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Planned Reoperation for Severe Trauma

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Objective
The authors review the physiologic basis, indications, techniques, and results of the planned reoperation approach to severe trauma.

Summary Background Data
Multivisceral trauma and exsanguinating hemorrhage lead to hypothermia, coagulopathy, and acidosis. Formal resections and reconstructions in these unstable patients often result in irreversible physiologic insult. A new surgical strategy addresses these physiologic concerns by staged control and repair of the injuries.

Method
The authors review the literature.

Results
Indications for planned reoperation include avoidance of irreversible physiologic insult and inability to obtain direct hemostasis or formal abdominal closure. The three phases of the strategy include initial control, stabilization, and delayed reconstruction. Various techniques are used to obtain rapid temporary control of bleeding and hollow visceral spillage. Hypothermia, coagulopathy, and the abdominal compartment syndrome are major postoperative concerns. Definitive repair of the injuries is undertaken after stabilization.

Conclusion
Planned reoperation offers a simple and effective alternative to the traditional surgical management of complex or multiple injuries in critically wounded patients.

Surgery for major trauma typically follows a well-defined sequence of steps consisting of access, exposure, control of bleeding, and reconstruction. This sequence represents a priority-oriented surgical approach whereby the most life-threatening injuries are addressed first and reconstructions are performed in a stabilized patient.

During the last decade, civilian wounding patterns have shifted to multivisceral, high-energy transfers (from automatic weapons and fast motor vehicles). Consequently, surgeons are confronted by increasing numbers of trauma patients with multiple injuries and exsanguinating hemorrhage, in whom the traditional operative sequence is inappropriate. The extensive multivisceral damage often requires lengthy and complex repairs in a

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shock patient whose bleeding (often from several sources) is difficult to control. Rapid surgical "bailout" tactics are the only option.

The planned reoperation approach, previously limited to packing of high-grade liver injuries,[1,2] was extended to nonhepatic abdominal trauma in 1982 by Stone et al.[3] They used rapid temporary measures to abruptly terminate a laparotomy and pack the peritoneal cavity of coagulopathic patients. Definitive repair was undertaken only after the coagulopathy was corrected and the patient was stabilized in the intensive care unit (ICU).[4–6]

The aim of this review is to present the physiologic rationale and application of this new approach and summarize the current clinical experience.

PHYSIOLOGIC CONSIDERATIONS

Several mechanisms combine to make surgery for major trauma an ongoing physiologic insult even in a seemingly "stabilized" normotensive patient. Bleeding may be difficult to control directly, as in a high-grade liver injury or a pelvic fracture. Unattended injuries, often in another body cavity, continue to bleed while one or two major sources of hemorrhage are being controlled. The need for vigorous blood replacement exposes the patient to the adverse effects of massive transfusion, including hypothermia and coagulopathy.[7,8]

Hypothermia is an inevitable and ominous process in the critically wounded.[9] Heat loss in the field and in the emergency room correlates with injury severity[10] and is further aggravated during surgery by exposure of body cavities, impaired thermogenesis, and massive transfusion.[11] A core temperature of 32 C or less was associated with a 100% mortality in trauma patients undergoing laparotomy.[9] From the operative viewpoint, the most relevant physiologic effect of hypothermia is coagulopathy. This primarily is because of platelet dysfunction and sequestration, although impairment of the coagulation cascade and increased fibrinolytic activity also have been implicated.[11,12] Other factors contributing to the rapid development of coagulopathy in trauma victims are massive transfusion,[7,8] hemodilution, and hypotension.[13]

Metabolic acidosis results from inadequate tissue perfusion. It has been shown to adversely affect myocardial contractility and cardiac output in animal models,[14,15] but there is little direct documentation of its physiologic consequences in the context of major trauma.[16] Nevertheless, severe metabolic acidosis is a reliable predictor of impending death in critically injured patients.[4,17]

The increased capillary permeability of traumatic shock and current aggressive fluid resuscitation practices combine to produce progressive visceral edema.[18] From the perspective of the operating surgeon, the resulting swollen bowel and thickened abdominal wall make formal abdominal closure increasingly difficult or even impossible.

The triad of hypothermia, coagulopathy, and acidosis creates a vicious circle whereby these derangements augment each other,[13,16] up to a certain point beyond which the physiologic insult becomes irreversible. Diffuse oozing in the operative field is followed by refractory ventricular arrhythmias and death.[4] An attempt has been made to predict the point of physiologic irreversibility based on transfusion rate and the patient's pH,[4] but currently, no precise and validated clinical guidelines exist.

INDICATIONS AND PATIENT SELECTION

The three distinct indications for planned reoperation in severely injured patients are as follows[19,20]:

1. Avoidance of irreversible physiologic insult in a hypothermic coagulopathic patient by rapid termination of the surgical procedure;
2. Inability to obtain direct hemostasis (by ligation, suture, or vascular repair), necessitating indirect control of bleeding by packing or balloon tamponade;
3. Massive visceral edema precluding formal closure of the abdomen or chest.

The first indication is the most problematic. In the absence of precisely defined guidelines, the decision to abruptly terminate an operation relies heavily on surgical judgment. Carrillo et al.[21] used an early blood loss of 4 to 5 L, a core temperature of 34 C, and a pH of 7.25 as indicative of the need to rapidly abort the operation. A similar set of criteria was advocated by Sharp and Locicero.[22] Others[3,19] used the onset of clinical coagulopathy for the same purpose. However, an early decision to bail out before the onset of coagulopathy may significantly improve the outcome.[19,20]

Ideally, the decision to embark on the planned reoperation approach should be made within the first few minutes of the operation, as soon as the magnitude of the visceral damage is assessed. The decision must be based on injury pattern recognition rather than on physiologic endpoints. The combination of a major abdominal vascular injury with hollow or solid visceral damage is such a pattern.[5,21] Injury to the pancreatic head requiring pancreaticoduodenectomy,[21] a high-grade hepatic wound,[19,22,23] retrohepatic caval injury,[19,23] and a ruptured pelvic hematoma[22] or open pelvic fracture[24] are all examples of injury patterns where an early decision to employ temporary bailout measures is appropriate.

TECHNICAL ASPECTS

The two indirect hemostatic techniques currently in use are packing and balloon catheter tamponade. Packing is a time-honored technique that currently is used to treat high-grade liver injuries[1,23,25] and also to effectively control uncontained retroperitoneal,[22,26] pelvic,[22,24] and extremity[27] bleeding. Successful packing of retrohepatic vena caval and hepatic vein injuries also has been reported.[19,23] Balloon catheter tamponade[28–30] with a Foley or a Fogarty catheter temporarily controls bleeding from penetrating injuries to inaccessible sites such as the pelvis, zone III of the neck, and transfixing hepatic wounds.[31]

The abbreviated management of bowel injuries is based on rapid control of spillage while deliberately avoiding formal resection and anastomosis. The bowel is either ligated or stapled on both sides of the injured segment.[4,5,20,21] External tube drainage of duodenal, proximal pancreatic, and common bile duct injuries also are valid options, except in the presence of clinical coagulopathy, when isolation of the injuries by packing is preferred.[3] A rapid pancreaticoduodenectomy technique was described by Eastlick et al.[32] The pancreatic neck, pylorus, and proximal jejunum are stapled and transected, the common bile duct is ligated, and the biliary tract is drained by tube cholecystostomy. Reconstruction is performed at reoperation and does not include pancreaticojejunostomy. Five of seven patients treated via this technique survived.[21] Similar technical principles apply to the urinary tract.[3,20] Injured ureters are ligated, exteriorized, or "stented". A contained perinephric hematoma is not explored, and an actively bleeding kidney is rapidly removed without attempted repair.

In the chest, rapid nonanatomic resection of a bleeding lung segment is performed using a wide linear stapler across the pulmonary parenchyma.[20] Packing of the pleural cavity for diffuse bleeding may occasionally be necessary.

All injured vessels that are not vital to survival are ligated.[3] End-to-end anastomosis or graft interposition are deliberately avoided. Temporary intraluminal shunts are a simple technique for maintaining perfusion to vital vascular beds.[33,34] A carotid shunt or a similar plastic tube, secured in place by silk ligatures, may obviate the need for time-consuming reconstructions in coagulopathic patients. However, current experience with shunting for abdominal vascular injuries is limited.[4,20]

Formal closure of the abdomen or chest is unnecessary and wastes precious time. Rapid closure of the skin only is accomplished either by a running monofilament suture, or by towel clips sequentially applied to the skin edges 1 to 2 cm apart.[4,35] Massive visceral edema and midgut distention often preclude direct skin closure. A variety of techniques have been described to cover and accommodate the increased visceral volume.[4,35–38] Because no comparative studies exist, the choice of temporary closure technique is a matter of individual or institutional preference. In the largest reported experience to date, a soft intravenous fluid bag was used after being unfolded by cutting the seam and sterilized.[4,19,33] The plastic bag can be easily trimmed to the appropriate size and sutured to the skin edges with a running monofilament suture.

THE POSTOPERATIVE PHASE

The major immediate concern after a "damage control" operation is the correction of hypothermia by active external rewarming. Aggressive replacement of clotting factors and platelets, although important, will not restore normal coagulation until the core temperature exceeds 35 C.[39]

Most patients continue to bleed during the first few hours after damage control surgery, thus making bleeding another focus of attention during the early postoperative phase. The clinical dilemma revolves around the distinction between coagulopathic and surgical bleeding, because the latter may necessitate an urgent re-exploration.[40] Even when reoperation is required, every attempt should be made to correct the clotting abnormalities because the results of reoperation in the presence of uncorrected coagulopathy are dismal.[40] This correction is largely empiric because clotting studies (performed at 37 C) are misleading in the presence of hypothermia.[41]

The abdominal compartment syndrome, defined as organ failure secondary to increased intra-abdominal pressure,[39] is the most lethal complication of the planned reoperation approach. Clinical signs include a tensely distended abdomen, decreased cardiac output (from vena caval compression), oliguric renal failure and grossly impaired ventilatory mechanics caused by diaphragmatic elevation.[39,42–46] The perfusion of all abdominal viscera (except the adrenal glands) was shown to be decreased in a canine model.

In severely injured patients, elevated abdominal pressure develops very rapidly in the early postoperative period because of a combination of bleeding, midgut distention, and a noncompliant edematous abdominal wall.[47] The already depleted physiologic reserves of critically wounded patients make this an especially lethal complication unless immediately recognized and treated.[39]

Measurement of intra-abdominal pressure through an indwelling Foley catheter is a useful diagnostic aid[44,48] because a pressure greater than 30 mm Hg is diagnostic. However, most surgeons rely on the clinical signs of tense abdominal distention, oliguria, and excessively high peak inspiratory pressures to indicate the need for prompt decompression.

Although the concept of abdominal decompression is straightforward, its practical application often is not. Excessively high inspiratory pressures may preclude transfer of the patient to the operating room, so that the temporarily closed abdomen may have to be opened in the ICU.[39] Decompression also was associated with profound hypotension and asystole in 4 of 15 patients in one series,[39] presumably because of systemic reperfusion injury. Therefore, it is not surprising that the abdominal compartment syndrome carries a very high mortality in the context of damage control surgery. Of 19 patients reported, only 7 survived (63% mortality).[20,39]

REOPERATION

After stabilization in the ICU, the patient is returned to the operating room for definitive repair of visceral damage. The optimal timing of reoperation is controversial because neither the physiologic end points nor the increased risk of infection with prolonged packing are well defined. In some reported series,[3-5,22,26] reoperation was undertaken within 24 to 48 hours, whereas others[21] prefer to wait 48 to 96 hours.

The timing of reoperation often is dictated by the clinical circumstances. The presence of an ischemic limb[4] or bowel interruption at several sites (creating a closed loop obstruction) mandates early reoperation as soon as coagulopathy and hypothermia are corrected. If temporary abdominal closure includes a bridging technique to accommodate edematous bowel, formal closure may be possible only after mobilization of interstitial fluid (marked by a negative fluid balance) has occurred. This usually takes place after 48 hours or more.[37] Delay also may be warranted in patients who depend on very high positive end-expiratory pressure and high-dose inotropic amine support to maintain oxygen transport. Under these circumstances, each trip to the operating room is a complex and dangerous undertaking that provides ample opportunity for further deterioration.

The planned reoperation begins with a meticulous exploration of the injured cavities, with surgeons bearing in mind that missed injuries are common in these patients.[20] Hollow visceral, vascular, and nontruncal reconstructions are performed next. The cardinal principle is that the removal of packs should always be the last step before closure because it may result in recurrent bleeding and necessitate repacking, the so-called "pack and peek" sequence.[23]

The rationale of the damage control concept certainly applies to the reoperation as well.[20,39] Even though definitive hemostasis and reconstruction are the aims of reoperation, use of bailout techniques is sometimes appropriate. A typical problem is the patient who underwent gastrointestinal interruption and abdominal closure using synthetic coverage. The need to re-establish gastrointestinal continuity may dictate reoperation while formal abdominal closure without tension is still impossible. Under these circumstances, the synthetic cover can be trimmed (if redundant) and reused in anticipation of formal closure at a later date.

Urgent forced reoperation to control ongoing bleeding or to decompress the abdomen carries a mortality rate as high as 70%.[4,20,39,40] Many of these patients continue to bleed after the primary operation, making the decision to reoperate particularly difficult. Because this decision often is based on operative findings (i.e., the technical feasibility of improving hemostasis), it should always be made by the surgeon who performed the primary operation.[40]

The conduct of an urgent re-exploration differs from that of a planned reoperation. Bearing in mind the very high mortality of the situation, attention is immediately focused on obtaining hemostasis or abdominal decompression by the quickest means followed by rapid return of the patient to the ICU. Incomplete hemostasis was the most common cause of bleeding leading to urgent re-exploration in 11 of 23 patients in one series, although missed injuries (4 patients) and iatrogenic trauma (4 patients) also were contributing factors.[40]

LOGISTIC ASPECTS

Planned reoperation for severe trauma is a highly resource competitive endeavor. Although the magnitude of the logistic effort involved has never been directly addressed in the literature, it becomes apparent on review of several detailed series.[4,5,19,39] One prominent example is the massive transfusion requirements with a mean of 72 units of blood products per patient in the largest series published to date.[4]

Survivors of the primary damage control operation often undergo more than one reoperation. A mean of 1.7 reoperations per patient were required for definitive repair or management of associated injuries and complications in a series of 124 patients.[20]

The logistic profile of this surgical strategy consists of a rapid primary operation followed by 8 to 24 hours of very intensive resuscitation and thereafter, a prolonged stay in the ICU, with repeated trips to the operating room. One of the most critical concerns in these unstable patients is transfer. A well-orchestrated team effort is required to safely and rapidly transfer these patients to and from the operating room, where any delay immediately translates into additional loss of body heat and aggravates the physiologic insult.

All these logistic considerations result in the investment of vast hospital resources in a small group of severely injured patients who carry very high mortality rates.[6] Thus, exclusion criteria to define patients who are considered unsalvagable should be an important aspect of the strategy. This issue was addressed in one series[39] in which patients with massive head injury, patients with prehospital cardiac arrest from blunt trauma, and patients older than 70 years of age were deemed unsalvageable and did not undergo reoperation.

THE CURRENT CLINICAL EXPERIENCE

Use of planned reoperation for multivisceral or complex trauma was reported in three large series consisting of more than 100 patients each,[4,20,39] several smaller series,[3,5,19,21,22,26,36–38] and case reports.[27,32,49] Reports limited to packing of hepatic injuries were excluded from this analysis.

The reported mortality is high, ranging between 46% and 66% in the three largest series.[4,20,39] This high mortality reflects the often desperate clinical situation in which this surgical approach is employed. Most deaths occur either during or shortly after the primary operation, in patients who sustain an irreversible physiologic insult or who continue to bleed massively.[4,19,20,39] The mortality rate among patients who survive to reoperation still is 26% to 33%, but it is significantly lower in patients undergoing planned rather than forced reoperation.[4,20,39] Obtaining effective hemostasis either during or immediately after the primary abbreviated procedure clearly is the major determinant of a successful outcome.[4,39]

Major complications are very prevalent among patients who survive a staged repair.[3,4,19,20,39] However, this increased morbidity is acceptable if it comes in exchange for improved survival. A detailed analysis of complications and their impact on survival was reported by Burch et al.[4] The most lethal complication was multiple-organ failure (only 3 of 25 patients survived), and this finding is corroborated by others.[20,39] The reported incidence of intra-abdominal abscess[3,4,21,22,26] ranges between 12% and 67% and is much higher than that usually seen in patients with abdominal trauma. This and other septic complications in the chest and abdomen must be distinguished from the systemic inflammatory response (sepsis syndrome), which is a marker of poor prognosis.[4,39]

Comparison between the planned reoperation approach and traditional definitive surgery are few and retrospective. Stone et al.[3] compared both approaches in 31 coagulopathic patients. Whereas only 1 of 14 patients survived a definitive repair, 11 of 17 patients (65%) survived with planned reoperation. Another retrospective comparison of patients with penetrating abdominal trauma[5] demonstrated a significantly better survival in critically wounded patients with a major vascular injury and two or more visceral injuries when planned reoperation was employed. Because this approach represents the only practical bailout solution to very difficult (often desperate) operative situations, prospective, randomized clinical studies comparing definitive and staged repairs are not likely to be conducted.

CONCLUSION

Planned reoperation represents a critical stage in the modern extended concept of trauma resuscitation. Resuscitation of the patient with multivisceral injuries begins in the field and ends only on reaching a supported steady state in the ICU. Throughout this process, maintenance of oxygen transport and avoidance of irreversible physiologic insults are the primary objectives. When viewed in this context, it is clear why attempts to perform formal complex resections and reconstructions have dismal results[50,51]; simple temporary control measures that avoid crossing the patient's physiologic limits are more likely to succeed.

The technical simplicity of the primary abbreviated damage control operation makes it an ideal solution for surgeons who encounter major trauma only occasionally, for small hospitals in rural areas, and for field surgery in military conflicts.[6] However, the postoperative stabilization and reoperation are best undertaken at a trauma center, where specific experience and resources exist to address the complex surgical problems and unusual logistic effort involved.

Several crucial questions remain unanswered, such as the optimal timing of reoperation and the means of reducing late septic morbidity. There currently is no animal model in which answers to these questions can be sought. Despite these limitations, the planned reoperation approach is a rapidly evolving field. The emphasis on physiologic concerns rather than anatomic repair is changing existing algorithms and provides an effective answer to the challenge of multivisceral and complex trauma.

References

1. Svoboda JA, Peter ET, Dang CV, et al. Severe liver trauma in the face of coagulopathy: A case for temporary packing and early reexploration. Am J Surg 1982; 144:717–721.
2. Feliciano DV, Mattox KL, Burch JM. Packing for control of hepatic hemorrhage. J Trauma 1986; 26:738–743.
3. Stone HH, Strom PR, Mullins RJ. Management of the major coagulopathy with onset during laparotomy. Ann Surg 1983; 197: 532–535.
4. Burch JM, Ortiz VB, Richardson RJ, et al. Abbreviated laparotomy and planned reoperation for critically injured patients. Ann Surg 1992; 215:476–482.
5. Rotondo MF, Schwab CW, McGonigal MD, et al. "Damage control": an approach for improved survival in exsanguinating penetrating abdominal injury. J Trauma 1993; 35:375–382.
6. Hirshberg A, Mattox KL. "Damage control" in trauma surgery. Br J Surg 1993; 80:1501–1502.

7. Rutledge R, Sheldon GF, Collins ML. Massive transfusion. Crit Care Clin 1986; 2:791–805.
8. Collins JA. Recent developments in the area of massive transfusion. World J Surg 1987; 11:75–81.
9. Jurkovich GJ, Greiser WB, Luterman A, et al. Hypothermia in trauma victims: an ominous predictor of survival. J Trauma 1987; 27:1019–1024.
10. Gregory JS, Flancbaum L, Townsend MC, et al. Incidence and timing of hypothermia in trauma patients undergoing operations. J Trauma 1991; 31:795–798.
11. Bernabei AF, Levison MA, Bender JS. The effects of hypothermia and injury on severity of blood loss during trauma laparotomy. J Trauma 1992; 33:835–839.
12. Patt A, McCroskey BL, Moore E. Hypothermia-induced coagulopathies in trauma. Surg Clin North Am 1988; 68:775–785.
13. Hewson JR, Neame PB, Kumar N, et al. Coagulopathy related to dilution and hypotension during massive transfusion. Crit Care Med 1985; 13:387–391.
14. Wildenthal K, Mierzwiak DS, Myers RW, et al. Effects of acute lactic acidosis on left ventricular performance. Am J Physiol 1968; 214:1352–1359.
15. Yudkin J, Cohen RD, Slack B. The haemodynamic effects of metabolic acidosis in the rat. Clin Sci Mol Med 1976; 50:177–184.
16. Ferrara A, MacArthur JD, Wright HK, et al. Hypothermia and acidosis worsen coagulopathy in the patient requiring massive transfusion. Am J Surg 1990; 160:515–518.
17. Falcone RE, Santanello SA, Schulz MA, et al. Correlation of metabolic acidosis with outcome following injury and its value as a scoring tool. World J Surg 1993; 17:575–597.
18. Imm A, Carlson RW. Fluid resuscitation in circulatory shock. Crit Care Clin 1993;9:313–333.
19. Cuè Jl, Cryer HG, Miller FB, et al. Packing and planned reexploration for hepatic and retroperitoneal hemorrhage: critical refinements of a useful technique. J Trauma 1990; 30:1007–1011.
20. Hirshberg A, Wall MJ, Jr., Mattox KL. Planned reoperation for trauma: a two year experience with 124 consecutive patients. J Trauma (in press).
21. Carrillo C, Fogler RJ, Shaftan GW. Delayed gastrointestinal reconstruction following massive abdominal trauma. J Trauma 1993; 34:233–235.
22. Sharp KW, Locicero RJ. Abdominal packing for surgically uncontrollable hemorrhage. Ann Surg 1992; 215:467–474.
23. Beal SL. Fatal hepatic hemorrhage. An unresolved problem in the management of complex liver injuries. J Trauma 1990; 30:163–169.
24. Sinnott R, Rhodes M, Brader A. Open pelvic fracture: an injury for trauma centers. Am J Surg 1992; 163:283–287.
25. Feliciano DV, Pachter HL. Hepatic trauma revisited. Curr Probl Surg 1989; 26:453–524.
26. Talbert S, Trooskin SZ, Scalea T, et al. Packing and reexploration for patients with nonhepatic injuries. J Trauma 1992; 33:121–124.
27. Scalea TM, Mann R, Austin R, et al. Staged procedures for exsanguinating lower extremity trauma: an extension of a technique - case report. J Trauma 1994; 36:291–293.
28. Smiley K, Perry MO. Balloon catheter tamponade of major vascular wounds. Am J Surg 1971; 121:326–327.
29. Owen DR, Hodgson PE. Control of hemorrhage following missile wound to the pelvis. J Trauma 1980; 20:906–908.
30. Feliciano DV, Burch JM, Mattox KL, et al. Balloon catheter tamponade in cardiovascular wounds. Am J Surg 1990; 160:583–587.
31. Poggetti RS, Moore EE, Moore FA, et al. Balloon tamponade for bilobar transfixing hepatic gunshot wounds. J Trauma 1992; 33: 694–697.
32. Eastlick L, Fogler RJ, Shaftan GW. Pancreaticoduodenectomy for trauma: delayed reconstruction—a case report. J Trauma 1990; 30:503–505.
33. Johansen K. Bandyk D, Thiele B, et al. Temporary intraluminal shunts: resolution of a management dilemma in complex vascular injuries. J Trauma 1982; 22:395–401.
34. Nichols JG, Svoboda JA, Parks SN. Use of temporary intraluminal shunts in selected peripheral arterial injuries. J Trauma 1986; 26: 1094–1096.
35. Feliciano DV, Burch JM. Towel clips, silos, and heroic forms of wound closure. *In* Maull KI, ed. Advances in Trauma and Critical Care. Vol 6. Chicago: Mosby Year Book, 1991, pp 231–250.
36. Aprahamian C, Wittmann DH, Bergstein JM, et al. Temporary abdominal closure (TAC) for planned relaparotomy (Etappenlavage) in trauma. J Trauma 1990; 30:719–723.
37. Smith PC, Tweddell JS, Bessey PQ. Alternative approaches to abdominal wound closure in severely injured patients with massive visceral edema. J Trauma 1992; 32:16–20.
38. Bender JS, Bailey CE, Saxe JM, et al. The technique of visceral packing: recommended management of difficult fascial closure in trauma patients. J Trauma 1994; 36:182–185.
39. Morris JA, Jr., Eddy VA, Blinman TA, et al. The staged celiotomy for trauma: issues in unpacking and reconstruction. Ann Surg 1993; 217:576–586.
40. Hirshberg A, Wall MJ, Jr., Ramchandani MK, et al. Reoperation for bleeding in trauma. Arch Surg 1993; 128:1163–1167.
41. Reed RL, Johnston TD, Hudson JD, et al. The disparity between hypothermic coagulopathy and clotting studies. J Trauma 1992; 33:465–470.
42. Richards WO, Scovill W, Baekhyo S, et al. Acute renal failure associated with increased intra-abdominal pressure. Ann Surg 1983; 197:183–187.
43. Richardson JD, Trinkle JK. Hemodynamic and respiratory alterations with increased intra-abdominal pressure. J Surg Res 1976; 20:401–404.
44. Kron IL, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for abdominal reexploration. Ann Surg 1984; 199:28–30.
45. Bradley SE, Bradley GP. The effect of increased intra-abdominal pressure on renal function in man. J Clin Invest 1947; 26:1010–1022.
46. Kashtan J, Green JF, Parsons EQ, et al. Hemodynamic effects of increased abdominal pressure. J Surg Res 1981; 30:249–255.
47. Caldwell CB, Ricotta JJ. Changes in visceral blood flow with elevated intra-abdominal pressure. J Surg Res 1987; 43:14–20.
48. Iberti TJ, Kelly KM, Gentili DR, et al. A simple technique to accurately determine intra-abdominal pressure. Crit Care Med 1987; 15:1140–1142.
49. Shew GK, Rappaport W. Control of nonhepatic intra-abdominal hemorrhage with temporary packing. Surg Gynecol Obstet 1992; 174:411–413.
50. Cox EF, Flancbaum L, Dauterive AH. Blunt trauma to the liver: analysis of management and mortality in 323 consecutive patients. Ann Surg 1988; 207:126–134.
51. Stone HH, Fabian TC, Satiani B, et al. Experiences in the management of pancreatic trauma. J Trauma 1981; 21:257–261.