The Detrimental Effect of the G-Suit in Hemorrhagic Shock

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IN 1903, Crile described the use of a pessurized suit to control hypotension associated with blood loss during major surgical procedures.[1] More recently Gardner and Storer reported that the application of external counterpressure in the range of 20–40 mm. Hg by a modified G-suit reduces serious blood loss and controls hypotension.[2] Previous G-suit studies from this laboratory indicate no noticeable harmful effects on the intact circulation when used for a period of 3 hours.[7] Further, the G-suit prolonged survival in dogs with aortic lacerations [3] and decreased the amount of blood loss in animals with lacerations in smaller sized arteries and veins.[4, 8] The purpose of the present study was to assess the effects of external counterpressure applied by a modified G-suit in dogs subjected to hemorrhagic shock after hemorrhage was controlled.

Materials and Methods

Fifteen splenectomized mongrel dogs weighing 10 to 18 kilograms were anesthetized with sodium pentobarbital 25–30 mg. per Kg. intravenously. Two weeks recovery was allowed after splenectomy. Total blood volume was measured at the beginning of each experiment with radioactive iodinated serum albumin (RISA) and a Picker Hemolitre apparatus. Hemorrhagic shock was produced by bleeding 30 per cent of the measured blood volume in a 5-minute period from a single common carotid artery.

The studies were divided into three groups.

Group I consisted of five dogs subjected to hemorrhagic shock only and served as controls. Spontaneous respiration was allowed.

Group II consisted of five dogs subjected to hemorrhagic shock followed by the application of 40 mm. Hg external counterpressure with a modified G-suit immediately upon completion of bleeding. The G-suit was applied for 4 hours and was then deflated. Spontaneous breathing was also allowed in this group.

Group III consisted of five dogs subjected to hemorrhagic shock and 4 hours of G-suit treatment. In this group, respiration was assisted with a mechanical ventilator.

In all animals, data were obtained on cardiac output, arterial blood pressure, stroke volume, central venous pressure, total peripheral vascular resistance, blood gases, blood pH, blood lactates and pyruvates. All data were obtained before hemorrhage, immediately after hemorrhage, and at repeated intervals for a 5-hour period.

Cardiac output was measured by the dye dilution technic using indocyanine green and a Gilford densitometer. Arterial blood

Submitted for publication December 12, 1968.

This investigation was supported by the U. S. Army Medical Research and Development Command, Department of the Army, under Research Contract 17-67-C-7039 and Grant HE-02038 from the National Institutes of Health.

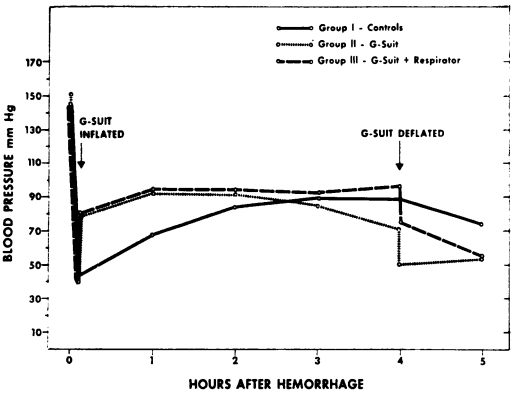

FIG. 1. Mean arterial blood pressure data.

pressure was measured through a polyethylene catheter passed from the carotid artery to the aortic arch. Central venous pressures were measured from catheters directed into the right atrium via the jugular vein. The pressures were recorded on a multichannel recorder from electronic transducers. Total peripheral vascular resistance (TPR) was calculated from arterial pressure, central venous pressure, and cardiac output values and was expressed in arbitrary units (PRU units). Stroke volume was calculated from cardiac output and heart rate. Blood gases (P_{O_2} and P_{CO_2}) and pH were determined on a Beckman model 160 gas analyzer. Lactates and pyruvates were determined by the enzymatic spectrophotometric method of Rosenberg and Rush and were expressed as lactate /pyruvates ratios.[5]

The pressurized garments used were circumferential plastic bladders which extended from the xiphoid process to the knee of the hind leg of the dog. The suit was inflated with compressed air and equipped with an exhaust valve which supported a column of water equivalent to 40 mm. Hg.

Results

All results are summarized in Figures 1–7.

Group I. The mean arterial blood pressure of the control dogs was 145 mm. Hg before bleeding began. Immediately after bleeding, the mean blood pressure was 43 mm. Hg and then gradually rose to a maximum value of 89 mm. Hg at 3 hours. During the next 2 hours, the blood pressure declined to a mean value of 74 mm. Hg 5 hours after hemorrhage. The average cardiac output prior to bleeding was 1.15 liters/min., and fell to 0.25 liters/min. immediately after bleeding. Cardiac output reached a maximum of 0.6 liters/min. 2 hours after bleeding and then declined to 0.39 liters/min. at 5 hours. The average initial calculated total peripheral vascular resistance (TPR) was 7.5 PRU and rose to 10.25 PRU after bleeding. The TPR fluctuated for the next three hours and reached a maximum of 11.75 PRU at 4 hours.

Arterial oxygen tension declined from a pre-bleeding value of 83 mm. to 72 mm. one hour after bleeding and remained lower than pre-bleeding values for the remainder of the experiment. The arterial carbon dioxide tension also steadily declined from a pre-bleeding value of 33 mm. to 15 mm. five hours after hemorrhage. Similarly the arterial pH declined steadily in control animals from 7.39 to 7.22 at 5 hours. The lactate/pyruvate ratio rose rapidly during the first hours after bleeding and continued at high levels throughout the experiment.

The mean survival time for control dogs was 30 hours (range 12–72 hours).

Group II. The average blood pressure

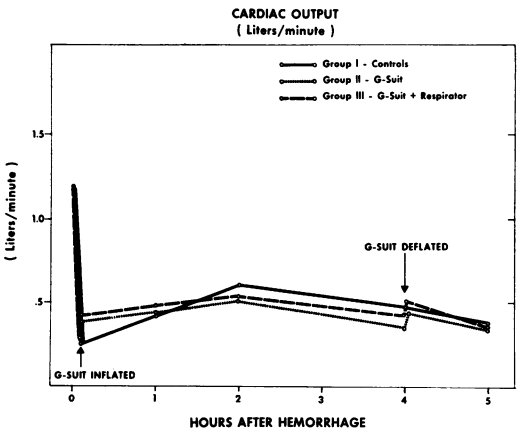

FIG. 2. Cardiac output data.

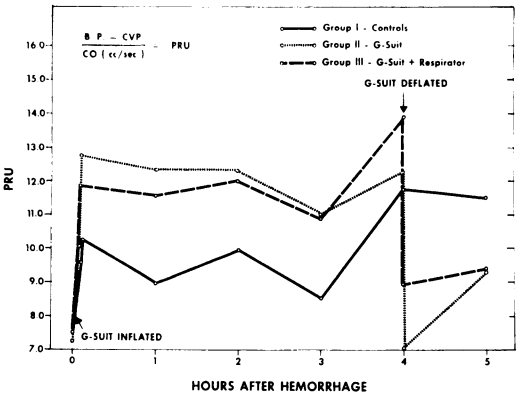

FIG. 3. Calculated total peripheral vascular resistance data.

of these dogs was 151 mm. Hg prior to bleeding and the blood pressure fell to 39 mm. Hg after hemorrhage. Upon inflation of the G-suit, the blood pressure rose immediately to a mean value of 78 mm. Hg and reached a value of 92 mm. Hg one hour after bleeding which was maintained during the second hour. Thereafter, the arterial blood pressure steadily declined and upon G-suit deflation at 4 hours immediately dropped 22 mm. Hg further. The average cardiac output was 1.20 liters/minute before bleeding and the value fell to 0.33 liters/minute after bleeding. With G-suit inflation, the cardiac output increased immediately to 0.47 liters/min., but after 4 hours the cardiac output gradually declined to 0.34 liters/min. Upon G-suit deflation, a rise in cardiac output from 0.34 to 0.45 liters/min. occurred. After hemorrhage, the calculated TPR increased to 12.7 PRU and remained relatively steady at this high value until the G-suit was deflated at 4 hours. Upon suit deflation, the TPR fell to 7.0 units.

The arterial oxygen tension dropped from a mean pre-bleeding value of 81 mm. Hg to 61 mm. Hg in the first hour after bleeding. After two hours, the arterial oxygen tension began to increase but declined again after suit deflation at four hours. The arterial P_{CO_2} declined from a pre-bleeding value of 36 mm. to 21 mm. one hour after bleeding and remained at that level throughout the experiment. The mean arterial pH prior to hemorrhage was 7.35 and exhibited a steady decline to 7.01 at 5 hours. The lactate/pyruvate ratios rose very rapidly in these animals.

The mean survival time in Group II dogs was 11.4 hours (range 3–24 hours).

Group III. In the animals that received external counterpressure and ventilatory assistance the mean arterial blood pressure prior to hemorrhage was 142 mm. Hg. The value fell to 40 mm. Hg with hemorrhage, but rose immediately with G-suit inflation to 80 mm. Hg. Blood pressure remained stable until the G-suit was deflated when a fall in blood pressure occurred. Before bleeding the average cardiac output was 1.15 liters/min. The cardiac output fell to 0.37 liters/min. after hemorrhage and then increased slightly to 0.43 liters/min. with suit inflation. After hemorrhage, the calculated TPR increased to 11.9 PRU and reached a maximum of 13.9 PRU at four hours. With suit deflation, the TPR fell to 8.9 PRU.

The arterial oxygen tension dropped from a pre-bleeding value of 84 mm. to 76 mm.

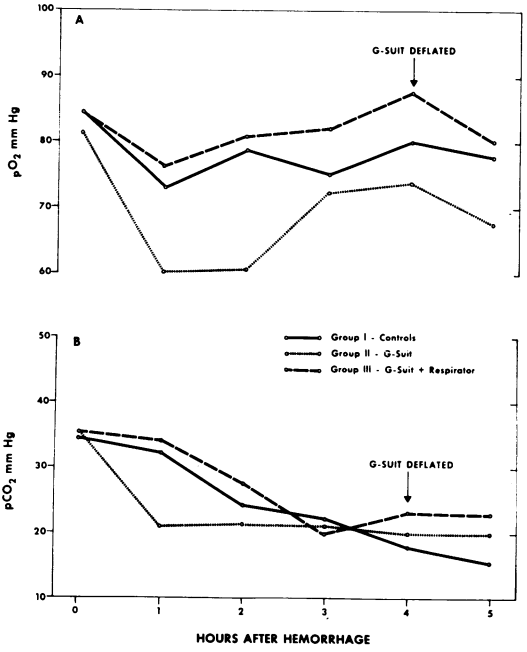

FIG. 4. Blood gas results. A. P_{O_2}, B. P_{CO_2}.

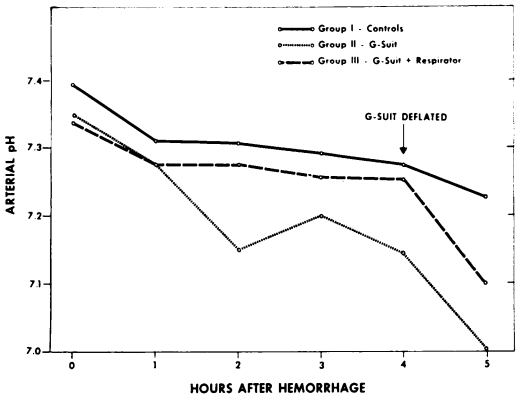

FIG. 5. Arterial pH data.

one hour after bleeding, but then increased to a maximum of 87 mm. at four hours, the time of suit deflation. Arterial P_{CO_2} declined from a pre-bleeding value of 35 mm. to a low of 20 mm. at 3 hours and increased slightly. Arterial pH fell from a pre-bleeding value of 7.33 to 7.27 one hour after hemorrhage and remained relatively stable until suit deflation. After suit deflation, the pH declined further to 7.10. Lactate/pyruvate ratios rose rapidly in the 2-hour period after hemorrhage and then declined until suit deflation when ratios again rose.

The mean survival time after hemorrhage was 15 hours (range from 3–24) in Group III.

With hemorrhage, the cardiac stroke volume fell markedly in all three groups and never returned to pre-bleeding levels. Immediately after G-suit inflation, the stroke volume was higher in the G-suit dogs, but after 2 hours the control animals had higher stroke volume values. The central venous pressure remained at zero throughout the five hour period of monitoring in all three groups.

Discussion

Since it was uncertain at the outset of these experiments whether or not the effects of the G-suit would cause a change in arterial blood pressure in shocked animals, we used a hemorrhage shock preparation that was not pressure dependent for a particular period of time. The hemorrhagic shock preparation chosen was a modification of that described by Swan.[6] In Swan's experience, removal of 40 per cent of the estimated blood volume from dogs in a 5-minute period resulted in approximately a 50 per cent mortality. In our laboratory, removal of 40 per cent of the blood volume measured by RISA in five minutes resulted in rapid death in all animals. We found that removal of the 30 per cent blood volume in 5 minutes gave more prolonged survival in animals without any treatment.

The results indicated the overall effect of the G-suit inflated to 40 mm. Hg pressure for 4 hours in dogs subjected to hemorrhagic shock was detrimental. Dogs in Group II demonstrated an immediate increase in arterial blood pressure, cardiac output, stroke volume, and peripheral vascular resistance with the application of the G-suit. However, after a period of 2 to 3 hours, the values for arterial blood pressure and cardiac output in control animals equalled or exceeded those in G-suit dogs. The dogs subjected to external counterpressure developed a more rapid and severe metabolic acidosis than controls as evidenced by the blood pH, P_{O_2}, P_{CO_2}, and lactate/pyruvate ratio data. Moreover, the survival time in G-suit dogs was considerably less than control dogs ($p < 05$).

Because the G-suit was suspected of

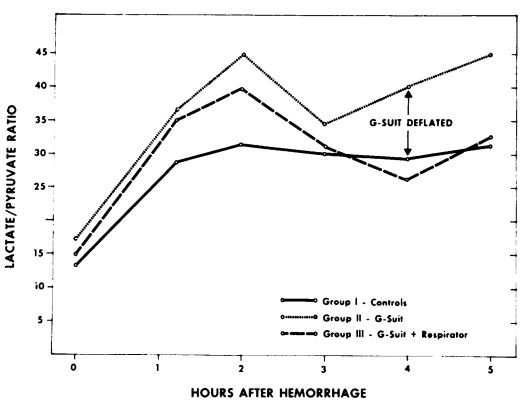

FIG. 6. Blood lactate/pyruvate ratio results.

causing respiratory embarrassment by mechanically interfering with respiratory movement, Group III animals were studied with ventilatory assistance. These animals also exhibited an immediate rise in arterial blood pressure, cardiac output, and TPR with inflation of the pneumatic suit. The arterial blood pH and blood gas pattern resembled more closely those of the control group, indicating a beneficial effect brought about by mechanical ventilation. The marked elevations in lactate/pyruvate ratios, however, coincided more closely with the G-suit dogs in Group II than controls. The mean survival time of the Group III animals was longer than Group II animals but less than controls.

In an attempt to determine a possible regional source contributing to the severe metabolic acidosis in the Group II dogs, two additional dogs were studied with pneumatic suit application and without mechanical ventilation. In these animals multiple venous blood samples were taken from the femoral vein, left renal vein, an hepatic vein, and the right ventricle. Blood pH determinations were performed on the samples and the results are shown in Figure 7. The results indicated that the blood returning from the lower extremities had a lower pH than blood from other sites including the mixed venous blood from the right ventricle and probably represented impaired perfusion to the lower extremities caused by the G-suit. These findings are in keeping with the observation in previous studies with the G-suit on non-shocked animals with an intact circulation that arterial blood flow to the lower extremities was reduced. As seen in Figure 7, it was interesting to note that the renal vein blood had a higher pH than blood obtained from other sites, suggesting some degree of autoregulation.

In previous studies from this laboratory, the ability of external counterpressure to control active hemorrhage was tested in controlled situations. Control dogs with a

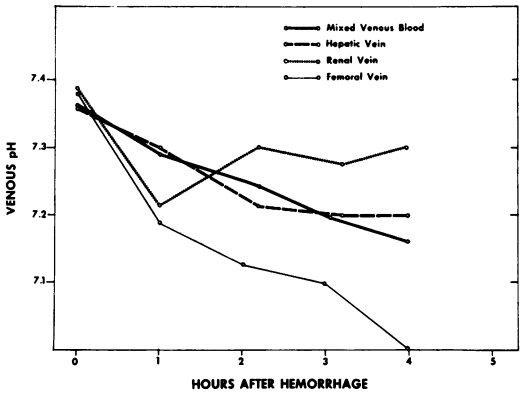

FIG. 7. Venous pH results from multiple venous sampling sites. Average values from two dogs subjected to G-suit without mechanical ventilation.

5 mm. aortic laceration bled rapidly and died within 15 minutes. When 40 mm. Hg G-suit pressure was applied, the blood pressure fell rapidly to 40 mm. Hg and then stabilized. When counterpressure of 80 mm. Hg was applied, blood pressure fell to 80 mm. Hg and stabilized. Bleeding apparently stopped when arterial blood pressure reached the level of applied external pressure or when the transmural pressure (arterial blood pressure minus external pressure) became negligible. Dogs with external counterpressure lived longer than controls.

In another study the effect of 40 mm. Hg external pressure was studied on arterial bleeding from the femoral artery and saphenous artery in dogs. Lacerations 5 mm. and 1 mm. in length were made in each size vessel in different groups of dogs with comparable controls. Before bleeding stopped, the blood pressure of control dogs fell to levels apparently determined by the size of the vessel and the size of the arteriotomy. The blood pressure of dogs with external pressure was approximately 40 mm. Hg higher than that of control dogs when bleeding stopped. Dogs subjected to external pressure bled less and at a slower rate than controls, although the results were of variable significance. There was no difference in total bleeding time between test dogs and controls.

Additional studies were done in dogs in which a 5 mm. longitudinal incision was made in the inferior vena cava in one group and a similar incision was made in the femoral vein of another group. Control dogs were allowed to bleed until hemorrhage stopped spontaneously. External pressure of 40 mm. Hg was applied to test dogs which were also allowed to bleed spontaneously. During hemorrhage, the arterial blood pressure of dogs with counterpressure remained substantially higher than controls, and test dogs bled less than controls. Dogs with femoral vein incisions bled less than dogs with inferior vena cava incisions. External pressure was believed to reduce the effective laceration size, the venous transmural pressure, and local venous blood flow.

In contrast to previous studies which were designed to assess the effects of external pressure on active hemorrhage, the present experiments were designed to assess the effects of external pressure applied by a pressurized suit on hemorrhagic shock after bleeding had stopped. Although external pressure exerts a salutary effect on active hemorrhage by causing a reduction in transmural blood pressure and laceration size, the modality exerted deleterious effects in hemorrhagic shock under the conditions of the experiment described. In these experiments, G-suit applications for a period of four hours accelerated the development of severe metabolic acidosis and led to a decreased survival time.

Summary

The effect of 40 mm. Hg external counterpressure applied by a modified G-suit was studied in dogs subjected to hemorrhagic shock by bleeding 30 per cent of the measured blood volume in 5 minutes. Dogs in Group I served as controls and received no treatment after bleeding. The G-suit was applied to dogs in Group II immediately after bleeding. In Group III, the dogs were mechanically ventilated and also had the G-suit applied.

After inflation of the G-suit in Groups II and III, there was immediate increase in arterial blood pressure, cardiac output, stroke volume, and peripheral vascular resistance. In control dogs, the values for arterial blood pressure and cardiac output equalled or exceeded those in G-suit dogs after a period of 2 to 3 hours. Dogs in Group II developed a more rapid and severe metabolic acidosis than controls. In Group III dogs, the arterial pH and blood gas value patterns resembled more closely those of the control group, indicating a beneficial effect of mechanical ventilation.

The mean survival time of Group II dogs was significantly less than control dogs and the mean survival time of Group III animals was longer than Group II animals but less than controls.

Results of venous pH measurements from blood samples obtained from several sites during G-suit inflation indicated significantly lower values from the lower extremities when compared to values from other sites. The G-suit probably impaired perfusion to the lower extremities.

In these experiments, the application of the G-suit for a period of 4 hours was detrimental.

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