Summary. This is a very brief, superficial and biased discussion of the pathophysiologic changes in shock. It was designed to provide some insight into the very complex changes that occur, with particular attention to a few examples of the impaired cell metabolism, including changes in ATP, cAMP, and calcium. Although inadequate tissue perfusion through nutrient capillaries is the main etiologic factor in most types of shock, it is not the primary problem in many patients, particularly those with early or hyperdynamic sepsis. The importance of oxygen consumption and the possible benefits of higher hemoglobin levels are discussed to some extent because of their possible clinical application.

Key words: Shock, ATP, Oxygen Consumption.

I. Pulmonary Changes

A. Ventilation

Even relatively minor trauma or blood and fluid loss will cause hyperventilation [2]. With shock, the minute ventilation is usually at least 1 1/2 to 2 times normal. Although the tidal volume is usually reduced, the respiratory rate generally increases two — three fold and provides much of the increased minute ventilation. This response is nonspecific and occurs even before there is evidence of increased lactate or hypoxemia. As a consequence, the PCO₂ frequently falls to 25 to 35 mm Hg or lower and, because the bicarbonate levels are still relatively normal, the pH rises to 7.45 to 7.55 or higher.

B. Oxygenation

Initially in shock, the arterial PO₂ (PaO₂) may remain relatively normal. In fact, during the early stages of hypovolemia, pulmonary compliance and function may be better than normal, because the decreased amount of blood in the lung increases the V/Q ratio in many portions of the lung. Later, however, the PaO₂ may fall rather rapidly, particularly if shock persists in spite of adequate fluid replacement. Hypoxia may develop swiftly in patients with acute myocardial infarction due to shock because these patients have a strong tendency to pulmonary congestion and edema. The combination of hypoxemia and decreased blood flow can rapidly result in severe damage to all vital organs.

II. Cardiovascular Changes

The complex pathophysiologic processes involved in shock have often been compared to simple plumbing.
problems with abnormalities involving either the pump, pipes or fluid in the system. However, many patients with early septic shock [3] and some patients with acute myocardial infarction (A.M.I.) shock [4] have a normal or increased cardiac output with little or no evidence of impaired tissue perfusion.

A. Cardiac Output

1. Normal Values
The normal cardiac output for the young healthy male adult averages about 6 liters per minute (3.3 L/min/M²), and the overall normal range is 2.5 - 4.0 L/min/M². The cardiac output of females, who generally have less muscle and more fat, is about 10 percent less than that of males of the same body size. With increasing age of the patient, the cardiac index decreases about 0.2 L/min/M² per decade. Thus, at 5 years of age, the C.I. is about 4.3; at 10 years it is 4.0; at 20 years - 3.7; at 40 years - 3.1; at 60 years - 2.7; and at 80 years - 2.5 L/min/M².

2. Factors Affecting Cardiac Function and Cardiac Output
The main factors that affect cardiac function and cardiac output include the pre-load (filling of the heart during diastole), the afterload (resistance against which the heart must pump), the contractility of the heart, the coordinated pattern of contraction and the heart rate.

a. Pre-Load. Pre-load refers to the load or tension on a muscle as it begins to contract. In the heart, this term refers to the pressure or quantity of blood in the ventricle at the end of diastole. In hypovolemic shock, the main cause of the reduced cardiac output is inadequate filling of the heart. To help correct this problem, vascular capacity, predominantly in the large veins, is reduced by up to 25%. This action is partially passive but may also be caused to some extent by increased sympathetic nervous activity.

b. After-Load. In certain respects it might be said that the main determinant of right ventricular output is the venous return and the main determinant of left ventricular output is the resistance against which it must pump. If total peripheral systemic resistance (TPR) is reduced to half, cardiac output will usually double (if venous return is adequate).

On the other hand, if the TPR is increased, stroke volume and cardiac output tend to fall. For example, if vasopressor agents are used to correct hypotension, the blood pressure can usually be made to rise, but the TPR may increase so much that the cardiac output falls and peripheral tissue perfusion is reduced. In this circumstance “cosmetic” improvement of the B.P. results in a reduction of effective cardiovascular function.

The mechanism relating ventricular activity to the blood pressure is the so-called baroreceptor reflex. Hypotension in the carotid sinus causes a reflex stimulation of the vasomotor center and sympathetic nervous system which in turn increases myocardial activity and peripheral vasoconstriction. In contrast, an increased B.P. tends to decrease the stimuli from the carotid sinus to the vasomotor center. This decreases the sympathetic stimulation of the heart thereby decreasing ventricular contractility. Vagal tone also tends to increase with the rise in B.P. thereby slowing the heart rate.

c. Myocardial Contractility. A heart that is capable of pumping greater quantities of blood than normal against a standard amount of resistance (such as a heart that is hypertrophied or is strongly stimulated by sympathetic impulses) is considered to have increased myocardial contractility.

Stimulation of the beta adrenergic receptors results in an increased force of myocardial contraction (inotropic effect) and a faster heart rate (chronotropic effect). It also causes a slight amount of vasodilation in the arteries in skeletal muscle and some venous constriction. The net result is an improved circulation to the tissues. This response is an important part of our defense against shock, and patients who fail to develop an appropriate tachycardia with shock or sepsis have a poor prognosis.

d. Coordinated Contraction. Failure of the individual muscle units in the heart to beat in a coordinated fashion greatly impairs cardiac function and output. Some of the more frequent causes of uncoordinated myocardial contraction include 1) ischemia or trauma to the conduction system of the heart (producing various types of bundle branch blocks), 2) ischemia or trauma to portions of the myocardium (causing them to beat slower and/or weaker than normal adjacent muscle), 3) various arrhythmias due to drugs, ischemia or unknown factors and 4) excessive dilatation of the ventricle.

e. Pulse Rate. During shock, the pulse usually increases to at least 120 - 130 per min due to stimulation of the sympathetic nervous system. This helps to obtain the best possible cardiac output with the blood volume that is available. If venous return is reduced or the stroke volume is limited, as may occur in patients with myocardial fibrosis or scarring, bradycar-