impaired tubular function is associated with light chain proteinuria even in patients not in renal failure. The present study, from a different set of observations, supports the conclusion that light chain alone is not sufficient to precipitate or sustain renal failure. Four patients already in renal failure reverted to normal glomerular filtration rates despite continued light chain production.

There may be several factors in the onset of renal failure in patients with light chain associated tubular dysfunction. Difficulty in conserving water and salt is likely to be of central importance. Dehydration will also aggravate and be aggravated by hypercalcaemia, proximal tubular acidosis, and cast formation. Casts are amply documented as a special feature of myelomatosis, but renal biopsy studies suggest that they are not invariably associated with renal failure.[16-18] Shutdown in glomerular filtration is not always associated with cast formation in other conditions.[19] Additional factors must therefore be considered. Cast formation is likely to be enhanced during phases of dehydration and reduced when a high fluid intake is maintained. Pyogenic infection may also help to precipitate renal failure. The similar incidence of pyogenic infection before treatment in the renal study patients and other patients in the trial implies that pyogenic infection in the former was not secondary to renal failure.

Giving supplements of bicarbonate was clearly not harmful. Continued high fluid intake itself, however, was associated with reversal of renal failure in many patients. In single case studies Kay *et al*[20] and Bryan and McIntyre[21] have reported reversal of renal failure in myelomatosis associated with giving alkali. One reason for advocating alkali administration is a possible association between cast formation and urinary pH. Clyne *et al* reported that in their experiments only human light chains with high isoelectric points could be induced to form casts in acidaemic water deprived rats.[22] Weiss *et al* have shown that the acidic protein β lactoglobulin will form casts in rat tubules, while the relatively basic protein myoglobin will not.[23] In our patients[24 25] correlation between renal failure and the dominant isoelectric point of urinary light chain has not been apparent. Hill *et al*, however, reported an association between acidic electrophoretic mobility of light chains and the induction of renal lesions.[26] At low pH light chain may be precipitated in the lumen of the tubules. In addition, the reabsorption of light chain by proximal tubules has been shown to be reduced.[24] In vitro studies show that light chain reabsorption is not affected by a number of agents affecting renal tubular transport mechanisms.[27]

In setting up this study the evaluation of fluid intake as a randomised variable in the management of renal failure could not be justified ethically, and therefore we do not know why patients presenting in renal failure survived longer than in other trials in which chemotherapy as well as the fluid intake policy was different. These may, however, have been less important than the physicians' belief in the poor prognosis associated with renal failure in myelomatosis. A renal study offering hope of effective treatment may have encouraged more energetic treatment of potentially fatal complications in this trial.

This study shows that in many cases renal failure may be reversed by relatively simple procedures, while in oliguric renal failure associated with myelomatosis the glomeruli may open up after a period of dialysis.[28] Bernstein and Humes report that prolonged survival is unlikely when renal failure does not resolve rapidly.[29] In this study, however, prolonged survival was seen in many patients whose renal failure was only partially reversed, and some of them led an active life.

In conclusion, renal failure in myelomatosis remains a highly complex condition. This study has been clinically useful by showing that in many cases a continued high fluid intake may reverse renal failure. It has also shown that while light chain is an essential component of renal disease in myelomatosis, other factors are important in precipitating renal failure. These other factors are accessible to treatment.

Antiserum and standard for the α_1 microglobulin assay were a gift of Behring Werke AG. All other antisera and standards were generously donated by Dr A R Bradwell and Mr R L Drew, of the immunodiagnostic research laboratory of the department of immunology, University of Birmingham.

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SHORT REPORTS

Therapeutic venous infarction of an aldosterone producing adenoma (Conn's tumour)

Primary aldosteronism accounts for about 1% of the hypertensive population.[1] It may be due to an aldosterone producing adenoma (Conn's tumour), adrenal hyperplasia, or occasionally morphologically normal adrenal glands. The usual management of Conn's tumours is surgical excision or maintenance treatment with spironolactone. Large doses of this drug may be needed, however, in combination with other antihypertensive agents and this may produce untoward side effects. We describe a patient whose Conn's tumour was treated by superselective catheterisation and venous infarction.

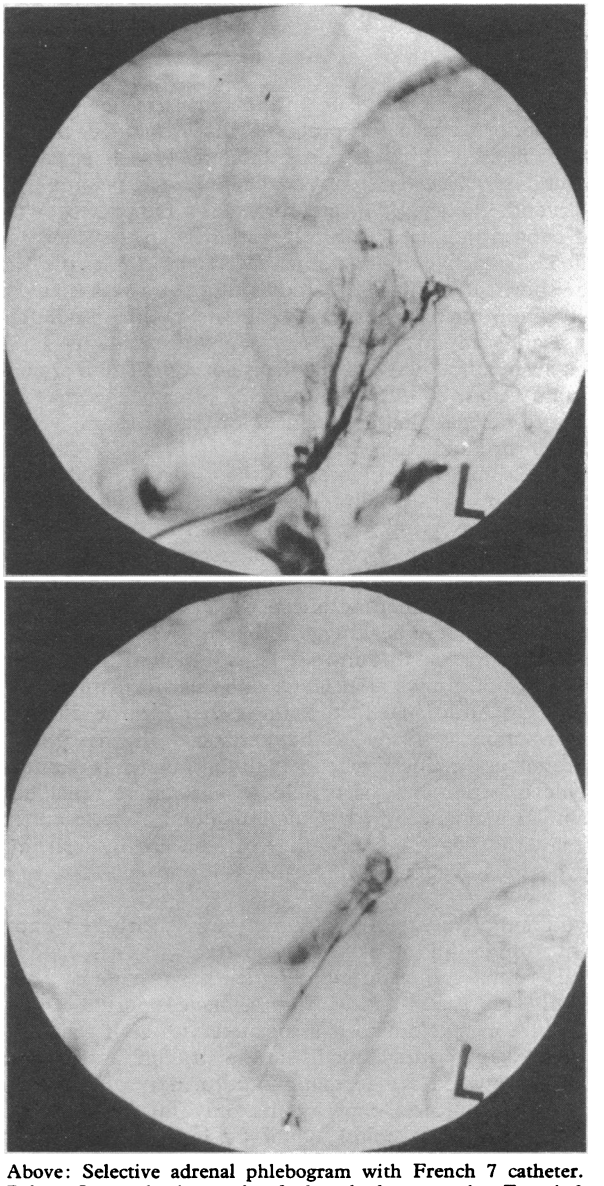
Case report

A 55 year old woman with an eight year history of hypertension had been found to be hypokalaemic two years previously, the lowest potassium concentration (1·7 mmol(mEq)/l) being recorded when she was not taking spironolactone. Her medical management was complicated by severe side effects of her drugs. Laboratory investigations suggested a diagnosis of primary aldosteronism. A left adrenal aldosterone producing adenoma was confirmed by computed tomography, ^{75}Se cholesterol scintiscanning, and venous sampling. She was thought to be a poor surgical risk and was referred to the Hammersmith Hospital for percutaneous tumour ablation.[2 3] Under local anaesthesia, using the Seldinger technique, a catheter (French 7) was directed from the right femoral vein into the left adrenal vein. The adenoma was clearly visualised (figure). A coaxial catheter (French 3) was inserted through the larger catheter, manipulated into the tumour vein (figure), and a mixture of 0·5 ml absolute alcohol and 0·5 ml contrast medium (iohexol) injected into the tumour. The sclerosant mixture filled the venous network of the tumour and rapidly abolished flow in the opacified vessels.

After the procedure the patient experienced a little discomfort in her left loin, which was controlled with mild analgesics, and a fever of 38°C subsided after three days without antibiotics. Labetalol and hydralazine were withdrawn and her blood pressure remained well controlled. She was discharged home taking spironolactone (200 mg daily) only, which was stopped six weeks later. Computed tomography six weeks after the procedure showed a reduction in tumour size, and a ^{75}Se cholesterol scintiscan showed normal uptake on the right but no uptake in the left adrenal gland. Blood pressure and plasma aldosterone and plasma and urine electrolyte concentrations remained normal when she had stopped all drugs. Twelve months after tumour ablation the patient remained normokalaemic without spironolactone and had needed only a small dose of a β adrenoceptor antagonist (oxprenolol 40 mg twice daily) to control her blood pressure.

Comment

Conn's tumours are usually hypovascular[4]; this and the fact that the adrenal gland receives its arterial supply from at least three different sources mean that it is not feasible to embolise these tumours by the arterial route, the approach normally employed for tumour embolisa-



Above: Selective adrenal phlebogram with French 7 catheter. Below: Superselective study of adrenal adenoma using French 3 catheter passed coaxially through larger catheter.