Acute Chemical Injury of the Airway and Lungs

Experience with Six Cases

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SUDDEN severe alteration of pulmonary function is a common medical emergency which requires the prompt institution of effective measures directed toward the restoration of function. Exposure of the respiratory apparatus to a variety of chemical substances or to superheated gas mixtures may precipitate such crises. The physiological and pathological responses to this type of injury follow a general pattern independent, to a large extent, of the specific nature of the chemical irritant, or the physical state of the respired mixture.

The anesthesiologist is well acquainted with methods for maintaining the functional integrity of the respiratory apparatus under a variety of circumstances. His knowledge and experience should be fully utilized in the management of patients exposed to pulmonary irritants.

This account may serve as a specific example of the utilization of the anesthesiologist as an important member of the team which cares for the pulmonary casualty.

Clinical Course and Management of Six Patients with Severe Pulmonary Injuries

At 6:00 A.M. on October 24, 1954, Philadelphia firemen were investigating a report of "leaking gas" or "ammonia fumes" from a 4,000 gallon aluminum tank used for the storage of a commercial solvent, DOW 421.*

* DOW 421 is a mixture of 4 parts ortho-dichlorobenzene; 2 parts propylene dichloride; 1 part ethylene dichloride.

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During the investigation, the tank exploded and approximately 3,000 gallons of the volatile mixture was liberated into a narrow court yard (approximately 5,500 sq. ft.) in which there were about 45 men.

Three men were killed by the blast and four died within 24 hours in other hospitals as a result of injuries and pulmonary disorders. Within two hours following the explosion, 39 firemen and policemen were admitted to the Philadelphia General Hospital with possible pulmonary, ocular or cutaneous injuries or with acute psychiatric problems. Six of these patients developed serious respiratory tract injury; three died. The other three survived, in large part because of the early recognition of the respiratory problems involved and the institution of 24-hour-a-day individualized management of the patients.

Clinical Observations, Physiological and Pathological Studies

The sequence of events following exposure to these chemical agents may be divided into four phases.

Phase 1 (0–6 Hours). Initially, there was choking and coughing which diminished in severity on removal of the victims from the site of exposure. They had only mild respiratory distress on admission. This was followed by a period of relative quiescence, lasting four to six hours. The patients showed signs of minimal bronchoconstriction and slight redness of the oropharngeal mucosa.

In light of the subsequent course, this relatively quiescent period was deceiving. The chemical agents (or the other chemicals evolved when they interacted with tissues) were relatively nonirritant to surface sensory receptors, but within six hours destroyed much of the lining of the entire upper and lower respiratory tracts.

Phase 2 (6 Hours–6 to 8 Days). This was the most critical phase. It began with edema of the oropharngeal mucosa and partial obstruction of the conducting airway (including the smaller bronchi). This was followed by a sequence of events which included pulmonary edema, atelectasis, acute emphysema and bronchopneumonia; all were present simultaneously in different parts of the lung by the end of 48–72 hours. This phase, in which three deaths occurred, was marked by the greatest impairment of pulmonary function.

Edema of the mucosa of the nasopharynx and oropharynx and larynx was apparent by inspection. A grayish-white membrane covered most of the swollen mucosa except for occasional large bullae. The lesions extended into the trachea and bronchi, narrowed the airway and led to signs of respiratory obstruction (retraction of the supraclavicular fossae and suprasternal notches on inspiration and use of accessory muscles of respiration) and of anoxemia (cyanosis, tachycardia, hypertension and restlessness).

The individuals were unable to cough effectively, probably because of the decreased velocity of gas flow that resulted from the increased airway resistance, and this resulted in further retention of exudates and secretion.

Atelectasis also developed in scattered areas distal to obstructed bronchi; these areas frequently coalesced resulting in collapse of entire bronchopulmonary segments. Late in the first day, the devitalized mucosa began to slough and tissue debris began to obstruct airways. The denuded areas of mucosa became covered by a loosely adherent fibrinous pseudomembrane.

In some areas, air appeared to enter alveoli during inspiration more readily than it could escape during expiration and acute overdistension or acute obstructive emphysema developed.

Bronchopneumonia resulted from bacterial invasion of collapsed segments and injured peribronchial structures. This occurred despite the use of antibiotic therapy. Bronchial obstruction, atelectasis, edema and pneumonia reduced the vital capacities of these patients' lungs to the range of their normal tidal volumes so that the patient was often breathing his full vital capacity with each breath.

Confirmation of the clinical diagnoses was afforded both by pathological and physiological studies (table 1). Two of the six patients died on the day after admission. Immediate autopsy showed that there was necrosis of the superficial layers of trachea, bronchi and bronchioles (fig. 1). The alveoli were not similarly affected but there was clear evidence of acute obstructive emphysema (fig. 2), atelectasis and hypostatic pneumonia in different areas of the lung. Static pressure volume curves of the lungs and thorax immediately after death (using a cuffed endotracheal tube, water or aneroid manometer, spirometer and anesthesia bag) showed such a marked decrease in the compliance of the system that no mechancial ventilator could have produced adequate pulmonary ventilation.

TABLE 1. Studies During Phase 2

	Normal Values	Patient 1 Died 30 hrs.	Patient 2 Died 34 hrs.	Patient 3 Died 6th day	Patient 4 Recov.	Patient 5 Recov.	Patient 6 Recov.
Age (years)		36	33	44	30	39	44
Height (inches)					72	68	57
Weight (pounds)					162	173	164
Surface area (m^2.)					1.95	1.91	1.84
Art. O_2 sat. % (patient breathing room air)	97	97(1)	82(1)	94(4)	90(2)	72.7(6)	84.7(6)
P_{aCO_2} (mm. Hg)	40	30	—	50	40	67	42
V_T (ml.)	500	—	—	320	640	400	400
Frequency (breaths/minute)	12–14	—	—	28–34	14	34	28
Vital capacity (ml.)	4,000	—	—	380	640	460	600
Pulm. Compliance (l./cm. H_2O)	0.2	—	0.026(D)	0.015(D)	0.137(7)	0.036(7)	0.086(11)
Pulm. resist. (cm. H_2O/l./sec.)	1.1	—	—	—	28	15.7	7.8
Work/breath (kg.-m.)	0.03	—	—	—	0.066	0.051	0.064

Number in ( ) indicates day after exposure on which it and tests below it were performed: (D) indicates test done after death.
V_T = Tidal volume.

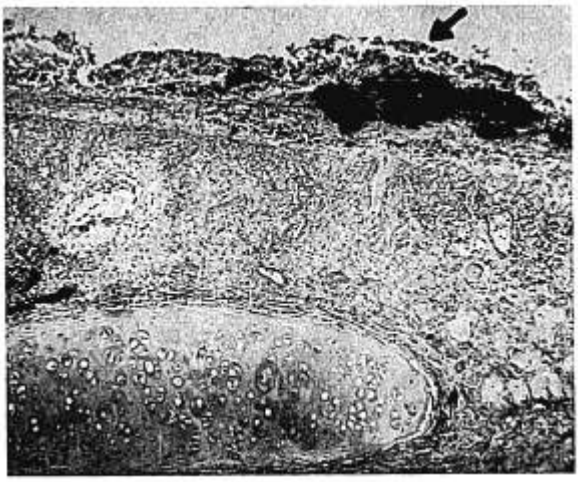

FIG. 1. Trachea in cross-section showing absence of mucosa with collapse and nonviability of underlying tissue invaded by polymorphonuclear leucocytes (case 1). Pseudo membrane consists of fibrin, cellular debris and polymorphonuclear leucocytes (arrow). (From × 10.)

The surviving patients were evaluated clinically and physiologically at frequent intervals. Physiological studies included measurements of tidal volume and frequency of breathing, arterial oxygen saturation (either by direct analysis of arterial blood or by the use of frequently calibrated ear oximeters), end-expired P_{CO_2} and pulmonary compliance and resistance (using an esophageal balloon).

The low values for compliance were probably due to exclusion of many alveoli from the ventilatory process. The restoration of the mechanical properties of the lung to near normal values was believed to result from regression of the tissue response to chemical injury and infection and removal of tissue debris and exudate. The main object of the management of these patients was then to provide adequate ventilation and maintain gas exchange until healing occurred.

Cardiac difficulties were most troublesome during phase 2. They included sinus tachycardia, right ventricular strain, congestive heart failure, ectopic auricular foci discharging the ventricle with minimal disturbance of ventricular rhythm (cases 3, 4 and 5) and auricular flutter.

Sinus tachycardia with rates of 120–130/minute developed early and was present on admission. The sinus tachycardia persisted and rates of 140–160 were observed by the end of the first 24 hours following exposure. Associated with these rapid rates were signs of congestive failure: hepatic tenderness and enlargement (2–3 cm. below the right costal margin) and engorgement of cervical veins while the patient was in a sitting position. Pulmonary symptoms caused by congestive failure were difficult to assess because of the primary pulmonary disease.

Digitalization was begun between 29 and 38 hours following exposure. Intravenous lanatoside C in 0.8, 0.4 and 0.4 mg. doses was administered at six hour intervals to achieve initial digitalization. Digalen, 1 U.S.P. digitalis unit (2 ml.) intravenously or intramuscularly once daily, was used for maintenance. One patient (case 2) died during this critical period primarily of cardiac failure.

Frequent electrocardiographic and clinical evaluation were necessary as guides to the effectiveness of the digitalis therapy; in two patients the electrical changes were indicative of digitalis toxicity at a time when clinical signs of digitalis effect or toxicity were not apparent. Quinidine in large doses was necessary to suppress ectopic auricular foci (case 5). Treatment with digitalis or quinidine was discontinued by the sixteenth day following injury.

Phase 3 (6–8 Days to 4 Weeks). During this phase, tissue repair occurred with restoration toward normal function. Visible repair consisted of granulation tissue, with no strong matrix and a relatively rich blood supply covering the areas where the normal mucosa of the upper respiratory tract had been destroyed. Later, the friable granulations frequently became dislodged, either naturally or as a result of treatment, and filled portions of the airway with debris and blood.

The presence of a troublesome "hacking" cough during phase 3 was attributed to the replacement of normal columnar epithelium with its mucus-producing cells by an abnormal tissue which failed to secrete a proper quality or quantity of mucus. Possibly the absence of cilia or of effective ciliary action was also a factor. Distressing symptoms of persistent cough ended by the time the patients were discharged from the hospital two and one-half to six and one-half weeks after exposure.

Phase 4 (After 4 Weeks). The three survivors were studied in the pulmonary function laboratory over periods of time up to 32 months after injury. The data are presented in table 2. Uneven airway obstruction persisted in case 4, even at 32 months after injury. Distribution of inspired gas was still nonuniform, airway resistance (body plethysmograph method) was twice normal and maximal flow rates were reduced moderately. However, in all cases the lung volumes and ventilation returned to normal, as did pulmonary diffusing capacity, arterial oxygen saturation and arterial P_{CO_2}. Arterial blood oxygen analyses done during the inhalation of 100 per cent oxygen showed that there were significant venous to arterial shunts present. All patients returned to work, although patient 4 was limited to work requiring only moderate exertion.

These studies show that no lasting alveolar or pulmonary capillary damage could be detected one to three years after the chemical injury. The destructive lesions of the upper and lower respiratory tract healed well in two patients and imperfectly in one, who was left with some residual increased airway resistance and uneven ventilation.

The survival of these three patients and their return to useful work shows the value of the individualized continuous therapy for such casualties.

Management of the Respiratory and Pulmonary Problems

The major task was to provide adequate ventilation for patients who had marked increase in pulmonary resistance and decrease in

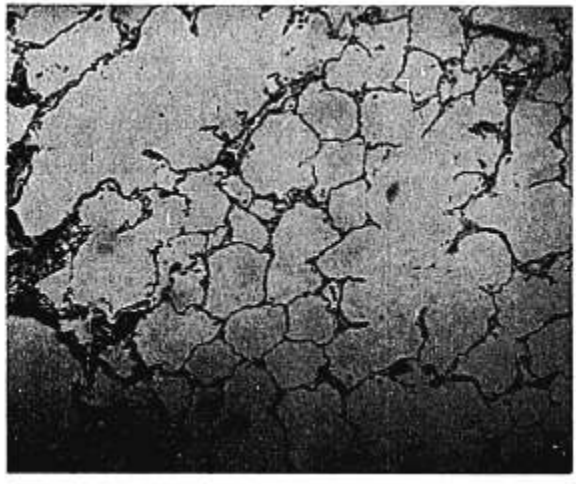

Fig. 2. Emphysema (acute) in patient surviving only 30 hours (case 1). (From × 40.)

TABLE 2. Pulmonary Function Studies 1–32 Months Following Injury

Test	Units	Patient 4					Patient 5		Patient 6		Approximate Normal Values
		1½ mo.	16–20 mo.	24 mo.	32 mo.		2 mo.	16–20 mo.	1½ mo.	16–20 mo.	
Art. O_2 sat.	%	94.6	95.5				99.6	94.9	92.2	95.3	97
Art. P_{aCO_2}	mm. Hg	33	41				32	38	36	43	40
Art. pH		7.45	7.30				7.19	7.42	7.30	7.36	7.40
I. C.	ml.	2,550	3,550	3,150	3,760		2,000	3,100	880	2,800	3,600
E. R. V.	ml.	1,000	860	790	945		1,680	1,080	1,106	790	1,200
V. C.	ml.	3,250	4,150	4,120	4,220		3,450	4,120	1,970	3,610	4,800
R. V.	ml.	2,380	2,716	2,480	2,555		1,770	1,760	1,874	2,820	1,200
T. L. C.	ml.	5,630	7,126	6,600	7,260		5,220	5,880	3,844	6,430	6,000
RV/TLC	%	42	33	37.5	35		33	30	49	44	20
F. R. C.	ml.	3,380	3,576	3,450	3,500		3,450	2,840	2,980	3,610	2,400
Resp. rate	r/minute	16	12				22	18	32	19	12
V_T	l.	0.77	0.73				0.86	0.67	0.41		0.5
$\dot{V}$	l./min.	12.3	8.8				18.9	11.7	12.9		5.0
Anat. Vp (anat.)	ml.	164							192		150
Physiol. Vp (Physiol.)	ml.	344							212		160
M. B. C.	l./min.	51	74	58	71		90	110	65	140	130
Max. Exp. Flow Rate	l./min.	115	206	101	102	226*	170	301	60	320	400
Max. Insp. Flow Rate	l./min.	130	260	159	178	216*	200	304	45	200	400
1 sec. V. C.	%	50	56	51	52	55*	66	91	60	70	83
Alv. gas uniformity	% N_2 750–1,250 ml.	3.0	3.0	4.5	0.5		5.5	2.7	5.0	2.1	1.5
Pulm. Compliance	l./cm. H_2O	0.17	0.23	0.18	0.17			0.15	0.08	0.10	0.20
Total pulm. resis.	cm. H_2O/l./sec.		3.74	4.02	3.34			2.60	2.39	1.77	1.10
Airway resis.	cm. H_2O/l./sec.	3.51	3.04	3.80	2.23		4.27	2.08	2.52	1.39	0.90
Dco	ml. CO/min./mm. Hg	27	23	27.9				34	9.8	30	22–41

* After Isuprel inhalation.

I. C. = Inspiratory Capacity
E. R. V. = Expiratory Reserve Volume
V. C. = Vital Capacity
R. V. = Residual Volume
T. L. C. = Total Lung Capacity
F. R. C. = Functional Residual Capacity
V_T = Tidal Volume
$\dot{V}$ = Minute Volume
Vp = Dead Space
M. B. C. = Maximum Breathing Capacity
Dco = Diffusing capacity for CO

vital capacity and pulmonary compliance. The following measures were used:

(1) Tracheostomy
(2) Continuous and intermittent positive pressure respiration
(3) Humidified oxygen or compressed air
(4) Bronchodilators
(5) Tracheobronchial aspiration
(6) Bronchoscopy
(7) Voluntary cough
(8) Enzymatic digestion of cellular debris
(9) Vigorous pounding on chest
(10) Frequent changes in position
(11) Compression of the thorax
(12) Antibiotics
(13) Cortisone and hydrocortisone administration.

(1) *Tracheostomy.* We recognized that some of the patients developed upper airway obstruction early, and we performed tracheostomy within four or five hours of exposure in four of the six most severely injured victims. Tracheostomy was delayed until nine and 32 hours in two who died (cases 1 and 3). Case 1 was temporized with because he did not seem, at first, to have as severe pulmonary injury as the other victims. Case 3 was transferred to the Philadelphia General Hospital 32 hours following exposure. Although dramatic relief of symptoms followed this operation, we believe that early, severe hypoxia may have caused irreversible changes which led to the ultimate deaths of these two patients.

No. 10 Magill rubber endotracheal tubes shortened to 8 cm. and fitted with inflatable cuffs and metal slip joints were more effective than conventional tracheostomy tubes. These were preferred because of (1) their larger bore, (2) their being readily adapted to conventional anesthesia apparatus, (3) the inflatable cuff prevented pharyngeal secretions

from passing into trachea and (4) a gas-tight fit was achieved. By the end of 48 hours, these tubes were no longer necessary and were replaced with conventional no. 8 metal tracheostomy tubes because continuous positive pressure was no longer needed. The patients had learned to swallow with the tracheostomy tube in place and pharyngeal secretions thereby failed to present a problem. After the first 12 hours, all tubes in tracheostomies were changed every six hours.

It should be emphasized that these patients developed severe upper *and* lower airway obstruction; tracheostomy corrected only the *upper* airway obstruction but it (1) decreased the respiratory dead space by nearly 50 per cent, (2) facilitated application of positive pressure to the lungs and (3) provided easy access to the tracheobronchial tree for aspiration of secretions and debris and for administration of nebulized drugs.

Disadvantages of a tracheostomy were that it prevented the patients from coughing properly and bypassed the normal passages for warming and humidifying inspired air or oxygen.

(2) *Continuous and Intermittent Positive Pressure Respiratory Assistance: 'Positive Pressure Breathing.'* In 1954, all of the mechanical equipment available for assisting ventilation was unsuitable. Tank respirators could not produce adequate tidal volume in the face of the unusual resistance to ventilation and further prevented the necessary frequent change in position (*vide infra* 10). Assistors, IPPB apparatus * and "suck and blow" type resuscitators or ventilators † which depend on pressure changes to actuate the valves did not function well in the presence of the high airway resistance and/or low lung and thoracic compliance observed in these casualties. The ineffectiveness of this equipment was judged by measurements of tidal volume, esophageal pressure and ear oximeter readings. Therefore, manual methods were used on these casualties. Conventional anesthesia equipment (Waters' To-and-Fro, 480 Gm. soda lime canister and 5-liter rebreathing bag) was connected directly to the improvised tracheostomy tubes (fig. 3) to produce and assist inspiration. Using a metal "Y" piece, an aneroid manometer and a clamp on a piece of rubber tubing (fig. 3) we could maintain positive expiratory pressure at 10–15 mm. of mercury. This

* Bennett IPPB.
† Emerson, Stephenson, and E. & J. resuscitators.

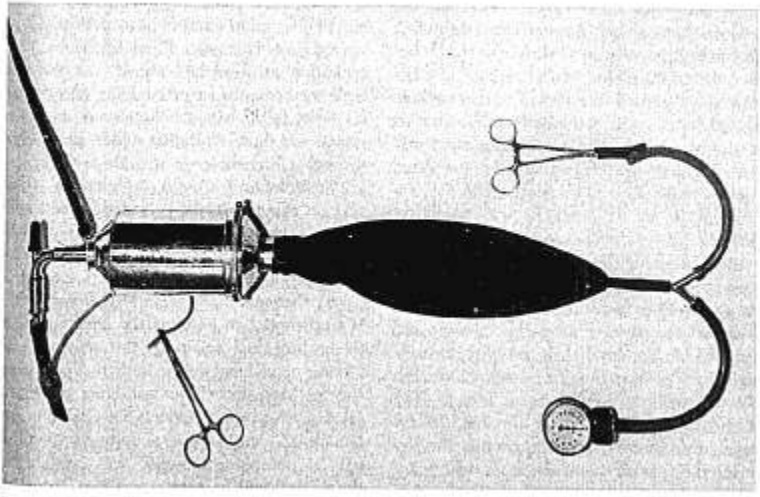

FIG. 3. Anesthesia equipment assembly for manual assistance of respiration and continuous positive pressure breathing.

manually operated apparatus was used to assist the patient's inspiratory efforts for nearly four days.

It was impossible to completely obtund the respiratory drive of these patients at any time so that respirations were "assisted" rather than "controlled." Pressures as high as 50 mm. of mercury were required occasionally to inflate the lungs. Despite these high airway pressures, arterial hypotension was not observed by clinical estimation of blood pressure. The positive pressure during the expiratory phase was discontinued after approximately 18 hours.

After four days, positive inspiratory pressure was provided for 15 minute periods every hour for several days thereafter, to rest the muscles of respiration. It is important to appreciate that work of the respiratory muscles was increased continuously in these patients; assisted breathing proved valuable in relieving fatigue of inspiratory muscles.

(3) *Humidified Oxygen or Compressed Air.* One hundred per cent oxygen was administered during the first 24 hours by the partially closed to-and-fro absorption technique. Early in the second day because of fear of oxygen toxicity (pulmonary congestion and irritation), oxygen was replaced by compressed air administered by the same technique, but it had to be abandoned because the patients' hemoglobin became less saturated. Helium-oxygen mixtures were also tried but were discontinued for similar reason. By the third day, normal saturation of hemoglobin was achieved in all patients by administration of various mixtures of compressed air and oxygen (oxygen mixtures gradually decreased from 60 to 30 per cent). By the fifth day, enrichment of air with oxygen was no longer necessary.

"Bubble-through" water bottles and nebulizers were used to humidify the administered gases. Neither proved adequate. Because much of the tissue lining the airways had sloughed off, we decided, in addition, to instill sterile physiological saline solution directly into the trachea (3–5 ml. every hour). This practice was maintained for three weeks following the injury. Better apparatus for producing aerosols is available at present ‡ and should be used in such cases to provide a moisture-rich atmosphere (see chapter on aerosols, this symposium).

(4) *Bronchodilator Drugs.* Nebulized isoproterenol (0.5 ml. of 1 : 200 solution 2.5 mg.) was administered into the tracheostomy at 30 minute intervals in the early part of phase 2, in an attempt to reduce the number of high resistance pathways.

Intravenous infusions of dilute solutions of theophyline ethylenediamine (0.5 Gm. in 500 ml. 10 per cent dextrose in distilled water) were administered during the first 24–30 hours following exposure. Total doses of as much as 2.5 Gm. were used in 24 hours. Although bronchiolar smooth muscle contriction may have occurred shortly after the accident, later much of the airway obstruction was due to plugs of tissue debris.

(5) *Tracheobronchial Aspiration.* It was necessary to aspirate the trachea and larger bronchi frequently (every 30 minutes at least) with large bore catheters (16–20 F). At first, whistle-tip catheters were used, but catheters with the tips removed were more satisfactory. During phase 2, the trachea was less sensitive to direct stimulation than normally, but the region of the carina maintained some sensitivity and cough could be produced. The risk of mechanical damage to insensitive areas must be borne in mind in such patients.

(6) *Bronchoscopy.* Blind tracheobronchial aspiration as described above was the principle method used in maintaining an open airway, but failed to open the airway in two instances; in these aspiration under direct vision through a bronchoscope was life-saving.

(7) *Voluntary Cough.* Voluntary cough was not effective in the presence of the tracheostomy opening. Occluding the opening momentarily at beginning expiration assisted in the production of a more effective cough.

(8) *Enzymatic Digestion of Cellular Debris.* We attempted to improve the effectiveness of tracheobronchial toilet by the enzymatic digestion of cellular debris with the proteolytic enzyme, trypsin. Two milliliters of trypsin solution (Tryptar, Armour & Co., solution containing 250 mg. tryptic activity in 25 ml. of *p*H 7.4 phosphate buffer §) was instilled into the trachea and bronchi every 30 minutes, and

‡ Croup-Aire. Air-Shields, Hatboro, Pennsylvania.

§ No longer commercially available in this form.

in five minutes the trachea was aspirated. This was carried out from 24 to 48 hours following exposure.

Little was known of the untoward effects of trypsin on normal or denuded tissues in 1954, though its use to liquefy cell debris was established. The use of trypsin solutions in the airway has since been evaluated more thoroughly, and its employment in this respect is condemned.[1] In light of this knowledge, we recommend withholding instillations in the tracheobronchial tree in such cases except physiological saline solution or nebulized isoproterenol or similar bronchodilators.

(9) *Vigorous Pounding on Chest.* Vigorous pounding on the thoracic cage was useful in loosening bronchial plugs not removed by catheter aspiration or assisted voluntary cough. These patients were breathing small volumes of gas at low expiratory velocities; these were not sufficient to dislodge plugs in the smaller air passages. The additional expulsive force supplied by sharp blows to the thorax appeared to be helpful.

(10) *Frequent Change in Position.* The patients observed that they could breathe more easily by lying on their sides, and they invariably turned so that the more severely damaged lung was dependent. We were misled by the apparent relief afforded by this position and it was not until one patient had developed widespread atelectasis in the "down" lung that we kept them from lying in their most comfortable position for long.

When the patients finally slept (with the aid of sedatives) we found it hazardous to allow them to remain in one position for any longer than one hour.

(11) *Compression of the Thorax.* Late in Phase 2, when obstructive emphysema had occurred and the thorax was fixed near the maximal inspiratory position, the patients had difficulty in deflating the lungs. By squeezing the thorax, we were able to reduce the functional residual capacity temporarily and the patients were subjectively improved.

(12) *Antibiotics.* All six patients received prophylactic doses of antibiotics (procaine penicillin 300,000 units daily and streptomycin 0.5 Gm., q.i.d.). The dose of penicillin was increased on the second day to one million units and by the end of five days it was discontinued. Antibiotic therapy was continued with intravenous tetracycline 0.5 Gm. daily.

(13) *Hydrocortisone and Cortisone.* Early in the management of these patients, we administered hydrocortisone 100 mg. intravenously in 500 ml. 5 per cent dextrose every 24 hours in an attempt to overcome bronchoconstriction and to decrease the fibrous tissue response to inflammation.

Discussion

An accident precisely like this one will occur infrequently. However, the physician and the anesthesiologist must have a 'prepared mind' to recognize and treat, promptly and effectively, injuries of the respiratory tract that may result from many different chemical agents.[2-8] For example, new manufacturing processes add effluents containing potentially hazardous chemicals to the atmosphere despite local and national regulations enacted to control such practices. In addition, persons may be exposed to respiratory irritants on their farms, in their homes or places of employment. Conflagrations, producing smoke, products of incomplete combustion of various flammable substances and superheated gases may be an additional source of pulmonary irritants. Large scale transport of such potentially hazardous chemicals as compressed gases, liquids or solids is becoming increasingly common. These chemicals, or their reaction products, if accidentally liberated into the atmosphere, may produce large quantities of irritating gases or vapors. The possibilities of a large number of casualties resulting from such accidents is readily apparent.

The Problem of Mass Casualties

This catastrophe resulted in the admission of 39 patients to the Philadelphia General Hospital. The care of the six men with severe respiratory problems required a manpower force of 12 persons continuously around the clock for the first four days. About 75 physicians and nurses were required for the full-time care of these patients on a 24-hour basis (including rotation and relief). This does not include the man hours spent by various consultants, laboratory technicians and nonprofessional persons whose services were required. We were fortunate to have 125 interns and

approximately 75 residents in various departments within the hospital. Nearly all served in some capacity in the care of these patients in addition to their primary obligations to other patients. In addition, volunteers from other hospitals served on the emergency rotation schedule set up to care for these men.

It is apparent that should an even greater catastrophe occur, resulting in hundreds of pulmonary casualties, one hospital would be unable to treat all of these even if it had satisfactory mechanical equipment to provide adequate pulmonary ventilation. It becomes important to decide then which patients may be saved by intensive, continuous care. This is a difficult problem, particularly when the chemical damage is delayed; considerable help is afforded by using modern, portable bedside methods for rapid evaluation of pulmonary function.

From our experience with these men we would like to make some recommendations concerning the overall management of casualties exposed to pulmonary irritants.

(1) Triage should begin in the reception area, the receiving ward or emergency room. The patients most severely injured should be sent to one designated area. The other patients should be sent to a second nearby area so that transfer to the first area is not physically difficult. The patients in the second area should be watched continuously for delayed obstruction of the respiratory tract, and tracheostomy trays should be on hand.

(2) A physician familiar with the major problems evidenced by the patients should be appointed to administer a preconceived but flexible disaster plan. He should have temporary authority of wide scope with administrative and service personnel at his disposal. The effective administration of any plan of medical management concerned with gravely injured patients requires that the director be present or readily available in the maximal care area, at least for the first 24-36 hours. When the severity of the patients' disease lessens, the director may make fuller use of his lieutenants.

(3) Pulmonary physiologists should be recruited, if possible, to monitor the function of the lungs and to determine the nature and severity of the dysfunction in each case.

(4) Hospitals in the geographic area should have on hand, for emergency use, tanks of 50 per cent oxygen–50 per cent nitrogen (or the means for mixing oxygen with air) and apparatus for administering the gas mixture to patients.

The Problem of Providing Assisted Ventilation

The design and production of equipment for the long-term ventilation of patients with a variety of cardiopulmonary or respiratory diseases has been based on physiological principles only within the last decade or two. Better mechanical equipment is available in 1962 than in 1954, so that now manual methods may not be needed throughout the critical phase.

Suitable equipment for application to cases such as these must meet certain specifications: (1) it must operate dependably over extended periods; (2) it must not be pressure limited to such an extent that it cannot produce the high pressures often required to overcome marked elevations in airway resistance and so fail to produce alveolar ventilation; (3) it must produce adequate flow rates during the inspiratory phase; (4) it should permit independent variation of pressure, volume and rate; either the mechanical ventilator must be synchronized to the patient's respiratory efforts or some means employed to deprive the patient of his own respiratory drive.

In a recent monograph,[9] 56 ventilators are described. All of them have advantages and disadvantages in specific applications. Their performance may be adequate when applied to the paralyzed, anesthetized or unconscious patient having a patent, normal airway, but inadequate for patients having high airway resistance, relatively stiff pulmonary structures and reduced surface area in the lung for gas exchange. Volume-cycled ventilators generally meet the requirements best, but a number of these have serious inadequacies. Recommendation of one specific mechanical respirator is undesirable because even patients injured by the same chemical agent may have different lesions at different times. One should be familiar with the design and operation of the equipment in his own and neighboring hos-

pitals and its intended application. Should it become desirable to use a mechanical ventilator in the management of patients with this type of acute pulmonary difficulty, one should patient-test the apparatus while carefully observing the patient's response. If the equipment does not function, there is no reason to abandon the patient, for the simple, manual means of caring for him as utilized on these six patients should be widely available.

The Problem of Rapid Physiological Evaluation of Pulmonary Function

We believe that the information obtained from the objective measurements of pulmonary function performed on these patients was a valuable supplement to clinical impressions of the pathologic physiology and of the effectiveness of therapy.

A simple volume recorder, such as a portable BMR apparatus, can provide accurate measurements of tidal volume and of vital capacity. A recording spirometer, bellows or ventilation meter, can provide data on maximal expiratory and inspiratory flow rates to determine the degree of airway obstruction throughout the respiratory cycle. A direct recording ear oximeter may be used to determine the degree of saturation of arterialized ear blood and may also be used to determine whether air, 30 per cent oxygen or 50 per cent oxygen is needed to provide maximal oxygenation of the arterial blood. An infrared carbon dioxide analyzer is useful to measure the end-expiratory P_{CO_2} and determine whether serious hypoventilation is present (one must be certain that the tidal volume is adequate to flush the respiratory dead space so that the recorded end-expired P_{CO_2} is, in fact, alveolar P_{CO_2}). The compliance of the lungs can be measured by the use of an esophageal balloon (to record changes in intrathoracic pressure) and a spirometer (to record changes in volume); a U-tube water manometer is quite satisfactory for the measurement of pressure change, and expensive electronic equipment is not necessary.

None of these procedures, with the possible exception of the passage of an esophageal balloon, is uncomfortable.

Summary

This is a report of the clinical course and medical management of six men who developed severe pulmonary dysfunction following exposure to a volatile commercial solvent during an explosion in 1954. It emphasizes the utility of pathophysiological findings as guides to effective therapy, the necessity for continuous, individual therapy and the probability of return of normal pulmonary function if gas exchange can be maintained until repair begins.

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