METHODS OF FLUID ADMINISTRATION IN THE TREATMENT OF SURGICAL SHOCK

AN EXPERIMENTAL COMPARISON

BY

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The present time of battlefield and air-raid casualties gives the subject of surgical shock an ever-increasing importance.

The frequent low level of pain and the cessation of haemorrhage in severe injury are beneficial effects of shock. Individuals, however, vary in their reaction to the causes of shock: the sensitive faint "at the sight of blood," while the phlegmatic remain unmoved. In many cases shock seems needlessly severe, so that failure of the circulation and death occur.

A great number of investigations have been made in the past into the pathology and treatment of shock, and the many somewhat conflicting facts and theories argued tend to produce confusion, with the result that recognition and treatment of shock are not above criticism. It cannot be too clearly emphasized that early treatment brings its reward even in severe cases, but the neglected case of severe shock may progress to the stage of peripheral circulatory failure, and energetic treatment may then be of no avail. It was towards these severe cases of secondary shock that this investigation was directed.

Pathology of Surgical Shock

Shock is a condition of cerebral anaemia with depression of the higher centres of the brain, consequent on a reduction in volume of the active circulating blood. It is commonly divided into two clinical types.

Primary shock is due to nervous impulses, either central or peripheral in origin, which, by reflex stimulation of the vasomotor centres, cause a sudden fall in blood volume and pressure. Recovery is the rule in a comparatively short time. Some cases, however, persist with sluggish circulation, and merge gradually into secondary shock. In rare instances primary shock has been fatal.

Secondary shock may follow after an interval of a few hours or be continuous with primary shock, the symptoms increasing slowly and insidiously. It is the result of several factors acting simultaneously, all of which tend to reduce the blood volume—e.g., pain, fear, toxæmia, and shortage or loss of body fluids—as a result of haemorrhage, oedema, vomiting, sweating, and diarrhoea. It is aggravated by long exposure to cold, fatigue, the effects of certain anaesthetics, and asphyxial states. Secondary shock is to be regarded as serious because, although many cases recover spontaneously, the altered conditions of circulation may persist and gradually become worse unless treatment is prompt and efficient. Occasionally a severe state of secondary shock occurs rapidly, and is termed "collapse."

Diminished Blood Volume.—The marked fall in circulating blood volume produces a corresponding fall in arterial blood pressure. Some cases of shock may have a normal blood pressure for a short time, but this always falls as shock increases. It has been found that the larger arteries and veins are relatively empty and contracted in shock and that the heart is not filled to capacity. There is evidence to show that most of the blood remains in the vast capillary network of the body (capillaries of muscles, intestines, skin, etc.). This capillary stagnation is brought about by an active dilatation of these minute

vessels. Much has been written as to the actual cause of this dilatation. It has been shown to occur experimentally by the action of toxins, histamine, or histamine-like substances. Such toxins, for instance, are absorbed in cases of burns, infections, and intestinal strangulation. It was thought until recently, after the work of Cannon, Bayliss, Dale, and others, that traumatized tissue, especially muscle, was the source of a histamine-like body. Capillary dilatation has also been shown to occur as a result of reflex nervous stimuli, as in primary shock. These nervous stimuli may be sensory—e.g., pain—or they may be central in origin—e.g., fear—and in the case of secondary shock may be long continued. The investigations of Slome and O'Shaughnessy support this view and discount tissue toxæmia as a cause of traumatic shock.

Peripheral Circulatory Failure.—As a result of capillary dilatation the volume of circulating blood is reduced in the great vessels and heart. Effects of this reduction are, first, that the vasomotor system attempts to compensate by an outpouring of adrenaline and a contraction of the arterial side of the circulation. This phenomenon may keep the blood pressure normal for a short time, but a fall in blood pressure occurs later in all cases of shock. A vicious circle is produced. The lowered blood pressure reduces the *vis a tergo* in the capillary circulation, which is also slowed as a direct result of the increased diameter of the capillaries. On account of this slow circulation the blood remains long in the capillaries and finds difficulty in reaching the veins. These are consequently poorly filled, and blood is hindered from reaching the heart. The heart is not filled to capacity and its output falls, further lowering the arterial blood pressure. Secondary effects occur as the result of stagnation of the capillary circulation: plasma passes out of the capillaries into the tissue spaces, leaving blood which is more viscid. The capillary cellular content rises: in severe cases of shock the red cell count from capillaries has been found as high as 9,000,000 per c.mm.

Anoxæmia.—Capillary blood is venous in shock, and as the result of prolonged anoxæmia the endothelium is damaged, becoming more permeable to fluids and salts. Plasma and salts enter the tissue spaces, leaving capillary blood viscid and the circulation further hindered. Other associated processes lower the volume of circulating fluids: direct loss of blood by haemorrhage (external, internal, or haematoma), and loss of body fluids locally by oedema at the site of trauma or by sweating, vomiting, and diarrhoea. The general effects of this stagnant circulation are seen in a lowered heat production, diminished basal metabolic rate, and a greatly lessened output of urine. These changes in the worst cases result in a failure of the circulation—so-called peripheral circulatory failure—and lead to death.

Spontaneous Recovery from Shock

In recovery, haemorrhage ceases owing to the favourable conditions for clotting. Rest and warmth allow the circulatory elements to recover their tone; the blood volume is restored by water drunk by the patient to alleviate thirst. With removal of the disturbing factors—pain and fright—recovery is the rule, slowly and gradually.

In this investigation a number of experiments were performed on laboratory animals with the object of comparing the results of different methods of treating traumatic shock. Cats were used in the following experiments. The animals were anaesthetized by intraperitoneal injections of urethane (1.5 grammes per kg. of body weight). A control tracing of the carotid blood pressure under this anaesthetic showed no fall in blood pressure over a period of three hours. A tracheal tube was

inserted in every animal after anaesthesia in order to eliminate variations in blood pressure due to temporary respiratory obstruction. Blood-pressure tracings were recorded by means of an indwelling carotid cannula. Shock was obtained by trauma. One hind leg of the anaesthetized animal was struck several times with a hammer. The nature of the trauma was constant, and consisted of laceration of the thigh muscles with comminution of the femur. The skin of the animal in most cases remained intact. This method of trauma was used after the manner of many early investigators. It produced a true picture of traumatic shock, the blood pressure falling on an average by 50%. In some cases the injury was augmented by movements of the injured limb until the blood pressure had fallen 50%.

These experiments were chiefly concerned with comparing methods of restoring blood pressure by the addition of fluids to the organism. In many cases the experiment was made much more severe by the formation of clot in the carotid cannula after shock had been present for some hours. Removal of the clot always produced additional haemorrhage, which in the circumstances had a serious adverse effect on the experimental animal. The use of anti-coagulants, however, was not considered advisable having regard to the method of causing shock. The animals were not allowed to regain consciousness, and were killed at the termination of each experiment. The temperature of the experimental animals was kept constant throughout.

Effect of Intravenous Infusions

1. *Normal Saline*.—Temporary recovery readily occurred on the addition of normal saline intravenously in most cases of shock. The rise in blood pressure usually lasted from a few minutes to half an hour, after which time it returned to the previous shock level or even lower. In severe shock intravenous normal saline had little or no effect whatever in raising blood pressure. These results confirm the findings of many early investigators, and are now widely accepted.

FIG. 1.—Temporary effect of intravenous normal saline in shock.

Fig. 1 shows a tracing of the carotid blood pressure of a shocked cat. Each step represents a period of two minutes. At A, 50 c.cm. of normal saline was given by the intravenous route; an immediate gradual rise in blood pressure occurred, which returned almost to the previous shock level after eighteen minutes. At B the process was repeated, with similar results.

Quantitative Limit of Intravenous Saline.—Where shock is well marked the blood pressure may have fallen 50%, and the circulating blood volume may be 60% to 70% of the normal total quantity of blood. In order to restore blood volume and pressure large quantities of normal saline would have to be given, but this fluid passes rapidly through vascular endothelium into the tissues, which explains the temporary beneficial effect. There is a practical limit, therefore, in using intravenous saline. When it is given in too large a quantity oedema of the lungs occurs. Often about 20% of the normal blood volume can be given in the early stages of shock. Cardio-vascular disease, and degenerative changes in the vascular endothelium from prolonged anaemia and anoxaemia, greatly increase the risk of lung oedema.

In order to avoid cardiac failure intravenous fluids as a rule should be given slowly by the drip method. The

risk of oedema of the lungs is greatest in the advanced stages of shock.

2. *Hypertonic Fluids*.—The administration of intravenous hypertonic saline in cases of shock seems to act in two ways:

(a) An immediate rise of blood pressure is obtained. The response is sudden, and is so marked that it bears little relation to the amount of hypertonic fluid given.

FIG. 2.

Fig. 2 is a tracing of carotid blood pressure in severe shock of five and a half hours' duration. Each step represents a five-minute period. At Y, 25 c.cm. of hypertonic 5% saline with 2% glucose solution was injected into a vein. An immediate rise of blood pressure occurred, which then fell gradually over a period of thirty-five minutes to a little above the original shock level. The pulse pressure during this period was increased, as is shown by the larger vertical depth of the tracing.

The sudden high rise in blood pressure noted in this series of experiments where intravenous hypertonic saline was given was thought to be due to a direct action on the blood vessels, probably arterioles and capillaries, causing them to contract, thus increasing the peripheral resistance of the circulation.

(b) The prolonged effect of intravenous hypertonic solutions in raising the blood pressure is partly due to the altered osmotic pressure of the plasma in the circulation, which causes reabsorption of tissue fluids.

Effect of Fluid Absorption by the Tissues

Water Absorption via Alimentary Canal.—In spontaneous recovery from shock water is taken to alleviate thirst and is rapidly absorbed, making up the deficient blood volume.

FIG. 3.—Recovery from mild shock by water absorption from colon.

Fig. 3 is a tracing of carotid blood pressure in mild shock which recovers from 100 to 130 mm. Hg. The rise in blood pressure is gradual and sustained, and of more than an hour's duration. It follows the introduction of 50 c.cm. of water into the colon at A. Blood pressure in shock can frequently be restored by water absorbed from the intestines. However, in many cases the alimentary canal does not tolerate fluids well and vomiting occurs; fluids may then be given subcutaneously.

Water Absorption via Subcutaneous Route.—Isotonic saline or glucose given by subcutaneous injection over a prolonged period restores blood volume and pressure in shock.

FIG. 4.—The beneficial effect of subcutaneous fluid.

Fig. 4 shows the result after normal saline was administered subcutaneously in large quantities to the shocked cat (A, B). The blood pressure rose from shock level continuously over a period of three hours; it then appeared

to be stabilized. Each step in the tracing represents a period of five minutes. This effect on the blood pressure in shock was repeatedly obtained in experiments, and contrasts remarkably with the temporary benefit on using intravenous normal saline. In certain experiments subcutaneous fluid produced no effect whatever on the blood pressure, and the animals died without showing any signs of recovery. These fatal cases of shock were always severe cases of long standing, in which the circulation was probably too stagnant for the mechanism of subcutaneous absorption to occur.

Hypotonic Saline

It has been found that hypotonic saline 0.5% when administered by the subcutaneous route is rapidly absorbed in most cases of shock, and causes an increase of blood pressure.

FIG. 5.

Fig. 5 is a tracing of carotid blood pressure in the shocked cat. Each step represents a five-minute period. At A, 300 c.cm. of 0.5% saline was given subcutaneously and caused a gradual sustained rise of blood pressure over a period of seventy minutes and with evidence of continuing.

This result was typical of all but the most severe cases of shock; in these, absorption from the tissues appeared to be absent.

FIG. 6.

Fig. 6 is a tracing of carotid blood pressure in severe shock. Each step represents a five-minute period. At A trauma was followed by a moderate fall in blood pressure over an hour; further injury at B caused a pronounced fall, which was maintained. At C, 100 c.cm. of 0.5% saline was injected subcutaneously; 200 c.cm. was also injected at D, but the blood pressure was not altered.

The Osmotic Gradient

From these results it is seen that the state of shock may be benefited by the addition of fluid to the circulation. Temporary effects are to be expected by giving intravenous normal saline, but far more prolonged raising of the blood pressure occurs by the absorption of water from intestines or saline from the subcutaneous tissues. It is probable, however, that in the severest cases the circulation is too stagnant for absorption of subcutaneous fluids or intestinal contents to occur. In these conditions it is possible to stimulate the circulation by intravenous addition of hypertonic fluids and so enable water to be absorbed by osmosis from tissues or intestines.

In Fig. 2 (a tracing of carotid blood pressure in severe shock of five and a half hours' duration), at X, 200 c.cm. of hypotonic 0.5% saline was given subcutaneously with no benefit, after fifteen minutes, to the blood pressure. At Y, 25 c.cm. of hypertonic 5% saline and glucose 2% was injected into a vein, with an immediate high rise of blood pressure. A gradual fall occurred in the following thirty-five minutes, but the blood pressure began to rise steadily and gradually from this point for a further twenty-five minutes owing to osmotic absorption.

The increased osmotic pressure of the active circulation brings about absorption of tissue fluids of lower osmotic

pressure: an osmotic gradient is present. Such a condition can be maintained artificially by slow intravenous drip of hypertonic saline coincident with the provision of an adequate fluid reserve in the tissues. The use of hypotonic saline 0.5% for a fluid reserve as well as the administration of water by natural channels would seem to be a logical method of preserving this osmotic gradient. On many occasions hypotonic saline measuring more than twice the normal blood volume was given subcutaneously without causing oedema of the lungs in the experimental animal; whereas normal saline amounting to a fraction of the normal blood volume given directly into a vein might produce this complication, although the heart appeared to be beating strongly.

Summary

Some methods of replacing fluids in the shocked experimental animal are compared.

The temporary beneficial effect of intravenous saline in shock is again shown.

Hypertonic saline given intravenously directly stimulates the circulation in shock. The rise in blood pressure is probably prolonged by osmotic absorption of tissue fluids also.

Fluids absorbed by the capillary circulation from intestines or subcutaneous tissues may restore blood pressure permanently in shock.

No absorption occurs by capillaries in severe shock.

Some evidence is brought to show that hypotonic saline may be absorbed by the tissues in severe shock and produce a sustained rise of blood pressure when the circulation is stimulated by hypertonic saline and an osmotic gradient is maintained.

The clinical application of the above principles of fluid administration is proceeding. Up to the present several cases of severe shock have been successfully treated, and in one instance the blood pressure recovered from the low level of 30 mm. Hg.

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Local education authorities have been asked to provide instruction in simple first-aid measures for older boys and girls, the instruction being given where possible by teachers or the school staff holding recognized certificates. Where no such teacher is available it may be possible to obtain suitably qualified instructors through the local A.R.P. authority. A Board of Education circular to this effect states that instruction should be given on simple dressings, burns, arrest of haemorrhage, artificial respiration, and, in general, how to render such immediate help as will prevent further injury.