

A NEW APPROACH TO THE CONTROL OF SHOCK

ELIZABETH M. MARTIN, M D *

THE solution to the problem of shock may have been within our grasp ever since sympathetic blockade first occurred as a corollary to spinal analgesia. Labat's monograph in 1931 cites the use of spinal analgesia without a pressor drug to combat shock resulting from crushing injury (1). Since the circulatory system is dependent chiefly on gravity in high subarachnoid block, anaemia of the brain is prevented by the Trendelenburg position. In this position it is both neurologically and anatomically impossible for the solution to diffuse to the respiratory centres (1)

We can produce autonomic block with "total spinal," with Hexamethonium compounds and Arfonad®, or with Chlorpromazine. The latter are easy to use, readily transportable and can be administered without further moving of the patient. We have done a large volume of clinical work with "total spinal" induced hypotension, using the technique introduced by Griffiths and Gillies (2, 3). The spinal induced hypotension is capable of blocking the sympathetics, establishing analgesia, giving muscle relaxation and control of haemorrhage by positioning, all in a single manoeuvre. The Hexamethonium technique calls for a relaxant and an anaesthetic drug if reparative surgery is contemplated (25, 26, 27). Chlorpromazine acts as an autonomic blockade, a hypothalamic depressant, and is an excellent pre-medication agent (4, 5, 6, 7)

Immediately following injury the alarm goes out to the circulatory system to preserve the blood volume. Anatomically one thinks of the vascular tree as a single system. Physiologically it immediately becomes two systems (10). a "peripheral" pool which can be spared, and a "central" pool, which must be preserved. After injury, a preferential blood shift takes place, and the peripheral blood pressure drops, as measured over the brachial artery by cuff and manometer. When a total sympathetic block is induced in man the peripheral blood pressure also drops, but for a wholly different reason. That is why one speaks of induced hypotension rather than ganglionic block, although this is only the most spectacular effect. This drop in peripheral pressure is just as capable of killing the patient as trauma or actual blood loss because the body no longer has the ability to decide where the blood is most needed. We must make the decision and can then maintain an adequate blood supply to the vital organs by a complete head-down tilt of the body in a 15-degree Trendelenburg position. If this is not possible, raising the legs above the level of the trunk with the head flat will suffice. In the unconscious intact human with a ganglionic block to the first thoracic nerve root, placed in a 15-degree Trendelenburg position, the systolic pressure is read by oscillation between 90 and 120 mm. Hg. This appears to be irrespective of previous hypertension or hypotension, as we have observed in 680 cases given spinal analgesia without a pressor drug to the level of the first

*Highland Hospital, Rochester, N.Y., U.S.A.

thoracic nerve root. This blood pressure measurement represents the total pressure under block of an apparently normal blood volume. Therefore, in replacing blood for the shocked patient, when the total blocked pressure, which may have been unobtainable, returns to the level of 100 mm. Hg. systolic, the blood volume has been adequately restored.

If a patient has a penetrating abdominal wound with internal bleeding, or one leg nearly torn off with the iliac artery pumping blood up behind the peritoneum, or if a woman has an obstetrical haemorrhage, the wound is central and the bleeding is increased by the peripheral vasoconstriction of the protective mechanism. If such a patient is given 100 mg of Hexamethonium Iodide intravenously, a fairly complete ganglionic block follows in 2-5 minutes. During this time, the bleeding area is elevated to the highest point if possible, while the patient is placed in Trendelenburg position until the pressure can be determined. If it is above 70 mm. Hg, then, while blood is given, it will be safe to reposition the patient in such a way that the operative site takes advantage of the ischemic effect of a posture in the patient with a block. If the total blocked pressure in Trendelenburg position is less than 50 mm Hg, then the arms may also be elevated to take advantage of the blood which has pooled there. Intravenous replacement is made as rapidly as possible until the pressure is read between 50 and 60 mm. Hg. Establish anaesthesia with the agent of choice or availability, giving oxygen wherever possible. We have used Cyclopropane, Nitrous Oxide-Oxygen, and even Thiopentone in very dilute solutions (gm. 1 in 1000 cc normal saline) given cautiously when gas was not available. Ether is contraindicated as it interferes with capillary vasomotion (12).

The surgeon takes advantage of the bloodless field to repair damage while vasodilatation averts anoxia of the vital organs. This technique will delay the onset of irreversible shock and will minimize the sequelae of anoxic tissue damage which are too often the price imposed by nature for survival (21). In the patient whose mechanism has compensated for his shock, a second insult, such as surgery, without the protection afforded by a vasodilated system may throw him into the phase of irreversibility, as in the second bleeding of the experimental animal.

This concept of the treatment and control of shock is supported by evidence of two kinds, that of experimental physiologists who have seen these phenomena for some time without attempting the clinical application, and that of the clinicians who have stumbled upon it, and been too frightened by its complete reversal of all we have previously been taught to put it into operation. Foldes (13) has pointed out that it is almost impossible to bleed a dog with ganglionic block into the state of shock provided the bleeding point does not lie below the level of the heart. It does not matter whether the block is with a spinal agent or a Methonium salt. The most striking manifestations of haemorrhagic shock will actually be inhibited by a total sympathectomy because the usual preferential blood shift to the vital organs no longer occurs (14; 28). Selye further states that, in the dog, Dibenamine decreased the incidence of irreversible shock after extensive haemorrhage or trauma (15). In experiments where various protective measures, such as Dibenamine or sympathectomy, have been introduced the venous outflow from the omentum into the portal system is remarkably well

sustained, even at blood pressures as low as 30 mm Hg. The essential difference between Dibenamine-treated and etherized dogs subjected to shock is the continued operation of the terminal vascular bed as a dynamic functional unit in the Dibenamine-treated animal which is resistant, and the functional deterioration of the peripheral circulation in the etherized animal which readily develops the irreversible type of shock (12). Dibenamine is no longer protective if given after shock has developed. This confines a promising drug to the laboratory. We do have, in its place, Chlorpromazine, which combines an effective adrenergic blockade with an autonomic blockade and a central depressant effect at the level of the hypothalamus. Experimental and clinical work carried out by Laborit and his co-workers in France proved it very effective in the control of shock by a mechanism similar to that of Dibenamine, sympathectomy, total spinal, and Methonium compounds (16, 17, 18, 19). From a neurosurgeon's experience with the central nervous system and shock, as reported by Franklin (20), comes an interesting observation which may shed additional light on the role of Chlorpromazine. Shock was not produced with the removal of, or trauma to the cerebrum as far down as the thalamus, or with increased intracranial pressure. Shock occurred only during operative interference in the region of the optic chiasma with attendant retraction of hypothalamic structures. Manipulation of the brain stem even at the supposed level of the vasomotor centre produced no effect upon blood pressure.

In the early stages of the shock phase, adrenergic peripheral vasoconstriction will expedite the onset of irreversible shock due to the elaboration of Ferritin (V D M.). Following even arterial transfusion, the arterioles become atonic and distended, the capillary circulation remains slow despite blood pressures as high as 100 mm Hg. systolic, and progressive pooling of blood on the venous side of the bed develops. If the transfusion is continued, pulmonary oedema and cardiac dilatation occur without resuscitating the animal (12, 20). This was also seen in battle casualties.

If one can put all of the shocked patient's available blood volume at the service of the vital organs, in such a manner that he can make full use of its oxygen carrying power through a non-resistant vascular bed, whose capillary vasomotion remains undisturbed (29), that patient can make better use of half his original blood volume with the vasodilatation provided by ganglionic blockade than he could with his reduced blood volume in the presence of vasoconstriction (21). Animals treated with Dibenamine can be recovered by replacement of as little as 50 per cent of the total blood withdrawn during haemorrhage (12, 20). By proper positioning we must see that we do not allow the patient to bleed into his own vascular reservoir.

Pitkin has reported two urgent emergencies: the first, a ruptured spleen and the second, a cut bowel with a severed internal iliac artery from a knife wound. Both of these were given high spinal anaesthetics, which he considered life-saving, as they permitted quick control of bleeding, response to intravenous infusions, and immediate surgery (11).

When Hexamethonium Chloride is used clinically, Rollason (22) says, "In the acute head-up position with a 30-40 degree tilt in reverse Trendelenburg position,

the systolic blood pressure should not be allowed to fall below 79-80 mm. Hg. to avoid an oxygen debt to the brain' In a 15-degree head-up position an average reduction of 44 per cent in mean arterial blood pressure failed to alter cerebral blood flow or oxygen consumption, which was maintained by a 46 per cent reduction in cerebrovascular resistance (29). (The head-up position may *never* be used in a high spinal block.) Always test the person before elevating the head. Therefore, if the wound be of the head and neck, the head and neck should be elevated and the blocked pressure determined in this position. If it is below 70 mm Hg, replacement with blood, plasma, or plasma substitutes should be rapidly accomplished until the pressure can be maintained at this level while the surgeon goes ahead in a comparatively bloodless field.

Because the blood vessels are dilated, they lie open on the cut surface and become thrombosed. When the block wears off, they constrict onto the clot, thereby reducing the likelihood of fresh haemorrhage when the pressure rises. Haemostasis must nevertheless be meticulous before wound closure.

There is evidence to show that Hexamethonium actually protects against shock, as shock has not supervened when it has been used with Cyclopropane and Flaxedil® for long and traumatic manoeuvres with fractured legs in placing Steinman pins or bone plating refractory fragments. This is in comparison to previous experience in using Cyclopropane alone for orthopaedic cases. Shock invariably occurred in these cases unless they were carried at the verge of respiratory arrest for the entire operation. This was before the days of relaxants.

There has been some argument about whether cardiac anoxia might occur with the induced hypotension technique. Rollason (23), citing Wigman, reports a few cases with an "inversion of the T wave lasting for several days before reverting back to normal" N. du Bochet and J LeBrigand report on a series of electrocardiograms done on decompensated cardiacs under spinal anaesthesia. The patients were all undergoing major abdominal surgery, 15 for ligation of the inferior vena cava. All showed slowing of the pulse rate once spinal block took effect. All QRS complexes increased from .02 to .04 second under spinal and 12 negative T waves became positive. Studies are in progress to correlate pulmonary artery pressure, minute volume, and arterial oxygen with striking clinical improvement of pulmonary oedema, tachycardia, urinary output, cyanosis, and dyspnoea under spinal anaesthesia. Pulmonary artery pressure was seen to drop 25-42 mm. in two cases, the minute volume to increase up to 1½ litres (24).

After September, 1952, I deliberately applied the ganglionic block technique to control shock in patients felt to be in extremis. Some representative case histories follow.

The first of these histories concerns a case of obstetrical haemorrhage from a retained placenta. The brachial blood pressure was unobtainable, yet blood was still running in a steady stream from the patient's vagina. A transfusion using a vein at the wrist and a No 18 needle was barely dripping because of the extreme vasospasm, in spite of pressure in the flask. Hexathide® 100 mg. were injected in the intravenous needle *in situ*. The patient was in lithotomy position, which was changed to Trendelenburg-lithotomy position. Anaesthesia was begun with Cyclopropane. Two minutes after Hexathide®, the blood was running freely into her

vein with the release of vasospasm. The blood pressure could be read at 40/10 with a pulse rate of 100. A second 500 cc. of blood brought the patient's pressure in Trendelenburg position to 90/30. We felt that she was out of danger as there was no further bleeding. Her recovery was uneventful. The problem here was not the total blood loss but the fact that the patient was losing blood faster than it could be replaced. Blood replacement should be available as soon as possible, but if it is not yet available, rapid saline infusion or blood substitute will serve as a temporary stop-gap. If the Hexamethonium can be introduced into a small vein with a hypodermic needle, the resulting vasodilatation will enable one to place a needle in a large vein by letting the arm or leg drop over the edge of the table. This saves the time lost performing a cut-down. At the same time, the dangers of arterial transfusion are avoided.

The second case was that of a boy who had suffered a head injury but was just beginning to regain consciousness. The surgeon desired to plate a compound comminuted fracture of the tibia and fibula which appeared to be keeping him in shock in spite of blood replacement. Further brain anoxia was to be avoided. He was given a spinal block with Nupercaine® 10 mg and Procaine 100 mg in 4 cc of spinal fluid, between the second and third lumbar nerve roots. He was immediately tilted into Trendelenburg position. After five minutes, his pressure was 100/30, although it had been 70/40 before the block was induced. He had been trapping some of his available blood supply in his extremities with vasoconstriction from tissue injury. The leg to be plated was elevated on a pillow with a marked reduction in bleeding at the site of fracture, no increase in bleeding was noted from the Cyclopropane with which he was kept asleep. The Cyclopropane was used to assure sleep, thus preventing vomiting as a reflex protective mechanism when a high subarachnoid block is in effect. When he awoke, he appeared much clearer mentally than before, and he made an uneventful recovery.

The third case was an almost exsanguinated ectopic gestation. As the patient was being prepared for the operating room, intravenous saline was begun while blood was being typed and cross-matched. She was barely put to sleep with Pentothal 200 mg in the intravenous tubing, Cyclopropane anaesthesia was induced, while 100 mg. of Hexathide® and 20 mg. Flaxedil® were given. By the time the surgeons had scrubbed, the Flaxedil® was effective, anaesthesia was established, and a slight Trendelenburg position had been effected. Her blood pressure was still unobtainable, but the intravenous saline had begun to run rapidly and the blood was on its way. Liquid blood, 1500 cc., was suctioned out of her abdomen (measured) besides clots and soaked abdominal sponges. The Fallopian tube was no longer bleeding. By this time, a pressure of about 30/? mm Hg. was obtainable and the blood arrived. Under pressure we replaced 1500 cc. of blood loss in three-quarters of an hour, and her blood pressure in Trendelenburg position was 100/40, with complete recovery.

The next extremely dramatic case was that of a patient with a ruptured ectopic gestation, arriving at the hospital unconscious, with no measurable blood pressure and an impalpable radial pulse. Fortunately, the gynaecologist had seen her some days previously and was aware of the possibility of an ectopic gestation. She was undressed on the stretcher in the operating room to save time. It was

impossible to locate a vein. She was placed on the right side and Nupercaine® 10 mg (1:200 in 2 cc solution) added to Procaine 100 mg. made up to 4 cc. with spinal fluid was given between the 3rd and 4th lumbar nerve roots in a 10-degree Trendelenburg position. She was placed supine on the table and tilted further to 15 degrees. Oxygen with Cyclopropane was started, again to assure sleep where more depressant narcotics were to be avoided. Her respirations which had been gasping and irregular became normal and regular, her colour improved, and when the left arm was dropped over the side of the table, a large vein became readily visible. Subtosan® 500 cc was started with a No. 18 needle. When 500 cc of Group O, RH negative blood arrived it was started in the right arm. Her blood pressure at this time was 40/20 mm. Hg. A further 1000 cc. of blood (typed and cross-matched with donors as well as patient) resulted in a blood pressure of 95/40 mm. Hg. A further 1000 cc. of saline was given on the ward. The amount of blood loss could not be determined as a good deal of it went on the floor when the abdomen was opened. We considered that, in this case, spinal anaesthesia had been truly life-saving. Her blood volume had been adequately restored, and the following morning one would never have known she had been in extremis.

In another case of a ruptured uterus where adequate blood replacement had been made for the patient's constricted vascular system to be in compensation at a blood pressure of 130/70, the method of "total" spinal without a pressor drug was chosen in order to minimize further blood loss and prevent the irreversible phase which is seen to supervene after a "second bleeding" of the experimental animal. This was the type of case so often reported from the battlefield with disastrous results even where massive blood transfusions were available. Again the patient recovered without further incident.

We then became bolder and used Hexamethonium Iodide for incomplete abortions if the patients showed signs of shock or were still bleeding. We used it for patients with fractures who had traumatic shock if they did not show adequate response to blood transfusion. Several times when the surgery promised to be minor, but turned out to be major, we saved the patient's blood and prevented shock with "Hexathide®" added to the established anaesthetic. One of these occasions was with a case of abdominal pregnancy, where, after the block became effective, I noticed that the placenta rubbed off the bowel like wet blotting paper, without damaging the serosa. Several patients with extensive abdominal carcinoma were kept alive through perilous operations that allowed them some further months of fairly comfortable life. Radical breast resections were done with an estimated loss of less than 100 cc. of blood.

The next case is an example of one where blood could not be given. We were consulted after the patient, still shocked, and bleeding from the uterus, had developed hives from the first 50 cc of plasma, and chills and fever following the first 100 cc. of blood. We had some plasma substitute kindly given us by Poulenc Ltd for such a contingency. To this Subtosan® was added 130 mg of Pentothal®, 100 mg of Hexathide®, and anaesthesia was established with Cyclopropane. She was in lithotomy-Trendelenburg position and in spite of this, her pressure was about 30/10 mm. Hg. She stopped bleeding immediately and did

not lose much more with the curettage, but the surgeon asked for Ergotrate®. We had not been using it with these cases because the patients did not appear to need it. With one ampoule, her blood pressure rose to 70/40, whereupon she began to bleed again, and had to be packed. The blood pressure then dropped off just as rapidly, and by the time we had given two bottles of plasma substitute, her pressure was 60/30 in Trendelenburg position. She was given oxygen by tent and continuous saline infusion. After 5 hours, her blood pressure again dropped below 50 mm. Hg. although her pupils were dilated and she appeared still to have some block present. Since she was no longer bleeding we decided to try decreasing the size of her vascular tree. We gave Methedrine 5 mg at five-minute intervals until 20 mg had been given, and her blood pressure was 80/40. We put another 10 mg. of Methedrine® into the intravenous drip and her blood pressure stayed constant at about 100 mm Hg for the next several days. She had a very stormy course owing largely to a transfusion reaction, having both hives and herpes. She did not sleep with her sedative, and became irrational. In 48 hours she appeared to be normal except for weakness from anaemia. Her haemoglobin was then 40 per cent. She tolerated liver and intravenous iron and progressed well, showing no mental damage from her prolonged hypotension.

We have, during the past year, been investigating Chlorpromazine as an adjuvant to anaesthesia, and in its relation to the control of shock, comparing it with the two methods previously described. Actually it may be used alone or in combination with hypotensive spinal anaesthesia techniques or Hexamethonium. Again, this is an agent whose use results in a vasodilated system. The principles governing its use remain the same as those for high spinal block or Hexamethonium salts.

The following case history is illustrative. A gentleman of some 50 years who had been hit by a car, with a resulting compound comminuted fracture of his right tibia and fibula, right femur and humerus, a fracture of his pelvis and a head injury, in spite of morphine and blood, was in severe shock when seen. He had been given two units of blood and Levophed® without any improvement. He was given 50 mg of Chlorpromazine intravenously and another 50 mg intramuscularly. The tibia was plated and the fractures of the femur and right humerus were set temporarily under Cyclopropane anaesthesia. At the end of the operation he was warm and dry with a blood pressure of 100/70. He complained very little of pain. A minimal amount of Demerol® kept him comfortable. This patient had two subsequent anaesthetics, one for Kunchner nail to the femur and another for setting the humerus. He showed no gross mental changes in spite of his head injury. Chlorpromazine was chosen here to offset the effect of Levophed® and establish sympathetic block.

It would have been of immense scientific interest to have been able to determine the presence or absence of VDM in the blood streams of these shocked persons. Clinically, there is no doubt in our minds that a hypotensive technique provides a wider margin of safety for the injured, and may completely prevent the occurrence of the irreversible phase of shock.

Over the past two years, we have lost only two patients for whom we have been called when the surgeons felt the situation was out of control. Both of these

patients were children with post-tonsillectomy haemorrhage, who were seen so early in our work that we lacked the courage of our convictions and attempted resuscitation by arterial transfusion. In the first case this resulted in pulmonary œdema and cardiac dilatation and death for the child without any improvement having occurred in his circulatory status. The second child died of a transfusion reaction although the blood given was correctly matched as far as our laboratory could determine even on rechecking. Our later experience taught us that while the survival of the first child might have been problematical, the second child's circulatory system could have been stabilized and his bleeding stopped by the use of Hexamethonium.

I should like to add one more striking case history, that of a female, age 52, thought to have been hit by a car. There was an obvious fracture of the left femur and left humerus. She was said to have Hodgkin's Disease as well. When seen she was deeply shocked, and lapsed into coma just after being seen. Demerol® 100 mg and Chlorpromazine 50 mg were given intravenously, one after the other. The X-ray table was placed in 10-degree Trendelenburg and oxygen was given by mask from the gas machine. The patient stopped breathing spontaneously for 10 minutes. At the end of that time a facial pulse was again felt and she began to breathe spontaneously. The X-ray revealed pathological fractures of multiple myeloma. It subsequently developed that she had fractured each one of her long bones. With Nitrous Oxide-Oxygen anaesthesia the patient was put in traction in Trendelenburg position and supportive fluids only were given—no blood. She maintained a pressure of 70/40 which gradually rose to 100/80 in the next 24 hours. She was kept on Chlorpromazine 25 mg. every 4 hours, with Demerol® as needed. On the second hospital day she was given blood because of the severe anaemia and low Hematocrit. She had at no time any kidney block and no mental deterioration. This patient is still alive. While I must apologize for being available to save such a patient, perhaps it was permitted in order to convince us that the impossible is possible.

DISCUSSION

It has been pointed out that the blood pressure seen in sympathetic blockade for hypotensive anaesthesia may frequently be anywhere from 50 to 30 mm Hg systolic over the brachial artery. This low pressure is obtained by pooling some of the patient's blood into the lower extremities while the head and trunk are kept in a down-tilt. In fact the anaesthetist has artificially produced a situation in which 1000 to 1500 cc of the patient's available blood volume are lost to him in terms of actual use by the vital organs. This loss does not cause the anaesthetist any alarm, nor does it harm the patient. If with a spinal block there is a decrease in pulse rate below 60 per minute, or if with the drug-induced blockade there is a further blood pressure drop below the established level, then more blood is required and may be instantly given by elevating the feet. Therefore when the patient who has lost, from traumatic injury, some undetermined portion of his blood volume is given a sympathetic block and placed in Trendelenburg position, the resulting blood pressure reading will immediately indicate how severe that

loss has been, and how urgent is the need for blood replacement. At the same time it makes the technical problem of replacing it much easier.

This is a method whereby we can regain control of the shocked patient's circulatory system when shock has progressed beyond our control and appears to be approaching the irreversible phase. In these patients where one must trust clinical judgment we hold that there is now a further chance for life. If this method should prove itself safe in other hands than mine for the patient who is lost without it, then increasing experience may widen the scope to a point where it may be used as first choice in severe trauma rather than a last resort.

SUMMARY

A method for the control and alleviation of traumatic and haemorrhagic shock is presented, using the principle of vasodilatation by sympathetic blockade obtained variously by high subarachnoid block, Hexamethonium compounds, or Chlorpromazine. By using position and blood replacement therapy in conjunction with sympathetic blockade it is possible to arrest and reverse the shock phenomena more promptly and safely for the patient and render immediate surgical interference feasible for repair of injury.

RÉSUMÉ

Il a été démontré que le choc résultant d'un traumatisme et de l'hémorragie peut être contrôlé par un blocage sympathique produit par une infiltration sub-arachnoïde spinale haute, ou encore, par les composés de l'hexaméthonium, l'arfonad et le chlorpromazine pourvu que l'on place le patient de façon à utiliser la vasodilatation résultante.

Le traumatisme est suivi d'un déplacement sanguin qui se traduit par une chute de la pression périphérique comme on peut le constater au sphygmomanomètre au niveau de l'artère brachiale. Ceci est dû à la vasoconstriction périphérique. De même, quand on produit un bloc total du sympathique chez l'homme, la pression artérielle périphérique tombe aussi mais cette fois pour une raison complètement différente. Il est possible, en cette circonstance, de maintenir un apport sanguin adéquat aux organes vitaux, en plaçant le patient en Trendelenburg à 15° ou bien en élevant les membres inférieurs au dessus du niveau du thorax, la tête étant maintenue à plat. De cette façon, la circulation se maintient à une pression diminuée et la réduction de la résistance vasculaire compense et permet de préserver une oxygénation adéquate.

Dans les cas d'une blessure centrale où l'hémorragie est augmentée par la vasoconstriction périphérique produite par les mécanismes de défense, une injection intraveineuse de 100 mgm d'iodure d'hexaméthonium réalise un blocage ganglionnaire presque total en 2 à 5 minutes. La vasodilatation résultante permet de trouver une veine, de commencer une transfusion de remplacement et aussi d'élever la région qui saigne en maintenant la tête et le thorax abaissés. Une anesthésie peut être donnée avec n'importe lequel agent excepté l'éther qui perturbe la vasomotricité capillaire.

Chez le patient dont le mécanisme protecteur a compensé pour le choc, une intervention chirurgicale qui se priverait de la protection procurée par une vasodilatation systémique, peut le conduire dans une phase de choc irréversible.

Quand on utilise les composés de l'Hexaméthonium ou le Chlorpromazine, on peut maintenir la position tête élevée, à condition que la pression brachiale artérielle ne descende pas en bas de 70 mm de Hg. S'il y a chute, une restauration rapide au niveau désiré s'obtient par le plasma ou ses substituts.

Depuis septembre 1952, j'ai employé cette méthode pour contrôler le choc de patients que les chirurgiens ne croyaient pas devoir survivre en défit des mesures thérapeutiques qu'ils avaient déjà instituées. Ces cas étaient traumatiques, hémorragiques ou encore, mixtes. Ils furent traités par un blocage rachidien haut, aucune médication vasoconstrictive n'étant employée; ou encore par l'iodure d'Hexaméthonium ou le Chlorpromazine, selon les circonstances. Par cette méthode, il est possible de reprendre contrôle du système circulatoire du patient en état de choc quand ce choc a progressé au delà de notre contrôle et semble approcher la phase irréversible.

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