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Shock

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Shock is a term that is used with various meanings. By the laity it may refer to an emotional upset such as the shock of hearing of a misfortune to a friend or an acute experience as for instance an electric shock, or a sudden subjective feeling such as the shock of hearing a loud noise.

In its purely medical application, shock refers to a clinico-pathological state, a symptom complex brought about in various ways and a fundamental feature of which is an acute and persisting deficiency of blood supply to the body tissues.

Clinically

The state of shock is recognisable as a clinical entity: in shock the patient may be restless or in coma, the skin is pale or cyanosed and may have a blotchy appearance, it is cold particularly in the extremities, the nail bed, blanched by pressure over the nail, regains colour slowly, the skin feels clammy and there may be beads of sweat on the brow, the patient's pulse rate is rapid, the quality poor and easily obliterated by a palpating finger, the blood pressure is much reduced and may be unrecordable. All these features are manifestations of a reduced peripheral circulation.

Essential Feature

Shock is the penultimate stage to death, as it is on an adequate circulation that the life of each cell in the body depends. Unicellular forms of life such as the amoeba are able to assimilate nutrient materials direct from their environment as also can simple multicellular forms. For more complex organisms, in which specialisation of tissue has occurred, the development of a circulatory system has been necessary to distribute nutrients throughout the body to the tissue cells, the most immediate requirement of which at any time is for oxygen which should be properly regarded as a food. A failure of adequate circulation results in anoxia for the tissues which are then obliged to metabolise anaerobically. Cellular metabolic disorder is consequently a fundamental feature of the shock state leading to cellular acidosis and ultimately the death of the cell.

Definition

Shock may therefore be defined as any acute haemodynamic disturbance which causes such a degree of reduced capillary flow that tissue hypoxia, of a degree leading to functional and or morphological changes, occurs.

This is an important subject as shock is a condition liable to be encountered by all practitioners regardless of speciality. Although Mr. Lean is to follow with a talk on "Obstetric Shock" it must not be thought that there is a type of shock exclusive to obstetrics. More properly perhaps the term "shock as encountered in obstetric practice" should be used. Mostly the causes of shock in obstetric practice are blood loss (hypovolaemic shock) and sepsis (septicaemic shock) for example due to septic abortion.

Normal Circulation

A normal circulation depends on three factors—the actions of the cardiac pump, vaso motor tone and an adequate blood volume—the latter two factors together constitute the peripheral resistance in the familiar equation BP=cardiac output x peripheral resistance. Blood pressure then is seen to be an expression of an adequate circulation.

The situation can be likened to a three legged stool which will stand up provided the three legs are intact; take any of the legs away and it will collapse. In like manner the circulation will collapse if any of its three props are removed and a state of shock results.

Shock then is the common end point of the action of various mechanisms operating in different clinical conditions.

Shock as a term is less informative then when it is qualified by a word which indicates the mechanism by which it was brought about, for instance, cardiogenic (cardiac pump), seques tration (vasomotor tone) and oligaemic (blood volume).

As much of the shock met with clinically is due to reduced blood volume a useful working classification of shock is a broad subdivision into normovolaemic and hypovolaemic.

Sequelae

It has been said of shock that "not only does it stop the works but it wrecks the machinery", referring to disordered cellular metabolism leading to death of the cells. In this respect the different tissues of the body vary in their susceptibility to a depleted circulation and hypoxia; highly specialised tissue such as nervous tissue and tissues of high metabolic activity such as the myocardium rapidly succumb in the face of hypoxia whereas connective tissue and skin for example are more resistant, but parenchymatous organs such as kidney and liver are much less so. There is then a range, or spectrum, of susceptibility to hypoxia exhibited by the various body tissue. Again tissues vary in their immediate importance to the organism for survival, the heart and brain for instance are of paramount importance and for this reason compensatory haemodynamic adjustments are made to ensure adequate perfusion of these vital structures, in the face of circulatory failure generally, at the expense of further depleting circulation to the rest of the body. Such conservation of blood flow for the coronary and cerebral circulations is achieved by selective vasoconstriction elsewhere. While the immediate objective of keeping alive the heart and brain is gained other organs suffer from the effect of oxygen lack and undergo progressive damage which may be irreperable if hypoxia is not relieved by timely restoration of the circulation. The importance of prompt and adequate treatment of shock is based on the anticipation of this sequence of events.

Among the organs liable to be damaged by sustained depleted circulation are the kidneys. In varying degrees of severity tubular necrosis and cortical necrosis may occur resulting in acute renal failure and uraemia as a complication of shock. Similar liver necrosis may take place with consequents liver failure.

Normovolaemic Shock

The blood volume is unchanged in shock states brought about by reduction of the cardiac pump action and vasomotor tone. In the first instance cardiac output may suddenly fall due to such causes as myocardial infarction, toxic myocarditis of bacterial toxaemia, for example diphhteria, hypo and hyper kalaemia, pulmonary insufficiency of diverse origin leading to respiratory acidosis, hypercarbia and anoxia. Secondly, loss of vasomotor tone arising from bacterial toxaemia, so called septic shock, especially encountered with gram negative organisms, spinal cord injury and hypoadrenalism leads to pooling of blood in the periphery, a fall of venous pressure and consequent reduced filling of the heart in diastole, which is a passive process, and lowered cardiac output.

Hypovolaemic Shock

Reduction of blood volume results from the loss of any of the fluid elements of the blood. Most obviously, loss of whole blood by haemorrhage externally or internally leads to hypovolaemic shock. Transudation of plasma from burn sites and water and electrolyte loss via the gastrointestinal tract in intestinal obstruction, fistulae and diarrhoeic states such as cholera are other ways in which the blood volume may be gravely depleted.

Treatment of hypovolaemic shock aims at restoring the blood volume to normal by intra venous administration of the appropriate fluid, blood, plasma, saline or a macro molecular so-called plasma expander such as dextraven. Recently a plasma expander called Rheomacrodex has become available with the desirable quality of preventing sludging of blood in the tissue

capillaries and thus maintaining blood flow in the smallest vessels.

How much fluid should these patients be given? Various criteria can be used. Simply and universally applicable, sufficent fluid to restore circulation to normal as manifest by the improved clinical state of the patient. A circulation has been restored when the patient's blood pressure and pulse quality and rate returns to values normal for him, bearing in mind the patient may be normally hypertensive. Usually the aim should be to maintain the patient's systolic blood pressure over 100 mm Hg. The following are also indications of a satisfactory circulation—warm skin surface especially of the nose, ear, hands and feet, rapid capillary filling, healthy colour and bloom of skin and mucous membranes and absence of sweating, urinary output of more than 30 ml. an hour. Continuous catheter drainage of the bladder is helpful in order to accurately measure urinary output in these cases.

The blood volume depletion requiring correction may be calculated using I^{131} albumen. A known quantity of this radioactive material which equilibrates within the vascular space is injected intravenously. From the dilution of radioactivity assessed by scintillation counting of a test specimen of the patient's blood taken after a short interval the blood volume can be calculated. An apparatus, the volumetron, is now commercially available to enable this estimation to be carried out within ten minutes at the patient's bed side.

Adequacy of blood replacement can furthermore be gauged by monitoring the central venous pressure. This is simply accomplished by passing a polythene tube from the antecubital vein into the superior vena cava and placing it in communication with a saline infusion flask

TABLE

Causes of Systemic Arterial Hypotension	Classification of Shock
Blood loss	Exanguination hypotension
Loss of body fluids	Dyhydration hypotension
Venodilatation: Orthostatic venous distension	Sequestration hypotension
Pericardial tamponade	Ventricular compression hypotension
Myocardial infarction	Coronary insufficiency hypotension
Vasovagal fainting	Autonomic imbalance hypotension
Traumatic injury	Visceral afferent hypotension; somatic afferent hypotension
Peritonitis	Splanchnic dilatation hypotension
Crush syndrome	Reactive hyperaemia hypotension
Anaphylactic reaction	Allergic hypotension
Bacterial (septicaemic)	Septicaemic hypotension

and a manometer by which pressure is recorded in terms of cms of saline with reference to the manubrium sternum. Transfusion of the patient can safely continue until the central venous pressure starts to rise above the normal 3 cms of saline beyond which a state of overfilling of the circulation can also be observed clinically as jugular vein engorgement.

"Irreversible" Shock

Patients who are suffering from shock which fails to respond to treatment are said to be in a state of irreversible shock. In the past there has been some doubt as to whether such an entity existed or whether it could always be attributable to misdiagnosis or inadequate treatment. Conditions which have masqueraded under the title of irreversible shock are cardiac tamponade, fat embolism, and continued or unsuspected internal haemorrhage. More recently, however, liver damage with the release of vasodilator material (V.D.M.) and changes in the reticulo-endothelial system interfering with its ability to deal with endotoxins of bacterial, particularly invaders from ischaemic gut, have been postulated as mechanisms leading to irreversibility. It follows that the shock state must not be regarded as irreversible until every diagnostic possibility has been entertained and every therapeutic possibility fully exploited. It is a diagnosis arrived at by exclusion rather than on any positive grounds.

Treatment Of Shock

The principles of the treatment of shock are firstly that it must be expeditious and secondly that the circumstances which have lead to circulatory failure must be rectified. This presupposes that the cause is known, but it is not always so. The clinical features of shock are virtually the same, irrespective of the mode of production, and ascertaining the cause may pose a difficulty in some cases. Usually, however, the antecedent history, the events leading to circulatory collapse and the manner of onset are strongly indicative. Investigations may be called for such as electrocardiography, Chest X-ray, haematocrit and haemoglobin estimations, blood chemistry and culture, and peritoneal aspiration.

Oligaemia can be corrected by blood or plasma or by so-called plasma expanders such as Dextraven or the newly available Rheomacrodex.

Restoration of vasomotor tone may require the administration of vasopressor drugs for example nor-adrenalin Vasoxine, Methedrine, Aromine, and Angiotensin or hydrocortisone.

The action of the cardiac pump will be supported by digitalisation and measures designed to improve oxygenation of the blood in the pulmonary circulation such as clearing obstructed bronchi by bronchosocopy suction and positive pressure automatic respiration; also correction of electrolyte deviations and accidosis.

Antibiotics are indicated when circulatory failure has a septicaemic basis and hypothermia is of value in a non-specific way by reducing the requirements of the tissues for oxygen.

Surgical intervention may be required for the arrest of haemorrhage or draingae of septic foci.