MEDICAL GRAND ROUNDS

Parkland Memorial Hospital

May 20, 1965

ACUTE PULMONARY EDEMA AND RELATED FORMS OF ACUTE RESPIRATORY INSUFFICIENCY

I. Definition - Pulmonary edema is a state of excessive accumulation of fluid in the extravascular spaces of the lungs. The fluid may be located in the interstitial spaces only, or in both interstitial spaces and intra-alveolar spaces. The colloidal content of the fluid may be high or low and may be accompanied by a variable amount of blood elements due to hemorrhage or diapedesis.

II. Pathogenesis

A. Normal fluid balance relationships - The only force tending to keep fluid within the capillary is the plasma oncotic pressure, ranges 30-37 cm. H_2O, which nevertheless counters the sum of the forces tending to move fluid out of the capillary:

1. Capillary blood pressure 10-15 cm. or less
2. Tissue fluid oncotic pressure 18 cm. or less
3. Surface tension pressure 4 cm. or less
4. Intrapulmonary pressure 4-8 cm.

No simple consideration of these forces will explain the appearance of pulmonary edema in every case but rather there is usually a complex interplay of a number of mechanisms.

B. Mechanisms

1. Hydraulic - Low colloid content
   - a. Excessive infusion
   - b. Hypertensive agents and CNS reflexes
   - c. Cardiac failure
   - d. Vagotomy
   - e. Vascular obstruction
   - f. Increased alveolar surface tension
   - g. Decreased intra-alveolar pressure
2. Osmotic - Low colloid content
   - a. Impair protein production
   - b. Dilution
   - c. Protein loss
   - d. Salt water drowning
3. Altered Permeability - High colloid content
   - a. Excess capillary distension
   - b. Capillary damage
     - (1) External
     - (2) Internal
   - c. Tissue alteration by intrinsic and extrinsic chemical agents

d. Ischemia
e. Other less certain mechanisms such as genetic and neurohumoral disorders

4. Altered Lymphatic Function

III. Physiological Alterations
Figure 1

IV. Clinical Manifestations
A. "By the bed-side, how many false diagnostics have I not witnessed - - - What is the source of such mistakes? I repeat, it is the deficiency of correct physiology." J.N. Corvisart: An Essay On The Organic Diseases And Lesions Of The Heart And Great Vessels.
B. Interstitial Phase
C. Intra-alveolar Phase
D. Complications
1. Angina
2. Asthma
3. Shock
4. Arrhythmias
E. Roentgenological Features
F. Cases

V. Treatment
A. Physiological
B. Pharmacological

Case #1: [redacted]

This 68 year old [redacted] man with moderately severe kyphosis scoliosis was first admitted to [redacted] in 1963 when he presented with acute dyspnea following a long history of progressive exertional dyspnea and orthopnea. Rales were heard in the lungs and a presumptive diagnosis of acute pulmonary edema was made. The patient was given morphine after which he became apneic, cyanotic, unresponsive and hypotensive. Blood gas studies revealed the patient to be suffering from severe respiratory insufficiency with respiratory acidosis and profound hypoxia. The patient improved immediately with effective oxygenation and ventilatory assistance by inspiratory IPPB and 100% oxygen by controlled rate. A tracheostomy was performed. Following a stormy course the patient was discharged, recovered and was able to resume his normal activities. No pulmonary function evaluation was made. Unfortunately, he failed to continue his pulmonary therapy program and he continued to smoke. Slowly exertional dyspnea, orthopnea, and daytime somnolence returned and on 10-20-64 the patient again was brought to the emergency room in severe respiratory distress. His blood pressure was found to be 220/120. Rales were heard in the lungs, and in the absence of the patient's previous record, again a diagnosis of acute pulmonary edema was made in spite of the fact that the patient's admitting complaint was asthma. Again he was given morphine 8 mg. and mercuhydrin 2 cc.. Following this the patient suddenly became apneic, hypotensive and unresponsive. Again controlled ventilation by inspiratory IPPB and high oxygen was instituted while a tracheostomy was performed. Arterial blood studies revealed a pH of 7.01, PCO_2 in excess of 100 mm. Hg., and since oxygen was administered the oxygen saturation was 97%. He rapidly improved with effective oxygenation and ventilatory assistance along with the removal of copious amounts of thick secretions. Respiratory distress was controlled with the administration of ventilatory assistance and bronchodilator agents, as well as an intensive bronchial hygiene program. Ventilatory function studies following recovery revealed a severe restrictive and obstructive ventilatory defect with vital capacity no more than 1 L.. Blood gas studies are listed below.

	[redacted]-64		[redacted]-65
	Rm Air Rest	100% O_2 Rest	Rm Air Rest
CO_2 tension, mm.Hg.	60	63	45
pH	7.4	7.36	7.43
CO_2 content, mm./L.	33.3	33.6	
Hematocrit	25	26	40
Hemoglobin	7	7	13
Oxygen saturation, %	83	99	92
Oxygen tension, mm.Hg.	56	429	
A-a O_2 gradient	30	277	

The studies of [redacted]-64 reveal evidence of a shunt of approximately 14%, evidence of moderate hypoventilation, and uneven distribution of ventilation with respect to blood flow.

This case is an example of the importance of instituting physiological therapy immediately and avoiding the use of the classical therapeutic procedures, especially sedation, for the treatment of what is presumably acute pulmonary edema. Aside from the fact that the primary indications for morphine in this case are not clear, the inherent dangers in using sedation in a patient with severe kyphoscoliosis and chronic obstructive pulmonary disease are forcefully illustrated. Moreover, this case clearly demonstrates how hypoxia is an important mechanism in the development of shock and how it responds dramatically to adequate oxygenation and ventilatory assistance.

Case #2: [redacted]

This 75 year old [redacted] male confined to a wheelchair in a nursing home which burned was brought to the emergency room in an unresponsive state with severe respiratory distress and inspiratory moist rales throughout the lungs, and frothy carbon flecked secretions being produced in large quantities. The clinical and radiological picture was that of acute pulmonary edema. His vital signs were stable with a blood pressure 90/75, pulse 120, and respirations labored at 36-40/min.. EKG revealed myocardial change compatible with an old posterior myocardial infarct. The patient's prognosis was considered guarded, largely because of his age and the magnitude of his injury, as well as the fact that he had a history of having had asthma for years prior to this event. He improved dramatically with a decrease in respiratory distress and slowing of the rate to 25/min.; his B.P. rose to 130/80 and pulse slowed to 96. His oxygen saturation on room air breathing was found to be 80%, on 100% oxygen breathing 96%. His lung compliance was 75 ml./cm. H_2O. A tracheostomy was performed, and he was placed on continuous assisted ventilation by IPPB at 20-25 cm. water pressure with oxygen concentrations in the inspired air between 60 and 70% which were found necessary to maintain adequate oxygenation. Intensive bronchial hygiene with continuous heated mist therapy, intermittent bronchodilator decongestant aerosol, postural drainage and tracheal aspiration were carried out. His course was followed by frequent measurements of volume exchange in relation to applied pressure and blood gas studies while continuing intensive steroid therapy, at first intravenously and subsequently orally, starting with 60 mg. Prednisone daily, and gradually tapering off during his hospital course. He was given maintenance parenteral fluid and electrolytes with intensive antibiotic therapy, including Kanamycin, Colymycin, and Methicillin. During the first few days of his hospital course, his pulmonary compliance gradually decreased as the magnitude of the pulmonary congestion reached its natural peak. At the same time the oxygen defect also increased as his oxygen saturation remained in the neighborhood of 73% when tested on room air breathing. The hypoxic defect was largely a result of uneven distribution of ventilation with respect to blood flow (blood flow through areas of non-ventilated lung). The magnitude of the defect is reflected by the fact that the arterial PO_2 was found to be 342 mm.Hg. on 100% oxygen breathing, representing approximately a 20% shunt. His condition

gradually improved over the next two weeks and his blood gas exchange gradually returned to near normal levels with an oxygen saturation on room air breathing of about 92% and his compliance gradually improved from a low of 30 cc./cm. water pressure. The cuff to the tracheostomy tube was removed. A smaller tube was placed in the trachea which was plugged, and the normal procedures for extubation were carried out and well tolerated.

This is an outstanding example of intensive and effective management of acute pulmonary edema due to smoke exposure with secondary pneumonitis in a very old, disabled man in whom the prognosis would ordinarily be virtually hopeless or at best extremely poor.

TYPES OF PULMONARY EDEMA

	Hypersympathetic	Hyposympathetic or Neuroparalytic
Anatomical:	immediate, generalized hyperemia with venous engorgement	late, patchy hyperemia without engorgement
Physiological:	initial high systemic and PA pressure	initial normal or low systemic and PA pressure
Pharmacological:	ameliorative effect by sympatholytic agents	no ameliorative effect by sympatholytic agents
Clinical:	hypertension and hypersympatheticotonia	hypotension or shock
Radiological:	uniformly dense lung fields	patchy and eventually reticulogranular
Examples:	epinephrine, asphyxia, CNS injury, NH_4 toxicity, hypertensive heart disease	vagotomy, silo filler's disease, bulbar polio, altitude, cardiac failure, blast, phosgene

Acc. to Ivanhoe and Meyers
Dis. Chest 46: Aug., 1964.

ALTITUDE - ACUTE PULMONARY EDEMA

	R.A.	15 min. 100% O_2	Sea level Recovery	Altitude Recovery
pA press., mm. Hg.	144/104	76/36	47/9	56/33
mean	117	57	22	47
pA wedge, mm. Hg.	4.3	6.5	9	4
HR	129	84	68	88
RR	34	15	17	36
SaO_2, %	76	100	97	89
C.I., L/min/m^2	2.6	3.8	3.7	-
PV resist., dynes---	2900	870	310	-

Acc. to Hultgren et al
Circ. 24: March, 1964.

ELECTRICAL SHOCK - PULMONARY EDEMA

	Air	100% O_2	IPPB - O_2 45 cm. H_2O
SaO_2	50	75	92
PaO_2	30	50	80
$PaCO_2$	26	30	24
pH	7.10	7.11	7.22
A-a $\triangle PO_2$	85	620	596
V_T	300	318	1100
RR	60	55	30
BP	50/20	60/40	90/60

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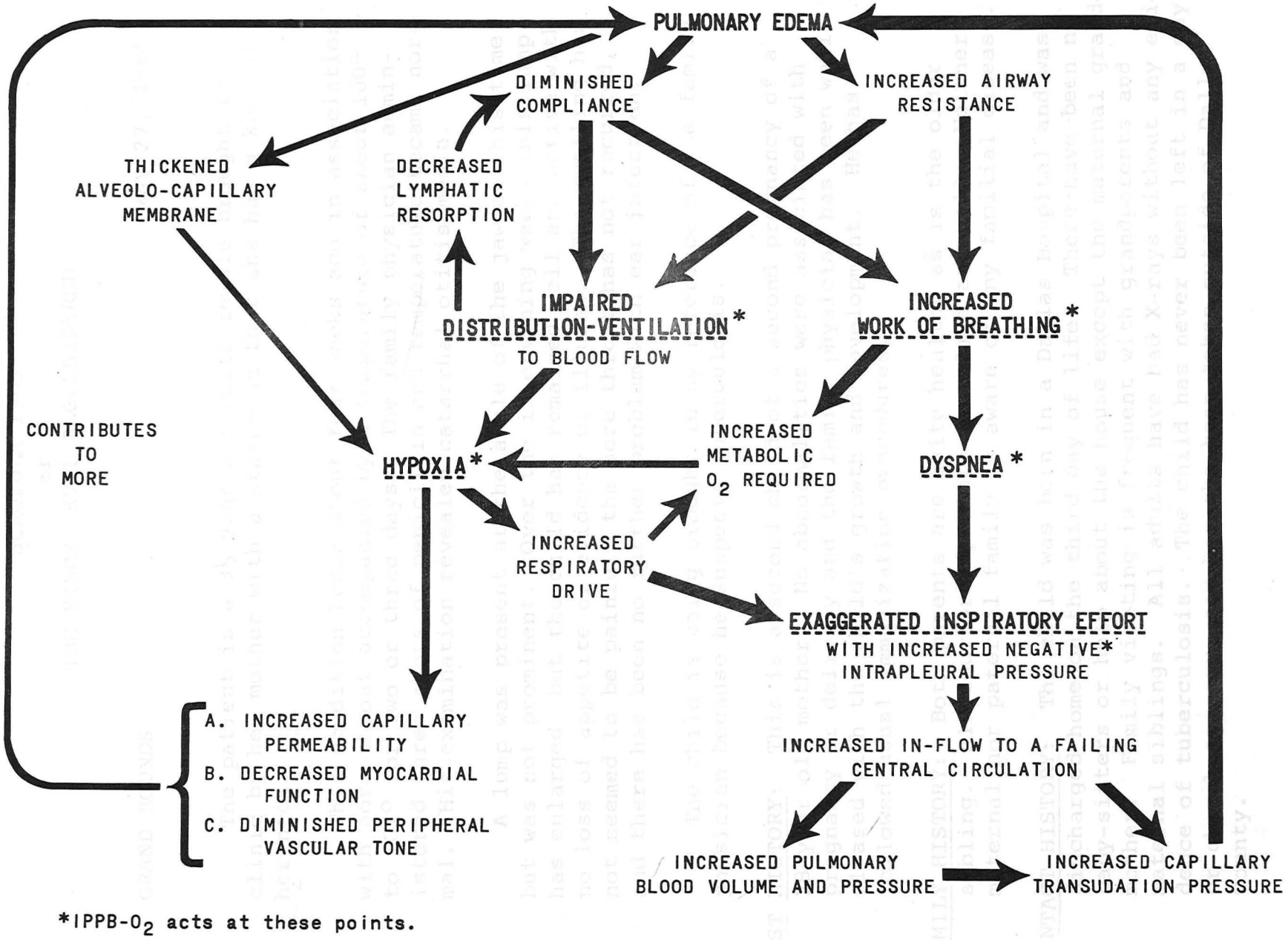

*IPPB-O_2 acts at these points.