animal is now 4 years old, it assumes the form of the perfect insect, and is capable of reproduction. The later moulting falls in the summer time, and so does fertilization and oviposition.

Male cockroaches may be distinguished from the females by their well-developed wings and wing covers. They stand higher on their legs than do the female, whose abdomen often trails upon the ground. In spite of the noxious secretion of their abdominal glands there are creatures who habitually feed on cockroaches—hedgehogs, for instance, are frequently imported into our houses to check these pests. Rats, cats, polecats, frogs, and wasps have been known to eat them, and some few of the digging wasps lay them down in their larders for the use of their progeny. Some birds will also tackle them. But even the most devoted friend of the cockroach can find little to say in its favour, except that it is currently reported to form the basis of the flavouring of a very popular sauce, but even wild cockroaches will not drag from me which particular sauce that is.

THE INCIPIENT PULMONARY PHTHISIS OF SCHOOL CHILDREN.

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I.

INTRODUCTION.

In the present communication the discussion will be mainly limited to the ages between 5 and 15, when the tuberculous process tends to become localized within the ducts and glands of the lymphatic system of the respiratory organs. This is practically the time of school life. During the earlier period of childhood, and particularly the first and second years—that of the first dentition—the conditions differ; the lymphatic system remains embryonic; its channels appear wider, and less able to prevent dissemination into the blood and distant areas.

Fig. 1.—Diagram of the lymphatic glands concerned, modified from Sukienikow. T.Br., Tracheo-bronchial. B., Bifurcation. H., Hilum, and P., pulmonary glands. Above the tracheo-bronchial group is a tracheo-bronchial chain of glands on each side of the trachea, reaching as high as the thyroid gland.

In other words, children of this age are practically defenceless, and the little resistance they may possess must be ascribed to increased efficiency of the leucocytes, since the opsonic indices are negligible.

The radiological appearances of the healthy infant's chest consist of a few indistinct bronchial streaks, radiating from the hilum towards the periphery. The hilum opacity and its ramifications become more obvious as age advances. Constant irritation of the walls of the lymphatic vessels, by particles, organized or inanimate, leads to a small-cell infiltration and a thickening of perivascular and peribronchial trunks—a process which emphasizes the central and arborescent opacities of the radiogram.

II.

ANATOMICAL AND PATHOLOGICAL CONSIDERATIONS.

Bartels¹ divides the glands into four categories: (a) Tracheo-bronchial, (b) bifurcation, (c) hilum, and (d) pulmonary glands.

(a) *The tracheo-bronchial glands* lie on each side of the thoracic portion of the trachea and on the outside of the extrapulmonary bronchi. They number from five to nine on the right, and from three to four on the left. Normally each is about the size of a large pea. The right glands lie nearer to the margin of the sternum. They receive branches from (b) and (c), from the posterior mediastinal glands, and from the trachea and bronchi. They send efferents to the broncho-mediastinal trunk on the right, to the thoracic duct on the left, and to the deep cervical glands (supra-clavicular). Since they lie at and above the level of the bifurcation of the trachea, they are situated in the superior and just above the middle mediastinum. In front of the right tracheo-bronchial glands lies the superior vena cava; behind, the right vagus; internally, the trachea; externally, the internal surface of the right lung; below lies the right bronchus, the right pulmonary artery and the terminal bend of the azygos major. The left tracheo-bronchial glands lie behind the ascending arch in front of the left vagus; above is the horizontal arch and left recurrent nerve; externally is the internal surface of the left lung. This group of glands is connected with the lymphatics of the upper and middle lobes of the lung.

(b) *The bifurcation glands* are ten to twelve in number and more closely related to the right than to the left bronchus. They lie between the pericardium and left auricle on the one hand, and the oesophagus, aorta, posterior pulmonary plexus and posterior borders of both lungs on the other. They receive ducts from the hilum glands and send branches to the tracheo-bronchial. They are connected with the lymphatics of the middle and lower lobes.

(c) *The hilum glands* lie buried in the pulmonary parenchyma at the root of the lung and occupy the angles of the first four divisions of the bronchi. They are in intimate connexion with branches of the pulmonary artery and receive afferent vessels from the pulmonary glands.

(d) *The pulmonary glands* lie outside the hilum in the substance of the lung at the divisions of the bronchi. Minute glands are even found just beneath the visceral pleura.

Surface Anatomy.—The trachea bifurcates at the level of the upper border of the fifth dorsal vertebra posteriorly, and about the junction of the manubrium with the sternum anteriorly. The extrapulmonary right bronchus is nearly vertical and reaches to the level of the upper part of the sixth vertebra. It gives one cparterial branch close to the hilum. The left extrapulmonary bronchus ends at the same level as the right. Each hilum is about 1½ inches broad and extends on either side at the level of the vertebral ends of the fifth, sixth, and seventh ribs. Within it are the branches of the bronchi, pulmonary artery and veins, bronchial arteries and veins, lymphatics and lymph glands, nerves and connective tissue. Robinson² gives three excellent figures of the mesial surface of the lung with sections of each hilum, showing the arrangement of these structures. The pulmonary artery and veins possess branches corresponding with the bronchi. After the cparterial branch is given off on the right side, the stem sends a ventral branch to the middle lobe. The tracheo-bronchial glands lie at the level of the vertebral ends of the third, fourth, and fifth ribs; the bifurcation at the level of the sixth vertebral extremity, and the fifth spine is dull from this cause and from the proximity of the right pulmonary artery (Ewart³). The apices of the upper lobes reach the level of the first rib; the apices of the lower lobes that of the vertebral ends of the fourth rib, but the left lower apex is a little higher (Ewart⁴). In front the middle lobe of the right lung occupies the third and fourth intercostal spaces. In front of the root of the lung lie the anterior pulmonary plexus and the phrenic nerve.

The lymphatics commence in irregular lacunae situated just beneath the walls of the air sacs; and also below the surface of the pleura. They form fine networks around the lobules, the bronchi, and the blood vessels. Superficial collecting trunks arise from the subpleural lobules and deep collecting ducts from the veins, arteries, and bronchi.

These trunks communicate. The lymphatics of the visceral pleura also empty into the superficial collecting vessels. Finally they all reach the hilum. The pressure within the vessels is not more than 15 mm. of mercury, but they possess valves which open only towards the hilum. The subpleural plexus communicates with the pleural cavity by means of stomata, and with the inter-alveolar lymph vessels. There are numerous adenoid nodules in the deeper parts of the bronchial mucous membrane. With regard to the minute anatomy, the lymphatics of the lung often contain large mononuclear phagocytes conveying carbon particles within their interior. In sections of the lung leucocytes are often visible within the alveoli (Schäfer⁵). Carbon pigment is taken up by these cells in the alveoli and transported through the lymph spaces towards the hilum. Sometimes they are caught on the way and produce proliferation of connective tissue, which may embarrass the lymphatic drainage. The endothelial leucocytes are more important as phagocytes than the polymorphs.⁶ They are not transient but normal scavengers. Beitzke thinks the pigment phagocytes are sometimes derived from the alveolar epithelium. Probably they remain for some time within the alveolus, at any rate, until they are well loaded with granules. Tubercle bacilli will be incorporated by these cells in a similar manner.

Post mortem the glands exhibit four stages: (1) Inflamed pink; (2) enlarged grey, studded with minute tubercles; (3) caseous, containing a cheesy deposit; and (4) calcareous, containing an accumulation of lime salts, with fibrous matting, due to periadenitis, uniting them to neighbouring glands, nerves, bronchi, and vessels.

III.

CHANNELS OF INFECTION AND DISSEMINATION.

The great channel or portal of entry is undoubtedly that of the respiratory tract. Bacilli enter with inspiration, the majority settle on the mucous membrane of the trachea, larger and smaller bronchi, and are usually driven outwards again by the activity of the ciliated epithelium. But some permeate the smallest bronchioles and the alveoli, becoming scattered freely over the periphery, and being transported thence along lymphatic trunks towards the hilum. Dissemination, as a rule, takes place gradatim, from one gland to the next in accordance with Cornet's⁷ law of localization. In spite of repeated investigations, undertaken with the idea of repudiation, this law still holds good. Some authorities have even denied the aërogenous origin of anthracosis in the hilum glands. Scepticism could scarcely be more obstinate, since pigment is never found in the mesenteric glands.⁸ After the ingestion of tuberculous food, the mesenteric glands may be attacked; after infection of the mouth, throat, and tonsil, the cervical; after abrasion of the limbs, the axillary and inguinal glands may become enlarged if the wound becomes infected with tuberculous material. Mesenteric infection under normal circumstances can only affect the bronchial glands indirectly—that is, by way of the thoracic duct, reaching the lungs through the pulmonary artery (Most and Beitzke⁹). It has been suggested that bacilli may enter the fetus from a tuberculous mother, penetrating the placenta, and pass a larval existence within the lymph nodes and the bone-marrow until favourable conditions obtain for future development. This could only be possible in the presence of tuberculous foci within the placenta itself; otherwise, we might relegate infection to the Garden of Eden—an obvious absurdity. Moreover, the newly-born do not react to tuberculin.

The child is really exposed to an infection, which may enter either by the respiratory or by the alimentary channel. Consequently mistaken conclusions are liable to be made as to the manner of infection and dissemination. There appears, however, to be no reason why bacilli should not be arrested within the lymphoid tissue of any one of the terminal bronchioles or in one of the pulmonary glands intervening between the alveoli and the hilum, and there lie dormant until an opportunity arises when they might, if superficial, infect the pleura.

There is much difference of opinion as to the manner in which the bacilli reach the bronchial glands, and as to whether a primary tuberculous affection of the glands really exists. Gohn¹⁰ has made a careful analysis of *post-mortem* investigations in the pulmonary tuberculosis

of children, and finds in general one, at times two, primary foci within the lung itself, situated just beneath the pleura. The focus is most frequently found in the upper, anterior, and inner half of the right and left upper lobes; behind, chiefly at the base and apex of the left lower lobe, and apex of the right lower lobe. Sometimes the focus is found on the mesial surface, especially at the right base. According to Gohn the hilum, bifurcation, and tracheo-bronchial glands are affected secondarily, even although the foci in these situations may be much larger than those within the lung itself. In 1898 Küss described a form of pulmonary tuberculosis in children which he recognized *post mortem* by the occurrence of small subpleural tuberculous foci. The condition might remain stationary for years or become entirely arrested. Gohn is a firm supporter of the aërogenous origin of the pulmonary foci, and can find no trace of retrograde infection. These pulmonary foci, however, are not necessarily primary, since their existence and formation may be due to conduction along other lines of transportation. By direct continuity from a tracheo-bronchial or bifurcation gland an infection—which has entered these structures through the mucous membrane of the trachea—may pass directly to one of the lobes of the lung, and induce the formation of a pulmonary focus. Gohn's investigations afford—by the proof of the very common occurrence of an extensive tuberculous pleurisy, particularly affecting the upper lobes, the hilum, and the base—a third method of dissemination which is well seen in the radiogram of Phyllis P. (Fig. 3), and will be described later. It remains for the radiologist to localize these pulmonary foci where possible, and to corroborate their existence by *post-mortem* control.

When the infection has reached the hilum and established itself within its glands, as well as in the tracheo-bronchial and bifurcation glands, the bacilli have proved victorious over the leucocytes, and have commenced to multiply. The result is the formation of tuberculous foci within the glands, consisting of epithelioid and giant cells. All the groups of glands appear liable to become involved, but in varying degrees. The tracheo-bronchial and bifurcation glands may be infected directly, that is, without implication of the lung, if the *materies morbi* penetrates the mucosa of the respiratory and alimentary tracts lying immediately adjacent to them (Walsham¹¹). The infection may be disseminated by simple continuity from the tracheo-bronchial and bifurcation glands, to the upper and lower lobes, and hilum, and subsequently infiltrate the lung.

There are several possibilities:

1. *Caseation of Glands.*—The glands may caseate, calcify, and becoming surrounded by a fibrous periadenitis, the process may be arrested or completely healed. Several glands may become matted together and form a prominent object within or outside the hilum. In the healthy adult it is quite common to see a few small fibrosed glands at the root of the lung.

2. *Caseous Pneumonia.*—The bacilli may escape through the capsules of the glands and induce a slow infiltration in the middle and lower lobes (caseous pneumonia). Walsham¹² described several instances in children where the disease was found to be confined to the parts of the lung, particularly the middle lobe, in immediate contact with the softened bronchial glands. There is a gland on the right side of the trachea—one of the right tracheo-bronchial group—which is often found to be caseous (Still).¹³ "From this gland the caseous process may pass directly by continuity to the adjacent pleura, forming local adhesions and spreading into the right lung towards the apex." No doubt the infection may spread by continuity from the tracheo-bronchial glands upwards along the chain into the neck, implicating amongst others the deep cervical glands. It has been stated that the thoracic glands may be infected from the abdomen by way of the hepatic and prepericardial glands.¹⁴ But this must be very exceptional.

3. *Irruption into a Bronchus.*—The bacilli may pass from the glands into the walls of the bronchi, particularly behind, where the cartilage is deficient, enter the mucosa, producing small tubercles, which subsequently ulcerate. The currents of air will then aspirate them into adjacent or even distant parts of the lung. "It is not uncommon for one of the diseased glands to perforate a bronchus, and if this occurs beyond the first division of the bronchus

the caseous debris of the gland may be discharged into one lobe only of the lung and set up an acute tuberculous process, sometimes with rapid cavitation throughout the whole of that lobe."¹⁵

4. *Irruption into a Blood Vessel.*—As a result of peradenitis and the formation of adhesions to an arterial or venous twig bacilli may enter the vessel and induce in the former case a localized miliary tuberculosis in the arterial area of distribution; in the case of the vein a wider dissemination takes place.

5. *Lymphatic Dissemination.*—In the more chronic cases, where resistance is sound, the infection may apparently spread centrifugally along the lymphatic sheaths of the vessels and bronchi to distal parts of the lungs, inducing a thickening and thrombosis in its course. For many years there has been a current belief that a lymphatic tuberculosis may pass along the ducts in a centrifugal direction opposed to the centripetal course of the lymph itself (retrograde flow, retroimpulsion). It has even been asserted that the lymphatic circulation is carried on in a manner not analogous to that of the blood. There is said to be a backward and forward flow of lymph within the chest, and this phenomenon has been invoked in order to explain the facts of dissemination within the lungs. If the flow is of this nature, it can be of little service as an effectual drain. Moreover, the presence of valves is forgotten. When a small lymphatic is blocked, a collateral circulation will be established, just as occurs in the venous system when a vein is thrombosed. It is really unnecessary to assume the existence of a normal retrograde current. It must be of infrequent occurrence, and the facts may be just as well explained by a dissemination along the walls of these vessels. Although the bacilli themselves, being immobile, cannot swim against the stream, the leucocytes containing them possess this power, and they can also creep along the intima and perforate it. Perhaps it is too dogmatically assumed that all the lymphatic striae on the radiogram are produced centrifugally. They may be formed early and in a centripetal direction from the alveolus towards the root. Since blocking of the ingress to the hilum glands would produce an engorgement and stasis in the implicated areas of the lymphatic circulation, even as far back as the alveoli themselves, a repetition of an aërogenous infection would then be more likely to lead to irritation and thickening of the walls (tuberculous lymphangitis). In some middle-aged healthy individuals radiograms show extensive mottling and numerous lymphatic striae, the latter sometimes reaching from the apex to a gland in the hilum (Stürtz). It is quite feasible to regard these as produced centripetally, as well as centrifugally. At the extreme apical end a few obsolete tubercles may be seen. If the affection had spread from the hilum in the reverse direction, and the disease was chronically progressive, why remain at the apex *in statu quo* for a couple of decades?

It is unreasonable, however, to deny the tendency of a hilum infection to extend outwards into the lung. Several methods by which such dissemination may take place have already been described. But it is never possible to exclude the incidence of second or even multiple aërogenous infections. Very many adults possess dense hilum opacities, containing cirrhotic or calcified glands, which must have been originally tuberculous. The infection, therefore, appears to be practically universal and the manner by which it has been acquired is naturally often obscure. Subsequent infections may just as readily occur. A second aërogenous infection might, however, not be so easily transferred to the hilum. If we accept Birch-Hirschfeld's¹⁶ conclusions with regard to the adult—namely, that there exists a particular area, in the upper lobes, of increased danger, produced after puberty by mechanical means, a "summation of bacterial supply" to this region might strain the leucocytic and bactericidal defences, and the nearest subalveolar glands would provide a suitable nidus for development, particularly if the deeper lymphatic channels should be closed. In this manner succeeding aërogenous infections would be more certain to infiltrate the lung. For a time the disease would remain apical, and the sequence would depend on the individual resistance. If there be a coexistent smouldering focus or bacillary residuum within the hilum glands, or if the second infection accidentally should also be carried thither,

the old infection would be more likely to burst into flame. It is difficult to see how the principle of centrifugal dissemination can explain the preponderating vulnerability of the apical lobe in adults. There is no anatomical design in the lymphatic system which would explain the fact. Moreover, the additional difficulty remains of determining the priority of an infection. It is possible to decide whether one focus is more advanced than another, but difficult to say which is primary, as the one may progress more quickly. And if we seek for an older lesion by the side of a new one, the search is often successful (Hutinel).¹⁷

In many instances positive proof can be afforded of the occurrence of second aërogenous infections after puberty. Von Pirquet¹⁸ states "that if an organism has once become infected, it has altered its reactivity towards the tubercle bacillus and its products. It has become allergic. The bacilli can no longer penetrate without hindrance the mucosa and reach the lymphatic glands, but they produce at the portal of entry inflammatory changes which usually heal. This pulmonary form, typical of adults, and found rarely in children, is therefore most probably a reinfection in individuals who have become allergic through a pulmonary infection in childhood." The facts are equally well explained by accepting Birch-Hirschfeld's views and by assuming the existence of stenosis within the lymphatic trunks. Von Pirquet adds, "the interesting statement that no tuberculous child reacts to tuberculin for a definite period—about a week—during an attack of measles and probably also of influenza." During this time the individual is anergic and deficient in necessary antibodies. A mobilization of the bacilli within the hilum takes place leading to an advance. At such a time, both in children and adolescents, a reinoculation may take place at the periphery.

IV.

CLINICAL SYMPTOMS AND SIGNS.

These are at first obscure, indefinite, and due to a slow and chronic absorption of the products of bacillary metabolism and to the compression exercised upon adjoining structures by enlargement of the glands. The toxæmia induces a delicacy of constitution, a debility, and disinclination for exercise; a loss of appetite and weight, anaemia, and myoidema. Such children are said to "catch cold" easily, and often suffer from "bronchitis." Where there is no apparent cause for feebleness of health, such as adenoids, enlarged tonsils, etc., suspicion should be directed to a possible affection of these glands. There may be swelling of the cervical, axillary, mesenteric and inguinal glands. The statement has been made that blonde children with long dark eyelashes and a blue iris are especially vulnerable.¹⁹ There may be pain, difficult of localization, within the chest on deep inspiration and vigorous exercise (pleuritic?). Some children exhibit scrofulous scars, otorrhoea, rickets, flat-foot, and spinal disease. Many are said to be "threatened with consumption."

Signs of pressure are not obvious in early cases. Cough and congestion of veins over the chest and upper three or four dorsal spines are usually present. The capillary veins over the spine are, without doubt, produced by compression of the superior intercostal veins by enlarged tracheo-bronchial glands.²⁰ Lombardi²¹ designates this area "the varicose zone of alarm." Tenderness over the dorsal spines,²² or spinalgia, to firm pressure may be present (first to eighth). Petruschky²³ ascribes it to disturbances of collateral circulation, to periosteal irritation, or to the presence of minute tuberculous foci within the spine. It is probably a referred pain, produced on irritation of the mediastinal nerves by the enlarged tracheo-bronchial and bifurcation glands. Noeggerath and Salle²⁴ found hyperalgesia in the region of C₄, D₂, and D₃ (Head's zones), and adduce this as a proof of incipient bronchial gland tuberculosis in children. They mapped out the areas by stroking lightly the skin with a smooth metal pencil or a blunt needle. There may also be tenderness on pressure in the interspaces.

Cough is often absent or present only at night. It may be intermittent and paroxysmal, due to pressure on the mucous membrane of the trachea at its bifurcation, or to irritation of twigs in the pulmonary plexuses. The vagi

* Are not these attacks due to exacerbations produced by the fluctuating size of the glands, by gradual involvement of neighbouring glands, and by periodical oozing of toxin?

carry both inspiratory and expiratory afferent fibres from the lungs to the medulla (Starling²⁵). There is no sputum. There may be dyspnoea and asthmatic attacks at night, the latter produced by irritation of afferent or efferent vagal branches.²⁶ French²⁷ thinks the chronic cough in town children may be due to irritation of the right phrenic nerve.

The temperature curve may be normal. It is often subnormal, with occasional fluctuations to 100° in the afternoon or evening. Cornet insists on a two-hour chart. Rest and exercise temperatures are necessary. Night sweats may occur. Pressure on the cardiac branches may induce a slowing of the heart (Dautwitz²⁸), but the usual condition is an increased rate, due to irritation of sympathetic fibres.

Physical Signs.

Riviere²⁹ describes these fully. Briefly they are:

(a) *Impairment to light percussion* at one apex, often the right, in front and behind, sometimes dullness over manubrium, the interscapular region, an increased oval interspinous area (Ewart), due to enlargement of tracheo-bronchial glands, and dullness over the fifth and sixth dorsal spines (bifurcation glands).

(b) *Auscultatory changes* produced by increased conduction are harshness of breath sounds over apices, with a prolonged expiration and increased vocal resonance; weak inspiration, possibly over the rest of the lung; vertebral bronchophony, with whispering echo on repeating the number 33, as far down as fourth and fifth dorsal spines,³⁰ or even lower still. Fine crepitations are to be heard occasionally at the apex and within the middle and lower lobes. They may be produced by a condition of slight oedema occurring within the air cells as a result of lymphatic and venous congestion.

V.

RADIOLOGICAL EXAMINATION.

Technique.

1. The little patient lies flat on the couch, and perfectly straight (in order to keep the manubrium and spine in the middle line), and the head is turned a little to one side. A small piece of lead is fastened to each nipple and the target is arranged midway between. It is always preferable to radiograph each child in the same way. The tube is of medium hardness (Bauer 6-7) and a moderate current is employed (2-3 milliamperes).

On the screen several points are noted:

- The movements of the diaphragm.
- The appearances of the apices on deep inspiration and coughing.
- The hilum opacities.
- The pulmonary fields, especially the second and third anterior intercostal areas during inspiration.

The screen is then raised 1 to 2 feet above the patient and the hilum again examined for glands.³¹ At any selected area, the diaphragm is focussed down. When the screening is finished, with the target in the original position, the diaphragm is opened wide and a radiogram taken on an ordinary plate, making use of a soft tube (6-7 Bauer), a current of 10 milliamperes, and an exposure of about 10 seconds. During this time the little patient fixes the chest in deep inspiration.

2. The patient then lies on the face, and similar precautions are again observed. The target is moved upwards to strike a small piece of lead on the fourth dorsal spine, opened out, and a second radiogram is taken as before. This will be a ventro-dorsal view.

3. The patient is then turned into the right oblique or semilateral position, in order that the mediastinum behind the aortic arch may be explored.

4. In cases with pronounced arborization stereoscopic plates are useful.

Examination of the Plate.—1. This shows the central mediastinal shadow, the hilum, and the lung fields. In order to explain the parasternal shadows I have examined several cases of lymphadenoma of the thorax. In

Fig. 2, taken from a boy aged 17, the intrathoracic glands are much enlarged. A diffuse homogeneous shadow is seen on each side of the upper mediastinal opacity extending from the vertebral ends of the third to the sixth rib. It is due to the enlargement of the tracheo-

Fig. 2.—Radiogram 1, Lymphadenoma of chest.

The letters in the radiograms are similar. T.B., Tracheo-bronchial opacity. T. Bg., Tracheo-bronchial gland. B., Bifurcation opacity. B.G., Bifurcation gland. H., Hilum opacities. 1S., 2S., etc., Sternal ends of ribs. 3V., 4V., 5V., etc., Vertebral ends of third, fourth, fifth ribs, etc. C., Clavicle. H.A., Heart apex. R.A., Right auricle. D., Diaphragm. L.N., Left nipple. R.N., Right nipple. R.B., Right bronchus. L.B., Left bronchus: A pleuritic shadow arising from tracheo-bronchial opacity.

bronchial glands, and may be termed the "tracheo-bronchial" or "superior opacity." It is visible in many cases of pulmonary tuberculosis in children and adults as a fine shadow, outside the sternum, sometimes on both sides, and at times more easily detected in a ventro-dorsal plate. It has been stated by many pathologists that of these three sets of glands those at the bifurcation are the most frequently attacked. They lie on the body of the fifth dorsal, a little more to the right than to the left of the middle line. A shadow just below the level of the vertebral end of the sixth rib, running directly into the hilum and obliterating the narrow isthmus which often intervenes between the hilum and the cardiac opacity, must be produced by congestion of these glands. It may be termed the "intermediate" or "bifurcation opacity." The hilum glands are so commonly affected, on the radiogram, that it makes one doubt the statement of the particular vulnerability of the bifurcation glands, and surmise that in some instances the condition of the hilum glands has been overlooked.

2. *The interspaces*—particularly the first, second, and third—are examined for signs of lymphatic mottling (which is perivascular as well as peribronchial), tuberculous infiltration, and pleurisy. Sometimes these spaces, although becoming more translucent during inspiration, seem, on the screen, to be veiled by a delicate haze or dimness, suggesting either a very close interlacement of lymphatics or a fine uniform but intangible pleuritic shadow. After a focal reaction, the lymphatics as well as the blood vessels would be congested and the interweaving more pronounced.

3. *The hilum* is now carefully observed for signs of swollen, caseous, and calcified glands, the density of the shadows increasing in this order. The hilum opacity lies at the level of the vertebral ends of the fifth, sixth, and seventh ribs. If it is abnormally dark, broad, and contains a large number of circumscribed shadows, these are enlarged and diseased glands. If the hilum is broad, irregular in outline, and uniformly diffuse or cloudy in appearance, the process is active. The branches running to the diaphragm should be carefully followed for signs of tubercles or localized patches of tuberculous bronchopneumonia.

4. *The sternal ends of the osseous ribs* are examined for signs of calcification, especially the first. In a young subject, if these edges are sharply cut and there is some

calcification of the first rib cartilage, the prognosis is more favourable. At the same time the width of the interspaces on each side is compared. Narrowing of these spaces on one side due to falling in of ribs produces a unilateral drooping of the shoulder. It leads to deficient expansion and weakened breath sounds.

5. The heart in older children may suggest cardiopneumosis (pendulous heart). It is apparently smaller than normal in some instances. There may be a convexity on the right side of the heart, due to a dilated right auricle—although this may in some measure be due to the negative pressure within the chest during a fixed inspiration (Walsham).

6. The pulmonary fields are to be explored for striae, networks, and small tubercles or tuberculous foci. In itself mottling is a sign of phthisis, but not of activity, since it may be merely evidence of a healed tuberculous lymphangitis. Morton and Owen³² think that mottling is due to the crossing shadows of thickened lymphatic vessels which are on different levels. The coarser the interlacements are, the more suggestive is the picture of an arrested lesion (Boardman³³). But the occurrence of

Fig. 3.—Radiogram 2, Phyllis P., aged 10 years.

small pinhead-sized foci standing separately or in conglomerate groups, producing a cloud effect, is diagnostic of activity (Rieder,³⁴ Morton³⁵). If they become fibrosed, the opacities will be denser and more sharply defined.

Interpretation.—Before attempting a diagnosis, the family, the personal, and clinical history, symptoms and physical signs should be examined in order to avoid errors and to command a more reliable prognosis. The radiologist endeavours to make a diagnosis:

1. As to the presence or absence of the disease (von Pirquet's reaction may here assist if it should be negative).
2. As to its activity or latency.
3. If active, as to whether it is open (manifest) or closed.
4. As to the probable direction or otherwise of its dissemination (prognosis).

In cases of difficulty the occurrence of an increased lymphocytosis after the administration of tuberculin may suggest a positive diagnosis as regards activity (Schulz³⁶). Or a second or third radiogram may be taken at intervals of a month.

The histories of two cases are now given:

CASE I.

A girl, aged 10 years; family history good; had measles and a septic throat four years ago; has a chronic cough; suffered from pains in chest—pleuritic?—four months ago.

Present Condition.

Cough little but hard; no sputum; no night sweats; no dyspnoea; no palpitation; a well nourished child. Examination reveals diminished expansion of left upper lobe, harsh

breath sounds and prolongation of expiration in this locality. At the right apex breath sounds are weak; vocal vibration diminished; dilated capillary veins over the spine. Temperature 97.8° mornings, 98.8°-99.5° evenings; spleen and liver normal; occasional attacks of vomiting; glands not palpable.

Radiological Examination.

Diaphragmatic movements good; both lungs appear dull, but light up well during inspiration, also the apices; increased hilum shadows. On the plate (Fig. 3) increased hilum opacity and arborizations; right tracheo-bronchial opacity containing one visible gland, bifurcation opacity, and gland visible on right; lymphatic mottling within upper interspaces with scattered tuberculous foci. Shadows and strands passing to right diaphragm are dense. Cardiopneumosis and dilated right auricle. The condition is active.

There is a dimness, radiating from the right tracheo-bronchial shadow, which is due to a fine pleuritic infiltration. Opacities are visible along the lower borders of vertebral ends of fifth and sixth ribs. The inner strands of Stürtz, running upwards and backwards towards the tracheo-bronchial glands, may be produced gradually by a centripetal dissemination from the pleuritic infection intervening between the glands and the striae. We consider that the subpleural foci of Gohn may arise through the medium of the tuberculous pleurisy, which has spread over the visceral pleura from affected tracheo-bronchial

Fig. 4.—Radiogram 3, Joseph Y., aged 12 years.

and bifurcation glands. Such a pleurisy may be ultra-clinical, but none the less effectual as a means for the dissemination of tubercle from paratracheal glands to the substance of the lung.

CASE II.

A boy, aged 12 years. Complaints of cough, shortness of breath, and general weakness; has always been delicate.

Present Condition.

Complexion is pale; tonsils enlarged (scraping is negative); glands in axillae, along sterno-mastoids, in anterior and posterior triangles; they are firm and discrete; spleen just palpable; blood count, 4,240,000 red; white 10,800; haemoglobin, 80 per cent.; polymorphs, 53 per cent.; lymphocytes, 47 per cent.; small, 36 per cent.; lymphocytosis; pulse 76-96; respiration 20-24; temperature, morning average 97.6°, evening, 98°. Chest expansion moderate; abdominal respiration increased; percussion note over left upper lobe in front and behind is impaired; breath sounds at apices harsh and expiration prolonged; vocal resonance increased; over the rest of lung the breath sounds are weak and vocal resonance is diminished. The radiogram (Fig. 4) shows massive shadows within the hilum on both sides; a right tracheo-bronchial opacity with a shadow just outside between vertebral ends of fourth and fifth ribs, which may be a pulmonary focus in right upper lobe; a right bifurcation shadow internal to the large right hilum opacity. There is not much mottling and no signs of activity. In the oblique radiogram some of the calcified glands are seen to lie along the left internal mammary vessels; the left axillary glands are also visible. The channel of infection appears to have been from the trachea via the tracheo-bronchial and bifurcation glands.

The coexistence of the five conditions:

1. Intermittent sudden cough in the absence of cases of pertussis;
2. Wasting, pallor, and tiredness;

3. A subfebrile temperature, rising occasionally to 100°;
4. The presence of capillary veins and a growth of hair over the upper thoracic spines; and
5. The absence of any obvious disease;

should induce the school medical officer to examine carefully for pulmonary tuberculosis and enlarged bronchial glands. If in doubt the little patient should be sent to the radiologist. The most difficult glands to materialize are the left bifurcation glands.⁸⁷ Special contrivances must often be adopted to bring them, as well as the tracheo-bronchial glands, into prominence.

VI.

TREATMENT.

If there is the slightest doubt, the patient should receive the benefit. All hygienic measures—such as proper diet, graduated exercise, sea air or sanatorium, open-air school or entire freedom from school routine for several months—should be adopted. Virol, red bone marrow, and cod-liver oil are said to increase the bactericidal powers of the blood. Ichthyol⁸⁸ (tabloids) and calcium sulphide are believed to hasten the fibrotic changes we desire; iron is useful to tone the erythrocytes, which, according to Spengler, manufacture the immune bodies. With regard to the curative value of the several varieties of tuberculin, the special treatises must be consulted. On the analogy of the cervical glands, the x rays may be suggested, making use of a hard tube and of filters, and treating a circular area around the spine of the fourth dorsal vertebra with a third of a pastille dose once a week.

A radiogram should certainly be taken first in order to localize the situation of the affected glands.

In conclusion, my best thanks are due to Dr. Walsham, who provided me with several typical cases and assisted me throughout with valuable advice.

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LIEUTENANT-COLONEL ANDREW ARTHUR MACROBIN, R.A.M.C., left estate valued at £13,335.

IN the *Revue Médicale de la Suisse Romande* there is a note from the President of the Swiss Medical Commission stating that certain members of the medical profession have taken advantage of the absence of doctors called out for service with the army to establish themselves within the area of practice of their absent brethren. The President calls upon all the cantonal medical societies to do whatever lies in their power to safeguard the interests of the mobilized practitioners. He deplores the fact that men capable of such conduct should be found in the ranks of the profession, and expresses regret that there are no means at present available to prevent such action.

THE "ELDERTON" METHOD OF CONTROL IN TESTING THE VALUE OF NEW TUBERCULIN.

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PROFESSOR SAHLI has published a criticism¹ of the way in which an endeavour has been made by myself and fellow-workers to arrive at definite conclusions as to the therapeutic value of tuberculin. Such criticism is the main topic of his contribution; but he has also referred to other matters in which we evidently are at variance, and I feel it is due to Professor Sahli that a reply should be made, not only because of the extreme interest of the arguments he puts forward, but also because his point of view is different from the one which, I think, activates practice in this country.

The first duty is to give answers to some of Professor Sahli's remarks: that I have "not the least chronological sense" in reverting "time after time to the experiments of Koch"; that I am wrong in assuming that "no one bases himself now . . . on these far-distant experiments"; that "there is, therefore, a grave injustice" in my "comparison between the quack doctor's lack of criticism and the similar lack which" I assume "in the tuberculin therapist"; that I see "only harmful action in these anaphylactic phenomena"; that my view, "that in anaphylactic phenomena . . . no curative side is to be recognized, should find little approval"; that "the significance of the modern reactionless tuberculin treatment appears to be little recognized" by me.

Professor Sahli does "not think that any thoughtful adherent of tuberculin treatment can nowadays be found to contend that tuberculin produces immunity against tuberculosis." It is an "old argument, which is scarcely used nowadays, that tuberculin treatment is an immunization, and that from the possibility of a protective inoculation against typhoid . . . it might be concluded that such an inoculation must also succeed in the case of tuberculin."

I am sorry that Professor Sahli thinks I have failed in the chronological sense. This is my answer: that I went to the fountain source of Koch's experiments under date of 1890, that I then read Löwenstein's and Römer's articles in Kraus and Levaditi's *Handbuch*, 1908; Cornet's *Die Tuberculose*, 1907; Bandelier and Roepke's *Lehrbuch*, 1908; and Wolff-Eisner's *Handbuch*, 1910, which I had hoped had brought all the experimental work on tuberculin up to that date.

It is my misfortune if I have missed any reference to the experimental proof of the specific cure by means of tuberculin of any animals since the year 1910. At the time of delivering my lecture none of my friends in this country who are following the matter with the greatest acumen knew of any further experimental work supporting Koch's original contention (reiterated by authorities abroad as shown above, and shown by Römer to be wrong) that tuberculin could cure animals of tuberculosis. I submit to Professor Sahli that his charge is not justified, and I fail to find in his own publication any reference to further experimental cure. I am obliged to revert to Koch's experiments, as they are the only ones which it is stated prove the specific curative value of tuberculin. The advocates of the use of tuberculin also revert to these experiments repeatedly as a foundation for their practice.

Now as to the grave injustice done to the tuberculin therapist. As shown in my lecture, all the experimental proof put forward by Koch and reverted to by modern workers is of doubtful value, and Professor Sahli joins me in saying Koch's experiments have never been confirmed. Then I submit that a tuberculin therapist who, in face of the inability to repeat and confirm Koch's experiments, says that he can cure tuberculosis of the lungs or any other organ in human beings by means of tuberculin, lays himself open to the charge of quackery—it makes no difference whether he is a qualified practitioner or not. The injustice so severely commented on by Professor Sahli can only be felt by those who say they "treat" rather than "cure" the disease. My lecture was