INTRODUCTION

Since World War II, the study of patients in shock, has become the subjects of numerous researches and has sparked new enthusiasm to improve the present success in the management of this dreadful emergency. So, regardless of medical speciality, it has become a subject of common interest and constant discussion at various conferences and symposia. It has been agreed that shock whether associated with blood or fluid loss, trauma or neurological injury, anaesthesia or thromboembolic catastrophe, heart attack or severe infection, effective management requires a thorough understanding of the underline deranged mechanism.

Though the term "Shock" is very familiar to every clinician who treats critically ill patients, yet this physiopathological state has been defined by various workers in various ways. Some have defined it as a reversible death and therefore considered as a general homeostatic reaction to all organisms to severe and sudden stress (Adloph 1964). Whereas others used this term to denote a physiopathological syndrome occasioned by the inability to meet the needs of the tissues for oxygen and nutrients (Dietzman and Lillehei 1968). Such wide and divergent range of meaning they interpret by their definitions that most of them suffers considerable ambiguity. Voluminous literatures reflect intense disagreement over the meaning and significance of the term. Most of them, however, results from the use of a single name to describe a condition produced by many unrelated causes (Weil 1967) and can disappear if the term "Shock" is used
within the limits of its definitions (Weil 1967). Whatever may be the definition, the concept of shock as a syndrome, is well established, widely used, and if no more, it communicates the dire clinical state of the patient common to a number of diseases. It also denotes failure of circulation to meet tissue need for which three basic factors are considered responsible. The patient may be hypovolaemic (due to haemorrhage & dehydration from vomitting, diarrhoea and burns) and consequently there is insufficient fluid available to perfuse the tissues. Secondly, the shock may be of cardiac origin i.e. the heart is incapable to circulate the perfusion fluid. Thirdly, the shock may be due to altered capillary permeability and impaired function of cells to such extent that they may no longer be efficiently capable of oxygen exchange or removal of metabolites (traumatic shock). Out of the several changes occurring in shock state, those occurring at the microcirculation level in the body, are most important. They include number of haemodynamic and biochemical alterations. These changes are inter related with each other and can be considered as cause and effect.

In shock a number of biochemical changes occur due to disturbances in micro-circulation and thus in cell metabolism. It is interesting to note that though these changes appear early in shock yet they are less readily recognised than respiratory and haemodynamic changes (Ledingham 1978). Carbohydrate metabolism is impaired at an early stage. There is fall in intracellular oxygen and impairment of tissue perfusion. Thus, pyruvate the main product of anaerobic cycle,
accumulates in the blood. The major part of this pyruvate is converted into lactic acid but the rest remains in the blood. These leads to rise of lactic acid and pyruvic acid level in blood with lowering of blood pH and plasma bicarbonate level. Therefore, the increased concentration of lactic acid in blood is used as an index of the degree of intracellular anoxia (Lillehei & Dietzman 1974). But it is also reported that myocardial depression which occurs in shock, is dependent upon the blood pH level and not on the absolute amount of lactic acid present (Orkin 1965). During the period of stagnant anoxia glucocorticoid level is raised and measurable shift of sodium and water occur into the cells with leakage of potassium ions outside the cells. This cause corresponding change in their concentration in blood. There is also increase mobilisation of fatty acids with formation of significant quantities of ketone bodies. In shock, usually these substances are produced in small amounts and are metabolised by liver and peripheral tissues. These ketone bodies together with hypoxia cause further metabolic acidosis (Lillehei and Dietzman 1974). Beside this, fatty acid molecules may coalesce and form fat embolism (Ledingham, Lillehei 1978 & Dietzman 1974), which may lodge in the linings causing further oxygen deprivation. This view has been supported by clinical observation of Fleck (1976) when he demonstrated lessening of fat embolism by infusion of high concentration of oxygen. Beside adrenaline and noradrenaline, shock is associated with the release of other vaso active substances like histamine, plasma kinine, prostaglandine and angiotensin I and so forth.
In respect of biochemical changes the chief difference between haemorrhagic and endotoxic shock, lies on the length of time they require to develop stagnant anoxia. In haemorrhagic shock it may take several hours but in endotoxic shock the above changes may be evident within minutes depending on the dose of endotoxin and susceptibility of the host animal. The above biochemical changes from anaerobic metabolism are also seen in traumatic shock. There is decrease in protein formation (immunoglobulin). There is early appearance of DIG with consequent depletion of some of the coagulation factors like fibrinogen, platelets, factors II, VI and VII. Beside traumatic shock this type of bleeding diathesis is also seen in endotoxic shock. Before an effective treatment is undertaken, the clinical assessment, of the degree of the shock particularly early detection of its refractory stage, evaluation of its nature and estimation of the blood component loss, are mandatory. Previously the attention of many investigators was focused on more direct studies of clinical shock in patients. With the introduction and improvement of cardiac catheterisation technique, availability of different types of blood gas analysing equipment, improvement of pH meters with micropipette, invention of better techniques of pulmonary and kidney function tests, it is now not only possible but also practicable to carry out detailed physiological measurements on shock patients. Recording of intra arterial and central venous pressure, estimation of arterial and venous blood pH, carbon dioxide and oxygen tension recording in arterial
and venous blood and estimation of total base deficit or excess, are now actually and repeatedly carried out on critically ill patients. The availability of these dates provides the clinicians with much better informations. On the basis of these he may plan and monitor treatment with efficiency far exceeding than that previously possible.

Several research work for further improvement are in progress in different shock research centres. But a complete solution of this problem is yet to come.

All facilities were not available here yet with limited resources a project was undertaken to study the different biochemical changes that occur in the microcirculation of the patients in shock and to find out their values in the prognosis and management.