

AN EVALUATION OF THE PNEUMATIC ANTI-SHOCK GARMENT (PASG) IN VARIOUS CLINICAL SETTINGS

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Few devices used in EMS engender such vehement differences of opinion as the pneumatic anti-shock garment (PASG), otherwise known as the military anti-shock trouser (MAST). The device achieved widespread civilian use in EMS following its successful field use on casualties suffering from hemorrhagic shock in the Vietnam conflict. Since that time, the PASG has alternately enjoyed tacit acceptance and endured harsh criticism. While the PASG has become one of the most widely studied devices used in EMS, interpretation of these studies is intensely debated, with wide divergence of opinions.

Initially, the PASG was widely used in the treatment of hemorrhagic shock, where, in the opinion of many, acceptance preceded scientific validation of its clinical efficacy.¹⁻³ This tacit acceptance at one time extended to a variety of shock states, including cardiac arrest,⁴⁻¹¹ anaphylaxis,¹²⁻¹⁶ and paroxysmal supraventricular tachycardia.¹⁷⁻¹⁹ The PASG has even been successfully used to increase the exercise tolerance of paraplegics.²⁰

Recently, two studies reported increased mortality associated with PASG use in victims of penetrating trauma.^{21,22} Another study refutes this claim in demonstrating increased survival with the PASG in victims of hemorrhagic shock whose initial systolic blood pressures are less than 50 mm Hg.²³ Studies of other applications of the PASG, such as in cardiac arrest and spinal shock, failed to reveal any difference in mortality whether or not the device was used.⁴⁻¹¹ The in-

evitable backlash occurred, and the PASG fell into disfavor. Ironically, this widespread condemnation of the PASG took place in the absence of scientific validation that it was detrimental in all situations. Negative outcomes in a limited number of restricted applications have been extrapolated to the broad gamut of clinical situations. Today, the use of the PASG is widely taught to EMS providers,²⁴⁻²⁶ and the ability to use it is required for NREMT-P certification.²⁷ This requirement for certification is in place despite the fact that many systems have sharply restricted PASG use, or have eliminated it altogether.

The following discussion summarizes the physiologic effects of the PASG on hemodynamic parameters, pulmonary function, and intracranial pressure. The effects on intravenous fluid therapy, drug delivery, and field treatment time are then discussed. Effects of the environment on inflation pressures, the use of a wide range of inflation pressures, and their impacts on physiologic parameters are explored next. The uses of the PASG in hemorrhagic shock, low cardiac reserve, the elderly, anaphylactic shock, septic shock, hypovolemic (non-hemorrhagic) shock, CPR, abdominal aortic aneurysm (AAA), spinal shock, hypothermia, cardiac tamponade, paroxysmal supraventricular tachycardia (PSVT), pelvic fracture, and local hemorrhage control are summarized. Finally, the safety of using PASG in pregnancy, evisceration, and lower extremity trauma is assessed.

HEMODYNAMIC EFFECTS

When the PASG is initially applied, venous return increases and results in increased cardiac output. Full inflation of all compartments to 90 mm Hg produces the greatest initial rise in cardiac output, accompanied by increased afterload.²⁸⁻³³ With time, venous return, preload, cardiac output, and stroke volume decrease, an effect that is more pronounced if lower PASG inflation pressures are used.²⁸⁻³³ With aortic blood flow reduced to 25% of normal, mean arterial pressure (MAP) is

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maintained proportionate to the increase in total peripheral resistance (TPR).³⁴ Autotransfusion occurs to a limited degree, and does not contribute to maintaining TPR.^{32,33} Of note, one study suggested that the application and inflation of the PASG resulted in impaired oxygen utilization.³⁰

Sequential inflation and deflation of the PASG confirms these findings. Following inflation, stroke volume and cardiac output transiently increase. However, both stroke volume and cardiac output decrease with time, while heart rate and TPR increase further, resulting in preservation of MAP. Regional blood flow to abdominal organs decreases substantially, while flow to the adrenals, renal cortex, heart, and brain increase.³⁵⁻³⁷ In addition, the increased MAP response can be completely blocked by administering trimethaphan camsylate, suggesting that the increase in TPR is due to activation of vasomotor reflexes and not direct compression.³⁸ Following rapid deflation, stroke volume and cardiac output increase, while heart rate, TPR, and MAP return to baseline levels.³⁵⁻³⁷

The minimum inflation pressure needed to displace blood centrally is 40 mm Hg, however, higher inflation pressures displaced incrementally less volume.³⁹⁻⁴¹ No more than a 5% increase in the central volume was achieved, regardless of inflation pressure or the sequence of compartment inflation.⁴² Sequential inflation of the compartments produced greater increases in stroke volume, end-diastolic volume and cardiac output than did simultaneous inflation, which produced the greatest increase in blood pressure.³⁹⁻⁴¹

It is of interest to note that the Trendelenburg position, which has also been used to maintain MAP, does so by increasing cardiac output and increasing venous return. Compared with the PASG, a greater percentage of volume is displaced centrally, and TPR is not affected. The Trendelenburg position and the PASG equally increase carotid sinus diameter. Since the Trendelenburg position primarily displaces volume centrally, and the PASG primarily results in increased TPR, it is logical that their combined effect on MAP appears additive.⁴³⁻⁴⁴

While changes in hemodynamic parameters are probably not due to direct pressure, compression can nonetheless be of benefit in certain settings.⁴⁵⁻⁴⁸ External pressure is capable of stopping arterial bleeding in small lacerations and venous bleeding in all lacerations, so long as coagulation, vasoconstriction, and hypotension proceed unimpeded. External application of pressures less than systolic offers no advantage in stopping bleeding due to large arterial lacerations.⁴⁸

PULMONARY FUNCTION

One study demonstrated that pulmonary function may be impaired with PASG use, since a 13.8% decrease in vital capacity was seen with inflation to a

pressure of 100 mm Hg.⁴⁹ Another study reported no effect on minute ventilation in a study of ten supine patients, but did note a significant increase in transdiaphragmatic pressure with the PASG.⁵⁰ Another study showed decreases in forced expiratory volume, vital capacity, functional residual capacity, and tidal volume without effect on minute ventilation due to an increase in respiratory rate.⁵¹ Harmful effects in swine with experimentally induced diaphragmatic rupture were reported.⁵² The negative effects on pulmonary function in these studies were small, but were observed in healthy volunteers. One might speculate that the effects on pulmonary function in a trauma patient might be amplified; however, this has not been studied.

INTRACRANIAL PRESSURE

Three studies evaluated the effect of the PASG on intracranial pressure (ICP) in hypovolemic animals. All found no significant change in ICP with PASG inflation.⁵³⁻⁵⁵ In a study of 12 patients with ICP monitors in place and ICPs less than 20 mm Hg, small incremental increases in intracranial pressure were observed with PASG inflation to 100 mm Hg.⁵⁶ Two patients had inflation discontinued due to ICP increases to more than 24 mm Hg. Increased cerebral perfusion pressure appeared to more than compensate for the small increases in ICP. A report of 24 patients with ICP monitors and intermittent pneumatic compression of the legs noted no significant change in ICP or cerebral perfusion pressure.⁵⁷ The use of the PASG in patients who have closed head injuries does not appear to produce significant elevations in ICP; however, patients who have significantly elevated baseline ICPs have not been studied.

IV FLUID AND DRUG DELIVERY

No significant delay in drug delivery to the central circulation was observed in a canine study of infusion distal to a PASG inflated to 60 mm Hg in either normal or cardiac arrest states.⁵⁸ In a canine hypotensive model, significant delivery of distally infused intravenous fluid to the central circulation of IV fluid occurred despite PASG inflation to 100 mm Hg.⁵⁹ Similar results were reported in human volunteers.⁶⁰ Whether the flow of intravenous fluids infused distal to the PASG is impeded from reaching the central circulation in patients with hemorrhagic shock has not been specifically studied.

ENVIRONMENTAL CONSIDERATIONS

As with any closed vessel, the internal pressure in the PASG is directly related to the ambient temperature and inversely related to pressure. The internal pressure of the PASG increases in linear proportion to increases

in altitude (as pressure decreases).⁶¹ As temperature increases, the internal pressure of the PASG slowly increases proportionately.⁶¹

INFLATION PRESSURE

The effects of various inflation pressures on MAP have been studied. Inflation pressures as low as 20–30 mm Hg can significantly increase MAP in hemorrhagic models, with higher pressures having more marked effects. Pressures above 80–100 mm Hg, however, cease to have additive effects. In a study of 91 hypotensive trauma patients, PASG inflation to 30 mm Hg increased MAP in 27 patients.⁶² Subsequent inflation of the PASG to 60–80 mm Hg generated increased MAP in an additional 37 patients. Overall, 70% of the patients showed increased MAP with stepwise PASG inflation.

TIME CONSIDERATIONS

A recurrent concern with PASG application is delay in transport. Whether or not application of the PASG delays out-of-hospital care has not been independently studied. Three studies that examined the times for ALS procedures in trauma were unable to show an increased scene time with PASG application.^{63–65} However, none of these studies controlled for other potential sources of delay, such as level of provider training, need for airway management, or extrication.

HEMORRHAGIC SHOCK

Animal models of hemorrhagic shock can be divided into models of controlled and uncontrolled hemorrhage. In controlled hemorrhage, the animal is bled a specific amount of the blood volume or to a specific blood pressure, the blood loss is stopped, and the animal is maintained at that pressure through various means giving reproducible mortality rates. In uncontrolled hemorrhage, the animal is bled to specific blood pressure and then a vascular defect is created, allowing the animal to continue to hemorrhage, giving a more physiologic model of injury, also with reproducible mortality rates.

Cardiovascular dynamics were evaluated in 60 hypovolemic dogs bled 30 ml/kg. PASGs were applied and inflated to 30 mm Hg, producing an increased MAP and cardiac output. Increased SVR was noted 15 minutes after hemorrhage, which decreased slightly with PASG inflation. The PASG caused a slight redistribution of blood flow to the noncompressed areas.⁶⁶

Effects of the PASG were compared in two groups of dogs. The first had a sequential bleed to MAPs of 75 and 40 mm Hg with a spontaneous physiologic return of MAP to 74 mm Hg prior to PASG inflation, which the investigators referred to as *compensated hemorrhage*. The second had a sequential bleed first to MAP 75 mm

Hg and then to a level that required a 5% transfusion to bring the pressure up to 40 mm Hg, after which there was no physiologic recovery, which the investigators called *decompensated hemorrhage*. The PASGs were inflated stepwise from 30 to 90 mm Hg. In the compensated-hemorrhage group, the PASG significantly increased MAP and CO, without a change in TPR. In the decompensated-hemorrhage group, MAP and TPR increased, without change in CO. In this model, the PASG appeared to have a significant effect of MAP independent of SVR in moderate hemorrhage, but in severe hemorrhage the increase in MAP was solely due to an increase in SVR.⁶⁷

In another dog model where the animals were bled to a stabilized MAP of 40 mm Hg, SVR was noted to have risen significantly after the initial hemorrhage. With subsequent PASG inflation to 20 mm Hg, SVR rose even higher. The investigators also noted increases in MAP, CVP, and CO.⁶⁸

A significant survival benefit was observed with PASG use in a porcine model of fatal hemorrhage. The animals were bled 45 ml/kg and PASGs were inflated to 60 mm Hg. Although these models helped to elucidate the effects of the PASG in various hypovolemic states, the uncontrolled-hemorrhage model is more applicable to human trauma.⁶⁹

Using a canine model of uncontrolled abdominal hemorrhage created by crushing the spleens of the test animals, a significant decrease in blood loss was demonstrated using the PASG inflated to 100 mm Hg.⁷⁰ A rat model of uncontrolled hemorrhage demonstrated that the combination of external counter-pressure and hypertonic saline provided a survival benefit unrealized with hypertonic saline alone.⁷¹ The external pressure converted an uncontrolled hemorrhage into a controlled one. A report of a porcine model of uncontrolled hemorrhage secondary to an abdominal aortic laceration demonstrated improved survival and decreased blood loss with PASG inflation to 60 mm Hg.⁷² Additional studies also demonstrated improved survival of abdominal uncontrolled hemorrhage with the PASG.^{73–76}

Studies with uncontrolled hemorrhage in areas not compressed by the PASG yield quite different results. A study of uncontrolled hemorrhage involving a descending thoracic aortic injury showed a significant increase in blood loss with the PASG inflation. There was also a decrease in survival time from better than one hour without the PASG to between 10 and 18 minutes in animals with the PASG inflated to a degree sufficient to return blood pressure to baseline level.⁷⁷

The animal models demonstrate consistently that in all types of hemorrhage, MAP is improved with the application of the PASG. The investigators also demonstrated that if the hemorrhage was compressed by the PASG, decreased blood loss and improved survival were observed. Conversely, if hemorrhage could not

be directly compressed by the PASG, increased volumes of blood were lost and survival was shortened.

DIMINISHED CARDIAC RESERVE

The PASG was studied in patients with impaired cardiac reserves, and was found to be associated with increased afterload, left and right ventricular workload, and pulmonary capillary wedge pressure.^{78,79} Patients who had preexisting coronary artery disease had more pronounced effects. PASG inflation did not appear to precipitate symptoms of pulmonary edema in patients who had histories of congestive heart failure, especially when inflation times were kept to a minimum.^{80,81}

THE ELDERLY

In elderly patients without preexisting cardiac disease, PASG application and inflation were associated with increased MAP and TPR. In addition, diminished left ventricular performance, stroke volume, and cardiac output were observed. Left ventricular dysfunction was greatest when higher inflation pressures were used, and when inflation times exceeded 10 to 15 minutes.⁸²

ANAPHYLACTIC SHOCK

The only reported uses of the PASG in patients with anaphylactic shock are four case reports of successful treatment of refractory anaphylactic shock.¹²⁻¹⁶ All patients were refractory to intravenous epinephrine and vigorous fluid resuscitation. All showed hemodynamic improvement with inflation and subsequently recovered without sequelae. While use in this setting appears promising, the lack of a controlled clinical trial limits the recommendation.

SEPTIC SHOCK

There is no report studying the use of the PASG in a large series of patients with septic shock. The only case report that could be found utilized a device so different from the conventional PASG that no recommendation could be made. The device applied 150 mm Hg pressure to the lower extremities intermittently during peak systolic arterial pressure. The device fully deflated during diastole.⁸³

The PASG inflation results in increased TPR, which may be of theoretical benefit in septic shock. Whether or not this beneficial increase in TPR is offset by reductions in cardiac output and stroke volume remains to be determined.

HEMORRHAGIC SHOCK

None of the limited number of controlled studies of the use of the PASG in humans has been as conclusive as

the animal studies. Examination of the effect of the PASG on presenting emergency-department trauma scores (TSs) demonstrated no significant difference between control and PASG-treated patients.⁸⁴ Patients with thoracic injuries were not excluded from the study. Twenty-five of the 68 study patients had penetrating chest trauma. Nine blunt-trauma patients were in the no-PASG group, compared with four in the PASG group. All PASG compartments were inflated simultaneously. Another study of 201 patients with penetrating abdominal injuries reported no significant difference in survivals between treatment groups. There were 81 survivors in the 104 patients without PASG treatment, and 67 survivors in the 97 patients treated with the PASG.^{85,86}

One prospective randomized study of trauma patients was reported in 1989, with 345 patients in the PASG group and 439 patients in the non-PASG group (911 total). There was a 31% mortality in the PASG group compared with a 25% mortality in the non-PASG group. There were 320 patients with thoracic injuries entered into this study.⁸⁷

A retrospective study of 147 severely hypotensive trauma patients reported that survival of patients treated with the PASG was significantly better than that of non-PASG patients. No improvement in survival was found in patients with blood pressures of 50–70 mm Hg or in patients with blood pressures of 90 mm Hg or less. The same results were reported for both blunt trauma and penetrating trauma. The blunt-trauma rate was 64%.⁸⁸ Another retrospective review of 70 patients with penetrating cardiac wounds found significantly lower survival in patients treated with the PASG, with eight of 44 surviving, compared with 13 of 26 patients surviving when not treated with the PASG.⁸⁹

There is sufficient evidence based on animal and human data to indicate that the use of PASG in injuries to the thorax is potentially harmful. PASG use in hypotensive patients with abdominal trauma has not been shown to be beneficial, but has not yet been shown to be clearly harmful. PASG use in patients with severe hypotension, as evidenced by a thready pulse but unobtainable blood pressure, may be of benefit, although no prospective study has been done on this class of patients.

NONTRAUMATIC HYPOVOLEMIC SHOCK

The treatment of nontraumatic hypovolemia with the PASG has not been widely studied. Use of the PASG in patients with simulated hypotension was associated with a significant increase in MAP with combined PASG inflation of 80 mm Hg in the leg and 40 mm Hg in the abdominal compartments.⁹⁰ An evaluation of the effect of the PASG on hemodialysis-induced hypotension in seven patients failed to demonstrate a benefit

for the use of the PASG.⁹¹ In medical hypotension, the PASG may have a positive effect on blood pressure, and has not been shown to be harmful. Intravenous fluid probably offers better treatment.

ADJUNCT TO CPR

As an adjunct to CPR in the setting of cardiac arrest, inflation of the PASG has been reported to produce increases in MAP, systolic arterial pressure, cerebral perfusion pressure, common carotid artery flow, and arterial partial pressure of CO₂. Tidal volume, pH, and arterial partial pressure of O₂ were decreased. Cardiac output remained unchanged. One study reported a slightly increased survival rate for patients with pulseless electrical activity (PEA) and ventricular fibrillation (VF), but the sample size was small, comparison groups were not standardized, and the difference was not statistically significant.⁴⁻⁶

Two studies reported that abdominal binding was associated with increased pressure in the right atrium and the aorta during systole and diastole.^{4, 11} Common carotid flow and flows to the brain, heart, and kidney all transiently increased. However, binding resulted in decreased coronary perfusion pressure, which has been independently correlated with survival.⁷⁻¹¹ This finding raises significant doubt that either the PASG or abdominal binding is effective as an adjunct to CPR. The decrease in coronary perfusion pressure is worrisome and raises great concern that the PASG is harmful when used to treat patients who are in cardiac arrest.

ABDOMINAL AORTIC ANEURYSM (AAA)

One very compelling study examined outcomes in 18 patients with ruptured AAAs and systolic blood pressures (SBPs) less than 80 mm Hg who were allocated to control and to PASG-treatment arms. The PASG group had a lower average SBP (54 mm Hg vs 76 mm Hg), but had increased survival to the operating room (88% vs 60%), and increased postoperative survival (75% vs 0%). Despite the fact that the PASG-treatment group appeared more gravely ill, their outcomes were much improved.⁹¹

In another study using a porcine model to study uncontrolled hemorrhage from the abdominal aorta, the group treated with the PASG survived longer than either the control group or the group treated with aggressive volumes of intravenous fluids (8–10 mL/kg/min). In addition, the use of the PASG was associated with a fourfold reduction in blood loss.⁹²

Given these dramatic results, the PASG appears to limit blood loss in cases of ruptured AAA. Use of the PASG should not delay operative intervention. If definitive surgery is unavailable or delayed, PASG application may be indicated.

SPINAL SHOCK

Studies of the PASG and exercise tolerance in paraplegic patients demonstrate a decrease in lower body venous pooling and increased blood pressure over baseline.^{93, 94} There has been no such study in trauma patients. In spinal shock, the PASG has not been shown to be of harm and may be helpful.

HYPOTHERMIA-INDUCED HYPOTENSION

One study showed PASG to be of no benefit in canine hypotension induced by hypothermia.⁹⁵ No benefit has been shown for PASG in hypothermia.

CARDIAC TAMPONADE

In a study using the PASG in a canine model of decompensated pericardial tamponade, improvements of cardiac filling, MAP, and CO was reported.⁹⁶ Findings using a sheep model showed neither beneficial or harmful effect from PASG inflation up to 80 mm Hg.⁹⁷ Considering that the diagnosis of cardiac tamponade is extremely difficult in the out-of-hospital setting, the fact that tamponade is rarely an isolated injury in trauma, and the previous information indicating that the PASG is contraindicated in thoracic trauma, PASG use in pericardial tamponade is without supporting scientific data and may be harmful.

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA (PSVT)

There are a number of case reports of PSVT conversion with PASG use simulating the Valsalva maneuver.¹⁷⁻¹⁹ Another study reported four conversions in 24 attempts with PASG inflation.⁹⁸ Use of the PASG for the treatment of PSVT may be helpful and is probably not harmful. One should not consider the PASG as primary treatment for PSVT, since there are currently much better out-of-hospital treatments for this condition.

PELVIC FRACTURE

While there has been no prospective study of the use of the PASG in pelvic fracture, retrospective reviews and case reports support PASG use. A report on the use of the PASG in ten patients with bleeding in excess of 2,000 ml after other surgically repairable lesions had been excluded found that prompt hemostasis and reduction in mortality were achieved compared with historical controls.⁹⁹ Another study reported improved survival using the PASG as part of a graded approach to open pelvic fractures. The PASG was used for two patients in combination with external packing and resulted in hemostasis.¹⁰⁰

These reports support the use of the PASG for he-

mostasis in patients with pelvic fracture and uncontrolled bleeding or hypotension. There is sufficient information to recommend the use of the PASG in hypotensive patients with suspected pelvic fracture as the primary cause of their hypotension.¹⁰¹⁻¹⁰⁶ There is not enough information to support universal use in the management of pelvic fractures, as most do not require intervention for hemostasis and PASG use is not without complications.

INTRAVENOUS ACCESS

No study has directly addressed the question of the usefulness of the PASG to improve IV start percentage. EMS providers often give anecdotal reports of improved IV accessibility with PASG inflation. Many providers believe the PASG improves the ability to start an IV line in the hypotensive trauma patient. Considering the hemodynamic studies that indicate improved cardiac filling with the PASG, it seems a reasonable assumption that the device would increase peripheral venous filling as well as providing a more visible target for IV access. Mattox et al., in commenting on their PASG study, indicated that there were improved IV start rates in the PASG group.²¹ The PASG seems to improve IV success rates; however, this should be considered a secondary benefit of its use and not a primary indication for PASG use.

EVIScerATION

The use of the PASG in the setting of abdominal organ evisceration is theoretically harmful due to peritoneal contamination and visceral ischemia, but has not been reported. One case where traumatic diaphragmatic rupture was exacerbated has been reported.¹⁰⁷

PREGNANCY

A series of cases involving the successful treatment of spontaneous rupture of the liver in pregnancy has been reported.¹⁰⁸ No control group was used, and all patients were stabilized to permit operative intervention. Another case series reported the successful use of the PASG in the setting of ruptured ectopic pregnancy with uncontrolled hemorrhage and shock.¹⁰⁹ Use in uncontrolled gynecologic hemorrhage has also been speculated.¹¹⁰ Use in pregnant patients in shock for reasons other than isolated pelvic or intra-abdominal hemorrhage has not been studied, and use of the abdominal compartment in the setting of a gravid uterus is believed to be harmful.

UROLOGY

Case reports had appeared describing the use of the PASG in the setting of otherwise uncontrollable urologic hemorrhage following transurethral resection of

the prostate, nephrectomy, prostatectomy, and renal biopsy.^{111, 112} In one case, the PASG remained in place for 36 hours, with the device deflated for 10 minutes every hour to prevent ischemia.¹¹²

LOCAL HEMORRHAGE CONTROL

A number of case reports have described the use of the PASG in the setting of lower-extremity injury. One case involved massive degloving of the perineum and buttocks.¹¹³ Use of the PASG on patients with lower extremity injuries must be weighed against the risk of pressure injury. While only 2% of patients tested positive for myoglobinuria, muscle damage, as evidenced by increased creatine phosphokinase, begins within two hours of application.¹¹⁴ Intermittent release of pressure appears to prevent damage. Digital blood flow to the lower extremities is absent in 90% of patients at inflation pressures as low as 100 mm Hg, and at even lower pressures if the extremity has been fractured.^{115, 116} In addition, compartment pressure appears to approximate PASG compartment inflation pressure so that prolonged exposure to high pressures increases the risk of compartment syndrome.¹¹⁷⁻¹¹⁹

CONCLUSIONS

The PASG was initially advocated for the treatment of shock for a variety of reasons. Examination of the literature reveals that the therapeutic effects of the PASG, and the settings in which it should or should not be used, are unclear. While beneficial in certain instances, PASGs are harmful in other closely related, clinical conditions. Finally, there are many areas for further research, such as in their use in AAAs, in the severely hypotensive, and for local hemorrhage control. In addition, newer compression devices are appearing that could have applications in situations where the conventional PASG is impractical or if prolonged application is required. The decision whether or not to use the PASG in out-of-hospital care is a difficult one, with many questions yet unanswered. We have attempted to answer such questions whenever the information needed to do so was available, but have perhaps raised additional issues to be addressed in the future.

References

1. Randall P, Banks J, Little RA. Medical (military) anti-shock trousers—a short review. *Arch Emerg Med.* 1984;1:39-51.
2. Randall PE. Medical anti-shock trousers (MAST): a review. *Injury.* 1986;17:395-98.
3. Davis SM. Anti-shock trousers: a collective review. *J Emerg Med.* 1986;4:145-55.
4. Bircher N, Safar P, Stewart R. A comparison of standard, "MAST"-augmented, and open-chest CPR in dogs. *Crit Care Med.* 1980;8:147-52.
5. Lee HR, Wilder RJ, Downs P, et al. MAST augmentation of external cardiac compression: role of changing intrapleural pres-

- sure. *Ann Emerg Med.* 1981;10:560-5.
6. Warren ET, Pass HI, Crawford FA. External cardiopulmonary resuscitation augmented by the military anti-shock trousers. *Am Surg.* 1983;49:651-4.
 7. Lilja GP, Long RS, Ruiz E. Augmentation of systolic blood pressure during external cardiac compression by use of the MAST suit. *Ann Emerg Med.* 1981;10:182-4.
 8. Chandra N, Snyder LD, Weisfeldt ML. Abdominal binding during cardiopulmonary resuscitation in man. *JAMA.* 1981;246:351-3.
 9. Unger RJ, Feiner JR. Hemodynamic effects of intermittent pneumatic compression of the legs. *Anesthesiology.* 1987;67:266-8.
 10. Mahoney BD, Mirick MJ. Efficacy of pneumatic trousers in refractory prehospital cardiopulmonary arrest. *Ann Emerg Med.* 1983;12:8-12.
 11. Niemann JT, Rosborough JP, Ung S, et al. Hemodynamic effects of continuous abdominal binding during cardiac arrest and resuscitation. *Am J Cardiol.* 1984;53:269-74.
 12. Bickell WH, Dice WH. Military anti-shock trousers in a patient with adrenergic-resistant anaphylaxis. *Ann Emerg Med.* 1984;13:189-90.
 13. Oertel T, Loehr MM. Bee-sting anaphylaxis: the use of medical anti-shock trousers. *Ann Emerg Med.* 1984;13:459-61.
 14. Low RB. MAST and adrenergic-resistant anaphylaxis [letter]. *Ann Emerg Med.* 1985;14:373-4.
 15. Granata AV, Halickman JF, Borak J. Utility of military anti-shock trousers (MAST) in anaphylactic shock—a case report. *J Emerg Med.* 1985;2:349-51.
 16. Perkin RM, Anas NG. Mechanisms and management of anaphylactic shock not responding to traditional therapy. *Ann Allergy.* 1985;54:202-8.
 17. Tandberg D, Rusnak R, Sklar D, et al. Successful treatment of paroxysmal supraventricular tachycardia with MAST. *Ann Emerg Med.* 1984;13:1068-70.
 18. Tandberg D, Hauswald M, Rusnak R. MAST conversion of paroxysmal supraventricular tachycardia in Wolff-Parkinson-White syndrome. *Ann Emerg Med.* 1987;16:712-14.
 19. Walker LA, MacMath TL, Chipman H, et al. MAST application in the treatment of paroxysmal supraventricular tachycardia in a child. *Ann Emerg Med.* 1988;17:529-31,1097.
 20. Effect of lower body positive pressure on exercise capacity in spinal cord injury patients. *Med Science in Sports and Exercise.* 1994;26:463-8.
 21. Mattox KL, Bickell W, Pepe PE, et al. Prospective MAST study in 911 patients. *J Trauma.* 1989;29:1104-12.
 22. Honigman B, Lowenstein SR, Moore EE, et al. The role of the pneumatic anti-shock garment in penetrating cardiac wounds. *JAMA.* 1991;266:2398-401.
 23. Cayten CG, Berendt BM, Byrne DW, et al. A study of pneumatic anti-shock garments in severely hypotensive trauma patients. *J Trauma.* 1993;34:728-35.
 24. American College of Surgeons, Committee on Trauma: *Advanced Trauma Life Support.* 5th ed. Chicago, IL: American College of Surgeons, 1993.
 25. Prehospital Trauma Life Support Committee of the National Association of Emergency Medical Technicians in Cooperation with the American College of Surgeons, Committee on Trauma: *Basic and Advanced Prehospital Trauma Life Support.* 3rd ed. St. Louis, MO: Mosby-Lifeline, 1994.
 26. Campbell JE. *BTLS: Basic Prehospital Trauma Care.* Englewood Cliffs, NJ: Prentice-Hall, 1988.
 27. U.S. Department of Transportation, National Highway Safety Administration: *Emergency Medical Technician: National Standard Curriculum.* Washington, DC: Department of Transportation, 1985.
 28. Ali J, Duke K. Timing and interpretation of the hemodynamic effects of the pneumatic anti-shock garment. *Ann Emerg Med.* 1991;20:1183-7.
 29. Goldstein A, Phillips T, Scafani SJ, et al. Early open reduction and internal fixation of the disrupted pelvic ring. *J Trauma.* 1986;26:325-33.
 30. Abraham E, Cobo JC, Bland RD, et al. Cardiorespiratory effects of pneumatic trousers in critically ill patients. *Arch Surg.* 1984;119:912-6.
 31. Neimann JT, Stapczynski JS, Rosborough JP, et al. Hemodynamic effects of pneumatic external counterpressure in canine hemorrhagic shock. *Ann Emerg Med.* 1983;12:661-7.
 32. Goldsmith SR. Comparative hemodynamic effects of anti-shock suit and volume expansion in normal human beings. *Ann Emerg Med.* 1983;12:348-50.
 33. Ali J, Vanderby B, Purcell C. The effect of the pneumatic anti-shock garment (PASC) on hemodynamics, hemorrhage, and survival in penetrating thoracic aortic injury. *J Trauma.* 1991;31:846-51.
 34. Hauswald M, Greene ER. Aortic blood flow during sequential MAST inflation. *Ann Emerg Med.* 1986;15:1297-9.
 35. Bellamy RF, DeGuzman LR, Pedersen DC. Immediate hemodynamic consequences of MAST inflation in normo- and hypovolemic anesthetized swine. *J Trauma.* 1984;24:889-95.
 36. Caldwell CB, Ricotta JJ. Changes in visceral blood flow with elevated intraabdominal pressure. *J Surg Res.* 1987;43:14-20.
 37. Julius S, Sanchez R, Malayan S, et al. Sustained blood pressure elevation to lower body compression in pigs and dogs. *Hypertension.* 1982;4:782-8.
 38. Eich RH, Smulyan H, Chaffee WR. Hemodynamic response to G-suit inflation with and without ganglionic blockade. *Aerospace Med.* 1966;37:247-50.
 39. Bivins HG, Knopp R, Tiernan C, et al. Blood volume displacement with inflation of anti-shock trousers. *Ann Emerg Med.* 1982;11:409-12.
 40. Hanke BK, Bivins HG, Knopp R, et al. Anti-shock trousers: a comparison of inflation techniques and inflation pressures. *Ann Emerg Med.* 1985;14:636-40.
 41. Jennings TJ, Seaworth JF, Howell LL, et al. The effects of various anti-shock trouser inflation sequences on hemodynamics in normovolemic subjects. *Ann Emerg Med.* 1986;15:1193-7.
 42. Begin R, Dougherty R, Michael ED, et al. Effect of sequential anti-G suit inflation on pulmonary capillary blood flow in man. *Aviat Space Environ Med.* 1976;47:937-41.
 43. Hauswald M, Tandberg D. The effect of patient position and MAST inflation on carotid sinus diameter. *Ann Emerg Med.* 1985;14:1065-8.
 44. Zippe C, Burchard KW, Gann DS. Trendelenburg versus PASG application in moderate hemorrhagic hypoperfusion. *J Trauma.* 1985;25:923-932.
 45. Eddy DM, Wangenstein SL, Ludewig RM. The kinetics of fluid loss from leaks in arteries tested by an experimental *ex vivo* preparation and external counterpressure. *Surgery.* 1968;64:451-8.
 46. Wangenstein SL, Ludewig RM, Cox JM, et al. The effect of external counterpressure on arterial bleeding. *Surgery.* 1968;64:922-7.
 47. Ludewig RM, Wangenstein SL. Effect of external counterpressure on venous bleeding. *Surgery.* 1969;66:515-20.
 48. Ludewig RM, Wangenstein SL. Aortic bleeding and the effect of external counterpressure. *Surg Gynecol Obstet.* 1969;128:252-8.
 49. McCabe JB, Seidel DR, Jagger JA. Anti-shock trouser inflation and pulmonary vital capacity. *Ann Emerg Med.* 1983;12:290-3.
 50. Abraham E, Cobo JC, Bland RD, et al. Cardiorespiratory effects of pneumatic trousers in critically ill patients. *Arch Surg.* 1984;119:912-6.
 51. Riou B, Pansard JL, Lazard T, et al. Ventilatory effects of medical anti-shock trousers in healthy volunteers. *J Trauma.* 1991;31:1495-502.
 52. Maull KI, Krahwinkel DJJ, Rozycki GS, et al. Cardiopulmonary effects of the pneumatic anti-shock garment on swine with diaphragmatic hernia. *Surg Gynecol Obstet.* 1986;162:17-24.
 53. Dannewitz SR, Lilja GP, Ruiz E. Effect of pneumatic trousers on intracranial pressure in hypovolemic dogs with an intracra-

- nial mass. *Ann Emerg Med.* 1981;10:176–81.
54. Cram AE, Davis JW, Kealey GP, et al. Effects of pneumatic anti-shock trousers on canine intracranial pressure. *Ann Emerg Med.* 1981;10:28–31.
 55. Palafox BA, Johnson MN, McEwen DK, et al. ICP changes following application of the MAST suit. *J Trauma.* 1981;21:55–9.
 56. Gardner SR, Maull KI, Swensson EE, et al. The effects of the pneumatic anti-shock garment on intracranial pressure in man: a prospective study of 12 patients with severe head injury. *J Trauma.* 1984;24:896–900.
 57. Davidson JE, Willms DC, Hoffman MS. Effect of intermittent pneumatic leg compression on intracranial pressure in brain-injured patients. *Crit Care Med.* 1993;21:224–7.
 58. Joyce SM, Barsan WG, Hedges JR, et al. Effect of a pneumatic anti-shock garment on drug delivery via distal venous access. *Ann Emerg Med.* 1984;13:885–90.
 59. Mullin MJ, Krohmer JR, McCabe JB. Intravenous fluid flow beneath inflated anti-shock trousers in a canine hemorrhagic shock model. *Ann Emerg Med.* 1987;16:153–5.
 60. Tucker JF, Danzl DF, Teague E, et al. Infusion of intravenous fluids distal to pneumatic anti-shock trousers. *J Emerg Med.* 1984;2:79–83.
 61. Sanders AB, Meislin HW. Effect of altitude change on MAST suit pressure. *Ann Emerg Med.* 1983;12:140–4.
 62. Wayne MA, Macdonald SC. Clinical evaluation of the anti-shock trouser: prospective study of low-pressure inflation. *Ann Emerg Med.* 1983;12:285–9.
 63. Cwinn AA, Pons PT, Moore EE, et al. Prehospital advanced trauma life support for critical blunt trauma victims. *Ann Emerg Med.* 1987;16:399–403.
 64. Honigman B, Rohweder K, Moore EE, et al. Prehospital advanced trauma life support for penetrating cardiac wounds. *Ann Emerg Med.* 1990;19:145–50.
 65. Pons PT, Honigman B, Moore EE, et al. Prehospital advanced trauma life support for critical penetrating wounds to the thorax and abdomen. *J Trauma.* 1985;25:828–32.
 66. Ferrario CM, Nadzam G, Fernandez LA, et al. Effects of pneumatic compression on the cardiovascular dynamics in the dog after hemorrhage. *Aerospace Med.* 1970;41:411–5.
 67. Johnson G, 3d, Bond RF, Stack LB, et al. Efficacy of military anti-shock trousers in compensatory and decompensatory hemorrhagic hypotension. *Circ Shock.* 1987;21:233–45.
 68. Roth JA, Rutherford RB. Regional blood flow effects of G suit application during hemorrhagic shock. *Surg Gynecol Obstet.* 1971;133:637–43.
 69. Traverso LW, Lee WP, DeGuzman LR, et al. Military anti-shock trousers prolong survival after otherwise fatal hemorrhage in pigs. *J Trauma.* 1985;25:1054–8.
 70. Low RB, Schmidt C, Wilder RJ, et al. Control of intraabdominal hemorrhage and shock: a comparison of fluid resuscitation, MAST, and balloon occlusion. *Ann Emerg Med.* 1985;14:540–6.
 71. Landau EH, Gross D, Assalia A, et al. Treatment of uncontrolled hemorrhagic shock by hypertonic saline and external counterpressure. *Ann Emerg Med.* 1989;18:1039–43.
 72. Ali J, Purcell C, Vanderby B. Effect of intraabdominal pressure and saline infusion on abdominal aortic hemorrhage. *J Cardiovasc Surg.* 1991;32:653.
 73. Ali J, Duke K. Pneumatic anti-shock garment decreases hemorrhage and mortality from splenic injury. *Can J Surg.* 1991;34:496–501.
 74. Aberg T, Steen S, al Othman K, et al. The effect of pneumatic anti-shock garments in the treatment of lethal combined hepatic and caval injuries in rats. *J Trauma.* 1986;26:727–32.
 75. Aberg T, Steen S, Vagianos C, et al. The effects of pneumatic anti-shock garments in the treatment of critical abdominal injuries in rats. *J Trauma.* 1988;28:772–8.
 76. Aberg T, Rosen I, Walther B, et al. Cerebral function monitoring in rats with a critical hepatic injury treated with pneumatic anti-shock garment and infusion. *J Trauma.* 1989;29:168–74.
 77. Ali J, Vanderby B, Purcell C. The effect of the pneumatic anti-shock garment (PASG) on hemodynamics, hemorrhage, and survival in penetrating thoracic aortic injury. *J Trauma.* 1991;31:846–51.
 78. Rubal BJ, Geer MR, Bickell WH. Effects of pneumatic anti-shock garment inflation in normovolemic subjects. *J Appl Physiol.* 1989;67:339–45.
 79. Gaffney FA, Thal ER, Taylor WF, et al. Hemodynamic effects of medical anti-shock trousers (MAST garment). *J Trauma.* 1981;21:931–7.
 80. Jabbour I, Savino JA, Agarwal N, et al. Pneumatic anti-shock garments detrimental in elderly with diminished myocardial reserve. *Curr Surg.* 1986;43:498–501.
 81. Bain RJ, Tan LB, Murray RG, et al. Central hemodynamic changes during lower body positive pressure in patients with cardiac failure. *Cardiovasc Res.* 1989;23:833–7.
 82. Savino JA, Jabbour I, Agarwal N, et al. Overinflation of pneumatic anti-shock garments in the elderly. *Am J Surg.* 1988;155:572–7.
 83. Gunter JP, deBoisblanc BP, Rust BS, et al. Effect of synchronized, systolic, lower body, positive pressure on hemodynamics in human septic shock: a pilot study. *Am J Resp Crit Care.* 1995;151:719–23.
 84. Bickell WH, Pepe PE, Wyatt CH, et al. Effect of anti-shock trousers on the trauma score: a prospective analysis in the urban setting. *Ann Emerg Med.* 1985;14:218–22.
 85. Bickell WH, Pepe PE, Bailey ML, et al. Randomized trial of pneumatic anti-shock garments in the prehospital management of penetrating abdominal injuries. *Ann Emerg Med.* 1987;16:653–8.
 86. Bickell WH, Pepe PE, Bailey ML, et al. Efficacy of pneumatic trousers in the prehospital management of penetrating abdominal injuries. *Ann Emerg Med.* 1987;16:653–8.
 87. Mattox KL, Bickell WH, Pepe PE, et al. Prospective randomized evaluation of anti-shock MAST in post-traumatic hypotension. *J Trauma.* 1986;26:779–86.
 88. Cayten CG, Berendt BM, Byrne DW, et al. A study of pneumatic anti-shock garments in severely hypotensive trauma patients. *J Trauma.* 1993;34:728–35.
 89. Honigman B, Lowenstein SR, Moore EE, et al. The role of the pneumatic anti-shock garment in penetrating cardiac wounds. *JAMA.* 1991;266:2398–401.
 90. Mannering D, Bennett ED, Mehta N, et al. Application of the medical anti-shock (MAST) increases cardiac output and tissue perfusion in simulated, mild hypovolemia. *Intens Care Med.* 1986;12:143–6.
 91. Ali J, Purcell C, Vanderby B. Effect of intraabdominal pressure and saline infusion on abdominal aortic hemorrhage. *J Cardiovasc Surg.* 1991;32:653–9.
 92. Gustafson RA, McDowell DE, Savrin RA. The use of the MAST suit in ruptured abdominal aortic aneurysms. *Am Surg.* 1983;49:454–9.
 93. Hopman MT, Oeseburg B, Binkhorst RA. The effect of an anti-G suit on cardiovascular response to exercise in persons with paraplegia. *Med Sci Sports Exerc.* 1992;24:984–90.
 94. Hopman MT, Kamerbeek IC, Pistorius M, Binkhorst RA. The effect of an anti-G suit on the maximal performance of individuals with paraplegia. *Int J Sports Med.* 1993;14:357–61.
 95. Kolodzik PW, Mullin MJ, Krohmer JR, et al. The effects of anti-shock trouser inflation during hypothermic cardiovascular depression in the canine model. *Am J Emerg Med.* 1988;6:584–90.
 96. Davis JW, McKone TK, Cram AE. Hemodynamic effects of military anti-shock trousers (MAST) in experimental cardiac tamponade. *Ann Emerg Med.* 1981;10:185–6.
 97. Girotti MJ, Low DE, Lambros S. The pneumatic anti-shock garment: use in experimental acute cardiac tamponade. *Circ Shock.* 1986;19:203–9.

98. O'Toole KS, Heller MB, Menegazzi JJ, et al. Intravenous verapamil in the prehospital treatment of paroxysmal supraventricular tachycardia. *Ann Emerg Med.* 1990;19:291-4.
99. Flint LM, Brown A, Richardson JD, et al. Definitive control of bleeding from severe pelvic fractures. *Ann Surg.* 1979;189:709-16.
100. Richardson JD, Harty J, Amin M, et al. Open pelvic fractures. *J Trauma.* 1982;22:533-8.
101. Moreno C, Moore EE, Rosenberger A, et al. Hemorrhage associated with major pelvic fracture: a multispecialty challenge. *J Trauma.* 1986;26:987-94.
102. Bruining HA, Eeftinck Schattenkerk M, De Vries JE, et al. Clinical experience with the medical anti-shock trousers (MAST) treatment of hemorrhage, especially from compound pelvic fracture. *Neth J Surg.* 1980;32:102-7.
103. Mulcha PJ, Welch TJ. Hemorrhage in major pelvic fractures. *Surg Clin North Am.* 1988;68:757-73.
104. Brown JJ, Greene FL, McMillin RD. Vascular injuries associated with pelvic fractures. *Am Surg.* 1984;50:150-4.
105. Flint L, Babikian G, Anders M, et al. Definitive control of mortality from severe pelvic fracture. *Ann Surg.* 1990;221:703-6.
106. Evers BM, Cryer HM, Miller FB. Pelvic fracture hemorrhage. Priorities in management. *Arch Surg.* 1989;124:422-4.
107. Hagman J, Iguchi R, Kinsley J, et al. Diaphragmatic rupture following blunt trauma. *Ann Emerg Med.* 1984;13:49-52.
108. Hibbard LT. Spontaneous rupture of the liver in pregnancy: a report of eight cases. *Am J Obstet Gynecol.* 1976;126:334-8.
109. Sandberg EC, Pelligra R. The medical antigravity suit for management of surgically uncontrollable bleeding associated with abdominal pregnancy. *Am J Obstet Gynecol.* 1983;146:519-25.
110. Hall M, Marshall JR. The gravity suit: a major advance in management of gynecologic blood loss. *Obstet Gynecol.* 1979;53:247-50.
111. McCullough DL, McLaughlin AP, Warshawsky AB. The gravity suit: a useful device in complicated urologic hemorrhage. *Urology.* 1975;6:468-70.
112. Ryan DW, Johnston PP, Powell PH, et al. The G-suit in controlling massive urological haemorrhage. *Br J Urol.* 1986;58:226-7.
113. Pezzi C, Brotman S, Deitrick J. Massive degloving injury of the trunk. *Am J Emerg Med.* 1986;4:233-4.
114. Weiss LD, Lauro AJ, Derks FW, et al. The incidence of myoglobinuria in patients with pneumatic trousers. *Am J Emerg Med.* 1984;2:115-8.
115. Aprahamian C, Towne JB, Thompson BM, et al. Effect of circumferential pneumatic compression devices on digital flow. *Ann Emerg Med.* 1984;13:1092-5.
116. Chiu D, Wang HH, Blumenthal MR. Creatine phosphokinase release as a measure of tourniquet effect on skeletal muscle. *Arch Surg.* 1976;111:71-4.
117. Chisholm CD, Clark DE. Effect of the pneumatic anti-shock garment on intramuscular pressure. *Ann Emerg Med.* 1984;13:581-3.
118. Shakespeare DT, Henderson NJ, Sherman KP. Transmission of pressure into the human limb from pneumatic splints. *Injury.* 1984;16:38-40.
119. Hedges JR, Baker PB, Dalsey WC. Compartmental pressure measurements during application of the pneumatic anti-shock garment. *J Emerg Med.* 1984;1:377-85.