developing such strategies is to identify the patterns and characteristics of trauma patients in various regions.

This study has revealed the trauma profile of our region. Road traffic accidents and falls are the major trauma types encountered in our region. A few simple measures such as appropriate intervention and referrals, education of physicians and medical personnel, and increasing public awareness in these types of trauma causes can significantly reduce injury-related burden, suffering, and mortality.

This study has also revealed the importance of keeping medical records of trauma patients. We know that this study would have been much better if we had had all the information pertaining to the trauma patients in their trauma charts. Trauma registry should be taken more seriously in our institution and in our country as well as in other developing countries.

References

1. Ozyurt G, ed. *Yogun bakım*. Bursa: Uludag Universitesi Basımevi; 1992.
2. Landon BA, Driscoll PA, Goodall JD, eds. *An Atlas of Trauma Management: The First Hour.* New York: Parthenon Publishers; 1994.
3. Lewis FR, Mathewson C Jr. Management of the injured patient. In: Way LW, ed. *Current Surgical Diagnosis and Treatment.* 6th ed. Los Altos, Cal: Lange Medical Publications; 1983.
4. Kynerd RE, Rodnet WM, Bullock K. Trauma. In: Rakel RE, ed. *Textbook of Family Practice.* 6th ed. Philadelphia: W.B. Saunders; 2002.
5. Baker CC, Oppenheimer L, Stephens B, Lewis FR, Trunkey DD. Epidemiology of trauma deaths. *Am J Surg* 1980;140:144-50.
6. American College of Surgeons, Committee on Trauma. Triage Decision Scheme in Advanced Trauma Life Support for Doctors. *ATLS Course Manual.* 6th ed. Chicago: American College of Surgeons; 1997, p 24.
7. Ihtiyar E, Unluoglu I, Sahin A, Yilmaz S, Caga T, Karahuseyinoglu E. The evaluation of multi-trauma patients with GCS, TS, AIS at Osmangazi University Faculty of Medicine emergency service: prospective evaluation of 734 patients. *Turkish J Trauma Emergency Surg* 1998;4(3):176-9.
8. Moini M, Rezaishiraz H, Zafarghandi MR. Characteristics and outcome of injured patients treated in urban trauma centers in Iran. *J Trauma* 2000;48:503-7
9. World Health Organization. *The World Health Report 1998: Life in the 21st Century. A Vision for All.* Geneva: World Health Organization; 1998.
10. Parkkari J, Kannus P, Niemi S, et al. Childhood deaths and injuries in Finland in 1971-1995. *Int J Epidemiol* 2000;29:516-23.
11. Watson WL, Ozanne-Smith J. Injury surveillance in Victoria, Australia: developing comprehensive injury incidence estimates. *Accid Anal Prev* 2000;32(2):277-86.
12. Lerner EB, Jehle DV, Billittier AJ 4th, Moscati RM, Connery CM, Stiller G. The influence of demographic factors on seatbelt use by adults injured in motor vehicle crashes. *Accid Anal Prev* 2001;33(5):659-62.
13. Scuffham P, Alsop J, Cryer C, Langley JD. Head injuries to bicyclists and the New Zealand bicycle helmet law. *Accid Anal Prev* 2000;32(4):565-73.
14. Kobusingye CO, Lett RR. Hospital-based trauma registries in Uganda. *J Trauma* 2000;48:498-502.
15. Bergman AB, Rivara FP. Sweden's experience in reducing childhood injuries. *Pediatrics* 1991;88:69-74.
16. Gaillard M, Herve C. Emergency medical care and severe home accidents in children. Study of 630 cases over 5 years. Their significance in traumatic accidents [in French]. *Ann Pediatr (Paris)* 1991;38(5):311-7.
17. Mosenthal AC, Livingston DH, Elcavage J, Merritt S, Stucker S. Falls: epidemiology and strategies for prevention. *J Trauma* 1995;38:753-6.
18. Hijar M, Chu LD, Kraus JF. Cross-national comparison of injury mortality: Los Angeles County, California and Mexico City, Mexico. *Int J Epidemiol* 2000;29:715-21.
19. del Ciampo LA, Ricco RG, De Almeida CA, Mucillo G. Incidence of childhood accidents determined in a study based on home surveys. *Ann Trop Paediatr* 2001;21(3):239-43.
20. Tepas JJ 3rd, Ramenofsky ML, Barlow B, et al. National Pediatric Trauma Registry. *J Pediatr Surg* 1989;24:156-8.
21. Lallier M, Bouchard S, St-Vil D, Dupont J, Tucci M. Falls from heights among children: a retrospective review. *J Pediatr Surg* 1999;34:1060-3.
22. Benoit R, Watts DD, Dwyer K, Kaufmann C, Fakhry S. Windows 99: a source of suburban pediatric trauma. *J Trauma* 2000;49:477-82.
23. Adesunkanmi AR, Oginni LM, Oyelami OA, Badru OS. Road traffic accidents to African children: assessment of severity using the Injury Severity Score (ISS). *Injury* 2000;31:225-8.
24. Katkıci U, Orsal M, Ozkok SM. Trafik kazası ile yaralanarak Cumhuriyet Universitesi Tıp Fakültesi Hastanesi'ne başvuran adli olgular. *C. U. Tıp Fak Derg* 1993;15(4):221-4.
25. Orsal M, Katkıcı U, Ozkok SM. C.Ü.T.F. Hastanesi'ne başvuran trafik dışı kaza sonucu yaralanmaların adli tıp yönü. *C. U. Tıp Fak Derg* 1993;15(2):105-9.
26. McDonald A, Duncan ND, Mitchell DI, Fletcher PR. Trauma aetiology and cost in the accident and emergency unit of the University Hospital of the West Indies. *West Indian Med J* 1999;48:141-2.

Uncontrolled Hemorrhage in a Patient with Pelvic Fracture: A Case Report

Magnus Södergren, MD, and Robert G. Hahn, MD, PhD
Department of Anesthesiology & Intensive Care, South Hospital, S-118 83 Stockholm, Sweden
robert.hahn@sodersjukhuset.se

Learning Objectives: 1) To be aware of the potential influence of episodic rebleeding on hemodynamic stability in patients with traumatic pelvic fracture, and 2) to be aware that animal experiments demonstrate a better outcome if fluid resuscitation does not completely restore the arterial blood pressure in the presence of major vascular injury.

Neither author has any conflicts of interest to disclose.

Abstract
A 73-year-old woman arrived at hospital with multitrauma, including a nonoperative pelvic fracture. Fluid resuscitation aimed at restoring normal hemodynamics was started, but hypotensive events developed on four occasions when intensive fluid resuscitation had raised the systolic pressure to just above 100 mm Hg. The blood volume expansion resulting from transfusion of erythrocytes and plasma and the infusion of clear fluid corresponded to six times her blood volume during the first 21 hours in hospital. The clinical course suggests the presence of episodic rebleeding from the pelvic fracture.

Pelvic fractures are often accompanied by major bleeding, which is potentially life-threatening.[1,2] Current recommendations for treatment are to stabilize the bony pelvis (if anatomically possible) while restoring normal tissue perfusion through administration of fluids and blood products.[3,4] In recent years, however, animal experiments have raised concerns as to whether such fluid treatment may aggravate ongoing acute bleeding or lead to rebleeding from surgically inaccessible vessels.[5,6] While most orthopaedic injuries will stop bleeding spontaneously, allowing for completion of resuscitation, venous bleeding from the posterior presacral plexus is a notable exception. We report on a patient with pelvic fracture whose clinical course showed a striking resemblance to the "transient responders" seen in animal studies of aggressive volume resuscitation during acute hemorrhage.

Case Report

A 73-year-old woman was found below a third-floor window after an attempted suicidal jump. At the arrival of the ambulance she was conscious, not in pain, but confused. Her left arm had two open fractures (wrist and elbow) and was severely distorted. The left thigh was bruised. The blood pressure was unrecordable and the heart rate high. Breathing was not affected at first, but she became slightly dyspneic during transport to hospital, and was treated with 10 liters of O_2 100% delivered by a nonrebreather face mask. On arrival in the emergency department (ED), her systolic blood pressure was 45 mm Hg (external cuff); SaO_2, 75%; body temperature, 37°C; and capillary Hb, 12.1 g/dL. Gross examination revealed a malpositioned left shoulder, an open wrist fracture of the radius and ulna, crepitational fracture of costa IV, and an unstable fracture of the pelvis involving the sacrum and the left acetabulum, ala of ilium, and inferior ramus. Past medical history was not available.

Fluid resuscitation began with 1 liter of Ringer's acetate solution and, within 10 minutes, the systolic pressure rose to 95 mm Hg. During the subsequent 30 minutes, another 1 liter of Ringer's solution and 2 liters of a Ringer's-dextran mixture were infused. After tracheal intubation under ketamine anesthesia, the patient was transferred to the operating room (OR), where the pelvic fracture was stabilized with a Hoffman instrument. Bilateral pleural drainage was also applied, without significant return of blood or air from either chest cavity. Ultrasound showed minimal fluid in the abdominal cavity. Hemodynamic instability persisted and was treated with ongoing fluid resuscitation; angiographic embolization was not an available option at that time. During the operation, the patient developed ventricular fibrillation, which required four defibrillations.

Four hours later, the patient was transferred to the intensive care unit (ICU) where intravascular catheters for monitoring the arterial and central venous pressures were inserted. Computed tomography (CT) on day 2 showed anterior displacement of the spleen and left kidney owing to massive retroperiotoneal hematoma. There was no damage to the internal organs, including the aorta. Peripheral edema was particularly pronounced around the shoulders. On day 5, the fracture of the humerus was fixed and the dislocated shoulder was repositioned. The patient was weaned off the ventilator after a long period in the ICU, but eventually died 4 months after the injury, owing to respiratory failure.

After the initial fluid resuscitation, the patient had four markedly hypotensive events despite liberal volume support, which, during the first 21 hours in hospital, comprised 10.8 liters of erythrocytes, 7.2 liters of stored plasma, 3.2 liters of fresh-frozen plasma, 8 liters of of Ringer's solution, 2 liters of of Ringer-dextran, 1 liter of glucose 2.5%, 600 mL of bicarbonate buffer, and 350 mL of thrombocytes (Fig. 1). The total fluid losses in surgical drains and external bleeding amounted to 3.6 liters.

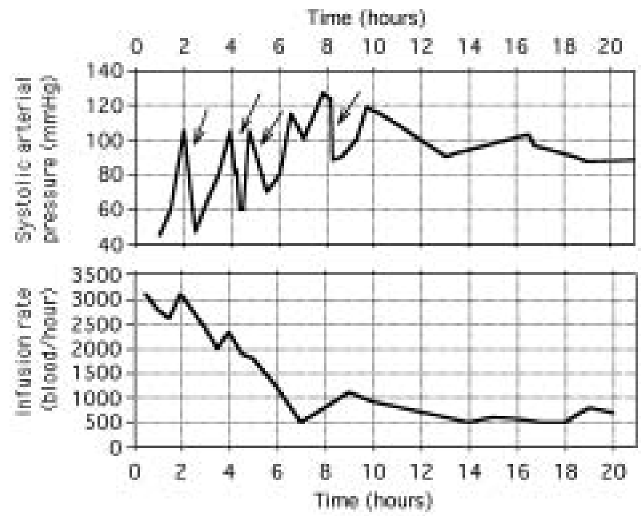

Figure 1. Systolic arterial pressure (top) and the rate of volume support (bottom) in a multitraumatized woman with pelvic fracture. To obtain the latter parameter, we converted each type of fluid into its corresponding blood volume effect by using the following weights: erythrocytes and plasma = 1, Ringer-dextran = 0.5, and Ringer's solution = 0.3. The result is expressed as the sum of the volume effect of all fluids given per hour. Arrows indicate sudden development of hypotension after intensified fluid therapy.

Discussion

The survival of patients with a pelvic fracture is greatly affected by their hemodynamic stability on arrival in hospital.[7] While there is a general consensus that an aggressive multidisciplinary approach to hemorrhage control is mandatory,[1] the immediate goals of fluid resuscitation are controversial. Restoration of intravascular volume and composition is clearly important in the long term to sustain tissue perfusion, yet clinical trials have not consistently documented improved survival with prehospital volume support in patients with uncontrolled hemorrhage.[8,9] A large body of animal research suggests that early aggressive volume support, in the presence of ongoing hemorrhage, possibly increases mortality through rebleeding, dilution of clotting factors and the hematocrit, and induced hypothermia.[5,6,10-14]

Stern et al[6] resuscitated pigs with a 4-mm laceration in the lower aorta to mean arterial pressures of 40, 60, and 80 mm Hg and found that attempts to restore a normal blood pressure markedly increased mortality. Even when the arterial pressure remained low, survival could be improved by limiting resuscitation with crystalloid[5] and hypertonic[10] fluid to between 30% and 50% of the recommended amount, and by infusing the fluid at a lower rate.[10,11] The benefits of a limited fluid program in pigs received support from experiments in rats.[12-14] The increased blood flow rate and/or increased transmural pressure following fluid resuscitation apparently disturbs immature blood clots in the walls of bleeding vessels[6,14,15] and the subsequent rebleeding is characterized by a sudden drop in flow rate after the initial rise.[10,16] These experiences from animal experiments are similar to the clinical course of the patient reported here, to whom fluid was given vigorously. The volume support administered during the first 21 hours in hospital was sufficient to expand the blood volume to 30 liters, six times her normal blood volume. The measured blood loss was quite small. The clinical course, together with the CT scan, suggests that "third-space" loss of fluid was substantial, while blood gradually accumulated in the extravascular retroperitoneal space. The blood pressure curve shows that the patient repeatedly became hypotensive despite (or perhaps because of) the rapid administration of fluids. These data are consistent with repeated rebleeding from surgically inaccessible wounds.

Figure 1 illustrates the intensity of the volume support and the systolic pressure during the day of admission. Apart from the short period of cardiac arrest, four events with a rapid fall in blood pressure occurred when enough volume support was given to raise the systolic pressure to 100 mm Hg. These events were most probably due to relative hypovolemia, which is surprising as the rate of infusion of fluid was high, accessible sources of bleeding had been arrested, and a Hoffman instrument had been applied early on to fix the pelvic fracture. Stabilization using an external frame, a pelvic binder, or percutaneous fixation is often employed to stop bleeding from a pelvic fracture, although in a lateral compression injury such as this patient's, it may not be of great benefit. Early stabilization does allow for improved nursing care and earlier patient mobility. Damage to major arteries may require a direct surgical approach[2] or angiographically directed embolization; these options could have been helpful in the present case, but were not available in the hospital at the time.[17] The patient eventually stabilized at a systolic pressure of 90 mm Hg and was allowed to remain at this level without further fluid administration.

Vigorous fluid therapy, resulting in a rise of arterial pressure followed by a marked drop—the classic "transient responder" pattern—causes the disruption of immature blood clots. Such disturbances result in rebleeding, which increases the total blood loss and prolongs the period of hemodynamic instability. Hemodilution-induced changes in hemostasis and blood viscosity may contribute to such rebleeding, but mechanical forces associated with a rise in the blood flow rate and/or arterial pressure associated with vigorous fluid resuscitation constitute the most important mechanism.[5,10-15,18]

Hypoperfusion leading to organ system failure is a significant concern in any patient, particularly an elderly one, and older age is a relative contraindication to deliberate hypotensive resuscitation. In this case of difficult-to-control pelvic bleeding, however, where there were no surgical or angiographic options available, targeting a lower than normal blood pressure early in the resuscitation might have prevented significant rebleeding and avoided the necessity for such massive resuscitation. Hypotensive resuscitation is already being practiced by many clinicians in patients with gastric bleeding or a ruptured aorta, but its application in unselected trauma patients is controversial.[19,20] The advent of systemic procoagulant agents (e.g., recombinant human factor VIIa) may someday contribute to the care of patients such as the one reported here, but at present this therapy is expensive and unproven.[21]

Summary

In summary, this case presents an interesting and all too common clinical dilemma, which invites a solution in contradiction to the usual standard for resuscitation from shock. Tolerance of a lower-than-normal blood pressure and deliberate slowing of fluid administration should be considered in any case of ongoing hemorrhage in which an immediate anatomic solution is not available. In all patients, but particularly the elderly, close clinical observation and moment-to-moment titration of therapies is required. Early and frequent assessment of arterial blood gases and serum lactate level will provide evidence of the depth of shock, and can help the clinician to achieve a balance between adequate perfusion and the risk of rebleeding. New monitoring technology currently under clinical study may make this process more rapid and reliable in the near future.[22,23] While individual resuscitation strategy will always depend on details of the patient and presentation, deliberate hypotension is one option the clinician should consider when confronted by persistent or recurrent hemorrhage that is not anatomically correctable.

References

1. Poole GV, Ward EF, Muakkassa FF, Hsu HS, Griswold JA, Rhodes RS. Pelvic fracture from major blunt trauma. Outcome is determined by associated injuries. *Ann Surg* 1991;213:532-9.
2. Agnew AG. Hemodynamically unstable pelvic fractures. *Orthop Clin North Am* 1994;25:715-21.
3. Alonso JE, Lee J, Burgess AR, Browner BD. The management of complex orthopedic injuries. *Surg Clin North Am* 1996;76:879-903.
4. Bircher MD. Indications and techniques of external fixation of the injured pelvis. *Injury* 1996;27(suppl 2): B3-19.
5. Riddez L, Johnson L, Hahn RG. Central and regional hemodynamics during crystalloid fluid therapy after uncontrolled intra-abdominal bleeding. *J Trauma* 1998;44:433-9.
6. Stern SA, Dronen SC, Birrer P, Wang X. Effect of blood pressure on hemorrhage volume and survival in a near-fatal hemorrhage model incorporating a vascular injury. *Ann Emerg Med* 1993;22:155-63.
7. Mucha P Jr, Farnell M. Analysis of pelvic fracture management. *J Trauma* 1984;24:379-86.
8. Bickell WH, Wall MJ Jr, Pepe PE, et al. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. *New Engl J Med* 1994;331:1105-9.
9. Kaweski SM, Sise MJ, Virgilio RW. The effect of prehospital fluids on survival in trauma patients. *J Trauma* 1990;30:1215-8.
10. Bickell WH, Bruttig SP, Wade CE. Hemodynamic response to abdominal aortotomy in the anesthetized swine. *Circ Shock* 1989;28:321-32.
11. Riddez L, Drobin D, Sjöstrand F, Svensen C, Hahn RG. Lower dose of hypertonic saline dextran reduces the risk of lethal rebleeding in uncontrolled hemorrhage. *Shock* 2002;17:377-82.
12. Capone AC, Safar P, Stezoski W, Tisherman S, Peitzman AB. Improved outcome with fluid restriction in treatment of uncontrolled hemorrhagic shock. *J Am Coll Surg* 1995;180:49-56.
13. Krausz MM, Landau EH, Klin B, Gross D. Hypertonic saline treatment of uncontrolled hemorrhagic shock at different periods from bleeding. *Arch Surg* 1992;127:93-6.
14. Wangensteen SL, Eddy DM, Ludewig RM. The hydrodynamics of arterial hemorrhage. *Surgery* 1968;64:912-21.
15. Shaftan GW, Chiu C-J, Dennis C, et al. Fundamentals of physiologic control of arterial hemorrhage. *Surgery* 1965;58:851-6.
16. Riddez L, Hahn RG, Suneson A, Hjelmqvist H. Central and regional hemodynamics during uncontrolled bleeding using hypertonic saline dextran for resuscitation. *Shock* 1998;10:176-81.
17. Evers BM, Cryer HM, Miller FB. Pelvic fracture hemorrhage. Priorities in management. *Arch Surg* 1989;124:422-4.
18. Soucy DM, Rudé M, Hsia WC, Hagedorn FN, Illner H, Shires GT. The effects of varying fluid volume and rate of resuscitation during uncontrolled hemorrhage. *J Trauma* 1999;46:209-15.
19. Dutton RP, Mackenzie CF, Scalea TM. Hypotensive resuscitation during active hemorrhage: impact on in-hospital mortality. *J Trauma* 2002;52:1141-6.
20. Dutton RP. Low-pressure resuscitation from hemorrhagic shock. *Int Anaesthesiol Clin* 2002;40:19-30.
21. Martinowitz U, Kenet G, Segal E, et al. Recombinant activated factor VII for adjunctive hemorrhage control in trauma. *J Trauma* 2001;51:431-9.
22. Weil MH, Nadagawa Y, Tang W, et al. Sublingual capnometry: a new noninvasive measurement for diagnosis and quantification of severity of circulatory shock. *Crit Care Med* 1999;27:1225-9.
23. Crookes BA, Cohn SM, Burton EA, Nelson J, Proctor KG. Noninvasive muscle oxygenation to guide fluid resuscitation after traumatic shock. *Surgery* 2004;135:662-70.

THE INTERNATIONAL TRAUMA ANESTHESIA & CRITICAL CARE SOCIETY

(International Trauma Care)

POB 2797, Pasco, WA. USA 99302
PH (509) 547-7065, (800) 222-6927
FAX (509) 547-1265 email info@nwas.com
Internet http://www.nwas.com/itaccs

PRESENTS

TRAUMA: THE TEAM APPROACH TO THE CLINICAL CHALLENGE

The Latest Information on Current Care of the Trauma Victim

While each program title is the same, the content varies.
Please request the individual location brochure for complete details.

2005

March 20-27
Eastern Caribbean Spring Bread Cruise

April 7-10
Miami, Florida

April 30-May 3
Hilton Head Island, South Carolina

May 21-28
Tahiti & French Polynesian Cruise

June 19-24
Grand Tetons National Park, Wyoming

June 20-July 2
London>Copenhagen North Cape/Arctic Circle Cruise

July 3-10
Alaska Discovery Cruise

August 14-19
Grand Canyon National Park, Arizona

September 6-9
Las Vegas, Nevada (The New Wynn Hotel)

September 12-16
Yosemite National Park, California

October 2-5
Atlantis, Paradise Island, Bahamas

October 2-12
Rome to Barcelona Mediterranean Cruise

October 8-15
Bonaire, Dutch Antilles

November 10-13
Key West, Florida

December 3-10
St. Thomas, Virgin Islands

FOR ADDITIONAL INFORMATION VISIT OUR WEB SITE

www.nwas.com/itaccs

Call (inside U.S.) (800) 222-6927 or (509) 547-7065 FAX 509 547-1265

email info@nwas.com