Anesthesiology
51:218–221, 1979

# *Postoperative Renal Failure in Trauma Patients*

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**Since 1975 the authors had observed a sudden increase in the incidence of nonoliguric renal failure following anesthesia in trauma victims. In an attempt to find the possible causes they reviewed postoperative fluid management of 2,191 patients. During the period 1974–1975, fluid intake was increased and diuretics were administered in 960 postoperative trauma patients with oliguria (Group I). In those patients in Group I with respiratory insufficiency and oliguria, fluid therapy was restricted, and furosemide and albumin were administered. During the period of 1976–1977 postoperative creatinine and free-water clearance were monitored in 1,231 trauma victims (Group II). In patients with abnormal renal clearance values or oliguria, optimal cardiac output was maintained by maximizing preload. Diuretics were not administered unless circulatory overload was confirmed. There were 17 cases of acute renal failure in Group I and 18 in Group II. All of the 18 affected patients in Group II, but only three in Group I, were nonoliguric. Duration and severity of azotemia, complications, number of patients dialyzed, and mortality were significantly less ($P < 0.05$) in Group II than in Group I. It is concluded that when postoperative renal dysfunction is recognized early and therapy is directed toward attaining optimal blood volume, oliguria in acute renal failure may be avoided, thus minimizing morbidity and mortality. (Key words: Complications: trauma. Kidney: failure; function.)**

ACUTE NONOLIGURIC RENAL FAILURE (ANORF),[1–3] a benign variation of acute renal failure (ARF), is characterized by steady increases in blood urea nitrogen (BUN) and serum creatinine in the absence of oliguria. In contrast to prerenal azotemia, the urine–plasma creatinine ratio is less than 10 and the urine–plasma osmolality ratio less than 1.1. Morbidity, frequency of dialysis, and mortality are significantly less than in acute oliguric renal failure. In 1975 we began maximizing preload[4] in patients with oliguria using measurement of balloon-occluded pulmonary arterial pressure and cardiac output as a means of assessing adequacy of fluid replacement. Since then we had noticed a significant increase in the incidence of ANORF and a decrease in the number of patients experiencing oliguric renal failure. To determine whether this could be attributed to different fluid management or to other factors, a retrospective study was conducted.

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## Materials and Methods

From July 1973 to June 1977, 2,191 trauma patients received general anesthesia on the day of admission to the hospital. General anesthesia was administered by use of a nonrebreathing system with an Engström® ventilator. Following induction with thiopental and succinylcholine, anesthesia was maintained with nitrous oxide and oxygen supplemented by halothane or droperidol–fentanyl. *d*-Tubocurarine was employed for muscle relaxation when needed. The admission and anesthesia records were reviewed, together with postoperative laboratory data. The complete medical records of 35 patients in whom ARF developed were examined in detail. Criteria for the diagnosis of ARF were: BUN more than 40 mg/dl urine–plasma creatinine ratio less than 10, and urine–plasma osmolality ratio less than 1.1. ARF developing one week or more after admission was not considered to be related directly to trauma or anesthesia, and was not included.

Patients were divided into two groups. Group I included 960 patients who were admitted during the period July 1973 to June 1975. Following initial resuscitation and stabilization of vital signs, fluid intake was maintained at a rate of 50 ml/hour plus replacement of urinary output and other fluid loss during anesthesia and the postoperative period. Whenever a patient became oliguric (<20 ml/hour) and azotemic, fluid intake was increased until urinary output increased to 50 ml/hour or the central venous pressure (CVP) increased to 10 torr. In patients who had oliguria and increased CVP, a double-lumen Swan-Ganz catheter was inserted and fluid intake was increased until the pulmonary capillary wedge pressure (PCWP) increased to 18 torr. In oliguric patients with PCWPs higher than 18 torr with or without respiratory failure ($Pa_{O_2} < 60$ torr at $F_{I_{O_2}} > 0.35$), fluid was restricted and albumin, 25 per cent, and furosemide were administered. Whenever oliguria persisted despite high doses of furosemide (as much as 2 g/day), patients underwent dialysis.

Group II consisted to 1,231 patients admitted from July 1975 to June 1977. During this period, intraoperative and postoperative fluid management remained essentially the same as for Group I, but in addition, all patients were followed postoperatively by measurement of 24-hour creatinine and free-water clearance values. For patients with abnormal clearance

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values, fluid intake was increased to 100 ml/hour plus replacement of urinary output and other fluid losses, whether or not there was evidence of respiratory failure. In patients with oliguria and CVPs higher than 10 torr, a triple-lumen thermistor-tipped pulmonary-artery catheter was inserted. Fluids were then given to increase cardiac preload. Plasma protein fraction was infused at a rate of 50 ml/min for 5 min. The response of PCWP was observed. Provided PCWP remained the same or returned to within 2 torr of the control level within 15 min, the procedure was repeated. When PCWP remained 4 to 5 torr above the control, cardiac output was measured by a thermodilution technique. Whenever cardiac output increased then, further infusion of plasma protein fraction at the same rate was repeated in an attempt to maximize cardiac preload until cardiac output no longer increased or urinary flow rate increased to 50 ml/hour. This procedure was carried out despite the presence of respiratory failure or PCWP values greater than 18 torr; however, in the event total lung–thorax compliance (tidal volume/end-inspiratory airway pressure) decreased, suggesting pulmonary edema, this procedure was terminated. Whenever oliguria persisted, furosemide was administered. When cardiac output was deemed inadequate, as evidenced by increasing arteriovenous oxygen content difference, dopamine or sodium nitroprusside was administered, depending on the systemic vascular resistance.

A comparison was made of the patient populations, severities of injury, fluid volumes necessary for resuscitation and homeostasis during operation, and the incidences and clinical courses of ARF in the two groups.

## Results

There was no significant difference in ages or severities of injuries between the two groups (table 1). There were, however, significant differences in the fluid volumes necessary for resuscitation and in the durations of resuscitations. The durations of anesthesia and operation were longer and the fluid volumes infused during anesthesia were greater in Group II. The difference in the incidences of ARF between the two groups was not significant (table 2). The mean age of patients with ARF in Group II was significantly greater than that in Group I. With the exception of one patient in Group II, patients in both groups needed mechanical ventilation because of respiratory insufficiency. $Pa_{O_2}$ was not less than 60 torr for any patient with ARF following resuscitation. During this study period, there were four cases in Group I and five in Group II in which ARF developed a week or more after admission. These were excluded.

TABLE 1. Total Population, Description and Initial Therapy (Mean ± SE)

| | Group I (960 Patients) | Group II (1,231 Patients) |
|---|---|---|
| Age, years | 30 ± 0.5 | 31 ± 0.5 |
| Number of body sections injured* | 1.9 ± 0.03 | 1.9 ± 0.02 |
| Fluid volume for resuscitation, ml | 2087 ± 74 | 2333 ± 85† |
| Duration of resuscitation, hours | 2.2 ± 0.03 | 2.4 ± 0.03† |
| Duration of anesthesia, hours | 3.2 ± 0.03 | 4.1 ± 0.07† |
| Fluid volume during anesthesia, ml | 4105 ± 152 | 4760 ± 131† |

* Head, chest, abdomen, extremity, pelvis, and spine are each defined as one body section. One body section may include multiple organ injury (*i.e.*, heart and lung within chest section = 1).
† $P < 0.05$ compared with Group I.

All 18 ARF patients in Group II, but only three of 17 in Group I, were nonoliguric. In Group II, one patient received furosemide after volume loading, and another, sodium nitroprusside infusion because of a low cardiac output. One patient in Group II became oliguric following six days without oliguria. Of 17 ARF patients in Group I, 14 became oliguric despite high doses of furosemide and albumin. Daily fluid intake was significantly greater in Group II than in Group I (fig. 1). The severity and duration of azotemia (>40 mg/dl) were significantly less in Group II.

Fourteen of 17 ARF patients in Group I and five of 18 in Group II underwent dialysis. Complications in ARF were more frequent in Group I than in Group II (table 3). Hyperkalemia occurred in eight of 17 patients in Group I, but did not occur in Group II. Respiratory failure developing during ARF was also less frequent in Group II. All deaths in association with ARF in Group I occurred during ARF, while two

TABLE 2. Clinical Findings for Patients in Whom Acute Renal Failure Developed

| | Group I (960 Patients) | Group II (1,231 Patients) |
|---|---|---|
| Number of patients with ARF | 17 (1.8 per cent*) | 18 (1.5 per cent)* |
| Age (mean ± SE) | 30 ± 3 years | 41 ± 4 years† |
| Section injured (number of patients) | | |
| Head | 7 | 8 |
| Chest | 7 | 11 |
| Abdomen | 11 | 13 |
| Others | 15 | 12 |
| Fluid volume for resuscitation (mean ± SE) | 572 ± 48 ml/hour | 601 ± 65 ml/hour |
| Number of patients in shock | 9 | 9 |

* Incidence.
† $P < 0.05$ compared with Group I.

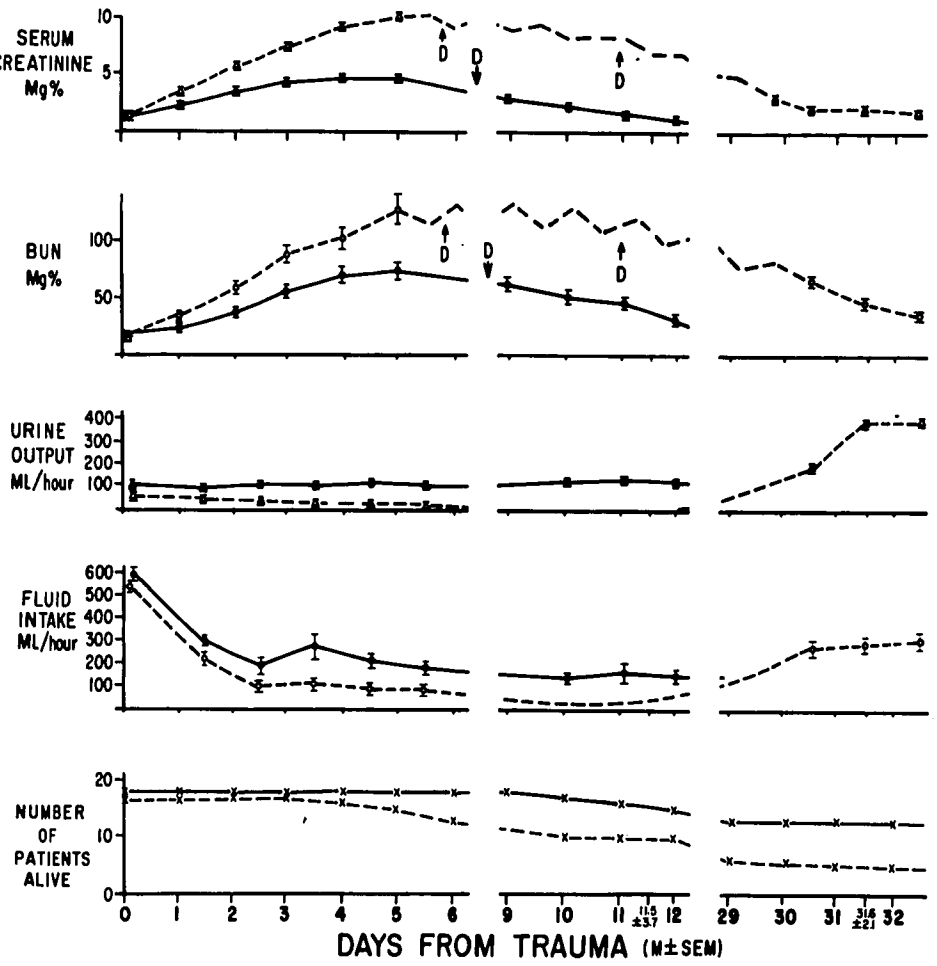


FIG. 1. Values (mean ± SEM) for renal function in patients with acute renal failure. ------ = Group I, —— = Group II. D = dialysis. Recovery of Group II patients occurred between 9 and 12 days and that of Group I patients, between 29 and 32 days.

of five deaths in Group II occurred after full recovery from ARF. None of the deaths in ARF patients in Group II was due to respiratory failure. Nine of 14 patients treated by dialysis in Group I, and three of five treated by dialysis in Group II, died.

TABLE 3. Complications and Deaths

| | Group I (960 Patients) | Group II (1,231 Patients) |
|---|---|---|
| Number of patients with ARF | 17 | 18 |
| Complications (number of patients) | | |
| Hyperkalemia (>7 mEq/l) | 8 | 0* |
| Pulmonary edema | 7 | 1* |
| Jaundice | 6 | 5 |
| Sepsis | 5 | 9 |
| Bleeding tendency | 3 | 1 |
| Pneumonia | 2 | 3 |
| Dehiscence | 3 | 0 |
| Pericardial effusion | 1 | 0 |
| Deaths | 12 | 5* |
| Cause of death | | |
| Sepsis | 3 | 2 |
| Heart failure | 1 | 3 |
| Cardiac arrest | 3 | 0 |
| Respiratory failure | 5 | 0* |

* $P < 0.05$ compared with Group I.

## Discussion

Oliguria has been commonly associated with ARF. Recently, there has been a growing number of reports suggesting that ANORF is not uncommon.[2,5] Lordon and Burton[5] reported a 43 per cent incidence of ANORF in combat casualties in the Vietnam War. Anderson and co-workers[2] reported 45 per cent of patients who had postoperative ARF had the nonoliguric type. However, it has not been clear in these reports whether ANORF may actually have been an early diuretic phase of oliguric renal failure.[6] Our study confirms that ANORF is not a rare, occasional variation, but rather a common form of ARF in trauma patients.

The etiology of ANORF is not known. Nephrotoxic drugs,[7,8] mild renal insults,[2] administration of diuretics,[9] and volume expansion with lactated Ringer's solution[3] have all been suggested as potential causes of the sustained urinary output in ARF. Our study suggests that oliguria in ARF in trauma patients can be avoided; that this can be done without administration of potent diuretics; and that aggressive fluid infusion in ARF is not necessarily associated with res-

piratory failure. It is not clear from our study whether lactated Ringer's solution played a role in ANORF. It has been our policy to use plasma protein fraction for volume replacement in patients with oliguria and to use crystalloid solution for hourly fluid maintenance. Since renal function in the patients with ANORF was significantly better than in those with oliguric renal failure, it appears that those in ANORF had suffered a lesser renal insult. However, the similarity of severities of injuries in patients in both groups in whom ARF developed suggests that extents of renal damage at the time of admission may have been the same.

If renal damage had been more severe in patients with oliguric renal failure, it probably should have occurred during resuscitation or anesthesia, or in the immediate postoperative period. The causes of greater renal damage may include hypoxemia,[10] nephrotoxic drugs, anesthesia,[11] and poor myocardial function following hemorrhagic shock. None of the patients who had ARF had hypoxemia ($Pa_{O_2} < 60$ torr) during anesthesia and operation or in the postoperative period, although they needed high inspired concentrations of oxygen and positive end-expiratory pressures. Nephrotoxic antibiotics were not given in sufficient doses and for sufficient periods to cause renal failure. Anesthesia has been known to depress renal function, particularly in hypovolemic patients.[11] Lordon and Burton[5] reported that patients with ANORF who underwent anesthesia experienced oliguria frequently. It is not clear, however, whether the longer resuscitation and the greater volume infused before anesthesia in Group II patients did, in fact, minimize renal failure.

A vasomotor renal insult from either heart failure or fluid restriction is another possible cause of oliguria in patients with ARF in Group I. Myocardial depression is not unusual in patients who recover from hemorrhagic shock.[12] Overzealous fluid infusion may overload such patients.[5] To determine whether the renal dysfunction was due to heart failure or hypovolemia, we monitored the responses of PCWP and cardiac output to rapid fluid infusion. Absolute values of PCWP[13] may be misleading in patients whose lungs are being ventilated with positive end-expiratory pressure and in patients with abnormal pulmonary function. Patients with depressed myocardial contractility may need an even higher than normal PCWP to maintain optimal cardiac output.[14] Whenever oliguria and renal dysfunction are recognized, we believe that cardiac output should be maximized prior to administration of diuretics even though the patients may have respiratory failure. Indiscrete use of diuretics before volume replacement has been effected may aggravate hypovolemia and lead to renal failure. We postulate that the high incidence of ANORF in Group II might have been due to early detection of the factors causing further renal damage and maintenance of optimal blood volume. This should be confirmed by further study.

Respiratory failure occurring with oliguric renal failure is common. Intensive monitoring of pulmonary function during fluid infusion avoids aggravation of respiratory failure while effectively minimizing the renal damage. When fluid restriction is necessary to prevent further deterioration of respiratory function, renal function should be monitored closely.[15]

The results of dialysis in posttraumatic and postoperative renal failure have been disappointing.[16,17] Maintaining urinary output in ARF appears to be extremely important for survival. This study strongly suggests that oliguria can be prevented in posttraumatic renal failure provided renal clearance values are monitored closely and optimal cardiac output is maintained by maximizing preload. A prospective study is in progress to confirm this.

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