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Review article

Acute renal failure in the intensive care unit today

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Although there are isolated reports of what would now be called acute reversible renal failure dating back a century, particularly in the German literature describing injuries in the First World War [1], it was not until the second major conflict that a number of sets of clinical and experimental data were brought together to produce the current concept: an acute, potentially-reversible failure of renal function in previously normal, unobstructed kidneys, in response to events as diverse as mismatched transfusion, abortions, cardiovascular collapse, sepsis, crush injuries, and a variety of nephtrotoxic substances [2, 3]. Unfortunately there is still no satisfactory term for this condition, since "acute renal failure" is too broad, and "acute tubular necrosis" too narrow.

At this time the mortality of "acute tubular necrosis" was very high, especially in those injured: in the Second World War, the death rate amongst wounded servicemen was 91% [4]. A major leap forward was the introduction of haemodialysis by Kolff during this same conflict: the first patient (who had sulphonamide toxicity) to survive acute renal failure thanks to haemodialysis was treated 42 years ago. Immediately upon the introduction of haemodialysis the mortality in both civilian post-surgical renal failure [5] and military trauma [6] fell to about 50% or 60%, and the oliguric and diuretic stages of acute renal failure were observed and studied.

It is distressing to find that thirty years on the mortality of the more severe forms of acute renal failure has remained approximately the same, only one patient in three surviving on average. Why is this? First, many patients with "uncomplicated" acute renal failure are managed in general wards or renal units, and these patients have a very low mortality. The most recent figures, those of the EDTA-European Renal Association's 1985 report [7] notes that in a survey of 474 patients with acute renal failure from 114 renal units treated during a six week period in 1984, the mortality in those where renal failure was the only problem was 8%. However, these were only 61 patients out of 474, the other 413 (87%) carrying an average mortality of over 60% (Fig. 1). These are, of course, just those patients who are treated in intensive care units.

Thus, despite what most of us would regard as improvements in ventilation, nutrition, anaesthesia, antibiotic chemotherapy and the diagnosis of infection, cardiovascular monitoring and management, and techniques of substitution for renal failure, we appear to be doing no better than 30 years ago [8]. Of course, one possible explanation is that although the mortality remains high, the proportion of more severe cases is greater, and the group of those going into renal failure are a higher risk group than in 1964 or 1974. We know, of course, that there are patients who go into renal failure now after procedures undreamt of in the past, and a few causes – like septic abortions – are now uncommon in the developed world;

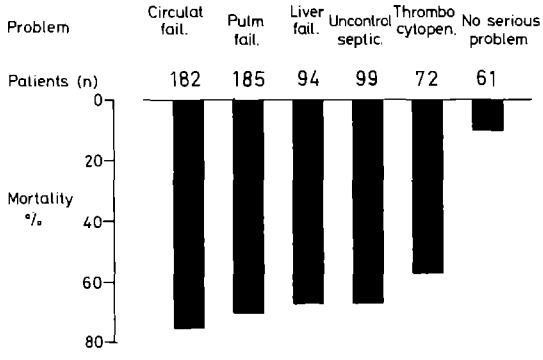

Fig. 1. Mortality in acute renal failure in relation to the associated clinical problems present at the onset. Data of the EDTA-European Renal Association Registry, reproduced with permission of the authors and Ballière Tindall, Eastbourne, UK [from 8]

Table 1. Mortality of surgical acute renal failure by site

	Number of patients	Died	Mortality (%)
From Kerr [11]			
Stomach and duodenum	81	51	36
Biliary tract	31	20	63
Appendix and bowel	52	85	61
Vascular and cardiac	33	18	55
Urological	34	13	37
Miscellaneous (mainly gynaecological)	46	19	41
From McMurray [12]			
Gastrointestinal	38	21	56
Aneurysms	42	21	50
Cardiac	21	8	39
Other	16	4	25

but in general the causes are depressingly similar. A number of papers in the early and middle 1970's [9–12], analysed the risk factors for survival in acute renal failure, including post-surgical cases; the presence of sepsis, especially intra-abdominal sepsis, appeared to be the major determinant, but surprisingly age is not. In our 1972 paper [8] the proportion of patients aged over 60 (25%) was exactly the same as our recent series, and age emerges as either a weak determinant of risk in some series, including our own, or to have no effect at all, as in the recent EDTA-European Renal Association data [7].

In addition, if one compares the mortality of acute renal failure in *truly comparable* patients who appear in both recent and earlier series – such as complications of biliary surgery, gastrointestinal disasters both natural and iatrogenous, ruptured aortic aneurysms and so on, the mortality is almost identical 10–20 years on – and even though there *have* been reports of a decline from 90% to 60% in mortality of post-aortic operation cases from other units [13], our own mortality remains the same in this group of patients.

Another risk factor is the site of the surgery. Several analyses, two of which [12, 13] are summarised in Table 1, have shown that the mortality from acute renal failure in a setting of gastrointestinal operations is particularly high, especially when the bowel rather than stomach is involved. In many units now, as illustrated in Table 1 which is similar to our own data, the mortality of patients having even complex contemporary cardiac surgery is no higher (66%), and in some series lower than those in acute renal failure following other surgery; previously these patients had an unenviably high mortality.

One area in which progress *has* been made however, is the disappearance of gastrointestinal haemorrhage as an immediate cause of death. This complication was, after sepsis, the second commonest cause of death in earlier series [10, 11], but has now all but disappeared. Better nutrition, more intensive dialysis and the prophylactic use of Cimetidine or Ranitidine together with oral antacids all may play a role in this decline. In general, today patients live longer in acute renal failue before dying in the third, fourth or fifth week, whereas 10–15 years ago death most commonly occurred within two weeks of onset [10]. This has important consequences for workload and costs, which are discussed further below.

This brings us to a consideration of both respiratory failure and sepsis as (perhaps interrelated) causes of death [20]. Table 2 presents analyses of surgical patients treated in our own unit done at three different

Table 2. Acute renal failure after surgery at Guy's hospital

		Overall				Ventilated cases		
		Treatment	*n*	Died	Mortality (%)	Ventilated	Died	Mortality (%)
Stott [9]/Brown [45]	1969–73	HP/PD	109	67	62	25[c]	22	91
Neild (unpublished)	1977–78	HD	28	14	50	21	15	71
Taube (unpublished)	1981–84	HF/HD	143[a]	91[a]	64[a]	29[b]	22[b]	76[b]

Selected analyses from 739 patients treated for ARF 1964–1984 [47]. [a] *All* cases, including surgical; [b] 1984 only; [c] 1971–73 only. HD = haemodialysis; PD = peritoneal dialysis; HF = haemofiltration

Table 3. Mortality in multiple organ failure following emergency operations (from [24]) and in intra-abdominal sepsis (from [25])

	n	Deaths	Mortality (%)
No. of organs failed following emergency operations			
1	31	7	23
2	15	8	53
3	29	23	79
4	5	5	100
No. of organ systems failing[a] in intra-abdominal sepsis			
0	75	2	4
1	10	1	10
2	6	3	50
3–5	15	15	100
Total	106	21	

[a] Heart (11), lung (30), liver (9), kidney (16), CNS (10), any (31)

points in the history of our own unit by successive Senior Registrars. Forty per cent of such patients required ventilation and the mortality in the early 1970's was 91%. In 1984, the mortality of ventilated patients was 76%, compared with 9% in unventilated patients ($p \ll 0.01$); and 70% of post-surgical acute renal failures required ventilation, similar to the data of Sweet et al [14].

Of course, all this does not represent only the effects of ventilatory failure, since it is just those patients most severely ill from their primary surgery and previous state who require ventilation; a multivariate analysis of the various factors involved would be necessary to sort out the individual contribution of the respiratory failure, and such analyses have begun [13, 15–19].

Once a patient has two systems malfunctioning – renal and respiratory – he or she qualifies for the ominous title of "multiple organ system failure" [21–23]. This has attracted much attention in the past decade, and in a surgical setting with acute renal failure as one of its components, carries a very high mortality. Table 3 shows the data from Fry's [24] study of multiple organ failure after emergency operations of all types and the data from Pine et al. [25].

In identifying patients who are likely to die or likely to survive [15–19] a simple count of the number of organ systems "down" gives useful, if gloomy information. Two systems failing carries a high mortality of around 50%, which interestingly is just about that for the average series of acute renal failure. When three systems are malfunctioning, then immediately the mortality rises towards, or equals 100% [24, 25]. Thus, the failure of only *one* other system in a patient already in acute renal failure and on a ventilator is a very ominous sign. Once four systems are affected, recovery is virtually unknown.

These figures are not only of humanitarian and scientitic interest, but have major significance for planning of staff and financial resources in renal and intensive care units. As mentioned above, although the mortality in acute renal failure has not decreased, the patients survive longer. There are few data on the costs of acute renal failure as part of multiple organ failure, and we need these data rather badly. One analysis from the United States is that of Eisman et al. [23], which suggested that for an average survival of 30.5 days, the total hospital costs were $22,000 in 1977, excluding physician's fees; a similar study by McMurray et al. [13] gave a figure of $470 per day in 1975. Allowing only for inflation (and medical costs have increased at a greater rate than general inflation for some time), it is likely that these figures would need to be doubled at least to give contemporary costs (inflation went from 100% to 230% from 1974 to 1984 in the United Kingdom). We rather badly need some British data on the costs of patients in acute renal failure in intensive care; for those with both respiratory and renal failure, surviving for four weeks on average, the cost cannot be less than about £40,000 per patient. One of our own patients, who survived early last year after more than three months in intensive care, alone cost upwards of £250,000.

These data raise all sorts of difficult and contentious ethical and practical questions: for example, is it justifiable to proceed with a patient with four or more systems malfunctioning, if two of these are the kidneys and the lungs? Can one perform cost benefit calculations if life, rather than quality of life, is at stake?

Prevention of surgical acute renal failure

Naturally, having failed to reduce the mortality of established acute renal failure, attention turns – as it

should in any case – to prevention. In a rational world, preventitive measures should arise from an understanding of the pathogenesis of the acute renal failure state. Unfortunately, despite a vast number of studies, the events mediating acute renal failure are ill-understood and contentious; they have been reviewed a number of times recently [26, 27]. An important general point is that *all too many cases of acute renal failure arise through positive action, or delay and neglect on the part of doctors.* Thus doctors can, by thinking about the patient's general status, avoiding nephrotoxic drugs, and maintaining a watchful readiness to act rapidly if things should go wrong, avoid a large number of cases of acute renal failure; once the patient has crossed this particular Rubicon, the chances of return are at best only fifty: fifty, and so time, effort and money expended at this point are indeed well spent.

One new factor in acute renal failure over recent years has been the role of non-steroidal anti-inflammatory agents [28]. A large proportion of the elderly population are now taking these drugs – some of which are available off prescription – and this fact is not always known to the admitting physician. All of them act by inhibiting the cyclo-oxygenase enzyme which catalyses the first step in the conversion of arachidonic acid into prostaglandins. It appears that in normal circumstances, and especially in conditions of renal hypoperfusion, the medulla elaborates vasodilator prostaglandins [principally PGI_2 (prostacyclin) in man], which raise the perfusion and GFR back towards normal. In the presence of indomethacin, ibuprofen or similar drugs, this protective mechanism is eliminated and renal failure may appear. In addition, some of the group, notably fenoprofen, may lead to an interstitial nephritis, with a more prolonged acute renal failure, sometimes accompanied by a rather acute nephrotic syndrome.

Standard measures of cardiovascular resuscitation should help preserve renal bloodflow in patients with hypovolaemia or cardiogenic shock, and many of these patients will start passing urine: with the obvious proviso that an overfull circulation may precipitate or worsen pulmonary oedema in those who do not. Attention has also been paid to specific pharmacological measures to achieve an increase in renal bloodflow and/or glomerular filtrate. The earliest studied was the use of *mannitol,* which is an osmotic diuretic, and which appears to protect when given in jaundiced patients subjected to surgery [24] and in aortic cross-clamping [30]. In retrospect it may have been that mannitol achieved this effect by expanding blood volume and thus increasing renal bloodflow, rather than by any effect on filtration or increased passage of fluid down the nephron; reversal of the "no reflow" phenomenon of prolonged vascular endothelial swelling [26, 27] is another possibility.

More attention recently has been given to the use of *dopamine* in low doses (0.5 – 2.0 μg/kg per min intravenously) [31, 32], with or without the concomitant use of *frusemide,* itself alleged to be able to reverse incipient acute tubular necrosis [33]. This state of "incipient acute renal failure" is thought to be identifiable by the finding of oliguria of about 300 – 500 ml/24 h, the urine being highly concentrated with respect to plasma with a low sodium concentration below 20 mmol/l or even below 10 mmol/l [34], in contrast to the dilute urine with a high urinary Na^+ of established acute tubular necrosis. However, even these distinguishing features have been called into question recently [35], and the boundaries of immediate reversibility (minutes or hours) and established tubular necrosis (days or weeks) are certainly not precise in functional or practical terms. If the patient has already received frusemide (as is almost invariable today) the boundary is further blurred. None of these pharmacological agents has much effect on those patients believed to be in "established" acute tubular necrosis, although we [36] managed to induce a useful increase in urine volume in a minority of such patients using large intravenous doses of frusemide.

Attention has also turned to the metabolic abnormalities of the renal tubules, however induced, in search of prevention of acute renal failure. These abnormalities include a fall-off in oxygen consumption an increase in intracellular Ca^{++} concentration, and a run-down in ATP. This has led to attempts to alter the Ca^{++} fluxes with verapamil, and boost the ATP using $MgCl_2$-ATP, inosine and/or allopurinol. Whether these experimental approaches will turn out to be useful in clinical acute renal failure in man is not clear yet; verapamil appears particularly promising.

Despite the high mortality amongst those who do go into renal failure, it is clear that *some* progress has been achieved in reducing the number who actually do develop the complication. The incidence of acute renal failure in battle casualties fell from 1:20 in World War II, to 1:800 in the Korean conflict, and 1:1800 in Vietnam [37]. The incidence of acute renal failure following complicated cardiovascular surgical procedures also fell, from 30% to 1.7% between 1965 and 1978 [38].

Sepsis in acute renal failure

This leaves us with the vitally important question of *sepsis* [10, 11, 16, 17, 22 – 24, 39, 40] if sepsis is important in both the genesis of the acute renal failure, through vasoconstriction from endotoxin [39] and the induction of disseminated intravascular coagulation,

Table 4. Survival in ARF with intra-abdominal sepsis

		Practice	*n*	Deaths	Mortality (%)
Kornhall [41]	(1972)	Civilian	50	50	100
Lordon and Burton [42]	(1972)	Military	37	24	88
Milligan et al. [43]	(1978)	Civilian	76	52	68

and also as a cause of death, then surely here is an avenue for prevention, or at least a reduction of mortality? There is little doubt that sepsis is an important determinant of survival, above all intra-abdominal sepsis [16, 17, 24, 25]. Table 4 shows the mortality in threee series [41 – 43] of military and civilian cases of acute renal failure complicated or precipitated by intra-abdominal infection.

Obviously, all the usual steps should be taken to avoid or limit sepsis, with particular attention to any breach of the integument, especially intravenous cannulae for feeding, monitoring and dialysis which are contaminated on average after three days with bacteria, and three weeks with *candida*. Deep sepsis should be suspected and sought vigorously; ultrasound is cheap and easy to perform, but is perhaps inferior to CAT scanning in this respect; Ga^{67} scanning may also be useful, or the localisation of ^{111}In-oxime labelled white cells. The problem with these two types of study is that up to several days may elapse before the results are available. The problem of antibiotics is too large to discuss here, but almost all patients in combined renal and respiratory failure will end up on "cocktails" of broad spectrum antibiotics; one must not forget anaerobic organisms, and all patients with deep sepsis should be given metronidazole as part of their regime. Wardle [39] reminds us that bacterial destruction leads to release of further endotoxin and perhaps to worsening of the condition. Unfortunately, until recently, assays for endotoxin have been neither easy to perform, reproducible, nor readily available; the availability of radioimmunoassay may change this. Yeasts and fungi wait for a week or two in the wings before invading the patients, often from the end of the intravenous feeding cannula; the only effective treatment for systems candidaemia or fungaemia remains the toxic amphotericin.

Table 5. Findings at time of 70 re-explorations in infected abdomens + ARF [from 43]

	Survivors	Non-survivors	Total
Remediable lesions	29	37	66
Abscesses	19	23	42
Suture insufficiency	4	5	9
Perforations	4	5	9
Bleeding site	2	4	6
Non-remediable lesions	4	16	20
Diffuse peritonitis	2	8	10
Ischaemic/infarcted gut	1	6	7
Pancreatitis	0	1	1
Nothing	1	1	2

An important point which arises from all three series cited above [41 – 43] is that *re-exploration of the suspect abdomen is essential,* if necessary several times, however sick the patient may be. In Kornhall's series [41], 30% of patients had undiagnosed intra-abdominal problems at post-mortem, many remediable. Lordon and Burton [42] in Vietnam used loose deep sutures for almost daily laparotomy in many patients. Milligan et al. [43], who present the lowest mortality in this group of patients to date, re-opened 40 of 76 patients once, 25 twice and 5 patients on three occasions; Table 5 shows the findings at re-exploration. In 86 reexplorations in 40 patients, 66 showed potentially remediable problems; indeed, if one includes the 7 cases of infarcted bowel (which they did not) the total rises to 73/86.

How sepsis exerts its effects if still unclear. Many studies have been done in animals using the injection of endotoxin, but there is great species variation in sensitivity to this substance, and endotoxin may not be the only important mechanism in bacteraemia or local sepsis. In these animal models, it has not been possible to reproduce, with endotoxin alone, the typical early response of humans to the septic state: low blood pressure, low peripheral resistance with a dilated hyperdynamic circulation, before vasoconstriction and severe hypotension sets in. More appropriate models such as that employing caecal performation in the sheep, as studied by Linton and colleagues (T. F. Walker, R. M. Lindsay, W. T. Sibbold, K. Solez, A. L. Linton, unpublished work) should help us here.

The size of the problem has been little studied, but during 1982 the EDTA-European Renal Association [44] surveyed the numbers of patients with acute renal failure treated by the units contributing to its Registry – i. e. units also treating patients in chronic renal failure. A total of 16,600 patients with reversible acute renal failure were identified in Europe, of which 1,237 (22.2 per million population/year) were report-

ed from the United Kingdom. This figure was comparatively low when compared with France (1,625, 30.4/10^6/year), FRG (4,378, 71.4/10^6/y) and Italy (2,231, 39.3/10^6/y). Interestingly, the number of cases treated for acute renal failure paralleled those treated for chronic renal failure, which may suggest substantial under-reporting from countries with few units, such as the UK.

Acute renal failure remains a challenge to nephrologists and intensivists, and the relative neglect of clinical and experimental studies on acute renal failure in Europe when compared to the United States is striking. Also, in allocating budgets for both renal and intensive care units, the huge demands made upon staff and money by this small group of patients must not be forgotton. A recent combined conference of anaesthetists, intensivists and nephrologists on the topic of acute combined respiratory and renal failure proved a valuable forum, and we await its proceedings (A. Martin, unpublished work) with interest.

References

1. Minami S (1923) Über Nieren Veränderung nach Verschüttung. Virchow's Arch Pathol Anat 245:247
2. Bywaters EGL, Beall D (1941) Crush injuries with impairment of renal function. Br Med J 1:427
3. Swan RC, Merrill JP (1953) The clinical course of acute renal failure. Medicine (Baltimore) 32:215
4. The Board for the Study of the Severely Wounded (1952) Clinical, physiologic and biochemic correlation in lower nephron nephrosis; Chapt 2. Surgery in World War II. The physiologic effects of wounds. Office of the Surgeon General, Department of the Army, p 122
5. Alwall N (1954) On renal failure complicating surgical diseases (laparotomy etc) with special regard to conservative treatment and the need for the artificial kidney (dialyser, ultrafilter) in rational renal therapy. Acta Med Scand 108:95
6. Smith LH, Post RS, Teschan PE et al. (1955) Post-traumatic renal insufficiency in military casualties. II. Management, use of an artifical kidney, prognosis. Am J Med 18:187
7. Brunner F, Broyer M, Brynger H et al. (1986) Combined report on regular dialysis and transplantation in Europe XV, 1984. Proc Europ Dial Transpl Assoc 22: (in press)
8. Stott RB, Cameron JS, Ogg CS, Bewick M (1972) Why the persistently high mortality in acute renal failure? Lancet 1:75
9. Kennedy AC, Burton JA, Luke RG et al. (1973) Factors affecting the prognosis in acute renal failure. Q J Med 42:73
10. Kleinknecht D, Jungers P, Chanard J, Barbanel C, Ganeval D, Rondon-Nucete M (1971) Factors influencing immediate prognosis in acute renal failure, with special references to prophylactic hemodialysis. Adv Nephrol 1:207
11. Kerr DNS (1979) Acute renal failure. In: Black DAK, Jones NF (eds) Renal disease, Chap 15. Blackwell, Oxford, p 437
12. McMurray SD, Luft FC, Maxwell Dr et al. (1978) Prevailing patterns and predictor variables in patients with acute tubular necrosis. Arch Intern Med 138:950
13. Gornick GC, Kjellstrand CJ (1983) Acure renal failure complicating aortic aneurysm surgery. Nephron 35:145
14. Sweet SJ, Glenney CU, Fitzgibbons JP, Friedmann P, Teres D (1981) Synergistic effect of acute renal failure and respiratory failure in a surgical intensive care unit. Am J Surg 141:492
15. Rasmussen H, Ibels LS (1982) Multivariate analysis of causes and risk factors. Am J Med 73:211
16. Routh GS, Briggs JD, Mone JG, Ledingham JMH (1980) Survival from acute renal failure with and without multiple organ dysfunction. Postgrad Med J 56:244
17. Moyer C, Cena AF, Chenier R et al. (1981) Multiple systems failure. II Death predictors in the trauma septic state. The most critical determinants. J Trauma 81:862
18. Wilkins RG, Faragher EB (1983) Acute renal failure in an intensive care unit: incidence, prediction and outcome. Anaesthesia 38:628
19. Bullock ML, Finkelstein M, Keane WF (1985) The assessment of risk factors in 462 patients with acute renal failure. Am J Kidney Dis 5:97
20. Wardle EN (1984) Shock lungs: the post-traumatic respiratory distress syndrome. Q J Med 53:317
21. Baue AE (1975) Multiple, progressive, or sequential systems failure. A syndrome of the 1970's. Arch Surg 110:779
22. Fry DE, Pearlstein L, Fulton RL, Pol HC Jr (1980) Multiple system organ failure. The role of uncontrolled infection. Arch Surg 115:136
23. Eisman B, Beart R, Norton L (1977) Multiple organ failure. Surg Gynaec Obstet 144:323
24. Fry DE, Garrison RN, Heitsch RC, Calhoun K, Polk HC Jr (1980) Determinants of death in patients with intra-abdominal abscess. Surgery 88:517
25. Pine RW, Wertz MJ, Lennard ES, Dellinger EP, Carnico CJ, Minshew BH (1983) Determinants of organ malfunction or death in patients with intra-abdominal sepsis. A discriminant analysis. Arch Surg 118:242
26. Brenner BM, Lazarus (eds) (1983) Acute renal failure. Saunders, Philadelphia
27. Andreucci V (1984) Pathophysiology of acute renal failure. In: Andreucci V (ed) Acute renal failure: pahtophysiology, prevention, treatment. Martinus Nyhoff, The Hague, p 1
28. Stoff JS, Clive DM (1983) Role of prostaglandins and thromboxane in acute renal failure. In: Acute renal failure. Brenner GM, Lazarus MG (eds) Saunders, Philadelphia, p 157
29. Dawson JL (1965) Post-operative renal failure in obstructive jaundice-effect of a mannitol diuresis. Br Med J 1:82
30. Barry KJ (1963) Post-traumatic renal shut down in humans: its prevention and treatment by the intravenous infusion of mannitol. Milit Med 128:224
31. Henderson IA, Beattie TJ, Kennedy AC (1980) Dopamine hydrochloride in oliguric states. Lancet 2:1329
32. Graziani G, Cantaluppi A, Casati S (1984) Dopamine and frusemide in oliguric acute renal failure. Nephron 37:39
33. Levinsky NG, Bernard DB, Johnston PA (1983) Mannitol and loop diuretics in acute renal failure. In: Brenner GM, Lazarus MG (eds) Acute renal failure. Saunders, Philadelphia, p 712
34. Espinel CH, Gregory AW (1980) Differential diagnosis of acute renal failure. Clin Nephol 13:73
35. Pru C, Kjellstrand CM (1984) The FE_{NA} test is of no value in acute renal failure. Nephron 36:20
36. Brown CB, Ogg CS, Cameron JS (1981) High-dose frusemide in acute renal failure. A controlled trial. Clin Nephrol 35:90
37. Whelton A, Donadio JV Jr (1969) Post-traumatic acute renal failure in Vietnam. A comparison with the Korean War experience. Johns Hopk Med J 124:95
38. Hilberman M, Blyers BD, Carrie BJ et al. (1979) Acute renal failure following cardiac surgery. J Thora Cardiovasc Surg 77:880
39. Wardle N (1982) Acute renal failure in the 1980's: the importance of septic shock and endotoxaemia. Nephron 30:193

40. Zech P, Bouletreau R, Moskovtchenko JF et al. (1971) Infection in acute renal failure. Adv Nephrol 1:231
41. Kornhall S (1971) Acute renal failure in surgical disease with special regard to neglected complications. Acta Chir Scand (Suppl 419):7
42. Lordon RE, Burton JR (1972) Post-traumatic renal failure in military personnel in South-East Asia. Am J Med 53:137
43. Milligan SL, Luft FC, McMurray SD, Kleit SA (1978) Intra-abdominal infection in acute renal failure. Arch Surg 113:467
44. Broyer M, Brunner FP, Brynger H et al. (1983) Combined report on regular dialysis and transplantation in Europe, XIII 1982. Acute (reversible) renal failure. Proc Europ Dial Transp Ass 20:64
45. Brown CB, Cameron JS, Ogg CS, Bewick M, Stott RB (1973) Established acute renal failure following surgical operations. In: Friedman E, Eliahou H (eds) Proceedings of a conference on acute renal failure. Department of Health, Education and Welfare, Washington, p 187

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