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Intra-arterial transfusion

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INTRA-ARTERIAL TRANSFUSION

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INTRODUCTION

Irreversible shock has long been a much feared condition which physicians have sought to combat and prevent, knowing the only therapy is prophylaxis. The intravenous route of fluid administration has been popular, but in conditions of marked hypotension with peripheral venous collapse it is usually a difficult and most ineffective means of therapy. Under these conditions the technical difficulties in performing a vena puncture are great, and if successful, transfusing sufficient fluids to restore a clinically sound blood pressure is most difficult without overloading an already ischemic myocardium with resultant right heart failure. A means of restoring a physiologic blood pressure under conditions of severe hypotension is possible by utilizing the intra-arterial route. The definite value of intra-arterial transfusion has been proved by many investigators, and it has played an important part in resuscitation of patients in severe hemorrhagic shock.

HISTORY

In 1875, Landois (33) utilized transfusions via the intra-arterial route in cases of severe frostbite using the posterior tibial artery. Halsted (24) in 1883 reported the use of intra-arterial transfusion in cases of carbon monoxide poisoning; the patients were bled through a vena puncture, the blood being exposed to atmospheric oxygen and then returned to the patient intra-arterially. Pike (46) in 1908 discovered in animals that rapid infusion of blood intravenously resulted in pulmonary edema and

heart failure. He proceeded to perfuse blood via the carotid arteries in dogs in severe shock states with some success. The results of these earlier experiments were not encouraging and little was written in the literature of this treatment for the next thirty years. Birillo (5) in 1939 reported five patients who were in severe shock and given blood intra-arterially; all showed temporary improvement, but died in a span of three to twenty-four hours following treatment. Again, in 1941, Birillo (5) reported five cases of soldiers at the front in severe hemorrhagic shock who were treated with 200-300 cubic centimeters of blood given intra-arterially under 170-180 millimeters mercury pressure; four survived and one died. Negovski (38) in 1939-1942 described treatment of fifty-one soldiers at the front, in all degress of traumatic and hemorrhagic shock, with intra-arterial blood. He stated the needle was directed toward the heart and blood to which adrenalin and glucose had been added was given intra-arterially utilizing the most accessible artery. Twelve patients recovered with no residual damage. Davis (10) in 1941 described his experiment with dogs comparing the efficiency of intravenous and intra-arterial routes of blood administration, and stated that the intra-arterial routes were superior in cases of severe hemorrhagic shock. Kendrick (32) confirmed Davis's results but stated it could not be reduplicated using 5% glucose and water in place of blood. Petrovsky (45) in 1943 described four cases of cardiac arrest in which 200-300 cubic centimeters of blood were given under 200-250 millimeters of mercury pressure via the carotid artery. Three of

these patients survived, one died later of unrelated causes. Avedisou (1) in 1944 reported sixty cases successfully treated by intra-arterial blood administered by cannulas and needles. Kohlstaedt and Page (33) in 1943 following advice from Seely, began a series of experiments with hemorrhagic hypotension and its treatment using dogs. It was found that giving blood via the arteries yielded a higher rate of survival than intravenous therapy. At that time they reported a successful case of a patient with acute hypotension who was resuscitated by the intra-arterial route using a 14 gauge needle giving 1500 cubic centimeters of whole blood under 50 millimeters of mercury pressure. With the information available about the practicability of giving blood intra-arterially for hypotension, Gardner (18) in 1946 used induced hypotension in surgery to decrease blood loss and returned the patient's blood by the intra-arterial route following the surgical procedure. Glasser and Page (21) in 1947 produced a marked degree of hemorrhagic shock in dogs and resuscitated the animals by intra-arterial transfusions while studying the physiology of irreversible shock. Kay and Hacker (29) in 1947 encountered severe hemorrhage following injury to a right internal mammary artery; a severe state of shock developed. By inserting an 18 gauge needle into the ascending aorta they were able to transfuse 500 cubic centimeters of whole blood in three minutes, which resuscitated the patient. During the next two years much investigational work was done experimentally, as well as clinically, and the procedure of intra-arterial transfusion became more applicable

clinically. In 1948, Robertson, Trincher, and Dennis (48) bled dogs from the femoral artery to a controlled level of hypotension. Following a specific period of time, blood was returned via the femoral artery under 50 millimeters of mercury pressure; an equal number of dogs were bled and transfused intravenously; the conclusion was reached that more blood per unit of time could be given intra-arterially and that the resultant rise in blood pressure was more stable than that obtained from intravenous infusion. Since 1948 many reports of clinical application of the intra-arterial transfusion technique have been published by such men as Seely, Page, McKenzie, Morton and others which covered technique, transfusion rates and blood substitutes.

PHYSIOLOGY OF SHOCK

The shock state has many itiological factors, the most common being hemorrhage or severe trauma. The pictures become quite similar when disparity occurs between the volume of the circulatory tree and that volume of blood within the circulatory tree, namely, decreased cardiac output and oligemia which produces a low blood pressure and generalized anoxemia. Seely (51) considers the following classifications of shock:

1. "Hemorrhagic, caused by gross loss of blood either apparent or concealed."

2. "Traumatic, in which the predominant feature is (a) external violence to large muscle groups or the intestines (b) extensive freezing or (c) burning of tissues."

3. "Vasogenic, occuring in poisoning with rattlesnake vemon, in which the effect in the capillary bed is the most prominent feature."

4. "Neurogenic such as the syndrome of acute collapse resulting from a sudden blow to the epigastrium or testicle."

5. "Circulatory in which venous return to the heart is impeded."

Hemorrhagic shock differs from shock of traumatic origin in that in the latter, hemoconcentration is any early finding. Since plasma is readily lost through the capillary walls in traumatic shock, the cell mass concentration may be readily detected. In contrast, hemorrhagic shock involves loss of both plasma and cell mass with no resultant hemoconcentration until tissue anoxemia disturbs the permeability of the capillary wall allowing plasma to escape into the tissues. With the onset of plasma escape due to increased capillary permeability, a vicious cycle is set up which if not interrupted by intravenous or intra-arterial therapy progresses to an irreversible state and death. As the shock state approaches irreversibility, larger crystaloid particles pass through the capillary walls and in a short space of time the capillary damage is sufficient to produce irreversible changes. It is the hope that blood via the intra-arterial route will relieve the anoxemia promptly and spare the capillaries irreversible damage. It is known that tachycardia precedes the drop in blood pressure, especially in shock caused by trauma and hemorrhage. Seely (51) feels that active treatment

is essential when a pulse rate exceeds 120 in the presence of a normal blood pressure. Page (40) urges the condition of the peripheral arteries be established in making a diagnosis of shock. He cites the case of a patient with a blood pressure of 38/0 who was conscious, warm, responsive and ambulatory. No symptoms of shock were present except urinary cessation, the result of ingesting arsenic trioxide which gave rise to a great decrease in peripheral resistance in the arterial tree. Experimental work proved the peripheral arteries are constricted until shortly prior to death at which time the arterioles and heart dilate and display a decrease in response to chemical stimuli. Kohlstaedt and Page (33) further studied this phenomenon of arteriole response to pressure drugs. They induced hypotension in dogs and maintained them at hypotensive levels while periodically injecting standardized doses of angiotonin and epinephrine; the response varied with the state of shock. If no response to these chemical stimuli occurred, there was a low survival rate even after intra-arterial transfusion; if, however, a good response could be obtained, the hypotensive level could be reversed by intra-arterial transfusion with a resultant high survival rate.

As it is well known, vasopressor drugs have no theraputic value in the treatment of shock from decreased blood volume and should be used only for neurogenic shock state, since their action tends to decrease the volume of the vascular tree. Replacement therapy is a necessity in cases of shock with a decreased blood volume since the myocardium is capable of delivering sufficient blood to the tissues

if an adequate volume is available to the heart.

The problem of blood or plasma replacement via the intra-arterial route to disrupt the cyclic pattern of irreversible shock embodied many problems relative to the physiology of the shock state. The problem of what was responsible for the production of the irreversible state was studied by many individuals. Fine, Seligman and Frank (16) investigated the part played by the kidneys and liver in this condition. The theory that ischemia in the kidney tissue from hypotension results in increased renin production is accepted, but since the hypertensiongen level in the blood falls rapidly in shock states, the renin production would not be a factor in producing irreversible shock. Bilateral nephrectomies were done on dogs in which shock was subsequently induced; no alteration in the shock picture was noted. They found the liver under anoxic conditions liberates a toxin that depresses the vasomotor system. Cross circulation experiments in which oxygenated blood perfused the hepatic cells produced marked delay in the production of severe shock states, and in numerous animals produced resuscitation from a state of profound shock. These toxic products cause the heart to decrease its contractile strength and rapidly dilate with resulting heart failure.

Corcoran and Page (9) state that during shock, especially from traumatic causes, there results marked urinary shut down from hypotension. The result being the formation of acid urine which caused the precipitation of hematin from the breakdown products of myoglobin and hemoglobin.

Gold, Rosenthal and Schotz (22) described the effect on the

myocardium in shock states. Early in shock with a decreased venous return, low blood pressure, small pulse pressure, low diastolic pressure, and reduced minute output, the decreased stroke volume is insufficient to maintain aortic pressure even during the height of systole. Electrocardiograms at this stage are essentially negative. Later in the course of hypotension, the cardiodynamic alterations, which are characteristic of a progressively falling blood pressure become manifested; the force of ventricular contractions decrease, and the ejection rate of the blood is slowed. Electrocardiograms now reveal depression of the S-T segment. Subsequent intra-arterial transfusions may reverse these changes.

Negovski (38) described the electrocardiogram findings in severe shock states. To facilitate doing this, he developed this classification of phases of death:

1. "Agonal death: 4 - 6 minutes before clinical death, characterized by gasping respirations."

2. "Clinical death: period of final respirations with no obtainable pulse."

3. "Biologic death: stage when tissue decomposition begins."

The electrocardiogram findings in the agonal phase are distortion of the T wave, prolonged P-Q, S-T, and QRS intervals. In clinical death, the QRS waves are biphasic and the P wave is totally lacking. Both of these conditions may be reversed by intra-arterial transfusions provided treatment is begun within 4 - 6 minutes following the onset."

The physiologic response of the body in shock to the intra-arterial administration of blood follows Pascal's Law: "Pressure exerted on a confined liquid is transmitted equally to all parts of the enclosed liquid." Jones, Davis and Hubay (28) from results of animal experiments described the physiologic effect of intra-arterial transfusions: With intra-arterial infusion of blood, the pressure is rapidly built up in the arterial system thereby perfusing the coronary arteries with blood. The pressure in the aortic valves maintain them in a closed position until ventricular systole becomes forceful enough to overcome them. Also, due to this increase in pressure there is decreased stimulation of the sino aortic receptors with a resultant decrease in principal vasoconstriction. The blood flow is increased in the vertebral arteries supplying the respiratory centers with a subsequent increase in respiration. Likewise there is increased glomerular filtration rate with an increasing urinary output which lowers the level of waste products in the blood. This rapid administration that is possible using the intra-arterial route is most beneficial; rapid administration via the intravenous route is often accompanied by an overloading of the venous side placing additional strain on an already ischemic myocardium yielding pulmonary edema and right sided heart failure.

INDICATIONS

The indications for arterial transfusions vary with different investigators, but in general it is thought that in shock states from exsangiunating hemorrhage the arterial route is preferred.

When venous collapse is marked and intravenous therapy not feasible, the intra-arterial route is often life saving. An editorial in the New England Journal of Medicine (15) states that intravenous infusions should be attempted through many ports before intra-arterial therapy is instituted. White (54) feels that the intra-arterial route should be used in all cases of hemorrhagic shock with systolic blood pressure of 50 millimeters of mercury or less. Page (42) considers the intra-arterial route beneficial in cases of acute hemorrhagic shock, cardiovascular surgery, and as an adjunct in locating obscure bleeding points often missed in surgery when hypotension is treated by intravenous transfusions. McKenzie and Morton (37) consider the indications to be exsangiunating hemorrhage, profound non-hemorrhagic shock, and cases of cardiac arrest. The utilization of this method in surgically induced hypotension has accounted for much clinical research on this problem. Various surgical procedures known to be associated with considerable blood loss have been greatly aided by surgically induced hypotension with intra-arterial return of the patient's blood following the operation. Gardner (18) utilized this method in a case of an olfactory groove meningioma; the patient's blood pressure was reduced from 140-100 millimeters (systolic) after the withdrawal of 1600 cubic centimeters of blood. At the completion of surgery, 1100 cubic centimeters of blood was returned to the patient via the dorsalis pedis artery, the surgical site was then closed after checking for bleeding points. While returning to the ward another 500 cubic centimeters of blood was given. Similar use was made of this

method by Love, Courtin, Anderson and Lundy (36) in the removal of an epidermoid tumor of the third ventricle. Harris and Hale (26) did similarly in a fenestration operation to control bleeding.

APPARATUS

Numerous mechanical arrangements have been designed for intra-arterial transfusions; regardless of their complexity, certain basic equipment must be incorporated in them; the variations and individuality being the product of the operator. A source of blood, such as a standard blood storage bottle; pressure whether it be from a hand bulb, oxygen tank, or gravity; a manometer of the mercuric or aneroid type; connecting tubing, and a source of outlet into the artery, a cannula or needle. Variations in these basic pieces of equipment are numerous, such as means of controlling air emboli; devices to incorporate an apparatus for inducing hypotension in surgery, and others. Regardless of these variations, workers in all fields agree a type of apparatus that is convenient to set up for emergencies as well as readily cleaned and sterilized is of prime importance.

Glasser and Page (19) used blood storage bottles; they attached a bulb from a sphygmomanometer to a Y connection which supplied tubing to an aneroid manometer and to the air intake on the blood bottle. To the outlet of the blood bottle was attached a Murphy drip chamber, tubing and a cannula or neede for insertion into the artery of choice. Robertson, Trincher and Dennis (48) utilized a similar apparatus, but attached the sphygmomanometer

bulb directly to the air intake of the blood bottle, the Y connection with the aneroid in the outlet tubing between the Murphy drip chamber and the needle. Jones and co-workers (28) utilized a similar combination, but added another bottle in series with the blood bottle so changes in pressure would not be so abrupt. Porter, Sanders and Lockwood (47) designed specially made flasks, two in number, sharing a common drop chamber and outlet tube; the pressure was supplied by an oxygen tank with reduction valve. The apparatus was so constructed that by turning stopcocks either reservoir of blood could be turned into the circuit, thereby saving valuable time when a large quantity of blood was needed. In times of great emergency when no equipment is prepared for use, a standard intravenous setup may be utilized with a bulb connected directly to the air intake of the blood bottle and the pressure estimated by clinical response. If no bulb is available, merely elevating the height of the bottle above the needle site to facilitate gravity may be life saving. All connections in the pressure system should be secured by tape to insure against "blow outs."

SITES OF INJECTION

Selection of the site of injection is primarily a matter of convenience to the operator; numerous locations have been utilized for this purpose. One popular location is the radial artery, since it is apart from most operative fields, and is readily accessible. The femoral, dorsalis pedis, and posterior tibial arteries are others frequently used. In abdominal and thoracic surgery, close proximity

to major arteries allows emergency transfusions to be instituted in the aorta or one of its major branches.

SELECTION OF NEEDLES

Early in the history of arterial transfusions, cannulas were commonly employed; more recently an effort has been made to preserve the artery used for injection, hence needles are now more widely used. Keet, Halsted and Collins (30) prepared a 15 gauge spinal needle by grinding it down to a length of 1½ inches with no bevel or cutting edges; the stilet was left protruding slightly from the end of the prepared needle and was sharpened to pencil point sharpness. When this needle was inserted, no cutting of the arterial wall resulted. Because of the work done by Brew and Dill (6), the 15 gauge needle now is the most popular size. They ran a series of experiments using a 22 gauge needle with a flow of 4.75 cubic centimeters per minute under a pressure of 50 millimeters of mercury as a standard. Their results expressed in rate of flow are as follows:

Needle Size	Flow Rate Compared to Standard
20 gauge	2.8x
18 gauge	7.0x
15 gauge	16x
13 gauge	18x

As is readily seen in the above comparison, the greatest increase in flow is between the 18 gauge and the 15 gauge needle. Because of the little difference between the 13 and 15 gauge needles in flow, and the considerable difference in size, the 15 gauge needle was adapted.

PROCEDURE

If a peripheral artery is selected as the site of injection, a surgical preparation of the skin is done. An incision over the most accessible portion of the artery is made, and the artery is freed by blunt dissection. After assembling the apparatus, the needle is inserted into the artery directed toward the heart and loosely secured by a silk suture thru the adventitia. Pressure is developed in the system by manipulation of the bulb, or oxygen, if the latter is used; the amount of pressure to obtain is a disputed point. Pressures have ranged from 20 millimeters to 300 millimeters of mercury. Page (42) maintained the pressure in his system approximately 20 millimeters of mercury above that of the patients; this was gradually increased as the patient's blood pressure responded. Robertson and co-workers (48) maintained the pressure in their system about 50 millimeters of mercury above that of their patients. Porter, Sanders and Lockwood (47) perfused blood under a pressure of 250 - 300 millimeters of mercury stating that 500 cubic centimeters of blood could be administered in three minutes. Most investigators agree on a pressure in the range of 50 millimeters of mercury above that of the patients, gradually increasing it as the patient's pressure increases until the desired level is reached. If, however, no source of pressure for the system is available, White (54) proposes a set of fairly well standardized values applicable to gravitational force. He stated that a height of 50 centimeters above the needle was roughly equivalent to 30 millimeters of mercury pressure

in the system; at 100 centimeters above the needle an equivalent pressure of 60 millimeters; at 150 centimeters, 90 millimeters of mercury. Since intra-arterial transfusions are usually associated with spasm of the involved artery, infiltration with a few cubic centimeters of 1-2% novocaine in the area of puncture may help control this condition. Keet, Halsted, Collins and Rousselot (30) injected 5 - 10 cubic centimeters of 1-2% novocaine into the tubing near the needle and found it greatly reduced spasm which may occur for some distance beyond the point of the needle. However, this practice was carried out only in anesthetized patients; if not anesthetized, a stellate ganglion block was performed.

Pressure at the site of injection during transfusion may act as a tourniquet on the part of the extremity distal to the needle; hence only a period of 30 minutes should be used at one time for transfusions; at the end of that time the line should be clamped and time allowed for perfusion of blood into the extremity.

As the transfusion progresses, periodic checking of the venous pressure is a good safety measure employed by Beattie and Thistlethwaite (2). They recommend that if the venous pressure rises 5 centimeters of water pressure or more, the intra-arterial transfusion should be stopped to prevent over-transfusion.

As the arterial blood pressure approaches the pressure in the system, less blood will enter the artery, finally when equilibrium is reached, a pulsation will be visualized in the drip chamber. Should the patient's blood pressure fall, blood will again flow

readily into the artery. The usual pressure accepted by most men as acceptable is 100 millimeters of mercury pressure in the patient.

To reduce the hazard of clotting, various investigators have used a heparin solution which may be injected periodically into the tubing of the system.

On completion of the arterial transfusion, the needle is withdrawn and moderate pressure is applied to the artery to acquire hemostatis. If the opening in the artery is large, a few sutures may be placed in the defect. Every effort is made to spare the artery; due to anomalous conditions of the arteries which may occur, ligation may result in gangrene of the extremity distal to the site of the ligation.

During the intra-arterial transfusion, the prognosis may be judged with some degree of accuracy. Page (41) states the following are poor prognostic signs:

1. Gradually increasing hypotension with little tendency towards stabilization.

2. Large quantities of blood taken with the intra-arterial infusion (intra-arterial transfusions should require 30% less blood than intravenous transfusion for the same response).

3. Failure to respond to epinephrine or angiotonin following transfusions (indicative of the cardiovascular system becoming refractory to chemical stimuli).

BLOOD SUBSTITUTES

If blood is unavailable, plasma or parenteral fluids may be given until blood is available. The possibility of successful blood

substitutes was investigated by Davis and co-workers (10). They discovered that hypertonic solutions injected intravenously resulted in a drop in blood pressure, but if given intra-arterially a rise was noted; the extent of which depended on the substance given. 50% glucose, 30% gum acacia in isotonic saline, and 50% succrose were used on dogs in shock states. The 50% glucose caused the greatest rise; 30 - 50 millimeters of mercury lasting about three hours. The reason for the elevation was neurogenic, since sectioning of the spinal cord abolished this response. Kendrick and Wakin (31) later proved that by comparing the intravenous and intra-arterial routes, using 5% glucose in saline, that the intravenous route showed the most response in blood pressure rise.

HAZARDS

Air emboli are an ever present hazard when intra-arterial transfusions are utilized. Great care should be exercised to securely clamp the tubing before the blood storage bottle completely empties. This risk may be minimized by using 2 or more bottles of blood in series. Hale (23) utilizes a MacIntosh drip chamber which resembles a Murphy drip chamber, but contains floats that settle out when the fuild is gone and prevents the passage of air into the outlet tubing. Dornetti and co-workers (14) feel there is no certain means of preventing air emboli, so the equipment should be simplified enough that its progress can easily be watched. Ischemia is another hazard; as described earlier, anomalies of the arteries in the extremity increase the possibility of ishemia and gangrene if the artery used for injection is ligated. This is particularly true in

the radial artery due to anomalies in the ulnar arcade of the hand. This problem may be controlled if the vessel is spared. Over-transfusions are occasionally seen when the arterial route of transfusion is utilized. Beattie and co-workers (2) state the signs associated with over-transfusion are dependent upon not only the amount, but the rate which it was given. Using dogs, they divided them into classes of "slow rate," "moderate rate," and "fast rate" of transfusions. Using a slow rate of 30 - 60 minutes the blood volume could be increased 50 - 100% over normal before symptoms appeared; the symptoms were increased blood pressure initially, then a decrease below normal; increased pulse rate; and increased venous pressure. With a moderate rate of 15 - 30 minutes, elevation in blood volume of over 35% resulted in a high mortality. The symptoms were essentially the same as listed before but more marked. Using the fast rate of less than 15 minutes, the mortality rate was comparable to the moderate rate; the symptoms were the same but a marked rise in venous pressure was seen. From their experiments they determined that blood volume up to 35% greater than normal could be produced if the rate of flow was slow; even with fast rates there was little mortality if volume of blood was not increased above 35% of normal. When death resulted it was from marked congestion of the body organs with an associated cardiac failure. Attempts have been made to use Ouabain for prevention of cardiac failure due to over-transfusion.

CLINICAL APPLICATION

The clinical uses of intra-arterial transfusion can be roughly divided into two categories:

1. Emergency procedures for individuals in profound shock from hemorrhagic or non-hemorrhagic causes.

2. Cases in which hypotension has been induced for surgical purposes, the blood being returned to the patient intra-arterially on the completion of surgery.

Emergency procedures utilizing the arterial route of transfusion have been used for years as described earlier in the history. More recently it has been employed with more success due to the increased knowledge of the underlying physiology. Aside from the emergency treatment, when intravenous therapy fails, many investigators have been utilizing the intra-arterial route for prophylaxis against shock states in surgery. Seely (51) places a blood bottle at a height of six feet which delivers pressure by gravity equal to that of the patient's normal blood pressure. If hypotension becomes manifested during surgery, blood will flow into the artery as needed. Porter and co-workers (47) have on hand an apparatus for intra-arterial transfusion which they have used many times for prophylactic therapy. Conditions of non-hemorrhagic shock for which the intra-arterial route has been used are quite numerous: Bell (3) has had successful results treating shock associated with myocardial infarction by intra-arterial transfusion. McKenzie and Morton (37) utilized this method in cases of cardiac

arrest where cardiac massage and the administration of oxygen were carried on simultaneously; the results were usually successful.

Intra-arterial therapy in conjunction with induced hypotension was first described by Gardner (18) who successfully used it while removing an olfactory groove menningioma. Since then other types of cardiovascular and neurological procedures have been accomplished using this method. As described earlier, Love and co-workers (36) used it successfully in removal of an epidermoid tumor of the third ventricle.

SUMMARY

Intra-arterial transfusions were first used by Landois in 1875 for cases of shock associated with severe frostbite. Since that time this procedure has been used in various types of hemorrhagic and non-hemorrhagic shock. The important factor is a minimal loss of time in instituting therapy so that the shock does not become irreversible. A working rule of 50 millimeters of mercury pressure for systolic blood pressure has been used with success to determine when to begin therapy by the intra-arterial route.

Another rule is to try intravenous therapy in multiple ports before beginning intra-arterial transfusion, in cases of hemorrhagic and non-hemorrhagic shock. The advantages of blood given intra-arterially in shock states are:

1. Rapid administration (1000 cubic centimeters in very few minutes) which is faster than the intravenous route and is less likely to overload the heart.

2. Rapid elevation of the systemic blood pressure.

3. Dilation of the coronary arteries with resultant oxygenation of the myocardium.

4. Decrease in the sino aortic reflex with subsequent peripheral vasodilation.

5. Decreases the cerebral anoxemia and increases renal and hepatic blood supply which helps prevent irreversible shock.

6. Smaller amounts of blood are needed than in intravenous infusions.

7. It can be performed even in the face of marked venous collapse.

8. It allows rapid recognition of bleeding points at surgery which would otherwise remain obscure if hypotension had been treated by intravenous transfusion.

9. It permits rapid return of blood to patients in surgery who have had hypotension induced for surgical purposes.

The apparatus used should be of simple design and immediately available for use should the need arise. A sterile tray should contain the necessary tubing, connections, and adaptors so that a blood storage bottle may instantly be incorporated into the apparatus. The manometer and bulb may be taken from a sphygmomanometer when needed. Also included in the tray should be the necessary instruments to dissect out the artery, and a 15 gauge needle for insertion into the selected site. To reduce vasospasm, 1-2% novocaine should be available in this kit. A pressure of 20-50

millimeters of mercury above that of the patient may be used safely, gradually increasing this pressure as the patient's blood pressure responds. During the transfusion great care should be exercised to prevent occurence of air emboli. Signs of over transfusion should be watched and the transfusion discontinued before its occurence; a manometer for measuring venous pressure is most helpful in following the patient. In the event blood is not available, plasma or parenteral fluids may be used until blood is available; the clinical use of hypertonic solutions should be withheld until more information is obtained regarding their application to shock states. On completion of the transfusion, the needle is withdrawn and moderate pressure is exerted over the puncture site to control hemorrhage. Make every effort to spare the artery used for the transfusion to decrease the chance of ischemia and gangrene distal to the site of injection. If the puncture defect is large, a few sutures may be used to close it; gelfoam may be helpful in some cases. When the systemic blood pressure becomes sufficient to permit venapuncture, the intravenous route may then be employed. Poor prognostic signs are: persistent falling of blood pressure with no tendency towards stabilization; large amounts of blood readily taken by the patient in shock; and a decreased response to vasopressor drugs, all of which are indicative of vasomotor collapse.

CONCLUSION

I. There are definite indications for arterial transfusions.

II. In the face of severe hypotension which will not respond

to intravenous therapy, no contraindications exist to the use of intra-arterial transfusion.

III. The apparatus should be simplified but complete, and the ease of sterilization and availability of its use are of prime importance.

IV. This method may be utilized in conjunction with induced hypotension for surgery.

V. Any artery may be employed as the site of injection.

VI. The artery need not be ligated following transfusion.

VII. Blood substitutes such as plasma and parenteral fluids may be used if blood is not available for immediate use; however, blood should be obtained at the earliest time possible.

VIII. Hazards do exist - air emboli; eschemia gangrene, and over-transfusion. These may be minimized as described earlier.

IX. Intra-arterial transfusion presents a means of rapid elevation of a lowered blood pressure, and when properly used may be life saving.

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