mals which have died of rinderpest and tuberculosis. It may also be due to insufficient bleeding after death in animals which have died from natural causes.

Dark Reddish-Brown.—This color is due to imperfect oxidation of the blood, and is seen in animals which have been drowned, or suffocated in smoke. It is also occasionally seen in the flesh of overdriven or hunted animals.

Green or Violet.—This color is due to the beginning of putrefaction, or to the diffusion of vegetable coloring matter through the walls of the stomach after death.

The Consistency of Meat.—The consistency of flesh food is an index of its soundness. Good meat is firm, while unsound meat is usually flabby and exudes moisture. Coarse-grained meat which cannot be cut evenly is inferior to fine-grained meat.

Lehmann has devised an ingenious apparatus for determining the degree of toughness of meat (*Zeitschrift Fleisch- u. Milch-Hyg.*, 1898, viii., p. 32). It consists of a balance with arms of different lengths, the shorter being made on the plan of a pair of scissors with one fixed blade. The weights are placed in the pan of the longer arm, and the force required to cut through a layer of the meat 1 cm. thick is expressed in grams. By this means Lehmann found that the skin muscle of beef is two and one-half times as tough as the fillet. Flesh which contains much collagenous tissue becomes more tender on boiling, while meat which contains but little remains about the same as before boiling.

Lehmann obtained the following results:

FORCE REQUIRED FOR DIVISION EXPRESSED IN GRAMS.

	Raw.	Boiled.
Fillet of beef	83.4	84.0
Skin muscle of beef	236.4	88.8
Heart	104	88
Liver, ox	42	8
Liver, calf	35	6.6
Kidneys	40	24
Brain	7	2.4

The Odor of Meat.—Aside from ordinary rough inspection by the sense of smell, which is much more acutely developed in some persons than in others, the odor may also be observed by boiling fragments of flesh with water, and also by mixing the flesh with dilute sulphuric acid, distilling about one-fourth of the liquid, and noting the smell of the distillate; it may be:

1. The normal odor, characteristic of the animal.
2. The characteristic odor intensified, as in the case of uncastrated male animals. This is more marked with the flesh of the he-goat and boar than with that of the ram and bull.
3. An abnormal odor due to the substances eaten by the animal.
4. An odor due to chemical alteration or decomposition, as, for example, that of the volatile products formed during the putrefaction of flesh.
5. An odor of foreign substances, chloride of lime, carbolic acid, etc.

The Diseases of Animals Used as Food.—Animals should be inspected within twenty-four hours before slaughter. The principal diseases for which the inspecting officer should watch are:

Among cattle, 1. Pleuropneumonia; this disease is not easily recognized at first. The temperature soon rises to 104° or 105° F. and the animal refuses food. A short, dry cough develops and the breathing becomes labored and painful.

2. Cattle plague (Rinderpest). Recognized by early prostration, shivering, discharge from nose, eyes and mouth, cessation of rumination, abdominal pain and scouring.

3. *Anthrax.* This may be general or localized. If boils, pustules, or carbuncles form they are recognized at once. The peculiar organism of anthrax may be detected in the blood.

4. *Tuberculosis.* This disease has attracted more attention than any other, but the question whether it may be transmitted from animals to man does not yet appear to be fully settled. In cattle it may be acute or chronic. At first there may be no emaciation nor diminution of the milk; later, emaciation supervenes, and there are loss of appetite, shortness of breath, and cough, and these become intensified.

Three royal commissions have reported in England upon the subject of bovine tuberculosis, those of 1890, 1895, and 1898. The conclusions of the latter commission (1898), so far as meat is concerned, were as follows:

"We recommend that the Local Government Board be empowered to issue instructions from time to time for the guidance of meat inspectors, prescribing the degree of tuberculous disease which, in the opinion of the board, should cause a carcass, or part thereof, to be seized.

"Pending the issue of such instructions, we are of the opinion that the following principles should be observed in the inspection of tuberculous carcasses of cattle:

"*a.* When there is miliary tuberculosis of both lungs.
"*b.* When tuberculous lesions are present on the pleura and peritoneum.
"*c.* When tuberculous lesions are present in the muscular system, or in the lymphatic glands, embedded in or between the muscles.
"*d.* When tuberculous lesions exist in any part of an emaciated carcass.

} The entire carcass and all the organs *may be* seized.

"*a.* When the lesions are confined to the lungs, and the thoracic lymphatic glands.
"*b.* When the lesions are confined to the liver.
"*c.* When the lesions are confined to the pharyngeal lymphatic glands.
"*d.* When the lesions are confined to any combination of the foregoing, but are collectively small in extent.

} The carcass, if otherwise healthy, shall not be condemned, but every part of it containing tuberculous lesions *shall be* seized.

"In view of the greater tendency to generalization of tuberculosis of the pig, we consider that the presence of tubercular deposit in any degree should involve seizure of the whole carcass and of the organs. In respect of foreign dead meat, seizure shall ensue in every case where the pleura have been 'stripped.'"

5. *Actinomycosis.* Attacks by preference the lower jaw and tongue, also the lungs and bones. It leads to general malnutrition and is sometimes fatal.

6. *Texas Cattle Fever.* In this disease there is intense fever with a temperature of from 105° to 110° F., with great weakness and prostration. The ears and head droop, the hind legs are advanced under the body, giving the animal a characteristic attitude. The urine becomes deep colored, like undiluted venous blood. The liver and spleen are congested and enlarged, the kidneys also are congested and show numerous blood extravasations.

Sheep. In addition to the foregoing diseases sheep are subject to splenic apoplexy, or "braxy." The meat in this disease is dark and sometimes dropsical, and the weight of the spleen is increased, often to double its normal weight. When attacked the animal staggers, stretches out its head, and breathes rapidly.

Sheeppox is known by the high fever, especially in the pustular stage, by the flea-bitten appearance of the skin in the early stage, and by the rapid appearance of nodules or vesicles.

Liver flukes are large parasites, an inch or more in length, and about three-eighths of an inch wide, which are found in the bile ducts of the liver, occasioning the disease known as the "rot." The principal symptoms are

sluggishness, followed by wasting and pallor of the mucous membrane, diarrhœa, yellowness of the eyes, falling of the hair, and dropsical swellings.

Swine. The principal parasitic diseases of the hog which unfit the meat for use as food are the "measles" and trichinosis. The former is known by the appearance of small, egg-shaped bladders about one-quarter of an inch in length containing the larvæ known as cysticerci, which when eaten uncooked or nearly raw become tapeworms in human beings. In live hogs these little bladders may occasionally be seen beneath the tongue, or in the loose folds near the tail. Perroncito found that a temperature of 50° C. (122° F.) maintained for a minute or more destroyed the vitality of cysticerci.

Trichinosis. Trichinæ are found chiefly in the muscular tissue, though occasionally in the fat of swine. They are usually most abundant in the pillars of the diaphragm. With a low magnifying power they may be easily detected in a thin shaving of infected pork, either encysted and coiled up in the cyst or free and living. Swine affected with this disease do not necessarily present noticeable symptoms during life. Examinations made by the State Board of Health of Massachusetts showed that swine fed upon city offal, or garbage, and especially upon the entrails of infected animals, were far more subject to the disease than those which are fed upon healthy food (grain, vegetables, or cooked food).

This disease has assumed an international importance, as shown in a recently published pamphlet of the United States Department of Agriculture entitled "Trichinosis in Germany."

The danger to man lies in the eating of raw or imperfectly cooked pork, ham, bacon, sausages, or other meat of swine, and consequently the thorough cooking of such meat will prevent its occurrence.

Hog Cholera. Animals affected with this disease have fever, shivering, unwillingness to move, loss of appetite, a temperature of 106° to 107° F. They appear stupid and dull, and hide in the litter. The bowels may at first be constipated, but later there is usually a liquid and fetid diarrhœa, exhausting and persistent. There is rapid loss of flesh. The animal grows weak, stands with arched back and abdomen drawn up, and walks with tottering gait.

Horse Flesh. A law was enacted in England in 1889 which defines horse flesh to be such flesh cooked or uncooked, alone or mixed with other substances, and includes the flesh of asses and mules. This act provides that the flesh of horses, asses, or mules must not be sold or kept for sale as human food, except in a shop or stall over which is placed conspicuously, in legible characters four inches long, a statement that horse flesh is sold there. It also prohibits the sale of horse flesh for human food to any purchaser asking for other meat, or for a compound article not usually made of horse flesh.

There is no evidence that sound, healthy horse flesh is less wholesome than that of beef.

The Use of Preservatives.—For the purpose of preserving fresh meat, fish, canned meats, hams, and sausages various chemical agents are employed, and the tendency to use these substances appears to be increasing. Various opinions are expressed as to the propriety of using such agents as salicylic and boric acid and formaldehyde. Although the harm arising from the constant use of such substances may be less than that which might arise from using meat in a state of incipient putrefaction, the possibility still remains of harmful effect to the consumer from the frequent use of preservatives in meat and other kinds of food. The substances in most common use for this purpose are boric acid and borax, salicylic acid, sulphites, and formalin. The following are the recommendations of the recent British Parliamentary Commission upon this subject. This report was made to Parliament in 1901:

1. That the use of formaldehyde or formalin, or preparations thereof, in food or drinks, be absolutely prohibited, and that salicylic acid be not used in a greater proportion than one grain per pint in liquid food, and one grain per pound in solid food. Its presence in all cases to be declared.

2. That the use of any preservative or coloring matter whatever in milk offered for sale in the United Kingdom be constituted an offence under the Sale of Food and Drug Acts.

3. That the only preservative which it shall be lawful to use in cream be boric acid or mixtures of boric acid and borax, and in amount not exceeding 0.25 per cent., expressed as boric acid, the amount of such preservative to be notified upon the vessel by a label.

4. That the only preservative to be used in butter and margarin be boric acid or mixtures of boric acid and borax, to be used in proportions not exceeding 0.5 per cent., expressed as boric acid.

5. That in the case of all dietetic preparations intended for the use of invalids or infants chemical preservatives of all kinds be prohibited.

6. That the use of copper salts in the so-called greening of preserved foods be prohibited.

7. That means be provided, either by the establishment of a separate court of reference, or by the imposition of more direct obligation on the Local Government Board, to exercise supervision over the use of preservatives and coloring matters in foods, and to prepare schedules of such as may be considered inimical to the public health.

Samuel W. Abbott.

BIBLIOGRAPHY.

Atwater: Chemistry and Nutritive Value of Food. Century Magazine, 1887–1888.
Bigelow: Meat and Meat Products, in Bulletin No. 65, U. S. Dept. of Agriculture, Bureau of Chemistry, 1902.
C. A. Mitchell, B.A.: Flesh Foods, London, 1901.
Dr. O. Schwarz: Bau, Einrichtung und Betrieb von öff. Schlachthöfen, Berlin, 1894.
F. Vacher: Food Inspector's Handbook, London, 1898.
Gesundheitsbüchlein, Kais. Gesundheitsamt, Berlin, 1895.
Hime: Practical Guide to the Health Acts, London, 1901.
J. A. Dembo: Jewish Method of Slaughter, London, 1894.
König: Chemie der menschlichen Nahrungs- und Genussmittel, Berlin, 1889.
Lehmann: Methods of Practical Hygiene.
Notter: Theory and Practice of Hygiene, London and Philadelphia, 1900.
Ostertag: Handbuch der Fleischbeschau für Tierärzte, 1892.
Palmberg: Public Health and Its Applications, London, 1893.
Report of the Departmental Committee appointed to inquire into the Use of Preservatives and Coloring Matters in Food, London, 1901.
E. W. Hope: Slaughter-Houses and their Administration. Stevenson and Murphy's Hygiene, vol. i., p. 975.
Strohmer: Die Ernährung des Menschen, 1887.
Trichinosis in Germany, U. S. Dept. of Agriculture, Washington, D. C., 1901.
Zeitschrift für Fleisch- und Milch-Hygiene, Berlin, 1890–1902.
Zeitschrift für Nahrungsmittel-Untersuchung und Hygiene, Wien, 1887–1902.

MEDIASTINUM, DISEASES OF THE.—The mediastinum is a space left in the median portion of the chest by the non-approximation of the pleuræ; it is bounded in front by the sternum, behind by the vertebral column, and on either side by the pleural surfaces. It may be divided into two parts, the anterior mediastinum, including the space in front of the pericardium and trachea, and the posterior mediastinum, including the space behind these. The structures found in the anterior mediastinum are: the heart surrounded by the pericardium, the ascending aorta and the lower parts of its branches, the lower part of the superior vena cava, the greater azygos vein, the innominate veins, the pulmonary artery dividing into its two branches, the right and left pulmonary veins, the bifurcation of the trachea and the two bronchi, the phrenic nerves, the anterior mediastinal lymph glands, the bronchial lymph glands, and, in early life, the thymus gland.

The posterior mediastinum contains: the descending aorta, the greater and lesser azygos veins, the pneumogastric and splanchnic nerves, the œsophagus, the thoracic duct, and the posterior mediastinal lymph glands. In considering the diseases of the mediastinum, however, the heart and pericardium are not included, nor are the trachea, bronchi, œsophagus, blood-vessels, and nerves, except so far as they are secondarily involved. The structures in this region which chiefly concern us are the

thymus gland, and the lymph glands and vessels which are subject to degenerative changes, infection, hyperplasia, hemorrhage, and tumor formation.

Thymus Gland.—This is an organ of intra-uterine life and childhood, reaching its greatest development during the second year and from that time on undergoing a slow atrophy and fatty degeneration until it is finally transposed into a mass of fat, the so-called "thymic fat organ" (Waldeyer). The thymus arises from the entodermal layer of the third gill clefts, each of which sends down a tubular prolongation of epithelial cells on one side of the trachea; these tubes (which have a narrow lumen and a thick epithelial covering) then approach each other and coalesce in front of the trachea to form a solid flat organ. From the mesoderm a vascular stroma grows into the epithelial mass, dividing it into lobes and lobules, and forming a capsule around it. Small, round connective-tissue cells accumulate within the stroma and form follicles of lymphoid tissue, increasing at the expense of the epithelial cells, which are finally reduced to scattered islands of concentrically arranged squamous cells—the so-called Hassall's corpuscles. These corpuscles persist and may even be found after involution of the organ, in the thymic fat. Involution takes place by disappearance of the lymphoid cells and their replacement by epithelial cells derived apparently from the endothelium of the perivascular lymph spaces, which become filled with fat. The thymic fat organ containing Hassall's corpuscles can be found even in old age. It is important to remember that the capsule of the thymus gland is rich in lymph glands.

The thymus grows rapidly during intra-uterine life, attaining its greatest dimensions between the first and second years of infancy. At this time it consists of two flat lobes joined by delicate vascular connective tissue; it is soft and pinkish and has an abundant milky white secretion, which has often been mistaken for pus. Lying directly upon the trachea, it extends from 1 cm. above the sternum down to the lower border of the fourth costal cartilage, being in relation above with the thyroid gland and below with the pericardium, the arch of the aorta, the pulmonary artery, the superior vena cava, and both innominate veins, with all of which it is connected by delicate bands of fibrous tissue rich in lymph glands. As to the physiology of this organ, whether it is concerned in blood formation or in the development of bone or of the nervous system, we know practically nothing, and extirpation experimentally performed upon animals has as yet thrown no light on the question.

The dimensions of the thymus gland are very variable even within normal limitations, and this fact has given rise to great confusion as to what size the gland must attain in order to be regarded as abnormal. Friedleben's classical work ("Die Physiologie der Thymusdrüse," Frankfort a. M., 1858) remains to this day the source of most of our knowledge on this subject. Here are his statistics as to the weight of the gland at different ages: Three to five months intra-uterine life, 0.3 gm.; six to seven months intra-uterine life, 2.4 gm.; eight months intra-uterine life, 8.4 gm.; at term (measuring 6 by 4 cm.), 14.3 gm.; one to nine months after birth (this is the period of greatest secretory activity), 20.7 gm; ninth month to second year, 27.3 gm.; second year to fifteenth year, 27.0 gm.; fifteenth year to twenty-fifth year, 22.1 gm.; twenty-fifth year to thirty-fifth year, 3.1 gm.

As can be seen by these figures the atrophy of the thymus is not nearly so rapid as is usually supposed, beginning practically after the twentieth year instead of at puberty, as is so often stated. Vierordt, for instance, gives the weight at birth as 24 gm., reaching 26 gm. at the end of the second year and remaining at this weight until puberty, when atrophy begins and is complete by the twentieth year. But the figures given by Friedleben, depending as they do upon an enormous number of examinations, are generally accepted. Individual variations in size undoubtedly occur, and the nutrition of the child is a very important factor, as the thymus is invariably atrophied in marantic children; indeed Seydel considers this atrophy the most indisputable proof of death from inanition.

This variability in size makes it a matter of much difficulty to decide in a given case whether, in the absence of other pathological findings, a thymus gland exceeding the average in size may be regarded as the cause of death. The so-called "asthma thymicum" is absolutely denied by some authorities, by others regarded as a not very uncommon cause of death in childhood, and a rare cause in adult life. Statistics founded on the cases reported prior to Friedleben's work prove little and many cases since his time are open to objections; but even after a most searching review there remain a number of indisputable instances of sudden death, from strangulation, of healthy infants and children in whom the autopsy revealed no abnormality except a much enlarged thymus gland. Such are the four cases reported by Pott, in which all the patients died with symptoms of suffocation within two minutes after the attempt of the physician to insert a spatula into the mouth; the two cases of Grawitz; the case of Clar, who after performing tracheotomy on a child, was unable to insert the tube because of the narrowing of the trachea from pressure of an enlarged thymus.

Granting that the enlargement of this gland may cause death by suffocation, it remains to be explained how such a result is brought about. A direct mechanical compression of the trachea, evidenced by a flattening perceptible after death, has been shown only in the cases of Somma, of Benecke, and of Barach. More probable seems compression of the thin-walled, collapsible superior vena cava with its branches, which might be gradually compressed for some time without giving rise to symptoms. Hasse gives this explanation.

Another possibility is pressure on the recurrentes or vagi, in which case asthma thymicum would be spasma glottidis set up reflexly from the thymus. All of these explanations are open to the objection of not accounting for the suddenness of the symptoms. The puzzling fact is that death takes place apparently by suffocation within a few moments. If the large thymus is the cause, then it acts quite differently from other tumors by inducing, not a gradually increasing dyspnœa, but a spasma glottidis. Pott endeavors to explain it as due to a sudden bending back of the child's head, increasing the pressure; but in that case the child's instinct would be sufficient to cause it to right the position of the head at once. More plausible, but still unproved, is the theory of gradually increasing enlargement which finally reaches the fatal point, an enlargement which would seem not improbable in rachitic or lymphatic children, but would be hard to explain in the healthy; or it may be that the thymus is subject to great vascular engorgement and that the increase in size to the point of fatal compression is actually as sudden as are the resulting symptoms, the contraction of the vessels after death preventing the condition from being recognized. Jacobi considers this possible. Unfortunately for this theory the thymus is not a very vascular organ.

Cohnheim first pointed out the fact that most of the children who die of spasma glottidis are rachitic. He reported one such case, in a rachitic child with one lung partially atelectatic as a result of the pressure of a large thymus. The relation between spasma glottidis and rachitis has been emphasized by Jacobi also, but he is inclined to attribute death in these cases not to direct pressure but to the cerebral and meningeal hyperæmia and effusion resulting from the rachitis. In proof of this he points out the increase of thymic asthma in this country as coincident with the increase of rachitic children following the enormous immigration from the poor, ill-nourished classes of Europe. He is able to account in this way for all the cases of death from laryngismus stridulus which have come under his notice, except one in which the thymus weighed 410 gm. and extended from the thyroid gland down to and covering the pericardium.

Persistent thymus gland in middle life and old age has been given as a possible cause of death in some obscure

cases, but in most of these other possible causes were not eliminated. Among the least doubtful may be mentioned those of Bruce (1867), Jacobi (1883), and Glück (1894). Complete absence of the thymus was first observed by Bischoff in a still-born child otherwise perfectly normal; later on, four cases were reported by Friedleben. It is commonly absent in monstrosities, especially anencephalic monsters.

Inflammation of the thymus, with or without pus formation, is not common and many of the instances of thymic abscesses in the literature may have been simply normal glands, the milky secretion of which was mistaken for pus. Undoubted cases are those of Wittich, Hennig, Demmé, and Pürkhardt; that of the last-named was a thymic abscess which broke into the trachea. Jacobi found changes in the thymus in two cases of diphtheria, changes which from his description were evidently focal necroses. Simple inflammation by extension from a pleurisy or pericarditis has been also found. Small hemorrhages are often found in the thymus gland in healthy children who have died during or shortly after delivery. Friedleben reports one case of extensive hemorrhage in the thymus and elsewhere, the child dying suddenly while asleep, without spasm. Malnutrition was marked in this case. Péan's case was one of purpura hæmorrhagica in an eleven-year-old child; the thymus was very large and soft, resembling the spleen, and full of hemorrhages. Hoffmann thinks that this was probably a lymphoma with hemorrhages.

Cysts in the thymus have been described and have been accounted for by œdema, by softening of blood clots, or by softening of gummata. Tuberculosis is not rare, but is usually secondary to tuberculosis of the bronchial glands, although Demmé has reported a large tuberculous thymus in a child of six weeks with no tuberculosis in any other organ. Syphilis of the thymus gives rise to various lesions: to foci of suppuration (Dubois, Wells, Hanfsted, Mervis), or cysts (Jacobi, Hoffman), or syphilitic endarteritis with induration (Fürth, Jacobi), or gumma (Jacobi).

Tumors of the thymus will be described in the section on mediastinal tumors in general.

Diseases of the Bronchial and Mediastinal Glands.—Barety's division of the bronchial glands ("De l'adénopathie trachéobronchique," Thèse de Paris, 1875), which is very generally accepted, is the following:

1. Right pretracheobronchial group in relation with the superior vena cava, the arch of the aorta, vagi, trachea, and right bronchus.
2. Left pretracheobronchial group, at the angle of the bifurcation of the trachea and along the left bronchus. This is less large and important than the right group.
3. Intertracheobronchial group in the space below the bifurcation of the trachea between the main bronchi.
4. Peribronchial group, accompanying the bronchi.

In addition there is the small group of anterior mediastinal glands in the areolar tissue in front of the pericardium and the posterior mediastinal glands, which run in a chain in the posterior mediastinum parallel with the œsophagus.

Simple, non-infectious hyperplasia of the bronchial glands has been described by Thomas and by Biedert. The symptoms are the same as in tuberculous infection of these glands, and the authors in question base their belief in the non-infectious nature of the process on the apparent recovery of their cases. On the other hand, many authorities believe that all enlargements of the bronchial lymph glands are tuberculous; others distinguish a purely suppurative form which may end in resolution or in abscess formation, but which is not primarily or secondarily tuberculous. In every case of bronchitis the glands are probably involved in the inflammation, but usually this subsides; if, however, it goes on to enlargement of the gland by chronic productive inflammation we have the symptoms due to pressure or contraction, which will be considered under tuberculous glands. Measles, grippe, pneumonia, whooping-cough may give rise to such hyperplasia; indeed, Mussy believes that the pressure of these enlarged glands is the true anatomical cause of the attacks of coughing in whooping-cough. Aside from the possibility of chronic hyperplasia there is that of abscess formation, and it may be that certain mediastinal abscesses of unknown origin have formed in this way. On the other hand, healing may take place with contraction which may compress or form diverticula in the neighboring structures, as the bronchi, pleuræ, pericardium, or œsophagus. Such contracted glands are usually deeply pigmented from accumulations of coal dust, and it is suggested that the presence of large quantities of this dust may in itself be sufficient to cause the growth of connective tissue with contraction.

Tuberculosis is undoubtedly responsible for the great majority of cases of enlargement of the bronchial glands. It may appear either in the acute miliary form, giving rise to no clinical symptoms, or in the caseating form. It was long supposed to be secondary to pulmonary tuberculosis, but the opinion has been steadily gaining ground that the glands in children are more apt to be the seat of primary infection than of secondary. Biedert, in 1884, collected 84 cases of primary bronchial-gland tuberculosis in children as against three secondary to pulmonary or vertebral tuberculosis. Not only this, but the bronchial glands may show tuberculosis in the absence of any other focus of infection. Steiner and Neuretter found tuberculous bronchial glands in 275 out of 302 children, and in 36 of these there were no other tuberculous organs. The bacilli in such cases enter by the respiratory tract and follow, according to Weigert, the route taken by the coal dust, lodging in the bronchial glands, as is evidenced by the frequency of tuberculosis in these glands compared to the mesenteric. Berthelot found the proportion to be as 20 to 1.

Tuberculous infection in these glands may remain latent for long periods of time and indeed may never give rise to demonstrable lesions. The inoculation experiments of Loomis showed that six out of fifteen persons dying of acute infectious diseases, and in whom no tuberculous lesions were found post mortem, had living tubercle bacilli in their bronchial lymphatic glands. Loomis' work is open to criticism as he used no control animals; but the same cannot be said of Pizzini, who used all possible precautions and yet succeeded in proving the presence of tubercle bacilli in the bronchial glands of forty-two per cent. of healthy non-tuberculous adults dying from accident, suicide, or acute infectious diseases. According to Weigert, this latency of the germs is to be explained by closure of the outgoing lymphatics; according to others, by the slight tendency to caseation in the bronchial glands which are already fibrous and pigmented.

An appreciable amount of enlargement may be found post mortem, although no symptoms were observed during life; but usually there are symptoms of contraction, or pressure or inflammation. The tuberculous glands may become adherent to the bronchi, œsophagus, pericardium, or large vessels, and may open and discharge into these, as in a case reported by Powell in which a scar was formed at the site of a perforation of the trachea; or they may cause compression, as in Pitt's case of enlarged and caseated posterior mediastinal glands which compressed the trachea and right bronchus. Parker, Gulliver, Goodhart all report cases of sudden death from dyspnœa caused by the rupture of a caseous gland into the trachea or into a bronchus; Malsin, Coupland, and Gee report death from dyspnœa caused by compression of trachea or bronchus. More rarely rupture is into the mediastinum. Secondary infection from these glands involves the lungs, the pleuræ, pericardium, and perhaps most commonly the meninges.

The symptoms of tuberculosis of the bronchial glands with enlargement and cheesy degeneration are: vague pain which is apt to be at the level of the fourth dorsal vertebra on one side or both with tenderness on pressure. Rarely the pain is substernal ("pain in the stomach"). Or there may be simply a feeling of pressure without actual pain. The cough is paroxysmal, like that of whooping-cough, but differs from it in its non-infectious