

the root of the lung. A direct ending of the nerves in the immediate neighborhood of the epithelium lining the air sacs was not found. The mucous membrane and muscular tissue of the bronchi are innervated from the nerves following the bronchial artery, and also from the very considerable plexus of nerve fibres found in fibrous layers about the bronchi. In the muscle the nerves end in small, rounded bulbs upon, and not within, the muscle cells. In the larger bronchi nerves could not be followed through the fibrous layer immediately under the epithelium, but in the smaller bronchi the nerves could be followed to a well-marked interepithelial arborization. The nerve supply of the pulmonary blood-vessels is not so large as that to the bronchial.

William Snow Miller.

## BIBLIOGRAPHY.

- <sup>1</sup> Addison, W.: Observations on the Anatomy of the Lungs. Medico-Chir. Trans., 1840-41. (a) On the Ultimate Distribution of the Air Passages and the Formation of the Air Cells of the Lungs. Phil. Trans., 1842.
- <sup>2</sup> Adriani, A.: De subtiliori pulmonum structura. Trajecti ad Rhenum, 1847.
- <sup>3</sup> Aebly, C.: Der Bronchialbaum der Säugethiere und des Menschen. Leipzig, 1880.
- <sup>4</sup> Aigner, A.: Ueber Trugbilder von Poren in den Wänden normaler Lungenalveolen. Sitz. d. kais. Akad. d. Wiss. in Wien, Math.-nat. Cl., Bd. cviii., 1889.
- <sup>5</sup> Aristoteles: Hist. Animal., lib. 1., cap. 16, 17.
- <sup>6</sup> Arnold, J.: Zur Histologie der Lunge. Virchow's Archiv, Bd. 28, 1863.
- <sup>7</sup> Bartholin, T.: De pulmonum substantia et motu diatribe. Hafnia, 1693.
- <sup>8</sup> Bazin: Sur la Structure intime du Poupon. Comptes rendus de l'Académie Royale des Sciences—Mémoire du Dr. Bazin, t. ii. et ix.
- <sup>9</sup> Berkley, H. J.: The Intrinsic Pulmonary Nerves in Mammalia. Johns Hopkins Hospital Reports, vol. iv., 1895.
- <sup>10</sup> Bezzola, D.: Beiträge zur Histologie der fibrinösen Pneumonie. Virchow's Archiv, Bd. 136, 1894.
- <sup>11</sup> Bianchi e Cocchi: Sul rapporto dell'albero bronchiale colla parete posteriore del torace. Archives ital. de Biologie, t. xvi., 1891.
- <sup>12</sup> Birch-Hirschfeld, F. V.: Ueber den Sitz und die Entwicklung der primären Lungentuberkulose. Deutsches Archiv f. klin. Med., Bd. lxxiv.
- <sup>13</sup> B. N. A.: Die anatomische Nomenclatur, von W. His. Archiv f. Anat. u. Phys., Anat. Abthlg., 1900.
- <sup>14</sup> Braune u. Stahel: Ueber das Verhältniss der Lungen als zu ventilirender Lufträume, zu den Bronchien, als Luft zuleitenden Röhren. Archiv f. Anat. u. Phys., Anat. Abthlg., 1886.
- <sup>15</sup> Celsus, A. C.: De medicina, lib. iv., cap. 1.
- <sup>16</sup> Councilman, W. T.: The Lobule of the Lung and its Relations to the Lymphatics. Journ. Boston Soc. of Med. Sci., vol. iv., 1900.
- <sup>17</sup> Cruikshank, W.: The Anatomy of the Absorbing Vessels of the Human Body, London, 1790.
- <sup>18</sup> DeLafeld, F.: Studies in Path. Anat., vol. i., New York, 1882.
- <sup>19</sup> von Ebner, V.: Kölliker's Handbuch der Gewebelehre des Menschen, 6. Aufl., Bd. iii., Leipzig, 1896.
- <sup>20</sup> Elenz, E.: Ueber das Lungenepithel. Würzburger naturw. Zeitschr., Bd. iv., 1893.
- <sup>21</sup> Ewart, W.: The Bronchi and Pulmonary Blood-Vessels, London, 1889.
- <sup>22</sup> Galenus, C.: De usu partium Corp. hum., lib. vi., cap. 3.
- <sup>23</sup> Hansemann, D.: Ueber die Poren der normalen Lungenalveolen. Sitz. der preuss. Akad. der Wissensch., 1895. (a) Ueber V. von Ebner's Zweifel an der Existenz normaler Poren zwischen den Lungenalveolen. Archiv f. mik. Anat., 1900.
- <sup>24</sup> d'Hardiviller, A. D.: Développement et Homologation des Bronches Principales chez les Mammifères (Lapin), Nancy, 1897. (a) Origine des bronches lobaires du mouton. C. R. Soc. Biol., Paris, t. iv., 1897.
- <sup>25</sup> Harvey, W.: Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus, Frankfurt, 1628.
- <sup>26</sup> Hauser, G.: Ueber die Entstehung des fibrinösen Infiltrates bei der croupösen Pneumonie. Beitr. z. path. Anat. u. z. allg. Path., Bd. xv., 1894.
- <sup>27</sup> Heller und von Schrötter: Die Carina Tracheæ. Denksch. d. math.-naturw. Cl. d. kais. Akad. d. Wissensch., Bd. lxxiv., 1897.
- <sup>28</sup> Helvetius, J. C. A.: Observations sur le poupon de l'homme. Mém. de l'Acad. royale des Sci., 1716.
- <sup>29</sup> Henle, J.: Handbuch der systemat. Anat. des Menschen, Braunschweig, 1871.
- <sup>30</sup> Herbig, M.: Beiträge zur Histogenese der Lungen-Induration. Virchow's Archiv, Bd. 136, 1894.
- <sup>31</sup> Hippocrates: Opera Omnia, Geneva, 1657.
- <sup>32</sup> His, W.: Zur Bildungsgeschichte der Lungen beim menschlichen Embryo. Archiv f. Anat. u. Phys., 1887, Anat. Abthlg.
- <sup>33</sup> Huntington, G. S.: The Eparterial Bronchial System of the Mammalia. Ann. N. Y. Acad. Sci., vol. ii., 1898.
- <sup>34</sup> Huschke, E.: Sömmering's Lehre von den Eingeweiden, Leipzig, 1844.
- <sup>35</sup> Justesen, P. T.: Zur Entwicklung und Verzweigung des Bronchialbaumes des Säugethierlunge. Archiv f. mik. Anat., 1900.
- <sup>36</sup> Klein, E.: The Anatomy of the Lymphatic System, London, 1875, vol. ii. The Lung.
- <sup>37</sup> Kobler u. von Hovorka: Ueber den Neigungswinkel der Stammbronchus. Sitzber. Akad. Wiss. Wien, Math.-naturw. Kl., Bd. 102, 1893.
- <sup>38</sup> Kohn, H. N.: Zur Histologie der indurirenden fibrinösen Pneumonie. Münch. med. Wochenschr., 1893.
- <sup>39</sup> Kölliker, A.: Mikroskopische Anatomie oder Gewebelehre des Menschen, Leipzig, 1850-54. (a) Zur Kenntniss des Baues der Lunge des Menschen. Verhandl. d. phys.-med. Gesell. zu Würzburg, 1881.
- <sup>40</sup> Küttner, C.: Beitrag zur Kenntniss der Kreislaufverhältnisse der Säugethierlunge. Virchow's Archiv, Bd. 63, 1878. (a) Die Abschleudung des Indigenschwefelsauren Natron in den Geweben der Lunge. Medic. Centralbl., 1875.
- <sup>41</sup> Laguesse et d'Hardiviller: Sur la topographie du lobule pulmonaire. Bibliog. Anatomique, 1898.
- <sup>42</sup> Lereboullet, A.: Anatomie comparée de l'appareil respiratoire, Strasbourg, 1838.
- <sup>43</sup> Luschka, H.: Die Anatomie des Menschen, Tübingen, 1863.
- <sup>44</sup> Magendie, F.: Mémoires sur la structure du poupon de l'homme. Journ. de Physiol. exp., 1821. (a) Leçons sur les phénomènes physiques de la vie, Paris, 1836.
- <sup>45</sup> Mall, F. P.: Das reticulirte Gewebe und seine Beziehungen zu den Bindegewebsfortsätzen. Abhandl. Math.-phys. Klasse d. k. Säch. Gesell. d. Wiss., Bd. xvii.
- <sup>46</sup> Malpighi, M.: Opera omnia, Lugd. Batav., 1697. De pulmonibus.
- <sup>47</sup> Mandl, L.: Anatomie microscopique, Paris, 1838-57.
- <sup>48</sup> Mascagni, P.: Vasorum lymphaticorum corporis humani historia et ichnographia, Senis, 1787.
- <sup>49</sup> Miller, W. S.: The Lobule of the Lung and its Blood-Vessels, Anatom. Anz., 1892. (a) The Structure of the Lung. Journ. Morph., 1893. (b) The Anatomy of the Lungs, first edition of this work, vol. ix. (c) The Lymphatics of the Lung. Anatom. Anz., 1896. (d) Das Lungenlappchen, seine Blut- und Lymphgefässe. Archiv f. Anat. u. Phys., 1900, Anat. Abthlg.
- <sup>50</sup> Milne-Edwards, H.: Leçons sur la Physiologie et l'Anatomie comparée, t. ii., Paris, 1857.
- <sup>51</sup> Moleschott, J.: De Malpighianis pulmonum vesiculis, Heidelberg, 1845. (a) Ueber die letzten Endigungen der feinsten Bronchien. Hölland. Beiträge z. d. Anat. u. Phys. Wiss., t. i., 1846.
- <sup>52</sup> Narsath, A.: Vergleichende Anatomie des Bronchialbaumes. Verhandl. d. Anatom. Gesellsch., 1892. (a) Die Entwicklung der Lunge von Echidna aculeata. Semon's Zoolog. Forschungsreisen, ii., 1896. (b) Der Bronchialbaum der Säugethiere und des Menschen, Stuttgart, 1901.
- <sup>53</sup> Pappenheim: Sur les Lymphatiques des pmons et du diaphragme. Compt. rend., 1860, 30 Avr.
- <sup>54</sup> Rainey, G.: On the Minute Structure of the Lungs. Medico-Chir. Trans., 1845.
- <sup>55</sup> Reisseisen, F. D.: Ueber den Bau der Lungen, Berlin, 1822.
- <sup>56</sup> Remak, R.: Neurologische Erläuterungen. Archiv f. Anat., Physiol. u. wissensch. Medicin, 1844.
- <sup>57</sup> Retzius, G.: Zur Kenntniss der Nervenendigungen in den Lungen. Biol. Unters., Bd. v., N. F., S. 41, 1893.
- <sup>58</sup> Ribbert, H.: Zur Anatomie der Lungenentzündungen. Fortsch. d. Med., Bd. xii., 1894.
- <sup>59</sup> Roosevelt, J. W.: The Anatomy of the Thorax and Lungs. Medical Record, 1890. (a) The Anatomy of the Lungs as shown by Corrosion. New York Med. Jour., 1891.
- <sup>60</sup> Rossignol, M.: Recherches sur la structure intime du poupon de l'homme, Bruxelles, 1846.
- <sup>61</sup> Sappey, P. C.: Anatomie, physiologie, pathologie des vaisseaux lymphatiques, Paris, 1874.
- <sup>62</sup> Schrötter: Beitrag zur Aetiologie der Lungengangrän, nebst Bemerkungen zur Anatomie der grossen Bronchien. Wien. klin. Wochenschr., 1890.
- <sup>63</sup> Schulze, F. E.: Die Lungen. Stricker's Lehre von den Geweben, Leipzig, 1871.
- <sup>64</sup> Sikorsky, J.: Ueber die Lymphgefässe der Lungen. Centralbl. f. medicin. Wissensch., 1870. (a) The Lymphatics of the Lung (Russian). Kieff, 1872.
- <sup>65</sup> Smith, A. H.: Lobar Pneumonia. Twentieth Century Practice of Medicine, New York, 1890.
- <sup>66</sup> Sömmering, S. Th.: Vom Baue des menschlichen Körpers, Frankfurt, 1791. (a) Ueber die Structur, die Verrichtung, und den Gebrauch der Lungen, Berlin, 1808.
- <sup>67</sup> Teichmann, L.: Das Saugadersystem, Leipzig, 1861. (a) Ueber Lungenlymphgefässe. Anzeiger d. Akad. d. Wissensch. in Krakau, 1896.
- <sup>68</sup> Vesalius, A.: De corporis humani fabrica, Basel, 1555.
- <sup>69</sup> Waters, A. T. H.: The Anatomy of the Human Lung, London, 1890.
- <sup>70</sup> Williams, T.: Respiration. Cyclopaedia of Anat. and Phys., vol. v., London, 1859.
- <sup>71</sup> Willis, T.: Opera omnia, Geneva, 1676.
- <sup>72</sup> von Wittich, W.: Ueber die Beziehungen der Lungenalveolen zum Lymphsystem. Mitth. aus d. Königsberger phys. Laborat., 1878.
- <sup>73</sup> Wygodzoff: Die Lymphwege der Lunge. Wiener medicin. Jahrbücher, Bd. xl., 1836.
- <sup>74</sup> Zuckerkandl, E.: Ueber die Anastomosen der Venæ pulmonales mit den Bronchialvenen und mit dem mediastinalen Venennetze. Sitzungsber. der k. Akad. d. Wissensch., Math.-naturw. Classe, 1881. (a) Ueber die Verbindung zwischen den arteriellen Gefässen der menschlichen Lunge. Same publication, 1883.

**LUNGS, DISEASES OF: ABSCESS.**—Suppurative processes in the lungs are more common than is ordinarily supposed. The experienced clinician always keeps this fact in mind when dealing with acute inflammatory conditions, especially during convalescence.

**ETIOLOGY.**—An abscess always presupposes the presence of pus organisms.

1. Abscess occurs secondarily to diseases of the lungs proper, such as lobar and lobular pneumonia. It is not a common termination of lobar pneumonia; Osler found four cases in one hundred autopsies. On the other hand, it is extremely common in the aspiration and deglutition

forms of lobular pneumonia, such as occur following operations, under general anaesthesia, upon the mouth, nose, throat, and neck; also in wounds of the neck, suppurative processes of the nose, larynx, and even ear (Volkmann). Infective particles reach the bronchi, lodge there, and set up an inflammatory process, with abscess formation. Cancer of the oesophagus may penetrate a bronchus, with similar results.

Pulmonary tuberculosis at some time in its course usually becomes a mixed infection, with the development of abscess cavities, and symptoms of a septic process.

2. Metastatic abscesses develop in the course of a pyæmia, the infected material being distributed throughout the lungs by emboli. Similar abscesses are usually present in the various organs of the body, such as the brain, kidneys, spleen, etc.

3. Pulmonary abscess may develop as a result of the extension of the suppurative process from neighboring organs, or as an event secondary to the perforation of the lung by abscesses from without. Empyemas occasionally perforate the lung tissue and find a vent through the bronchi. Subdiaphragmatic and liver abscesses, associated with amebic dysentery or echinococcus disease, may perforate the diaphragm and lung. The bronchi may simply furnish drainage for these abscesses. In the passage of infective material there may or may not be an infection of the lung with abscess development.

**PATHOLOGY.**—Abscesses occurring in the later stages of lobar pneumonia are not large, but are likely to fuse, and thus involve a considerable portion of one lobe. Tuberculous abscesses are situated most often near the summit, while other varieties are near the base.

Metastatic abscesses are usually very numerous, and, though scattered throughout the lungs, are frequently situated underneath the pleura; they are small, about the size of a pea.

The septic emboli may be associated with suppurative middle-ear disease, diptheritic endometritis following childbirth, abortion, etc., or even with operations upon hemorrhoids.

**DIAGNOSIS.**—A consideration of the history of previous diseases throws a great deal of light upon the case. The symptomatology is also important. One is likely to secure a history of a septic temperature curve, with chills and fever, occurring several times during the day. The presence of leucocytosis and the character of the expectoration may be of great assistance. The sputum is purulent, offensive, and at times contains large quantities of elastic tissue. The sudden perforation of an empyema or a subdiaphragmatic abscess into the bronchi may simulate an abscess of the lung.

The physical signs are very unreliable. The lungs, though riddled with metastatic abscesses, often give no definite signs of the condition. There may be diminished expansion, impaired resonance, weak breath sounds, tactile fremitus, and voice sounds. If the abscess is large and parietal, as in some cases of tuberculosis, one might get the signs of a cavity.

**PROGNOSIS.**—Recovery occasionally occurs in the case of an abscess which has developed after pneumonia. The metastatic abscesses are of course almost invariably fatal. Single abscesses which discharge and are well drained may heal, with recovery of the patient.

**TREATMENT.**—Medical treatment is useless. If possible, such cases should be treated surgically with incision and drainage. James Rae Arneill.

**LUNGS, DISEASES OF: AFFECTIONS OF THE BRONCHIAL GLANDS.**—(Syn.: Fr., *Adénopathie Trachéo-bronchique*; Ger., *Krankheiten der Bronchialdrüsen*.)

**HISTORY.**—It is to M. Noël Guéneau de Mussy that we are specially indebted for our knowledge of the diseases of the bronchial glands. M. Baréty has supplemented his labors in a monograph, "L'Adénopathie Trachéo-bronchique," Tauchon (Paris, 1867), and others have described some of the changes in these glands which accompany phthisis in the adult, while MM. Rillet and Bar-

thez, in their work, "Traité des Maladies des Enfants" (Paris, 1861), and Dr. West, in his Lectures on Diseases of Infancy and Childhood, have given full descriptions of the same changes in children under the head of bronchial phthisis.

Tumors and enlargements are more especially considered in connection with intrathoracic tumors. (See article on *Mediastinum*, etc.)

**CLASSIFICATION.**—Upon a pathological basis, diseases of the bronchial glands are classified under the conditions affecting lymphatic glands generally. They are subject to the following changes:

1. Inflammations: (a) acute; (b) chronic; (c) specific.
2. Morbid deposits and growths: (a) pigmentation; (b) cancer; (c) tubercle; (d) syphilitic growths (tertiary); (e) albuminoid disease.

3. Hypertrophy and atrophy.

**ANATOMY.**—Since the greater portion of the symptoms arising from disease in the bronchial glands are due to implication of adjacent parts, either through inflammation or from pressure, exact knowledge of the anatomical relations is of the utmost value in determining the significance of any given symptom.

The largest group of glands lies just below the bifurcation of the trachea, between the right and the left bronchus. They are in relation, laterally, with the bronchi; anteriorly, with the pericardium, arch of the aorta, and pulmonary artery; and posteriorly with the aorta, vena azygos, oesophagus, and pulmonary plexus of nerves. Smaller ganglia are situated upon the anterior, posterior, and superior surfaces of the right and left bronchi. Those upon the right are the larger, and are in relation with the arch of the aorta, the brachiocephalic and subclavian arteries, the brachiocephalic and azygos veins, and the pneumogastric and recurrent laryngeal nerves. On the left they are in relation with the extremity of the arch of the aorta, the origin of the left subclavian and common carotid arteries, the subclavian vein, and the pneumogastric nerve with its recurrent branch.

Afferent vessels reach the glands from the lungs, pleura, neck, etc. The blood supply is through the bronchial arteries.

**PATHOLOGY AND MORBID ANATOMY.**—The pathological processes which occur in the bronchial glands are in no respect different from those which take place in other lymphatic glands. The resulting anatomical changes assume special importance through their mechanical effects.

In many instances, when the glandular disease is slight, the anatomical disturbances cause such marked symptoms as to obscure, or divert attention from, the more serious associated conditions.

1. *Acute inflammation* is attended by cellular infiltration, with increase of lymphoid elements and retention of lymph, resulting in enlargement of the glands and softening of their parenchyma. When this process is rapid, or if due to specific poisons, suppurative and necrotic changes may follow. More commonly resolution takes place, and the glands return to their normal size.

2. *Chronic inflammation* is characterized by similar but more gradual cellular and lymphoid changes. In connection with these, fibrous growth is more marked; the glands become greatly enlarged, in some cases permanently, and their capsules are thickened and form adhesions with surrounding tissues. Resolution is seldom complete, and if it is long delayed the glands become contracted and indurated.

When suppuration results, the pus may find its way, by an ulcerative process, to the free surface of a bronchus, into the oesophagus or pericardium, or into the substance of the lung or lumen of a blood-vessel; or the contents of the abscess may undergo caseous, calcareous, or cystic degeneration. Even in the acute form suppuration rarely takes place with sufficient rapidity to allow rupture directly into the connective tissue, and in the chronic forms protective inflammation with adhesions is always present.



3. *The specific inflammatory changes* present no peculiarities in pathological processes.

4. *Pigmentation* (see also *Lungs, Diseases of: Pneumonokoniosis*).—Carbonaceous and other deposits in the bronchial glands seldom cause more than the lightest grades of inflammatory changes, owing to the slight irritating nature of the foreign matter and the slowness with which it is deposited. The glands become more or less enlarged and variously pigmented. In extreme cases they are entirely black, firm, hard, and gritty on section, resembling a lump of coal. The glandular tissue is partially atrophied and absorbed. When suppurative changes supervene, the discharge from the resulting abscess is at first black, and, though gradually becoming lighter, is not entirely free from pigment until the entire gland has been removed by suppuration.

5. *Cancer*.—Cancerous developments in the bronchial glands follow similar disease of the lungs, pleura, or mediastinum, and are of like character. Primary cancer is infrequent.

6. *Tubercle*.—Secondary tuberculosis occurs, to a greater or less degree, in all cases of pulmonary phthisis in the adult. In children, on the contrary, the glandular changes are often the more extensive and important. The process is commonly one of general infiltration, evenly distributed throughout the gland, but may start from several centres or be confined to one extremity of the gland. It is seldom that the deposit presents the form of gray military tubercle. In connection with these changes the glands enlarge, and at first are softer than normal, but, as the process advances and implicates the entire gland, they become firm and resistant, resembling tuberculous pulmonary consolidation. In the second stage the usual softening takes place, and tuberculous glandular abscesses are formed which follow the course of other abscesses described above.

7. *Syphilis*.—Syphilitic deposits are usually tertiary. Gummy deposits may lead to extensive enlargement, with subsequent caseous or suppurative degeneration.

8. *Albuminoid degeneration* is exceedingly rare. When present, the glands are usually enlarged, firm, and tense; occasionally they are atrophied. On section they present the usual waxy, glistening, homogeneous appearance, and give the characteristic reaction with iodine.

*Etiology*.—The lymphatic diathesis, inherited tendencies, and general malnutrition are here, as elsewhere in the body, predisposing causes of glandular disease. Some statistics have seemed to show a slightly greater predisposition among females, and an increasing liability to such disease after puberty.

West and others consider the disease to be very frequent among infants and young children.

Of the exciting causes, acute inflammation of the pulmonary tissue or pleura is the most frequent. Thus, a simple bronchitis, a pneumonia, a pleurisy, or an empyema, etc., may each be followed by an acute or chronic inflammation of the bronchial glands, resulting in resolution, abscess, or caseous degeneration.

So frequently have these glands been found enlarged in cases of whooping-cough that Noël Guéneau de Mussy was led to regard the spasmodic element in the cough as due to pressure upon the pneumogastrics by the enlarged glands.

Other observers (Barlow, *Lancet*, 1879, vol. ii., p. 124), however, have reported cases in which the pneumogastrics were not only pressed upon, but even involved in the inflammatory processes surrounding the gland, without the presence of any cough. Still further, many cases of whooping-cough, in which the spasmodic element was specially marked, exhibited no change in the bronchial glands post mortem. More exact observation also shows but little resemblance in the cough of pertussis to that due to irritation of the recurrent laryngeal nerve.

Both acute and chronic inflammatory changes have been observed in the bronchial glands in connection with most of the infectious diseases, more especially in typhoid fever, measles, scarlet fever, and pyæmia. In these conditions the changes are part of a generally lymphatic in-

flammation, and are seldom of sufficient extent to attract attention during the life of the patient.

Absorption of various forms of dust to which certain classes of workmen are exposed, with the consequent filling and clogging of the glandular passages, may lead to either acute or chronic inflammation, ending in atrophy or suppuration and abscess. Such a result is exceedingly rare, however, if we consider the number of cases of pneumokoniosis in which the glands become partially or completely filled with extraneous matter.

As already indicated in the sections on classification and pathology, the specific causes of cancer, tubercle, syphilis, and amyloid degeneration are exciting causes of disease in these glands.

Finally, many cases of enlargement, induration, or suppuration with secondary changes will be found in which no exciting cause is apparent beyond the lymphatic diathesis. Simple inflammation seldom causes suppuration, such a result following more certainly from septic irritation, as in pyæmia or tubercle.

*Symptoms*.—In the earlier stages, and, indeed, throughout the entire course of the disease, unless the glands form decided tumors, the symptoms will be almost entirely rational. Since they are due solely to pressure, and as the glands involved in different cases will not be the same, nor always enlarge in the same direction, it is evident that the symptoms will vary greatly in their order of development and relative importance in different cases.

1. *Cough* is the most frequent as well as the earliest symptom. This may be due to pressure upon either a bronchial tube or the recurrent laryngeal nerve. In the former case it will resemble the cough of simple bronchitis. When due to pressure on the nerve it will be more harsh and laryngeal in character, and in some cases will have a distinct spasmodic element. When the irritation is severe it may be a persistent dry hacking, with or without paroxysmal exacerbations. More rarely it is deep, hollow, and metallic, or resembles the cough of an animal.

2. *Expectoration* attending the bronchial form of cough is quite constant. At first white and frothy, it gradually becomes muco-purulent when the glandular processes are acute and rapidly extending, or changes to a tenacious, mucous sputum with the more chronic processes. Should a glandular abscess open into the bronchial tubes, it will be evidenced by a more or less free purulent expectoration, mingled, it may be, with cheesy or even calcareous matter. After such an opening has occurred, an intermittent purulent discharge will continue indefinitely, or until the abscess has healed. When cough is due to nerve compression, expectoration is slight or entirely absent. No appreciable modification in the expectoration will be observed when the glandular disease is secondary to other pulmonary lesions.

3. *Hæmoptysis* is present in a small proportion of cases. When due to intense pulmonary congestion, resulting from prolonged paroxysms of coughing or obstruction to the pulmonary veins, it is usually capillary in character, and appears at first either as streaks in the sputa or in moderate amount as clear, bright-red blood, followed later by darker masses and small clots. When due to bronchial ulceration or erosion of a vessel in the wall of a glandular abscess, it is more profuse in character, appears suddenly, and may continue for several days, or even result in death.

4. *Pain* is one of the most frequent symptoms. In character it does not differ from that occurring with other forms of intrathoracic tumors. It has been described as dull and heavy, as a tightness or compression, and in a few cases has been spasmodic.

It is usually associated with some decided tenderness on pressure, and when once present is quite persistent, even when varying greatly in intensity.

It is most frequently located posteriorly between the spine and border of the scapula, opposite the bodies of the fourth, fifth, and, in a few cases, the sixth dorsal vertebra. Less commonly it is felt in front, near the edge of the sternum, or under the clavicle, with occa-

sionally a point of pain and tenderness in the axillary region, causing it to simulate intercostal neuralgia.

5. *Dyspnoea* is often a prominent symptom. One fatal case is reported in which it was the only symptom. Its intensity depends less upon the absolute size of the tumor than upon the direction and nature of the enlargement. A comparatively small tumor or rapid inflammatory exudation may compress a primary bronchus sufficiently to cause most intense dyspnoea.

When due to compression of a bronchial tube, or of the lung substance, the dyspnoea is persistent and unvarying. In a small proportion of cases it appears to depend upon implication of the laryngeal nerve and unilateral paralysis of the larynx. It may be paroxysmal, or even assume the characteristics of spasmodic asthma with decided nocturnal paroxysms, and it is occasionally so severe as to force the patient to assume the erect position. Quain gives the proportion in which this spasmodic element is well marked as one in fifteen.

6. *Dysphagia* is a quite common symptom, and is due simply to compression. It is present in about fifteen per cent. of cases. It comes on slowly, is persistent, and varies only with the changes in the size of the glandular tumor. It is first noticed and most marked as regards solid food, but in one or two cases it was almost confined to liquids.

7. *Change of voice* is present only when the recurrent nerves are implicated. There may be loss in volume and force in connection with the dyspnoea of bronchial obstruction, but distinct changes in character are probably always of nervous origin.

Hoarseness is the earlier and may be the only change. It occasionally passes into complete aphonia. In these cases paralysis of one or both vocal cords can be recognized by the laryngoscope.

8. *Nausea and vomiting* are rare symptoms, due to implication of the pneumogastrics. M. de Mussy considers them more frequent when the left nerve is affected.

9. *Venous Compression*.—Compression of the ascending veins seldom causes any marked symptoms. Anorexia and the general disturbances of digestion have only an indirect relation to venous obstruction.

Compression of the veins coming from the head is more frequent, causing cyanosis, congestion, and œdema of the face and neck, and rarely of the upper extremities. Epistaxis results from the same cause.

*Physical Signs*.—*Inspection* is usually negative. It may show: 1. The œdema, puffiness, etc., just mentioned, of the face. 2. Slight prominence of the upper sternal and infraclavicular regions. This is exceedingly infrequent; it was noticed in none of sixty cases reported by Quain. 3. Slight flattening of the affected side. It is the more frequent change, and is probably induced by bronchial occlusion and partial pulmonary collapse. 4. Diminished motion of the affected side. It may be present alone or in connection with either enlargement or contraction. 5. No change in either size or motion. Most cases will be of this nature.

*Palpation* will show decreased vocal fremitus when bronchial compression has resulted in occlusion of the tubes.

*Percussion*.—Dulness is the most constant physical sign, and will indicate, by the area over which it is present, and by its character, both the size of the glandular enlargement and its nearness to the surface. It is usually best marked behind, between the scapula and spine, extending in extreme cases from the fourth to the sixth, or even seventh, dorsal vertebra. Less frequently it may be obtained in front, over the manubrium sterni, and below the sternal end of the clavicle.

Rarely pulmonary collapse causes partial dulness over a greater or less area. Abscess cavities communicating with the bronchial tubes are seldom, if ever, of sufficient size to affect the percussion note.

A compensatory emphysema may possibly give a vesiculo-tympanic tone over the healthy lung.

*Auscultation*.—The respiratory sounds will be variously

modified by the size of the tumor and its relations to the pulmonary tissue and bronchial tubes.

Weakness or entire absence of vesicular murmur is the more frequent change. It is due principally to bronchial obstruction, but in some cases is caused by direct compression of the pulmonary tissue. In the former case the change may be observed over a considerable area, or even an entire lung, but in the latter it will be more localized.

In an almost equal number of cases the respiratory sounds are loud and harsh, or even distinctly tubular. These changes are found only over the seat of the disease, and depend upon compression and closure of the alveoli and smaller tubes.

A venous hum, heard best at the root of the neck, and more common in children, is usually present when there is decided compression of the descending venous trunks.

Bilateral examination of the chest with the x-ray will often reveal a thoracic tumor and locate it on or about the bronchial tubes.

*Diagnosis*.—It is evident from the foregoing description that, in the earlier stages at least, a positive diagnosis is impossible. In no two cases will the symptoms or their order of development be alike.

They indicate only some form of intrathoracic growth, and may all be present with mediastinal tumors or thoracic aneurism.

Mediastinal tumors are more frequently primary, those of the bronchial glands secondary.

Malignant growths are more common in the mediastinum, while inflammatory processes and tuberculous deposits more frequently affect the bronchial glands.

With mediastinal growths, especially of the anterior mediastinum, disturbances of circulation usually precede those of respiration, the contrary being the rule in disease of the bronchial glands.

Although both show a tendency to extend inward, mediastinal tumors are much more frequently attended by enlargement and bulging of the chest wall.

Distinct physical signs can usually be obtained earlier in mediastinal than in glandular disease.

In thoracic aneurism, also, the early symptoms are those connected with the circulation, while respiratory disturbances, both subjective and physical, are developed late. The arterial murmur, aneurismal bruit, with a thrill and heaving impulse on palpation, is a valuable point of differentiation. In aneurism the area of dullness increases along the course of the artery or rises into the neck, while in bronchial-gland enlargement it is more fixed, increases less laterally, and is more common behind than anteriorly. Diminution and delay of the radial pulse upon one side and cardiac hypertrophy are occasional symptoms of thoracic aneurism.

Erosion of the sternum, so frequent with aortic aneurism, does not result from disease of the bronchial glands.

*Prognosis*.—The most important element in prognosis will always be the nature of the pathological process.

Malignant disease here, as elsewhere, terminates fatally, and tuberculosis will have a similar ending. Syphilitic growths, simple enlargements of serofulous origin, and subacute inflammatory processes may often be arrested and a practical cure effected when the nature of the disease can be recognized early. In such cases the extent of the growth, the rapidity with which it is extending, and its relations to and effects upon adjacent tissue must form the basis of any prognosis. The more serious complications are those arising from implication of the laryngeal nerves and obstruction of the vessels. Glandular abscesses which open into bronchial tubes may be followed by recovery, but are more frequently fatal, either immediately or from prolonged suppuration and exhaustion.

*Treatment*.—The cases in which treatment has proved distinctly effective have been simple chronic enlargements. The iodides, with iron and cod-liver oil internally, and counter-irritation between the scapula, have been the most successful measures employed. The iron and iodine may be given separately or in combination. For syphilitic cases, in which large doses of iodide are



required, the former method is to be preferred, but in scrofulous disease the syrup of the iodide of iron may be given with equally good results.

Cod-liver oil is always a valuable remedy, and especially so with children and in the lymphatic diathesis. Even cases of tuberculosis may be delayed and greatly benefited for a time by its use, and, whatever the nature of the disease, the oil may be employed with success for its general nutritive effect.

The persistent use of small (gr.  $\frac{1}{10}$  to  $\frac{1}{15}$ ) doses of the bichloride of mercury has occasionally benefited some cases even when no syphilitic element was present, and this plan may be tried when the iodides are unavailing.

Counter-irritation over the seat of the disease is always of decided value. It may be obtained by the use of any of the more persistent counter-irritants, as the tincture of iodine, iodine liniment, blisters, or the actual cautery.

The special symptoms require palliative treatment. The cough is seldom relieved by expectorants, and is best controlled by sedatives and antispasmodics. Codeine, heroine, morphine, and chloroform inhalations, in the spasmodic form, are the most certain in their effects, but the bromides, belladonna, Hoffman's anodyne, or chlorodyne are of value, and may suffice in some instances.

Pain is best relieved by anodyne lotions, and when severe by hypodermics of morphine. For local applications, laudanum, belladonna, chloroform, or camphorated liniments may be employed.

Dyspnoea is more safely relieved by chloroform inhalations and the ethereal preparations than by opium or other narcotics.

The enforcement of general hygienic and tonic measures will greatly increase the efficacy of any form of treatment.

Charles E. Quimby.

**LUNGS, DISEASES OF: BRONCHIAL PNEUMONIA.** See *Pneumonia, Bronchial*.

**LUNGS, DISEASES OF: CHRONIC PNEUMONIA.** See *Pneumonia, Chronic*.

**LUNGS, DISEASES OF: EMPHYSEMA.** See *Emphysema of the Lungs*.

**LUNGS, DISEASES OF: GANGRENE.**—Gangrene of the lung takes place whenever the nutrient circulation in a given area is interrupted. It does not follow directly upon obstruction of the functional vessels, although obliteration of a considerable branch of the pulmonary artery may afford a nidus for putrefactive germs, and thus entail gangrene as a secondary result. In a considerable proportion of cases it occurs as a complication of pneumonia, the intensity of the infection at a particular point being such as to compromise the vessels that feed the tissues of the lung. If pneumonia were an "inflammation" of the lung substance, inducing such a disturbance of nutrition as the amount of exudation implies, gangrene would be the issue in every case.

**ETIOLOGY.**—Gangrene appears as the initial local condition in many forms of infectious disease. It may occur in the course of any debilitating disease or during convalescence from protracted fever. It is an occasional event in nearly all of the exanthemata, and also in diabetes mellitus. It is observed frequently in aspiration pneumonia, putrefactive material having been implanted in the air passages (Osler\*).

The putrid contents of bronchiectatic cavities may induce gangrene in neighboring parts of the lung. The breaking down of cancerous growths communicating with the air passages may produce a like effect. Sometimes there is no assignable cause. Embolism of a bronchial artery, which would be easily overlooked, might explain some of the cases.

**PATHOLOGY.**—Two forms of pulmonary gangrene are described, the diffuse and the circumscribed. The former may take in a large area of lung. It is more common

\* "Practice of Medicine."

in the lower lobe, and in the outer portions of the lung rather than in the centre. It tends to form an irregular cavity with ragged and sloughy contour. The putrid tissue is dark green in color, approaching black, and it drips with a greenish and exceedingly ill-smelling fluid. Surrounding this gangrenous area is one of intense congestion, and the lung beyond this is oedematous. The bronchial membrane throughout the entire lobe is infected by the ichorous fluid passing over its surface and is intensely congested and covered with muco-purulent material.

The destruction of tissue is likely to lay open vessels of considerable size, and sudden and profuse hemorrhage may occur. The pleura may be perforated, and, its cavity being infected with germs of the most virulent character, the pleurisy which follows is rapidly fatal.

From the gangrenous focus extensive embolic processes may occur, resulting in secondary abscesses in different localities.

In the circumscribed form the destruction of tissues is not so widespread, and the resulting cavities are more sharply defined. This form is most likely to occur in the course of lobar pneumonia, when the bacteria of putrefaction will usually be found associated with the pneumococcus. It is this association probably which gives increased virulence to the infection and induces stoppage of a branch of the bronchial artery.

**COURSE OF THE DISEASE.**—The accession of gangrene is usually announced by the fetid character of the breath. The odor is pungent and peculiarly sickening, and pervades the room and sometimes the whole house, making it almost intolerable for the attendants. This is soon followed by a greenish-black putrid expectoration containing shreds of lung tissue and particles of a more solid material. The lighter portions rise to the surface, forming a greasy layer in which the microscope shows abundant crystals of fatty acids. The heavier detritus, including elastic fibres from the broken-down tissues, sinks to the bottom, and between these two layers is a watery stratum of a greenish color. The quantity of expectoration is large, amounting to ten or even twenty ounces in twenty-four hours. There may be, however, small foci of gangrene discovered post mortem which have not communicated with a bronchial tube, and consequently have not been accompanied by fetid breath (Osler).

The physical signs are those of infiltrated lung tissue combined with those of a cavity. Cavernal respiration may be modified by the shreddy character of the walls, and usually lacks the distinct quality met with in tuberculous excavations with rigid boundaries. Small circumscribed areas may present no definite physical signs other than those of bronchial infection.

Fever of a moderate degree and variable in its course is usually present, but in encapsulated cases there may be no rise of temperature. The absorption of infective material may give rise to typhoidal symptoms.

Irritation of the stomach and intestines is apt to arise from swallowing putrid matter coughed up from the lung. Secondary abscesses may occur, especially in the brain.

The **DIAGNOSIS** is principally from fetid "bronchitis." In the latter the fetor is not so extreme, and the expectoration does not contain shreds of lung tissue. The affection is also not so acute and produces less constitutional disturbance. The secondary infections at distant points are absent.

In some cases of small encapsulated foci the diagnosis is extremely difficult and may be possible only at autopsy.

**PROGNOSIS.**—Gangrene of the lung when extensive is generally fatal. Death may be the result of sepsis from absorption of gangrenous material; of a rupture into the pleura, pericardium, or peritoneum; of hemorrhage; or of an exhausting infective diarrhoea. Occasionally even a large cavity heals completely, and the patient recovers. According to Strümpell,\* in encapsulated cases

the question of recovery or death may not be determined for months or even years. Small foci may be discovered at autopsy which have not been suspected during life.

**TREATMENT.**—As to treatment, much may be done in the way of prophylaxis in cases in which there is special danger of aspiration pneumonia, as, for example, in the insane and in the subjects of bulbar paralysis, and in recovering from anaesthesia. Care in the selection, preparation, and administration of the food in such cases will greatly lessen the liability of foreign matter being drawn into the air passages.

When the disease is actually present every effort should be made to keep up the strength of the patient, as in this lies our principal hope. Concentrated liquid food and alcoholic stimulants will be required. Quinine, strychnine, and carbonate of ammonia will be useful. Antiseptic remedies that are eliminated through the lungs promise more than any other form of medication. Among these are creosote, creosotal, turpentine, carbolic acid.

Inhalations of various antiseptics are usually employed, but it is difficult to make them penetrate into the affected portion of the lung.

Creosote, eucalyptol, menthol, iodine, bromine, formaldehyde have been recommended, but they exert their effect much more upon the healthy than upon the diseased areas, and unless greatly diluted will cause irritation which may be hurtful out of proportion to the good the inhalations accomplish. Inhalations of pure oxygen are indicated aside from any effect in relieving dyspnoea, as oxygen locally applied has been proved useful in improving the nutrition of foul, sloughing surfaces.\*

Sheets dampened with solutions of deodorizing substances, such as the chlorides, and hung about the room, will relieve in a measure the sickening fetor of the atmosphere, and contribute to the comfort of the attendants as well as of the patient.

There may be room for possible benefit from surgical interference, particularly in protracted cases.†

Andrew H. Smith.

**LUNGS, DISEASES OF: HYPERÆMIA.**—There are two kinds of congestion, active and passive.

1. **Active Congestion** of the lungs is a condition concerning which there is not a unanimity of opinion. Osler and other American and English authors believe that it is simply part and parcel of some other inflammatory disease of the lungs, such as pneumonia, bronchitis, tuberculosis, pleurisy, etc.

Acute congestion of the lungs and congestive chills were once familiar diagnoses. We now know that they are usually the initial symptoms of some acute infectious process, such as pneumonia. A case in point has recently come under my observation. An extremely severe chill followed by high fever was called a congestive chill. In a few hours distinct signs of pneumonia were present, with diplococci in the sputum. The disease, however, aborted in from twenty-four to thirty-six hours. Such a case would be termed acute congestion by the French writers, who give this condition the dignity of a disease *per se* (maladie de Woillez). They describe a definite symptomatology, such as initial chill, pain in the side, cough, dyspnoea, and slight elevation of temperature, 101°-103° F. The physical signs are indefinite, such as impaired resonance, weak vesicular or blowing breathing, with crackling râles. These signs, however, can all be associated with just such anomalous cases of pneumonia as above mentioned. In many epidemics these larval cases are common.

It is stated by some authors that a rapidly fatal congestion may follow extreme exertion, or exposure to excessive heat or cold. Leuf reports cases in which, in association with drunkenness, exposure, and cold, death occurred suddenly, or within twenty-four hours; post mortem an extreme congestion was the only pathological condition found.

\* Demarquay: "Pneumatologie Médicale."  
† Am. Journ. of the Medical Sciences, March, 1902, p. 375.

\*\*Text-book of Medicine," 3d American edition. D. Appleton & Co., New York, 1901.

2. **Passive Hyperæmia or Congestion.**—There are two forms: (a) Mechanical; (b) hypostatic.

**Mechanical.**—Etiology. In the passive form there is an excess of venous blood in the lungs due to obstruction to the flow of blood into the heart. Mechanically, this results from the presence of chronic valvular lesions of the heart in which incompenation has taken place. Lesions on both sides may produce this result. It will also occur in myocardial degeneration with incompenation and the development of relative insufficiencies. Emphysema may also be responsible for this condition. Brown induration of the lungs is a sequel. Osler describes such a lung as voluminous, russet-brown in color, cutting and tearing with great resistance. On section, it shows at first a brownish-red tinge, and then the cut surface, exposed to the air, becomes rapidly of a vivid red color from oxidation of the abundant hæmoglobin. Histologically, it is characterized by (a) great distention of the alveolar capillaries; (b) increase of the connective-tissue elements of the lungs; (c) the presence, in the alveolar walls, of many cells containing altered blood pigment; and (d) the presence, in the alveoli, of numerous epithelial cells containing blood pigment in all stages of alteration, which are also found in great numbers in the sputum.

The presence of tumors may cause a local congestion. The symptoms of this passive form develop with incompenation and are dyspnoea, cough, expectoration, etc.

(b) **Hypostatic Congestion.**—In conditions of great weakness of the heart, such as follow the various acute fevers, anæmias, cachexias, Bright's disease, prolonged coma, etc., there is a transudation of serum from the blood-vessels into the dependent tissues of the body. This is of course favored by gravity, but is not essentially dependent upon it, since a healthy man may be in bed for weeks without its development. If the thorax of such a patient be examined there will be found, posteriorly and in the axillæ, impaired resonance, feeble breath sounds, and numerous crackling râles. The tactile fremitus is perhaps somewhat diminished. When the transudation is extensive and of long duration the dulness may become marked, with weak blowing breathing; it is now termed hypostatic pneumonia. This condition is usually a part of a more general transudation of serum. It must be distinguished from atelectasis, since in all inactive individuals fine crackling râles are frequently heard at the lower borders of the lungs. Hydrothorax and pneumonia must also be excluded. There are no special symptoms of this condition and it is often discovered only by careful examination.

Osler refers to the forms of passive congestion which occur in injury to and diseases of the brain. In prolonged coma there may be an association of patches of consolidation along with the congestion, due to the aspiration of particles of food.

**Pathology.** The posterior portion of the lung is dark in color and engorged with blood and serum; part of it may even sink in water, when it is termed splenization, or hypostatic pneumonia.

**Treatment.** This consists in the treatment of the primary disease; with its improvement the signs and symptoms of the congestion disappear. If the symptoms are severe and are secondary to incompenated valvular affections, venesection, with the removal of from twenty to thirty ounces of blood, may reduce the congestion and relieve the right heart. James Rae Arneill.

**LUNGS, DISEASES OF: INFARCTION.**—Infarction (*in*, and *farvere*, to stuff) of the lung consists in a "hemorrhagic engorgement" of a circumscribed area of pulmonary tissue. Although first classically described by Laënnec in 1819, it appears that the condition was characterized by him as that of an apoplectic area, being compared to cerebral apoplexy. Laënnec dwelt upon the venous thrombosis in such cases, apparently disregarding completely the arterial occlusion upon which now so much stress is laid.

2. **Passive Hyperæmia or Congestion.**—There are two forms: (a) Mechanical; (b) hypostatic.

**Mechanical.**—Etiology. In the passive form there is an excess of venous blood in the lungs due to obstruction to the flow of blood into the heart. Mechanically, this results from the presence of chronic valvular lesions of the heart in which incompenation has taken place. Lesions on both sides may produce this result. It will also occur in myocardial degeneration with incompenation and the development of relative insufficiencies. Emphysema may also be responsible for this condition. Brown induration of the lungs is a sequel. Osler describes such a lung as voluminous, russet-brown in color, cutting and tearing with great resistance. On section, it shows at first a brownish-red tinge, and then the cut surface, exposed to the air, becomes rapidly of a vivid red color from oxidation of the abundant hæmoglobin. Histologically, it is characterized by (a) great distention of the alveolar capillaries; (b) increase of the connective-tissue elements of the lungs; (c) the presence, in the alveolar walls, of many cells containing altered blood pigment; and (d) the presence, in the alveoli, of numerous epithelial cells containing blood pigment in all stages of alteration, which are also found in great numbers in the sputum.

The presence of tumors may cause a local congestion. The symptoms of this passive form develop with incompenation and are dyspnoea, cough, expectoration, etc.

(b) **Hypostatic Congestion.**—In conditions of great weakness of the heart, such as follow the various acute fevers, anæmias, cachexias, Bright's disease, prolonged coma, etc., there is a transudation of serum from the blood-vessels into the dependent tissues of the body. This is of course favored by gravity, but is not essentially dependent upon it, since a healthy man may be in bed for weeks without its development. If the thorax of such a patient be examined there will be found, posteriorly and in the axillæ, impaired resonance, feeble breath sounds, and numerous crackling râles. The tactile fremitus is perhaps somewhat diminished. When the transudation is extensive and of long duration the dulness may become marked, with weak blowing breathing; it is now termed hypostatic pneumonia. This condition is usually a part of a more general transudation of serum. It must be distinguished from atelectasis, since in all inactive individuals fine crackling râles are frequently heard at the lower borders of the lungs. Hydrothorax and pneumonia must also be excluded. There are no special symptoms of this condition and it is often discovered only by careful examination.

Osler refers to the forms of passive congestion which occur in injury to and diseases of the brain. In prolonged coma there may be an association of patches of consolidation along with the congestion, due to the aspiration of particles of food.

**Pathology.** The posterior portion of the lung is dark in color and engorged with blood and serum; part of it may even sink in water, when it is termed splenization, or hypostatic pneumonia.

**Treatment.** This consists in the treatment of the primary disease; with its improvement the signs and symptoms of the congestion disappear. If the symptoms are severe and are secondary to incompenated valvular affections, venesection, with the removal of from twenty to thirty ounces of blood, may reduce the congestion and relieve the right heart. James Rae Arneill.

**LUNGS, DISEASES OF: INFARCTION.**—Infarction (*in*, and *farvere*, to stuff) of the lung consists in a "hemorrhagic engorgement" of a circumscribed area of pulmonary tissue. Although first classically described by Laënnec in 1819, it appears that the condition was characterized by him as that of an apoplectic area, being compared to cerebral apoplexy. Laënnec dwelt upon the venous thrombosis in such cases, apparently disregarding completely the arterial occlusion upon which now so much stress is laid.

2. **Passive Hyperæmia or Congestion.**—There are two forms: (a) Mechanical; (b) hypostatic.

**Mechanical.**—Etiology. In the passive form there is an excess of venous blood in the lungs due to obstruction to the flow of blood into the heart. Mechanically, this results from the presence of chronic valvular lesions of the heart in which incompenation has taken place. Lesions on both sides may produce this result. It will also occur in myocardial degeneration with incompenation and the development of relative insufficiencies. Emphysema may also be responsible for this condition. Brown induration of the lungs is a sequel. Osler describes such a lung as voluminous, russet-brown in color, cutting and tearing with great resistance. On section, it shows at first a brownish-red tinge, and then the cut surface, exposed to the air, becomes rapidly of a vivid red color from oxidation of the abundant hæmoglobin. Histologically, it is characterized by (a) great distention of the alveolar capillaries; (b) increase of the connective-tissue elements of the lungs; (c) the presence, in the alveolar walls, of many cells containing altered blood pigment; and (d) the presence, in the alveoli, of numerous epithelial cells containing blood pigment in all stages of alteration, which are also found in great numbers in the sputum.

The presence of tumors may cause a local congestion. The symptoms of this passive form develop with incompenation and are dyspnoea, cough, expectoration, etc.

(b) **Hypostatic Congestion.**—In conditions of great weakness of the heart, such as follow the various acute fevers, anæmias, cachexias, Bright's disease, prolonged coma, etc., there is a transudation of serum from the blood-vessels into the dependent tissues of the body. This is of course favored by gravity, but is not essentially dependent upon it, since a healthy man may be in bed for weeks without its development. If the thorax of such a patient be examined there will be found, posteriorly and in the axillæ, impaired resonance, feeble breath sounds, and numerous crackling râles. The tactile fremitus is perhaps somewhat diminished. When the transudation is extensive and of long duration the dulness may become marked, with weak blowing breathing; it is now termed hypostatic pneumonia. This condition is usually a part of a more general transudation of serum. It must be distinguished from atelectasis, since in all inactive individuals fine crackling râles are frequently heard at the lower borders of the lungs. Hydrothorax and pneumonia must also be excluded. There are no special symptoms of this condition and it is often discovered only by careful examination.

Osler refers to the forms of passive congestion which occur in injury to and diseases of the brain. In prolonged coma there may be an association of patches of consolidation along with the congestion, due to the aspiration of particles of food.

**Pathology.** The posterior portion of the lung is dark in color and engorged with blood and serum; part of it may even sink in water, when it is termed splenization, or hypostatic pneumonia.

**Treatment.** This consists in the treatment of the primary disease; with its improvement the signs and symptoms of the congestion disappear. If the symptoms are severe and are secondary to incompenated valvular affections, venesection, with the removal of from twenty to thirty ounces of blood, may reduce the congestion and relieve the right heart. James Rae Arneill.

**LUNGS, DISEASES OF: INFARCTION.**—Infarction (*in*, and *farvere*, to stuff) of the lung consists in a "hemorrhagic engorgement" of a circumscribed area of pulmonary tissue. Although first classically described by Laënnec in 1819, it appears that the condition was characterized by him as that of an apoplectic area, being compared to cerebral apoplexy. Laënnec dwelt upon the venous thrombosis in such cases, apparently disregarding completely the arterial occlusion upon which now so much stress is laid.

\* Demarquay: "Pneumatologie Médicale."  
† Am. Journ. of the Medical Sciences, March, 1902, p. 375.

\*\*Text-book of Medicine," 3d American edition. D. Appleton & Co., New York, 1901.