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Sequential System Failure after Rupture of Abdominal Aortic Aneurysms:

An Unsolved Problem in Postoperative Care

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CERTAIN COMBINATIONS of disease and trauma form lethal syndromes. Components of those syndromes, taken individually, can often be treated effectively, but when they occur together, therapy is unsuccessful. One of these combinations is acute renal failure and the otherwise successful surgical treatment of a ruptured aortic aneurysm. An example of particular interest, it is a worse-case challenge to present-day technics of postoperative care.

The mortality of ruptured abdominal aneurysm alone is 32%–85% for patients who live to reach the operating room. Some of these deaths occur during the operative and early postoperative periods in those individuals who are not completely resuscitated. Others go on to a phase of apparent stability. At this time, typically the first or second postoperative day, blood volume has been restored and hemostasis accomplished. The patient may have good cerebral and cardiovascular function and breathe spontaneously without assistance. Most patients reaching this point will survive, though subject to the ordinary vicissitudes of recovery from major operation. For one sub-group, those with fixed renal failure, survival is exceptional.

Alone, the mortality of uncomplicated renal failure is small, approaching the risk of chronic hemodialysis. This has been reported as only 3% when the underlying condition is "slight or cured."[1] The hazard of ruptured abdominal aortic aneurysm, and acute renal failure together is greater than the sum of their risks taken singly. Almost all patients subjected to this combination will experience progressive, sequential, organ system failure and eventually succumb. Our experience with patients in this category is reported to demonstrate the need for a more effective approach to their therapy, and in its absence, as an aid to judgment in the difficult balancing of expenditure of medical resources against probable gain to the patient.

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Clinical Material

Patient Selection

This series includes all patients treated by hemodialysis for postoperative acute renal failure after rupture of an abdominal aneurysm during the past 15 years.

TABLE 1. *Occurrence of Unfavorable Prognostic Factors in 18 Patients with Acute Renal Failure after Ruptured Abdominal Aneurysm*

	Number	Per Cent
Pre-existing Systemic Disease		
Chronic pulmonary insufficiency	9	50%
Heart disease	14	78
Infarct by history or EKG	11	
Congestive failure	2	
Arrhythmia	1	
Hypertension	10	56
Obesity	6	33
Intraoperative events		
Hypotension (<80 mm. Hg)	16	80
Cardiac arrest	4	22

Patients who did not survive for 5 postoperative days were excluded to eliminate those for whom operative resuscitation was unsuccessful. Clinical information has been taken from hospital and intensive care records; the authors participated in the care of the majority.

Composition of the Series

Eighteen patients meeting this description were found in the records of the Peter Bent Brigham Hospital for the 15-year period ending January 1, 1971. Only one patient survived. Fifteen (84%) were men, and three were (16%) women. Mean age of men was 63 years, of women 76 years. Five patients had been transferred postoperatively from other hospitals. For the majority, a number of factors correlating with increased risk were recognized preoperatively or occurred during operation. As shown in Table 1, 16 patients were hypotensive (systolic pressure less than 80 mm. Hg at some time). Cardiac arrest occurred during operation in four and a thoracotomy was performed in three of these patients. Ten patients underwent a second operative procedure of some sort at some time, exclusive of arteriovenous shunt insertion (Table 2). Seventeen patients died; the mortality thus exceeded 90%. The mean time to death was 18 postoperative days (range 5-48).

Analysis of Data

The objective in reviewing the clinical record was to learn at what time during the course, failure of an organ or organ system was first observed. Evidence was obtained from records of laboratory data, bedside observation, or the initiation of therapy in support of a particular system. Criteria defining failure were necessarily specific to the organ or system. For the pancreas it was serum amylase levels twice normal (beyond the expected renal failure range). Pulmonary failure was defined as need for mechanical ventilation. Liver failure was defined as observation of clinical jaundice (or serum bilirubin greater than 3 mg./100 ml.). Failure of the central nervous system (CNS) was defined as failure to respond to other than painful stimuli. Failure of the gastrointestinal tract was equated to bleeding that required transfusion. Gross blood from the nasogastric tube distinguished failure of the upper GI tract, bright red blood from the rectum signified failure of the lower tract. Cardiac failure was established either by hypotension with elevated right atrial pressure, an arrhythmia compromising cardiac output, or electrocardiographic evidence of myocardial infarction.

An effort was also made to assign an immediate cause of death by combining data from autopsy and clinical records. Though subject to judgment, for most of the patients this determination was made without difficulty.

TABLE 2. *Surgical Procedures after Aneurysmectomy*

Procedure	Number
Femoral Embolectomy	4
Laparotomy for Bleeding	2
Femoral Vein Ligation	1
Pyloroplasty, Vagotomy	1
Sigmoid Resection	1
Insertion of Pacemaker	1
Tracheostomy	12
Arteriovenous Shunt	18

Treatment

A maximum and concentrated therapeutic effort was made for every patient. Details of management varied over the period covered by the review. Antibiotic agents of increased effectiveness became available during this time (but have been matched by an increased frequency of significant infection by previously commensal organisms). Pressure cycled ventilators were replaced by volume limited machines. The incidence of tracheostomy decreased, and the use of arterial and central venous monitoring catheters became universal. The principles underlying use of dialysis have been little changed. They are early and frequent hemodialysis (4–6 hours every second day; maximum flow consistent with hemodynamic stability). Recently, the use of parenteral nutrition has slowed the rate of increase of blood urea and potassium concentration during intervals between dialyses, but the same treatment schedule is maintained in order to palliate the other components of the uremic syndrome: CNS depression, interference with wound healing, and increased susceptibility to infection.

Results

Although 17 of the 18 patients died, there was a wide distribution of survival times. One died on the 5th postoperative day, one on the 48th; the remaining 15 deaths occurred between these extremes. Despite these differ-

ences, a similar progression of organ system failure was observed in all. The sequence unfolded more slowly in patients who survived longer, and developed quickly in those surviving for shorter periods, but most patients developed all components of the pattern in approximately the same order before death occurred. This is shown in Figure 1 where the onset of organ system failure for all patients is compared on a common time scale. There was some variation from patient to patient in the manner of presentation and pathophysiology of specific organ system failure, as follows.

Central Nervous System

Some form of major neurologic abnormality was recorded in all patients but one. The commonest was coma, occurring in 11. It was manifested by lack of response to all but painful stimuli or to tracheal stimulation but without localizing signs or primitive reflexes. Five patients experienced six episodes that were marked by focal neurologic findings; three occurred early and were probably caused by local ischemia or low-flow thrombosis during the shock period. Three episodes occurred terminally.

Lungs

Respiratory failure which occurred in all of these patients was the leading cause of death (Table 3). All patients who died required mechanical ventilation before death. Contributory causes to impairment of pulmonary function, such as hepatic failure, tended to follow a pattern of early appearance, apparent improvement, and then secondary progressive failure. Morphologic changes at autopsy were extensive and nonspecific. Lungs were heavy; microscopically there was bronchopneumonia, interstitial edema, alveolar hemorrhage and hyaline membrane formation. Organisms were grown from all postmortem cultures, *Pseudomonas* or *Escherichia coli* from the majority, a mixed group of Gram-negative enteric organisms from the remainder, but in only one patient was there major tissue reaction to infection. None had significant pulmonary emboli.

Heart

It is accepted that an arteriosclerotic abdominal aortic aneurysm is a late manifestation of generalized cardiovascular disease, attested to by the presence of an old, healed myocardial infarction in more than half of these patients and by occurrence of a new infarct during the postoperative period in three. All occurred during the second postoperative week. Four other patients experienced significant arrhythmias requiring treatment by cardioversion, pacemaker, or drugs. Autopsy showed extensive coronary artery disease in all patients; uremic pericarditis was found in two.

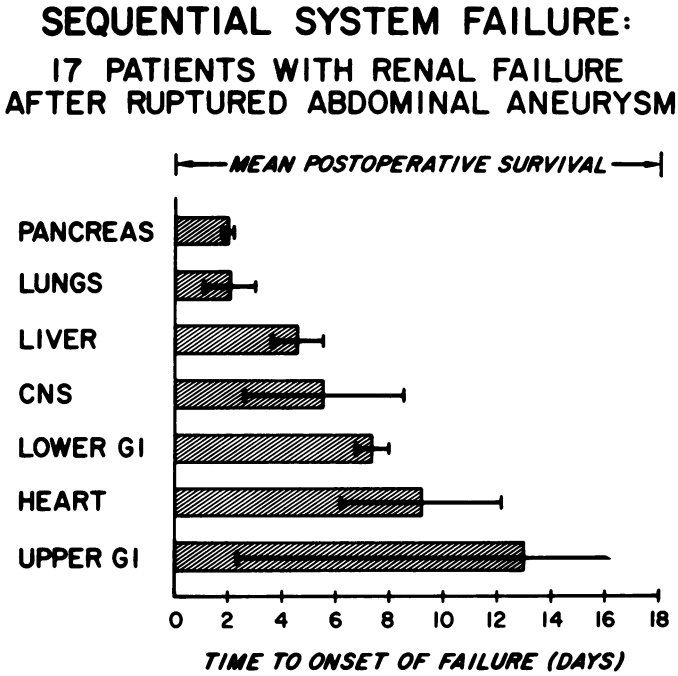

FIG. 1. The order of appearance of organ system failure is shown by normalizing survival time for each patient to mean duration of survival for the group.

Liver

All patients were jaundiced, chemically or clinically. Bilirubin and enzyme levels followed a characteristic pattern: early, transient elevation of hepatocellular enzymes, a later and more or less steady increase in bilirubin to very high levels (Fig. 2). Histologic changes were correspondingly severe—centrilobular necrosis, significant bile stasis, congestion and lipid infiltration. There was one instance of ductal obstruction by pancreatitis, one of multiple septic infarcts.

Pancreas

Fulminent pancreatitis was recognized early (on the second postoperative day) in two instances but could be seen retrospectively to have occurred in a total of nine patients, in whom severe pancreatitis was found at autopsy, and could be correlated with antemortem amylase elevation and a plasma requirement. Pan-

TABLE 3. *Proximate Causes of Death (17 Patients)*

	Primary	Secondary	Total
Respiratory Failure	9	4	13
Septicemia	3	7	10
Gastrointestinal Hemorrhage	1	1	2
Acute Pancreatitis	2	1	3
Cardiac Failure		2	2
Uremia		1	1
Multiple Factors	2	1	3

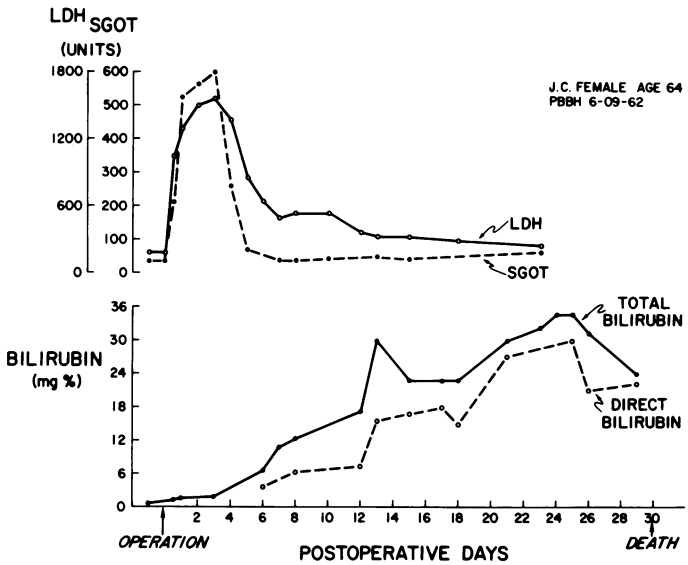

FIG. 2. The characteristic evolution of plasma enzyme and bilirubin levels as seen in one patient.

creatitis was hemorrhagic and necrotizing in four, in three there was inflammation, edema, and fat necrosis.

Gastrointestinal Tract

Gastrointestinal complications occurred late in these patients (Fig. 1) but were seen in all. Some complications were recognized prior to death: major upper gastrointestinal hemorrhage in eight patients (one died of recurrent bleeding after pyloroplasty and vagotomy); massive bleeding from the colon in two, sigmoid perforation in one. At autopsy previously unrecognized necrosis and perforation of the sigmoid was present in two patients, some degree of gastric erosion and ulceration was recognized in all.

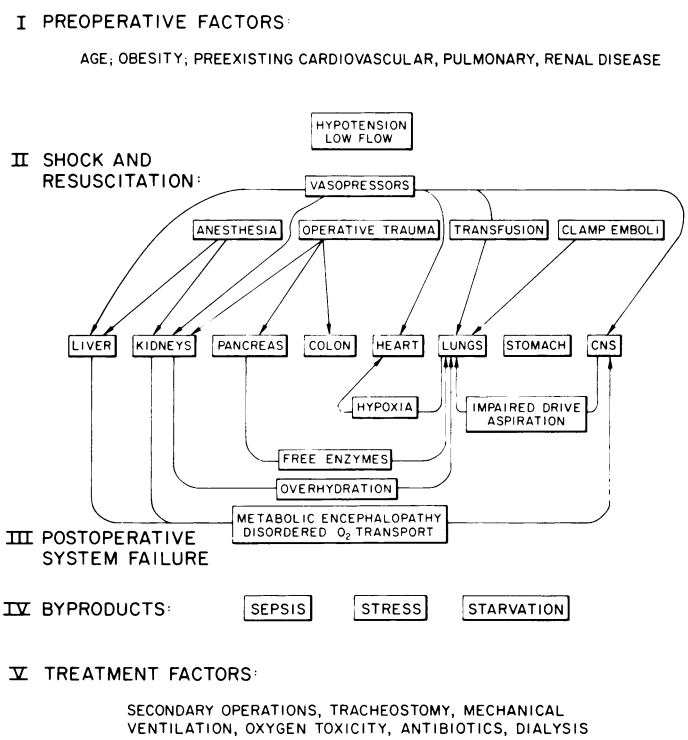

FIG. 3. How one occurrence leads to another: some of the pathophysiologic mechanisms interrelating system failure. Preoperative factors, and those associated with shock and resuscitation (I-II) are established at the beginning of the postoperative course.

Kidneys

Autopsy demonstrated bilateral acute tubular necrosis superimposed on varying degrees of nephrosclerosis in most of the patients. There were several exceptions: bilateral cortical necrosis in one patient, in two examinations cortical necrosis was found in one kidney, tubular necrosis in the other. One case emphasized a clinical principle, the importance of demonstrating bilateral patency of the ureters. A clear lower tract was shown by retrograde pyelography of the left kidney but the right ureteral orifice could not be intubated in this individual. Autopsy revealed renal infarction on the left, with ureteral ligation and hydronephrosis on the right.

Discussion

The question presented by these patients is familiar and fundamental: how do a number of physiologic liabilities, each in itself potentially reversible, combine to make survival exceptional, though not impossible; for even in this group there was one survivor. This individual did not differ from the others in age, preoperative cardiovascular status, or need for postoperative respiratory support.

There are established interrelationships between organ systems which help explain both the additional hazard created by renal failure, and the characteristic sequence of events during the postoperative course. Failure of one system challenges others and improvement in survival depends on identification of points where these cycles can be interrupted. Some of them are not controllable. For example, those existing preoperatively or occurring intraoperatively (Fig. 3) are difficult or impossible to modify. Many operative factors are common to survivors of hemorrhage, shock or trauma generally, a few are peculiar to aneurysmal resection. For example, aortic cross clamping may cause renovascular constriction, atheromatous embolism, parenchymatous arteriovenous shunting, and renal artery pressure changes.[3] Clamp release can initiate washout acidosis, increasing damage to already compromised renal tubules, and possibly allow a shower of microemboli to reach the lungs.[2] Direct operative trauma to the kidney or collecting system can occur, as it did in one patient in this series; similarly, operative trauma to the pancreas or the

inferior mesenteric circulation may set the stage for problems later, and pancreatitis occurred frequently.

Respiratory failure, however, was the commonest proximate cause of death. Successful resuscitation from massive hemorrhage and management of the metabolic consequences of renal failure allows development of what has been variously called shock lung, post-traumatic pulmonary insufficiency, or progressive pulmonary insufficiency.[5] Less clearly related to operative events, its etiology is probably mixed and possibly unknown; a long list of factors which may share responsibility can be made. The principal difficulty in dealing with the syndrome of post-traumatic pulmonary insufficiency lies in assessment of the relative importance of these possible causative factors. Some are part of the resuscitation and hence unavoidable: microembolism after aortic cross clamping, previously mentioned, the load of embolic material imposed by many transfusions, and the pulmonary vascular response to hypovolemia and to vasopressors which may be used in its treatment. After resuscitation, the lungs remain the principal target of changes initiated by failure of other organs and of the undesirable effects of necessary therapy. Figure 3 shows some of these relationships, in a necessarily abbreviated way. Not all can be modified by present day methods but some can. Of those influencing the lung, for example, overhydration can be avoided or treated by early dialysis. All of these patients had received large quantities of water and sodium for initial replacement of lost intravascular volume, intraoperative extracellular fluid expansion, and as a trial of volume loading when oliguria was first noted. Large positive water balances could be calculated at the time of admission to the intensive care unit. Simply restricting water leads to hypertonicity unless gastrointestinal losses are large: combining some water restriction with hemodialysis (massive water removal by ultra-filtration may not be tolerated hemodynamically) improves the chest X-ray and alveolar-arterial gradient for oxygen, but avoiding the early phase of hyperhydration might be an advance. Good principles of respiratory management are certainly important, but obviously not the whole answer, and need not be detailed here. Maintenance of nutrition parenterally may be important, at least to maintain respiratory muscle function and possibly in more indirect ways.

Gastric mucosal ulceration, like postoperative pulmonary insufficiency, is a standard surgical topic. Recent reports emphasize the importance of gastric resection in its treatment, and this series includes a patient who rebled after pyloroplasty and vagotomy. Other than this there was little to learn except that episodes of bleeding usually occurred in association with hemodialysis, even when regional heparinization was tightly controlled, and that massive upper GI bleeding was a late event, which in a setting of multiple organ system failure might have been interpreted as marking the point at which vigorous therapy should be discontinued.

Renal failure, once clearly established, is unlikely to resolve before hemodialysis is required. These patients have additional loading of potassium and nitrogen metabolites from hematoma and devitalized tissue even where hypercatabolic nutritional requirements are met parenterally. The objectives are to keep serum potassium within the normal range, avoid central depression, and to maintain dry lungs. The effect of intensive dialysis on wound healing has been established in man and animal[6,7]; there is also evidence from animal studies that dialysis can partially reverse the effects of uremia on leucocyte function, the gastrointestinal epithelium, and hematopoiesis.

The universal occurrence of profound increasing jaundice was a distinguishing feature. Explanations are available: a large pigment load from transfusions and hematoma with no renal clearance of plasma bilirubin; splanchnic vasoconstriction in hypovolemic shock profound enough to cause acute renal failure also results in hepatocellular damage.[6] In patients in whom acute renal failure followed operations other than aneurysm resection, where survival is more frequent, bilirubin levels often continued to be elevated for weeks after diuresis and return of BUN and creatine to normal levels.

Diagnoses were missed despite close observation and frequent re-evaluation; most often they were intraabdominal complications: *e.g.*, pancreatitis and sigmoid perforation. The treatment of pancreatitis presents few surgical options, although its frequency in this series illustrates the truth that not all amylase elevation in acute renal failure is due to impaired clearance. Ischemic necrosis and perforation of the sigmoid colon after aneurysm resection, such as massive bleeding from stress ulcers, carries an extremely high mortality.[4] Again, this event might in the whole setting of the patient's preoperative state of health, family wishes, and available resources allow some judgment to be made concerning the wisdom of further treatment.

Summary

Eighteen patients required hemodialysis after surgical treatment of ruptured abdominal aortic aneurysm. Review of the clinical events and autopsy findings showed a similar progression of organ system failure, beginning with pancreatic to pulmonary disease, and progressing to upper GI bleeding. A significant incidence of clinically silent (or at least undetected) major intraperitoneal disease was also noted. The lethality of renal failure after ruptured aneurysm is explained as a superimposition of pre-existing chronic cardiovascular disease

on the mechanical and metabolic consequences of the surgical procedure. Even though some of the mechanisms are well understood, their combination initiates a cycle which is difficult to interpret: mortality for this group of patients was greater than 90%.

References

1. Balslov, J. T. and Jorgensen, H. E.: A Survey of 499 Patients with Acute Anuric Renal Insufficiency. Am. J. Med., **34**:753, 1963.
2. Blaisdel, F. W., Lim, R. C., Jr., and Stallone, R. J.: The Mechanism of Pulmonary Damage Following Traumatic Shock. Surg. Gynecol. Obstet., **130**:15, 1970.
3. Christiansen, J. and Mouridsen, H. T.: Ruptured Aneurysm of the Abdominal Aorta. Acta. Chir. Scand., **133**:111, 1967.
4. Kantrowitz, P. A., Jones, W. A., Greenberger, N. J., Isselbacher, K. J.: Severe Postoperative Hyperbilirubinemia Simulating Obstructive Jaundice. N. Engl. J. Med., **276**:591, 1967.
5. Moore, F. D., Lyons, J. H., Jr., Pierce, E. C., Jr., Morgan, A. P., Drinker, P. A., MacArthur, J. D., and Dammin, G. J.: Post-Traumatic Pulmonary Insufficiency. W. B. Saunders Co., 1969.
6. Nayman, J.: Effect of Renal Failure on Wound Healing in Dogs. Response to Hemodialysis Following Uremia Induced by Uranium Nitrate. Ann. Surg., **164**:227, 1966.
7. Schreiner, G. E. and Maher, J. F.: The Patient with Chronic Renal Failure and Surgery. Am. J. Cardiol., **12**:31, 1963.