Renal Decapsulation in the Prevention of Post-ischemic Oliguria

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The delayed onset of anuria/oliguria in acute tubular necrosis has been theorized to represent a complicating compartment syndrome, *i.e.*, parenchymal swelling within an unyielding capsule. To test this proposition, 12 monkeys had suprarenal aortic cross-clamping, followed by unilateral renal decapsulation to create an experimental as well as a control kidney unit in the same animal. Histologic examination uniformly confirmed tubular necrosis at death or sacrifice. Subsequent split renal function studies (creatinine, urea, and free water clearances) indicated significantly greater maintenance of renal function by the decapsulated kidney than by its paired control. Clinical evaluation in 21 hemorrhagic shock patients, with the capsule of one kidney stripped, revealed on follow-up that 15 developed a renal failure consistent with acute tubular necrosis. Although three patients with polyuric failure died before split studies could be run and two others have been too recent for computer analysis to have been completed, nine of the remaining ten had significantly greater renal plasma flows (194 versus 121 ml/min M^2, $p < .01$) and significantly greater urine flows (.99 versus .18 ml/min M^2, $p < .01$) on the decapsulated side than on the control, as determined by differential renal scans. No significant difference in these same lateralized renal functions was noted in the tenth patient with renal failure and in the six survivors without renal failure. Renal decapsulation as prophylaxis reduced the anticipated incidence of oliguria/anuria from an expected 75% to 7% ($p < .01$) in these 21 shock patients. Such data suggest that delayed renal ischemia, possibly based on a compartment syndrome, may be the cause for a progression of acute tubular necrosis from polyuria to oliguria and then to anuria.

POST-TRAUMATIC ACUTE TUBULAR necrosis can appropriately be described as a disease of numbers. Since the lesion on histologic examination appears to be either present or absent, with few valid criteria of gradations between, pathologists can seldom be relied upon to accurately quantitate the degenerative process. Instead, severity of damage must be based almost entirely on some battery of so-called renal function tests. These describe the degree of renal injury in terms of finite numbers *i.e.*, renal blood flow, glomerular filtration rates according to clearance of creatinine or some foreign compound, and measurements of tubular excretion, absorption, or transport.[4] The result of each determination is then duly recorded as a number.

Still, despite all of these detailed renal function studies and despite generously equipped and intelligently staffed kidney failure units, acute tubular necrosis remains one of the most common causes of death following hypovolemic shock.[28] To date, only efforts at limiting the duration and depth of precipitating shock have appeared to reduce to any significant degree the incidence of this dreaded complication. Such measures have primarily centered about more energetic fluid therapy and earlier operation to arrest the hemorrhage. Certain other forms of treatment, supposedly both specific and adjunctive, have also been tried, e.g., diuretics, steroids, etc; yet there is no general agreement as to how they work or even as to whether they are actually of any benefit.[3,8–10,14,17,28]

Decapsulation of the kidney for bilateral cortical necrosis has been used sporatically during the past century with somewhat indifferent results.[1,2,13,16,21,23,–24,29] Nevertheless, the all too frequent progression from polyuria on the first and second postoperative days, to oliguria, and then to anuria by the fourth to sixth day does indeed mimic the course of events noted in a typical compartment syndrome, *i.e.*, parenchymal swelling within a non-yielding envelope of bone and/or fascia.[7] Because of this continually recurring lethal complication and yet our admitted inability to reverse the trend toward deterioration in renal function, it was deemed worthwhile to evaluate once again the possibility that anuria due to acute tubular necrosis might be a late and even avoidable stage of an otherwise self-limited polyuric state.

Three phases of study were involved: 1) a retro-

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TABLE 1. *Mortality of Acute Renal Failure (Grady Memorial Hospital 1970–1974)*

	Number	Died	Mortality
Traumatic Shock	42	33	78.6%
General Surgery	76	19	25.0%
Medicine*	1010	249	24.7%
Nephrology ICU	111	34	30.6%
Other Services	316	51	16.1%
Total	1555	386	24.8%

* Numbers somewhat inflated by the introduction of the problem oriented record on the Medical Service in 1972.

spective and somewhat superficial review of cases of acute renal failure at Grady Memorial Hospital; 2) a series of controlled laboratory experiments using monkeys as the test subjects; and 3) a clinical application of the evolved principles, should the animal work be productive, in a set of appropriately selected patients.

Retrospective Case Review

Over the five year period from 1970 thru 1974, acute renal failure was listed on the patient code-out sheet 1555 times (Table 1). The 386 deaths in this group produced an overall mortality of 24.8%. However, those individuals whose renal failure developed as a consequence of trauma had a mortality rate of 78.6%, a figure more than three times that for any other general category.

More detailed analysis of those patients acquiring acute tubular necrosis on the surgical services, thereby including both the elective surgery wards as well as instances secondary to traumatic shock, demonstrated that anuria carried the highest mortality of all, *i.e.*, 95% (Table 2). Polyuria, on the other hand, was associated with only a 10% mortality rate.

Anuria occurred just once after the sixth postoperative or post-trauma day (Table 3). By contrast, polyuria was usually present from the first day of injury or the day of surgery; yet seldom was the diagnosis of renal failure made until four or five additional days had passed. Cases of oliguria seemed to fall into an intermediate area between the two extremes. The overall pattern, nevertheless, suggested that a progression of the same lesion might well account for the differences in severity and in date of onset.

TABLE 2. *Mortality of Acute Renal Failure (Surgical Service 1970–1974)*

Least 24-Hour Output	Number	Died	Mortality
Polyuria (>500 ml)	29	3	10%
Oliguria (100–500 ml)	68	29	43%
Anuria (<100 ml)	21	20	95%

TABLE 3. *Onset of Acute Renal Failure (Surgical Service 1970–1974)*

Least-24-Hour Output	Hospital Day Diagnosis Made		
	<4	4–6	>6
Anuria (<100 ml)	13	7	1
Oliguria (100–500 ml)	9	41	18
Polyuria (>500 ml)	—	12	17

Since the mortality of anuria was essentially ninefold greater than that for polyuric renal failure, and since worsening of kidney function appeared to take place with the same degree of regularity during the first week following injury, efforts directed at prevention of such a decline might well be rewarded if the responsible event were to be specifically identified and controlled. Accordingly, creation of similar conditions in a near-human experimental animal seemed to represent the next logical step.

Animal Studies

Under intravenous ketamine anesthesia and controlled ventilation, 12 adult rhesus monkeys were subjected to transabdominal cross-clamping of the suprarenal aorta (Fig. 1). Strict aseptic precautions were used throughout. Prior to clamping the aorta, both kidneys were exposed to assure gross bilateral normality; and then the respective renal arterial pedicles were isolated for blood flow determinations (square wave flow probes and flow meter). In addition, an intra-arterial cannula was inserted at the aortic bifurcation in order to allow direct recording of aortic pressures. Base line measurements were next taken as were venous blood samples for determinations of serum electrolytes (sodium, potassium, chloride, and carbon dioxide combining power), blood urea nitrogen and serum creatinine. The suprarenal aorta was then occluded for periods of five to 15 minutes. Repeat determinations of individual renal artery flows as well as infrarenal aortic pressures were made during cross-clamping and at one, five, and/or 15 minutes after release of the clamp. The capsule of one kidney, usually the left, was incised and stripped medially. Once again, renal blood flows and aortic pressures were measured. Upon completion of these readings, the midline abdominal incision was closed with a single suture of 2-0 prolene, as was the skin.

One animal served as a control; for, instead of undergoing cross-clamping of the suprarenal aorta, the clamp was placed just below the renal take-offs. Otherwise, all details in the experimental procedure were the same as with the original 12 monkeys.

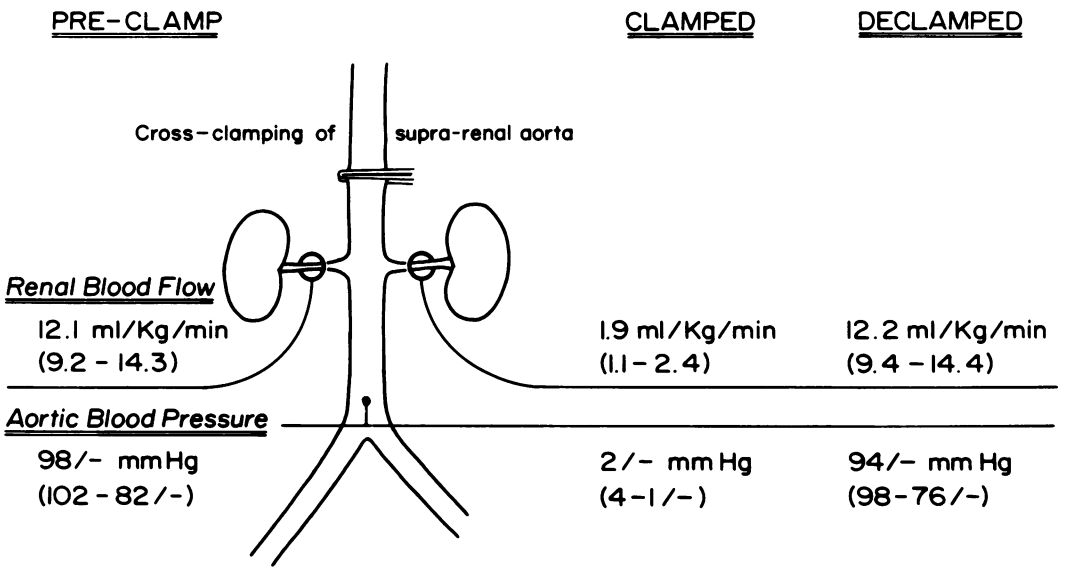

FIG. 1. Monkey model for study of renal decapsulation: Renal blood flows and aortic cross-clamping the suprarenal abdominal aorta.

Postoperatively, all animals were given water, standard monkey chow, and fresh fruit as desired. However, troublesome hypothermia demanded use of heat lamps in several of the animals whose procedures had run for more than 90 minutes.

The nine long term survivors among the experimentals and the single control animal were restudied at eight to 27 days later. Ketamine anesthesia, controlled ventilation, and sterile precautions were employed as before. Venous blood specimens were also taken for follow-up determinations of serum electrolytes, creatinine, and blood urea nitrogen. The previously used incision was reopened; both kidneys were inspected; and then each ureter was isolated, divided, and cannulated for controlled urine collection. Two of these ten monkeys, not included in subsequent function studies, had injury to a scarred kidney pedicle during vessel isolation for determination of renal blood flow and thus could no longer be considered to represent reliable preparations. It was for this same reason that follow-up renal blood flow determinations were not even attempted in the remaining eight animals.

Under the controlled conditions thus provided, a two to four hour collection of urine was taken for determination of creatinine, urea, and free water clearances on a split function basis. Measurement of clearances for sodium and potassium had also been planned, but the small volumes of urine produced by the kidney with an intact capsule prohibited such additional tests.

At completion of the urine collections, all animals were sacrificed by means of an intravenous bolus of concentrated potassium chloride. Gross and histologic examinations were then performed on each kidney, as well as on the kidneys of those monkeys dying prior to this second laparotomy.

Animal Study Results

On cross-clamping of the suprarenal abdominal aorta, average arterial blood pressures below that point fell from 98 mm Hg to 2 mm Hg systolic (Fig. 1). On declamping, pressures immediately increased to 80 mm Hg and eventually were maintained at 94 mm Hg. Renal blood flows paralleled these changes, with an average of 12.1 ml/kg/min before clamping, down to 1.9 ml/kg/min or 16% of the initial value during occlusion, and then back up to 12.1 ml/kg/min after release of the clamp. The one animal whose aorta had been cross-clamped below the renal arteries had similar blood pressure fluctuations, but no such alterations in renal blood flow.

Two monkeys with 55 minute occlusion times and one with a 45 minute clamping failed to survive the immediate post-operative phase. Hypothermia and unexplained recurrent shock appeared to be the causes for death. An autopsy on each revealed no other reason.

Eight of the nine long term survivors among the experimentals had no significant changes in serum electrolytes, serum creatinine, or blood urea nitrogen. The ninth, a monkey that had been subjected to 45 minutes of aortic occlusion, did have normal serum electrolytes, but definite elevations were noted in both serum creatinine and in the blood urea nitrogen.

Split ureteral function studies demonstrated significant differences ($P < .01$) for clearance of creatinine, urea, and free water (Fig. 2). The average value for each on the decapsulated side (2.42, 0.86, and 6.34 ml/kg/min, respectively) was more than triple that for the control kidney (0.57, 0.17, and 1.93 ml/kg/min, respectively).

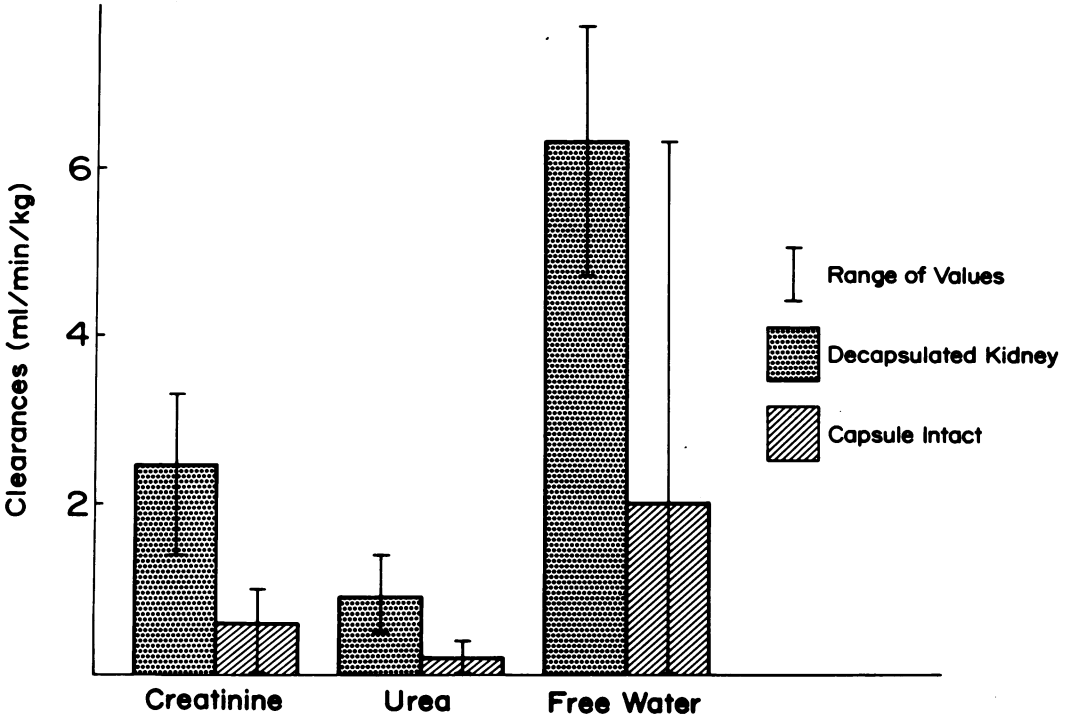

FIG. 2. Comparative renal clearances of creatinine, urea, and free water in the rhesus monkey following suprarenal aortic occlusion and unilateral kidney decapsulation.

Gross pathologic examination revealed that the decapsulated kidney appeared relatively normal if subjected to less than 45 minutes of reduced perfusion (Table 4). However, each of the kidneys with an intact capsule was either smaller than its contralateral partner or obviously atrophic. These gross changes correlated well with the results of a comparison of individual creatinine clearances to what had been published as the accepted norm.

Findings of acute tubular necrosis with varying degrees of glomerular degeneration were present on histologic study of the kidneys from each of the experimental animals. Among the several examiners, however, there was no absolute agreement as to whether the two sides had similar extents of involvement or whether the decapsulated kidney was decidedly less distorted.

TABLE 4. *Renal Decapsulation in Monkeys*

Animal (number)	Ischemia Time	Gross Pathology		Renal Function	
		Control	Experimental	Control	Experimental
1	15 min	atrophic	normal	0	normal
10	15 min	small	normal	30%*	normal
2	25 min	atrophic	normal	—	—
11	25 min	atrophic	normal	10%*	normal
12	25 min	small	normal	20%*	normal
3	35 min	small	normal	30%*	normal
7	35 min	small	normal	10%*	70%*
9	35 min	atrophic	normal	—	—
4	45 min	atrophic	small	10%*	30%*
6	45 min	Died acutely		—	—
5	55 min	Died acutely		—	—
8	55 min	Died acutely		—	—

* On comparison to normal values for creatinine clearance as cited by Pichering and Sussman.[25]

The single control animal that had aortic clamping below the renal arteries had no significant deviation from pre-study figures of the serum electrolytes, serum creatinine, blood urea nitrogen, and clearances of creatinine, urea, and free water. In addition, gross and microscopic examination of its kidneys revealed no overt abnormalities.

Clinical Study

Because of the documented benefits of renal decapsulation in the monkey experiencing an acute reduction in both renal perfusion as well as in renal perfusion pressure, a clinical trial was deemed to be appropriate. However, strict criteria were set in order that the variables might be kept to a minimum. In addition, clinical data needed to define the shock state itself had to be immediately at hand.

Criteria

Patients considered to represent potential candidates for the study were individuals who had been admitted in shock as emergencies to the Trauma Service of Grady Memorial Hospital. Seven specific criteria for patient inclusion had to have been satisfied before renal decapsulation could be considered. These were:

1) Shock unquestionably due to hemorrhagic hypovolemia.
2) Duration and/or depth of shock to be of sufficient magnitude to make post-ischemic acute tubular necrosis an almost certain complication, that is:
   a. profound shock (blood pressure 40/0) for ½ hour, or
   b. moderate shock (blood pressure 60/40) for one hour, or
   c. mild shock (blood pressure 80/60) for two hours.
3) A significantly massive pre-operative and intra-operative fluid replacement volume, that is:
   a. volume of blood equal to or greater than normal blood volume, or
   b. total fluid repletion volume of at least twice the normal blood volume.
4) Emergency abdominal exploration already required for treatment of shock.
5) Both kidneys present and grossly normal on inspection at the time of laparotomy.
6) Absence of any signs of established peritoneal sepsis, even though contamination by bowel contents has occurred.
7) No overt bleeding diathesis.

Once selected, the patient could receive no diuretic agents either at operation or during the immediate post-operative course. Otherwise, appropriate management for all injuries was rendered; and measures considered to be routine forms of post-operative support were given. As a general rule, all patients were observed in the surgical intensive care unit for at least five days following operation until it appeared certain that the major organ systems had stabilized.

Technique of Decapsulation

The kidney selected for decapsulation was usually the one that had been exposed during abdominal exploration or was the more easily reached through that particular surgical incision. Most commonly, this was on the right. After mobilization of the lateral border, a small nick was made in the capsule at its most lateral extreme, care being taken not to incise the cortex proper (Fig. 3A). Next, a mosquito forcep was slipped beneath the capsule so as to elevate it from the parenchyma below (Fig. 3B). Then the capsule was slit from pole to pole along its lateral convexity (Fig. 3C). Finally, the free edges of the capsule were grasped with thumb forceps and stripped medially, both front and back (Fig. 3D).

Bleeding from kidney substance was a problem only if the cortex had been incised or the capsule had not been separated from the cortex prior to extending the cut. Nevertheless, application of a dry sponge or a piece of hemostatic gauze for several minutes consistently stopped the ooze.

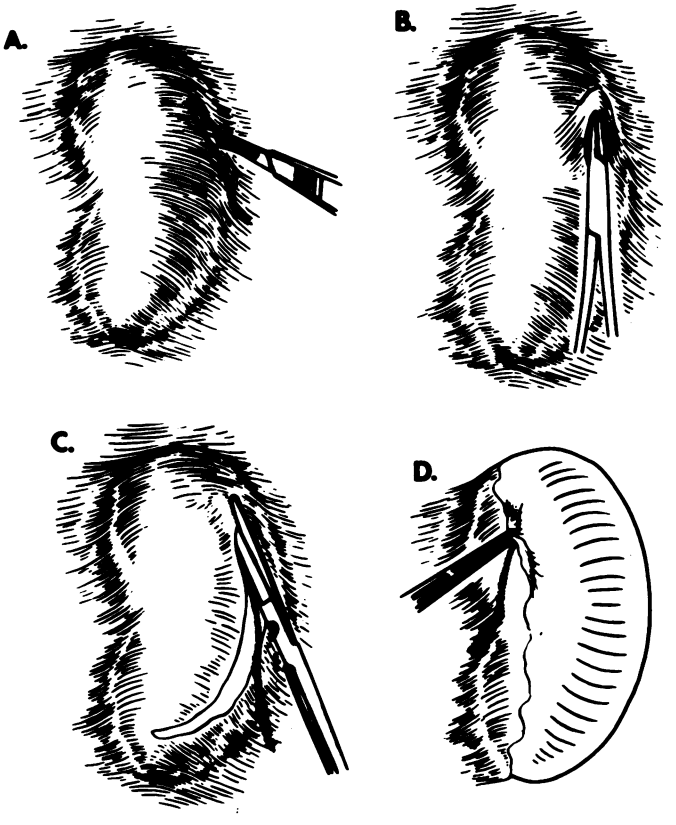

FIG. 3. Renal decapsulation: (A.) The capsule is incised at its lateral margin, care being taken not to cut the cortex below. (B.) A mosquito forcep is used to elevate the capsule. (C.) The capsule is cut from superior to inferior pole along its lateral extremity. (D.) The capsule is stripped medially, both in front and behind.

The kidney was next replaced into its rightful fossa, although frequently there would be a persistent tilt of the superior pole anteriorly. Gerota's fascia was never closed back over the kidney, but the reflected colon was routinely repositioned so as to seal off the retroperitoneal space. Drains were never used, at least not for the area of the decapsulated kidney.

Follow-up

Subsequent follow-up was appropriate to the injuries sustained and the individual operative procedures carried out. In addition, special attention was given to: hourly and then daily volumes of urine produced; urine and serum determinations of standard electrolytes (sodium, potassium, chloride, and carbon dioxide combining power), creatinine, and blood urea nitrogen; calculations of creatinine clearances; and concomitant comparisons of serum and urine osmolarities.

Split renal function studies were obtained at convenient intervals by intravenous pyelography in all patients, retrograde ureteral catheterization for lateral-

TABLE 5. *Renal Decapsulation (Shock)*

Fluid resuscitation	Blood	average	6.2 liters
		range	2.5–12.5
	Ringer's	average	4.6 liters
		range	2.5–11.0
Blood pressure	Less than 80/60	average	1.9 hours
	21 patients	range	0.5–4.0
	Less than 60/40	average	1.1 hours
	16 patients	range	0.5–2.0
	Less than 40/0	average	0.9 hours
	9 patients	range	0.5–2.0

ized determinations of urine osmolarity and sodium concentration in four patients, and differential renal scans with both technetium-99 and I-131 hippuran for calculations of individual renal plasma flows and urine flows, respectively, in 19 patients. Intravenous pyelograms, even if of the infusion variety, were found to be relatively inaccurate, so that no great emphasis was placed on the rapidity of opacification or dye excretion. Retrograde catheterization of the ureter imposed an additional hazard, plus considerable extra discomfort on the patient; and thus this particular method of follow-up was soon abandoned. Isotopic scan, however, was readily available and appeared to be relatively innocuous to the patient. Crude data gained were then transmitted to the Analytical Development Associates Corporation for detailed computerized radio renography. This MEDNET system provided relatively accurate determinations of split renal plasma flow and urine flow.

With respect to deaths, every attempt was made to obtain a full autopsy. Both gross and microscopic examination of the kidneys was considered to be essential in these cases.

Results of Clinical Study

After 14 months, only 21 patients had met the necessary criteria for inclusion in the study. There were 17 blacks and four whites, 17 males and four females. Ages ranged from nine to 51 years with an average of 35.8.

Trauma was responsible for the hemorrhagic shock in 20 of the 21 patients. In 14, it was due to a gunshot wound, blunt injury in four, and a stabbing in two. The final case was that of a ruptured abdominal aortic aneurysm.

Severity of shock was reflected in an average of 6.2 liters of blood and 4.6 liters of Ringer's lactate being used per patient for fluid resuscitation alone (Table 5). The duration of shock with respect to significant reduction in blood pressure was also in line with the criteria previously set. Nine patients had experienced profound shock (blood pressure of 40/0 or less) for an average of 0.9 hours and 16 had had a moderate shock (blood pressure between 40/0 and 60/40) for 1.1 hours, while

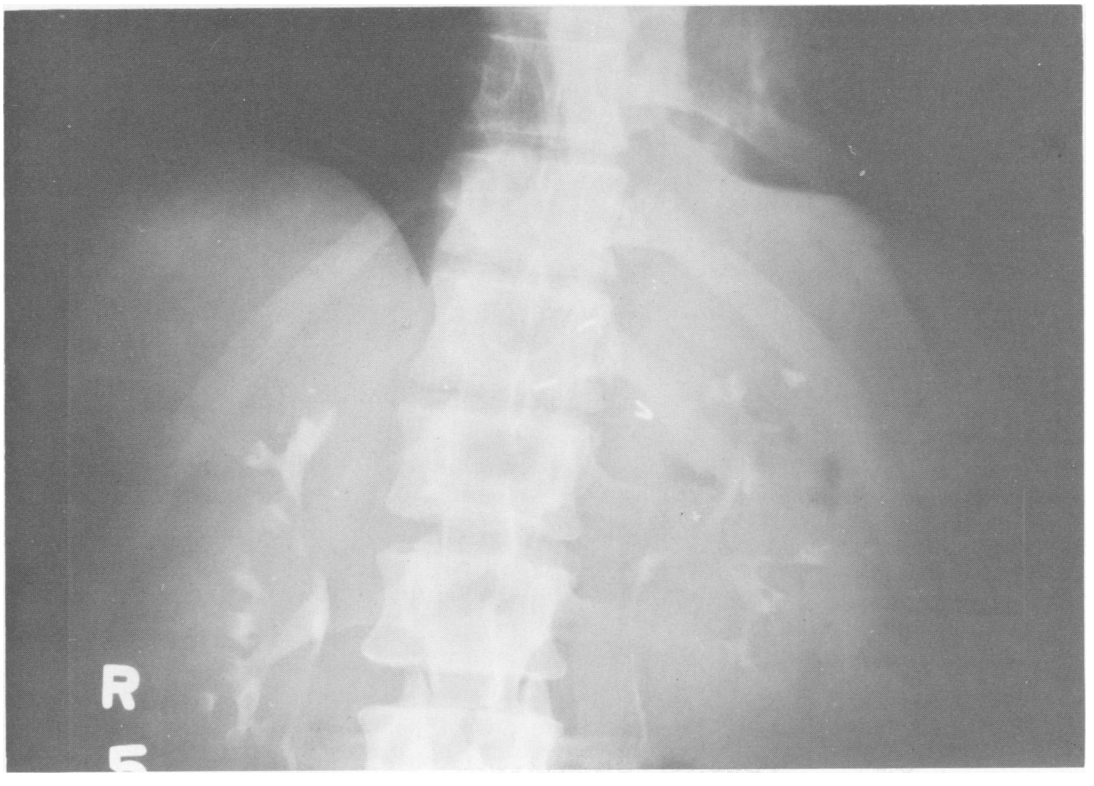

FIG. 4a. Patient J. T.: Intravenous pyelogram demonstrating delayed dye excretion on the left (prior right kidney decapsulation).

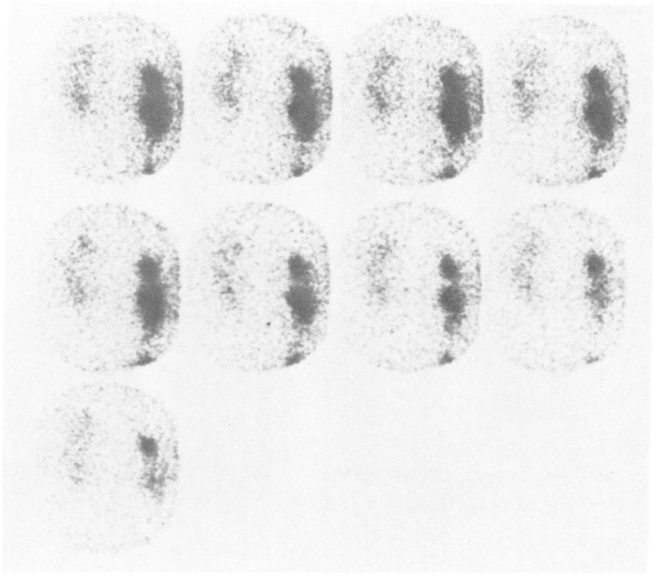

FIG. 4b. Patient J. T.: I-131 hippuran scan, showing impaired excretion of isotope by the left kidney (prior right kidney decapsulation).

all 21 had sustained at least a mild shock (blood pressure no greater than 80/60) for 1.9 hours.

There were three deaths. In one, the suture line of an aortic repair became infected, dehisced, and led to irreversible shock. The second patient, already in Child's class C liver failure, succumbed to hepatic insufficiency. The third death occurred late (*i.e.*, on the twenty-seventh hospital day) and was due to the sudden onset of a shock-like state that was never fully understood. Autopsies in the first two patients cited confirmed the clinical impression as to cause of death. Unfortunately, post mortem examination was refused in the final case.

Significant impairment in kidney function interpreted as a serum creatinine greater than 3 mg/100 ml and/or a creatinine clearance of less than 30 ml/min, could not be detected in six of the patients. However, each of the remaining 15 had classical polyuria of high output renal failure, with only one patient having a single day of supposed oliguria (327 ml of urine on the sixth postoperative day). This exception was believed to be the result of a careless collection error. Three of the 15 patients required renal dialysis just once, while two others were dialyzed three and five times, respectively, because of the extra metabolic demand created by a poorly controlled sepsis.

Split Function Studies

Intravenous pyelography was inconsistent at showing a difference between the two kidneys when renal failure was known to have developed. However, never did the non-decapsulated side appear to opacify sooner or to excrete a greater quantity of dye. Whenever an obvious difference was noted, the image on isotopic scan and the counts of isotope excreted were much more dramatic indications of better function by the decapsulated kidney (Figs. 4a–c).

Retrograde ureteral catheters failed to collect the entire output of urine from their corresponding kidneys because of an unreliable fit of the catheter tip into the ureteric orifice. Nevertheless, urine osmolarities and sodium concentrations varied more appropriately with the state of hydration on the decapsulated side than with the control side (Fig. 5). Forced hydration with an infusion of 5% glucose in water at a rate of 200 ml/hour produced significant differences ($P < .01$) in both sodium concentration and osmolarity within three hours. Subsequent water restriction created similar discrepancies, but in opposite directions, after a considerably longer interval of time, *i.e.*, ten hours. Differential renal scans were obtained on the third to twenty-seventh postoperative day in 19 of the cases and demonstrated no variance between the two kidneys in six patients who had evidenced no renal impairment

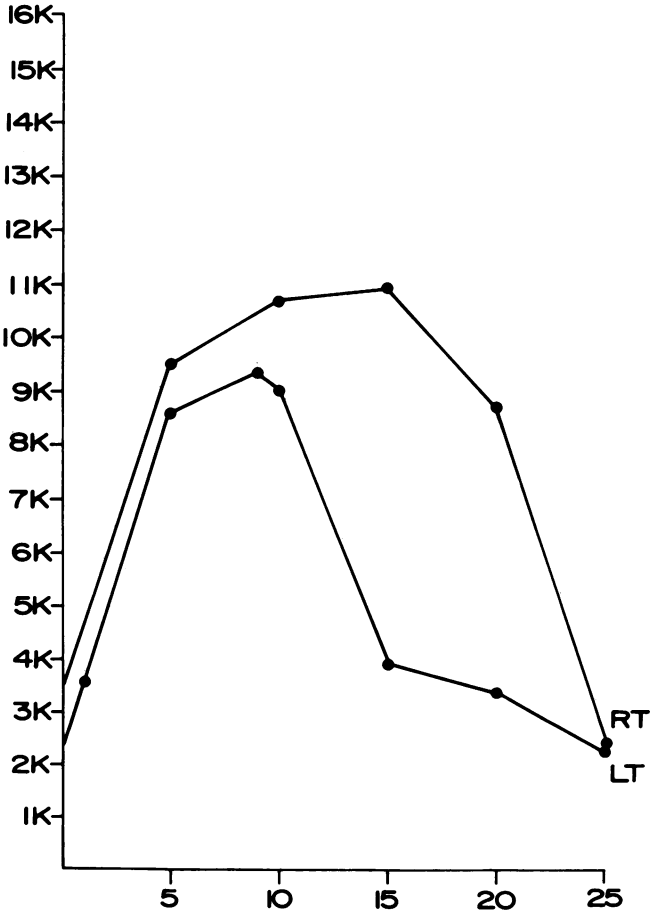

FIG. 4c. Patient J. T.: Graph of I-131 hippuran excretion, confirming reduced function of the left kidney (prior right kidney decapsulation).

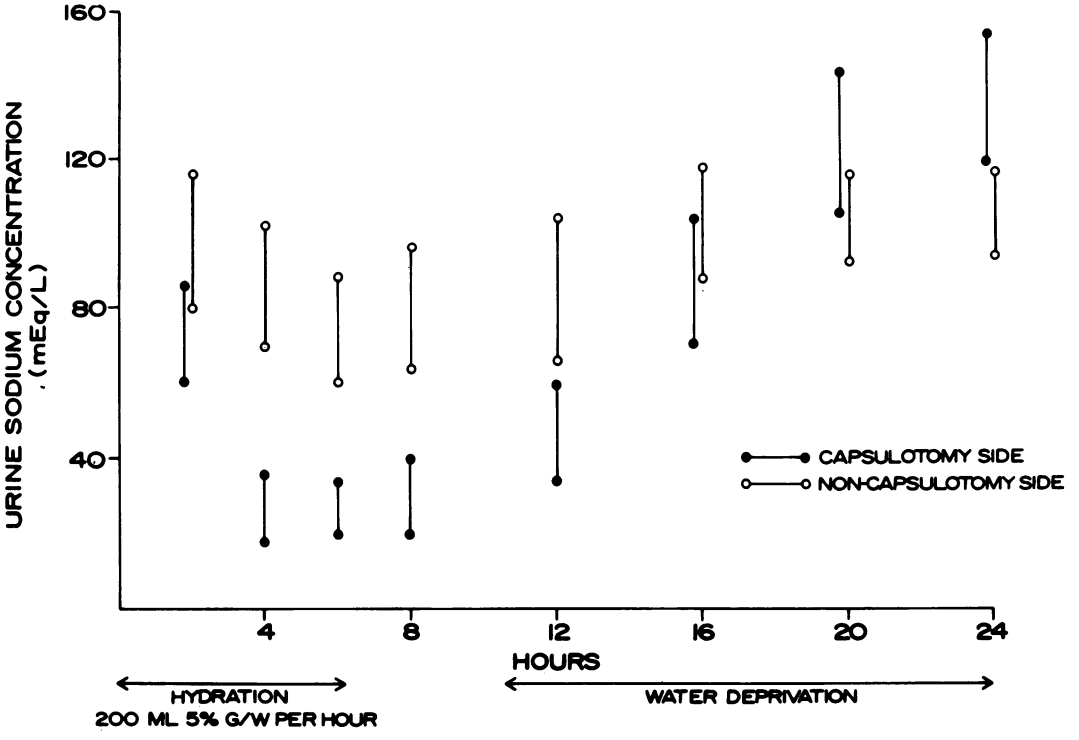

FIG. 5. Ability to vary sodium concentration as determined by differential ureteral urine collections. The decapsulated kidneys behaved more appropriately to the state of hydration than did the kidneys with capsules intact (average of four patients).

and in only one of the 13 patients with polyuric failure. The scans in two patients have only recently been obtained so that final analysis of their data by computer is still pending. However, of the ten remaining patients with polyuric renal failure, a significant difference ($p < .01$) between the two kidneys was noted (Table 6). Renal plasma flow was 160% greater and urine flow was 550 times greater on the decapsulated side than on the side with the capsule still intact.

An immediate diuretic response to renal decapsula-

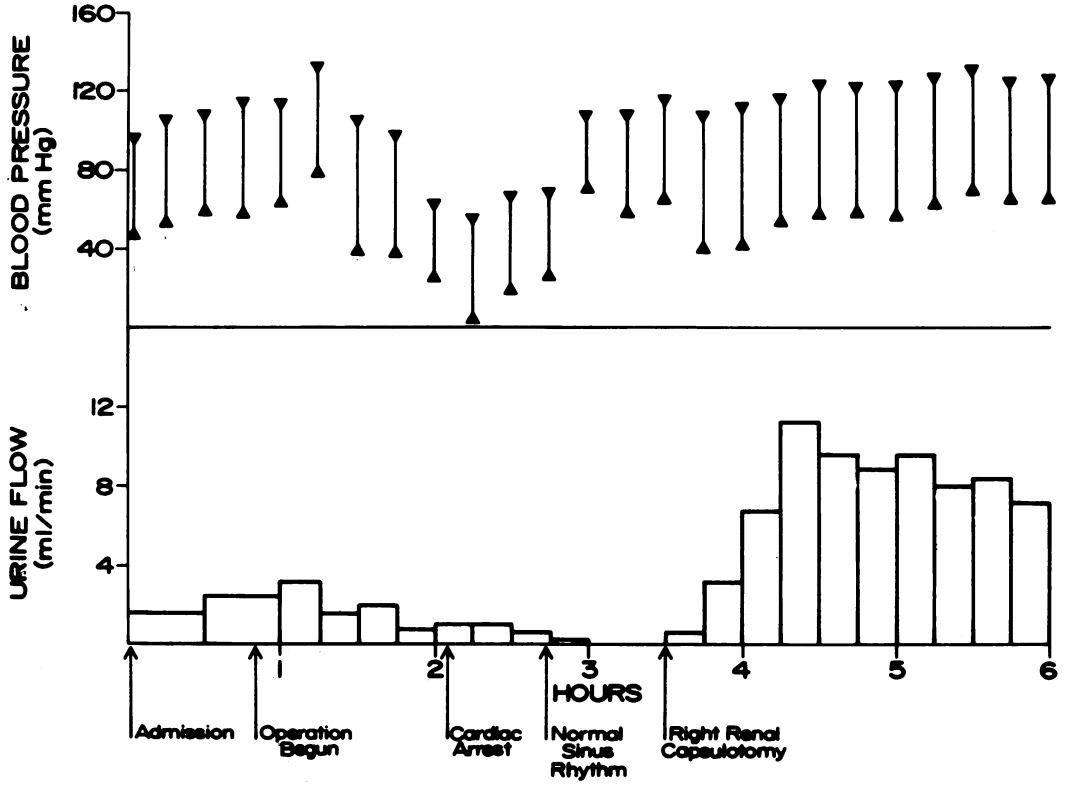

FIG. 6a. Patient T. C.: Sudden onset of diuresis, following three hours of hypovolemic anuria, when the right renal capsule was removed.

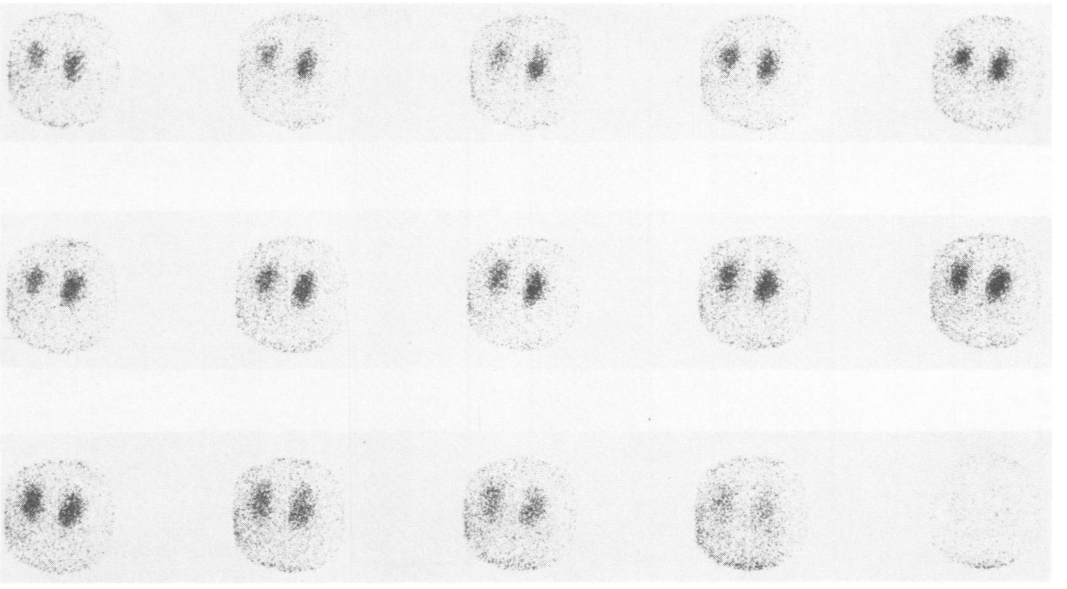

FIG. 6b. Patient T. C.: Follow-up renal scan, showing reduced excretion of I-131 hippuran by the left kidney (prior right kidney decapsulation).

tion was noted in only three patients, yet these were the same individuals who had been anuric from the beginning of their operation up until the time of capsule stripping. Not only did a dramatic flow of urine commence, but in addition the kidney parenchyma seemed almost to explode out of its incised capsule. Although decapsulation was carried out on just one side, late follow-up uniformly demonstrated significantly greater function of the kidney that had been decompressed surgically than that exhibited by its contralateral control (Figs. 6a–c).

During 1976, 18 patients underwent renal decapsulation while on the Trauma Service at Grady Memorial Hospital (Fig. 7). Six had no evidence of subsequent renal impairment, but each of the other 12 had some degree of polyuric failure. There was not a single instance of persisting oliguria or anuria among these patients. By contrast, non-polyuric renal failure was relatively common (37%) among 30 patients who, during the same 12 month interval, fell short of the strict criteria set for admission to the study, *i.e.*, hypovolemic shock not profound or long enough or sepsis also being present. Even more striking, however, was an 18% incidence of anuria and a 58% incidence of oliguria on the entire surgical service during the five preceeding years.

TABLE 6. *Renal Function (10 Patients with Polyuric Renal Failure)*

	Decapsulated Kidney	Capsule Intact	p Valve
Renal plasma flow (ml/min/M²)	194	121	<.01
Urine flow (ml/min/M²)	.99	.18	<.01

Autopsies were obtained on two of the three patients who died. In both, the decapsulated kidney appeared healthier, *i.e.*, grossly having a better blood supply. One set of kidneys was of approximately the same weight, while the kidney without its capsule weighed 39% more than its contralateral mate in the second case. Microscopically, however, no difference in extent of renal injury could be proven in either patient.

Discussion

There are three anatomic areas generally considered to be subject to the development of a compartmental

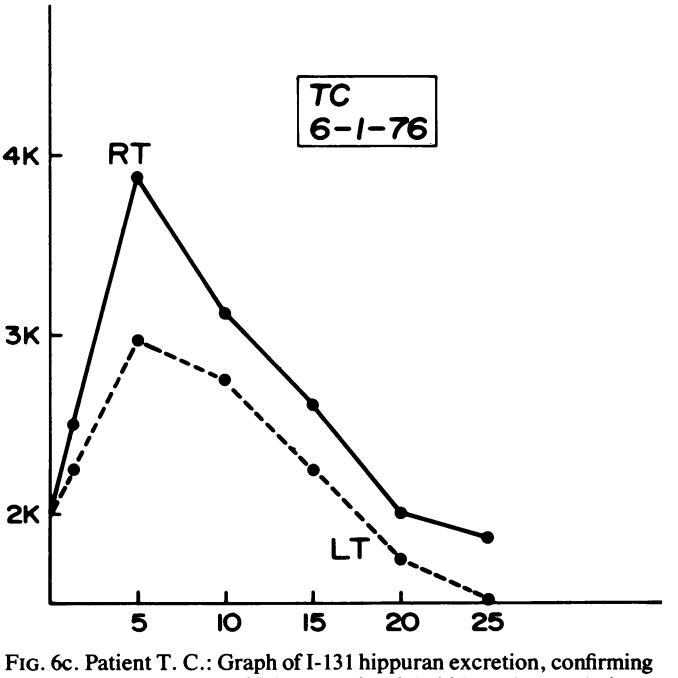

FIG. 6c. Patient T. C.: Graph of I-131 hippuran excretion, confirming reduced function of the left kidney (prior right kidney decapsulation).

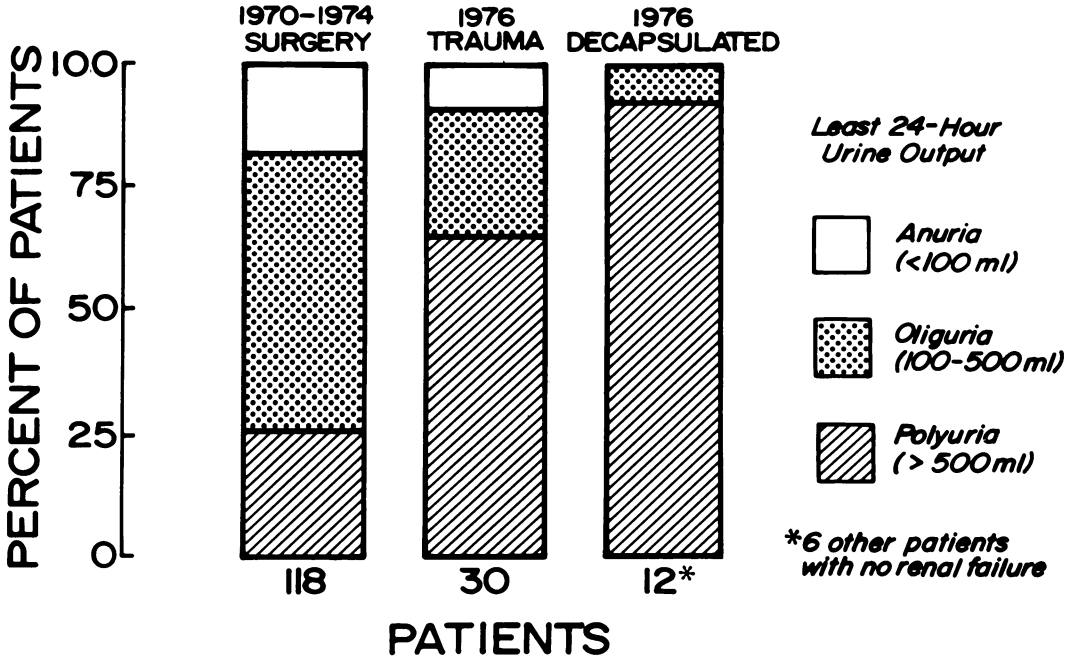

FIG. 7. Severity of renal failure according to daily volume of urine excreted.

swelling syndrome. These are the intracranial cavity, the orbital globe as in glaucoma, and the anterior compartment of the leg.[7,11,15] Other finite spaces, such as the posterior compartment of the leg, may on less frequent occasions manifest a similar set of findings.

The basic problem is an acute increase in volume, usually due to edema or hemorrhage, of the contents of a specific anatomic compartment.[7,11,15,22,34] Unfortunately, the wall presents an unyielding barrier of bone or mature fascia.[7,11,15,33] The end result is a reduction in the amount of arterial flow and thus the creation of a secondary ischemic injury.[19,20,22,26,30,32] The same process may well occur within the renal capsule as a consequence of ischemia, myoglobin or hemoglobin excretion in the urine, or one of a multitude of chemical toxins.[1-4,8-10,12-14,16,17,21,24,28,29]

TABLE 7. *Collection Review of Renal Decapsulation (Modified from Abeshouse—up to 1944)*

Diagnosis	Number	Improved	Per cent
Acute nephritis	246	176	71.5%
Chronic nephritis	565	390	69.0%
Acute or chronic nephritis	471	427	90.7%
Acute pyelonephritis	112	61	54.5%
Chronic pyelonephritis	2	2	100.0%
Chronic perinephritis	62	62	100.0%
Degenerative nephrosis	108	73	67.6%
Toxic nephrosis	613	255	41.6%
Cortical necrosis	13	4	30.8%
Transfusion reaction	3	3	100.0%
Non-obstructive anuria	32	26	81.3%
Miscellaneous	80	64	80.0%
Total	2307	1543	66.9%

Treatment of an impending or already established compartment syndrome generally includes: 1) efforts directed at fluid extraction or tissue excision, both being designed to diminish the absolute intracavitary volume and thus the intracavitary pressure, and 2) removal of some part of the confining wall, thereby allowing unrestricted swelling to take place. Diuretics,[3,8,10,14,15,19,20,26,32,34] corticosteroids,[5,22,27] and local or general hypothermia[9,15,31] have proven to be quite useful in reversing cerebral edema. Direct aspiration of accummulated fluid may instead be the more desirable course of action, as in cases of expanding intracranial hematomas or cerebrospinal fluid excess with hydrocephalus.[11,15,31,33] There are instances, however, when absolute reduction in tissue volume is preferable.[15,33] An example of this is when temporal lobectomy is done for relatively massive cerebral edema.

Decompression by elimination of a section of the restraining wall has been exceedingly useful in the prevention and early treatment of the anterior compartment syndrome.[7] In a similar fashion, subtemporal craniectomy has been performed to relieve increased intracranial pressure.[11,33] Renal decapsulation may produce its beneficial effects through this same mechanism whenever kidney swelling can be anticipated.[13,21,24]

Abeshouse has detailed quite thoroughly the history of renal decapsulation.[1] Harrison did the first recorded kidney puncture in 1878; Le Dentu, the first full capsulotomy in 1881; and Pousson, the first true decapsulation in 1901. A summary of the world experience with renal decapsulation up to 1944 has also been provided by

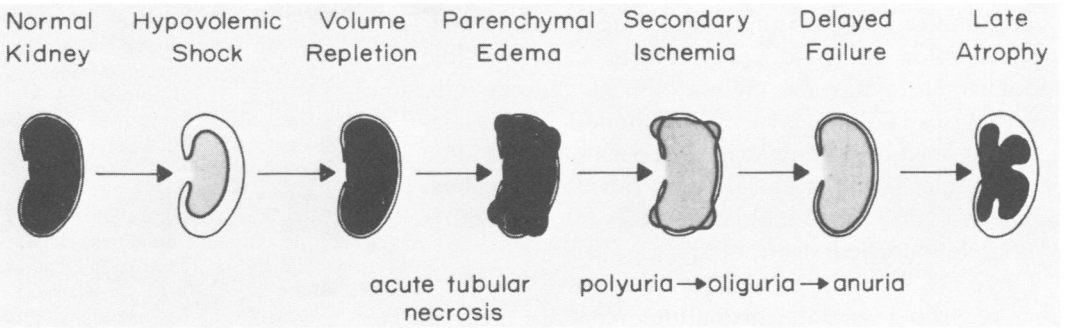

FIG. 8. Theorized steps in progression of polyuric acute tubular necrosis to anuric acute cortical necrosis.

Abeshouse (Table 7). He collected a total of 2307 cases. Some form of improvement in kidney function followed operation in 1543, or 66.9% of the entire group. Of the 13 patients with renal cortical necrosis, a good response was obtained in only four, or 31%. Nevertheless, a more accurate assessment of those results was not possible because of relatively nonspecific diagnoses, little in the way of objective data on renal function, and a somewhat limited follow-up.[2] In addition, a majority of the decapsulations performed for cortical necrosis were delayed until after a week of anuria had elapsed.[16]

Few animal studies have evaluated the relative merits of renal decapsulation. Two separate experiments in rats are somewhat conflicting, in that there was early benefit in preserving renal integrity in rat kidneys made edematous by choline deficiency,[6] while the life span of rats subjected to renal ischemia was shortened by removal of the kidney capsule.[23] The only other study was in pigs, where decapsulation decreased the amount of renin produced by an ischemic kidney.[18]

From that data presented in the above animal and clinical investigations, plus specific experiences with renal decapsulation as cited in the medical literature,[1,2,6,13,16,21,24,29] it would appear that removal of the resilient capsule of at least one kidney may well be indicated in most cases of profound hypovolemic shock due to intraperitoneal hemorrhage. No additional operation is necessary, for successful control of the bleeding has almost always required a formal laparotomy. Recorded failures in response have primarily been in those instances where decapsulation had been delayed until after a week or so of anuria and thus an already well established renal cortical necrosis.[1,2,16] Earlier intervention, that is, shortly after renal failure had been discovered, has given significantly better results.[1,13,21,24,29]

Up until 1945, there were many strong proponents of renal decapsulation.[21,24] However, peritoneal dialysis was then introduced; this was later replaced by a more sophisticated hemodialysis, and, accordingly, all thoughts of capsule stripping were eclipsed.[28,29] Like any other procedure, decapsulation must be used at the time when indicated and for a specific purpose. Otherwise, no real benefit should be expected.

There is now available a considerable amount of information to support the contention that polyuric renal failure may progress to oliguria and then anuria as a consequence of renal parenchymal swelling within its tough, unyielding capsule.[4,12,24] The initial insult can be ischemia, a transfusion reaction, or some chemical toxin; yet the response of the kidney is almost monotonous—interstitial and intracellular edema.[24,28] If a compartment syndrome is allowed to develop, the secondary ischemic injury will lead to extensive cortical necrosis (Fig. 8). At times, however, the original injury may have been so severe that necrosis of the renal cortex, intraparenchymal hemorrhage, and anuria are present from the outset.

To avoid this progression of events, either kidney swelling must be avoided or the renal capsule removed. Local hypothermia and various diuretics appear to have only a transient beneficial effect.[3,8–10,12,14,17,28]

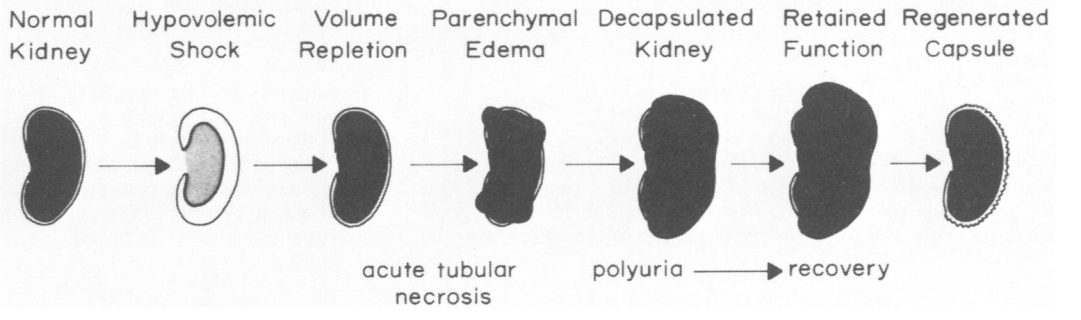

FIG. 9. Avoiding progression from polyuria to anuria. Theorized role of kidney decapsulation in prevention of secondary renal ischemia due to compartmental swelling.

Decapsulation, on the other hand, offers a more lasting protection against development of an irreversible compartment syndrome. When clinical findings indicate that renal failure may well be imminent, decapsulation of at least one kidney can then permit unrestricted parenchymal swelling and thereby obviate a progression of polyuric acute tubular necrosis into the anuria of complete cortical destruction (Fig. 9).

There are still a large number of unanswered questions regarding renal decapsulation. What are the long term effects, good or bad? Is unilateral decapsulation sufficient in most cases? How late can decapsulation be performed and yet a good response be obtained? However, probably the most pressing question of all is whether or not decapsulation should now be undertaken as a separate operative procedure itself in cases of impending or early renal failure not associated with abdominal surgery.

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Discussion

Dr. Hiram C. Polk, Jr. (Louisville, Kentucky): This is an extraordinarily innovative resurrection of an old idea. I simply wanted to ask Dr. Stone one question. We recognize the relatively good-prognosis polyuric form of renal failure as being a favorable situation further defined here. However, there is the oliguric variant with a very poor prognosis, as he showed, which more often than not seems to be associated with infection. I wonder if he has been able to show, either in the laboratory or in man, the ability to convert the poor-prognosis, oliguric form of renal failure into the better polyuric form, in an individual kidney, by decapsulation applied either early or late?