Medical (military) anti-shock trousers—a short review

P. RANDALL, J. BANKS AND R. A. LITTLE

MRC Trauma Unit, Hope Hospital, Salford

HISTORICAL INTRODUCTION

The first recorded use of pneumatic compression as a method to maintain blood pressure in hypovolaemic and hypotensive surgical patients was described by Crile in 1903. He used a pressurized rubber suit with good effect but acknowledged that the suits were prone to perish and leak. In 1909 he described its use in a patient with a cut throat and an unrecordable blood pressure where application of the suit maintained the blood pressure at 110 mm hg for 12 h. With the advent of safe methods of fluid replacement and later of blood transfusion pneumatic compression fell into disuse.

It was the use of the G-suit by fighter pilots in the 1940s and 1950s and its value in preventing retinal haemorrhage at altitude that reawoke medical interest in pneumatic compression. Gardner and Dohn (1956) reported the use of a G-suit in combating postural hypotension during surgery performed with the patient in the sitting position. Soon after this the use of a G-suit at a pressure of 20 mm Hg was described in a patient with post-partum intra-abdominal bleeding which had required 57 units of blood and two unsuccessful laparotomies (Gardner *et al.*, 1958). With the G-suit inflated for 24 h only one more unit of blood was required. Later it was shown that in anaesthetized dogs with arterial or venous lacerations the G-suit reduced bleeding dramatically when applied at pressures of 20–40 mm Hg and also elevated and maintained the blood pressure as long as the suit was inflated (Gardner & Storer, 1966; Wangenstein *et al.*, 1968b; Gardner, 1969; Ludewig & Wangenstein, 1969a,b). The control of bleeding by suit pressures well below mean arterial pressure was explained by the fact that the major determinant of tension in a vessel wall is the radius of that vessel (Law of Laplace, vide *infra*). The maintenance and elevation of blood pressure was thought to be due to redistribution of blood to areas above the suit. Gardner (1969) concluded that the suit should not be applied at pressures greater than 30 mm Hg in man or used to control bleeding above the diaphragm although no reasons for these statements were given.

Correspondence: Mr P. Randall, MRC Trauma Unit, Stopford Building, University of Manchester Medical School, Oxford Road, Manchester M13 9PT

The use of the G-suit at low pressure was found to be of use in transferring patients during the Vietnam war. Cutler & Daggett (1974) reported eight severely injured soldiers being transferred by helicopter at G-suit pressures of 30–40 cm of water. Four patients survived and all had an elevation of blood pressure while the G-suit was inflated. They concluded that the G-suit and its modification the MAST suit (military anti-shock trousers now medical anti-shock trousers) was the most effective form of resuscitation at a battle front, being without the risks attached to attempting intravenous infusion in surgically primitive conditions.

Kaplan and his colleagues first used MAST in civilian pre-hospital medical care (Kaplan *et al.*, 1973). They described 20 patients including eight cases of haemorrhagic shock with unrecordable blood pressure, seven of whom had an elevation of blood pressure when the trousers were applied. They do not report the pressure used but with its increasing use by the paramedical services in the USA for transferring all shocked patients (McSwain, 1977) the recommendation has become inflation to 100–110 mm Hg (i.e. until the safety valves open and the velcro straps are about to give way).

USES

The main value of the MAST suit appears to be in the transfer of shocked patients from the place of injury to hospital.

Bleeding

They have been used to manage traumatic retroperitoneal haemorrhage (McLaughlin *et al.*, 1972); to treat gun-shot wounds, fractures of the lower limbs and stab wounds (Kaplan *et al.*, 1973); in the management of intra-abdominal bleeding from aneurysms, gastrointestinal tract, ectopic pregnancy and ruptured liver (Espinosa & Updegrave, 1970; McSwain, 1976); for bleeding above the diaphragm (Crile, 1909; Lilja *et al.*, 1975; Wasserberger *et al.*, 1981), and the control of bleeding arising from hypo-coagulation (Lewis *et al.*, 1973).

Cardiogenic Shock

Wayne (1978) reported the use of MAST in cardiogenic shock. Of his 14 cases, six had no response and died. Eight of his patients survived but only five had any elevation of blood pressure when the suit was applied. This, coupled with a failure to define what was meant by cardiogenic shock in these cases must still cast doubt on whether MAST are of much value in these patients. More recently Mahoney and Mirick (1983) have shown that MAST (fully inflated) can be a useful adjunct in the treatment of refractory cardiac arrest. They suggest that MAST inflation during cardiopulmonary resuscitation increases peripheral vascular resistance which in turn will increase both diastolic aortic pressure and carotid artery blood flow.

Postural Hypotension

Rosenhamer & Thorstrand (1973) have described the use of the G-suit in the successful management of two cases of otherwise untreatable postural hypotension. This is confirmed by our own experience with a woman aged 34 with Crohns disease. She experienced dizziness on standing and could only maintain her blood pressure by an increase in heart rate. This situation was easily reversed by applying MAST at 30 mm Hg. Her symptoms resolved when she was fitted with elastic pressure stockings.

Septic Shock

There is, as yet, no report of MAST being used in such patients. However, they may be expected to be of some value in a shock state where peripheral vasodilation and pooling is a prominent feature.

Head Injuries

Wasserberger *et al.* (1981) have reviewed evidence which shows MAST to be effective in maintaining blood pressure in hypotension associated with some head injuries. Although intracranial pressure (ICP) is increased by MAST inflation there is a larger rise in mean arterial pressure (MAP), this means that cerebral perfusion pressure (MAP-ICP) is also increased and cerebral blood flow is maintained (Cram *et al.*, 1980).

EFFECTS OF MAST

There is general agreement that the application of MAST at pressures above 20 mm Hg in normal subjects and, more obviously, in shocked patients leads to a sustained rise in blood pressure (e.g. our own results, Table 1), stroke volume and cardiac output (Gray *et al.*, 1969; Barclay *et al.*, 1981; Gaffney *et al.*, 1981). The effects on peripheral blood flow and resistance in tissues above the trousers in uninjured subjects are not so clearcut and it is not obvious why different patterns of response are seen. An increase in forearm blood flow and a reduction in peripheral resistance at inflation pressures up to 30 mm Hg have been reported (Barclay *et al.*, 1981). Higher inflation pressures (40 and 100 mm Hg) were used by Gaffney and his colleagues (1981) and they also found an increase in forearm blood flow but this was accompanied by an increase in peripheral resistance. We have found a reduction in forearm blood flow at MAST pressures of 20, 40 and 60 mm Hg with a concomitant increase in peripheral resistance (Table 1). All the effects were maintained for as long as the trousers were inflated.

Table 1 Effect of MAST inflated to pressures of 20, 40 and 60 mm Hg (same pressure in all compartments) on mean arterial blood pressure, forearm blood flow and peripheral resistance in uninjured subjects.

	MAST Pressure (mm Hg)			
	0	20	40	60
A Mean arterial blood pressure (mm Hg)	86.6 ± 3.2 (7)	89.6 ± 4.1 (7)	92.4 ± 3.4* (7)	95.6 ± 2.7* (7)
B Forearm blood flow (ml min ⁻¹ 100 g tissue)	7.1 ± 0.8 (11)	6.5 ± 0.9 (11)	6.0 ± 0.8** (11)	5.5 ± 0.8** (11)
C Peripheral resistance (A/B)	16.9 ± 1.6 (7)	20.7 ± 2.4 (7)	21.9 ± 2.1* (7)	24.0 ± 3.5* (7)

Results expressed as mean ± sem; number of subjects shown in parentheses

Blood pressure and flow were measured 10 min after the MAST had been inflated to the pressure indicated

Blood flow was measured with a mercury in silastic strain-gauge.

* Significantly different from '0' value at $p < 0.05$ (Wilcoxon signed rank test)

** Significantly different from '0' value at $p < 0.01$ (Wilcoxon signed rank test)

MECHANISMS OF ACTION

Tamponade

Gardner and Storer (1966) showed that applying low abdominal pressure (40 mm Hg) reduced bleeding from a cut aorta in anaesthetized dogs. The Law of Laplace states that $T = P \times R$ (where T is the tension in the wall, P is the difference in hydrostatic pressure across the vessel wall and R is the radius of the lumen). When the normally low extra-luminal tissue pressure is increased by circumferential pneumatic compression the effective intra-luminal pressure is decreased correspondingly even though the actual blood pressure remains the same. Of greater importance is the fact that the pneumatic counterpressure is circumferential, tending to reduce the size of the lumen. Therefore, in the equation, P and R are reduced and hence T is reduced. T , the tension in the wall of the vessel, is tangential and so will tend to keep a hole open. If T is reduced then the size of the defect will be reduced thus slowing the blood loss. Wangenstein *et al.* (1968c) confirmed this by showing that, in anaesthetized dogs, application of an abdominal counterpressure of 40 mm Hg was transmitted to the retroperitoneal space, resulting in an increase in arterial and venous pressure. The vessel radius and flow were decreased in the area under pressure. This effect explains how low MAST pressures can lead to elevation of blood pressure by reducing bleeding from those areas directly compressed. It fails to explain how MAST can work when bleeding occurs in areas outside that covered by the trousers, e.g. supradiaphragmatic haemorrhage (Lilja *et al.*, 1975).

Splinting

It is well known that part of the shock effect of a fracture can be prevented by splinting the fractured part. There is no doubt that MAST immobilizes the area they cover and this must have some effect in attenuating the shock state in those patients with fractures of the femur and pelvis.

Nociceptive Stimuli

It is known that pain can lead to an elevation of blood pressure. Personal experience would indicate that, in the conscious subject, MAST pressures above 40 mm Hg for any length of time are extremely uncomfortable and this stimulus may well exert a pressor effect. This nociceptive stimulation by MAST may explain the peripheral vasoconstriction found in the forearm (Table 1). Also, as will be discussed in more detail below, inflation pressures lower than systolic pressure can markedly impair blood flow beneath MAST. This could lead to tissue ischaemia and it is well known that muscle ischaemia is a potent pressor stimulus (e.g. Alam & Smirk, 1938). Perhaps rather surprisingly we have not been able to show, in normal subjects, any increase in circulating catecholamine levels after MAST inflation (Table 2).

Table 2 Effect of MAST (limb compartments 60 mm Hg and abdominal compartment 40 mm Hg for 30 minutes) on plasma catecholamine concentrations in five normal subjects.

	Noradrenaline (nmol/l)	Adrenaline
Before MAST inflation	1.15 ± 0.11	0.16 ± 0.07
30 min after MAST inflation	1.29 ± 0.17	0.36 ± 0.16

Results expressed as mean ± SE

There is another interesting link between nociceptive stimuli from the compressed tissues and the persistent rise in blood pressure. The pressor response to MAST inflation is sometimes, but by no means always, accompanied by an increase in heart rate. Normally one would expect the rise in blood pressure to be buffered by a baroreceptor reflex mediated bradycardia. Thus the normal baroreceptor reflex may be inhibited during MAST inflation and it is perhaps relevant that such an inhibition has been shown during limb ischaemia in the rat (Little, 1981; Redfern *et al.*, 1980) and in man (Little *et al.*, 1983).

Redistribution of Blood Volume

There has been considerable work on this aspect of MAST physiology (e.g. Ferrario *et al.*, 1970). Does MAST cause a redistribution or ‘transfusion’ of blood from the area

under the trousers to the area above and is this effect big enough to cause a rise in blood pressure of its own accord? McSwain (1980) considers that the trousers work by squeezing blood from the legs centrally. How big an effect this is in the shocked patient is difficult to assess because one would expect that the normal homeostatic response to hypovolaemia would have already redistributed as much blood as possible centrally, by peripheral vasoconstriction, shutting down muscle blood flow, and reduction of capacitance (venous) vascular volume.

Many workers have shown an increase in blood flow to organs above the area of compression. Bondurant *et al.* (1957) showed a central and pulmonary vascular engorgement by means of radio-active scanning and sequential radiology of lungs following rapid inflation of the G-suit. An increase in the blood content of the pulmonary vascular bed might be expected to increase the diffusing capacity of the lung (Cotes, 1979) but we have been unable to demonstrate such a change (Table 3). Roth and Rutherford (1971) using radionuclide labelled microspheres in dogs showed enhancement of flow in organs above the MAST suit. Tenny & Honig (1955) calculated a shift of 250 ml of blood in normal subjects at 40 mm Hg inflation by measuring displacement of the centre of gravity in subjects balanced on a teeterboard. There have been other assessments of the amount of fluid that can be returned to the central circulation. McSwain (1976) and Wilder & Barber (1979) claim, without evidence, a transfusion of 750–1000 ml. The Committee on Trauma at the American College of Surgeons suggest that a 2–2.5 litre transfusion is possible! Gaffney *et al.* (1981) have shown that passive leg raising does not produce a significant or sustained 'autotransfusion' effect and they suggest that, at rest, there is very little displaceable blood in the legs.

Table 3 Effect of MAST inflation (limb compartments 60 mm Hg, abdominal compartment 40 mm Hg) on pulmonary diffusing capacity (transfer factor— T_{L}) for carbon monoxide.

Subject	T_L (ml CO min ⁻¹ mm Hg ⁻¹)	
	Initial	+ MAST
RAL	31.3	32.0
KF	36.4	34.4
SP	24.9	26.9
MA	35.1	32.7
MPY	39.2	40.4
MP	37.2	35.1

Results are the mean values of 2–3 readings. The coefficient of variation for measurements of T_L using the single breath method = 8.7%.

T_L was measured at approximately 2, 15 and 28 minutes after inflation of MAST.

Evidence of a shift of fluid from the area beneath the trousers to that above as shown by increases in organ flow, may not mean that fluid is being pushed back centrally but that cardiac output is being redistributed because flow to the area beneath the suit has been reduced or halted.

However, if a patient is hypovolaemic, even a small transfusion may be significant.

Autotransfusion

Hypovolaemia is compensated not only by a reduction in intravascular capacity and a redistribution of the remaining blood volume but also by the movement of tissue fluid into the circulation (Öberg, 1964). The magnitude of this autotransfusion is determined by the balance of forces across the microvascular endothelium first described by Starling (1896) and later amplified by Landis & Pappenheimer (1963).

It can be calculated that a net change in pressure across the vessel walls of only 2 mm Hg can lead to an autotransfusion of 400–500 ml in 30 minutes (Table 4). If the application of MAST raises tissue pressure sufficiently to favour the net movement of fluid into the circulation one would expect to see a fall in haematocrit. We have shown a fall in haematocrit in seven out of 10 normal subjects at a MAST pressure in all compartments of 15–20 mm Hg. This change may be due to posture and, indeed, three out of five subjects showed a similar fall in haematocrit after lying supine without the MAST! The haemodilution was prevented at MAST pressures above 20 mm Hg. Ashton (1966) showed, using a mercury strain gauge under a Jobst inflatable leg splint, that flow in the limbs of six out of 15 normal subjects could be abolished at pressures of 40 mm Hg and reduced by more than 50% in another seven cases. This was confirmed by later work (Ashton, 1975) where, at tissue pressures around 40 mm Hg, in subjects with a normal blood pressure, muscle blood flow showed a sharp decline or even complete cessation. Ashton considers that this is due to active closure of arterioles and passive collapse of capillaries. A less dramatic but still very significant reduction in flow has been demonstrated using a ¹³³Xe clearance technique in skeletal muscle (60% reduction) and subcutaneous adipose tissue (80% reduction) following inflation of a whole leg cuff to 60 mm Hg (Nielsen, 1982). We have been able to demonstrate using a Doppler probe placed over the posterior-tibial artery that arterial blood flow in the lower limb can be halted at MAST pressures below systolic pressures, and that the lower the subjects' arterial blood pressure the lower the pressure at which occlusion occurred. If tissue blood flow is markedly reduced or even stopped at such low inflation pressures in normotensive subjects then the possibility of occluding flow must be even greater in the hypotensive patient. There can, of course, be no autotransfusion of tissue fluid if there is no blood flow through the tissues. It would, therefore, seem very unlikely that at high MAST pressures

Table 4

Net change in pressure balance across microvascular endothelium	= 2 mm Hg
Capillary filtration coefficient	= 0.7 ml min ⁻¹ mm Hg ⁻¹ kg muscle ⁻¹
∴ Fluid movement	= 1.4 ml min ⁻¹ kg muscle ⁻¹
	= 42 ml kg muscle ⁻¹ in 30 minutes
Assume subjects weight 70 kg i.e. 28 kg muscle	
MAST encloses 40% muscle mass = 11.2 kg	
∴ Total fluid movement	= 470 ml in 30 minutes

autotransfusion is part of their action. Their main effect is probably the redistribution of the blood from the compressed tissues as the suit is inflated and once fully inflated the reduced intravascular volume in the lower body helps cardiac output to be redirected to vital organs such as the brain. The pressor action of MAST may then be reinforced by nociceptive stimuli from the ischaemic tissues under the suit.

Fig. 1 Effect of increasing pressure (mm Hg) in one limb compartment of MAST on flow in the enclosed limb assessed by the amplitude of the recording of a Doppler probe (Parks dual frequency bi-directional Doppler, Model 1010, Oregon, USA) placed over the posterior tibial artery in four subjects with different resting blood pressures ($\square$ 98/55, $\triangle$ 130/70, $\blacktriangle$ 180/90, $\blacksquare$ 230/110).

There is a situation in which blood flow could be occluded even at low inflation pressures. It has been shown that if a MAST suit is inflated to 30 mm Hg on a dummy at ground level in a helicopter the pressure increased to 80–90 mm Hg at an altitude of 9500 feet (Sanders & Meislin, 1983).

ADVERSE EFFECTS

Ischaemic Damage to Lower Limbs

The fact that blood supply to the lower limbs can be halted at MAST pressures considerably below systolic pressure (e.g. Fig. 1), raises the possibility of ischaemic tissue damage. We have not been able to detect an increase in plasma lactate concentration after release of MAST (inflated to 40 mm Hg abdomen and 60 mm Hg limbs for 30 minutes) and it is known that tourniquets can be applied for one–two hours without untoward effects (e.g. during orthopaedic operations, Klenerman *et al.*, 1980). The danger of

ischaemic damage may occur with longer periods of inflation. It is worth noting that the only successful prolonged applications of MAST (up to 72 h) have been at pressures below 30 mm Hg (Gardner & Storer, 1966; Lewis *et al.*, 1973; Pelligra & Sandberg, 1979) when the risk of ischaemia is slight, flow possibly being reduced but not halted even to the skin (Halperin *et al.*, 1948). The beneficial effect of MAST in these studies was probably one of tamponade as all the cases were of abdominal haemorrhage.

There is some clinical evidence that ischaemic damage can occur. Durand *et al.* (1982) advise care with pressure areas under the trousers as bullae and superficial skin necrosis may occur. Maull *et al.* (1981) report limb loss following MAST application although their cases all had compartment syndrome secondary to comminuted lower limb fractures and all occurred after prolonged MAST inflation. Bass *et al.* (1983) have reported thigh compartment syndrome in two patients who did not have lower extremity trauma. However, both the patients had the trousers on for a prolonged period of time—12 and 24 h respectively.

Effect on Renal Blood Flow

Laughlin *et al.* (1980) have shown with radionuclide labelled microspheres and a doppler probe over the renal arteries in miniature swine, that the application of a G-suit decreases renal blood flow and that the effect continued for some time after the suit was deflated. Shenasky & Gillenwater (1972) showed that in anaesthetized dogs a G-suit pressure of 30 mm Hg was easily transmitted to the retroperitoneum and that this caused a decrease in renal perfusion and urine output. This work raises the possibility of adverse renal affects with MAST even at low pressures.

Respiration

Espinosa & Updegrove (1970) showed that application of the G-suit in hypovolaemic patients, resulted in an increase in respiratory rate and a 17% fall in vital capacity. Ransom & McSwain (1978) concluded that although mechanical pulmonary function

Table 5 Effect of MAST on ventilatory capacity in nine uninjured subjects.

MAST pressure (mm Hg)	Forced expired volume (1) (FEV ₁)	Forced vital capacity (FVC)
0	3.81 ± 0.24	4.67 ± 0.33
20	3.66 ± 0.22	4.54 ± 0.31**
40	3.51 ± 0.20***	4.41 ± 0.29*
60	3.26 ± 0.26**	4.03 ± 0.37**

FEV_{T41} and FVC measured using a wedge spirometer (Vitalograph, Buckingham, U.K.). Results expressed as mean ± sem

* Significantly different from 'O' value at p < 0.05 (paired 't' test)

** Significantly different from 'O' value at p < 0.02 (paired 't' test)

*** Significantly different from 'O' value at p < 0.01 (paired 't' test)

may be affected, alveolar inflation was not impaired. Our own experience indicates that any effect on respiration is dependent upon the proximity of MAST to the costal margin. A person of small stature experiencing more respiratory embarrassment than a subject of larger size although in all subjects there was a reduction in forced expired volume (FEV₁) and forced vital capacity (FVC) at MAST inflation pressures of 40 and 60 mm (equal pressures in all compartments) (Table 5). It has recently been demonstrated that an increase in the size of the abdominal compartment leads to greater reduction in vital capacity than the standard MAST suit inflated to the same pressure (100 mm Hg) (McCabe *et al.*, 1983).

Acid/Base Balance

Wangensteen *et al.* (1968a) showed that, in hypovolaemic dogs metabolic acidosis developed after inflation of the G-suit unless the dog was ventilated. Unventilated dogs showed a significantly reduced mean survival time. Their conclusion was that the G-suit may impair perfusion to the lower limbs at pressures considerably below systolic blood pressure. Ransom & McSwain (1979) confirmed this finding, again in dogs, and noted that the metabolic response was greater if MAST pressure was greater than systolic pressure. We have been unable to show a similar response in human volunteers perhaps because they found high MAST pressures intolerable for the length of time required.

Other Effects

It has been reported that MAST inflation can induce vomiting, micturition and defaecation (Civetta *et al.* 1976; Silverston, 1980). They and others also point out that examination of parts covered by the trousers is difficult while they are inflated. This has been partly resolved by the development of transparent MAST although palpation of areas beneath the suit is still impossible.

The other danger of MAST is rapid decompression. McSwain (1980) recommends slow deflation over 30 minutes, transfusing fluid at the same time to compensate for the large increase in available vascular space caused by the reactive hyperaemia.

CONCLUSIONS

- MAST are effective in elevating blood pressure in various types of shock.
- In addition to tamponade and splinting (especially of the pelvis), they work on the redistribution of cardiac output by reducing or occluding input to the lower limbs. There is no evidence for an autotransfusion of tissue fluid even at low inflation pressures, indeed the haemodilution which occurs in the supine position is prevented at MAST pressures above 20 mm Hg.
- Blood flow to the lower limbs is occluded at pressures below systolic blood pressure for that subject. There is thus little point in inflating MAST to their full extent as

recommended to American paramedical services (Committee on Trauma, American College of Surgeons, 1977). A much lower pressure is quite sufficient in a shocked patient because once occlusion has occurred MAST can have no other effect.

- Because of the risks of ischaemia even at pressures below systolic, prolonged inflation is not recommended except when all that is required is the very low pressure tamponade effect, i.e. below 30 mm Hg.
- MAST are no substitute for transfusion. They are a first aid measure and should be reserved for patient transfer and specific medical indications, e.g. hypocoagulation states.

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REFERENCES

- Alam M. & Smirk F. H. (1938) Observations in man concerning the effects of different types of sensory stimulation upon the blood pressure. *Clinical Science* 3, 253–8.
- Ashton H. (1966) The effect of inflatable plastic splints on blood flow. *British Medical Journal* 2, 1427–30.
- Ashton H. (1975) The effect of tissue pressure on blood flow. *Clinical Orthopaedics* 113, 15–26.
- Barclay S., Bennett E. P. & Mehta N. (1981) The effect of lower body positive pressure on forearm and total vascular resistance. *Clinical Science* 62, 26.
- Bass R. R., Alison E. J., Reines H. O., Yeagar J. C. & Pryor W. H. (1983) Thigh compartment syndrome without lower extremity trauma following application of pneumatic anti-shock trousers. *Annals of Emergency Medicine* 12, 6, 382–387.
- Bondurant S., Hickman J. B. & Isley J. K. (1957) Pulmonary and circulatory effects of acute pulmonary vascular engorgement in normal subjects. *Journal of Clinical Investigation* 39, 59–66.
- Civetta J. M., Nussenfeld S. R., Rowe T. R., Hirschman J. C., McCullough K. E., Nagel E. L., Kaplan B. H. & Pettyjohn F. S. (1976) Pre-hospital use of the Military Anti-Shock Trouser (MAST). *Journal of the American College of Emergency Physicians* 5, 8, 581–7.
- Committee on Trauma, American College of Surgeons (1977) Essential equipment for ambulances. *Bulletin of American College of Surgeons*, pp. 7–12.
- Cotes J. E. (1979) Lung Function, *Assessment and Application in Medicine*. Oxford, Blackwell Scientific Publications.
- Cram A. E., Davis J. W., Kealey G. P. & Kassell N. F. (1980) Effects of Pneumatic Antishock Trousers on Canine Intracranial Pressure. *Annals of Emergency Medicine* 10, 1, 28–31.
- Crile G. W. (1903) Blood pressure in surgery, *An experimental and clinical research*. Philadelphia, J. B. Lippincot & Co.
- Crile G. W. (1909) Haemorrhage and transfusion, *An experimental and clinical research*. New York & London, D. Appleton & Co.
- Cutler B. S. & Daggett W. M. (1974) Application of the 'G-suit' to the control of haemorrhage in massive trauma. *Annals of Surgery* 173, 4, 511–14.

- Durand L. S., Thacker J., Rockwell D. & Edlich R. F. (1982) Pneumatic counter-pressure devices: how they work, when to use them. *Virginia Medical* **109**, 388–91.
- Espinosa M. H. & Updegrove J. H. (1970) Clinical experience with the G-suit. *Archives of Surgery* **101**, 36–9.
- Ferrario C. M., Nadzam G., Fernandez L. A. & Gardner W. J. (1970) Effects of pneumatic compression on the cardiovascular dynamics in the dog after haemorrhage. *Aerospace Medicine* **41**, 4, 411–15.
- Gaffney F. A., Thal E. R., Taylor W. F., Bastian B. C., Weigelt J. A., Atkins J. M. & Blomqvist C. G. (1981) Haemodynamic effects of Medical Anti-Shock Trousers (MAST garment). *The Journal of Trauma* **21**, 11, 931–6.
- Gardner W. J. (1969) Hemostasis by pneumatic compression. *The American Surgeon* **35**, 9, 635–7.
- Gardner W. J. & Dohn D. F. (1956) The antigravity suit (G-suit) in surgery. Control of blood pressure in the sitting position and in hypotensive anaesthesia. *Journal of American Medical Association* **162**, 274–6.
- Gardner W. J. & Storer J. (1966) The use of the G-suit in control of intra-abdominal bleeding. *Surgery, Gynaecology & Obstetrics* **123**, 792–8.
- Gardner W. J., Taylor H. P. & Dohn D. F. (1958) Acute blood loss requiring fifty-eight transfusions. The use of antigravity suit as aid in postpartum intra-abdominal haemorrhage. *Journal of American Medical Association* **167**, 8, 985–6.
- Gray S., Shaver J. A., Kroetz F. W. & Leonard J. J. (1969) Acute and prolonged effects of G-suit inflation on cardiovascular dynamics. *Aerospace Medicine* **40**, 1, 40–3.
- Halperin M. H., Friedland C. K. & Wilkins R. W. (1948) The effect of local compression upon blood flow in the extremities of man. *American Heart Journal* **35**, 221–37.
- Kaplan B. C., Civetta J. M., Nagel E. L., Nussenfeld S. R. & Hirschman J. C. (1973) The Military anti-shock trouser in civilian pre-hospital emergency care. *Journal of Trauma* **13**, 10, 843–848.
- Klenerman L., Biswas M., Hulands G. H. & Rhodes A. M. (1980) Systemic and local effects of the application of a tourniquet. *The Journal of Bone and Joint Surgery* **62B**, 385–8.
- Landis E. M. & Pappenheimer J. R. (1963) Exchange of substances through the capillary walls. In *Handbook of Physiology*, Section 2: Circulation II, p. 961. Washington, American Physiological Society.
- Laughlin M. H., Witt W. M. & Whittaker R. N. (1980) Renal blood flow in miniature swine during + G stress and anti-G-suit inflation. *Journal of Applied Physiology: Respiration & Environmental Exercise Physiology* **49**, 3, 471–5.
- Lewis D. G., Mackenzie A. & McNeill I. F. (1973) The use of the G-suit in the control of bleeding arising from hypocoagulation. *Annals of the Royal College of Surgeons of England* **52**, 53–8.
- Lilja G. P., Batalden D. J., Adams B. E., Long R. S. & Ruiz E. (1975) Value of the counterpressure suit (MAST) in pre-hospital care. *Minnesota Medicine* **58**, 540–3.
- Little R. A. (1981) Cardiovascular reflexes after injury. In *Advances in Physiological Sciences*, Homeostasis in injury and shock. Zs. Biro, A. G. B. Kovach, J. J. Spitzer & H. B. Stoner (eds) **26**, 93–7.
- Little R. A., Randall P., Redfern W. S., Stoner H. B. & Marshall H. W. (1983) *Components of injury affecting cardiovascular reflexes in man and rat*. (Submitted for publication.)
- Ludewig R. M. & Wangenstein S. L. (1969a) Effect of external counter pressure on venous bleeding. *Surgery* **66**, 3, 515–20.
- Ludewig R. M. & Wangenstein S. L. (1969b) Aortic bleeding and the effect of external counterpressure. *Surgery, Gynaecology & Obstetrics* **128**, 252–8.
- Mahoney B. D. & Mirik M. J. (1983) Efficacy of pneumatic trousers in refractory prehospital cardiopulmonary arrest. *Annals of Emergency Medicine* **12**, 1, 8–12.
- Maull K. I., Capehart J. E. & Cardea J. A. (1981) Limb loss following MAST application. *Journal of Trauma* **21**, 60–2.
- McCabe J. B., Seidel D. R. & Jagger J. A. (1983) Antishock trouser inflation and pulmonary vital capacity. *Annals of Emergency Medicine* **12**, 5, 290–3.
- McLaughlin A. P., McCullough D. L., Kerr W. S. & Darling R. C. (1972) The use of external counterpressure (G-suit) in the management of traumatic retroperitoneal haemorrhage. *Journal of Urology* **107**, 940–4.
- McSwain N. E. (1976) G-suits and shock. A non-invasive transfusion technique. *Journal of the Kansas Medical Society* **77**, 438–41.
- McSwain N. E. (1977) Pneumatic trousers and the management of shock. *The Journal of Trauma* **17**, 9, 719–24.
- McSwain N. E. (1980) Pneumatic Anti-shock Trousers. *Current Concepts in Trauma Care*, pp. 8–15.
- Nielsen H. V. (1982) Effects of externally applied compression on blood flow in subcutaneous and muscle tissue in the human supine leg. *Clinical Physiology* **2**, 447–57.

- Öberg B. (1964) Effects of cardiovascular reflexes on net capillary fluid transfer. *Acta Physiologica Scandinavica* **62**, Supplement 229.
- Pelligra R. & Sandberg E. C. (1979) Control of intractable abdominal bleeding by external counterpressure. *Journal of the American Medical Association* **241**, 7, 708–13.
- Ransom K. & McSwain N. E. (1978) Respiratory function following application of MAST trousers. *Journal of the American College of Emergency Physicians* **7**, 8, 297–9.
- Ransom K. J. & McSwain N. E. (1979) Metabolic acidosis with pneumatic trousers in hypovolaemic dogs. *Journal of the College of Emergency Physicians* **8**, 5, 184–7.
- Redfern W. S., Little R. A. & Stoner H. B. (1981) Resetting of the baroreceptor heart rate reflex in the unanaesthetised rat during and following a period of hind-limb ischaemia. In *Advances in Physiological Sciences*, Homeostasis in injury and shock. Zs. Biro, A. G. B. Kovach, J. J. Spitzer & H. B. Stoner (eds) **26**, 235–6.
- Rosenhamer G. & Thorstrand C. (1973) Effect of G-suit in treatment of postural hypotension. *Acta Medica Scandinavica* **193**, 277–80.
- Roth J. A. & Rutherford R. B. (1971) Regional blood flow effects of G-suit application during haemorrhagic shock. *Surgery, Gynaecology & Obstetrics* **133**, 637–43.
- Sanders A. B. & Meislin H. W. (1983) Effect of altitude change on MAST suit pressure. *Annals of Emergency Medicine* **12** (3) 140–4.
- Shenasky J. H. & Gillenwater J. Y. (1972) The renal haemodynamic and functional effects of external counterpressure. *Surgery, Gynaecology & Obstetrics* **134**, 253–8.
- Silveston P. P. (1980) The MAST suit. *Ambulance Journal*, July 1980, 153–60.
- Starling E. H. (1896) On the absorption of fluid from the connective tissue spaces. *Journal of Physiology* (London) **19**, 312–26.
- Tenny S. M. & Honig C. R. (1955) The effect of the anti-G-suit on the ballistocardiogram. *Journal of Aviation Medicine* **26**, 194–9.
- Wangensteen S. L., deHoll J. D., Ludewig R. M. & Madden J. J. (1968a) The detrimental effect of the G-suit in haemorrhagic shock. *Annals of Surgery* **170** (2), 187–92.
- Wangensteen S. L., Ludewig R. M., Cox J. M. & Lynk J. N. (1968b) The effect of external counterpressure on arterial bleeding. *Surgery* **64**, 5, 922–7.
- Wangensteen S. L., Ludewig R. M. & Eddy D. M. (1968c) The effect of external counterpressure on the intact circulation. *Surgery, Gynaecology & Obstetrics* **127**, 253–8.
- Wasserberger J., Balasubramaniam S. & Ordog G. (1981) Pneumatic anti-shock trousers: a widening spectrum of new uses. *E.R. Reports* **2**, 23, 105–10.
- Wayne M. A. (1978) The MAST suit in the treatment of cardiogenic shock. *Journal of the American College of Emergency Physicians* **7**, 3, 107–9.
- Wilder R. J. & Barber M. R. (1979) MAST pants—a successful adjunct for the critically injured patient. *Oklahoma State Medical Association Journal* **72**, 199–201.

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