[Pulmonary Edema]

MEDICAL GRAND ROUNDS

October 24, 1957

Case #1. A 22-year-old Negro male alcoholic was admitted with a one-day history of acute gastrointestinal bleeding. A chest x-ray made 2 months prior to admission revealed right upper lobe disease, probably pulmonary tuberculosis, but the patient refused treatment at that time. Physical examination revealed an acutely and chronically ill young man, blood pressure was not obtainable, respirations 30. There was bronchial breathing over the right upper lobe and scattered rhonchi and rales. Chest x-ray revealed a pneumonic process of the right upper lobe and diffuse nodular infiltration throughout the left upper lobe. Massive GI bleeding continued. In spite of transfusions totalling 3,500 cc., the hemoglobin never rose above 7 gm. and the patient remained markedly hypotensive. Eighteen hours after admission, his condition somewhat improved, general inhalational anesthesia and a subtotal gastrectomy were performed. Postoperatively, blood pressure was stabilized with pressor amines and blood transfusions. Progressive hyperpnea with moderate dyspnea were noted. Ultimately, severe dyspnea accompanied by bloody, frothy sputum, marked neck vein distension, and a blood pressure of 60/40 developed. 100 per cent oxygen by mask did not relieve the patient's dyspnea. IPPB with 100 per cent oxygen at 22 cm of water satisfactorily decreased his respiratory rate and relieved his dyspnea. This was continued for 4 hours until striking improvement had been achieved. Then high concentrations of 02 without pressure were used. However, it became necessary to reinstitute IPPB/1 for periods of 1/2 hour at a time every two hours to control dyspnea, until pulmonary edema gradually cleared.

Case #2. A 44-year-old Negro woman with disseminated lupus erythematosus and hypertensive cardiovascular disease was admitted to the hospital with severe dyspnea, chest pain and fever of several hours' duration. During the preceding 10 months, she had been hospitalized on several occasions for similar episodes. Each responded to antibiotics, parenteral hydrocortisone, low salt diet, and oxygen by IPPB/1. She was on digitalis. Physical examination revealed a blood pressure 200/110, temperature 101, pulse 120, respirations 40 per minute. She was gravely ill, profoundly dyspneic, was producing bloody, frothy sputum, and her neck veins were greatly distended. The heart was markedly enlarged; there was a gallop rhythm but no murmurs. The lungs were filled with bubbling rales and expiratory rhonchi. She was immediately started on oxygen and additional digitalis. Aminophylline, corticosteroids, and the hypotensive agent, Arfonad, were administered by slow intravenous drip. Improvement was only slight and the dyspnea continued to be profound. At this time the physiological studies shown in Table 2 were obtained.

SUBSEQUENTLY, THE PATIENT WAS MAINTAINED ON IPPB/I WITH 100 PER CENT OXYGEN FOR THE REMAINING 17 DAYS OF HER LIFE. PRESSURES UTILIZED VARIED, BUT GENERALLY HAD TO BE INCREASED GRADUALLY FROM 25 CM H_2O TO 45 CM H_2O IN ORDER TO RELIEVE THE PATIENT'S DYSPNEA. SHE COULD BE KEPT COMFORTABLE BY THIS METHOD, BUT UPON WITHDRAWAL OF ASSISTANCE, DYSPNEA BECAME SEVERE WITHIN A MATTER OF 5 TO 10 MINUTES AND THE PATIENT WOULD BECOME CONFUSED AND IRRATIONAL. SEVENTY PER CENT ALCOHOL WAS ADMINISTERED BY NEBULIZATION AS AN ANTI-FOAMING AGENT, AND ALTHOUGH THERE APPEARED TO BE SOME TRANSIENT IMPROVEMENT, THIS WAS SUBSEQUENTLY DISCONTINUED WITH NO SIGNIFICANT CHANGE. THE INCREASINGLY HIGH PRESSURES UTILIZED IN THIS CASE INDICATED THE PROGRESSIVE CHANGE THAT WAS OCCURRING IN HER PULMONARY SYSTEM, RESULTING IN INCREASED STIFFNESS OR DECREASED COMPLIANCE OF THE LUNG.

CASE #3. A 40-YEAR-OLD WHITE MALE 5 DAYS PRIOR TO ADMISSION HAD RECEIVED A 12,000 VOLT ELECTRICAL SHOCK WHICH RESULTED IN A 48 PER CENT SECOND- AND THIRD DEGREE BODY SURFACE BURN. DURING EMERGENCY TREATMENT AT AN OUTLYING HOSPITAL, HE DEVELOPED PROGRESSIVE RESPIRATORY DISTRESS AND WAS TRANSFERRED TO [redacted], SEMICOMATOSE WITH SEVERE RESPIRATORY DISTRESS CHARACTERIZED BY INSPIRATORY RETRACTION OF INTERCOSTAL SPACES AND EXPIRATORY GRUNTING. THERE WAS COPIOUS, THICK, SEROSANGUINOUS FLUID IN THE TRACHEA AND SEVERE RESPIRATORY OBSTRUCTION THAT COULD NOT BE CONTROLLED. TRACHEOSTOMY WAS PERFORMED WITH SLIGHT IMPROVEMENT. BLOOD PRESSURE WAS 70/40, RESPIRATIONS 40 PER MINUTE.

PHYSIOLOGICAL STUDIES WERE PERFORMED WITH THE RESULTS SHOWN IN TABLE 3. SUBSEQUENTLY, HE WAS MAINTAINED ON IPPB/I WITH 100 PER CENT OXYGEN HUMIDIFIED THROUGH A HEATED MAINSTREAM NEBULIZER IN ORDER TO AID IN THE EVACUATION OF THE THICK, SEROSANGUINOUS MATERIAL THAT FILLED THE TRACHEA. THE BLOOD PRESSURE ROSE TO 90/60, AND LATER TO 100 /70. BECAUSE OF THE PROFOUND DYSPNEA, CONTINUOUS ASSISTED RESPIRATION AT PRESSURE LEVELS RANGING MOST OF THE TIME BETWEEN 40 AND 45 CM H_2O WAS REQUIRED. THE LACK OF DIFFERENCE BETWEEN COLUMN 3 AND 4 IN THE TABLE INDICATES IT PROBABLY WAS NOT THE TIME FACTOR THAT PRODUCED THE CHANGE OBSERVED IN THE FINAL COLUMN. OVER THE NEXT 12 HOURS, THE PATIENT APPEARED TO IMPROVE CONSIDERABLY. HOWEVER, IT WAS OBVIOUS THAT HE WAS SUFFERING FROM AN OVERWHELMING SEPSIS AS WELL AS MASSIVE HEMORRHAGIC EDEMA OF THE LUNGS, AND HE EXPIRED SUDDENLY 2 DAYS AFTER ADMISSION.

POST MORTEM EXAMINATION CONFIRMED THE CLINICAL IMPRESSION OF DIFFUSE BRONCHOPNEUMONIA AND PROFOUND HEMORRHAGIC PULMONARY EDEMA. THE COMBINED WEIGHT OF THE RIGHT AND LEFT LUNGS WAS 4,680 GM. AND THEY SANK IN WATER.

Case 1

	Room Air*	100% O2 40 min.	IPPB-O2 15 min.	IPPB-O2 30 min.	100% O2 20 min.
Arterial blood					
O2 Sat. (%)	60	99	100	100	100
pO2 (mm. Hg)	36	124	188	275	140
pCO2 (mm. Hg)	36	38	28	29	--
pH	7.42	7.42	7.52	7.51	--
Alv. pO2 (mm. Hg)	108	664	674	674	--
A-a pO2 Diff. (mm. Hg)	72	540	486	399	--
Ventilation (L/min)	32.4	17.8	22.8	22.0	20.3
Respiratory rate	70	40	31	22	36
Tidal Vol.(ml) BTPS	459	445	719	998	563
CO2 production (ml) STPD	401	261	137	109	208
O2 consumption (ml) STPD	469	349	320	309	330
Blood pressure (mm Hg) (systolic/diastolic)	60/40	65/40	80/50	95/60	90/50

Case 2

	Room Air*	100% O2 60 min.	IPPB-O2 15 min.	IPPB-O2 45 min.	100% O2 20 min.
O2 Sat. (%)	31	86	95	100	95
pO2 (mm. Hg)	20	57	89	212	91
pCO2 (mm. Hg)	56	45	48	40	41
pH	7.28	7.39	7.34	7.41	7.37
Alv. pO2 (mm Hg)	95	662	660	668	665
A-a pO2 Diff. (mm. Hg)	75	605	571	457	574
Ventilation (L/min)	7.7	14.6	--	22.6	15.2
Respiratory rate	40	35	29	24	32
Tidal vol. (ml) BTPS	193	420	--	920	476
CO2 production (ml) STPD	129	242	--	200	220
O2 consumption (ml) STPD	154	251	--	340	275
Blood pressure	100/80	120/90	--	200/110	170/100

* On room air breathing for only a few minutes owing to severe asphyxia.

CASE 3

	Room Air	100% O_2 40 min.	IPPB-O_2 15 min. 25 cm H_2O	IPPB-O_2 45 min. 25 cm H_2O	IPPB-O_2 30 min. 45 cm H_2O
O_2 Sat. (%)	50	75	82	82	92
pO_2 (mm Hg)	30	50	60	62	80
pCO_2 (mm Hg)	26	30	25	26	24
pH	7.10	7.11	7.17	7.20	7.22
Alv. pO_2 (mm Hg)	115	670	690	690	695
A-a pO_2 Diff. (mm Hg)	85	620	630	628	615
Ventilation (L/min)	18.2	17.5	24.4	25.8	33
Respiratory rate	60	55	32	32	30
Tidal vol (ml) BTPS	300	318	762	805	1100
CO_2 production (ml) STPD	250	262	289	290	300
O_2 consumption (ml) STPD	264	276	320	325	340
Arterial pressure (mm Hg)	50/20	60/40	85/50	90/60	90/60

OXYGEN SATURATION DATA

Patient Diagnoses	Room Air	100% O_2	IPPB-O_2	Pressure cm H_2O
[redacted] Myocardial infarction, pulmonary edema, shock	65	80	100	15-20
[redacted] Myocardial infarction, pulmonary edema, shock	76	87	99	15-20
[redacted] Hypertensive cardiovascular disease, pulmonary edema, hypotension	74	85	100	25-30
[redacted] Poliomyelitis, pulmonary infarction, pulmonary edema, tank respirator, shock	60	75	97	20-35

In order to illustrate further the role of IPPB/I-O_2 in the treatment of pulmonary edema with shock, 4 additional cases studied by ear oximetry are summarized in the above table. In each instance, hypotension or shock was present and not corrected until after IPPB/I-O_2 was administered. In no instance was the hypotension or shock worsened by IPPB/I.