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THE USE OF ANTI-GRAVITY SUITS FOR THE CONTROL OF CRITICAL INTRA-ABDOMINAL HEMORRHAGE

by

Stein Kravik and Knud Landmark

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THE USE OF ANTI-GRAVITY SUITS FOR THE CONTROL OF CRITICAL INTRA-ABDOMINAL HEMORRHAGE

by

Stein Kravik and Knud Landmark

So far the use of a so-called anti-G suit in order to halt difficult /1498* to control intra-abdominal hemorrhage has hardly been accepted and has been very little practiced. The idea is however an old one. Already in 1903, Crile [5] used an inflatable rubber suit to apply pressure from the ankles to the waist. In that manner he was able to prevent hypotension during surgical operations in a sitting position. In 1909, he treated a patient in shock due to hemorrhaging according to the same principle [6]. The suit, which was inflated by means of a bicycle pump, was made of rubber and there were almost always leaks. In addition, blood transfusions soon became available and the method was abandoned.

The expression "anti-G" goes back to the early stages of World War II, when this suit was adopted by American airpilots to prevent hazardous unconsciousness as experienced during sharp turns, e.g., the pull-out from a dive. Under that condition, the pilot is subjected to a so-called "positive /1499 G-acceleration" because the load of the acceleration is added to the force of gravitation. Then the blood collects in the lower extremities with the result that the blood flow to the retina and the brain becomes deficient. The vision becomes fuzzy ("grey-out") and within seconds, there is a real syncope (black-out). Today, the anti-G suit is used by all fighter pilots and does effectively prevent pilot black-outs. A rubber sac, built into the suit, is connected to the airplane system and is automatically inflated during angular acceleration. During level, horizontally linear acceleration the anti-G suit is without effect. The pressure exerted by the suit counteracts the increased gravitation, hence, the term "anti-G" (= gravitation) suit.

* Numbers in the margin indicate pagination in the foreign text.

In this manner the vascular capacity of the lower part of the body is reduced, resulting in a considerably improved cerebral perfusion [7].

At the end of the 1950's, Gardner [11] started to experiment with the suit again and it became the target of a renewed medical interest, independently of the work by Crile 50 years previously. In 1958, Gardner [8] used the anti-G suit to halt a post-partum hemorrhage after all other attempts had failed. It was a case of *placenta percreta*. When the woman was dressed in the suit, she had already received 55 blood transfusions into four veins simultaneously within the course of 18.5 hours, been laparotomized twice, had had a hysterectomy and had had elastic bandages applied to her lower extremities. She was in a definite state of intravascular coagulation. The blood pressure was 80/40 in combination with hemorrhagic shock and her situation was considered hopeless. But only few minutes after applying a pressure by means of the suit at 20 mm Hg, the patient regained consciousness, the blood pressure rose to 114/80 and the diuresis started up again. The hemorrhage stopped gradually in the course of a few hours and the suit could be removed after 24 hours without new hemorrhaging occurring [8].

Later, the anti-G suit has been used in 174 reported cases in order to control intra-abdominal hemorrhage in connection with aneurysms of the aorta, fractures of the pelvis, disseminated intravascular coagulation and other kinds of coagulopathies, military trauma as well as obstetrical and gynecological complications [1, 8, 9, 11]. In the cases when the suit has been applied, the original situation has in general been hopeless and the battle considered just about lost. The anti-G suit has been considered as the ultimate life-saving device when no other alternative was available.

Until the present time, the treatment of orthostatic hypotension has been the most important area for an indication to use it (see also our article in another part of this issue of Tidsskr).

Why has the use of the anti-G suit not received more recognition? The most important cause seems to be that adequate clinical controls are lacking. All the reports are built on the *impression* that the anti-G suit contributed to hemostasis, but it is usually impossible to prove whether other factors

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could have been active. This has led to the efficiency of the suit being met with skepticism, even as an ultimate weapon, when the situation was catastrophic.

From 1909 to 1969, the use of the suit was reported in only five cases compared with 169 cases between 1970 and today. This clearly indicates the increased interest in the suit, which has grown during the last couple of years, first and foremost thanks to the fact that specially trained first-aid personnel in the U.S.A. (so-called "paramedics") have discovered the usefulness of the anti-G suit in acute situations. A commercial version of the suit, made of polyvinyl material, is used to control life-threatening hemorrhages and hypotensive crises, while the patients is on his way to further treatment (9).

Everybody agrees that the capacity of the suit to prevent low blood pressure depends upon its pressure against the skin below the diaphragm. and, thus, on a reduced vascular capacity of this area. There occurs an auto-transfusion from the lower part of the body to the organs above the diaphragm, probably of a magnitude of 750 - 1000 ml. [9, 11, 13].

Clinically, it is possible to observe an improved facial coloration and a distension of the neck veins. The increase in blood pressure is most spectacular in the case of hypovoluminous shock, but can also be observed on normovoluminous patients [12, 13].

Usually, the pressure of the suit is not above 20 - 25 mm Hg and the perfusion of the lower extremities is at all times satisfactory [12, 13]. It is difficult to explain the paradoxical fact that an external pressure against the skin so far below the average blood pressure can control an intra-abdominal hemorrhage both from veins as well as from *arteries*. Two well known physiological laws may offer a plausible explanation:

The law of La Flace $(T = P \cdot R)$, where T is the tension of the wall, P is the difference in pressure between the hydrostatic pressure within the blood vessel and the extravascular pressure, i.e. the transmural pressure, and R is the radius of the vessel) indicates - in a few words - that the tension in the

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wall of a flexible vessel is a function of the transmural pressure and the radius of the vessel. It is first and foremost the mural tension which makes the edges of an intramural lesion spread. The larger the tension, the bigger is the distance between the edges of the wound and the hemorrhage will continue. Because the suit exerts a pressure on the skin, the intraperitoneal pressure increases so that the intramural pressure diminishes. Thereby, the wall tension is reduced and the edges of the wound start to close up [1, 2, 9, 11, 13].

In addition, the pressure of the suit is evenly distributed over the entire abdomen and this leads to a narrowing of the vascular diameter [2, 13]. The reduction in vascular diameter will further contribute to the reduction in mural pressure (La Place's law).

Perhaps even more important is that the perfusion of blood in the area below the diaphragm is fairly much reduced: the law of Poiseuille ,(i.e. $Q = \Delta P r^4 / 8 n$ 1, where Q = perfusion, P = perfusion pressure, r = radius, n = the viscosity of the blood, and l = the length of the vessel) states that even a minor reduction of the vascular radius will lead to a considerably / 1500 reduced perfusion [9].

It seems therefore reasonable that the combined effect of reduced perfusion and mural tension will lead to the coagulation mechanism of the patient himself, although often marginal, being sufficient for taking over and making the bleeding stop [9].

Some authors have reported a reduced kidney function during the use of an anti-G suit, others have indicated an improved or unchanged diuresis. Espinosa [4] measured a reduced vital capacity of about 17% in healthy humans at a suit pressure of 20 mm Hg. Wangensteen [3] observed a serious metabolic acidosis in dogs during hypovoluminous shock at a suit pressure of 40 mm Hg, maintained for more than 4 hours. This observation has not been confirmed by other experiments on animals and never reported in respect to humans. The heart beat frequency slows down, something supposed to depend on the increased aortal pressure and the increased return-flow of venous blood, which can be

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measured by means of baroreceptors at arcus aortae, sinus caroticus, the lungs and on both sides of the heart [9].

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Because of the increased return-flow of venous blood and the consequently increased pressure due to the replenishment of the right atrium, the volume of the beat will increase (Starling's law). As a result of the increased volume of cardiac rhythm, an increased volume per minute would be expected. However, due to the simultaneous reduction in frequency of the heart beats, the volume per minute will usually remain unchanged [9]. The peripheralvascular resistance in the area covered by the suit increases due to the reduced vascular radius, the diminishing perfusion and the increased perfusion pressure. On the other hand, the total vascular resistance is dependent on the volume per minute and the possible distension of the venous vessels above the diaphragm [9]. At a suit pressure of 40 mm Hg, an increase of 20% in perfusion has been observed in *a. carotis* of dogs at the same time as there was a 33% reduction in *a. femoralis* [12].

The side effects connected with the use of the anti-G suit seem under no circumstances to represent a clinical problem. Most observations indicate thr' unfortunate effects increase together with increasing suit pressure and that no advantages are gained by exceeding 30 mm Hg. Perhaps in contrast to what could be expected, there is thus no clinical or theoretical basis for believing that the blood pressure will rise at the same rate as increased suit pressure. This is especially the case of hypovoluminous patients [9].

The anti-G suit should be used with great discretion in the case of patients suffering from basic kidney or lung ailments.

Due to the shunting effect of blood from the lower to the upper parts of the body, the use of the suit is normally contra-indicated in cases of cerebral edema, pulmonary edema, cardiac failure and hemorrhages above the diaphragm. However, McSwain [10] has observed a favorable response in connection with hemorrhaging from *a. pulmonaris*. He adds that this effect improved the oxygenation and the perfusion of the tissues in spite of the fact that, theoretically, there ought to have occurred an increased bleeding from the wound.

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He has also put forward a very interesting hypothesis considering that since intracranial lesions and hypotension give rise to cerebral edema, the use of anti-G suite could in reality reduce cerebral ischemia. The effect of the anti-G suite on hemorrhages above the diaphragm has, however, not been fully explained and here, further clinical tests are necessary.

Acute diffuse erosive hemorrhagic gastritis is an often fatal disease of patients suffering from a physiological stress-condition (e.g. serious burns, etc.). Multiple and diffuse sources of hemorrhages make surgery a doubtful means of treatment. The use of an anti-G suit under such conditions has not been tried. However, before resorting to such a drastic operation as total gastrectomy, there are many possible reasons for trying out this suit.

After having perused some literature regarding the anti-G suit, we feel it is evident that i_t offers a safe, effective and real supplement to existing surgical methods to be used in an acute crisis of hemorrhaging but mainly in first-aid situations. In the latter case, the suit can be inflated by means of an ordinary hand-operated pump and the control of the pressure is not critical. However, a low pressure is definitely preferable [9]. When the suit is used over a prolonged period of time, e.g. in connection with hemorrhagic crises which cannot be controlled surgically, it is necessary to check the suit pressure exactly. In most cases, it is not necessary to exceed a pressure of 20 - 25 mm Hg in order to achieve hemostasis. The pressure is then gradually reduced after about 24 hours, but this must depend on the individual case. It is very important that there is an adequate correction of the fluid/electrolyte relation and that a possible coagulopathy is treated before the suit is depressed [9].

The Equipment Department of the Air Defence at Kjeller has constructed a control unit, enabling an exact setting of the correct suit pressure. This control unit and an anti-G suit are now stored at the Akershus Central Hospital and can, when necessary, be transported by helicopter.

Below follow point-by-point guidelines for the use of the suit [9]:

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- 1) Thorough examination of the skin to be covered by the suit incisions, decubitus.
- 2) Insertion of bladder catheters.
- 3) If possible, mechanical ventilation.
- 4) Maximum suit pressures: $20 25 \text{ mm Hg} = 272 340 \text{ mm H}_20$.
- 5) Duration of pressure: this varies according to the medical literature between 24 and 48 hours.
- 6) To be observed:

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- a) blood pressure, pulse, respiration;
- b) intake of liquids/diuresis;
- c) arterial acid/base status.
- 7) Eefore the pressure is reduced:
 - a) check that there has been adequate fluid/electrolyte correction; IMPORTANT
 - b) check that there is laboratory confirmation in respect to the correction of any possible coagulopathy.
- 8) The suit shall be gradually depressed over a period of 30 60 minutes.

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