THE DIAGNOSIS AND TREATMENT OF SHOCK

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Shock is a physiological disturbance characterized by a discrepancy between effective circulating blood volume and the total capacity of the peripheral vascular bed. The key here is "effective circulating blood volume," for clinical and experimental evidence demonstrates that death from shock may occur in the absence of any external losses of body fluids. Rather, the pathological disturbance involves a failure of appropriate distribution of oxygenated blood to vital body organs, particularly the liver, kidney and gastro-intestinal tract.

It is obvious that death from shock must be due to a failure in energy production at a cellular level. A unifying concept of all types of shock is that this tissue failure is due to ischemic hypoxia regardless of whether the initiating event is hemorrhage, fluid or plasma loss, endo- or exo-toxemia or myocardial failure.

Pathophysiology of Haemorrhagic Shock - It is well recognized that trauma alone is sufficient to cause an outpouring of adrenaline and nor-adrenaline from the adrenal medulla. Trauma coupled with blood loss is even more potent and the amount of circulating catecholamines rises to very high levels. Increased sympathetic activity also results in liberation of excess nor-adrenaline at myoneural junctions. Together this results in intense vasoconstriction in precapillary arteriolar and postcapillary venular sphincters, particularly in the skin which becomes pale, cold and clammy, and in the viscera, and to a lesser extent in muscle. This increase in peripheral resistance initially offsets the failing cardiac output and the central arterial blood pressure is maintained. This protects the perfusion of heart and brain whose vessels are not affected by this sympathetic discharge. If fluid loss continues the intense vasoconstriction will not be sufficient to prevent a fall in blood pressure. Ischemic hypoxia of the tissues forces the use of the inefficient anoerobic pathway of metabolism with production of elevated blood pyruvic and lactic acids, an increase in serum inorganic phosphorus, and a reduction in blood pH, pCO_2 and ketone levels. Acidosis results in the peripheral arterioles being less responsive to catecholamines, although the venules are less affected. More catecholamines are liberated to counteract this. Eventually the arteriolar constriction fails and blood pours into the capillary beds. It cannot get out, however, because of the venular constriction and stagnation occurs. Hydrostatic pressure increases and fluid begins to leave the capillaries with increasing rapidity and amounts. Venous return falls lower and lower, reflected in decreasing cardiac output and diminishing coronary perfusion. Inadequate myocardial oxygenation, coupled with inefficient cardiac metabolism in the face of acidosis may result in abrupt death due to arrhythmias or gradually, due to myocardial failure.

Replacement of fluid losses early in this sequence of events will reverse the vicious chain and recovery will occur. If replacement is delayed until the period of stagnant anoxia and pooling has ensued it is ineffective, although it will delay the inevitable for a few hours.

Endotoxin Shock - The toxin produced by the Gram negative bacteria responsible for this syndrome is a complex lipopolysaccharide. It produces its effect by its sympathomimetic properties resulting in intense vasospasm, both by increasing plasma levels of catecholamines and secondly, by combining with some substance in the blood to form a potent vasoconstricting material. The deleterious action is due to the same mechanisms responsible for irreversible haemorrhagic shock. It is well known that infusion of adrenaline can cause exactly the same result.

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Coronary Shock - The shock accompanying myocardial infarction is fatal in approximately 70% of cases. Oddly enough, the same mechanisms as outlined above again come into play. That is, there is an abrupt increase in plasma catecholamine levels with resultant increase in total peripheral resistance. It is not hard to visualize the deleterious effects that increased resistance coupled with diminished venous return would have on a damaged heart.

The Clinical Syndrome of Shock - In this age of mass slaughter on our highways and internecine warfare in our cities the most common cause of shock is blood loss. The history and the nature of the injuries makes this diagnosis fairly straightforward. Similarly the shock accompanying gross plasma and blood loss in burns, and extracellular fluid loss in severe diarrhoea and/or vomiting is usually diagnosed without difficulty.

The major problems arise with shock developing in already ill patients in hospital for treatment of illnesses of varying severity. This is particularly obscured in post-operative patients. Here one is most often dealing with elderly arteriosclerotic people who often have had previous myocardial infarctions and who have undergone extensive surgery for malignancy or other serious illnesses. The picture is often confused by the presence of infection, or chronic respiratory insufficiency, or both. Nevertheless, it has been our experience that the most common initiating factor is still unrecognized blood loss, often unappreciated at the time of surgery or oozing or hidden bleeding post-operatively. It must be pointed out, however, that endotoxemia is potentiated by haemorrhage and this may add to the shock state. Hidden infection may manifest itself by sudden vascular collapse without any warning of its presence. Acute gastric dilation or pseudomembranous enterocolitis must be kept in mind, particularly in patients having abdominal surgery.

Treatment of Shock - Accepting the unified concept of shock that has been presented, one must direct treatment towards, firstly, replacing fluid that has been lost by haemorrhage, burns, gastric dilation, diarrhoea, vomiting, etc., plus that fluid effectively lost due to stagnant pooling and oedema formation in the viscera. This latter may account for 1/3 to 1/2 or more of the total normal blood volume especially in endotoxic shock.

Secondly, an attempt must be made to overcome the severe vasospasm responsible for the stagnation and diminished venous return.

Thirdly, one must protect the vital organs, particularly the heart, brain and kidneys.

Fourthly, one must overcome existing infection by diligent search for hidden loci using blood, urine, throat, and fecal cultures, and instituting immediate antibiotic therapy.

Fifthly, the vital function of respiration must be adequately maintained and the work of breathing, which may be totally exhausting in these patients, must be eased by use of respirators and tracheostomy where indicated.

Lastly, the associated metabolic acidosis should be corrected by infusion of sodium bicarbonate or Tris amine buffer.

Estimation of blood or fluid loss is notoriously inaccurate and the danger of circulatory overload is often present. It has been our practice to initiate therapy by inserting a No. 14 or No. 16 plastic needle or polyethylene tubing into the external jugular or antecubital vein and threading this catheter into the superior vena cava. This is attached to a Y connector, one limb of the Y used to deliver the intravenous fluid, the other to monitor central venous pressure. Normal venous pressure at this level is approximately 10 - 15 cm. of water. In hypovolemic shock, and indeed in the other types of shock discussed, the CVP is low, often only 2 or 3 cm. of water. Fluids can be run in rapidly and safely without fear of overloading a damaged heart as long as the CVP does not go above 15 cm. of H_2O. As long as the CVP is low the volume of blood returning to the heart is well within the pumping capacity of the heart.

The type of fluid given will, of course, depend to some extent on the underlying condition. Immediate cross matching is usually done and in most clinical situations blood will be required. If, however, the shock is due to myocardial infarction or to endotoxic shock in the presence of pre-existing normovolemic and normal hemogram, then plasma or plasma expanders are used as well.

We prefer low molecular weight dextran (Rheomacrodex, Mol. wt. 40,000) because of its small anticoagulant and antisludging properties and most of our patients receive 500 cc

of this, either initially or after blood replacement.

The use of vasopressors has been given up almost entirely on the surgical services at the Victoria General Hospital. Low doses of a vasodilator, usually chlorpromazine 12.5 to 25 mgm., are given intravenously after the fluid therapy is well underway. It was our surprising experience that the hypotension which had not responded to fluids alone or with aramine, promptly began to climb once vasodilators were given. Central venous pressure drops but can be returned to normal by increasing the fluid delivery. The chlorpromazine may be repeated every 1 or 2 hrs. as necessary. The point must be emphasized that vasodilators should not be used unless adequate fluid replacement is possible, as the total intravascular capacity may increase by a third to a half greater than normal. Often 5 or more litres of replacement fluid is required. An indwelling catheter is always inserted and hourly urine output determinations are done. A rise in urinary output indicates adequate fluid replacement. Unfortunately, many patients do not receive adequate treatment early enough and have developed anuria or oliguria. In these situations the CVP and B.P. and pulse are the only guides to adequate therapy.

In patients with cardiovascular disease oxygen by nasal catheter should be given. Digitalization should be carried out with a rapidly acting preparation. In a few cases of haemorrhagic shock we have used Tris amine buffer. This is a proton acceptor which quickly corrects acidosis both extra- and intra-cellularly. There is good experimental evidence that it will prevent or delay the development of kidney shutdown in these cases. It must be given with care to patients with respiratory problems as it will depress respirations, particularly if overcorrection to an alkalotic state occurs.

Once therapy has commenced a diligent search for hidden loci of infection must be done. The presence of a temperature is strong evidence that shock is due to or abetted by sepsis. If septic shock is suspected one cannot wait for proof but must treat immediately. Of course, blood, urine, throat and stool cultures are done. A broad spectrum antibiotics, e.g., chloramphenicol, is the drug of choice. Previous cultures are of value in picking the drug to be used.

In many patients the above measures will fail because alleviation of severe respiratory insufficiency has not been done. Mechanical ventilation will often relieve the patient of an intolerable burden of work, in addition to correcting any gas exchange problems that may be present. This is particularly true in shock associated with peritonitis or abdominal distension.

A great deal of attention has been paid to the acidosis which develops during shock. The correction of this defect by infusion of sodium bicarbonate has been considered to be of prime importance. There is no question that moderate or severe acidosis interferes with heart function and may precipitate arrhythmias. But otherwise acidosis should be considered an indication of the degree of stagnant anoxia of tissues and it will correct itself quickly if the tissue perfusion is restored. The drug should not be withheld if acidosis is severe or if tissue anoxia will be prolonged or if cardiac disease is present.

Hydrocortisone has been used for years on an empirical basis in shocked patients. There was a general clinical impression that it was helpful. Recently there has been good experimental evidence to show that its beneficial effects are due, at least partly, to its potent vasodilating action when given in large doses. We use it routinely in doses of 25 mgm/kg/ 24 hrs.

In the past year McLean of McGill has used Isuprel in the treatment of endotoxic shock. We have no experience with it for this syndrome but from a theoretical point of view it has the advantage of being inotropic to the heart, as well as being a vasodilator.

Success in the treatment of shock depends on the early diagnosis and early and continued treatment. These patients require one physician's constant care, usually for a prolonged period of many hours to many days.

Although not emphasized in the foregoing discussion there is no substitution for thorough physical examination in the initial assessment. Particular attention should be directed towards the observation of jugular venous pressure, rate and character of the pulse, careful auscultation of the chest and heart and a search for intra-abdominal pathology. The insertion of a nasogastric tube is a diagnostic procedure which should not be neglected. The electrocardiograph and

blood volume machines have been extremely helpful ancillary aids.

Summary - A concept of shock as being due to the deleterious effects of excess circulating catecholamines from a variety of causes has been presented. The use of vasodilators to overcome this has been emphasized. The importance of central venous pressure to monitor fluid administration and the use of Rheomacrodex, hydrocortisone, sodium bicarbonate and Tris amine buffer have been discussed. Cardinal clinical and diagnostic procedures have been mentioned.