The Shock Lung
Pathophysiology and Treatment

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The term SHOCK LUNG is used to designate the catastrophic clinical syndrome of progressive respiratory failure.

The lung is one of the organs most severely damaged by the shock state. It is estimated that more than 50% of the patients who die following shock have severe pulmonary complications and die in respiratory failure. The term shock lung was used for the first time in 1927. The synonyms for the shock lung are:

— Wet Lung
— Progressive pulmonary insufficiency
— RDS — Respiratory distress syndrome
— DIC Lung
— Posttraumatic pulmonary collapse
— Septic Lung
— Congestive atelectasis
— Da — Nang Lung
— Vietnam Lung
— Blast lung

Shock lung follows diverse conditions as:

— Shock syndrome
— Sepsis
— Aspiration pneumonia
— Fatty embolism
— Massive transfusion
— Hyperhydration — fluid overload
— Oxygen toxicity
— DIC
— Haemorrhagic pancreatitis
— Narcotic overdoses
— Virus pneumonia
— Chemical poisons

After a latent period following the shock state, or other life threatening conditions, shock lung syndrome is usually present — characterized by hypoxia with markedly deceased oxygen tension.

The progressive respiratory failure in shock lung is related to the response of pulmonary cells with a change in a cellular membrane metabolic phenomenon and pathophysiologic changes in the respiratory membrane. Inadequate blood flow to the lungs interferes with local cellular metabolism, causing multiple pathologic changes. One such alteration is intravascular microaggregation involving platelets, leukocytes, erythrocytes and formation of fibrin — containing thrombi which further impede blood flow. Another cellular change occurs in the endothlial cells of pulmonary capillaries. The first abnormality noted is swelling and disruption of the mitochondria in the pulmonary capillary endothelial cells. The endothelial cells begin to swell and, as in inflammatory process, the intracellular spaces open, permitting the escape of fluid, protein molecules, platelets, white blood cells, and even red blood cells into the interstitial spaces.

FIG. 1.   Disturbances of Pulmonary Microcirculation

Cellular aggregates of leucocytes, which adhere to the pulmonary epithelium, may act as mediators of hydrolytic enzymes due to their lysosomal breakdown.

FIG. 2.   Cellular damage of Shock Lung

The lysosome's enzymes can destroy normal cell membranes and damage capillaries. Furthermore, lysosome's enzymes can activate M.D.F. (myocardial depressant factor), and can cause activation of complement factor in alternative pathway called chaotic complement activation with its destructive effects to pulmonary endothelial cells. The platelet aggregates accumulating in pulmonary capillary beds may release substances such as: kinins, histamine, prostaglandins and serotonin, which increase capillary permeability. Through the damaged pulmonary capillary wall transudation of fluid into pulmonary interstitial space and into the alveoli occurs, resulting in dilution and inactivation of surfactant leading to the characteristic change in compliance (alveolar atelectasis).

FIG. 3.   Cycle leading to Shock Lung

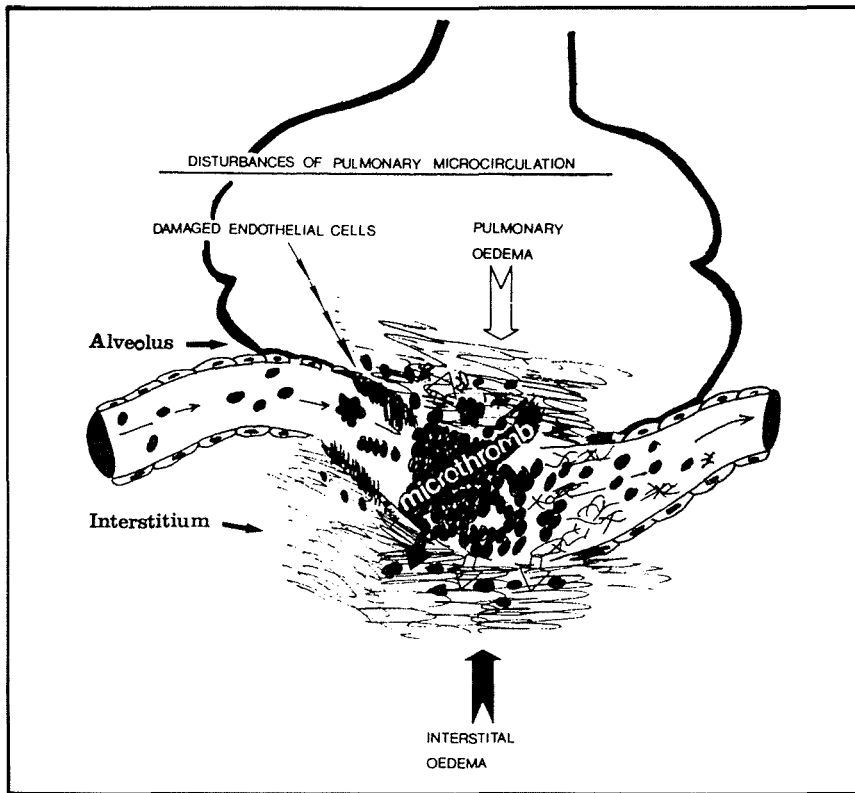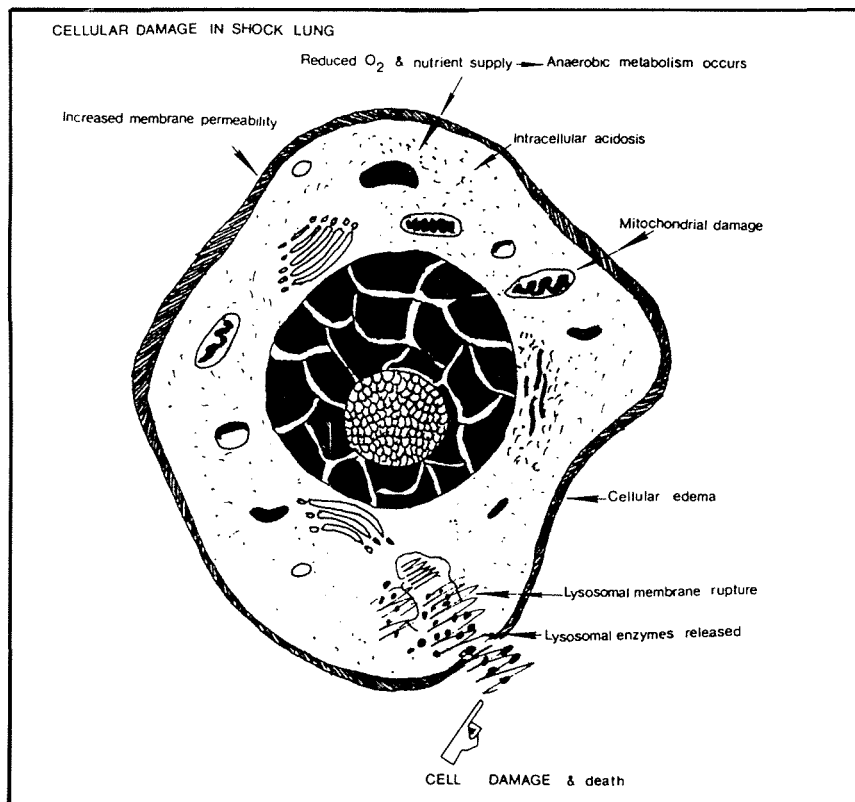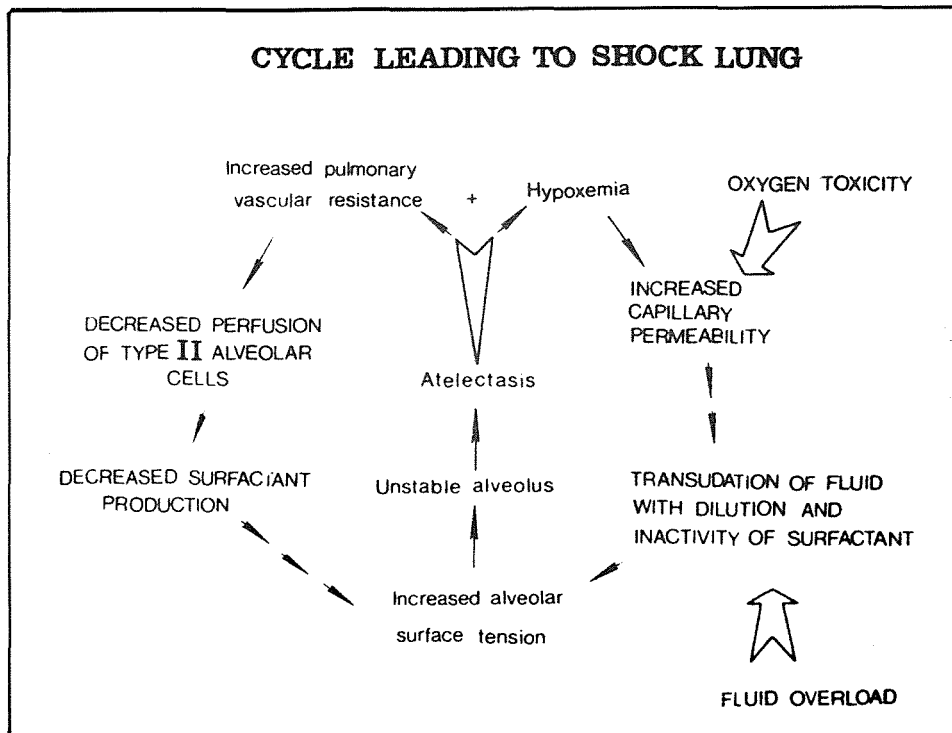Alveolar atelectasis and consolidation in areas of both lung fields, together with pulmonary oedema

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FIG. 1

DISTURBANCES OF PULMONARY MICROCIRCULATION

DAMAGED ENDOTHELIAL CELLS

PULMONARY
OEDEMA

Alveolus →

microthromb

Interstitium →

INTERSTITAL
OEDEMA

FIG. 2

CELLULAR DAMAGE IN SHOCK LUNG

Reduced O_2 & nutrient supply → Anaerobic metabolism occurs

Increased membrane permeability

Intracellular acidosis

Mitochondrial damage

Cellular edema

Lysosomal membrane rupture

Lysosomal enzymes released

CELL DAMAGE & death

FIG. 3

CYCLE LEADING TO SHOCK LUNG

Increased pulmonary vascular resistance + Hypoxemia

OXYGEN TOXICITY

DECREASED PERFUSION OF TYPE II ALVEOLAR CELLS

Atelectasis

INCREASED CAPILLARY PERMEABILITY

DECREASED SURFACTANT PRODUCTION

Unstable alveolus

TRANSUDATION OF FLUID WITH DILUTION AND INACTIVITY OF SURFACTANT

Increased alveolar surface tension

FLUID OVERLOAD

leads on to a decreasing respiratory lung field which cause acute progressive respiratory failure resulting in hypoxia and hypercarbia.

Scattered and confluent areas of consolidation and atalectasis in both lung field can be seen in the X-Ray examination of the lungs.

FIG. 4.   Chest X-Ray of two patients in Shock Lung

According to blood gas changes, chest X-Ray examination and, clinical evidence Shock Lung syndrome can be divided into four phases.

FIG. 5.   Phases of the Shock Lung

If patients have been intubated and mechanically ventilated following operative procedures or during resuscitating, the typical clinical manifestation of shock lung may be absent. In these circumstances, frequent investigations of arterial blood gases is necessary.

Management of patient in clinical syndrome of shock lung should consist of:

1. Supporting the respiratory system
2. Supporting the cardiovascular system
3. IVI fluid regime
4. Control of infection
5. Management of condition which was caused

shock lung
6. Frequent chest physiotherapy
7. Use of adjunctive drugs, e.g. Methylprednisolone 30mg/kg every 6 hours for 48 to 72 hours.

The treatment should be started as early as possible, as soon as the diagnosis is made.

FIG. 6.   Indication for commencement of artificial ventilation

During treatment of shock lung, we should pay attention to two frequent complications from vigorous treatment: fluid overload and oxygen toxicity which may deteriorate clinical syndrome of Shock Lung.

During treatment of severely injured patients, lots of "weak links" were discovered during resuscitation of patients. During World War I, the "weak link" was hypovolaemic shock.

During World War II, patients who recovered after severe injury, were resuscitated successfully from the circulatory impairment due to haemorrhage. The patients have been suffering from failure to the next "weak link", the kidneys – acute renal failure. In the Vietnam conflict, renal failure was understood much better and better treatment salvaged more severely injured patients. The lung has become now a major clinical problem.
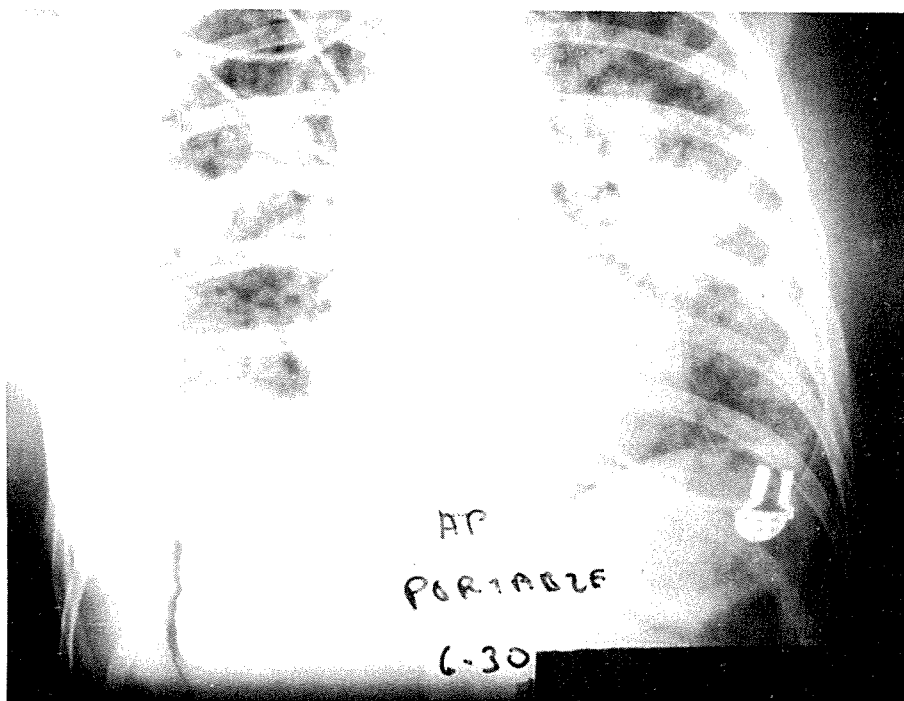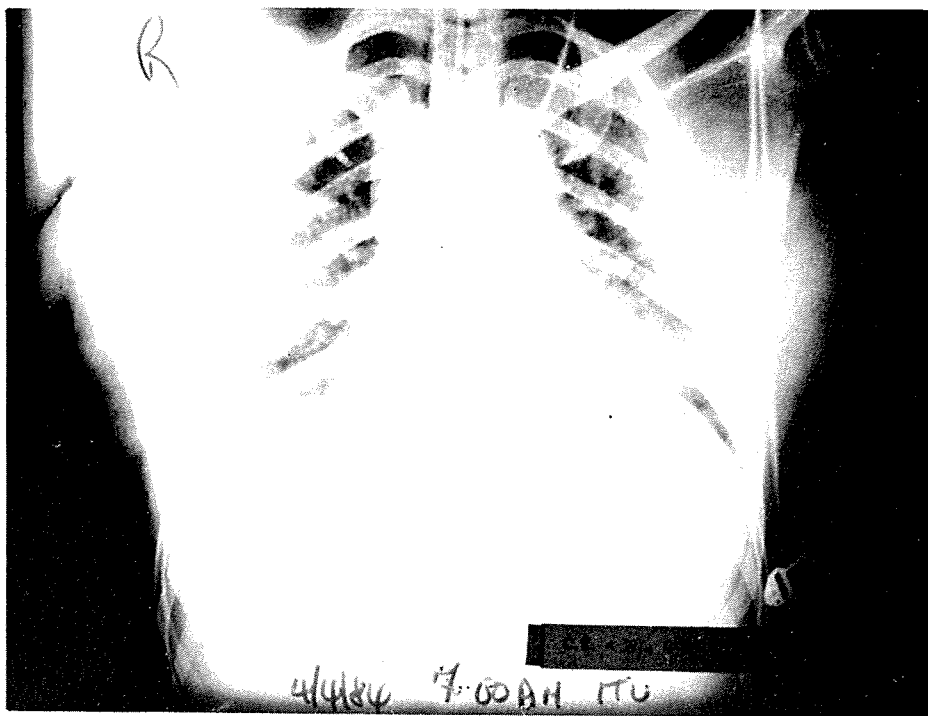
FIG. 4

X - RAY In SHOCK LUNG

PHASES	CLINICAL EVIDENCE OF SHOCK LUNG	CHEST X-RAY	WHILE BREATHING ROOM AIR FiO_2 .21			
			PaO_2 kPa	$PaCO_2$ kPa	$A\text{-}aDO_2$ kPa	QS/QT %
NORMAL	Ø	Ø	10.6-13.3	4.6-6.0	0.7-13	5-8%
1 Injury and Resuscitation	None	Normal	9.3-12.0	4.0-5.3	2.7-5.3	10-20%
2 Subclinical SHOCK LUNG	Mild to moderate tachypnea	Minimal or no infiltrate	8.0-10.6	3.3-4.6	4.0-6.6	15-25%
3 Established SHOCK LUNG	Increasing tachypnea	Increasing edema and confluence of infiltrates	6.6-8.0	2.7-4.6	5.3-8.0	25-40%
4 Severe SHOCK LUNG 3 stages	Obvious RESPIRATORY FAILURE	INCREASING OPACIFICATION OF THE LUNG	6.0-8.0 5.3-7.2 4.6-6.6	3.3-5.3 4.6-6.6 6.0-8.0	6.6-9.3 7.2-10.0 8.0-10.6	30-50% 40.60% 50-70%

This concept of "multiple links in a chain" has been well described by Bane, who states that we are now about to see the next "weak link" in the shock syndrome, the brain.

In experimental haemorrhagic shock, cortical and cerebrospinal fluid (CSF) pO_2 fell to approximately one third of the basal levels. The brain lactate levels doubled, within five minutes and continued to rise. Cerebral cellular integrity could not be maintained because intracellular components "leaked", and there was a rise in CNS potassium and pseudocholinesterase.

In more severe shock state, cerebral anatomical damage and death were demonstrable histologically.

Pulmonary effects of brain disfunction in shock syndrome were demonstrated by a lot of authors in their experiments.

Gerald Moss, MD, PhD and colleagues experimenting with dogs, produced isolated cerebral hypoxia by perfusing the brain with the animal's own mixed venous blood instead of arterial blood.

After two hours of perfusion, with perfusate pO_2 approximately 35 mmHg, 4.5 kPa, lethal progressive respiratory failure — Shock Lung developed.

The gross and microscopic examination of the canine's lungs showed the pattern of shock lung. Similar results were confirmed by Webb and by the anaesthesiology research group at the University of Texas at Golveston. Webb's group noted that the physiological and anatomical derangements of shock lung could be prevented by pulmonary denervation. Webb's group subjected denervation of canine left lung prior to haemorrhagic shock. The denervated lung remained normal, while the innervated right lung developed the complete pattern of shock lung.

Neurological impairement may persist long after the initial hypoxic insult is over.

It can be postulated that CNS disfunction in autonomic control of the pulmonary vasculature is a major factor in the development of the acute progressive respiratory failure — Shock Lung — so frequently seen following severe trauma and shock.

FIG. 6

INDICATION FOR COMMENCEMENT OF ARTIFICIAL VENTILATION

RESPIRATION PARAMETERS		NORMAL VALUE	VENTILATION	
			ELECTIVE	VITAL INDICATION
Oxygenation	PO_2 kPa FiO_2 0.21	10.7-12.0	6.7-8.0	<6.7
PCO_2 & Dead space	PCO_2 kPa VD/VT	4.0-5.3 0.3-0.4	6.0-7.3 0.5-0.6	>7.3 >0.6
Oxygenation	PO_2 kPa FiO_2 1.0	73.3-84.0	26.7-40.0	<26.7
Inspiratory force	kPa (Cm H_2O)	7.3-9.8	2.4-4.9	<2.4
Alveolar-Arterial Oxygen differences	$P(A-aDO_2)$ kPa FiO_2 0.21	0.7-2.7	7.3-7.9	>8.0
Alveolar-Arterial Oxygen differences	$P(A-aDO_2)$ kPa FiO_2 1.0	2.7-8.0	46.7-60.0	>60.0
R-L Physiological shnt	QS/QT (%)	3%-8%	30%-40%	>40%
Tidal volume	ml/kg	6-8	3.5-4.5	<3.5
Vital capacity	ml/kg	50-60	10-15	<10
Respiration rate	Respiration/min	12-16	30-35	>35

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