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. This tacit acceptance at one time extended to a variety of shock states, including cardiac arrest, anaphylaxis, and paroxysmal supraventricular tachycardia. The PASG has even been successfully used to increase the exercise tolerance of paraplegics.

Recently, two studies reported increased mortality associated with PASG use in victims of penetrating trauma. 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. 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. The inevitable 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 and the ability to use it is required for NREMT-P certification. 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. With time, venous return, preload, cardiac output, and stroke volume decrease, an effect that is more pronounced if lower PASG inflation pressures are used. With aortic blood flow reduced to 25% of normal, mean arterial pressure (MAP) is...
maintained proportionate to the increase in total peripheral resistance (TPR). Autotransfusion occurs to a limited degree, and does not contribute to maintaining TPR. Of note, one study suggested that the application and inflation of the PAG resulted in impaired oxygen utilization.

Sequential inflation and deflation of the PAG 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. In addition, the increased MAP response can be completely blocked by administering trimethaphan camysylate, suggesting that the increase in TPR is due to activation of vasomotor reflexes and not direct compression. Following rapid deflation, stroke volume and cardiac output increase, while heart rate, TPR, and MAP return to baseline levels.

The minimum inflation pressure needed to displace blood centrally is 40 mm Hg, however, higher inflation pressures displaced incrementally less volume. No more than a 5% increase in the central volume was achieved, regardless of inflation pressure or the sequence of compartment inflation. 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.

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 PAG, a greater percentage of volume is displaced centrally, and TPR is not affected. The Trendelenburg position and the PAG equally increase carotid sinus diameter. Since the Trendelenburg position primarily displaces volume centrally, and the PAG primarily results in increased TPR, it is logical that their combined effect on MAP appears additive.

While changes in hemodynamic parameters are probably not due to direct pressure, compression can nonetheless be of benefit in certain settings. 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.

**PULMONARY FUNCTION**

One study demonstrated that pulmonary function may be impaired with PAG use, since a 13.8% decrease in vital capacity was seen with inflation to a pressure of 100 mm Hg. 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 PAG. 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. Harmful effects in swine with experimentally induced diaphragmatic rupture were reported. 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 PAG on intracranial pressure (ICP) in hypovolemic animals. All found no significant change in ICP with PAG inflation. 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 PAG inflation to 100 mm Hg. 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. The use of the PAG 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 PAG inflated to 60 mm Hg in either normal or cardiac arrest states. In a canine hypotensive model, significant delivery of distally infused intravenous fluid to the central circulation of IV fluid occurred despite PAG inflation to 100 mm Hg. Similar results were reported in human volunteers. Whether the flow of intravenous fluids infused distal to the PAG 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 PAG is directly related to the ambient temperature and inversely related to pressure. The internal pressure of the PAG increases in linear proportion to increases
in altitude (as pressure decreases). As temperature increases, the internal pressure of the PASG slowly increases proportionately.

**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. 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. 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.

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.

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.

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.

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. 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. 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.

Additional studies also demonstrated improved survival of abdominal uncontrolled hemorrhage with the PASG.

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.

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. 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 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 (984 total). There was a 31% mortality in the PASG group compared with a 25% mortality in the no-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 inparaplegic 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 decompenated 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 uncontrollable 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.\textsuperscript{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. Mattax et al., in commenting on their PASG study, indicated that there were improved IV start rates in the PASG group.\textsuperscript{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.\textsuperscript{107}

**Pregnancy**

A series of cases involving the successful treatment of spontaneous rupture of the liver in pregnancy has been reported.\textsuperscript{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.\textsuperscript{109} Use in uncontrolled gynecologic hemorrhage has also been speculated.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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**

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