TRAUMA IN RELATION TO CONDITIONS OF LUNG AND THORAX

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INTRODUCTION

The lungs are particularly susceptible to injury because of their large size, their delicacy of structure and their constant contact with the air. Because of their complicated physiology and their importance in body economy, extensive damage to the lungs or interference with their function may result in serious, if not fatal, consequences. Disease or injury outside the lungs, i.e., in the brain[1], spinal cord[2], kidney[3], heart[4] and blood,[5] may result in marked derangements in pulmonary function with consequent severe disability or death; the present discussion will, however, omit consideration of these causes of respiratory dysfunction and will concern itself only with those forms of injury which cause organic lesions in the lungs, respiratory passages[6] and thoracic cage[7] which are known to occur or may occur in industry or through accident. Accordingly, discussion will be limited to diseases of the lung consequent to harmful stimuli which act directly on the lungs and in conformance with the following analysis:

I. *Irritant Dusts*
   A. Those causing allergic[8] reactions in the lungs
   B. Those irritating the lungs directly

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1. For instance, cessation of respiration following injury to the brain.
2. For instance, paralysis of muscles of respiration following injury to the spinal cord.
3. For instance, severe shortness of breath in acidosis due to renal (kidney) disease.
4. For instance, severe congestion of the lungs in certain types of heart disease. See White, Paul Dudley and Smith, Hubert Winston: Scientific Proof in Respect to Injuries of the Heart (Medicolegal Aspects of the Heart), (1946) 24 N.C.L. Rev. 106, a study in this Symposium series.
5. For instance, shortness of breath in severe anemia.
6. For instance, the trachea (wind-pipe) and bronchi (main branches of the trachea).
7. For instance, the ribs, sternum (breast bone) and that portion of the spine in the region of the chest.
8. Allergic reactions: The hypersensitiveness of an individual to an antigen (i.e., a foreign protein.)

II. *Irritant Gases*
III. *Aspirated Liquids*[9] (excluding drowning)
  A. Irritating liquids
  B. Oils
IV. *Foreign Body*
  A Aspirated
  B. Forced in or blown in through the chest wall, diaphragm or neck
  C. Entering through the oesophagus or stomach
V. *Penetrating Injury*
  A. Fracture of ribs
  B. Destruction of lung tissue
  C. Hemorrhage
  D. Hemothorax
  E. Pneumothorax
  F. Interstitial and mediastinal emphysema
IV. *Non-Penetrating Injury*
  A. Crushing
  B. Blast
  C. A blow or fall
VII. *Muscular Strain*
  A. Pneumothorax
  B. Mediastinal emphysema
  C. Hemoptysis
  D. Activation of antecedent disease
VIII. *Trauma Elsewhere*
  A. Embolization (excluding septic embolization following abortion)
    1. Due to phlebitis
      a. post-traumatic
      b. induced by treatment
    2. Air embolus
    3. Fat embolus
    4. Amniotic fluid embolus
  B. Post-traumatic pneumonia
IX. *Pneumonia Following Exposure*
X. *Thermal Change*
  A. Burning
  B. Freezing

Several of these groups, i.e., the injurious dusts, drown-

9. Aspirated liquids: Liquids drawn into the trachea (wind pipe) and lungs in course of breathing.

ing and septic embolization following abortion, have been the subject of so much study for so long a time, that a huge literature, ranging from brief reports to voluminous monographs, on the occurence, pathogenesis,[10] manifestations, treatment, prevention and medico-legal importance of injury consequent to the action of these factors is available. Indeed, these topics are already considered in an extensive and complicated body of law. Accordingly, and also because of the fact that some of these aspects of injury to the lungs are covered in the companion articles by Dr. Norbert Enzer and Dr. Leslie Silverman, no discussion will be undertaken here of these factors. Similarly, consideration of burning and freezing will not be included.

Although a large number of different conditions caused by many different factors will be touched on, this discussion should not be considered encyclopedic; the intention is rather to illustrate the wide variety of pulmonary lesions caused by trauma of one sort or another under different circumstances. Reference will be made to current medical literature only in those instances in which the subjects discussed may not be covered adequately in standard medical texts.

ANATOMY AND PHYSIOLOGY OF THE LUNGS

The lungs, two in number, lie to either side of the heart and occupy most of the chest. The right lung consists of three lobes and is larger than the left, which contains only two.

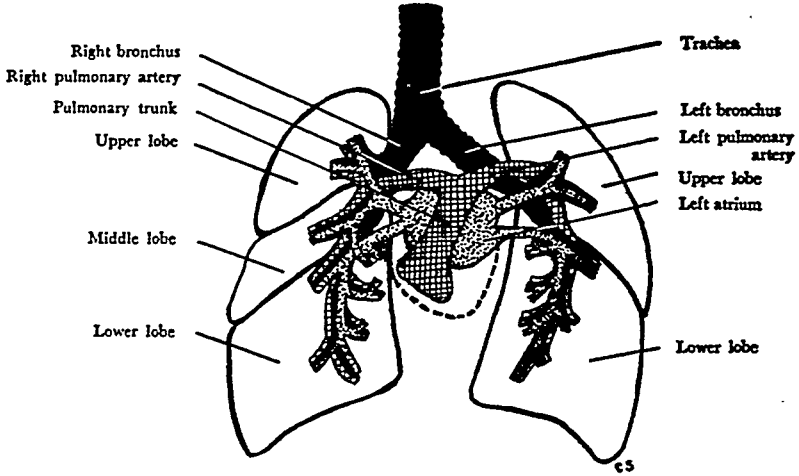

Fig. 1 Diagram of lungs with the branch; and blood vessels.

10. Pathogenesis: The development of morbid conditions or of disease.

The lobes are further subdivided into lobules. The lungs lie in a closed space, the thorax, surrounded outwardly by the ribs, breastbone and chest muscles, and bounded below by a large circular muscle called the diaphragm. Each lung is surrounded by a membrane-lined potential space, normally empty, called the pleural cavity after its lining, the pleura. Air entering the nose and mouth is conveyed internally by the trachea, which divides to form the two main bronchi, one of which goes to each lung, where further subdivisions corresponding to the lobes and then the lobules occur. Thence the air is conveyed to all parts of the lungs by dichotomously dividing bronchioles, finally entering the alveoli. The trachea and bronchi are tubes consisting of fibrous and muscular walls, strengthened by plates of cartilage and lined by an epithelial membrane[11] which secretes mucus[12] and also by means of ciliary[13] action conveys the mucus and its contents of entrapped dust and bacteria toward the throat. In the bronchioles, which are smaller ramifications of the bronchi, the muscular tissue in the walls is more prominent and cartilage is lacking. The alveoli are tiny air sacs lined by a layer of flat cells in contact with which are the capillaries containing blood to be aerated. Essentially the alveolar wall is a membrane separating the air in the lungs from the blood in the capillaries; diffusion occurs across this membrane. All of the blood of the body returning to the heart via the veins is carried from the right ventricle toward the lungs by the pulmonary artery which subdivides so that its branches follow the bronchi and bronchioles into the alveoli. Here the pulmonary arterioles give off the alveolar capillaries. Blood returning from the lungs enters small pulmonary venules, each of which lies parallel with a bronchiole and an arteriole. The venules unite to form veins which become larger as they approach the heart, which they finally enter via the left auricle. Thence the blood, now aerated by passage through the lungs, flows into the left ventricle, whence it is distributed throughout the body. About four liters (4.224 quarts) of blood pass through the lungs

11. Epithelial membrane: A thin layer of tissue which covers a surface or divides an organ.
12. Mucus: The viscid watery secretion of mucous glands.
13. Ciliary: Pertaining to the activity of minute lash-like processes on the surface of the mucous membranes.

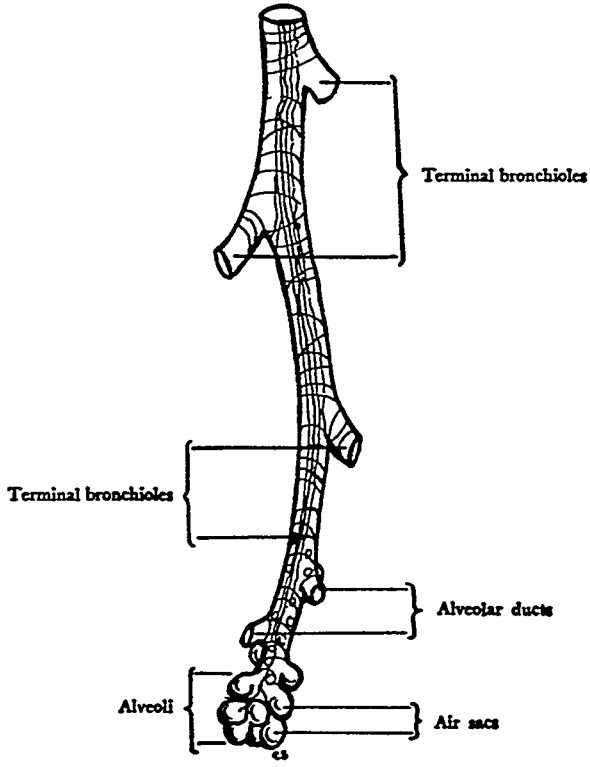

Fig. 2. Arthetecture Structional units of the lung tissue.

every minute while the organism is at rest. Exercise increases the flow greatly.

Functions of the Lungs. The lungs have a number of functions. Blood from all parts of the body, made deficient in oxygen and laden with carbon dioxide as a consequence of passage through the tissues, comes into contact with the air in the lungs, discharging its excess of carbon dioxide and taking up oxygen. Since carbon dioxide dissolved in the blood acts as an acid,[14] it is apparent that changes in respiration which cause variations in carbon dioxide content of the blood are important in regulating the acidity of the blood. A further function of the lungs is in relation to maintaining the body temperature at a normal level. Heat,

14. $CO_2 + H_2O = H_2CO_3$, which is an acid in that it has hydrogen atoms which are replaceable by positive atoms.

constantly produced in the body by the metabolic processes, must be dissipated. Most of it is lost through the skin, some through the urine (water, largely drunk cold and execreted warm) and about a quarter of the total via the lungs. This is accompanied by warming the air inhaled and, more particularly, by the vaporization of water in the lungs. Air is inhaled containing a small amount of water vaporized in it and exhaled saturated with water derived from the blood. The vaporization of this water, about a quart a day, requires just as much heat as would be required for the evaporation of a quart of water in 24 hours from a pan.

The Respiratory Movements. All of these functions are accomplished by the respiratory movements. The diaphragm (the flat circular muscle which separates the thorax from the abdominal cavity) arched when relaxed, contracts, becoming shortened and consequently flattened, thereby creating a negative pressure within the thorax and sucking air into the lungs via the trachea and bronchi. Contraction of some of the chest muscles elevates the ribs aiding in the creation of the negative pressure. About 400 cc. of air are sucked in at each inspiration at rest; several times that volume is inhaled at each inspiration during exertion. Relaxation of the diaphragm and the muscles of the chest initiates expiration. Expiration is accomplished by the intrinsic elasticity of the lungs, the muscles of respiration normally remaining relaxed during this phase of respiration. Consequently these muscles must be drawn back into position for the next inspiration by the negative pressure created by the return of the lungs to a collapsed state, effected by the elasticity of these organs. The rate and depth of respiration are regulated by a respiratory center in the brain whose activity varies in response to nerve-borne impulses which bombard it, by changes in oxygen and carbon dioxide content and acidity of the blood which bathes it, by the need for aiding in the dispersal of the body heat and by the state of distension of the lungs. The respiratory center emits nervous impulses which are carried down the spinal cord to the neck where the *phrenic nerve*, one on each side, leaves the spinal cord and passes down through the chest to the lungs. It is apparent that respiration is an extremely important and complex process. Increased respiratory activity can be expected to occur when there is increased need for the

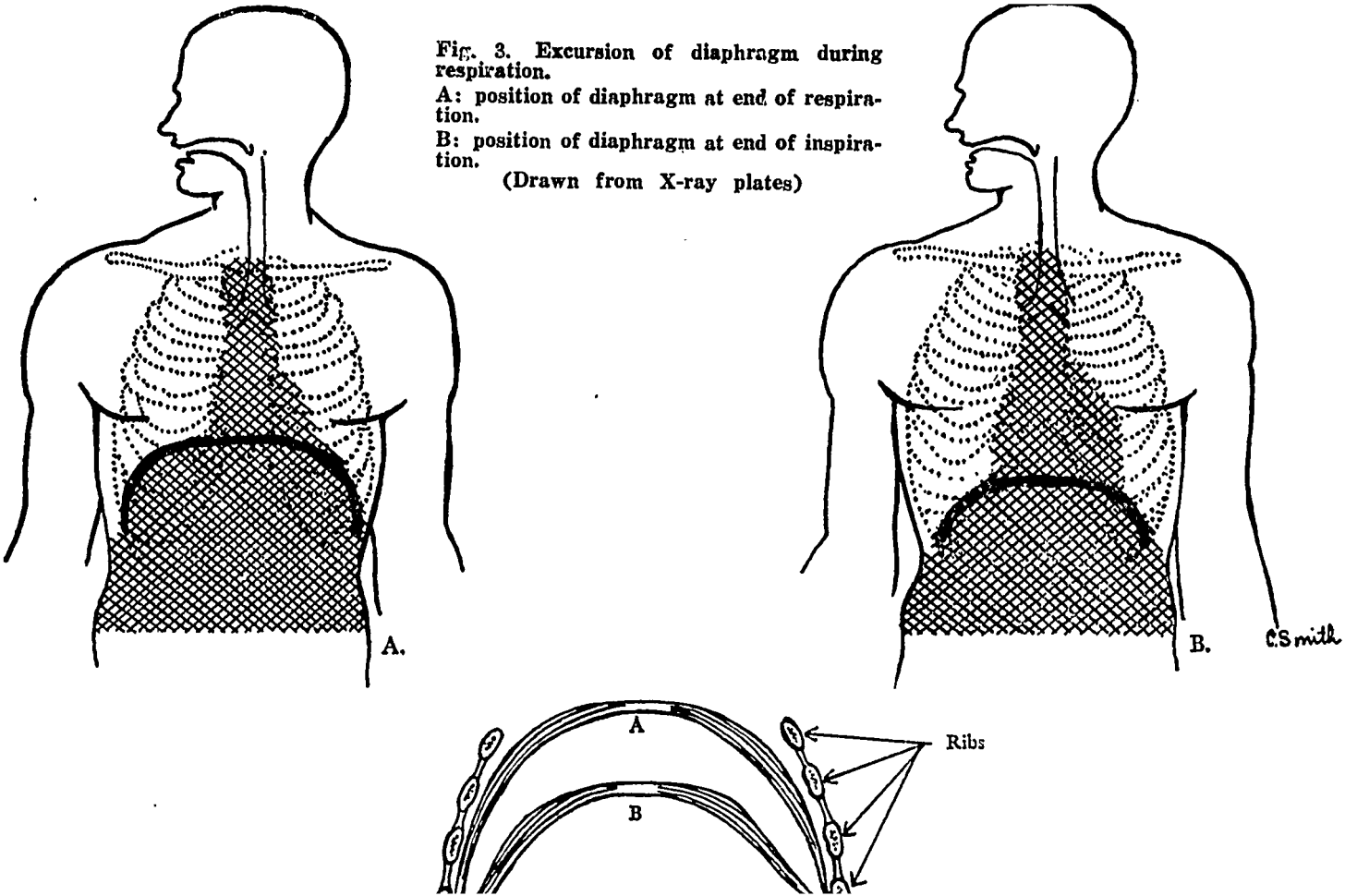

Fig. 3. Excursion of diaphragm during respiration.

A: position of diaphragm at end of respiration.

B: position of diaphragm at end of inspiration.

(Drawn from X-ray plates)

uptake of oxygen or the excretion of carbon dioxide (i.e., in fever, thyroid disease, etc., and during exercise), when the uptake of oxygen and relcase of carbon dioxide are impaired by pulmonary (lung) disease, when there is increased need for heat dispersal (i.e., in fever and thyroid disease and when the elimination of heat through the skin is diminished) and when the blood becomes abnormally acid (i.e., as a consequence of certain phases of diabetes, kidney disease or gastrointestinal disease).

IRRITATING GASES

Sources of Noxious Gases. Distinction must be made between gases which injure the lungs and those which, like carbon monoxide, enter through yet do not injure the lungs but act on some other organ; only the former will be discussed here. Although there is widespread recognition of the fact that pulmonary injury due to gases was a common cause of severe and/or prolonged disability, or even death, during the War of 1914-1918, it is not generally appreciated that equally severe damage to the lungs may result from gases used in industry (such as chlorine) or liberated normally during industrial processes (such as nitrogen dioxide or tetroxide formed during etching, metal pickling, photoengraving or oxy-acetylene welding). Some substances, normally liquid, may give off irritating vapors which may act, at least in part, on the lungs; these include some refrigerants and also liquid fuels such as gasoline and kerosene. In addition, irritating gases may be generated during certain types of accidents. For instance, the contact of brine or sea water with exposed electrical installations may under some circumstances result in the liberation of chlorine[15] gas which may reach a sufficiently great concentration in a closed space, such as a compartment in a partially sunken ship, to be injurious. Fire is especially likely to result in the formation of irritating gases, either in the form of flame itself, or as some product of combustion. An example of the latter is the nitrogen dioxide or tetroxide[16] formed when x-ray film burned during the Cleveland Clinic fire of 1929. Complex organic materials, when burned in ordinary air or in atmos-

15. Chlorine: A greenish-yellow gas with a characteristic sharp odor.
16. Tetroxide: NO_2 or N_2O_4, a poisonous volatile liquid, giving off brownish irritant fumes.

pheres partly depleted of oxygen, may give off irritating gases which are not yet identified. The increasing use of plastics in building in the future may result in an increased incidence of serious pulmonary damage during accidental fires. Another way in which injury to the lungs may occur as a consequence of fire is by the formation of irritating gases during attempts to extinguish a fire. Thus, carbon tetrachloride, used in some types of hand fire-extinguishers, may be converted into phosgene[17] gas by contact with open electrical installations or possibly with a very hot fire; the formation of this gas in a poorly ventilated space may result seriously for the occupants of that space, although the fire itself may be trifling.

The irritating gases, irrespective of mode of formation, may cause three different types of injury: (1) tracheobronchitis[18], (2) pulmonary edema[19], (3) central respiratory[20], vasomotor[21] and/or cardiomotor paralysis.[22]

Some gases cause solely or predominantly one type of injury, while others may cause more than one type to approximately equal degrees. The third of the above three types of injury, paralysis of the nervous centers in the brain which regulate breathing, blood pressure and cardiac action, is immediately or rapidly fatal, only occasionally responding to treatment. Strictly speaking, it is not a form of pulmonary injury. It is included here only to point out that gases, such as phosgene or the oxides of nitrogen, which damage the lungs may also be absorbed through them and act internally; in some instances the effects of the pulmonary injury may be successfully combatted, only to have the victim develop signs of collapse and die of injury to some vital center in the brain. No further discussion of this action of certain gases will be made here. Similarly, the injury to the skin, eyes and mucous membranes of the nose and throat, caused

17. Phosgene gas: A poisonous gas, $COCL_2$, with an odor of musty hay, green corn or the inside of a silo.
18. Tracheobronchitis: Inflammation of the trachea and bronchi.
19. Pulmonary edema: Accumulation of fluid within the lung tissue and also in the air within the lungs.
20. Central respiratory: Centers in the brain which regulate resporation may be damaged.
21. Vasomotor: Centers in the brain which regulate the body's blood pressure may be damaged.
22. Cardimotor paralysis: Damage to centers in the brain which regulate heart action.

by some of the gases in this group simultaneously with their action on the lungs, will not be considered here.[23]

TRACHEOBRONCHITIS

Mechanism, Pathology and Clinical Picture. Exposure to an irritating gas which acts on the main respiratory passages results in the death of, or serious injury to, the cells lining these passages. The damage may extend beyond the superficial cells into the deeper tissues. The thin, smooth, somewhat moist mucous membrane, provided with actively beating cells, is replaced by a thick, swollen layer of necrotic

23. Editor: In the following cases, poisonous gases, fumes or vapors were inhaled and caused direct injury to the lungs or were transported in the blood, causing injurious effects elsewhere. The principal questions litigated in these cases are the actuality of injury, causal connection between the alleged injury and the alleged exposure to a noxious gas or vapor, whether the injury was an accidental one compensable under Workmen's Compensation Act of the particular state or involved an occupational disease compensable only under special provisions of any existing occupational disease act. Some of the cases also involve common law liability of employers and a discussion of the legal effects of violating safety appliance acts. The cases are cited principally to give the interested practicing lawyer ready reference to a variety of decisions involving problems of proof and substantive law aspects of situations much as Dr. Altschule has dealt with on the medical side. In the cases cited, the gases fumes or vapors allegedly inhaled with resultant injury to the claimant were as follows: Gustafson v. Parlier Winery, 2 Cal. Ind. Acc. Comm. Dec. 101 (1915) (gases from tank or vat of winery): Industrial Commission of Colorado v. Ule. 97. Colo. 253, 38 P(2d) 803 (1935) (dope solution applied to bodies and wings of airplanes by spraygun); National Casualty Co. v. Hoage, 64 App. D.C. 33, 73 F.(2d) 850 (1934) sulphur dioxide escaping from Frigidaire system in apartment house); Sullivan Mining Co. v. Aschenbach, 33 F. (2d) 1 (1929) cert. denied 280 U.S. 586, 74 L.Ed. 635, 50 Sup. Ct. 35 (1929) (carbon bisulphide in thinner used by painter); Doherty v. Western United Gas and Electric Co., 188 Ill. App. 494 (1914) (gas fumes escaping in gas works); McBeth-Evans Glass Co. v. Brunson, 70 Ind. App. 513, 122 N.E. 439 (1919) (glass blower exposed to fumes); Brewer v. Veedersburg Paver Co., 92 Ind. App. 547, 177 N.E. 74 (1931) (gas, smoke and dust inhaled in brick kiln over a period of twelve to fourteen years); Dille v. Plainview Coal Co., 217 Iowa 827, 250 N.W. 607 (1933) (carbon monoxide and carbon dioxide in improperly ventilated mine); Jellico Coal Mining Co. v. Walls, 160 Ky. 730, 170 S.W. 19 (1914) (miner allegedly poisoned by carbonic acid gas or by carbon monoxide); T. M. Crutcher Dental Depot, Inc. v. Miller, 251 Ky. 201, 64 S.W. (2d) 466 (1933) (cromic acid gas allegedly inhaled by operator of chromium plating machine); Faulkner v. Milner-Fuller, Inc. (La. App.) 154 So. 507 (1934) (fumes from paints and lacquers); Thompson v. United Laboratories Co., 221 Mass. 276, 108 N.E. 1042 (1915) (volatile arsenic given off by cans of rat poison was allegedly inhaled by 17-year old girl employed to fill cans); Johnson's Case, 279 Mass. 481, 181 N.E. 761 (1932) (chronic hronchitis and emphysema allegedly aggravated by inhalation of coal gas and dense tar smoke); Baltimore & O. R. Co. v. Bran-

tissues[24] exuding large amounts of plasma[25] and later pus.[26] The diameter of the airway is consequently narrowed as a whole and may be plugged locally by masses of debris. The muscle cells which make up much of the walls of the respiratory passages may be thrown into spasm, further narrowing the airway. Accordingly, the patient experiences varying degrees of pain, and more particularly, shortness of breath

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son, 128 Md. 678, 98A 225 (1916), reversed 242 U.S. 623, 37 S. Ct. 244, 61 L. Ed. 534 (1916) (inhalation of paint spray from "paint gun"); Tomlanovich v. American Boston Mining Co., 272 Mich. 493, 262 N.W. 293 (1935) (tuberculosis allegedly activated by breathing gas, smoke and dust produced by blasting in mine); Adler v. Interstate Power Co., 180 Minn. 192, 230 N.W. 486 (1930) (coal and coke fumes in power plant); Jackson v. Euclid-Pine Inv. Co., 223 Mo. App. 805, 22 S.W. (2d) 849 (1930) (carbon monoxide poisoning of garage mechanic); Bender v. Midwest Pipe and Supply Co. (Mo. App.) 57 S.W. (2d) 707 (1933) (sulphur dioxide from burning coke in welding department); Decker v. Raymond Concrete Pile Co., 336 Mo. 1116, 82 S.W. (2d) 267 (1935) (Acetylene gas inhaled by user of acetylene torch); Thomson v. Amoskeag Mfg. Co., 86 N.H. 436, 170 Atl. 769 (1934) (Dormant tuberculosis allegedly aggravated by poisonous gases); Bove v. Donner-Hanna Coke Corp., 236 N.Y. App. Div. 37, 258 N.Y. Supp. 229 (1929) (Coke oven gases); O'Connor v. Consolidated Gas Co. of New York, 243 N.Y. App. Div. 661, 276 N.Y. Supp. 998 (1935) (Gas inhaled by foreman of gang working on gas pipes as result of gas main bursting); Dixon v. Gaso Pump and Burner Mfg. Co. 167 Okla. 401, 29 P. (2d) 764 (1934) (Carbon monoxide poisoning); Coca-Cola Bottling Co. v. Mowry, 167 Okla. 644, 31 P.(2d) 562 (1934) (Inhalation of caustic soda fumes, long continued); Johnston v. E. E. Orcutt Garage, 103 Pa. Super. 507, 157 Atl. 46 (1931) (Carbon monoxide gas); Sinkiewicz v. Susquehanna Collieries Co., 115 Pa. Super. 377, 175 Atl. 757 (1934) (Gases in smoke-filled room of mine where dynamite had been fired); Katora v. N.J. Zinc Co., 116 Pa. Super. 257, 176 Atl. 762 (1935) (Carbon Monoxide poison in gas plant); Alston v. Virginia-Carolina Chemical Co., 104 S.C. 410, 89 S.E. 497 (1916) (Fumes and gases in acid tower of fertilizer factory); Consolidated Kansas City Smelting and Refining Co. v. Dill (Tex. Civ. App.), 188 S.W. 439 (1916) (Fumes in smelter inhaled by carpenter); Commercial Standard Ins. Co. v. Noack (Tex. Civ. App.), 45 S.W. (2d) 798 (1931), Reversed, Com. App. 62 S.W. (2d) 72 (Carbon monoxide poisoning); Associated Indemnity Corp. v. Baker (Tex. Civ. App.) 76 S.W. (2d) 153 (1934) (Sulphuric acid fumes allegedly inhaled while cleaning and blowing out tubes of a steam condenser in an electric light and power plant); Depre v. Pacific Coast Forge Co. 151 Wash. 430, 276 Pac. 89 (1929) (Noxious gases from mixture of muriatic acid, sulphuric acid and water used to remove scale from metal preparatory to galvanizing it); Pellerin v. Washington Veneer Co., 163 Wash. 555, 2 P.(2d) 658 (1931) (Carbon bisulphide poisoning slowly contracted from exposure to gases and vapors generated in mixing glue in veneer mill).

24. Necrotic tissue: A tissue is an aggregation of fibers and cells composing a structural element; necrotic tissue is dead tissue.
25. Plasma: The liquid component of blood.
26. Pus: A liquid product of inflammation made up of white blood cells, plasma and debris.

(dyspnea) and cough. The dyspnea may be severe enough to require oxygen. In some instances attempts have been made to suck out the obstructing debris through a bronchoscope.[27] The cough may be dry, or may be productive of a mixture of mucus, serum and debris. If spasm of the bronchi is severe, asthmatic wheezing may be noted. Physical examination should show the signs of bronchitis, but the x-ray may not reveal the abnormality.[28]

The denuded bronchi, filled with debris, are extremely susceptible to infection by bacteria in the air or in the breath of neighboring individuals. A superimposed bacterial bronchitis or bronchopneumonia may develop, making the damage more extensive and the patient much more sick.

Within a period of hours or days, depending on the toxicity of the gas and the amount of exposure to it and the presence or absence of bacterial invasion, healing begins. The necrotic debris is sloughed off, to be replaced partly or wholly by normal tissue. Where damage to deeper layers of the trachea and bronchi has occurred, with consequent greater degrees of disorganization of structure, complete healing may be retarded or may never occur. A state of chronic inflammation may persist. Muscle cells, in particular, regenerate poorly and may be replaced by inelastic scar tissue. In some areas, due to the pressure changes within the bronchi during respiration, a thin patch of scar tissue may balloon out, forming a sac; this state is called bronchitectasis. Its occurrence is favored by the development of strictures, consequent to contraction and puckering of scar tissue, higher in the bronchial tree. Examination may show a few signs of congestion in the bronchi or may be entirely negative. Similarly, ordinary x-ray studies may also fail to reveal the disease; recourse must be had to the instillation of lipiodol[29] into the bronchi to outline the dilated bronchi. These saccular areas retain secretion which becomes infected and the patient develops a chronic productive cough and a greatly increased susceptibility to respiratory infection; common colds, instead of passing off in three or four days, descend into the lungs,

27. Bronchoscope: A tubular instrument for inspecting the interior of the bronchi; it is inserted via the mouth and trachea.

28. Schatzki, R.: Management of the Cocoanut Grove Burns at the Massachusetts General Hospital. Roentgenologic Report of the Pulmonary Lesions, Ann. Surg. 117: 834, 1943.

29. Lipiodol: An oil which is opaque to x-rays.

causing severe coughs which may last for weeks. Additional parts of the bronchial tree may thereby be successively involved. In some instances small patches of inflamed lung tissue (*pneumonitis*) develop about bronchiectatic areas. They may result in chest pain and irregular fever for prolonged periods.

Complications. If the bronchitis and bronchiectasis are associated with considerable obstruction to respiration, either because of extensive damage to the bronchi or because of associated asthma, the patient will develop a state of overinflation of the lungs (*emphysema*). In a previous paragraph it was pointed out that inhalation is an active and exhalation a passive process; when obstruction is present, it is easier to inhale than to exhale, so that obstruction ultimately leads to overdistension of the lungs. When this occurs, the entire respiratory mechanism is upset: the patient has to work harder to breath and mixing is poor within the lungs, so that the blood is poorly oxygenated and does not give off as much carbon dioxide as normally. The patient may become short of breath, may become blue (cyanotic) and may develop a train of symptoms involving various organs which are now bathed in inadequately aerated blood. These symptoms include mental sluggishness, intolerance of heat, and a variety of poorly defined gastrointestinal complaints.

In some cases the accumulation of secretion in a bronchus, or the formation of a stricture across it, may cause a lobule or lobe of lung to become completely functionless and collapsed (*atelectasis*). This usually is readily detected on examination and always by x-ray.

Another complication of bronchiectasis is sudden hemorrhage (*hemoptysis*), which may occur in a previously apparently healthy individual. A large hemoptysis may be fatal.

In extreme instances, the heart muscle bathed in poorly aerated blood and forced to work harder pumping the blood through the abnormal lung may fail; *cor pulmonale* is said to have developed. Patients with this condition are usually invalids and have a short life expectancy.[30]

Some patients in whom none of the above sequelae to

30. See, in this Symposium series: White, Paul Dudley and Smith, Hubert Winston: Scientific Proof in Respect to Injuries of the Heart (Medicolegal Aspects of the Heart) (1946) 24 N.C.L. Rev. 106.

gassing occur may suffer in another way, i.e., by the *lighting up of a latent disease,* usually tuberculosis, or by its exacerbation if already overt.

PULMONARY EDEMA

Contact of a sufficiently great concentration of certain gases with the walls of the alveoli may so damage them that the capillaries they contain no longer retain blood and large amounts, i.e., pints, of plasma, mixed with variable amounts of red blood cells, pour out into the air spaces. This may occur soon after gassing or several hours later; in the latter case the patient may feel entirely well in the interim. The patient at once commences to drown in his own secretions. He becomes intensely dyspneic (breathless) and cyanotic (blue) and coughs up large amounts of colorless or pink frothy fluid. Unless treatment, including the administration of oxygen, is instituted at once, the patient is almost certain to die; he may die in spite of treatment, either because the treatment is ineffective in his case or because he goes into shock following the loss of so much blood and plasma into and through his lungs. He may remain in a state of pulmonary edema for hours or days; during this time his lungs are unusually susceptible to bacteria in the air or in the breath of nearby individuals and a fulminating extensive pneumonia may supervene.

After the subsidence of the pulmonary edema, the lungs may heal completely or else emphysema may develop. It was pointed out previously that exhalation is a passive process and is accomplished by the elasticity of the lungs. This elasticity may be lost following gassing either by the destruction of the universally present fine elastic fibrils in the pulmonary tissue or because of the overgrowth of a fine diffuse scar tissue in the alveolar walls. Whatever the mechanism, loss of elasticity results in a state of overinflation of the lungs (*pulmonary emphysema*). The consequences of pulmonary emphysema have already been discussed. If diffuse scarring (*fibrosis*) occurs, the lungs become abnormally rigid, making breathing more difficult. In addition, the scarring may interpose a thin wall of relatively impermeable material between the blood in the capillaries and the air in the alveolar spaces, so that the normal gaseous exchange across the alveolar walls cannot occur. In this case all the symptoms of

oxygen lack (anoxia) will occur. Physical examination of the lungs may be perfectly normal, and the x-ray may even in the presence of extensive fine scarring reveal no abnormality.[31] *Cor pulmonale*[32] may develop later.

PROBLEMS OF IDENTIFICATION FOLLOWING GASSING

Proof that claimant was gassed. In attempting to establish beyond doubt the occurrence of gassing in civil life, the ideal situation would be one in which the supposed victim was in contact for a known period of time with a gas of known composition present in a concentration known to be toxic. This is clearly an impossibility: gases are too evanescent to allow investigators to return to the scene hours or days later to measure their concentration at the time of the accident. Moreover, not all of the irritant gasses have been identified or can be identified.[33] Accordingly, attempts to establish the fact of gassing must be based upon indirect evidence. Some of this evidence may have a high order of validity: if a gas known to be toxic is used in industry or is formed during an industrial process, its accidental escape into or its accumulation within a space occupied by men may be detected by a characteristic smell or color. This would be true of chlorine certainly[34] and probably of many other gases. On the other hand, establishment of the fact of the presence in the air of the gas in concentrations sufficient to be *detectable* does not necessarily in the case of all gases establish the fact of gassing, i.e., the attainment *in the lungs* of a concentration sufficient to be toxic. Also, some gases, such as phosgene, may be toxic when present in concentrations too small to be detected with certainty; this gas is *fatal* in con-

31. Altschule, M.D.; Linenthal, H. and Zamcheck, N.: Lung Volume and Pulmonary Dynamics in Raynaud's Disease. Effect of Exposure to Cold, Proc. Soc. Exper. Biol. and Med., 48: 503, 1941.
32. Cor pulmonale: Dilation of the right side of the heart from pulmonary embolism (obstruction of a pulmonary artery by a blood clot brought to it in the blood stream) or obstructng pulmonary disease. The condition is attended by cyanosis (bluish coloration of the skin.) See White, P.D. and H.W. Smith, op. cit. supra, f.n. 30.
33. Aub. J.C.; Pittman, H., and Brues, A.M.; Management of the Cocoanut Grove Burns at the Massachusetts General Hospital. Pulmonary Complications: A Clinical Description, Ann. Surg. 117; 834, 1943.
34. See f.n. 11, supra.

centrations of 25 parts in a million. Or because of panic or loss of consciousness, the victim may not have noted the presence of the gas. It would appear, therefore, that medical testimony as to the occurrence of the symptoms of gassing is of the greatest importance in situations involving questions of exposure to irritant gases. The physician's records should include a note as to odors persisting in the victim's clothes or hair; this may, however, be misleading in the case of an individual gassed during a fire, since the clothes or hair may themselves be charred, or may smell only of smoke. Note should be made of the condition of the victim's skin, eyes, nose and mouth. Some gases are extremely irritating to these organs, particularly the eyes. Ordinary smoke may be very irritating to the eyes, but its effects wear off more rapidly than those of most of the irritant gases. The presence of charred tissue in the nose and throat establishes the inhalation of flame. The occurrence of the previously discussed symptoms of tracheobronchitis, pulmonary edema[35] or central vasomotor or respiratory paralysis should be noted. It is to be remembered that they need not appear immediately, but may become overt only after hours or even a day or two. Medical testimony as to the presence of symptoms of gassing in a person who was in a situation in which a toxic gas might have been generated would appear to be as close as one can come in civil life to establishing the fact of gassing. If the victim is dead, postmortem examination of the lungs would be very helpful.

Proof that Specific Lung Conditions Were Caused by Gassing. Attempts to prove conclusively that the manifestations of bronchitis, bronchiectasis, asthma, pulmonary emphysema, pulmonary fibrosis or cor pulmonale, which a patient may show, are consequent to his having been gassed previously may be very difficult. All of these disorders, when consequent to gassing, are indistinguishable from their manifestations when due to some other cause, such as allergy or the ordinary varieties of pneumonia, bronchitis and influenza. Statistical studies as to the incidence of these sequelae are not very helpful, since statistics are of little help in evaluating a single case. In addition, most of them are based on

35. Pulmonary edema: Congestion of the lungs resulting from an effusion of serous fluid from the blood into the air vesicles and interstitial tissue of the lungs.

data derived from the War of 1914-1918, and gassing in warfare is likely to be quite different from gassing in civil life. Not only may the gases involved be different, although this is not necessarily true, but the military are provided with sensitive detecting devices and effective protective mechanisms, so that while the numbers involved may be much greater, the average exposure would be milder. The latter was not of course true of the first gas attacks, such as that on a front of three and three-quarters miles at Ypres on April 22, 1915, when a force of Canadians and others were gassed, five thousand dying outright and ten thousand others becoming casualties.[36] Moreover, such studies as have been published have as their authors members of the military forces who consciously or unconsciously attempted to minimize the frequency of persistent pulmonary disorders following gassing in soldiers. Thus examination of the case reports of Gilchrist and Matz[37] makes it appear to the present author, as well as to more distinguished medical authorities, i.e., the editors of the British Medical Journal,[38] that Gilchrist and Matz[37] may have been too conservative in their analysis of the numerical frequency of the sequelae to gassing. Similarly, the studies of Berghof[39] on 2000 men (one-third of them gassed with chlorine) three or four months after gassing, led him to conclude that 50 per cent had no permanent pulmonary damage, although he states that his "normals" frequently exhibited cough or dyspnea on exertion; his conclusions were based solely on physical examination which, as pointed out above,[31] may be misleading. Meakins and Priestley[40] found changes in the blood indicative of marked impairment of aeration and therefore proving severe damage in the lungs,

36. Prentiss, A.M.: Chemicals in War, New York, McGraw-Hill Book Co., Inc., 1937.

37. Gilchrist, H. L., and Matz, P.B.: The Residual Effects of Warfare Gases: The Use of Clorine Gas, With Report of Cases, Med. Bull. Vet. Admin. 9:229, 1933.
Gilchrist, H. L., and Matz, P.B.: The Use of Mustard Gas, With Report of Cases, id. at p. 339.
Gilchrist, H.L., and Matz, P.B.: The Use of Phosgene Gas, With Report of Cases, Med. Bull. Vet. Adminis. 10: 1, 1933.
Gilchrist, H.L., and Matz, P.B.: The Use of Arsenical Compounds, with Report of Cases, id. at p. 79.

38. Editorial: Residual Effects of Chlorine Gassing, Brit. Med. J. 1: 196, 1933.

39. Berghoff, R.S.: The More Common Gases; Their Effects on the Respiratory Tract, Arch. Int. Med. 24: 678, 1919.

40. Official History of the War, Medical Services, British, vol. II, 1923.

but concluded that these changes were a neurotic manifestation,[41] a conclusion which evokes only amazement in modern physiological and clinical students of pulmonary disease. Nevertheless, all of these studies, the data of earlier authors analyzed in those reports, and various discussions of civilian gassing such as the remarkably well annotated studies of Von Oettingen[42] on nitrous fumes, prove that sequelae to gassing, in the form of bronchitis, bronchiectasis, pulmonary emphysema, pulmonary fibrosis, cor pulmonale and activation of latent tuberculosis, are of common occurrence.

The establishment of these disorders as sequelae to gassing is very difficult without the previous establishment of the fact of gassing as outlined above. Nevertheless it must be remembered that such antecedent evidence of gassing may be lacking: the patient may have been unconscious or delirious because of other trauma (injury) following exposure to the gas and unable to complain of the symptoms; he may not have had competent medical attention or any attention following exposure to the gas, etc. If the fact of gassing has been established and if a patient previously shown by competent medical testimony to have been without pulmonary disease before gassing, shows a few weeks after the accident evidence on physical or x-ray examination one of the sequelae listed above, it can be safely concluded that the gassing caused the sequela. That is as close as one can come to perfect proof as to the causal relation between exposure to gas and the development of pulmonary disease thereafter.

Special Diagnostic Procedures. In the case of bronchitis and bronchiectasis physical examination and ordinary x-ray studies may reveal nothing abnormal and bronchoscopy and the introduction of the opaque oil, lipiodol, may be necessary to establish the diagnosis. It is doubtful if a patient can be made to submit to these procedures; he might be persuaded to do so, if he is not neurotic or malingering, on the basis that the results of such studies would be beneficial to him. In the ab-

41. Neurotic Manifestation: That is, a symtomatic expression of a neurosis, a disturbance believed to be due to psychological causes or maladjustment without any organic basis or lesion. Psychoneurosis involves a mental disorder caused primarily by some psychic conflict or maladjustment, without disturbance of intellectual functions, without distortion of reality, and without primary mood pathology (abnormality).

42. von Oettingen, W.F.: The Toxicity and Potential Dangers of Nitrous Fumes, Pub. Health Bull., No. 272, 1941.

sence of such studies the diagnosis would have to rest on the history and an estimate of the patient's personality.[43]

Late Effects: Problem of Proving Causal Connection. Where a period of months or years elapses between exposure to a gas and the development of one of the conditions considered to be sequelae to gassing, establishment of a causal relation is very difficult, if not impossible. Nevertheless, such situations might arise. For instance, a patient may feel entirely well after gassing, but yet have within his lungs an area of bronchiectasis which might make its presence known long after the gassing only by a massive hemorrhage. This hemorrhage might very well be a sequel of the exposure to gas several years before, or it might not in that he may have had the bronchiectasis before he was gassed, or it may have developed consequent to a respiratory infection which occurred some time after the episode of gassing. The same situation might arise in the case of the other sequelae of exposure to gases. Even the most meticulous medical study may not suffice to establish the origin of the manifestations in some patients of this type, although in other instances careful medical history may be very helpful.

ASPIRATION OF LIQUIDS

Cause and Effects of Aspiration. The entry of large volumes of any liquid into the lungs results in drowning and suffocation, if this material is not very soon expelled. This pulmonary disorder will not be discussed here.[44] Certain liquids are extremely irritating when aspirated even in small amounts, while others cause no immediately detectable reaction but, after the passage of time, may cause extensive disease of the lungs.

Substances which might with some frequency cause immediately detectable damage when aspirated include kerosene and gasoline[45,46] and gastric contents. In the case of the first two it is not clearly established that pulmonary lesions

43. The instillation of lipiodol has been performed thousands of times in patients, with only mild discomfort and extremely rare reactions. The introduction of a bronchoscope is more discomforting, but, in good hands, is not a dangerous procedure.

44. See, in this Symposium series, Helpern, M.: The Medicolegal Examination in Cases of Suspected Homicide, J. of Crim. L. & Crim. (March-April, 1946).

45. Machle, W.: Gasoline Intoxication, J.A.M.A. 117: 1965, 1941.

46. Cope, C. L.: Aspiration of Petrol, Lancet 1: 469, 1942.

found after aspiration are due to the action of these liquids directly on the lungs: their vapors may act like irritant gases and these substances in addition are systemic poisons[47] and may act on the lungs after having first entered the blood stream. Whatever the preliminary mechanism, we can say with certainty that following aspiration of gasoline or kerosene, extensive destruction of the lining of the bronchi and of the alveolar spaces may occur, causing bronchopneumonia with the production of a large amount of brownish sputum,[48] smelling characteristically. In severe poisoning, this is not important, as the systemic intoxication is usually fatal; in milder poisoning after aspiration, the pneumonia may be the chief or only manifestation.[46] In instances in which healing is taking place, invasion by bacteria may occur, with resulting exacerbation or even fatality. There is no record of the subsequent course of patients in the group with symptoms which are predominantly or solely pulmonary, although there is no reason to believe that all escape the occasionally occurring, more lasting, consequences of pneumonia, i.e., bronchitis, bronchiectasis, emphysema and fibrosis.

Aspiration of gastric contents occurs in individuals who vomit while unconscious and the entry of the gastric acid, digestive juices and food particles into the lungs results in a fulminating type of pneumonia which is resistant to treatment ordinarily successful in pneumonia. Secondary bacterial infection usually is part of the picture. The diagnosis of aspiration pneumonia is made with certainty only at autopsy and, accordingly, there are no data available on the subsequent course of patients who may recover from it.

A not uncommonly encountered lesion in hospital practice is oil aspiration pneumonia. The oils implicated are usually medicinal oils,[49] i.e., mineral oil, castor oil and cod liver oil, a smaller number of instances occurring after aspiration of food oils such as cream, olive oil, cottonseed oil, etc. It is possible that the large number of marine disasters in recent years may give rise to the recognition of pulmonary complications following aspiration of fuel or lubricating oils.

47. Causing collapse, disorientation, jaundice, signs of renal irritation, etc.

48. Sputum: Matter ejected from the mouth, having its origin in the mouth, trachea, bronchi or lungs.

49. Kaplan, L.: Combined Cod Liver Oil and Liquid Petrolatum Pneumonia in a Child, Am. J. Dis. Child. 62: 1217, 1941.

Aspiration of oils may occur in patients who are unconscious or who, because of depression of the activity of parts of the brain, have lost the cough reflex. An important group of cases of oil pneumonia occurs as the consequence of the protracted use of oily medications in the nose, with a slow irregular trickle of the oil into the lungs. Oil in the lungs sets up an irritative process which results in bronchitis and patchy pneumonia associated with marked fibrosis. Once established, the disease runs an irregular course of exacerbations and remissions, apparently unrelated to subsequent aspirations of oil, with a good deal of disability because of cough, fever and chest pain. The outlook appears to be unfavorable, although this is not conclusively true, since the diaguosis is with certainty made only at autopsy.[50]

Problems of Identification Following Aspiration of Liquids. Proving that pulmonary injury is due to the aspiration of an irritating liquid involves many of the same problems as in the case of inhalation of gases, with the important difference that liquids, being more stable and more easily handled, can be more readily identified. Here again, if sigus of bronchitis and/or pneumonia develop within a day or two after the accidental aspiration of a liquid which is known to be irritating, the pulmonary lesion should be regarded as the consequence of such aspiration. Aspiration of any liquid is more readily appreciated by the victim than the inhalation of some gases, since the immediate response to the former is coughing, choking and the production of sputum, unless the patient is unconscious. In the latter instance, the sputum may have a characteristic and readily identifiable odor or the liquid may be detected in the hair, about the face or on the clothing. The establishment of the

50. Editor: For medicolegal cases involving aspiration of irritating fluids (excluding drowning), see the following:
Baltimore & O.R. Co. v. Branson, 128 Md. 678, 98 Atl. 225 (1916), reversed 242 U.S. 623, 37 Sup. Ct. 244 61 L. Ed. 534 (1916) (Spray from paint gun); Evans v. Chevrolet Motor Co., 232 Mo. App. 927, 105 S.W. (2d) 1081 (1937) (Inhalation of spray solution containing soap, water and whale oil used to settle dust); Depre v. Pacific Coast Forge Co., 145 Wash. 263, 259 Pac. 720 (1927) (Fumes from sulphuric and muriatic acids); MacRae v. Unemployment Compensation Commission, 217 N.C. 769, 9 S.E. (2d) 595 (1940) (Sputum expectorated by tubercular employee entered mouth of X, a fellow employee and two months later he was found to be suffering from tuberculosis. Held: a compensable accidental injury arising out of and in the course of employment.)

fact that bronchitis, bronchiectasis, pulmonary emphysema and fibrosis, or their exacerbation if previously present, are the consequences of aspiration of an irritant liquid is subject to the same difficulties as in the case of gassing (*vide supra*); the same is true in regard to the question of the lighting up of latent tuberculosis. Attempts to prove such a relationship should be based on the type of reasoning and study already outlined for gases.

The case of oil aspiration pneumonia offers many more difficulties in that there may be a period of days, weeks or possibly a longer time before *any* manifestation of pulmonary disease becomes apparent. The property which the oils have of inducing fibrosis[51] may make x-ray studies valuable in this respect, since the appearance and progression of a fibrotic process in the lungs, where none was present before, within a period of months after known inhalation of oil, would be most suggestive. Where the history of aspiration of oil is not obtainable because of unconsciousness of the victim at the time of the accident, physician's data as to the presence of oil in the hair, in the nose, about the face and on the clothing may be helpful. If the disease is fatal, autopsy affords conclusive proof as to the nature of the process.

FOREIGN BODY IN THE LUNG

Modes of Entrance. Foreign bodies may enter the lungs as missiles or debris blown in through the chest wall, downward through the neck or upward through the abdomen. Foreign bodies may also enter the lungs by aspiration (a) through industrial accident (pins, nails or screws held between the lips), (b) dental procedures or trauma to the mouth, (c) operations on the nose or throat (fragments of tonsil or other tissue) or (d) other accidents. In rare instances a foreign body in the oesophagus or stomach, i.e., a fishbone, may work through into the lung.

Symptomatology. The symptomatology of foreign body in the lungs varies to some extent with the mode of entrance and with the nature of the body. Aspirated solid material at first may cause pain and strangulation as it enters the larynx[52]; proceeding downward into the trachea, it may cause

51. Fibrosis: The formation of fibrous tissue.
52. Larynx: Air passage containing the vocal cords, situated between the mouth and the trachea.

pain, cough and severe spasm resulting in wheezing, and the patient may feel it bobbing about within the trachea. Thence it passes into a bronchus, almost always a lower bronchus and usually in the right lung.[53] Once past the trachea and in a bronchus, all symptoms cease for a greater or lesser period of time. Accordingly, if the patient is excited or unconscious while inhaling the body, he may be unaware of its passage through the larynx and trachea, and if, when he is quieted down or restored to consciousness, it is in a bronchus, he may not know that he aspirated it. Once lodged in a bronchus, the body may cause symptoms after a period of a few hours to over twenty years. If obstruction of a bronchus occurs, loss of function of the lobe of the lung into which it leads results. The air trapped in this lobe is absorbed into the blood stream and the lobe collapses (atelectasis). This functionless lung tissue is very susceptible to infection. In other instances the local irritation may give rise to the coughing of blood (hemoptysis), in larger or smaller volume. Vegetable matter, such as nuts and grains, is extremely irritating and commonly causes a fulminating tracheobronchitis[54] and pneumonia, with severe prostration, high fever and the production of a large amount of thick tenacious sputum; unless treated, this condition may be rapidly fatal. Less irritating materials may give rise to a variety of other conditions. In some instances the foreign body may cause local damage to a bronchus, giving rise to localized bronchitis which occasionally heals over without further incident; usually, however, the patient is left with evidences of localized bronchitis and/or bronchiectasis in the form of chronic cough with localized evidences of congestion on physical examination. The symptoms of bronchitis due to foreign body may not become apparent for months after its aspiration. If the bronchitis is more severe, sputum may be profuse, fever high and prostration and wasting prominent. In some instances the surrounding lung is inflamed also (*pneumonitis*) and the signs of inflammation on examination are more widespread; the x-ray shows a patch of inflammation in the lung substance. The center of an area of pneumonitis may liquefy,

53. The right lower bronchus leads almost straight down from the trachea instead of at a sharp angle, as in the case of the other bronchi.

54. Fulminating tracheobronchitis: Inflamation of the trachea (wind pipe) and bronchi which begins suddenly with an intense severity.

becoming an abscess, and the patient shows high irregular fever, rapid wasting, profuse, extremely foul sputum (malodorous enough to be disgusting at a distance of ten feet or more) and characteristic changes in the fingertips known by the descriptive term of "clubbing".[55] If untreated, a lung abscess may rupture into the pleural space, causing empyema[56] or through the diaphragm into the liver or into the abdominal cavity. The severe forms of bronchitis, pneumonitis and lung abscess give symptoms usually within a few days to a few weeks after aspiration of the foreign body. Any suppurative[57] pulmonary disease may give rise to abscess of the brain. In rare instances an aspirated foreign body may cause no symptoms whatsoever, making its presence manifest months or years later, only when it is coughed up. X-ray studies are frequently helpful, for almost invariably they reveal the foreign body if it is metallic or calciferous (bones, teeth, some pebbles); unfortunately, vegetable matter is not detectable by this procedure.[58]

Problems associated with foreigu material forced in or blown in through the chest wall or diaphragm may be discussed under two heads, namely: (a) those associated with penetrating wounds of the chest (*vide infra*) and (b) those associated with damage which may occur if the foreigu body remains in the lungs for any length of time after the accident. There are some resemblances and some differences in the nature of the processes set up when foreign bodies enter the lungs through the chest wall as opposed to the trachea. In the former the picture may be colored by the occurrence of

55. "Clubbing": A bulbous enlargement of the ends of the fingers, with curvature of the nails.

56. Empyema: Pus within the pleural cavity.

57. Suppurative: Giving rise to the formation of pus or associated with its presence.

58. Editor: Mediocolegal cases involving aspiration of a foreign body seem most frequently to be malpractice actions against dentists for alleged injury to the lungs caused by aspiration of a tooth or other material during course of treatment, usually while the patient is under an anesthetic. See, for instance, the following: McGehee v. Schiffman, 4 Cal. App. 50, 87 Pac. 290 (1906); Nelson v. Parker, 104 Cal. App. 770, 286 Pac. 1078 (1930); Bollenbach v. Bloomenthal, 341 Ill. 539, 173 N.E. 670 (1930), reversing 255 Ill. App. 305 (1930); Toy v. Mackintosh, 222 Mass. 430, 110 N.E. 1034 (1916), Ann. Cas. 1918 C1188; Yarrington v. Pittenger, 6 N.J. Misc. 710, 142 Atl. 565 (1928); 8 N.J. Misc. 143, 149 Atl. 347 (1930); Schamoni v. Semler, 147 Ore. 353, 31 P. (2d) 776 (1934); Goehring v. McDiarmid, 289 Pa. 193, 137 Atl. 187 (1927).

manifestations of penetrating wounds of the chest (*vide infra*) and also by differences in localization. Foreign bodies entering through the chest wall and coming to rest in the substance of the lung may give rise to pneumonitis or to lung abscess, as discussed above. However, foreign bodies entering through the chest wall or diaphragm only very uncommonly come to rest in a bronchus and accordingly do not usually give rise to manifestations of localized bronchitis and/or bronchiectasis. Similarly, such a foreign body may not enter the lung at all, remaining within the pleural space, causing pleurisy[59] with stabbing pain on coughing or deep inspiration, and a friction rub[60] audible over the area.

Foreign Material in Pleural Space: Clinical Manifestations. The presence of foreign material in a pleural space

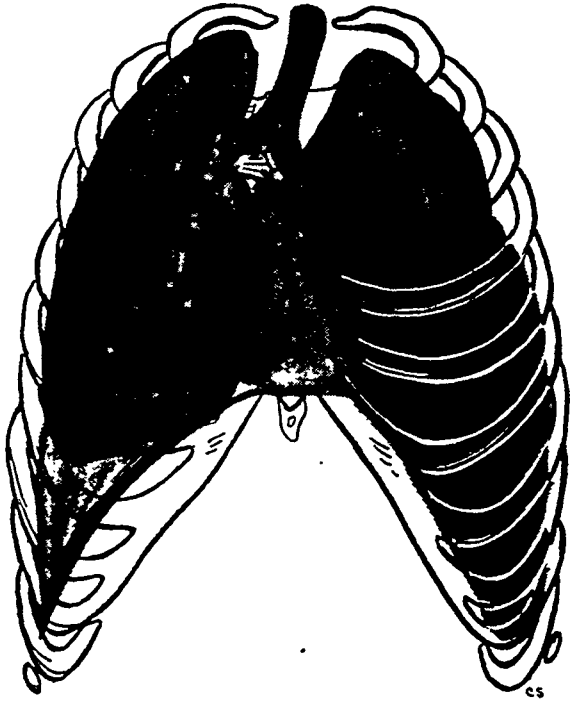

Fig. 4. Pleural Effusion. Note how collection of fluid (solid black area) in pleural space has collapsed lung and pushed the thoracic (chest) contents toward the side opposite to the effusion.

59. Pleurisy: Inflammation or irritation of a pleural surface.
60. Friction rub: A scratching or rubbing sound heard through the stethoscope over an area of pleurisy.

causes an outpouring of fluid from the blood stream into this space, giving rise to typical manifestations. The patient complains of a heavy sensation on that side, slight cough and variable degrees of shortness of breath; there may be slight fever. Since the fluid collapses the lung on that side and may push the entire thoracic contents toward the other side, the trachea, heart and lungs are accordingly displaced. No breath sounds or other sounds are audible over the fluid when one listens with the stethoscope, and the percussion note[61] is duller than over the portion of the chest containing aerated lung. In extreme cases the fluid may accumulate to such a degree as to compress the blood vessels leading to the heart, thus impeding the return of blood to the heart, or it may interfere with cardiac action itself; this may result in fatality if the condition is not treated. Treatment consists in the evacuation of the fluid through a hollow trocar.[62] The fluid may recur until the foreign body is removed. Some care must be exercised in removing the fluid, since too rapid removal of too large an amount may so derange cardiac and/or pulmonary function as to cause collapse or even death. If infection is introduced with the foreign body, or subsequently, pus may accumulate in the pleural space in large amount, i.e., a pint to a quart or more, with all the manifestations of empyema, i.e., those of pleural fluid plus the fever, wasting and prostration of severe, intractable, continued infection. This is treated by drainage to remove the pus, as sulfonamides are useless; now that penicillin has become freely available it is likely that the use of that substance will make treatment simpler and more effective. At present it is sometimes necessary to do a number of operations, including removal of ribs (*thoracoplasty*), for drainage and to collapse the empyema cavity, so that is not uncommon for the patient to be quite ill for many weeks or months.

In any case, the symptoms due to foreign body in the lungs do not clear up until the object is removed. Occasionally, the foreign body will be coughed up spontaneously, but usually it must be removed either by bronchoscopy,[63] if as-

61. Percussion note: Sound made by tapping the chest.
62. Trocar: Sharp, pointed, hollow tube of metal.
63. The insertion of a bronchoscope (see f.n. 27, supra) and examination of the visible lung through it.

pirated,[64] or by operation if otherwise introduced into the lungs.

Problems of Identification which Arise From Foreign Body in the Lung. The entry of a foreign body into the lung is usually a dramatic episode not often overlooked by a normal individual. Accordingly, a history of the entry of such a body into the chest, either by aspiration or by penetration, followed within a period of a few hours or days by the onset of some of the symptoms of foreign body in the lung described above, suggests a causal relation which can be corroborated by x-ray demonstration of the body and finally by its removal. However, as was pointed out above, an aspirated body in a bronchus need cause no symptoms for a period of some hours to twenty or more years, and unconsciousness, intoxication or excessive excitement or distraction might result in unawareness on the part of an individual that he has inhaled the foreign body. In some instances the long latent period between the accident when recognized and the first occurrence of symptoms may confuse the diagnosis. In other instances where the aspiration of several bodies is recognized several might be coughed up, leaving the victim with the belief that he had brought up all he had inhaled, so that when he developed pulmonary symptoms some months later, he might overlook the fact that foreign body might be at fault. The development of evidence of localized bronchitis[65] or bronchiectasis,[66] atelectasis,[67] pneumonitis[68] or lung abscess should cause inquiry as to possible aspirations, however remote in time, and should make the physician undertake x-ray studies which might reveal the foreign body. Indeed, many physicians feel that the occurrence of any of the above disorders, with or without a history of inhalation of a foreign body, is an indication for bronchoscopy, to examine the region involved and remove any body there detected. Where

64. If aspirated, the foreign body will be in the trachea (wind-pipe) or in the small branches (bronchi) which subdivide to carry air to and from the lung tissue.
65. Bronchitis: Inflamation of the bronchial tubes. See discussion supra.
66. Bronchiectasis: Dilation of the bronchi or of a bronchus. It may affect the tube uniformly or it may occur in irregular pockets. It is marked by fetid breath and paroxysmal coughing, with the expectoration of mucopurulent matter. See discussion supra.
67. Atelectasis: Partial collapse of the lung. See discussion supra.
68. Pneumonitis: A form of pneumonia. See discussion supra.

a definite history of aspiration is not obtained, the nature of a foreign body found may throw some light on the circumstances of its entry into the lungs. Thus a history of injury to the mouth with the breaking of teeth or dentures, with the subsequent finding of such fragments in the lung, should be fairly conclusive evidence as to aspiration at the time of accident, even though the victim was unaware of it. Similarly, the finding in the lungs of tacks, screws or small parts of machinery which might have been held between the lips of the victims while at work should be sufficient proof of aspiration during an accident which occurred during work. Or the finding of a pebble in the lung of the victim of an accident which resulted in the victim's being thrown on his face to the ground in pebbly soil might be considered as proof of aspiration during the accident, even though the victim was unaware of having inhaled the foreign body. A foreign body which might disintegrate and be coughed up in fragments or become merged into the pus of a lung abscess, i.e., fragments of tonsil or a piece of meat, might be undetectable at the time of bronchoscopy several weeks after aspiration, when the complete clinical picture is present. Without a history of the aspiration of such material within a period of a few weeks or months previously, it is not possible to demonstrate that the lesion in the lung is due to a foreign body.

In the case of foreign bodies blown in or forced through the chest wall, the immediate problem is that of a penetrating wound, and observations made at that time should demonstrate the presence of foreign bodies. In this connection it is to be remembered that a very small abdominal wound or neck wound may be the point of entry of a pulmonary foreign body which may occasionally be overlooked; in most such cases the pulmonary symptoms would call attention to the entry of such material into the chest even though no wound is visible over the chest (*vide infra.*)

After the removal of a foreign body, the inflammatory reaction in the bronchi, lungs or pleural space provoked by it should subside in a longer or shorter time. In the case of an infected pleural space the course may be long and sequelae, in the form of pain, shortness of breath and susceptibility to infection, important. Most patients with localized bronchitis, pneumonitis or lung abscess due to foreign

body experience recovery, but some remain with chronic changes in the lungs in the form of bronchiectasis or fibrosis. When chronic symptoms develop immediately after an acute episode due to aspirated foreign body, they should be considered a consequence of its aspiration. If a long latent period exists between the appearance of chronic manifestations and the subsidence of the acute manifestations, a causal relation may be impossible to prove unless x-ray evidence, based on ordinary x-ray studies or on films made after the introduction of lipiodol,[69] demonstrate that the chronic symptoms are due to a lesion in the location of an antecedently demonstrated but subsequently removed foreign body.

Lighting up of a latent tuberculous process during reaction to a foreign body is theoretically possible but would be difficult to prove, unless the exacerbation of the tuberculosis occurred during or shortly after the acute illness due to inhalation of the body. Pulmonary tuberculosis does not become manifest for several weeks at least after the infection sets in and, accordingly, the finding of active tuberculosis a few days after aspiration of a foreign body indicates no causal relation between the two. This does not, however, rule out exacerbation of a previously existing tuberculous lesion.

PENETRATING WOUNDS

Depending on its size, depth and location, a penetrating chest wound may have a number of different effects singly or in combination. A common consequence of such wounds is fracture of one or more ribs. This may be so painful as to limit respiratory movements and, in addition, the broken ends of the ribs may tear the lining of the pleural space,[70] giving rise to complications described below.[71]

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69. See f.n. 29, supra.

70. Pleural space: Each lung is surrounded by a serous membrane called the pleura. This has two layers: the visceral pleura which immediately invests the lungs and an outer layer called the parietal pleura which lines the inner wall of the chest cavity or thorax. The pleural space is the space between the visceral and the parietal pleura.

71. Editor: For medicolegal cases involving penetrating injuries of the thorax and lungs, see the following: Denver and R.G.R. Co. v. Mitchell, 42 Colo. 43, 94 Pac. 289 (1908) (Horseback rider thrown from horse frightened by train); H. L. Hunt, Inc., v. Frisby, 185 Ark. 1188, 51 S.W. (2d) 516 (1932) (Fracture of ribs and injury to lungs: verdict for $25,000 reduced to $20,000.);

A certain amount of destruction of, or at least damage to, lung tissue occurs in patients with penetrating wounds of the lungs. If severe, death or severe disability may occur quickly. The cause of the death or disability may be the rendering of a large part of the pulmonary tissue functionless, so that the victim dies of asphyxia, or it may be due to shock consequent to extensive tissue damage. The damaged lung provides a favorable culture medium for bacterial growth, and secondary infection may become a problem. If recovery ensues under these circumstances, healing is usually associated with a severe degree of disability for many months, with some permanent residual disability in some cases. Specific treatment consists in operative removal of dead or non-viable lung, removal of foreign bodies and taking of certain measures against complications.

The lung tissue is richly supplied by blood vessels and, accordingly, a good deal of hemorrhage usually occurs. This may occur into the damaged lung or into the remaining nor-

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Mulcahay v. Larson, 130 Conn. 112, 32 A. (2d) 161 (1943) (Five fractured ribs causing penetrating injury of lungs); Hannaher v. Blue Cab Co., 322 Ill. App. 277, 54 N.E. (2d) 257 (1944) (Fractured ribs punctured lungs); Niemeyer v. McCarty (Ind. App.) 48 N.E. (2d) 829 (1943) (Fractured ribs allegedly resulting in injury to lungs); Birmingham v. Lehigh & W. Coal Co. (N.J.L.) 95 Atl. 242 (1915) (Consolidation of lung tissues allegedly occurring after accidental injury); Schott v. Weiss, 92 N.J.L. 494, 105 Atl. 192 (1918) (Passenger thrown from defendant's jitney bus while alighting—fractured rib and punctured lung); Kalogerakas v. Public Service Coordinated Transport, 10 N.J.Misc., 175, 158 Atl. 408 (1932) (Three fractured ribs resulting in pleurisy); Upton v. Bell Cabs, 154 So. 359 (La. App. 1934) (Fracture of five ribs on one side and two on the other resulted in traumatic pneumonia and pleurisy); Clemens v. Southern Advance Bag and Paper Co. (La. App.) 20 So. (2d) 749 (1944) (15% pneumothorax of left lung and pleural effusion following injuries); Fittin v. Sumner, 176 N.Y. App. Div. 617, 163 N.Y. Supp. 443 (1917) (Fourteen year-old male pedestrian struck by automobile sustained fracture of three ribs and puncture of lung); Fagerile v. New York Life Ins. Co., 129 Ore. 485, 278 Pac. 104 (1929) (Gunshot wound through left lung; question of total and permanent disability under insurance policy.); Poneitawcki v. Harres, 200 Wis. 504, 228 N.W. 126 (1930) (Fracture of rib sustained by woman allegedly caused injury to lung and resultant tuberculosis.); Williams v. City of Spokane, 73 Wash. 237, 131 Pac. 833 (1913) (1400 pound cement form fell on bridge carpenter; resultant fracture of eight ribs allegedly caused pleural adhesions which greatly interfered with respiration); Lanahan v. Hydraulic-Press Brick Co. (Mo. App.) 55 S.W. (2d) 327 (1932) (Watchdog ran into night watchman and knocked him down; watchman fractured ninth, tenth and eleventh ribs and this caused him to develop lobar pneumonia, from which he died, according to autopsy reports. Held: a compensable accident under the Workmen's Compensation Act.).

mal tissue, and the victim may drown in his own blood or die of shock due to blood loss. A variable amount of blood may be lost when coughed up or as it runs from the external wound. The treatment of this complication is to control the hemorrhage and restore the lost blood by transfusion.

Hemothorax. A small wound of the pleural surface may result in a good deal of bleeding into the pleural space (*hemothorax*). This is a serious complication in that the victim may look deceptively well at first, with little or no external bleeding, only to go into a rapid decline after a period of one-half to several hours. This occurs after a considerable amount of blood has accumulated within the pleural space, causing embarrassment of circulation and respiration by compression, in the same manner as described above as due to pleural fluid. In addition, since the potential capacity of the pleural space is several quarts, the patient may bleed to death internally, with no indication of outward hemorrhage.[72] The treatment is to control bleeding if possible, transfuse to make up for blood lost and to remove blood from the pleural space by means of a hollow needle, rinsing the space with sterile salt solution. During the accident itself or as a consequence of manipulation designed to relieve hemothorax, the pleural space may become infected (empyema), with the development, as described above, of the manifestations of infection. Hemothorax is likely to be associated with severe prolonged disability which is made even longer by the development of empyema.

Pneumothorax. It was pointed out in the discussion of pulmonary physiology (*vide supra*) that there normally exists in the pleural spaces a negative pressure which is necessary for effective respiration and which maintains the lungs in their distended state. Penetrating wounds allow the entry of air into the pleural space either through the perforated chest wall or by puncturing the lung; the lung in some cases may be punctured by the sharp ends of broken ribs. The entry of air into the pleural space destroys the negative intrapleural pressure, the pressure becoming that of the atmosphere. This is known as *pneumothorax.* The lung on that side accordingly collapses and shortness of breath (dyspnea) and cyanosis (bluish coloration of the skin due to insufficient

72. The total blood volume is 4.5 liters. Loss of a liter or more of blood may have serious consequences, including death.

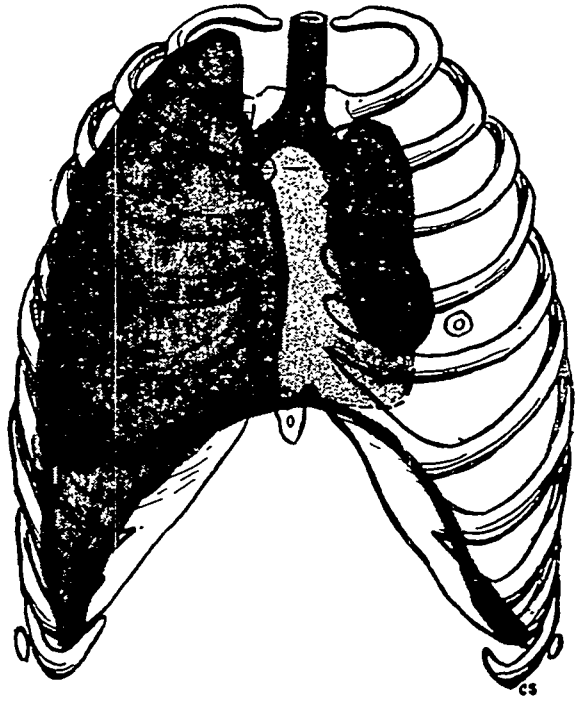

Fig. 5. Pneumothorax. Entry of air into the left pleural space has restored the negative intrapleural pressure normally present with the result that the left lung has collapsed.

oxygenation) may develop. If the wounds are sealed off, the air is slowly absorbed and the lung re-expands. Pneumothorax of this type in not uncomfortable in a patient at rest, although it makes strenuous exertion impossible. Indeed, it may act in a beneficent manner in that it is always associated with a collapsed, motionless lung on the same side; a bruised or oozing lung heals more readily when collapsed and at rest. On the other hand, if the pressure of the air in the pleural space increases, the function of the other lung and of the heart is impaired and death may result. This increase in pressure in the pneumothorax is known as *tension pneumothorax* and results from the check-valve action of certain small wounds. Respiration was described in the section on physiology (*vide supra*) as consisting of two phases, an active muscular phase during inhalation and a phase of relaxation during exhalation. The forceful muscular action of inhalation may draw such air through a narrow wound, air

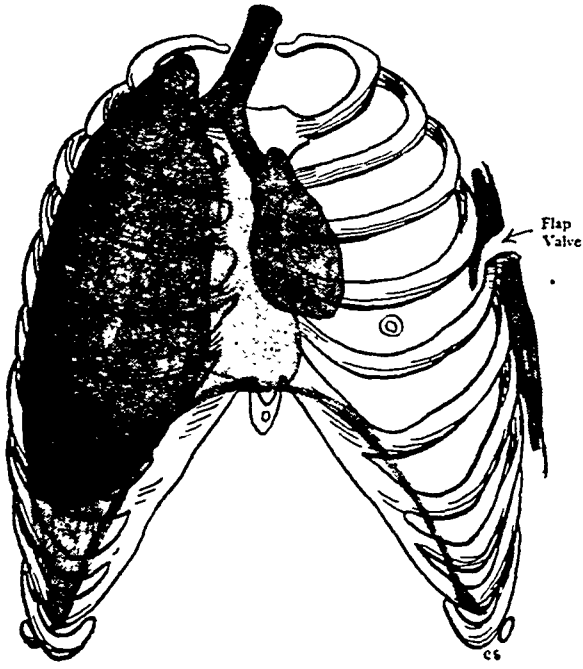

Fig. 6. Tension Pnemothorax. The flap value action of the penetrating wound enables air to be drawn in during inspiration which cannot be expelled during expiration. The increasive pressure may impair function of the heart and opposite lung, and in extreme cases, cause death if not relieved.

which cannot be expelled during exhalation. Treatment consists of the introduction of a hollow needle into the chest, connected with a rubber tube leading below the surface of water in a bottle, so that air under pressure in the pneumothorax may escape while air from the outside is kept out by the water in the bottle. After 48 hours this may be removed, as by then the wounds giving rise to a check-valve action will have closed over. A pneumothorax may become infected, giving rise to *empyema.*[73]

Interstitial Emphysema. Air from a bruised lung may be forced into the tissues of the thorax and spread up into the neck (*mediastinal emphysema*) or spread through the tissues of the chest wall (*interstitial emphysema*). The latter is usually not troublesome, unless the air works its way up

73. Empyema: Accumulation of pus in a cavity of the body, especially in the chest.

around the trachea, impairing respiration. It is readily detected by a crackling sensation on palpating the area and is relieved by introducing needles into the skin. Mediastinal emphysema may cause more severe respiratory embarrassment, requiring operation to release the trapped air. It is detected by characteristic crackling sounds drowning out the heart sounds on auscultation.[74]

Until relatively recently most patients with serious chest wounds died; recent advances in operative technique, anesthesia, oxygen therapy and control of shock, hemorrhage and infection have made the outlook much more favorable. Nevertheless, patients with such wounds have long periods of disability. The handling of chest wounds is at present undergoing many changes and, accordingly, it is not possible to make any conclusive statement regarding the amount of prolonged or permanent disability occurring after such wounds. Infection, extensive removal of lung tissue and resection[75] of ribs to secure exposure for operation are factors that might be expected to leave the patient with a greater or lesser degree of impairment of pulmonary function.

Problems of Identification in Penetrating Chest Wounds. Patients with penetrating chest wounds are almost always so severely disabled as to require immediate medical attention. Accordingly, the relation of the injury to the subsequent course is likely to be established by the close medical supervision which these patients require. Moreover, the period of observation is usually so long that sequelae will develop under the eyes of the attending physician and will be recognized as such. A small number of patients with chest wounds due to the penetration downward of a very small missile of high velocity which has entered the neck may die of hemothorax, but appear to die a non-traumatic death. The finding of a neck wound, however insignificant in size and appearance, should be an indication for postmortem examination.

NON-PENETRATING TRAUMA TO THE CHEST

Non-penetrating trauma to the chest may be discussed under three heads: the effects of (a) crushing, (b) blast and (c) a simple blow or fall.

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74. Auscultation: Listening for sounds within the body, usually by means of a stethoscope.

75. Resection: Excision of a part of an organ.

Crushing injuries. Crush injuries involving fractures of many ribs in several parts of the chest are likely to be serious and are usually associated with various degrees of hemorrhage, shock and the destruction or rendering functionless of portions of the lungs; the damaged lungs are very susceptible to infection and accordingly bacterial invasion may be a complication. Hemothorax,[76] pneumothorax,[77] and mediastinal emphysema[78] may also be part of the picture. The manifestations of shock, i.e., pallor, sweating, restlessness changing to stupor, dyspnea, rising pulse rate and falling blood pressure, indicate a serious prognosis; only energetic treatment, including transfusion of blood or plasma, may save the patient. Hemothorax, pneumothorax and interstitial or mediastinal emphysema may aggravate the patient's condition (*vida supra*) and require immediate treatment. Severe pain, exacerbated by breathing and involving several portions of the chest wall, is important not only because of the discomfort but also because of the tendency to make respiration very shallow and therefore inefficient. Treatment should include strapping of the chest and the use of analgesic drugs[79]; the latter must be used cautiously, since they depress respiration and may thereby cause fatality. The crushing of a good deal of the thoracic cage so that it loses its rigidity in several places may produce a respiratory abnormality which is fatal in itself; one side of the chest may be sufficiently strong to make possible relatively normal respiration on that side, while the other side of the chest, badly crushed, makes it impossible to secure the pressure changes on that side necessary for respiration. Respiration may therefore occur back and forth from the relatively well functioning lung into the poorly functioning lung. With the shallow respiration consequent to crush injuries, little or no respiratory exchange through the trachea to the outside air may occur. The patient in such cases uses up more and

76. Hemothorax: A collection of blood in the thoracic (chest) cavity.
77. Pneumothorax: An accumulation of air or gas in the pleural cavity. The air enters either by an external wound, a lung perforation, burrowing abscesses, or from the rupture of a superficial lung cavity. Pneumothorax is attended with sudden and severe pain and rapidly increasing dyspnea (breathlessness).
78. Mediastinal emphysema: The presence of air in the mediastinal tissues. The mediastinum is the space between the two pleural sacs which invest the left and right lungs respectively.
79. Analgesic drugs: Used for relief of pain.

more of the oxygen in the air and adds more and more carbon dioxide to it as it is passed back and forth between the two lungs, until finally symptoms of oxygen lack and/or carbon dioxide retention develop and the patient may die. A similar course of events may occur when one side of the diaphragm is paralyzed because of injury to it or to the phrenic nerve[80] on that side. Treatment calls for the use of oxygen and a tight strapping; too tight a binder may be harmful and it may restrict respiration. Because of pain and the difficulties involved in building up a high pressure in the chest following crushing injuries, victims of such injuries find it impossible to cough satisfactorily, so that secretions or blood accumulate in the lungs and respiratory passages obstructing the entrance and exit of air. These materials provide an excellent culture medium for bacteria, favoring the development of extensive bronchitis and pneumonia. To prevent the accumulation of blood and secretion in the lungs, the patient must be made to cough every few hours, one or more attendants supporting manually the broken parts of the chest. In any case, the onset of infection is associated with a high sustained fever, sweating and increased prostration. The infection which follows a crush injury may be pneumonitis, pneumonia, abscess or empyema. Depending on the severity of the damage and the development of infection, the victim will be seriously disabled for a variable period of time; the disability is almost always prolonged and usually requires many weeks of hospitalization. Late sequelae have not been studied, since in former years fatality following crush injuries to the chest was the rule and only recently have appreciable numbers of survivals been obtained. Nevertheless, it seems reasonable to assume that the sequelae of fibrosis, bronchitis and bronchiectasis and emphysema, and those of empyema will be recognized in the future.

Blast Injuries. Although the effects on the lungs produced by blast were recognized many years ago, there was no widespread appreciation until recently of the severity of the injury experienced by individuals exposed to explosion. Severe trauma to the brain and intestines, the latter especially severe in the case of individuals in the water during an underwater explosion, may dominate the clinical picture; these

80. Phrenic nerve: The nerve which regulates the activity of the diaphragm.

manifestations will not be discussed here. Injury to the lungs[81] is apparently produced through the chest wall, since closure of the mouth and nose does not prevent it. A number of changes may occur in the lungs, consisting mainly in rupture of alveolar walls and hemorrhage; hemorrhage in a given area may be so extensive as to replace an entire lobe or lobule with blood clot, while hemorrhage into the pleura may give rise to massive hemothorax. Air contained in the alveoli may be forced into or through the tissues of the lungs by the explosion and with production of pneumothorax or the formation of air-filled blebs on the surface of the lungs. The function of the lungs is greatly impaired, and in addition the blood in the air spaces impedes aeration of the blood, so that depletion of oxygen in the blood plus carbon-dioxide retention occurs. The damage also acts to set up many foci of irritation in the lungs, so that cough and very rapid respiration result. The patients also have dyspnea (breathlessness) and appear cyanotic (bluish); signs of congestion in the lungs are detectable by physical and/or x-ray examination. If hemorrhage or tissue damage is severe, the manifestations of shock may appear and in some instances the signs and symptoms[82] of hemothorax or pneumothorax may develop. The lungs appear greatly ballooned out for reasons which are not clear. The injury caused by blast may be immediately or rapidly fatal, although recovery, on the other hand, may be rapid. Statistical studies of the after-effects of blast are not available, but scattered reports of pain and shortness of breath persisting for as long a period of 26 months after the injury have appeared.[83]

Blow or Fall. Injury to the chest caused by a blow or fall may take a variety of forms. One complaint may be bruising of and pain in the muscles of the chest which may disappear in a few days, never to recur and leaving no sequelae. In other instances, bruising of the lung may occur

81. Dean, D.M.: Thomas, A.R., and Allison, R.S.: Effects of High-Explosive Blast on the Lungs, Lancet. 2: 224, 1940.

82. Signs and symptoms: In medical diagnosis the term signs refers to physical evidences of abnormality, injury or disease perceptible to the examining physician as, for instance, a swollen joint, a fractured bone, cyanosis (bluish coloration of the skin). Symptoms are subjective feeling states which the patient must usually describe to his physician, as for instance pain, emotional disturbance and the like.

83. Correspondence, Lancet 1: 241, 1942.

and the patient exhibits stabbing pain on deep inspiration and/or coughing and a friction rub may be heard over the injured area. After a few days to a week or two, all of these manifestations disappear. There is no evidence that permanent damage to the lung occurs in this type of injury.

One or more ribs may be fractured as a consequence of external trauma (injury); pain, exacerbated by deep breathing and/or coughing is the chief complaint and indicates irritation of the pleural surfaces. There is tenderness to palpation (touch) over the area involved and the x-ray reveals the fracture. Fracture of a rib cartilage may be overlooked during x-ray studies, although the symptoms may be quite severe. A small area of interstitial emphysema overlying the fractured rib is not uncommon. The symptoms of fractured rib are usually well controlled by adhesive strapping and after a period of a few weeks cure results, with no sequelae. Infrequently, extensive interstitial emphysema extending up into the neck, or pneumothorax or hemothorax may be a complication of fractured rib, prolonging the period of disability. A condition known as "pathologic fracture" of a rib is not uncommon and is a consequence of antecedent disease of the rib involved. The ribs may be the seat of a variety of bone diseases, including cancer, which may have spread from some other part of the body. Such ribs may be fractured by very slight trauma, or even by a sudden movement, and heal poorly. This may be the first indication that a patient is suffering from a disease of the bones, including cancer.

Pneumothorax and/or mediastinal emphysema may occur following trauma to the chest by a blow or fall even in the absence of fracture of ribs. The onset of these conditions is usually indicated by severe pain, more severe often than is to be expected from the degree of trauma, and radiation of pain to a shoulder. In the case of pneumothorax temporary collapse may occur and dyspnea and cyanosis may be prominent. The manner in which these may cause serious disability (tension pneumothorax, etc.) has been discussed above, as has their treatment. Disability may last for several weeks and, in the absence of infection, should leave no residual. Pneumothorax and mediastinal emphysema may be recurrent.

Antecedent disease of the lung (tuberculosis with cavitation) or of the bronchi (bronchitis and bronchiectasis) is

usually associated with increased vascularity (blood supply) and frequently loss of elasticity of the part involved. Accordingly, transmission of the force of a blow to such areas may result in rupture of blood vessels and the coughing up of blood in varying amount, i.e., *hemoptysis.* The hemorrhage may be so severe as to be incapacitating in itself for several weeks; even if smaller in amount, hospitalization for several weeks is to be recommended. The process usually subsides, although scattered authenticated reports of activation of tuberculosis following non-penetrating trauma to the chest have been recorded. As was pointed out above, the course of tuberculosis is such that signs of activation of tuberculosis, with the exception of hemorrhage, take several weeks to develop

Problems of Identification in Non-Penetrating Trauma to the Chest. The manifestations of crush injury are so striking as to make it unlikely that they will be overlooked. These injuries are so severe and hospitalization accordingly so prolonged that the development of sequelae, which may cause severe disability, occurs under observation. Causal relationships are therefore established quite readily. The same is likely to be true of blast lung, although here, because of injury to the brain and/or rupture of intestines, the pulmonary lesion may be overlooked or underestimated at the time of accident. However, the cerebral (brain) and intestinal lesions may ultimately, i.e., after several months, heal satisfactorily, leaving respiratory symptoms as the only lasting disorder. The persistence of chest pain, congh and dyspnea (breathlessness) in a patient known to be the victim of blast should be enough to make one consider these pulmonary manifestations as a sequel of injury following the explosion. As in the case of sequelae of war gas poisoning, the evaluation of disability first becoming manifest some time after the accident may be difficult; this is particularly true where neurosis[84] has also been precipitated by the trauma.

Bruising of the lung should not be followed by sequelae. In that condition and following fracture of ribs, the relation

84. Neurosis: See f.n. 41, supra. For a full discussion of medicolegal aspects of traumatic neuroses see: Smith, Hubert Winston and Harry C. Solomon: Traumatic Neuroses in Court, Va.L.Rev. 30: 87, 1943; Annals Int. Med. 21: 367, 1944.

of the symptoms to the accident is readily established. Even in the case of a patient unconscious for some time after the accident, the diagnosis of fractured ribs can be made by x-ray. In the absence of infection, secondary to the complications of pneumothorax, hemothorax or mediastinal emphysema, disability is usually of short duration and sequelae should not occur. In the case of fracture of ribs which are the seat of a previously existing bone disease, the presence of antecedent partial destruction of the rib is detectable by x-ray and the subsequent course of the patient will be that of a victim of that bone disease.

The proof that hemorrhage[85] from the lungs, although associated with antecedent tuberculosis or bronchiectasis, is a consequence of a blow or a fall is often inconclusive. The occurrence of the hemorrhage immediately or at least within a few hours after the accident strongly suggests a causal relationship. It is well known that spontaneous hemorrhage is common in those diseases and, accordingly, its occurrence some time after an accident makes it unlikely that the latter caused it. The evidence regarding the hemorrhage should be fortified by the testimony of competent witnesses, including preferably a physician. In the case of an unconscious patient, the finding of frothy blood, i.e., blood mixed with air, on the face and clothiug, is helpful. In some instances following the initial massive hemorrhage, there is continued coughing up of blood-tinged sputum or of small amounts of blood; this phenomenon may serve to corroborate, where witnesses to the accident are not available, the patient's story of having had a hemorrhage from the lungs.

The question of the lighting up of latent tuberculosis or of its aggravation where overt by a blow is always difficult to settle because of the unpredictable course of the disease. If the blow or fall, by rupturing a small cavity, causes spread of the disease, weeks may elapse before the manifestations become severe enough to make the patient seek medical attention. On the other hand, tuberculosis occasionally manifests rapid and unexpected spontaneous exacerbations. Absolute proof of the relation of a blow or fall to the appearance or aggravation of tuberculosis is, in the

85. Hemorrhage: Blood flowing from a ruptured vessel, in this case coughed up.

absence of hemoptysis immediately after the accident, impossible of realization, except in the rare case where by chance x-ray studies of the chest were made before the accident and found negative. In the absence of this rare coincidence, competent medical testimony as to the time relation between the trauma and the appearance or exacerbation of symptoms, such as cough, fever, weight loss, weakness, a positive x-ray, etc., should be relied on to establish the fact that a short period, i.e., several weeks, elapsed between the two. However, if too short a period, i.e., several days, elapsed between the two, a causal relation is unlikely.[86]

TRAUMA TO THE CHEST CAUSED BY MUSCULAR STRAIN

A variety of disorders of the lungs may occur after a muscular strain: these include spontaneous pneumothorax,[87] spontaneous mediastinal emphysema, hemoptysis,[88] frac-

86. Editor: For cases involving alleged causation or aggravation of lung conditions, including tuberculosis, by a blow, see the following: Hayhurst v. Boyd Hospital, 43 Idaho 661, 254 Pac. 528 (1927) (Pneumonia and tuberculosis; verdict for $15,250, upheld); Malloy v. Chicago Great Western R. Co., 185 Iowa 846, 170 N.W. 481 (1919) (Rupture of blood vessels in lung); Consolidated Kansas City Smelting and Refining Co. v. Tinchert, 5 Kan. App. 130, 48 Pac. 889 (1897) (Crushing injuries of chest and lungs impairing respiratory functions); Manhattan Const. Co. v. Tottress, 161 Okla. 69, 17 P.(2d) 407 (1932) (Fall injuring back and allegedly aggravating pre-existing lung condition); Victor Gasoline Co. v. Weatherman, 163 Okla. 113, 21 P.(2d) 35 (1933) (Empyema resulting from sharp edge of barrel striking claimant's side; held: evidence sustained award of Workmen's Compensation benefits); Graham v. Kimmel Drilling Co., 113 Pa. Super. 235, 173 Atl. 721 (1934) (Pleurisy attributed by doctor to one of two falls sustained in course of employment, there being no other discoverable cause; award of Workmen's Compensation affirmed.); Mitchell v. San Antonio Pnblic Service Co. (Tex. Civ. App.), 15 S.W. (2d) 694 (1929), reversed in (Tex. Com. App.) 35 S.W. (2d) 140 (1931) (Tuberculosis in fifteen year-old girl allegedly caused by a blow when automobile in which she was riding was struck by defendant's streetcar.).

87. Editor: For cases of pneumothorax following muscular strain, see: Pacific Employers' Ins. Co. v. Pillsbury, 61 Fed. (2d) 101 1932); Delaune v. Young (La. App.) 166 So. 149 (1936); Quigley v. Industrial Accident Commission (Cal. App.), 35 P. (2d) 544 (1934) (Hemothorax) subsequent opinion; 3 Cal. (2d) 46, 43 P.(2d) 289; Kelce v. Swift and Co., 115 Pa. Super, 570, 176 Atl. 66 (1935)) (Hydrohemothorax). In all the cases cited the precipitating cause was the lifting of a heavy weight in course of employment.

88. Editor: For cases involving hemoptysis (spitting of blood) allegedly caused by muscular strain, see: Kangas' Case, 282 Mass. 155, 184 N.E. 380 (1933) (Employee suffering from pulmonary tuberculosis had serious pulmonary hemorrhage immediately after

ture of ribs which are the seat of previously established disease, and possibly activation of tuberculosis.[89]

Spontaneous Pneumothorax. Spontaneous pneumothorax may occur in individuals with disease of the lung, i.e., active or latent tuberculosis or pulmonary emphysema, and in normal individuals. The mechanism which causes the rupture of the pleura thus allowing air to enter the pleural space is not clearly understood except in the case of pulmonary emphysema. In this disease the lungs are overdistended with air and the pleura in a given area may become very thin, so that an air-filled thin-walled bleb gradually forms; this may rupture. In any case the manifestations are sudden onset of chest pain, frequently referred to a shoulder, and sudden dyspnea with cyanosis; collapse may occur. In the case of tension pneumothorax, the dyspnea and cyanosis increase rapidly, the visible veins in the neck become greatly distended, the blood pressure may fall and the patient may die. The treatment has been discussed earlier in this paper. Disability lasts for a few weeks usually. Spontaneous pneumothorax may be recurrent. The amount of strain necessary to cause this disorder cannot be stated, since not infrequently it occurs when the patient is not exerting himself or even when he is at rest; it is not necessarily a consequence of strain.

Spontaneous Mediastinal Emphysema. Spontaneous mediastinal emphysema[90] usually occurs in apparently normal individuals and its mechanism is not known. It may be associated with or followed by pneumothorax. It is ushered in by severe pain which may be referred to a shoulder and may be made worse on lying down; it may be confused

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overexerting herself in turning an overhead wheel while working at her loom in a factory.); McCluskey v. Stock Exch. Bldg. Corp., 100 Pa. Super. 136 (1930); Southwestern Surety Ins. Co. v. Owens (Tex. Civ. App.) 198 S.W. 662 (1917).

89. Editor: For cases allegedly involving activation of pre-existing dormant tuberculosis by muscular strain, see: Hartford Accident and Indemnity Co. v. Industrial Accident Commission, 140 Cal. App. 482, 35 P.(2d) 366 (1934); Bosdyck v. Rochester Folding Box Co., 245 N.Y. App. Div. 880, 282 N.Y. Supp. 146 (1935); Industrial Commission of Ohio v. Lathrop, 52 Ohio App. 55, 2 N.E. (2d) 828 (1935).

90. Mediastinal emphysema: See f.n. 78, supra.

with the pain of coronary thrombosis.[91] On auscultation[92] the typical crackling sounds, drowning out the heart sounds, are heard. The treatment is bed-rest, the disability lasting for a few weeks. The amount of strain necessary to cause this disorder cannot be stated, since it is not always the consequence of strain, occurring in many instances while the patient is at rest or asleep.

Hemoptysis. Hemoptysis, i.e., hemorrhage from the lung, may occur during straining in patients with tuberculosis cavitation or bronchiectasis. During severe muscular strain the glottis[93] is usually closed, preventing egress of air from the lungs, while the abdominal musculature is tightened, pressing the diaphragm upward and compressing the lungs. This compression is, however, resisted by the air trapped in the lungs and a high pressure may be built up, which may rupture a cavity lined by inelastic scar tissue. A blood vessel in the wall of such a cavity may be torn and hemorrhage may result. The treatment of hemoptysis is bed-rest for several weeks; occasionally, the loss of blood is so severe as to make transfusion necessary. Here again the amount of strain necessary to produce the condition cannot be stated, since hemoptysis not infrequently occurs spontaneously, even while the patient with tuberculosis or bronchiectasis is at rest of asleep.

Activation of Tuberculosis by Strain. Activation or exacerbation of tuberculosis consequent to a strain has been the subject of contention for years. It is theoretically possible that, because of changes in pressure within the lung associated with straining while the breath is held, rupture of a tuberculous cavity may occur with dissemination of the disease.

Problems of Identification in Trauma Due to Strain. The establishment of causal relationships between a strain

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91. Hamman, L.: Spontaneous Mediastinal Emphysema, Bull. Johns Hopkins Hosp. 64: 1, 1939. See, in this Symposium series, White, Paul Dudley and Hubert Winston Smith, op. cit. supra., f.n. 30.

92. Auscultation: The act of listening for sounds within the body, chiefly for ascertaining the condition of the lungs, heart, pleura, and other organs, and for the detection of pregnancy. As a rule, auscultation is performed by using a stethoscope applied to the appropriate area of the body. The stethoscope makes the sound more audible to the examining physician.

93. Glottis: The aperture, or chink, between the vocal cords, together with all that part of the larynx which is concerned in voice production.

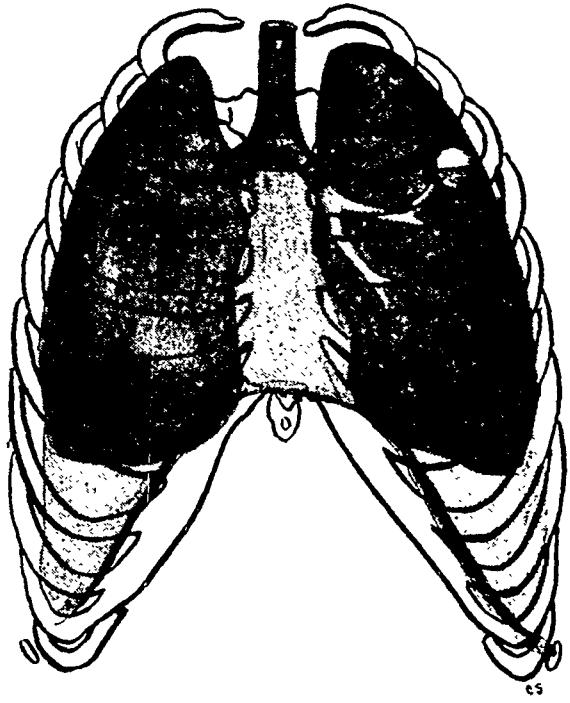

Fig. 7. Tuberculous cavity with fluid level.

and the occurrence of the disorders discussed in this section can never be considered as absolutely conclusive, since all of them may occur spontaneously. If pneumothorax, mediastinal emphysema or hemoptysis occurs during an unusually severe straining activity, such as heavy lifting or pushing, it is reasonable to conclude that a relation between the strain and the pulmonary disorder exists. Care must be taken to distinguish this type of physical strain from emotional or mental strain and from the strain of prolonged work at a task which is not straining in the sense here used. Evidence offered by reliable witnesses as to the development of the dramatic symptoms here described in association with an unusual strain, together with competent medical testimony based on examinations, including x-ray studies, made shortly after the accident, combine to form a very strong case in support of the concept that the disorder is related to the strain. The occurrence of one of these disorders some time after the strain, on the other hand, makes it very unlikely that the strain caused it.

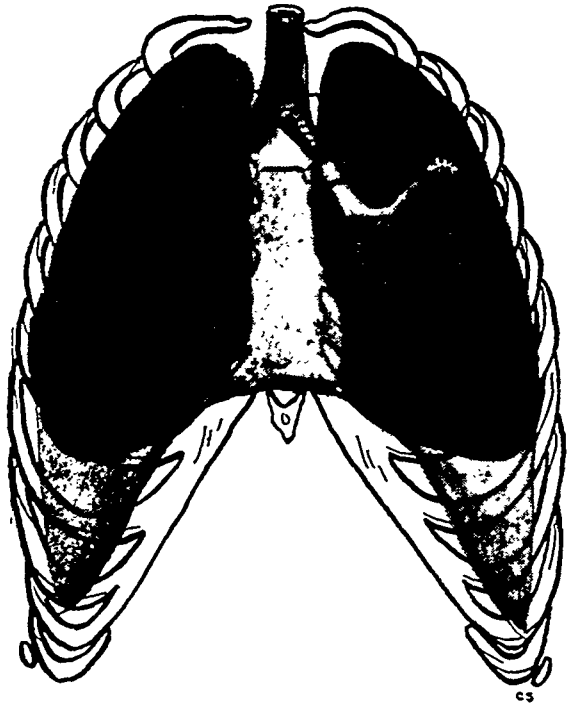

Fig. 8. Rupture of tuberculous cavity into brouchus during strain with discharge of infected material.

Evaluation as to the role of a strain in the exacerbation or activation of a tuberculous process is even more difficult than in the case of a blow or fall. The latter are likely to be witnessed by others, to be recorded and to be easily identifiable as to day and hour of occurrence. A strain, on the other hand, is usually not recognized as such by onlookers or in many cases by the patient himself; the latter may some time after the exacerbation or activation of his tuberculous process remember having experienced a strain shortly before. The value of such testimony from both the medical and legal point of view is small. No general rule can be formulated in regard to the criteria to be used in assessing the part played by a strain in activation or exacerbation of tuberculous processes. The time relation between the alleged strain and the development of signs of tuberculosis is important: too short or too long an elapsed period between the two makes a causal relation unlikely.

LESIONS OF THE LUNGS SECONDARY TO TRAUMA ELSEWHERE IN THE BODY

Pneumonia. Pulmonary complications may follow trauma to other parts of the body. The development of pneumonia following aspiration into the lungs of gastric contents in unconscious individuals has already been discussed.[94] In addition any individual who is made to remain in bed for long periods of time, i.e., weeks, may develop pneumonia spontaneously. This is especially likely to occur if the individual is elderly, greatly debilitated or a cardiac (heart) patient, and more particularly if he is immobilized by a large cast or some mechanical appliance used in the treatment of fractures. The pneumonia becomes manifest when fever, chest pain, cough and possibly dyspnea (breathlessness) are detected, together with the characteristic physical and x-ray findings in the lungs on examination. The development of pneumonia complicates and prolongs the course of the primary illness, may result fatally and may be followed by the sequelae already discussed in relation to other forms of pulmonary infection, i.e., bronchitis, bronchiectasis, fibrosis and emphysema.[95]

Embolization. Another form of pulmonary lesion occurring after trauma (injury) is embolization, i.e., the plugging by a blood clot or other material of one or more arteries leading to the lung, and infarction, i.e., the death of those portions of the lung fed by such arteries. If embolization is extensive, death of the patient may occur due to blockage of the circulation before infarction can be detected. The emboli which give rise to the disorder may consist of a variety of different substances and may originate in different ways. The most common cause of pulmonary embolization is the formation of thrombi, i.e., clots, in the veins which ultimately drain into the right auricle and ventricle of the heart and then pass with the blood through the pul-

94. See discussion, supra.

95. Editor: For cases involving pneumonia allegedly due to trauma, usually to some other part of the body, see: Meyer v. Krauss Laundry and Dry Cleaners 98 Ind. App. 388, 189 N.E. 431 (1934); Black Mountain Corp. v. Black, 220 Ky. 85, 294 S.W. 830 (1937); Ricci's Case, 294 Mass. 67, 200 N.E. 568 (1936); Anderson v. Fisher Body Corp., 239 Mich. 506, 214 N.W. 938 (1927); Zimmerman v. Goodfellow Lumber Co. (Mo. App.) 56 S.W. (2d) 608 (1933).

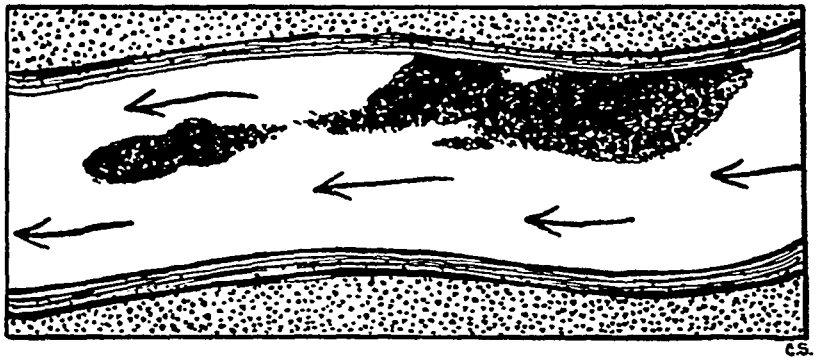

Fig. 9. Mechanism of emholus formation: a part of a blood clot (thromhus) within a vessel breaks off and is carried in the blood stream to a new site, as, for instance the lungs, where it lodges in and blocks a vessel, reducing or cutting off the blood supply to tissues served by that vessel.

monary artery into the lungs. Following infected abortions, the veins in the pelvis become thrombosed and the breaking off of portions of these clots may give rise to embolization; septic abortion will not be discussed here since an extensive body of legal and medical writing on this subject already exists. The veins in the legs may become thrombosed following relatively simple abdominal surgery even in patients in good condition, while patients who have to remain in bed for long periods of time, especially if debilitated, may develop the condition without having been operated upon. This condition, thrombophlebitis of the leg veins, may make itself manifest by pain and/or tenderness in the calves or may never be overt, suggesting its presence only by embolization or by its accidental finding at autopsy. The manifestations of pulmonary embolization are varied, including sudden death, collapse with the picture of shock, sudden dyspnea and/or cyanosis, sudden onset of fever, hemoptysis and chest pain. If infarction occurs, the chest pain may be of the pleuritic type, i.e., made worse by deep breathing and coughing and referred to the shoulder. The presence of infarction can be detected by the typical signs on physical and x-ray examination, and the electrocardiogram may be helpful.[96] Hydro-

96. The electrocardiogram is a device based upon the string galvanometer which enables the action current generated by the functioning heart to be recorded on a moving tape. See Smith, Hubert W. and J. E. F. Riseman: Applied Use of the Electrocardiogram in Legal Proceedings, 15 Rocky Mt. L. Rev. 251, 1943; Riseman, J.E.F. and Hubert W. Smith: Some Legal Aspects of Heart Disease and the Electrocardiogram, Ann. Int. Med. 19: 81, 1943.

thorax or hemothorax may occur. Pulmonary embolization may be fatal, and if not, it may prolong and complicate the patient's primary illness. Since it may occur after even minor abdominal surgery, its occurrence may create legal problems. Thus, a patient operated on for hernia regarded as occupational in origin may die of pulmonary embolization or at least have his hospital stay greatly prolonged.[97] The occurrence of thrombophlebitis in the legs giving rise to pulmonary embolization may require that he also be operated on for ligation of the femoral veins. Pulmonary embolization is not commonly followed by sequelae; in very rare instances enough of the circulation through the lungs may be permanently blocked to cause increased work of the heart in forcing the blood through and the heart may fail (chronic cor pulmonale).[98]

Another, much less common cause of pulmonary embolization is phlebitis which follows intravenous therapy of various sorts. Thus, varicose veins are commonly treated by injecting a solution into them which causes formation of clots, closing off the veins; in rare instances such a clot may break off and be carried to theluns. Phlebitis may occasionally develop in the arm veins following injection of solutions of various sorts, particularly those containing high concentrations of glucose, i.e., 25 to 50 percent. Very rarely pulmonary embolization may follow.

Fat Embolism. Another uncommon type of embolization is that due to free fat in the blood reaching the lungs. Fat in the cells in adipose tissue in various parts of the body, including the bone marrow, may be broken down by crushing trauma, including fracture of a bone, and the finely divided fat forced into the veins where the particles agglutinate. When this mass of fat is carried to the lungs, it may block off many of the small vessels and even the larger ones, causing shock or sudden death. The diagnosis is made with

97. Editor: If an original injury is compensable, the untoward effects of medical treatment are also compensable. The courts extend this doctrine to include even those injuries which are due to medical malpractice, provided the plaintiff or claimant selected the attending physician without notice of his incompetency.

98. For medicolegal cases involving post-traumatic embolization, see: Black Mountain Corp. v. Black, 220 Ky. 85, 294 S.W. 820 (1927); Geagan's Case, 301 Mass. 319, 17 N.E. (2d) 172 (1938); Consolidated Underwriters v. Langley, 141 Texas 78, 170 S.W. (2d) 463 (1943).

certainty only at autopsy. An apparently uncomplicated broken leg or a simple crushing injury to a fatty part of the body may therefore result fatally. In rare instances fat embolism may occur as a consequence of damage to the liver caused by a commercial solvent, carbon tetrachloride.[99]

Air Embolism. Air embolism may occur during trauma or without trauma in situations where atmospheric pressure is rapidly lowered, i.e., in rapid ascent in fighter planes or in caisson disease;[100] the embolic manifestations usually involve the brain and spinal cord. Following trauma (injury) to or operation on areas containing many large veins, air may, however, enter and be carried in the form of small bubbles to the lungs, blocking off small vessels and causing shock or sudden death. The condition is diagnosed with certainty only at autopsy.

A recently described type of embolization is that of meconium[101] through the placenta[102] into the lung, causing shock and death. It is diagnosed at autopsy. Its relation to trauma has not been established, although in one case it occurred after an operation on the pregnant uterus.[103]

Problems of Identification in Lesions of the Lungs Following Trauma Elsewhere. The occurrence of pneumonia or of pulmonary embolism due to phlebitis is easily recognized;

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99. MacMahon, H. E., and Weiss, S.: Carbon Tetrachloride Poisoning with Macroscopic Fat in the Pulmonary Artery, Am. J. Path. 5: 623, 1929.

100. Caisson disease: A condition which occurs when individuals are exposed to a sudden lowering of the atmospheric pressure, as in workers coming from high pressure caissons into the ordinary atmosphere. The sudden change in pressure causes the release of gases dissolved in the blood in the form of bubbles everywhere throughout the body. For medicolegal cases involving caisson disease, see: Knock v. Industrial Acc. Comm. of Calif., 200 Cal. 456, 253 Pac. 712 (1927); Quail v. Ind. Acc. Comm., 138 Cal. App. 412, 32 P(2d) 402 (1934); Maryland Casualty Co. v. Gerlaske, 68 F (2d) 497 (1934); Taylor v. List and Weatherly Const. Co., (La. App.), 146 So. 353 (1933); Williams v. Mo. Bridge and Iron Co., 212 Mich. 150, 180 N.W. 357 (1920); Beaty v. Foundation Co., 245 Mich 256, 222 N.W. 77 (1928); Dochler Die Castings Co. v. McNeely, 21 Ohio App. 148, 152 N.E. 792 (1926); Missouri Valley Bridge & Iron Co. v. Ballard, 53 Tex. Civ. App. 110, 116 S.W. 93 (1909).

101. Meconium: Fecal matter discharged into the amniotic cavity by the foetus.

102. Placenta: The very vascular organ in the uterus connecting the mother and foetus.

103. Steiner, P. E., and Lushbaugh, C. C.: Maternal Pulmonary Embolism by Amniotic Fluid as a Cause of Obstetric Shock and Unexpected Deaths in Obstetrics, J.A.M.A. 117: 1245 and 1340, 1941.

its relation to the trauma which required the hospitalization in the first place is clear. In the case of embolization, sequelae are rare; *cor pulmonale* may occur rarely and should be readily detected. The sequelae of pneumonia, i.e., bronchitis, bronchiectasis, fibrosis and empyema have already been discussed elsewhere in this chapter. The more uncommon types of embolization, those due to fat, air and meconium, are diagnosed only at autopsy.

PULMONARY DISEASE DEVELOPING AFTER EXPOSURE

Prolonged unusual exposure to cold and wet without adequate protection may be followed by an upper respiratory infection[104] and may occasionally, especially in the elderly, debilitated, unconscious or intoxicated, result in bronchitis or pneumonia. There is usually a period of one to a few days between the start of the exposure and the development of bronchitis or pneumonia, so that a short period of exposure might be followed by a latent period of a day or so, during which only a mild upper respiratory infection was manifest, before the onset of the more serious complications. After a longer period of exposure, however, the bronchitis or pneumonia might be found fully established when the individual is brought in. Pneumonia of this type may be followed by the usual complications and sequelae, as outlined previously in this discussion, including exacerbation of tuberculosis.[105]

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104. Editor: In a number of cases, student nurses, interns or physicians have contracted tuberculosis or other diseases presumably by inhaling bacteria while in attendance on sick patients and the interesting problem has arisen whether or not the evidence justifies a finding that this was an accidental injury sustained in the course and scope of employment and so compensable under the Workmen's Compensation Law. See, for instance: Ayers v. Hoage, 61 App. D.C. 388, 63 F (2d) 364 (1933); Smith's Case, 307 Mass. 516, 30 N.E. (2d) 536 (1940); Miller v. City of New York, 282 N.Y. 707, 26 N.E. (2d) 821 (1940), reversing 257 App. Div. 1092, 14 N.Y. Supp. (2d) 680 (1939), and 258 App. Div. 820, 15 N.Y.S. (2d) 718 (1939); Vanore v. Mary Immaculate Hospital, 260 App. Div. 820, 22 N.Y.S. (2d) 350 (1940); Milwaukee County v. Industrial Commission, 224 Wis. 302, 272 N.W. 46 (1937).

105. Editor: For medicolegal cases involving pneumonia allegedly due to exposure to cold and/or wet, see the following: Goble v. Clark, 56 F. (2d) 170 (1931); Anderson v. Hoage, 63 App. D.C. 169, 70 F. (2d) 773 (1934); Strauss National Bank & Trust Co. v. Marcus, 274 Ill. App. 597 (1934); Platisa v. Inland Steel Co. 100 Ind. App. 278, 195 N.E. 294 (1935); Cincinnati Times Star

Problems of Identification in Pulmonary Disease Developing After Exposure. The presence of bronchitis or pneumonia is detected with relative ease and with a high degree of accuracy. The cause of such an infection is not nearly so readily ascertained. The existence of a latent period of upper respiratory infection in most cases before the onset of bronchitis or pneumonia is helpful in evaluating causal relationships. If a brief period of exposure were followed within an hour or two by full-blown pneumonia, the valid conclusion would be that the patient was already on his way to having pneumonia before the exposure began. On the other hand, if the pulmonary complication developed more than a few days after the end of the exposure, it would usually not be considered a consequence of the exposure. Data as to the presence of an epidemic of pulmonary

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Co. v. Clay's Admr., 195 Ky. 465, 243 S.W. 16 (1922) (Illegal employment of minor, a ten year old newsboy, allegedly caused his death from pneumonia. Damages were recovered in a suit brought by an administrator but for the sole benefit of the boy's father. Held, on appeal: recovery of damages for the father's benefit could not be allowed if he knew of or suffered the employment of the minor in the work in question.); Warfield Natural Gas Co. v. Clark's Adm'x., 257 Ky. 724, 79 S.W. (2d) 21 97 A.L.R. 971 (1934) (On February 6 defendant gas company cut off widow's gas for non-payment of bill nine days before it had a right to do so. Widow's son, a newsboy, as a result of staying all night in the icy house, developed a chill and pneumonia from which he died. In an action against the gas company, the mother recovered a judgment of $11,500, for her son's death: held, on appeal: affirmed.); Carter v. Priebe and Sons (Mo. App.), 77 S.W. (2d) 171 (1934); Morgan v. Simplot (Idaho) 155 P. (2d) 917 (1945); Kardos v. American Smelting and Refining Co., 133 N.J.L. 39, 42 Atl. (2d) 271 (1945), affirming 132 N.J.L. 579, 39 A(2d) 509 (1944); Kern v. Premier Coal Saving Device Corp. 246 N.Y. App. Div. 661, 283 N.Y. Supp. 281 (1935); Lacey v. Washburn and Williams Co. 105 Pa. Super. Ct. 43, 160 Atl. 455 (1932); Senlock v. Phila. and Reading Coal and Iron Co., 104 Pa. Super Ct. 156, 158 Atl. 663 (1932); Poklembo v. Hazle Brook Coal Co. 116 Pa. Super. Ct. 532, 176 Atl. 850 (1935); Waleski v. Susquehanna Collieries Co. 108 Pa. Super. Ct. 342, 164 Atl. 355 (1935); Wilkins v. McSorely 119 Pa. Super. Ct. 442, 179 Atl. 759 (1935); Mussman v. Steele, 126 Neb. 353, 253 N.W. 347 (1934) (X, negligently driving ambulance, overturned it while conveying Y, a person suffering from septicemia, to a hospital. There was evidence offered that Y contracted pneumonia as a result of exposure following the accident. Held: whether X's negligence caused or hastened Y's death was a question for the jury.); Packard v. Industrial Commission, 213 Wis. 1, 250 N.W. 768 (1933). And see, Carson v. Fort Smith Light and Traction Co., 108 Ark. 452, 158 S.W. 129, Ann. Cas. 1915B 92 (1913) (Asthma allegedly caused or aggravated by exposure to cold as result of defendant's wrongfully turning off gas supply to plaintiff's house.).

infections in the community, or of an antecedent cases in the patient's own household, should be secured in attempting to establish the cause of bronchitis or pneumonia in a patient claiming compensation after exposure.

PROBLEMS OF EFFECTS FOLLOWING TRAUMA TO THE LUNGS

All of the conditions listed above as sequelae of trauma are, with the exception of tuberculosis, incurable. Their symptoms may be ameliorated and their progression retarded or stopped by adequate medical treatment, but the damage done remains. Accordingly, problems of disability and of compensation are important.

Disability Resulting From Tuberculosis. In the case of tuberculosis, the patient may require sanatorium care or its equivalent for a period of six or eight months to a few years before he is discharged as cured or arrested; during this time he must be regarded as completely disabled. Following this, if a good deal of scarring has occurred in his lungs as the tuberculosis healed, he may suffer varying degrees of disability, as will be discussed below in relation to fibrosis in general. If such scarring is minimal (the more common occurrence), he may appear to be entirely well, the only abnormal finding being a small patch of fibrosis in the lungs on x-ray examination. However, the patient will thereafter, probably for the rest of his life, have to follow the well known regime for cured tuberculars in regard to food, rest, hygienic conditions, etc.. The imposition of this regime may constitute partial disability in the case of a busy sales-manager, for instance, whose business activities normally involved long hours, great tension, irregular eating and sleeping, ingestion of large amounts of alcohol, etc., and who is therefore forced to take another job with a considerably smaller income after recovery from tuberculosis. On the other hand, if the patient had previously held a quiet or sedentary job as watchman or time-keeper, for instance, he could readily resume his previous work without hazard to himself and therefore need not be considered, from the occupational point of view at least, as having any disability.

Disability Resulting from Fibrosis, Emphysema, Asthma. Individual Variations. In the case of pulmonary fibrosis[106]

106. See f.n. 31, supra.

and also pulmonary emphysema and asthma, neither physical examination nor x-ray studies will yield data which in themselves permit an accurate estimate of disability. Measurement of the vital capacity, i.e., the expansibility of the lungs,[107] is highly regarded by some authors in this respect, but has proved disappointing in the hands of others, including the present author. An important element in the estimation of disability is, therefore, the symptomatology of the patient himself. Reliance entirely on the history of his symptoms can, however, be misleading. Neurosis[108] itself may result in dyspnea, (breathlessness) even in the presence of completely normal lungs. Neurosis or malingering may result in exaggeration of symptoms. Even when neither is present, a large variation in the severity of symptoms will occur among a group of patients with the same amount of pulmonary disease, depending on emotional makeup, upbringing, etc.. The stable, stolid individual, or one who has been brought up to minimize his complaints, should not be penalized for not complaining vociferously. Accordingly, interpretation of the patient's history must be done in the light of what is known concerning his personality. Evaluation of the symptoms may be difficult because of seasonal variations: patients with extensive diffuse pulmonary disease may telerate hot weather poorly, while asthmatics may be made worse by cold raw weather which may also increase the frequency and severity of flareups in a patient with chronic bronchitis. The symptom of dyspnea itself may be difficult to evaluate in relation to attempts to return to a former occupation. Since dyspnea is aggravated by exertion, it might be almost impossible to estimate accurately the degree of disability in a man with severe pulmonary disease who was more comfortable sitting quietly for hours at a time at his work as a watchman than he might be while up and about at home. In some instances, the patient's complaints may be those of gastrointestinal disease, or of severe mental sluggishness, as was pointed out previously. In the latter case the differentiation of sluggishness due to anoxia from that due to neurosis is important. Evidences

107. Performed by having the patient inhale maximally from the outside air and then exhale maximally into a measuring device. Normal persons should have volumes of over four liters.

108. Neurosis: See f.n. 41, supra.

of deficient oxygenation of the blood are helpful in this differentiation; these evidences consist in cyanosis (bluish coloration of the skin) visible on examination and a decrease in oxygen saturation of the arterial blood and an increase in the number of circulating red blood cells, detectable in the laboratory.

Reactivation or Exaberbation of Antecedent Disease. The problem of reactivation or exacerbation of antecedent disease is also important. Medical testimony as to the degree of disability before the trauma (injury) must serve as the basis for any evaluation of disability after trauma. Even so, great difficulty may be encountered. For instance, an individual with a given degree of pulmonary disease might feel entirely well and be able to live a normal life and yet, after experiencing minimal additional damage to his lungs during gassing, might then have enough pulmonary damage to push him over the line which separates the asymptomatic from the partly disabled patient. This is an extreme case, but the problem might very well arise.

It is apparent that no generalization can be made concerning the occurrence of disability or its degree after trauma. Accuracy of evaluation in a given case can be approached only by a cooperative study of the circumstances on the part of honest and competent medical and legal authorities.