



Management of Acute Renal Failure

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ABSTRACT

Management of ARF entails close monitoring of the patient, supportive therapy, nutritional management, dialysis, and target treatment of specific complications that may arise) The signs and symptoms of ARF reflect loss of the regulatory, excretory, and endocrine functions of the kidney. The loss of excretory ability of the kidney is reflected in a rise in the plasma concentration of specific substances, which are normally excreted by the kidney. The ARF management includes correction of fluid and electrolyte levels: avoidance of nephrotoxins, and a kidney replacement therapy.

Keywords: Dietary management, Dopamine, Mannitol, Loop diuretics, Dialysis

INTRODUCTION

Acute renal failure is an acute loss of kidney function that occurs over days to weeks and results in an inability to appropriately excrete nitrogenous wastes and creatinine. Electrolyte disturbances and loss of fluid homeostasis may occur. In spite of this rapid decline in kidney function, patients with acute renal failure often have few symptoms.

The cause of acute renal failure usually can be identified through an appropriate history, a physical examination, and selected laboratory tests. The initial laboratory evaluation should include urinalysis, a determination of the fractional excretion of sodium, a blood urea nitrogen to creatinine ratio, and a basic metabolic panel. Management includes correction of fluid and electrolyte levels; avoidance of nephrotoxins; and kidney replacement therapy, when appropriate.

Pathophysiology

Creatinine is a metabolic waste product excreted by the kidneys. When the Glomerular Filtration Rate (GFR) is normal, creatinine is filtered through the glomerulus into the tubules and then excreted. Creatinine also is secreted by tubular cells. Medications such as trimethoprim (Proloprim; with sulfamethoxazole [Bactrim, Septra]) and cimetidine (Tagamet) can inhibit tubular secretion and falsely elevate the serum creatinine level. Formulas to estimate the GFR in patients with acute renal

failure should not be used to adjust medication dosages because the serum creatinine level is not in a steady state and continues to fluctuate.

Management

Acute renal failure often is reversible. Risk factors for this condition include diabetes mellitus, chronic renal insufficiency, heart failure, and advanced age.

Many medications can injure the kidneys. Dosing schedules can help prevent acute renal failure. For example, acute renal failure is less likely to develop with a once-daily dose of an aminoglycoside than with multiple daily doses. When acute renal failure is diagnosed, the cause(s) must be identified and treated. Critical measures include maintaining adequate intravascular volume and mean arterial pressure, discontinuing all nephrotoxic drugs, and eliminating exposure to any other nephrotoxins. Electrolyte abnormalities must be corrected, and urine output should be monitored closely. Pigment or uric acid exposure can be treated with alkaline diuresis. Ethylene glycol or methanol poisoning should be treated with an alcohol drip or with fomepizole (Antizol).

ARF is often preventable once it is diagnosed, the causes must be identified and treated. Critical measures include maintaining adequate intravascular volume mean arterial pressure, discontinuing all nephrotoxic drugs, and eliminating exposure to any Electrolyte other nephrotoxins. (Agrawal and Swartz, 2001). abnormalities must be corrected, and urine output should be monitored closely, Pigment or uric acid exposure can be treated with alkaline diuresis. Ethylene glycol or methanol poisoning should be treated with an alcohol drip or with fomepizole (Antizol) (Brent *et al.*, 200 Fluid management is based on careful physical examination and invasive monitoring if appropriate. The decision to administer or remove fluids, however, is often difficult for the physician. Since both strategies have detrimental consequences if perused inappropriately. Although resuscitation is ineffective in restoring renal function once ATN is established. Volume replacement remains our most effective prophylactic strategy. (Conger, 1995).

Nutritional Management of ARF

Nutritional management requires collaboration patient, physicians, nurses and dieticians. The main

objective of nutritional therapy during the maintenance phase of ARF is to provide sufficient calories to avoid catabolism and starvation ketoacidosis while minimizing production of nitrogenous waste.) This can be best achieved by restricting dietary protein intake to approximately 0.5g/kg of body weight per day of protein of high biologic value (i.e, rich in essential amino acids) and to provide most calories in the form of carbohydrate (approximately 100g/day). Management of nutrition is easier in non-oliguric patients and after institution of dialysis.

Dietary management

Dietary treatment is directed toward correction of fluid and electrolyte imbalance and maintenance of adequate nutritional status in order to minimize endogenous protein catabolism and subsequent uremia.

Energy required-

A minimum of 600-1000 k cal is necessary. Thus, a higher calorie intake of carbohydrates and fats is desirable.

Proteins required-

The protein containing foods are not to be administered if the patient is under conservative treatment and if the level of BUN is rising. The diet can be raised to 2000-3000k cal and 40gm of protein. However, 40gm protein is allowed if the patient is on peritoneal dialysis or hemodialysis as it will reduce the endogenous protein breakdown and will maintain the health of the patient. During dialysis if the patient cannot take oral feeds, intravenous fat and amino acid infusion can be given. High protein food such as pulses, mango, tea, fish, milk and coca should be avoided.

Carbohydrates-

A minimum of 100gm/day is essential to minimize tissue protein breakdown 2 liters of 5% glucose meets this need.) If the patient is not fed by mouth a nasogastric tube feeding of 700ml of 15% glucose is administered.

Fluids-

The fluid is regulated in relation with the urinary output, any additional losses from vomiting or diarrhea and an allowance for insensible water losses. The total fluid permitted is 500ml + previous days urine output + total loss from the above causes.

Sodium-

The dietary sodium allowance is based on frequent measurement of the sodium ion in serum and urine. For the non dialysed patient restriction of sodium is necessary whereas on dialysis patients sodium intake is allowed. High sodium foods such as Chinese, tin food, pickles and papads should be avoided.

Potassium-

Potassium intoxication (hyperkalemis) occurs in ARF. It has deleterious effects on heart. A bowel wash may remove 1000m Eq of potassium. Potassium rich sources such as tomato juice, coffee, tea, coca, should be avoided.

Dopamine therapy

Low dose dopamine (1-3 ug/kg/min) is prescribed world-wide for the prevention and treatment of ARF, to correct oliguria, and to preserve renal perfusion in patients receiving systemic vasopressors, studies in

the early 1970s showed that low-dose dopamine infusion caused selective renal vasodilation, increased renal blood flow, and induced a natriuresis and diuresis in animals and healthy humans (Thompson, Cockrill, 1994) dopamine is synthesized by the kidney and is a critical regulator of sodium excretion. (Lee, 1993). (Aperia, 2000) Dopamine may improve the outcome of ATN by improving renal perfusion, inhibiting tubular transport processes and therefore improving the oxygen supply/demand relationship and flushing out renal tubules by inducing a diuresis.

Mannitol and treatment

Multiple pathophysiologic factors contribute to renal injury in ARF, including vasoconstriction, reduced glomerular capillary permeability, tubular obstruction by casts and swollen epithelial cells, and back leak of filtrate through an altered epithelium (Thadani *et al.*, 1996), (Kellum, 1998) Mannitol increases renal blood flow in both the renal cortex and medulla by reducing

Initial Evaluation of Acute Renal Failure

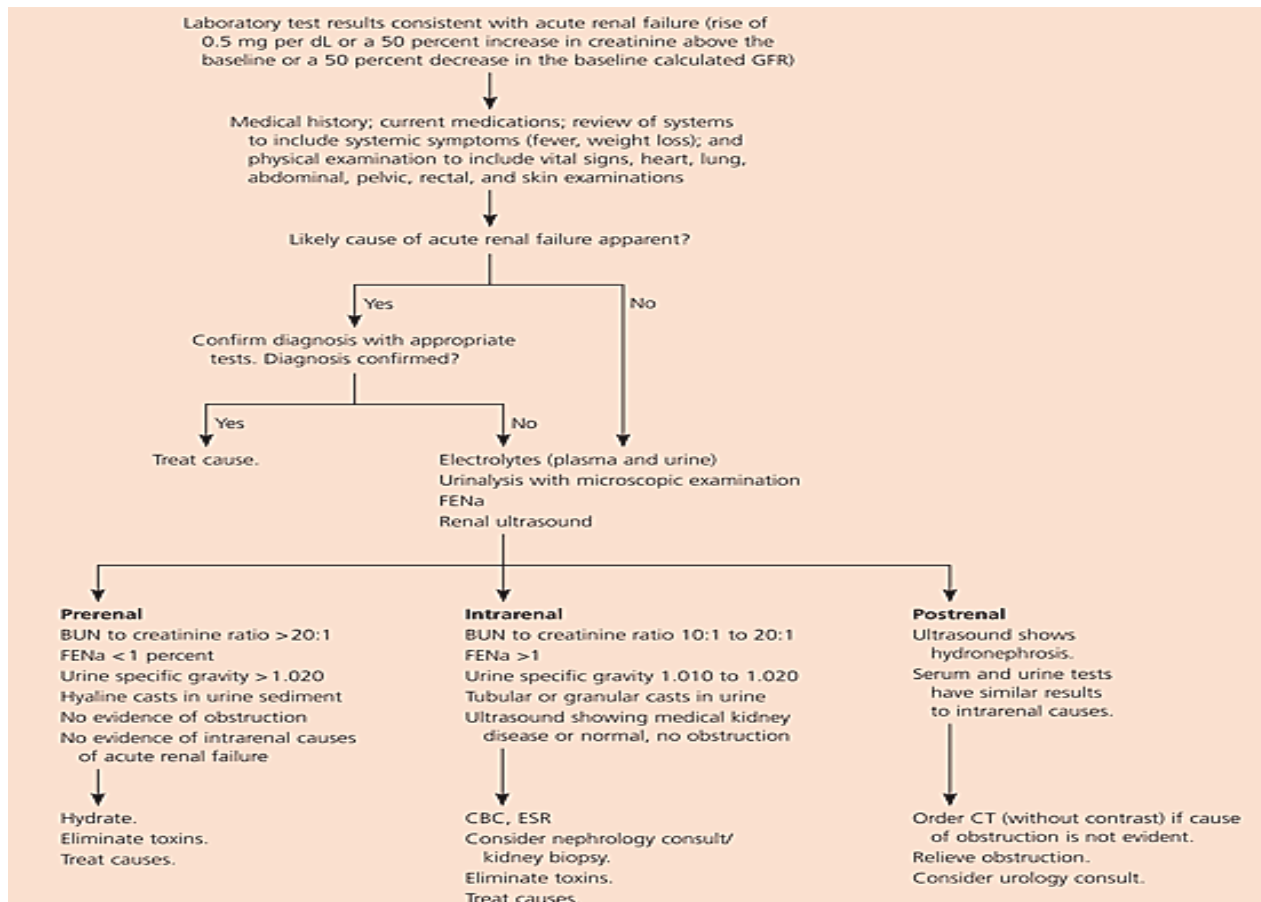


Figure 1.

Algorithm for the initial evaluation of acute renal failure. (GFR = glomerular filtration rate; FENa = fractional excretion of sodium; BUN = blood urea nitrogen; CBC = complete blood count; ESR = erythrocyte sedimentation rate; CT = computed tomography.)

renal vascular resistance, (Velasquez *et al.*, 1973) By increasing urine flow, mannitol could lead to relief of tubular obstruction by casts and cellular debris and to a reduction in the concentration of tubular toxins such as myoglobin or hemoglobin. (Star RA 1998) Finally, mannitol may reduce epithelial cell swelling as well as scavenge harmful free radicals, thereby ameliorating hypoxic reperfusion injury (Mason *et al.*, 1989), (Magovern *et al.*, 1984).

The prophylactic use of mannitol began in the 1960s when it was introduced for use in patients undergoing cardiovascular surgery to maintain intraoperative urine flow (Barry *et al.*, 1961) Since that time prophylactic use of mannitol has been recommended for patients at high risk of ARF in various clinical settings, including cardiac or vascular surgery, radiocontrast administration, obstruction jaundice, However severe rhabdomyolysis, and renal transplantation. prospective clinical trials examining the use of mannitol for prophylaxis failed to show improvement in renal function or mortality.

Loop diuretics

The loop diuretics also have vasodilatory properties and may act to increase renal blood flow. However, it has been postulated that the increased renal blood flow induced by loop diuretics may be maldistributed and potentially harmful (Solomon, 1994).

Dialysis

1. Hemodialysis

The initial care of the patients with ARF is focused on reversing the underlying cause and correcting fluid and electrolyte imbalances. Fluid management is based on careful physical examination and invasive monitoring if appropriate. The decision to administer or remove fluids, however, is often difficult for the clinician, since both strategies have detrimental consequences if pursued inappropriately. Continuous dialysis modalities are better tolerated in ICU patients, especially those with septic shock. The use of hemodialysis for the treatment of patients with ARF was introduced by Kolff in 1943 (Kolff WJ, Berk HT 1944). Hemodialysis should be initiated at the level of residual renal function above, which the major symptoms of uremia usually supervene. Among the criteria for initiating dialysis recognized by the funding entity for dialysis in the United States are residual creatinine clearances of 15mL/minute and

10mL/minute for diabetic and non-diabetics, respectively. K/DOQI guidelines suggest that dialysis should be initiated at a creatinine clearance between 9 and 14mL/minute. Heparin is administered as an anti-coagulant during hemodialysis. The process takes 3-4 hours for a good creatinine clearance. Ultrafiltration can be controlled in hemodialysis and it can be repeated number of times The dialysis machine is shown in Fig.no.4.

2. Peritoneal dialysis

Ganter (Ganter G, 1923) described the first clinical use of peritoneal dialysis in 1923. Peritoneum acts as filtering unit (kidney). It is possible even when the patient has low BP. Anticoagulants are not required, the process continues for days altogether.

Hyperkalemia is a common complication of acute renal failure. Potassium levels below 6 mEq per L (6 mmol per L) usually can be managed with dietary restriction and resin binders. Caloric intake should come primarily from carbohydrates. Protein intake should be balanced to minimize nitrogenous waste production while limiting starvation ketosis and subsequent production of ketoacids. This balance is achieved best with a protein intake of 0.6 g per kg per day.

Sodium bicarbonate therapy should be reserved for the treatment of severe metabolic acidosis (i.e., pH below 7.2 or a bicarbonate level below 10 to 15 mEq per dL [10 to 15 mmol per L]) with or without associated hyperkalemia. It is important to note that sodium bicarbonate and sodium polystyrene sulfonate have a large sodium load and may worsen fluid status in patients with acute renal failure.

When hyperkalemia is severe and unresponsive to treatment, kidney replacement therapy may be indicated. The use of intermittent or continuous hemodialysis (multiple techniques) continues to be debated. Both approaches are effective, and studies have not demonstrated either approach to be superior to the other. Intermittent hemodialysis requires less anticoagulation than does continuous hemodialysis; however continuous hemodialysis can be performed in patients with less hemodynamic stability.

Indications for Kidney Replacement Therapy

- Acidosis unresponsive to medical therapy
- Acute, severe, refractory electrolyte changes (e.g., hyperkalemia)
- Encephalopathy
- Significant azotemia (blood urea nitrogen level >100 mg per dL [36 mmol per L])
- Significant bleeding
- Uremic pericarditis
- Volume overload

Although renal biopsy rarely is performed, it may be indicated for patients with acute renal failure who do not respond to therapy or for assistance in the diagnosis of glomerulonephritis.

Acetylcysteine

Evidence exists that the prophylactic use of acetylcysteine (Mucomyst) before radiocontrast-media procedures decreases the incidence of acute renal failure.

In one randomized trial of 83 patients with chronic renal insufficiency, patients were assigned to receive 0.45 percent saline plus oral acetylcysteine (600 mg twice daily) or 0.45 percent saline alone before undergoing computed tomographic scanning. Within 48 hours after the imaging test, creatinine levels increased by 0.5 mg per dL or more in nine of the 42 patients in the saline-only group but increased in just one of the 41 patients in the acetylcysteine group ($P = .01$, relative risk = 0.11, absolute risk reduction = 19 %, number needed to treat = 5).

A second randomized controlled trial evaluated acetylcysteine pretreatment in patients scheduled to undergo coronary angiography and angioplasty. All patients had stable, moderate renal insufficiency and a GFR of less than 60 mL per minute. Patients randomly received acetylcysteine (600 mg twice daily) the day before the coronary procedure and the day of the procedure. All patients received an infusion of 0.9 percent normal saline. Within 48 hours of the procedure, serum creatinine levels increased by more than 25 percent in 12 of 98 patients in the saline-only group and in four of 102 patients in the acetylcysteine group ($P = 0.03$, relative risk = 0.33, absolute risk reduction = 8 %, number needed to treat = 12).

A third study showed that preprocedural acetylcysteine was neither helpful nor harmful.

Dopamine

Dopamine traditionally has been used to promote renal perfusion. However, systematic reviews of dopamine treatment in critically ill patients and in patients with sepsis do not support the use of dopamine to prevent renal insufficiency, morbidity, or mortality.

A multicenter, randomized, double-blind, placebo-controlled trial of low-dose dopamine therapy was conducted in patients with clinical evidence of early renal dysfunction who met two criteria for systemic inflammatory response syndrome (sepsis). In this study, 328 patients from 23 ICUs were assigned to receive dopamine (2 mcg per kg per minute) or placebo. The primary endpoint was elevation of the serum creatinine level during the infusion. No statistical differences were found between the two groups in elevation of creatinine levels, need for dialysis, duration of ICU stay, or length of hospital stay. There were 69 deaths in the dopamine group and 66 deaths in the placebo group. The study showed no benefit for dopamine.

A recent meta-analysis was conducted on the use of dopamine to reduce the incidence or severity of acute renal failure, the need for dialysis, or mortality in critically ill patients. Of the 58 studies that were identified, 17 were randomized clinical trials. Dopamine did not prevent mortality, onset of acute renal failure, or need for dialysis. A literature review reached a similar conclusion.

Oliguric vs. Nonoliguric Acute Renal Failure

Historically, nonoliguric renal failure has been assumed to have a better outcome than oliguric renal failure. As a result, diuretics commonly have been given in an attempt to convert the oliguric state to a nonoliguric state. However, diuretics have not been shown to be beneficial, and they may worsen outcomes.

An observational study of 552 patients with acute renal failure in four ICUs found that 326 of the patients were given diuretics at the time of nephrology consultation. The patients initially given diuretics were older; were more likely to have a lower serum blood urea nitrogen concentration; and were more

likely to have a history of heart failure, nephrotoxic renal failure, or acute respiratory failure. The main outcome measures were all-cause hospital mortality, nonrecovery of renal function, or both. Diuretic use in these higher risk patients was associated with a significant risk of death or nonrecovery of renal function (odds ratio [OR] = 1.77; 95 % confidence interval [CI] = 1.14 to 2.76). In the patients who survived one week past the initial nephrology consultation, the risk of death and nonrecovery of renal function was significantly increased (OR = 3.12; 95 % CI = 1.73 to 5.62).

Sodium Bicarbonate

A recent placebo-controlled trial involving 119 patients found an absolute risk reduction of 11.9 percent and a relative risk of 0.13 for elevated serum creatinine levels (from contrast-induced nephropathy) in patients who were given a sodium bicarbonate infusion before a radiocontrast-media procedure compared with those who were given only saline. This single-center study was stopped early because of the degree of benefit demonstrated for sodium bicarbonate infusion.

Conflict of interest

The author declares that there is no conflict of interest.

REFERENCES

Abuelo JG. Diagnosing vascular causes of renal failure [published correction appears in *Ann Intern Med* 1995;124(pt 1):78]. *Ann Intern Med*. 1995;123:601-14.

Agrawal M, Swartz R. Acute renal failure [published correction appears in *Am Fam Physician* 2001;63:445]. *Am Fam Physician*. 2000;61:2077-88.

Albright RC Jr. Acute renal failure: a practical update. *Mayo Clin Proc*. 2001;76:67-74.

Bellomo R, Chapman M, Finfer S, Hickling K, Myburgh J. Low-dose dopamine in patients with early renal dysfunction: a placebo-controlled randomised trial. Australian and New Zealand Intensive Care Society (ANZICS) Clinical Trials Group. *Lancet*. 2000;356:2139-43.

Brady H, Brenner B. Acute renal failure. In: Kasper DL, et al., eds. *Harrison's Principles of internal medicine*. 16th ed. New York: McGraw-Hill, 2001:1644-53.

Brent J, McMartin K, Phillips S, Aaron C, Kulig K. Methylpyrazole for Toxic Alcohols Study Group. Fomepizole for the treatment of methanol poisoning. *N Engl J Med*. 2001;344:424-9.

Briguori C, Manganelli F, Scarpato P, Elia PP, Golia B, Riviezzo G, et al. Acetylcysteine and contrast agent-

associated nephrotoxicity. *J Am Coll Cardiol*. 2002;40:298-303.

Cantarovich F, Rangoonwala B, Lorenz H, Verho M, Esnault VL. High-dose furosemide for established ARF: a prospective, randomized, double-blind, placebo-controlled, multicenter trial. *Am J Kidney Dis*. 2004;44:402-9.

Denton MD, Chertow GM, Brady HR. "Renal-dose" dopamine for the treatment of acute renal failure: scientific rationale, experimental studies and clinical trials. *Kidney Int*. 1996;50:4-14.

Fomepizole for the treatment of ethylene glycol poisoning. Methylpyrazole for Toxic Alcohols Study Group. *N Engl J Med*. 1999;340:832-8.

Galpin JE, Shinaberger JH, Stanley TM, Blumenkrantz MJ, Bayer AS, Friedman GS, et al. Acute interstitial nephritis due to methicillin. *Am J Med*. 1978;65:756-65.

Green GB, Coyne D. Renal disease. In: Green GB, Harris IS, Lin GA, Moylan KC, eds. *The Washington manual of medical therapeutics*. 31st ed. Philadelphia: Lippincott Williams & Wilkins, 2004:252-71.

Kay J, Chow WH, Chan TM, Lo SK, Kwok OH, Yip A, et al. Acetylcysteine for prevention of acute deterioration of renal function following elective coronary angiography and intervention: a randomized controlled trial. *JAMA*. 2003;289:553-8.

Kellum JA, Decker MJ. Use of dopamine in acute renal failure: a meta-analysis. *Crit Care Med*. 2001;29:1526-31.

Kodner CM, Kudrimoti A. Diagnosis and management of acute interstitial nephritis. *Am Fam Physician*. 2003;67:2527-34.

Martinez-Maldonado M, Kumjian DA. Acute renal failure due to urinary tract obstruction. *Med Clin North Am*. 1990;74:919-32.

Mehta RL, McDonald B, Gabbai FB, Pahl M, Pascual MT, Farkas A, et al. Collaborative Group for Treatment of ARF in the ICU. A randomized clinical trial of continuous versus intermittent dialysis for acute renal failure. *Kidney Int*. 2001;60:1154-63.

Mehta RL, Pascual MT, Soroko S, Chertow GM, PICARD Study Group. Diuretics, mortality, and nonrecovery of renal function in acute renal failure. *JAMA*. 2002;288:2547-53.

Merten GJ, Burgess WP, Gray LV, Holleman JH, Roush TS, Kowalchuk GJ, et al. Prevention of contrast-induced nephropathy with sodium bicarbonate: a randomized controlled trial. *JAMA*. 2004;291:2328-34.

Metnitz PG, Krenn CG, Steltzer H, Lang T, Ploder J, Lenz K, et al. Effect of acute renal failure requiring renal replacement therapy on outcome in critically ill patients. *Crit Care Med*. 2002;30:2051-8.

Prins JM, Buller HR, Kuijper EJ, Tange RA, Speelman P. Once versus thrice daily gentamicin in patients with serious infections. *Lancet*. 1993;341:335-9.

Pusey CD, Saltissi D, Bloodworth L, Rainford DJ, Christie JL. Drug associated acute interstitial nephritis: clinical and pathological features and the response to high dose steroid therapy. *Q J Med*. 1983;52:194-211.

- Schoolwerth AC, Sica DA, Ballermann BJ, Wilcox CS. Renal considerations in angiotensin converting enzyme inhibitor therapy: a statement for healthcare professionals from the Council on the Kidney in Cardiovascular Disease and the Council for High Blood Pressure Research of the American Heart Association. *Circulation*. 2001;104:1985-91.
- Singri N, Ahya SN, Levin ML. Acute renal failure. *JAMA*. 2003;289:747-51.
- Star RA. Treatment of acute renal failure. *Kidney Int*. 1998;54:1817-31.
- Tepel M, van der Giet M, Schwarzfeld C, Laufer U, Liermann D, Zidek W. Prevention of radiographic-contrast-agent-induced reductions in renal function by acetylcysteine. *N Engl J Med*. 2000;343:180-4.
- Thadhani R, Pascual M, Bonventre JV. Acute renal failure. *N Engl J Med*. 1996;334:1448-60.
- Venturini CM, Isakson P, Needleman P. Non-steroidal anti-inflammatory drug-induced renal failure: a brief review of the role of cyclo-oxygenase isoforms. *Curr Opin Nephrol Hypertens*. 1998;7:79-82.