

Resuscitative Fluids in Penetrating Trauma: When and When not to Give?

Khaqan Jahangir Janjua (Trauma Fellow, Royal Perth Hospital, WA, Australia.)

Conventional treatment of the shocked trauma patient involves intravenous fluid administration to bring the blood pressure back to “normal”. This management principle is outlined in the Advanced Trauma Life Support (ATLS) instructor manual¹. The practice of normotensive resuscitation is based on animal studies where hypovolemia was produced by controlled exsanguination techniques to a predetermined end point of volume or blood pressure^{2,3}. Aggressive volume resuscitation was necessary for survival of these animals^{2,3}. The rationale for normotensive resuscitation has been to maintain tissue perfusion and vital organ function while diagnostic and therapeutic procedures are being performed.

Until recently the dominant controversies in fluid resuscitation have focused on the appropriateness of either crystalloids or colloids. Traditionally for every 1 ml of estimated blood loss, 3 ml of crystalloid has been recommended if complete fluid resuscitation is to be achieved. Recently, hypertonic saline and dextran have been used to expand intravascular volume and reduce the total volume of fluid needed for resuscitation⁴. Enthusiasm about this modality of fluid resuscitation has been tempered by concern over potential adverse metabolic, haematologic and allergic effects.

The universal principle of early normotensive resuscitation has been unsuccessfully challenged in the past on the basis of animal studies and experience of treatment of wounded soldiers in World War I and II. Cannon in a study of wounded soldiers in 1918 observed and concluded “haemorrhage in the case of shock may not have occurred to a marked degree because blood pressure has been too low and flow too scant to overcome the obstacle offered by a clot. If the pressure is raised before the Surgeon is ready to check any bleeding that may take place, blood that is still needed may be lost. The same principle of surgical haemostasis before fluid resuscitation was practised by US army in World War II⁶

Subsequently this concept of hypotensive resuscitation was supported by a variety of animal studies where it has been demonstrated that in vascular injury, intravenous fluid administration, by elevating the systemic pressure, hydrostatically disrupts the thrombus leading to increased haemorrhage and decreasing survival^{7,8}. In addition, intravenous infusions of crystalloid may promote haemorrhage by diluting coagulation factors and by lowering the blood viscosity, hence decreasing the resistance to flow around an incomplete thrombus⁹. The concept of surgical correction of haemorrhage before fluid resuscitation is further supported by the fact that fluid resuscitation is often delayed in patients with ruptured abdominal aortic aneurysm until surgical exploration is performed¹⁰.

Despite this evidence, normotensive resuscitation has retained its pre-eminence until recently when Bickell et al tested the hypothesis that fluid resuscitation may be detrimental when given before haemorrhage control in trauma patients^{9,11,12}. In his study on swine in which a 5 mm aortotomy had been performed to simulate uncontrolled bleeding, the animals who were not resuscitated with fluid all survived. At autopsy, each animal was found to have a large firm extraluminal firm thrombus tamponading the aortotomy. Animals that were resuscitated with a hypertonic solution of saline and dextran had a substantial rate of mortality¹. The greatest challenge to conventional fluid therapy was provided by Bickell's 1994 prospective controlled randomised study on 598 adult trauma patients with systolic blood pressure <90 mmHg¹². In this study patients with penetrating injuries to the torso who were treated at an Urban Level I Trauma Centre (Ben Taub General Hospital in Houston), were divided into two groups. Group I received immediate fluid resuscitation and group II were allocated to delayed resuscitation until they reached the operating theatre. Among the delayed resuscitation group of 289

patients, 203 (70%) survived as compared with 193 of the 309 patients (62%) who received immediate resuscitation (p 0.04). When the analysis was limited to patients with Injury Severity Scores of 25 or higher, the findings were even more striking³; the survival rates were 48% in the immediate—resuscitation group and 61% in the delayed- resuscitation group (P<0.01). There was a trend toward increased intra operative blood loss (pM.I I) and a higher rate of post-operative complications (p=0.08) in the immediate resuscitation group. Moreover the duration of hospitalisation was also shorter in the delayed resuscitation group. This study suggested that for hypotensive patients with penetrating torso injuries in an organised urban environment, delay of aggressive fluid resuscitation until operative intervention improves the outcome. In recent animal experiments on rats with standardised liver injury, it was demonstrated that large volume isotonic crystalloid resuscitation increased bleeding and transiently expanded intra vascular volume when compared with no resuscitation or small volume isotonic resuscitation. Mortality in animals resuscitated with conventional large volume isotonic crystalloid was not increased compared with the mortality of animals given no resuscitation fluid. Hypertonic saline resuscitation in the same setting produced a sustained increase in blood pressure, maintained intra vascular volume in spite of increased intra peritoneal bleeding. Survival times and mortality rates were significantly lower in hypertonic saline animals (10%) than in no resuscitation animals (50%) or small volume resuscitation (47%) animals¹⁴.

Life results of Bickell's studies should be thought provoking but have some deficiencies. The situation is yet not clear at the extremes of age, with long transit times and patients with pre-existing illnesses. Moreover the study covered only penetrating injuries to the torso, so the findings may not be applicable to blunt trauma or injuries at other sites. Further prospective trials are needed to look into the effects of delayed or immediate fluid resuscitation in relation to specific organs injured, the number of organs injured, severity of injury, different age groups and to find out the outcomes of different intervals between injury and the start of fluid resuscitation.

The concept of hypotensive resuscitation is particularly relevant to the pre—hospital treatment of penetrating trauma or uncontrolled haemorrhage. A study by O'Gorman and colleagues from the University of California in 1989 analysed the time taken for establishment of an intravenous cannula versus the time taken for transport to hospital⁵. They concluded that seriously injured hypovolemic patients should not have resuscitative effects at the scene but should be subjected to immediate evacuation. En route intravenous access in a moving ambulance has been shown to be as successful as when attempted at the scene and under these circumstances there is less delay in arriving at the hospital¹⁶. A delay in patient transport to major trauma centres may result in an increased morbidity and mortality¹⁷. In severely injured patients, it is estimated that for every 10 minutes of delay in the definitive treatment, survival is reduced by 10%. In a recent study, Demetriades and colleagues showed that patients with severe trauma transported by private means have better survival than those transported via the Emergency Medical Services (EMS) system¹⁹. They suggested two plausible explanations. The first one is the time factor, assuming that victims transported by private means may have a much shorter prehospital time than those transported by EMS ambulances. The second plausible explanation for lower survival in EMS group is therapeutic interventions performed during the pre—hospital time. Thus on the basis of Bickell et al and Demetriades studies, patients with penetrating trauma to the torso would do better with shorter pre—hospital timings and minimal interventions including hypotensive resuscitation.

The concept of hypotensive resuscitation may have a role in urban situations where time to definitive care is comparable to that in North American urban trauma services. In rural areas or in situations where prolonged transit time is expected, then resuscitation with colloids and blood should be considered to maintain the perfusion to vital organs.

In conclusion the challenge and present state of debate of hypotensive resuscitation does not relate to the value of fluid resuscitation but rather to the volume, timing and extent of the resuscitation for

patients with penetrating injuries to the torso. Further human studies are needed to determine the safe level of blood pressure maintenance. Hence the question arises, what advice should be given to emergency personnel and paramedics?

In general, for patients with blunt or penetrating torso trauma, immediate fluid resuscitation at this time remains appropriate in situations where there is long transit time to definitive care or if the surgical skills are not available promptly. In an urban situation where patients can receive surgical care expeditiously, a serious consideration should be given to hypotensive resuscitation.

One of the most important messages from all recent articles is that early surgery remains the key to improved outcomes in treating bleeding trauma patients.

References

1. Shock. In: American College of Surgeons, Committee on Trauma. Advanced trauma life support program for physicians; instructor manual. Chicago American College of Surgeons, 1993:75-110.
2. Taverso L, W. Lee WP, Langford MJ. Fluid resuscitation after an otherwise fatal haemorrhage: Parts I and II. *J. Trauma*, 1986;28:169-82.
3. Wiggers CJ. Experimental haemorrhagic shock. In *physiology of Shock*, New York; Commonwealth Fund, 1950. pp. 121-46.
4. Vassar MJ, Holcroft JW. Use of hypertonic-hyperoncotic fluids for resuscitation of trauma patients. *J. Intensive Care Med.*, 1992;7:189-98.
5. Cannon WB, Fraser J, Cowell EM. The preventive treatment of wound shock, *J.A.M.A.*, 1918;70:618-20.
6. *Surgery in World War II, General Surgery*. Office of the Surgeon General, Department of the Army. Washington, D.C., US Government Printing Office, 1952, pp. 6.
7. Milles B, Koucky CJ, Zachmeis HG. Experimental uncontrolled arterial haemorrhage Surgery, 1966;60:851.
8. Shaftan GW, Chin CJ, Dennis C'. et al. Fundamentals of physiologic control of arterial haemorrhage. *Surgery*, 1965;58:851-52.
9. Bickell WH, Bruttig SP, Millnamow GA, et al. The detrimental effects of intravenous crystalloid after aortotomy in swine Surgery, 1991 ;110:529-36.
10. Crawford ES. Ruptured abdominal aortic aneurysm. *J Vasc. Surg.* 1991;13:348-50.
11. Bickell WH, Bruttig SP, Millnamow GA, et al. Use of hypertonic saline/dextran versus lactated Ringer's solution as a resuscitation fluid after uncontrolled aortic haemorrhage in anaesthetised swine *Am Emerg Med*, 1992;21:1077-85.
12. Bickell WH, Wall MJ, Pepe PE. et al. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. *N, Engl, J. Med.*, 1994;331: 1105-85.
13. Bicket WHI, Pepe PE, Mattox KL. Immediate versus delayed fluid resuscitation in patients with trauma *N. Engl. J. Med.* 1995;332: 681-83.
14. Metsuoka T, Hildreth J, Wisner DH. Uncontrolled Haemorrhage from Parenchymal Injury: Is Resuscitation Helpful? *J. Trauma* 1996;40:915-22.
15. O'Gorman M, Trabulsi P, Pilcher DB. Zero-time pre-hospital IV. *J. Trauma*, 1989;29:84-86.
16. Jacobs T.M, Sinclair A, Beiser A, et al. Pre-hospital advanced life support benefits in trauma *J. Trauma*, 1984. 24:8- 13.
17. Smith JP, Bodai BL., Hill AS. et al. pre-hospital stabilisation of critically injured patients: a failed concept. *J. Trauma*, 1985,25:65-70.
18. Brill JC, Geiderman JM. A rationale for scoop and run identifying a subset of time-critical patients. In: Brill, JC Geiderman JM, eds *Topics in Emergency Medicine*. Rockville, Md Aspen Systems Corp; 1981:37-43.

19. Demetrios D, Linda Chain, Edward C, et al. Paramedic vs Private Transportation of Trauma Patients Effect on Outcome Arch. Surg. 1996;131:133-38.