The Elderly Patient With Acute Renal Failure[1]

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ABSTRACT

Structural and functional changes observed in the aging kidney predispose the elderly patient to acute renal failure. Up to 36% of the patients with acute renal failure from this institution were over 70 yr, and the literature is full of similar experiences. The elderly patient with abrupt cessation of adequate renal function requires a special work-up in diagnosis and treatment. Prerenal and obstructive causes are of particular interest. Although the question of whether or not age has an independent prognostic importance during an episode of acute renal failure remains debated; when these and other authors compared the outcome of young and old populations with these disorders, a similar evolution was always observed. Age should not be used as a discriminant factor in therapeutic decisions concerning acute renal failure.

Key Words: *Acute renal failure, elderly, prognosis*

Acute renal failure (ARF) supervenes when the kidney is acutely unable to maintain the homeostasis of the organism. In the multicenter study recently carried out in our city (about 4 million habitants), 274 (36%) of 748 patients with ARF seen in the 13 hospitals with nephrology departments during a 9-month period were over 70 yr (1). Because they account for 7% of the general population and 10.5% of all hospital admissions in our country, the prevalence of ARF in older people appears to be over threefold more frequent than expected. A similar proportion of older patients have been attended in our unit suffering from ARF during the past 9 yr (2). The medical literature is full of similar experiences, to the extent that, recently, ARF was considered to be a typical geriatric disease (3).

It is not surprising that the incidence of ARF in the elderly is much greater than in the general population. Of the explanations for this high incidence, in our opinion, four are probably the most important ones: (*1*) the anatomical and physiologic changes occurring in the aging kidney; (*2*) the elevated risk of ARF in the presence of frequent systemic diseases such as arteriosclerosis, hypertension, diabetes, or heart failure; (*3*) the abuse of nephrotoxic drugs in the elderly; and (*4*) the obvious prevalence of obstructive uropathy. The review of the structural and functional changes observed in the aging kidney is essential to understand the high frequency of this condition in the elderly.

STRUCTURE AND FUNCTION OF THE AGING KIDNEY

Early studies in rats demonstrated the development of proteinuria associated with important structural changes in the kidney during the course of aging (4–6). The most striking features were focal or segmental glomerulosclerosis, diffusely increased mesangial matrix, and glomerular and tubular basal membrane thickening. It is unknown whether the proteinuria is the cause or effect of these structural lesions, and other authors suggest a predominant role for immunologic or environmental factors (7–9).

In healthy individuals, the aging kidney is associated with important structural and functional changes. There is a reduction in renal mass of up to 30% by the eighth decade (10–13). There is a 30 to 50% reduction in the number of glomeruli (14–16), with a substantial number of the rest developing focal sclerosis (14,15,17,18). The filtering surface is diminished by a progressive increase in the number of mesangial cells and a reciprocal decrease in the percentage of epithelial cells after the fifth decade; the mesangium increases from 8 to 12% of the total glomeruli volume (10,12,19). There is also a reduction in the size and number of tubules (11) and a significant deposition of connective tissue in the medulla. Glomerular and tubular basement membrane thickening appears (11,20). Diverticula of the distal nephron, almost absent in young individuals, become apparent in aged kidneys (11,20) and may represent the origin of the cysts frequently seen in the elderly (21).

Vascular changes are also important with age. The elegant studies by Takazakura and his colleagues (22) demonstrated that, with aging, there is obliteration of the cortical afferent arterioles with complete atrophy of the glomerular tuft. In addition, spiraling of the juxtamedullary arterioles with subsequent shunting of blood from the afferent to the efferent arterioles with redistribution of blood favoring the medulla was also observed. These structural changes may explain some

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of the physiologic changes observed in the aging kidney:

1. **Up to 50% decrease in RBF from 20 to 80 yr (23,24), probably due to anatomical changes and functional vasoconstriction.**

2. **A progressive decline in GFR (24–27).** Four decades ago, Davies and Shock observed a 50% reduction in GFR between 30 and 90 yr of age (24). In the Baltimore Longitudinal Study of Aging (25), Rowe *et al.* observed a linear reduction of 0.8 mL/min per 1.73 m^2 per year. In that study, however, there was considerable variation in normal values at various ages, with important overlap. Follow-up studies in this cohort with serial creatinine clearance (CrC) determinations obtained during a period of 23 yr showed a mean decrease in CrC with age of 0.75 mL/min per 1.73 m^2 per year (26). However, Rowe *et al.* (25) emphasized that some individuals did not demonstrate a decrease in GFR with aging, even into the ninth decade. It has been postulated that, at least partially, the decrease in GFR with aging may be related to mildly elevated blood pressure (28) or to the dietary intake of protein (29). The recent observation by Fliser *et al.* (30) of an almost normal GFR in normotensive elderly individuals on normal protein intake suggests that age *per se* is not the most relevant factor responsible for the decline of GFR with age. Moreover, those and other authors (31) have demonstrated preservation of renal functional reserve in elderly subjects. Because muscle mass decreases at a rate similar to GFR, the loss of renal function is not reflected by an increase in serum creatinine (SCr) (25). SCr is then inadequate to estimate GFR in the elderly, and as a result, it is necessary to measure or estimate CrC adjusting for age, weight, and sex, when SCr is either unknown (32) or known (33).

Other important factors implicated in the development of age-related glomerulosclerosis are atherosclerotic disease of renal vessels (34) and perhaps an excess of cholesterol or lipid abnormalities (35). Recently, Sonaka *et al.* have observed in rats an age-related decrease in the synthesis of nitric oxide associated with chronic nephropathy (36).

3. **Decreased urinary concentrating ability (11,25, 37–40).** Rowe *et al.* have observed a maximum urinary osmolality of 1,109 mosm/kg at 20 to 39 yr of age and of 882 mosm/kg at 60 to 79 yr (39). Lindeman *et al.* have observed a normal response of the kidney to low-dose vasopressin but an inability to maximally concentrate urine (37). This alteration partially explains the increased incidence of volume depletion and hypernatremia in the elderly (41). The deficit in thirst and the regulation of fluid intake could further contribute (42). The decreased GFR is also probably related to the concentration defect, because remnant functioning nephrons are under the stimulus of osmotic diuresis. An important point revealed by the studies of Macías-Núñez *et al.* (43,44) is a blunted sodium chloride reabsorption in the ascending limb of the loop of Henle in elderly people.

4. **Decreased diluting capacity (37), with a high risk of water intoxication and hyponatremia after moderate administrations of water or diuretics (45–47).**

5. **Inability to retain sodium when the aged individual is salt deprived (38), possibly due to nephron loss or impaired aldosterone secretion (48).** Despite a lower sodium tubular load as a result of lower GFR, 24-h urinary sodium and fractional excretion of sodium are greater in the elderly (44). As mentioned above, the alteration of the tubular handling of sodium appears to be related to a decreased capacity to reabsorb sodium by the thick ascending limb of the loop of Henle (43).

6. **Impaired renal acid excretion (49), with less efficacy in buffering.**

7. **Decreased PRA (50), with subsequent decreased aldosterone (48).**

In spite of all of the aforementioned anatomical and physiologic alterations, the aging kidney is quite capable of maintaining fluid and electrolyte balance under normal conditions. However, its adaptative capacity is restricted, and during systemic disease, hemodynamic or abrupt intravascular volume changes, the administration of toxic drugs, or other insults, significant disorders may emerge.

PRERENAL ARF

Prerenal or functional ARF occurs when poor perfusion is causing the failure of renal function. This type of ARF is of special importance in the elderly population, and the physiologic changes detailed above have particular significance in this setting. In a patient with

TABLE 1. Causes of ARF in the elderly occur more frequently than in younger populations

Prerenal ARF
External loss of fluids
Vomiting/diarrhea
Insufficient fluid replacement
Uncontrolled use of diuretics
Internal volume redistribution to interstitial spaces
Decreased cardiac output
Myocardial dysfunction
Pericardial disease
Use of drugs that alter intrarenal hemodynamics
ACEI
NSAID
Intrinsic ARF
Postischemic established prerenal failure
Nephrotoxic
antibiotics
radiocontrast dyes
Rapidly progressive glomerulonephritis
Renal artery occlusion or thromboembolism
Atheroembolic renal disease
Obstructive ARF
Prostatic hypertrophy or carcinoma
Other urologic or gynecologic neoplasms

TABLE 2. Renal biopsy in 180 elderly patients presenting with ARF and rapidly progressive renal failure (73–77)

Disease	N (%)
Rapidly Progressive Glomerulonephritis	61 (33.9)
Vascular Disease	41 (22.8)
Other Glomerulonephritis	32 (17.8)
ATN	19 (10.5)
AIN	14 (7.8)
Other Diseases	13 (7.2)

reduced RBF and GFR, a physiologic situation in the elderly, a subsequent decrease in renal perfusion associated with stimulated sympathetic activity and the release of vasoconstrictor substances during acute hypotension can lead to a further reduction of GFR and ARF. The decreased perfusion of the kidneys easily leads to a reversible state of renal failure, recovered after the correction of hemodynamic disturbances (51).

Loss of fluids, internal redistribution, decreased cardiac output, and certain drugs are responsible for the vast majority of cases of prerenal ARF in older patients. The most frequent causes are the external loss of fluids due to vomiting, diarrhea, bleeding, or excessive sweating, with insufficient fluid replacement and/or uncontrolled use of potent diuretics (51). We might include here those cases produced by dehydration of either origin, in a particularly susceptible geriatric population. More than half of prerenal ARF cases in patients aged over 70 at our institution were secondary to dehydration (2). Accordingly, in a series of 122 elderly patients from 1961 to 1972 (52), the most common cause of ARF was dehydration. Obviously, the decreased urinary concentrating ability (37–39), the impairment of thirst regulation (42), and the inability to retain sodium (38), all characteristics of the aged patient, contribute to this high prevalence.

Internal redistribution from intravascular to interstitial space in states of hypoproteinemia (nephrotic syndrome, cirrhosis or malnutrition) or during important episodes of tissue injury (burns or pancreatitis) is also a major cause of decreased effective circulating volume and prerenal ARF, particularly in geriatric patients. Surgical procedures and the use of nephrotoxic agents are evident aggravating factors. Decreased cardiac output after myocardial dysfunction or pericardial disease may also induce a similar prerenal ARF (Table 1).

The use of drugs that alter intrarenal hemodynamics is a growing cause of prerenal ARF in the elderly. Angiotensin-converting enzyme inhibitors (ACEI) are now widely used to treat hypertension, congestive heart failure, and (as secondary prevention) myocardial infarction. Functional ARF is a well-known complication, suggesting bilateral renovascular disease or arterial stenosis in a solitary kidney (53). Of particular interest is the increasing frequency of ARF secondary to ACEI use in patients without renal artery stenosis (54–56). In a recent French series (56), 67.5% of all ACEI-related ARF cases during a 4-yr period occurred in patients without renal arterial stenosis. Mean age was 65 yr, and volume depletion, concomitant treatment with diuretics, and/or a low-salt diet were commonly associated. Other facilitating factors were cardiac failure, combined treatment with nonsteroidal anti-inflammatory drugs (NSAID) or the presence of diabetes mellitus. Sodium repletion and drug withdrawal of the ACEI usually result in prompt renal recovery, but ARF is not always reversible, particularly if diabetes mellitus is associated (56).

Elderly patients are frequently prescribed NSAID to treat rheumatic diseases and are at risk for NSAID-related ARF (57–63). In these patients, the regulation of RBF and glomerular filtration depends on prostacyclin production. NSAID inhibits this production, and impairment of renal function can result (57). Blackshear *et al.* (58) described seven patients with a mean age of 70 yr who developed reversible ARF after NSAID exposure. Six of them were on concomitant diuretic therapy. In a recent prospective evaluation of 114 patients with a mean age of 87 yr exposed to NSAID, Gurwitz *et al.* (62) demonstrated reversible azotemia in most of the individuals. In addition, concurrent loop diuretic therapy and high NSAID dose were significant predictors of a more than 50% increase in serum urea nitrogen level. As with the use of aminoglycosides, the best approach to reduce NSAID-related renal toxicity in the elderly is to avoid the use of these agents whenever possible. If treatment becomes necessary, the patient must be closely monitored. The unusual metabolism of sulindac may make it less nephrotoxic than other NSAID (64).

A urinary osmolality of more than 500 mosm/kg, a urinary sodium of less than 20 mEq/L, a urine-to-plasma creatinine ratio of more than 40, and a fractional excretion of sodium of less than 1 suggest prerenal ARF (65). The value of these indices has been questioned (66), and other causes of ARF have been reported to show very low fractional excretion of sodium (67–70). Although prerenal ARF is usually associated with oliguria, urinary concentrating defects in the setting of reversible ARF might result in prerenal polyuric ARF (71). Consequently, urinary indices do not always differentiate prerenal ARF from other forms, and in addition, it is unknown whether these parameters are reliable indicators of prerenal failure or if they merely reflect age-related disturbances in the tubular handling of sodium and water. Response to treatment in terms of reversibility after a few days is probably the most reliable indicator of the prerenal state. It should be noted however that old patients may have a delayed response to volume expansion (72).

PARENCHYMATOUS ARF

Acute tubular necrosis (ATN), acute interstitial nephritis, acute glomerulonephritis, and vascular dis-

ease are the most common causes of intrinsic ARF in the elderly when only biopsy cases are considered (73–77) (Table 2). One-third of elderly patients who are biopsied suffer from ARF or rapidly progressive renal failure, as has been shown in a recent review of 314 biopsies performed in patients over 65 yr at a single institution over a period of 20 yr (77). Vasculitis and idiopathic crescentic glomerulonephritis make up nearly half of these cases. However, many more patients who are not biopsied suffer from other forms of intrinsic ARF. In elderly patients with prerenal ARF secondary to volume depletion, delayed restitution of normal extracellular volume may lead to postischemic ARF. It is very important to differentiate between prerenal ARF or established ATN. This can usually be achieved with the aid of history, clinical examination, urinary indices and even central venous catheterization.

ATN Secondary to Surgery or Sepsis

Surgical interventions account for about one-third of ATN cases in elderly patients (2,3), and ATN after cardiac surgery (78–80) and ruptured aortic aneurysms (81,82) is associated with poor prognosis. Hypotension during and after surgery, postoperative fluid loss, and arrythmias are common features in the elderly and may induce hemodynamically mediated ARF. Vigorous volume loading before and during surgery are critical to reduce the high incidence of ARF in these settings (83–86). Careful attention to nutritional issues and the prevention and treatment of postoperative infection are of special interest in older patients, because severe infections often result in ATN with quite a poor prognosis (1–3,87–103). The problem of sepsis and renal failure remains a great challenge to nephrologists (104–106).

Nephrotoxic Agents

Besides ACEI and NSAID, agents known to cause functional ARF, multipharmacy, and the easy availability of multiple nephrotoxic drugs have increased the incidence of nephrotoxic ATN in the elderly (3,107–110). Most of the potent antibiotics currently used to treat ambulatory and, particularly, in-hospital infections are frequently associated with iatrogenic ATN. Age is a well-known risk factor for developing aminoglycoside nephrotoxicity (111,112). Preexisting age-related disturbances and renal disease, volume depletion, and overdosage are contributory factors. Emphasis must be placed on prevention, and physicians should avoid aminoglycoside treatment in older patients. When absolutely necessary for certain infections, monitoring levels of the drug in the blood seems essential.

The elderly are also at increased risk of radiocontrast-induced ARF (113–121). In a prospective study of 183 patients aged over 70 yr undergoing 199 cardiac catheterizations, 11% had an increase in SCr of more than 44 μmol/L above baseline (121). The evolution appears to be similar to that of contrast nephropathy among younger individuals. Other factors such as preexisting renal failure, vascular disease, volume depletion, or diabetes could predispose elderly patients to enhanced renal toxicity. These high-risk patients should not undergo contrast procedures unless strictly necessary. The protective effects of mannitol (122,123) are not clear (124), and its use in elderly patients may even induce severe ARF (125). Low-dose dopamine and/or furosemide (126,127) have been added with variable results. The use of recently available low-osmolality contrast agents has also been associated with ARF (128). Saline infusion before and after the procedure could be of value in preventing contrast-induced ARF, as has recently been demonstrated by our group in high-risk patients (129).

Acute Interstitial Nephritis

Acute interstitial nephritis (AIN), a syndrome caused by a variety of agents, is characterized by ARF and pathologic features such as edema and inflammatory infiltrates in the interstitium. At present, allergic AIN secondary to drugs is the most commonly reported (130,131). It is sometimes difficult to distinguish whether the ARF episode is due to AIN or to the underlying disease. AIN affects all ages and probably does not have any special implications for the elderly, except for its relation with the aging kidney and a possible higher prevalence of NSAID-induced AIN (131).

Acute Glomerulonephritis

Acute glomerulonephritis is a well-known cause of ARF in the elderly (73,75–77,132–136). Diffuse proliferative forms usually occur associated with major infections (134), and the good outcome is similar to that in younger populations (73,132,134,136). Circulatory congestion and renal failure are frequent features in elderly patients, whereas hypertension and edema are important in younger individuals. Because presenting symptoms are frequently masked by coexistent disease, the diagnosis can be overlooked and the prevalence might be higher than described. Rapidly progressive forms appear to be more prevalent in geriatric populations (73,77,134,136). Summarizing four renal biopsy studies (73,75–77), this was the single most commonly made diagnosis in elderly patients presenting with ARF and undergoing renal biopsy (Table 2). Rapidly progressive glomerulonephritis in the elderly carries a poor prognosis. Of seven patients described in a single institution, four died during the recovery phase and the remaining three required maintenance hemodialysis (136). Although stabilization or improvement with high-dose steroids has been reported (136,137), no specific studies on this therapy in the elderly are available.

Vascular Disease

Renal artery occlusion or thromboembolism may cause ARF and is usually associated with atheroscle-

rosis, atrial fibrillation, or myocardial infarction (138–143). Probably, the most frequent presentation of ARF with underlying renal vascular disease in the elderly is after the administration of ACEI (53–56), as has been mentioned above. Less frequent is ARF secondary to acute arterial obstruction. During 12 yr, we have attended 18 arteriographically confirmed such cases (142). The mean age was 62 years, and 10 patients had atrial fibrillation. Mortality was 50%, and five of six patients aged over 70 yr died, whereas only two of nine patients under 60 yr died during the study period. Therefore, the prognosis seems to be quite poor for elderly patients with this severe form of ARF.

Atheroembolic renal disease is gaining increased interest as a major form of ARF in the elderly. Cholesterol crystals dislodged from atherosclerotic plaques occlude intralobular arteries of the kidney and other organs such as the brain or retina after procedures such as aortic catheterization, arteriography, or vascular surgery, or even after minor abdominal trauma (144–148). "Spontaneous" cases have also been reported (145). ARF develops within 1 to 4 wk, and progression to ESRD is frequent (144). A variable combination of signs and symptoms can be present, disclosing distal or organic ischemia. The authors of several recent large studies have emphasized that atheroembolic renal disease is more common than was previously thought and must always be considered in the differential diagnosis of a multisystemic disease in elderly patients (146–148). At present, no treatment is available, and it is advisable to avoid invasive procedures in elderly patients whenever possible.

Renal vasculitis is gaining importance as a cause of ARF in the elderly (82,149). In this population, diagnosis is difficult because of the frequent onset in the form of a febrile, nonspecific syndrome. The prognosis has improved considerably with steroid and immunosuppressive treatment but remains quite poor.

Rhabdomyolysis

Older patients are susceptible to developing rhabdomyolysis in the setting of acute immobilization, infectious disease, cerebrovascular accidents, hyperosmolar state, hyponatremia, hypernatremia, hypothermia, and a fall. Marcus *et al.* have reported a series of 17 old patients with ARF secondary to rhabdomyolysis (150). These patients had a mild form of ARF, none of them required dialysis, and all but one—who died of respiratory failure—had an uneventful recovery. This form of ARF is probably much more frequent than it is diagnosed, because acute immobilization resulting from acute illness is particularly prevalent in the geriatric population.

OBSTRUCTIVE ARF

Urinary obstruction is one of the most significant causes of ARF in the elderly (151,152). Prostatic hypertrophy or carcinoma, retroperitoneal or pelvic neoplasia such as lymphoma, and carcinoma of the bladder, cervix, uterus, ovaries, or rectum are frequent causes of obstructive ARF in the geriatric population. Benign prostatic hyperplasia is the most common neoplasm in men, affecting 50% of men aged 50 yr and increasing to 90% by the ninth decade of life (153). A small percentage of these patients develop severe obstructive ARF, some of them progressing to irreversible renal failure, whereas others recover quite well. A longer mean duration of symptoms and a higher incidence of small kidneys with increased parenchymal echogenicity appear to be related to irreversible renal dysfunction in elderly patients presenting with obstructive benign prostatic hyperplasia (154).

It is always imperative to exclude a possible obstructive cause in a patient presenting with ARF, especially in those situations of previous uropathy or recent abdominal surgery. Prompt intervention may result in improvement or complete recovery of renal function. Elderly men with unexplained ARF should undergo bladder catheterization. A brisk diuresis usually begins in patients with obstructive ARF at the bladder neck level, but the physician must verify concomitant improvement in renal function, because some patients with large volumes of urine after catheterization do not show a fall in SCr.

Unfortunately, all of the laboratory findings with obstructive ARF are nonspecific and difficult to interpret. Usually, they are similar to those seen with ATN: high urinary sodium and decreased osmolality (65,151,155,156). However, some patients may have high urinary osmolality and low urinary sodium concentration, simulating prerenal ARF (155,156). Urinalysis is normal or reveals only a few red or white blood cells or mild proteinuria.

Ultrasonography is safe, sensible, and readily available and has become the initial investigation of choice in this setting (157–162). The diagnosis of obstruction is based on a dilated collecting system filled with fluid. Retrograde ureteropyelography usually makes the etiologic diagnosis if ureteral catheterization is feasible. If this is not the case, percutaneous or even surgical nephrostomy with anterograde pyelography has both diagnostic and therapeutic usefulness (163–165). However, a few cases of urinary tract obstruction have been reported to be associated with minimal or no dilation of the collecting system (163–167). Most of them are men with a prior history of malignancy, microscopic or gross hematuria without casts, and severe anuric or oliguric ARF of unknown origin. Curiously, some of these patients did have minimal dilation (166,168), but it was thought to be not severe enough to explain the degree of renal failure. If ultrasonography reveals nondilated kidneys but obstructive ARF is still suspected, duplex-Doppler sonography is probably a useful tool (169). Renal arterial resistance rises with obstruction of the kidneys. A resistive index above 0.7 suggests obstructive ARF and requires mandatory ureteral catheterization. Computed tomography plays a minor role in this

diagnostic work-up but can be a valuable adjunct in patients with inadequate ultrasonographic visualization or with an unidentified cause of obstruction (160,168).

PROGNOSIS

The question as to whether or not age has independent prognostic importance during an episode of ARF remains unanswered. In our study on the prognosis of ARF in patients over 70 yr (2), although overall mortality was slightly greater in the elderly, the difference from younger patients did not reach statistical significance by the use of univariate analysis (45 versus 41%). In this extensive group of patients suffering from all types of ARF, we failed to show any relationship between advanced age and prognosis of ARF, in agreement with some authors (52,78,88,92,96,97,99, 100,103,170–184) but in conflict with others (81,82, 87,89–91,93–95,98,105,185–194). When considering only ATN cases in an initial forward analysis of 228 patients at our institution (102), age was similar in survivors (57 yr) to that of nonsurvivors (60 yr). When three age groups were considered (<30, 30 to 60, and >60 yr), mortality rates were comparable (102). Additionally, age had no significant influence on mortality when developing a multiple linear regression model (102). However, in a further prospective evaluation including 100 consecutive additional cases (synthetic phase comprising 328 ATN patients), age was an independent determinant of outcome by the use of a multiple linear regression model (195). The survival rate fell slowly but steadily along with each decade of life. As can be seen, the influence of age on outcome of ARF is debated, even within our own experience. Of interest, when we and other authors have compared the outcome of old and young patients with ARF, a similar evolution was always observed (2,52,173,179, 182,183,176,184). It seems reasonable to conclude that elderly patients have a moderately worse prognosis than younger patients, but age should not be used as a discriminant factor in therapeutic decisions concerning ARF. Instead, early clinical features such as hypotension, assisted respiration, or coma are decidedly related to mortality in ARF patients of any age (79,98,102,186,190,191,195,196).

Three recent studies have explored the evolution of ARF prognosis during the last 15 (176,197) and 30 yr (194). Biesenbach *et al.* (197) showed that the mortality of all patients with ARF attended in their dialysis center was reduced from 69% in the years 1975 to 1979 to 48% in 1985 to 1989, although the mean age of the patients had increased from 44 to 58 yr, respectively. Druml *et al.* (176) examined the historical evolution in the elderly (>65 yr) population with ARF treated at their intensive care unit. Prognosis improved significantly during the years since 1975, despite the fact that the disease severity increased. Essentially similar results were observed by Turney *et al.* (194).

There are few data concerning patients' survival and renal long-term prognosis in ARF in the aged. We have observed that elderly patients who survived after an ARF episode needed more time for total recovery and showed a lower level of renal function than did younger survivors (2). All of our survivors recovered enough renal function to be removed from dialysis (2), in agreement with a very recent study (179) and in contrast with an earlier one, in which age was found to be a determinant of the recovery of renal function (92). Long-term prognosis seems to be quite good for elderly survivors (179). Large prospective, preferably multicenter, studies are needed to reveal the predictive value of age and other demographic and clinical parameters in the early and late outcome of ARF in the elderly.

REFERENCES

1. **Liaño F, Pascual J, and the Madrid Acute Renal Failure Study Group:** Course and prognosis of acute renal failure in the very old: results of a multicenter survey in Madrid, Spain [Abstract]. J Am Soc Nephrol 1994;5: 400.
2. **Pascual J, Orofino L, Liaño F, *et al.*:** Incidence and prognosis of acute renal failure in older patients. J Am Geriatr Soc 1990;38:25–30.
3. **Rosenfeld JB, Shohat J, Grosskopf I, Boner G:** Acute renal failure: a disease of the elderly? Adv Nephrol 1987;16:159–167.
4. **Berg BN:** Spontaneous nephrosis with proteinuria, hyperglobinemia and hypercholesterolemia in the rat. Proc Soc Exp Biol Med 1965;119:417–420.
5. **Everitt AV:** The urinary excretion of protein, nonprotein nitrogen, creatinine and uric acid in aging male rats. Gerontology 1958;2:33–46.
6. **Scott EB:** Modification of the basal architecture of renal tubule cells in aged rats. Proc Soc Exp Biol Med 1964; 117:586–598.
7. **Bolton WK, Benton FA, McLay JG, *et al.*:** Spontaneous glomerular sclerosis in aging Sprague Dawley rats. I. Lesion associated with mesangial IgM deposits. Am J Pathol 1976;85:277–302.
8. **Bolton WK, Sturgill BC:** Spontaneous glomerular sclerosis in aging Sprague Dawley rats. II. Ultrastructural studies. Am J Pathol 1980;98:339–356.
9. **Linder E, Pasternack A, Edgington IS:** Pathology and immunology of age-associated disease in mice and evidence for an autologous immune pathogenesis of the associated renal diseases. Clin Immunol Immunopathol 1972;1:104–121.
10. **Tauchi H, Tsuboi K, Okutomi J:** Age changes in the human kidney of the different races. Gerontology 1971; 17:87–97.
11. **Lindeman RD, Goldman R:** Anatomic and physiologic age changes in the kidney. Exp Gerontol 1986;21:379–406.
12. **Levi M, Rowe JW:** Aging and the kidney. In: Schrier RW, Gottschalk CW, Eds. Diseases of the Kidney. 5th Ed. Boston: Little Brown; 1993:2405–2432.
13. **Lindeman RD:** Overview: Renal physiology and pathophysiology of aging. Am J Kidney Dis 1990;16:275–282.
14. **Frocht A, Fillit H:** Renal disease in the geriatric patient. J Am Geriatr Soc 1984;32:28–43.
15. **McLachlan MSF, Guthrie JC, Anderson CK, *et al.*:** Vascular and glomerular changes in the aging kidney. J Pathol 1977;121:65–78.
16. **Goyal VK:** Changes with age in the human kidney. Exp Gerontol 1982;17:321–331.
17. **Kaplan C, Pasternack B, Shah H, *et al.*:** Age-related incidence of sclerotic glomeruli in human kidneys. Am J Pathol 1975;80:227–234.
18. **Sworn MJ, Fox M:** Donor kidney selection for trans-

plantation-relationships between glomerular structure, vascular supply and age. Br J Surg 1972;59:310–311.
19. **Sorenson FH:** Quantitative studies of the renal corpuscles IV. Acta Pathol Microbiol Immunol Scand 1977;85: 356–366.
20. **Darmady EM, Offer J, Woodhouse MA:** The parameters of the aging kidney. J Pathol 1973;109:195–209.
21. **Baert L, Steg A:** Is the diverticulum of the distal and collecting tubules a preliminary stage of the simple cyst in the adult? J Urol 1977;118:707–710.
22. **Takazakura E, Wasabu N, Handa A, *et al.*:** Intrarenal vascular changes with age and disease. Kidney Int 1972;2:224–230.
23. **Hollenberg NK, Adams DF, Solomon HS:** Senescence and renal vasculature in normal man. Circ Res 1974; 34:309–316.
24. **Davies DF, Shock NW:** Age change in glomerular filtration rate, effective renal plasma flow, and tubular excretory capacity in adult males. J Clin Invest 1950;29: 496–507.
25. **Rowe JW, Andres R, Tobin JD, Norris AH, Shock NW:** The effect of age on creatinine clearance in men: a cross-sectional and longitudinal study. J Gerontol 1976;31:155–163.
26. **Lindeman RD, Tobin J, Shock NW:** Longitudinal studies on the rate of decline in renal function with age. J Am Geriatr Soc 1985;33:278–285.
27. **Hadj-Aissa A, Dumarest C, Maire P, Pozet N:** Renal function in the elderly. Nephron 1990;54:364–365.
28. **Lindeman RD, Tobin J, Shock NW:** Association between blood pressure and the rate of decline in renal function with age. Kidney Int 1984;26:861–868.
29. **Lew SQ, Bosch JP:** Effect of diet on creatinine clearance and excretion in young and elderly healthy subjects and in patients with renal disease. J Am Soc Nephrol 1991; 2:856–865.
30. **Fliser D, Zeier M, Nowack R, Ritz E:** Renal functional reserve in healthy elderly subjects. J Am Soc Nephrol 1993;3:1371–1377.
31. **Böhler J, Glöer D, Reetze-Bonorden P, Keller E, Schollmeyer PJ:** Renal functional reserve in elderly patients. Clin Nephrol 1993;39:145–150.
32. **Rowe JW, Andres R, Tobin JD, *et al.*:** Age-adjusted standards for creatinine clearance. Ann Intern Med 1976;84:567–569.
33. **Cockcroft DW, Gault MH:** Prediction of creatinine clearance from serum creatinine. Nephron 1976;16:31–41.
34. **Kasiske BL:** Relationship between vascular disease and age-associated changes in the human kidney. Kidney Int 1987;31:1153–1159.
35. **Levi M, Jameson DM, Van Der Meer BW:** Role of BBM lipid composition and fluidity in impaired renal Pi transport in the aged rat. Am J Physiol 1989;256:F85–F94.
36. **Sonaka I, Futami Y, Maki T:** L-arginine nitric oxide pathway and chronic nephropathy in rats. J Gerontol 1994;49:B157–B161.
37. **Lindeman RD, Lee TD, Yiengst MJ, *et al.*:** Influence of age, renal disease, hypertension, diuretics and calcium on the antidiuretic responses to suboptimal infusions of vasopressin. J Lab Clin Med 1968;68:206–223.
38. **Epstein M, Hollenberg NK:** Age as a determinant of renal sodium conservation in normal man. J Lab Clin Med 1972;87:411–417.
39. **Rowe JW, Shock NW, De Fronzo RA:** The influence of age on the renal response to water deprivation in man. Nephron 1976;17:270–278.
40. **Helderman JH, Vestal RE, Rowe JW, *et al.*:** The response of arginine vasopressin to intravenous ethanol and hypertonic saline in man: The impact of aging. J Gerontol 1978;33:39–47.
41. **Snyder NA, Feigal DW, Arieff AI:** Hypernatremia in elderly patients. Ann Intern Med 1987;107:309–319.
42. **Miller PD, Krebs RA, Neal BJ, McIntyre DO:** Hypodipsia in geriatric patients. Am J Med 1982;73:354–356.
43. **Macías Núñez JF, García Iglesias A, Bonda Roman JL, *et al.*:** Renal handling of sodium in old people: a functional study. Age Aging 1978;7:178–181.
44. **Macías Núñez JF, García Iglesias C, Tabernero Romo JM, *et al.*:** Renal management of sodium under indomethacin and aldosterone in the elderly. Age Aging 1980;9:165–172.
45. **Booker JA:** Severe symptomatic hyponatremia in elderly outpatients: The role of thiazide therapy and stress. J Am Geriatr Soc 1984;32:108–113.
46. **Kleinfeld J, Casimir M, Borra S:** Hyponatremia as observed in a chronic disease facility. J Am Geriatr Soc 1979;27:156–161.
47. **Sunderam SG, Mankikar GD:** Hyponatremia in the elderly. Age Aging 1983;12:77–80.
48. **Flood C, Gherondache C, Pincus G, *et al.*:** The metabolism and secretion of aldosterone in elderly subjects. J Clin Invest 1967;46:960–966.
49. **Adler S, Lindeman RD, Yiengst MJ, *et al.*:** Effect of acute acid loading on urinary acid excretion by the aging human kidney. J Lab Clin Med 1968;72:278–289.
50. **Crane MC, Harris JJ:** Effects of aging on renin activity and aldosterone excretion. J Lab Clin Med 1976;87: 947–959.
51. **Burnett JC:** Acute renal failure associated with cardiac failure and hypovolemia. In: Lazarus JM, Brenner BM, Eds. Acute Renal Failure. 3rd Ed. New York: Churchill Livingstone; 1993:193–206.
52. **Kumar R, Hill CM, McGeown MG:** Acute renal failure in the elderly. Lancet 1973;1:90–91.
53. **Hricik DE, Browning PJ, Kopelman R, *et al.*:** Captopril-induced functional renal insufficiency in patients with bilateral renal-artery stenoses or renal-artery stenosis in a solitary kidney. N Engl J Med 1983;308:373–376.
54. **Murphy BF, Whitworth JA, Kincaid-Smith P:** Renal insufficiency with combinations of angiotensin-converting inhibitors and diuretics. BMJ 1984;288: 844–845.
55. **Paker M, Lee WH, Medina N, Yushak M, Kessler PD:** Functional renal insufficiency during long-term therapy with captopril and enalapril in severe chronic heart failure. Ann Intern Med 1987;106:346–354.
56. **Bridoux F, Hazzan M, Pallot JL, *et al.*:** Acute renal failure after the use of angiotensin-converting-enzyme inhibitors in patients without renal artery stenosis. Nephrol Dial Transplant 1992;7:100–104.
57. **Blackshear JL, Napier JS, Davidman M, Stillman MT:** Renal complications of nonsteroidal antiinflammatory drugs: Identification and monitoring of those at risk. Semin Arthritis Rheum 1985;14:163–175.
58. **Blackshear JL, Davidman M, Stillman M:** Identification of risk for renal insufficiency from nonsteroidal antiinflammatory drugs. Arch Intern Med 1983;143:1130–1134.
59. **Garella S, Matarese RA:** Renal effects of prostaglandins and clinical adverse effects of nonsteroidal antiinflammatory agents. Medicine 1984;63:165–181.
60. **Lamy PP:** Renal effects of nonsteroidal antiinflammatory drugs. Heightened risk to the elderly? J Am Geriatr Soc 1986;34:361–367.
61. **Clive DM, Stoff JS:** Renal syndromes associated with nonsteroidal antiinflammatory drugs. N Engl J Med 1984;310:563–572.
62. **Gurwitz JH, Avorn J, Ross-Degnan D, Lipsitz LA:** Nonsteroidal anti-inflammatory drug-associated azotemia in the very old. JAMA 1990;264:471–475.
63. Nephrotoxicity of non-steroidal anti-inflammatory drugs. Lancet 1994;2:515–518.
64. **Whelton A, Stout RL, Spilman PS, Klassen DK:** Renal effects of ibuprofen, piroxicam and sulindac in patients with asymptomatic renal failure. A prospective, randomized, crossover comparison. Ann Intern Med 1990; 112:568–576.
65. **Miller TR, Anderson RJ, Linas SL, *et al.*:** Urinary diagnostic indices in acute renal failure. A prospective study. Ann Intern Med 1978;89:47–50.
66. **Pru C, Kjellstrand CM:** Indices and urinary chemistries in the differential diagnosis of pre-renal failure and acute tubular necrosis. Semin Nephrol 1985;5:224–233.

67. **Van Ypersele de Strihou C:** Acute oliguric interstitial nephritis. Kidney Int 1979;16:751–765.
68. **Diamond JR, Yoburn DC:** Nonoliguric acute renal failure. Arch Intern Med 1982;142:1882–1898.
69. **Domínguez F, Orofino L, Quereda C, Ortuño J:** Excreción fraccional de sodio baja en la necrosis tubular aguda por rabdomiolisis. Nefrología 1987;4:409–410.
70. **Zarich S, Fang LST, Diamond JR:** Fractional excretion of sodium: exceptions to its use. Arch Intern Med 1985;145:108–112.
71. **Miller PD, Krebs RA, Neal BJ, McIntyre DO:** Polyuric prerenal failure. Arch Intern Med 1980;190:907–909.
72. **Macías Núñez JF, Sánchez Tomero JA:** Acute renal failure in old people. In: Macías Núñez JF, Cameron JS, Eds. Renal Function and Disease in the Elderly. London: Butterworths; 1987:461–484.
73. **Moorthy AV, Zimmerman SW:** Renal disease in the elderly: Clinicopathological analysis of renal disease in 115 elderly patients. Clin Nephrol 1980;14:223–229.
74. **Hariharan S, Date A, Kirubakaran MG, *et al.*:** Medical renal disease in the elderly in a southern Indian hospital. Nephron 1988;49:119–121.
75. **Kingswood JC, Banks RA, Tribe CR, *et al.*:** Renal biopsy in the elderly: Clinicopathological correlations in 143 patients. Clin Nephrol 1984;22:183–187.
76. **Preston RA, Stemmer CL, Materson BJ, Pérez-Stable, Pardo V:** Renal biopsy in patients 65 years of age or older. An analysis of the results of 334 biopsies. J Am Geriatr Soc 1990;38:669–674.
77. **Modesto-Segonds A, Ah-Soune MF, Durand D, Suc JM:** Renal biopsy in the elderly. Am J Nephrol 1993;13:27–34.
78. **Lange HW, Aepli DM, Brown DC:** Survival of patients with acute renal failure requiring dialysis after open heart surgery: Early prognostic indicators. Am Heart J 1987;113:1138–1143.
79. **Corwin HL, Sprague SM, De Laria GA, Norusis MJ:** Acute renal failure associated with cardiac operations. J Thorac Cardiovasc Surg 1989;98:1107–1112.
80. **Salomon NW, Page S, Bigelow JC, Krause AH, Okies JE, Metzdorff MT:** Coronary artery bypass grafting in elderly patients. J Thorac Cardiovasc Surg 1991;101: 209–218.
81. **Gornick CC, Kjellstrand CM:** Acute renal failure complicating aortic aneurysm surgery. Nephron 1983;35: 145–157.
82. **Berisa F, Beaman M, Adu D, *et al.*:** Prognostic factors in acute renal failure following aortic aneurysm surgery. Q J Med 1990;76:689–698.
83. **Bush HL, Huse JB, Johnson WC, *et al.*:** Prevention of renal insufficiency after abdominal aortic aneurysm resection by optimal volume loading. Arch Surg 1981; 116:1517–1524.
84. **Hesdorffer CS, Milne JF, Meyers AM, Clinton C, Botha R:** The value of Swan-Ganz catheterization and volume loading in preventing renal failure in patients undergoing abdominal aneurisnectomy. Clin Nephrol 1987;28: 272–276.
85. **Beck LH:** Perioperative renal, fluid and electrolyte management. Clin Geriatr Med 1990;6:557–569.
86. **Kellerman PS:** Perioperative care of the renal patient. Arch Intern Med 1994;154:1674–1688.
87. **Swann RC, Merrill JP:** The clinical course of acute renal failure. Medicine 1953;32:215–292.
88. **Bluemle LW, Webster GD, Elkinton JR:** Acute tubular necrosis. Analysis of 100 cases with respect to mortality, complications and treatment with and without dialysis. Arch Intern Med 1959;104:180–197.
89. **Kiley JE, Powers SR, Beebe R:** Acute renal failure: Eighty cases of renal tubular necrosis. N Engl J Med 1960;262:481–486.
90. **Balslov JT, Jorgensen HE:** A survey of 499 patients with acute anuric renal insufficiency. Am J Med 1963; 34:753–764.
91. **Lunding M, Steiness I, Thaysen JS:** Acute renal failure due to tubular necrosis. Immediate prognosis and complications. Acta Med Scand 1964;176:103–119.
92. **Hall JW, Johnson WJ, Maher FT, Hunt JC:** Immediate and long-term prognosis in acute renal failure. Ann Intern Med 1970;73:515–521.
93. **Kleinknecht D, Jungers P, Chanard J, *et al.*:** Factors influencing immediate prognosis in acute renal failure with special reference to prophylactic hemodialysis. Adv Nephrol 1971;1:207–230.
94. **Stott RB, Ogg CS, Cameron JS, Bewick M:** Why the persistently high mortality in acute renal failure? Lancet 1972;2:75–79.
95. **Kennedy AC, Burton JA, Luke RG, *et al.*:** Factors affecting the prognosis in acute renal failure. A survey of 251 cases. Q J Med 1973;42:73–86.
96. **Minuth AN, Terrell JB, Suki WN:** Acute renal failure. A study of the course and prognosis of 104 patients and the role of furosemide. Am J Med Sci 1976;271:317–324.
97. **Hou HS, Bushinsky DA, Wish JB, Cohen JJ, Harring JT:** Hospital-acquired renal insufficiency. A prospective study. Am J Med 1983;74:243–246.
98. **Bullock ML, Umen AJ, Finkelstein M, Keane WF:** The assessment of risk factors in 462 patients with acute renal failure. Am J Kidney Dis 1985;5:97–103.
99. **Abreo K, Moorthy V, Osborne M:** Changing patterns and outcome of acute renal failure requiring hemodialysis. Arch Intern Med 1986;146:1338–1341.
100. **Corwin HL, Teplick RS, Schreiber MJ, Fang LST, Bonventure JV:** Prediction of outcome in acute renal failure. Am J Nephrol 1987;7:8–12.
101. **Rayner BL, Wilcox PA, Pascoe MD:** Acute renal failure in community-acquired bacteremia. Nephron 1990;54: 32–35.
102. **Liaño F, García-Martin F, Gallego A, *et al.*:** Easy and early prognosis in acute tubular necrosis: A forward analysis of 228 cases. Nephron 1989;51:307–313.
103. **Spurney RF, Fulkerson WJ, Schwab SJ:** Acute renal failure in critically ill patients: Prognosis for recovery of kidney function after prolonged dialysis support. Crit Care Med 1991;19:8–11.
104. **Brezis M, Rosen S, Epstein FH:** Acute renal failure due to ischemia (Acute tubular necrosis). In: Lazarus JM, Brenner BM, Eds. Acute Renal Failure. 3rd Ed. New York: Churchill Livingstone; 1993:207–229.
105. **Kjellstrand CM, Ebben J, Davin T:** Time of death, recovery of renal function, development of chronic renal failure and need for chronic hemodialysis in patients with acute tubular necrosis. Trans Am Soc Artif Int Organs 1981;27:45–50.
106. **Schiffl H, Lang SM, Konig A, Strasser T, Haider MC, Held E:** Biocompatible membranes in acute renal failure: prospective case-control study. Lancet 1994;2: 570–572.
107. **Cooper K, Bennet WM:** Nephrotoxicity of common drugs used in clinical practice. Arch Intern Med 1987; 147:1213–1218.
108. **Ouslander JG:** Drug therapy in the elderly. Ann Intern Med 1981;95:711–722.
109. **Williamson J, Chopin JM:** Adverse reactions to prescribed drugs in the elderly: A multicentre investigation. Age Aging 1980;9:73–80.
110. **Rasmussen HH, Ibels LS:** Acute renal failure. Multivariate analysis of causes and risk factors. Arch Intern Med 1982;73:211–218.
111. **Moore RD, Smith CR, Lipsky JJ, *et al.*:** Risk factors for nephrotoxicity in patients treated with aminoglycosides. Ann Intern Med 1984;100:352–357.
112. **Bennet WM, Elzinga LW, Porter GA:** Tubulointerstitial disease and toxic nephropathies. In: Brenner BM, Rector FC, Eds. The Kidney. 4th Ed. Philadelphia: WB Saunders; 1991:1430–1496.
113. **Eisenberg RL, Bank WO, Hedgock MW:** Renal failure after major angiography. Am J Med 1980;68:43–46.
114. **Cochran ST, Wong WS, Roe DJ:** Predicting angiography-induced acute renal function impairment: Clinical risk model. AJR Am J Roentgenol 1983;14:1027–1033.
115. **Martin-Paredero V, Dixon SM, Baker JD, *et al.*:** Risk of renal failure after major angiography. Arch Surg 1983; 118:1417–1420.
116. **Van Zee BE, Hoy WE, Talley TE, Jaenike JR:** Renal

injury associated with intravenous pyelography in nondiabetic and diabetic patients. Ann Intern Med 1978; 89:51–54.
117. **Alexander RD, Berkes SL, Abuelo G:** Contrast media-induced oliguric renal failure. Arch Intern Med 1978; 138:381–384.
118. **Swartz RD, Rubin JE, Leeming BW, Silva P:** Renal failure following major angiography. Am J Med 1978; 65:31–37.
119. **Krumlovsky FA, Simon N, Santhanam S, *et al.*:** Acute renal failure associated with administration of radiographic contrast material. JAMA 1978;239:125–127.
120. **Fang LS, Sirota RA, Ebert TH, Lichtenstein NS:** Low-fractional excretion of sodium with contrast media-induced acute renal failure. Arch Intern Med 1980;140: 531–533.
121. **Rich MW, Crecelius CA:** Incidence, risk factors and clinical course of acute renal insufficiency after cardiac catheterization in patients 70 years of age or older. A prospective study. Arch Intern Med 1990;150:1237–1242.
122. **Anto HR, Chou SY, Porush JG, Shapiro WB:** Infusion intravenous pyelography and renal function effects of hypertonic mannitol in patients with chronic renal insufficiency. Arch Intern Med 1981;141:1652–1656.
123. **Old CW, Lehrner LM:** Prevention of radiocontrast induced acute renal failure with mannitol. Lancet 1980; 1:885.
124. **Levitz CS, Friedman EA:** Failure of protective measures to prevent contrast media-induced renal failure. Arch Intern Med 1982;142:642–643.
125. **Dorman HR, Sondheimer JH, Cadnapahornchai P:** Mannitol-induced acute renal failure. Medicine (Balt) 1990;69:153–159.
126. **Hans B, Hans SS, Mittal UK, *et al.*:** Renal functional response to dopamine during and after arteriography in patients with chronic renal failure. Radiology 1990; 176:651–654.
127. **Lindner A, Cutler RE, Goodman WG:** Synergism of dopamine plus furosemide in preventing acute renal failure in the dog. Kidney Int 1979;16:158–166.
128. **Aron NB, Feinfeld DA, Peters AT, Lynn RI:** Acute renal failure associated with ioxaglate, a low-osmolality radiocontrast agent. Am J Kidney Dis 1989;13:189–193.
129. **Teruel JL, Marcén R, Herrero JA, Felipe C, Ortuño J:** An easy and effective procedure to prevent radiocontrast agent nephrotoxicity in high risk patients. Nephron 1989;51:282.
130. **Cameron JS:** Immunologically mediated interstitial nephritis: primary and secondary. Adv Nephrol 1989;18: 207–248.
131. **Kleinknecht D, Droz D:** Acute renal failure from interstitial disease. In: Cameron JS, Davison AM, Grunfeld JP, Kerr D, Ritz E, Eds. Oxford Textbook of Clinical Nephrology. Oxford: Oxford University Press; 1992: 1084–1098.
132. **Arieff AI, Anderson RJ, Massry SG:** Acute glomerulonephritis in the elderly. Geriatrics 1971;26:74–84.
133. **Samiy AH, Field RA, Merrill JP:** Acute glomerulonephritis in elderly patients: report of seven cases over sixty years of age. Ann Intern Med 1961;54:603–609.
134. **Potvliege PR, De Roy G, Dupuis F:** Necropsy study on glomerulonephritis in the elderly. J Clin Pathol 1975; 28:891–898.
135. **Abrass CK:** Glomerulonephritis in the elderly. Am J Nephrol 1985;5:409–418.
136. **Montoliu J, Darnell A, Torras A, Revert L:** Acute and rapidly progressive forms of glomerulonephritis in the elderly. J Am Geriatr Soc 1981;29:108–116.
137. **Galpin JE, Shinaberger JH, Stanley TM, *et al.*:** Acute interstitial nephritis due to methicillin. Am J Med 1978;65:756–765.
138. **Rowe JW, Resnick NW:** Disorders of the kidney and urinary tract. In: Andres R, Bierman EL, Hazzard WR, Eds. Principles of Geriatric Medicine. New York: McGraw Hill; 1985:614–628.
139. **Baird RJ, Yendt ER, Firor WB:** Anuria due to acute occlusion of the artery to a solitary kidney. N Engl J Med 1965;272:1012–1014.
140. **Duncan DA, Dexter RN:** Anuria secondary to bilateral renal artery occlusion. N Engl J Med 1961;266:971–973.
141. **Lessman RK, Johnson SF, Coburn JW, Kaufman JJ:** Renal artery embolism. Clinical features and long-term follow-up of 17 cases. Ann Intern Med 1978;89:477–482.
142. **Liaño F, Gámez C, Pascual J, *et al.*:** Use of urinary parameters in the diagnosis of acute renal artery occlusion. Nephron 1994;66:170–175.
143. **Pontremoli R, Rampoldi V, Morbidelli A, Fiorini F, Ranise A, Garibotto G:** Acute renal failure due to acute bilateral renal artery thrombosis: Successful surgical revascularization after prolonged anuria. Nephron 1990;56:322–324.
144. **Kassirer JP:** Atheroembolic renal disease. N Engl J Med 1969;280:812–818.
145. **Varanasi UR, Moorthy AV, Beirne GJ:** "Spontaneous" atheroembolic disease as a cause of renal failure in the elderly. J Am Geriatr Soc 1979;27:407–409.
146. **Smith MC, Ghose MK, Henry AR:** The clinical spectrum of renal cholesterol embolization. Am J Med 1981;71: 174–180.
147. **Fine MJ, Kapoor W, Falenga V:** Cholesterol crystal embolization: a review of 221 cases in the English literature. Angiology 1987;38:769–784.
148. **Dahlberg PJ, Frecentese DF, Gogbill TH:** Cholesterol embolism: experience with 22 histologically proven cases. Surgery 1989;105:737–746.
149. **Wilkowski MJ, Velosa LA, Holley KE, *et al.*:** Risk factors in idiopathic renal vasculitis and glomerulonephritis. Kidney Int 1989;36:1133–1141.
150. **Marcus EL, Rudensky B, Sonnenblick M:** Occult elevation of CK as a manifestation of rhabdomyolysis in the elderly. J Am Geriatr Soc 1992;40:454–456.
151. **Orofino L, García Martín F, Quereda C, *et al.*:** Incidencia, evolución y pronóstico del fracaso renal agudo de etiologia obstructiva. Actas Urol Esp 1987;11:379–383.
152. **Wilson DR, Klahr S:** Urinary tract obstruction. In: Schrier RW, Gottschalk CW, Eds. Diseases of the Kidney. 5th Ed. Boston: Little Brown, 1993:657–688.
153. **Walsh PC:** Benign prostatic hyperplasia. In: Walsh PC, Gittes RF, Perlmutter AD, Stamey TA, Eds. Campbell's Urology. 5th Ed. Philadelphia: WB Saunders; 1986: 1248–1265.
154. **Sarmina I, Resnick MI:** Obstructive uropathy in patients with benign prostatic hyperplasia. J Urol 1989; 141:866–869.
155. **Hoffman LM, Suki WN:** Obstructive uropathy mimicking volume depletion. JAMA 1976;236:2096–2097.
156. **Gillenwater JY, Westervelt FB, Vaughan E, Howards SS:** Renal function after release of chronic ureteral hydronephrosis in man. Kidney Int 1975;7:179–186.
157. **Ellenbogen PH, Scheible FW, Talner LB, Leopold GR:** Sensitivity of gray scale ultrasound in detecting urinary tract obstruction. AJR Am J Roentgenol 1978;130:731–733.
158. **Malave SR, Neiman HL, Spies SM, *et al.*:** Diagnosis of hydronephrosis. Comparison of radionuclide scanning and sonography. AJR Am J Roentgenol 1980;135: 1179–1185.
159. **Talner LB, Scheible FW, Ellenbogen PH, *et al.*:** How accurate is ultrasonography in detecting hydronephrosis in azotemic patients? Urol Radiol 1981;3:1–6.
160. **Arafa NM, Fathi MM, Safwat M, *et al.*:** Accuracy of ultrasound in the diagnosis of nonfunctioning kidneys. J Urol 1982;128:1165–1169.
161. **Webb JAW, Reznek RH, White SE, *et al.*:** Can ultrasound and computed tomography replace high-dose urography in patients with impaired renal function? Q J Med 1984;53:411–425.
162. **Denton T, Evans C, Cochlin DL:** Ultrasound in previously undiagnosed renal failure. Br J Radiol 1984;57: 673–675.
163. **Maillet PJ, Pelle-Francoz D, Laville M, Gay F, Pinet A:** Nondilated obstructive acute renal failure. Diagnostic procedures and therapeutic management. Radiology

1986;160:659–662.
164. **Naidich JB, Rackson ME, Massey RT, Stein HL:** Nondilated obstructive uropathy: Percutaneous nephrostomy performed to reverse renal failure. Radiology 1986;160:653–657.
165. **Roscoff JH, Golden RA, Spinowitz BS, Charyton C:** Nondilated obstructive uropathy. Arch Intern Med 1983;143:696–698.
166. **Lyons K, Matthews P, Evans C:** Obstructive uropathy without dilatation: A potential diagnostic pitfall. BMJ 1988;296:1517–1518.
167. **Gornish M, Lurie Y, Wysenbeek AJ:** Nondilated obstructive uropathy causing acute renal failure. Case report and review of the literature. Isr J Med Sci 1990; 26:50–52.
168. **Megibow AJ, Mitnick JS, Bosniak MA:** The contribution of computed tomography to the evaluation of the obstructed ureter. Urol Radiol 1982;4:95–104.
169. **Platt JF, Rubin JM, Ellis JH, Di Pietro M:** Duplex Doppler US of the kidney: Differentiation of obstructive from nonobstructive dilatation. Radiology 1989;171: 515–517.
170. **Fischer RP, Griffen WP, Reiser M, Clark DS:** Early dialysis in the treatment of acute renal failure. Surg Gynecol Obstet 1966;123:1019–1023.
171. **Baek SM, Makabali GG, Shoemaker WC:** Clinical determinants of survival from postoperative renal failure. Surg Gynecol Obstet 1975;140:685–689.
172. **Mallinson WJ, Fleming SJ, Shaw JEH, *et al.*:** Survival in elderly patients presenting with uremia. Q J Med 1984;53:301–310.
173. **Làmeire N, Mattys E, Vanholder R, *et al.*:** Causes and prognosis of acute renal failure in elderly patients. Nephrol Dial Transplant 1987;2:316–322.
174. **Pedersen RS:** A retrospective investigation of acute tubulointerstitial nephropathy treated with hemodialysis. Scand J Urol Nephrol 1981;57[Suppl]:59–64.
175. **Routh GS, Briggs JD, Mone JG, Ledingham IMA:** Survival from acute renal failure with and without multiple organ dysfunction. Postgrad Med J 1980;56:244–247.
176. **Druml W, Lax F, Grimm G, Schneeweiss B, Lenz K, Laggner AN:** Acute renal failure in the elderly 1975–1990. Clin Nephrol 1994;41:342–349.
177. **Sonnenblick M, Slotki IN, Friedlander Y, Kramer MR:** Acute renal failure in the elderly treated by one-time peritoneal dialysis. J Am Geriatr Soc 1988;36:1039–1044.
178. **Rasmussen HR, Pitt EA, Ibels LS, McNeil DR:** Prediction of outcome in acute renal failure by discriminant analysis of clinical variables. Arch Intern Med 1985; 145:2015–2018.
179. **Gentric A, Ciedes J:** Immediate and long-term prognosis in acute renal failure in the elderly. Nephrol Dial Transplant 1991;6:86–90.
180. **Kaufman J, Dhakai M, Patel B, Hamburger R:** Community-acquired acute renal failure. Am J Kidney Dis 1991;17:191–198.
181. **Schaefer JM, Jochimsen F, Keller F, Wegscheider K, Distler A:** Outcome prediction of acute renal failure in medical intensive care. Intensive Care Med 1991;17: 19–24.
182. **Rodgers H, Staniland JR, Lipkin GW, Turney JH:** Acute renal failure: a study in elderly patients. Age Aging 1990;19:36–42.
183. **Arora P, Kher V, Kohli HS, Sharma RK, Gupta A, Jha R:** Acute renal failure in the elderly: Experience in a single centre in India. Nephrol Dial Transplant 1993;8: 827–830.
184. **Baldyga AP, Paganini EP, Chaff C, Higgins TL:** Acute dialytic support of the octogenarian: Is it worth it?. ASAIO J 1993;39:M805–M808.
185. **McMurray SD, Luft FC, Maxwell DR, *et al.*:** Prevailing patterns and prediction variables in patients with acute tubular necrosis. Arch Intern Med 1978;139:950–955.
186. **Oliveira DBG, Winearls CG:** Acute renal failure in the elderly can have a good prognosis. Age Aging 1984;13: 304–308.
187. **Cioffi HG, Ashikaga T, Gamelli RL:** Probability of surviving postoperative acute renal failure. Development of a prognostic index. Ann Surg 1984;200:205–211.
188. **Maher ER, Robinson KN, Scoble JE, *et al.*:** Prognosis of critically-ill patients with acute renal failure: APACHE II Score and other predictive factors. Q J Med 1989;72: 857–866.
189. **Wheeler RG, Faragher EB:** Acute renal failure in an intensive care unit: incidence, prediction and outcome. Anaesthesia 1983;8:628–634.
190. **Lien J, Chan V:** Risk factors influencing survival in acute renal failure treated by hemodialysis. Arch Intern Med 1985;145:2067–2069.
191. **Montoliu J, Campistol JM, Cases A, Lens XM, Revert L:** Mortalidad y factores pronosticos de supervivencia en la insuficiencia renal aguda grave que requiere diàlisis. Nefrologia 1989;9:152–158.
192. **Tran DD, Groeneveld ABJ, Van der Meulen J, Nauta JJP, Van Schijndel RJMS, Thijs LG:** Age, chronic disease, sepsis, organ system failure, and mortality in a medical intensive care unit. Crit Care Med 1990;18: 474–479.
193. **Guly UM, Turney JH:** Post-traumatic acute renal failure 1956–1988. Clin Nephrol 1990;34:79–83.
194. **Turney JH, Marshall DH, Brownjohn AM, Ellis CM, Parsons FM:** The evolution of acute renal failure 1956–1988. Q J Med 1990;73:83–104.
195. **Liaño F, Gallego A, Pascual J, *et al.*:** Prognosis of acute tubular necrosis: an extended prospectively contrasted study. Nephron 1993;63:21–31.
196. **Liaño F:** Severity of acute renal failure: The need of measurement. Nephrol Dial Transplant 1994;9[Suppl 4]:229–238.
197. **Biesenbach G, Zazgornik J, Kaiser W, Grafinger P, Stuby U, Necek S:** Improvement in prognosis of patients with acute renal failure over a period of 15 years: An analysis of 710 cases in a dialysis center. Am J Nephrol 1992;12:319–325.