Contamination from the Lash Area.

The last point I wish to mention is contamination from the lash area, which is the neighbourhood most difficult to preserve clean when an eye has to be kept bandaged for any length of time. It is not, I am thankful to say, the most important, because there is not the same risk of infection of the operation field from the ciliary border as there is from the other sources of contamination—the conjunctiva, the instruments and accessories, and the surgeon's fingers. On the other hand, I think it is quite impossible, unless special means are taken, to keep the lash area from becoming dirty, and often very quickly so when the eye is kept tied up. In other words, it is quite impossible to sterilize the ciliary borders satisfactorily on account of the organisms which lie beneath the surface round and about the lash follicles. The testing pad worn before operation may be entirely free from all discharge, and the lashes themselves be apparently perfectly clean, but soon after operation, even if they have been cut short, as they should have been, the stumps are apt to get sticky and dirty, and the discharge will then tend to get more and more objectionable, unless the source of trouble can be checked.

It is, therefore, in this regard, as well as in the dealing with sutures, that a non-irritating, efficient antiseptic becomes of extreme value, and once more I urge the use of flavine, which will generally do its work admirably.

After every operation I cover the affected eye with a gamgee pad well sprinkled with a 1 in 4,000 solution of flavine. It makes a delightfully comfortable dressing, which I change twice daily; if this procedure is followed the lash area will rarely cause any trouble whatever. If trouble does occur it will be of a mild character and easily controlled. Something must then be used in addition to, or as a substitute for, flavine, and I know of nothing better than a 2 per cent. solution of protargol employed twice daily as drops. Used at this strength protargol never irritates the wound, and it acts very efficaciously in arresting the discharge.

To sum up, then, the gist of these few remarks is to urge that the maintenance of a healthy operation field with quick normal healing can be obtained by the simplest methods before, during, and after operation. Antiseptics of any kind are only needed as *extra* measures, especially with regard to needles, sutures, and the lash area. If to these simple measures is added meticulous attention to detail, suppuration in the operation field can be eliminated with absolute certainty.

One exception must, however, be made to this assertion, and that arises when force of circumstances prevents the taking of routine precautions. It is occasionally necessary to operate on an acute glaucoma when there is intense congestion and obvious sepsis. In such an exceptional case I think the best antiseptic to use is hydrogen peroxide, with which the eye is flooded immediately before the knife section is made. It is much better than flavine, because the latter is strictly an antiseptic in the true sense of the word, and is not of much value when sepsis is already present.

Finally it is, perhaps, legitimate to quote personal experience in these matters, and I can claim that, in my private practice, I have never experienced the tragedy of intra-ocular suppuration after operation. In hospital practice I must, I think, have occasionally experienced this misfortune, though I cannot recall any instances, and certainly I have no single case impressed on my mind. In hospital work, however, it is not nearly so easy, for many reasons, to impress attention on certain details or to see that such details are carried through, and consequently accidents are more likely to occur there than in private work, where one assumes sole and entire responsibility.

PATHOLOGY OF THE TUBERCULOSIS OF CHILDHOOD AND ITS BEARING ON CLINICAL WORK.*

BY

EUGENE L. OPIE, M.D.

(From the Henry Phipps Institute, University of Pennsylvania, Philadelphia.)

RECOGNITION of the widespread occurrence of tuberculous infection acquired during childhood introduces into study of the disease two conflicting factors, one or other of which is often forgotten: (1) the increased resistance to the disease from a somewhat ineffective immunization, and (2) the danger that latent infection may cause manifest and perhaps fatal disease.

If attention is limited to tuberculous infection which is so advanced that it produces cough, fever, loss of weight, and obvious physical signs, while less advanced infection which remains concealed, for a time at least, is ignored, spread of the disease in families and in the community is studied under a heavy handicap, and ancient problems concerning its contagion remain unsolved. We might with equal reason study the epidemiology of measles by limiting our attention to instances of the disease followed by pneumonia, or of small-pox by excluding all cases except those with haemorrhagic lesions.

The Pathology of Latent Tuberculosis.

I am inclined to believe that more knowledge of the pathology of tuberculosis can be obtained by careful examination of the tuberculous lesions of those who die from causes other than tuberculosis than from fatal instances of the disease. The frequency of concealed tuberculous infection is thoroughly recognized, in great part as the result of almost innumerable studies made with the tuberculin test, but the variety and severity of latent tuberculous lesions is not fully understood. Sir Arthur Newsholme[1] assembled evidence to show that there is a long latent period between exposure to infection and the appearance of the symptoms of phthisis. The frequency of calcified pulmonary nodules which may be regarded as scars of an infection acquired in childhood is well known, but it is less fully realized that one in five of adults[2] who die from causes other than tuberculosis have apical tuberculous lesions which are caseous or partly calcified.

X-ray photographs of lungs excised at autopsy furnish a satisfactory means of depicting the distribution of tuberculous lesions, particularly when calcium salts have been deposited in caseous material, or new formation of fibrous tissue has increased the density of lung tissue. They serve as a guide in dissection to scattered nodules and reveal lesions which would otherwise be discovered with much difficulty. All types of lesions which are discoverable in *x*-ray plates prepared from excised lungs can be identified in plates prepared from the living chest. Radiologists who are doubtful concerning the interpretation of latent tuberculous lesions evident in *x*-ray plates of the living chest should prepare a series of plates from lungs removed at autopsy and should identify by dissection the lesions which are revealed.

First Infection with Tuberculosis.

Tuberculosis of infancy and early childhood, which resembles that of an animal inoculated with tubercle bacilli, forms a lesion at the site of infection and quickly affects the nearest lymph nodes. The severity of tuberculosis in early infancy seems to indicate great susceptibility to the disease, but exposure of the infant to massive infection from those who tend it may explain the high mortality from tuberculosis during the first year of life. At this period general dissemination through the body accompanied by tuberculous meningitis is common. During the second year of life the mortality from tuberculosis falls rapidly, and during the next few years gradually. From the fifth to the twelfth year the death rate is lower than at any other period. It is noteworthy that during this interval of low mortality infection with tuberculosis, indicated by the tuberculin reaction, makes its appearance with

* A paper read in opening a discussion in the Section of Tuberculosis at the Annual Meeting of the British Medical Association, Edinburgh, 1927.

great frequency, but the lesions with few exceptions heal. A nodule is formed in the lung substance, and foci which are usually much larger are formed in the nearest lymph nodes. Occasionally, particularly when disease of bronchial lymph nodes is advanced, a few tubercles which undergo encapsulation are formed in distant organs such as spleen and liver,[3] but general dissemination and death are uncommon. With healing, encapsulation is followed by calcification, and scars of these first infections remain throughout adult life in lung, bronchial lymph nodes, and sometimes in other organs.

Tuberculosis in young children usually proceeds more rapidly in the lymph nodes than in the lung parenchyma, so that the lesion of bronchial lymph nodes at the hilum of the lung is often very conspicuous. Many have thought that the disease begins in the lymph nodes and spreads peripherally into the lung substance. A nodule, occasionally found with difficulty, is, as Küss[4] showed, invariably present in the lung substance, and is presumably the primary site of lodgement of tubercle bacilli which have entered by way of the bronchi. The term "hilum tuberculosis" is, it seems to me, unfortunately chosen, because it suggests that the disease has its origin at the root of the lung. In *x*-ray plates made during the progress of pulmonary tuberculosis in children the lesion occasionally seems to extend from the hilum to the periphery of the lung, but this deceptive appearance is caused

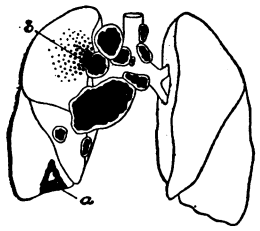

FIG. 1.

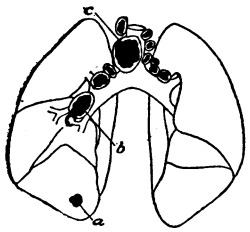

FIG. 2.

FIG. 1.—Diagram showing tuberculosis of the lungs and tracheobronchial lymph nodes with secondary extension to adjacent lung substance in a child aged 1 year. The primary focus (*a*), which contained a small cavity, was in the right lower lobe. The caseous tuberculous centre of a lymph node had penetrated the capsule at *b* and produced a secondary lesion of the adjacent right upper lobe. Death occurred with tuberculosis widely disseminated throughout the body.

FIG. 2.—Diagram showing tuberculosis of lung (*a*) and of tracheobronchial lymph nodes (*b*, *c*) with secondary extension of tuberculosis from lymph node to lung substance, in a girl aged 6. The primary focus of infection was the caseous encapsulated nodule (*a*, 1 by 1.2 cm.). The caseous centre of the lymph node (*b*) had penetrated the capsule and extended into the lung substance. The caseous centre of the large lymphoid mass (*c*) had penetrated the capsule of the node and the adjacent tracheal wall so that caseous material protruded into the lumen of the trachea just above the orifice of the right bronchus. There were tuberculous ulcers of the intestine, tuberculous peritonitis, and tuberculous salpingitis.

by the projection of three dimensions on one plane. *Post-mortem* examination occasionally shows that caseous lesions of bronchial lymph nodes have penetrated the capsule of the node and produced tuberculous pneumonia of the adjacent lung (Fig. 1), or have ulcerated through the wall of a bronchus (Fig. 2), and perhaps caused dissemination of the disease in the domain of tributary bronchial trunks, but in such instances a primary focus of infection can be found in the peripheral lung field. (Fig. 1, *a*; Fig. 2, *a*.)

Experimental studies indicate that tuberculosis is conveyed to susceptible animals with much more difficulty by way of the gastro-intestinal tract than by way of the lungs. Several years ago I had the opportunity to compare the frequency of latent tuberculosis of mesenteric lymph nodes in British and American adults.[5] In slightly more than one-fourth of young British adults examined who had died from causes other than tuberculosis, there were healed or healing tuberculous lesions of the mesenteric lymph nodes, whereas in Montreal Klotz[6] found obsolescent lesions in 8 per cent. of those examined, and in San Francisco Ophüls[7] found them in 5 per cent. I found that focal lesions of the lung were usually absent when mesenteric tuberculosis had occurred. Todd[8] has recently found that the incidence of mesenteric tuberculosis is very high in young persons in Edinburgh, and has observed that pulmonary lesions are uncommon when mesenteric lesions are numerous.

Reinfection with Tuberculosis.

Phthisis of adults, unlike the first infection of childhood, has little resemblance to tuberculosis of susceptible animals, but resembles the disease of reinfected animals. Why it selects the apex of the lung is not yet evident, but once established it tends to pursue a chronic course and remains, save in the last stages, a localized disease of the lung. The apical lesions which heal so completely that they never produce symptoms are much more numerous than those which cause progressive disease. Less than 1 in 10 of all deaths is caused by tuberculosis, whereas approximately 1 in 5 of those who die from causes other than tuberculosis have characteristic tuberculous, caseous or calcified, lesions of the pulmonary apex. The number of those having fibrous apical scars with no gross microscopic characters of tuberculosis is even greater.

Once infected, the experimental animal undergoes changes which alter its reaction to the bacteria and bacterial products (tuberculin). We have no clear understanding of the relation between hypersusceptibility in the infected animal and the resistance conferred upon it by immunization, but analogy with similar changes produced by more readily studied protein substances[9] suggests that accentuation of the inflammatory reaction following infection by the tubercle bacillus is a beneficial process which tends to overcome the second invasion.

This conception concerning the pathogenesis of phthisis has little value unless it is in harmony with changes which are evident in the human body. (1) When phthisis of adults occurs the calcified scars of a first infection can be found in the lung substance and in adjacent bronchial lymph nodes.[10] (2) Like a second infection of experimental animals, phthisis of adults pursues a chronic course, remains localized, and does not implicate the nearest lymphatic nodes. (3) When people who have not come into contact with European civilization escape tuberculosis during childhood they suffer, if infected during adult life, with a type of tuberculosis which resembles that of childhood. The susceptibility to acute forms of tuberculosis of native African troops serving in Europe has been clearly shown by Dr. Lyle Cummins.[11] The American negro not infrequently escapes infection during childhood, and exhibits acutely fatal lesions of first infection during adult life. (4) When reinfection occurs at an early age, after a still earlier first infection, it exhibits the characters of the disease of adults. The adult type of tuberculosis with localization in the apex, chronic course, and cavity formation is seldom seen during the first decade of life, but during the second decade with increasing frequency as age advances. In adolescent children after 15 years of age pulmonary tuberculosis in most instances has the characters of the adult disease.

There are two peaks upon the curve representing mortality from tuberculosis. There is one in infancy reaching a maximum during the first year of life. It is due to first infection having its origin in the lungs and bronchial lymph nodes, or, in a smaller number of cases, in the intestine and mesenteric lymph nodes. Mortality from these lesions of first infection diminishes rapidly during the second year, and then gradually during subsequent years until the fifth year. After the fifth year and during the greater part of childhood mortality is lower than at any other period of life. After the twelfth year in girls, and somewhat later in boys, the curve of mortality begins to rise and reaches a second peak in early adult life or even later. It is noteworthy that this second wave of mortality from tuberculosis is caused by lesions of reinfection having their origin with a few exceptions in the apices of the lungs.

Bacteriology of Latent Tuberculous Lesions.

My associate Dr. Aronson[12] and I have attempted to determine the frequency with which living tubercle bacilli can be recovered from latent lesions of first infection present as encapsulated and calcified nodules in the lungs and bronchial lymph nodes. Tubercle bacilli are seldom recovered from these healing nodules unless there is a latent apical lesion in the same lung, and when latent apical lesions are present living tubercle bacilli are

recovered not only when material from calcified nodules is injected into guinea-pigs, but when material from parts of the lung which contain no evidence of tuberculosis is used for inoculation. These observations indicate that living tubercle bacilli are present in the surrounding lung tissue rather than in the nodule itself. In latent lesions of the apex, on the contrary, tubercle bacilli are almost invariably found when the lesion is fibroid and caseous, and persist even after apparent healing, so that they are found in one-fourth of the fibrous apical scars which have none of the gross or microscopic characters of tuberculosis. Tubercle bacilli in most, if not all, instances seem to have disappeared from the lesion of childhood by the time the lesion of reinfection makes its appearance. Furthermore, anatomical evidence furnishes little support to the opinion that the apical lesion of adults is derived by extension from the focal lesions of childhood, for the one is seldom in continuity with the other, and not infrequently the lesion of first infection is in one lung, whereas the apical lesion of reinfection is in the opposite organ. The available evidence

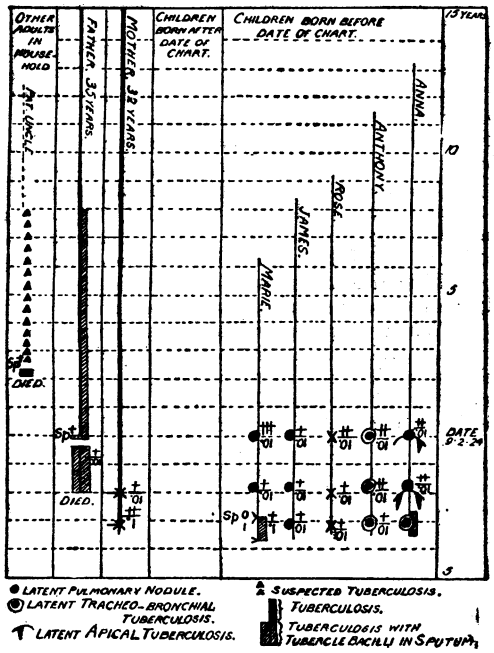

FIG. 3.—Graphic chart used to depict the occurrence of latent and clinically manifest tuberculosis in families. Vertical lines represent each member of the family, and their length corresponds with the duration in years of residence within the household. They are erected upon a horizontal line, which represents the date at which the family came under observation. Later observations are recorded below this line. Tuberculin reactions are shown by 0 (negative) or from one to three *plus* signs over the quantity in milligrams (0.01 to 1) of tuberculin used. The result of sputum examination (Sp) is indicated by zero or *plus* placed above the number of examinations.

indicates that pulmonary tuberculosis of adults is not the continuation of an infection acquired in childhood, but is an exogenous reinfection.

Clinical Significance of Latent Tuberculosis.

Concealed foci of tuberculosis in lungs and lymph nodes which give none of the manifestations of pulmonary disease may be the source of serious tuberculosis in distant organs. Ophüls[13] has recently attempted to determine the primary focus of infection when tuberculosis causes recognizable disease of the genito-urinary tract, adrenals, bones, joints, etc. In seven cases of Addison's disease tuberculosis of the adrenal had apparently had its origin in a pulmonary focus, and in six instances this focus had healed. In twenty-seven cases of tuberculosis of bones and joints the primary focus of infection was in the lungs, and in one-third of these it was healed. Genito-urinary tuberculosis is always secondary to tuberculosis of lung or intestinal tract, and in 20 per cent. of instances referable to the lung the pulmonary lesion was healed.

Latent tuberculosis may be defined as tuberculous infection which is unaccompanied by significant symptoms evident to the patient or by physical signs discovered by the physician. The means at our disposal for the discovery of latent tuberculosis are the tuberculin test, which shows whether tubercle bacilli have found lodgement in some part of the body, and *x*-ray examination, which, within certain limitations, discloses the character and extent of the lesion. The term "latent," in the sense in which I have used it, has long been used in medical literature, and may be found in the writings of Laennec and Louis. Some authors have assumed that it is applicable only to lesions which are dormant or arrested, but long accepted usage gives no support to this interpretation of the word. Lesions which are unaccompanied by symptoms or physical signs are often progressive, and manifest disease is invariably preceded by a long or short period of latency.

My associate Dr. McPhedran and I[14] have undertaken a study of latent tuberculous lesions in children of families exposed to tuberculosis, and have attempted to determine whether they produce manifest disease. By means of *x*-ray examination we found that it was possible to discover all types of lesions which can be recognized in *x*-ray plates

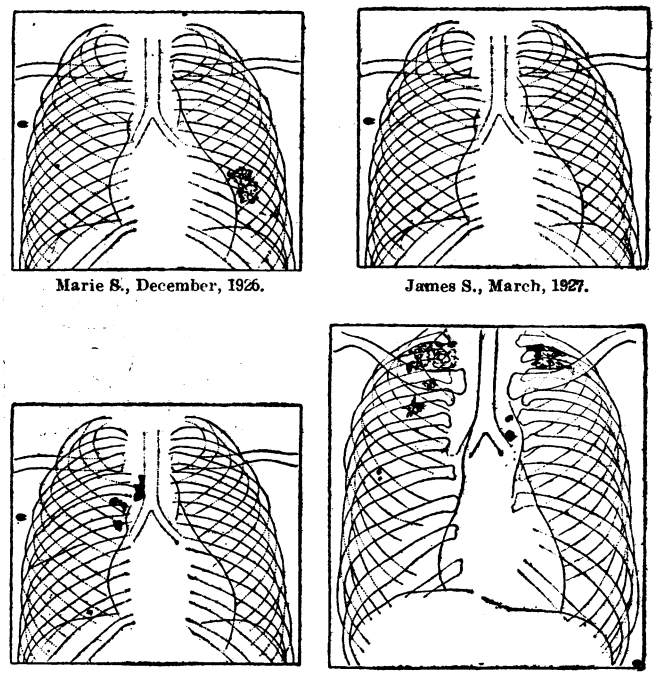

FIG. 4.—Diagrams prepared from *x*-ray films to show the position of the lesion in the lungs of children of the family shown in Fig. 3.

of excised lungs and identified by subsequent dissection. Examination of members of families exposed to contact with tuberculosis showed that latent lesions were more numerous and more extensive among them than in families which had no contact with the disease. The severity of infection bore a measurable relation to the elimination of tubercle bacilli by the tuberculous member of the family, and to the duration of exposure to contact with the micro-organism. Transitional stages from latent to manifest disease occurred, and it became evident that it is often possible within certain limitations to predict clinically manifest disease long before it makes its appearance.

These observations have brought about important changes in our procedures for the prophylactic treatment of tuberculosis. Families are studied as units and the progress of infection in them determined. The usual method of sputum examination is the best means for determining whether members of a family are exposed to present danger of contagion, but latent infection in members of a family may serve as an index of past exposure to open tuberculosis. We have prepared for each family under the care of our out-patient department a graphic chart (Fig 3) which records how long each member of the household has been exposed to contact with tuberculosis, and shows for each the presence or absence of a tuberculin reaction, the character of any latent tuberculous lesions revealed by *x*-ray examination (Fig. 4), and the presence or absence

of manifest disease which has been recognized by symptoms and physical signs.

The children of the family shown in the chart (Fig. 3) were exposed to tuberculosis of the father and of the uncle. Sputum of both contained tubercle bacilli and they were careless in their habits. Marie, when first seen, had an intense tuberculin reaction and a small nodule in the lung. Two years later a focus of infiltration had appeared in the left lung and râles were heard over it. Anthony had advanced tracheo-bronchial tuberculosis. Anna, when first seen, had nodules in the lungs and a latent apical lesion on the right side. One year later there were bilateral apical lesions. After another year these lesions had extended and râles were heard over the apices.

This study has emphasized the value of the tuberculin reaction as a means of recognizing tuberculosis in children. Its significance, as is well known, diminishes with increasing age from infancy to adult life, when among inhabitants of large cities almost all react. In our group of families exposed to open tuberculosis over 80 per cent. have acquired an active tuberculin reaction before the age of 5 years, and 90 per cent. at the age of 10, whereas at the same time among "non-contact" families only 23 per cent. react at the age of 5, and 62 per cent. between the ages of 15 and 20 years.

Examination by x rays gives trustworthy evidence concerning the frequency of latent tuberculosis among children exposed to contact with open tuberculosis and furnishes a means by which the severity of infection may be measured. Focal lesions within the lung substance are readily recognized if the characters of the lesions found at autopsy are kept in mind. Blood vessels directed in the axis of the x ray may be mistaken for calcified nodules. Thirty per cent. of our children who were exposed to tuberculosis in their families had pulmonary nodules, as compared with 17 per cent. of the children who were not thus exposed. More significant is the recognition of lesions of the tracheo-bronchial lymph nodes, for although anatomical study (Fig. 5, from the lungs of a youth who had been in contact with open tuberculosis) shows that they invariably accompany focal pulmonary lesions, they are seldom recognizable in x-ray films unless they have attained considerable size. Tuberculosis of tracheo-bronchial lymph nodes was found in 23 per cent. of children in contact with open tuberculosis, and in only 4.5 per cent. of children of non-contact families. The average duration of contact with open tuberculosis when tuberculosis of tracheo-bronchial lymph nodes is recognized has been four years and five months, and the lesion is seldom found in those who have been exposed during a period of less than two years.

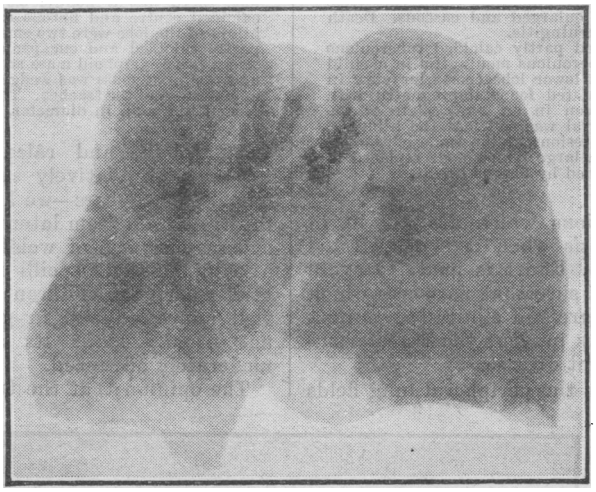

FIG. 5.—Reproduction of an x-ray film prepared from the lungs, removed at necropsy, of a youth of 19 years, who died with purpura haemorrhagica and anaemia. His father died of pulmonary tuberculosis at the age of 44. Five calcified nodules from 2 to 4 mm. in diameter were found in the upper lobe of the right lung (reversed in the figure), and there were several lymph nodes containing calcified masses at the hilum of the lung. At the right of the bifurcation of the trachea there was lymphoid tissue replaced in great part by irregularly distributed calcified material (4 by 1.9 cm.). Caseous, partly calcified and encapsulated tubercles were sparsely scattered in the liver and spleen.

Of graver import is the occurrence of latent apical tuberculosis in adolescent children. At the time of our report one year ago this lesion was found in 1.6 per cent. of the children of families with open tuberculosis, but subsequently the incidence has increased to 3.2 per cent. Latent apical disease has not been found in children of non-contact families. Among those in whom latent apical tuberculosis has made its appearance, including adolescent children and adults, the average duration of contact has been seven years and 11 months, but this is evidently not the minimal period of contact required to produce the lesion.

Parallel with the frequent occurrence of such latent lesions of tuberculosis in families with open tuberculosis, there is high incidence of clinically manifest tuberculosis with symptoms and physical signs. In the group of contact families 9 per cent. of children among those examined had manifest tuberculosis. No manifest disease has appeared in the non-contact families.

This study has shown that the usual method of sputum examination, repeated at short intervals, is an effective means of measuring the danger to which an infected individual subjects his family. The frequency of tuberculin reactions, of lesions recognized by x-ray examination, and of clinically manifest disease, was much lower in families in which a member suffered from pulmonary tuberculosis but had no tubercle bacilli in his sputum than in families exposed to open tuberculosis, and was only slightly greater than in those having no contact with the disease. There was another group of families in which some members had symptoms and other evidence suggesting tuberculosis. In these families with suspected tuberculosis the incidence of infection was not materially greater than in non-contact families.

Isolated nodules recognized in the peripheral lung field in most instances represent well circumscribed infections and have little clinical significance. Although they indicate that tuberculous infection has occurred and has doubtless conferred some degree of immunity upon the child, they do not exclude the possibility of progressive disease or of reinfection.

Tuberculosis of Tracheo-bronchial Lymph Nodes.

Tuberculosis of the tracheo-bronchial lymph nodes is recognized by clearly defined, oval, granular shadows in contact with the larger bronchial trunks at the hilum of the lung or in immediate contact with the trachea and often below the bifurcation (Fig. 4). Accentuation of the hilum shadow or thickening of the mediastinum is seldom due to tuberculosis of lymph nodes and does not aid in the recognition of the disease. Lesions of the tracheo-bronchial lymph nodes often attain considerable size, and in such instances indicate that infection is advanced. Occasionally very large groups of caseous lymph nodes are well defined in x-ray plates, but comparison with x-ray films prepared from lungs removed at autopsy shows that tuberculous lesions of lymph nodes with few exceptions are recognizable only when calcium salts have been deposited within the caseous material. It has been claimed that these lesions of bronchial lymph nodes are never visible except when they are calcified and completely healed and no longer a source of danger. This view is certainly incorrect. When there is impregnation with calcium salts scarcely sufficient to increase the consistency of caseous material, lesions become clearly defined in x-ray films. Furthermore, a lesion which is arrested and calcified in one part may be progressive in another. Figure 6 represents the lung of a child; there are in one part nodules of lungs and of lymph nodes (a, b) which are encapsulated and calcified, and in another part progressive caseous lesions (c, d) which have produced death by general dissemination. It is probable that the two kinds of lesions, since they occur in a child 2 years of age, began at approximately the same time. Figure 7 represents the lungs of a child 9 months of age with caseous and calcified foci in the lungs and bronchial lymph nodes. Death occurred with disseminated tuberculosis and tuberculous meningitis.

Although I believe that recognizable lesions of the tracheo-bronchial lymph nodes in many cases cause no disturbance of the health and heal completely, I think that they should be regarded with grave concern, especially when they appear during the first decade or the beginning of the second decade of life. This opinion is

based upon the frequent coexistence of tuberculosis of the tracheo-bronchial lymph nodes and clinically manifest lesions of the lungs in children of families exposed to open tuberculosis, and the development of manifest tuberculosis in another group of children in whom tuberculosis of the tracheo-bronchial lymph nodes has been recognized. All of our children who exhibit this lesion have been placed

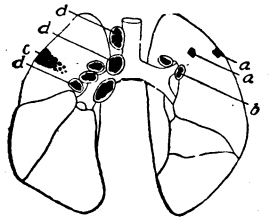

FIG. 6.

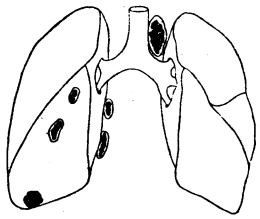

FIG. 7.

FIG. 6.—Diagram of lungs with caseous calcified lymph nodes in one lung (*b*) and with progressive caseous lesions in the other (*c*, *d*) in a child 2 years of age. In the left lung there were two small caseous encapsulated nodules at the hilum of the lung; nearby there was a lymph node replaced in great part by a tuberculous lesion with caseous, partly calcified centre and fibrous capsule. In the right upper lobe below the pleura there was a wedge-shaped area of consolidation, in large part caseous and surrounded by numerous small tubercles. The adjacent lymph nodes were much enlarged and caseous. Death occurred as the result of tuberculous meningitis.

FIG. 7.—Diagram showing encapsulated partly calcified tuberculous foci in lungs and lymph nodes with tuberculous meningitis in a child aged 9 months. At the base of the left lower lobe (posterior view in diagram) there was a caseous encapsulated focus impregnated with calcium, so that opaque spots were seen in an *x*-ray plate of the excised lung, though the caseous material was soft. At the hilum of the lobe there were lymph nodes with lesions of the same character. On the right of the trachea there was a large lymph node replaced in great part by caseous material surrounded by fibrous tissue.

so far as possible under conditions which are known to retard the progress of tuberculosis when it is manifested by symptoms and physical signs. Effort is made to avoid malnutrition and fatigue. I feel sure that measures which are effective in retarding the progress of clinically manifest tuberculosis will in most instances prevent the transformation of latent infection into evident disease.

The presence of soft shadows in the peripheral lung fields recognizable in *x*-ray plates of the chest is often difficult to interpret, and introduces a consideration of the diagnosis of pulmonary tuberculosis in children. They are at times unaccompanied by symptoms or physical signs. The following criteria indicate that these soft shadows are the result of tuberculous infiltration: (1) occurrence in a child in contact with tuberculosis; (2) an active tuberculin reaction (with 0.01 mg. of old tuberculin); (3) association with lesions of adjacent lymph nodes recognizable in the *x*-ray plates.

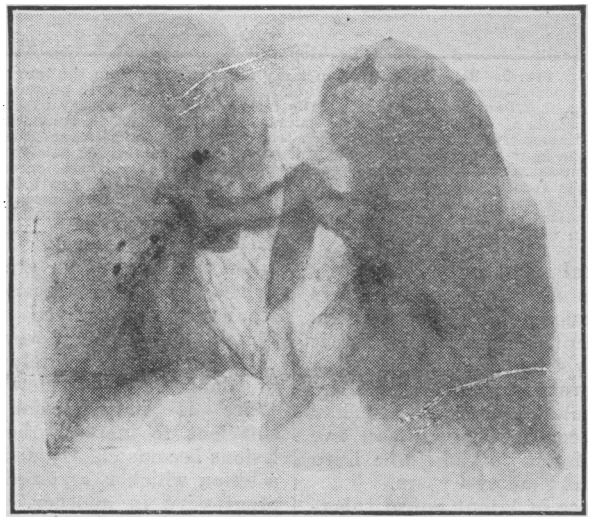

FIG. 8.—Reproduction of an *x*-ray film prepared from the lungs of a boy aged 18, showing latent apical tuberculosis on the right side (reversed in the figure) and calcified nodules in the lung and adjacent lymph nodes on the left side. Death occurred as the result of peritonitis following laparotomy. At the right apex in continuity with the puckered pleura there was fibrous and caseous tissue forming a mass 3 cm. across. In the left upper lobe there was a small calcified nodule and larger firmly calcified nodules in lymph nodes at the hilum of the lobe. In the substance of the left lower lobe and in adjacent lymph nodes there were similar lesions.

Latent Apical Tuberculosis of Adolescent Children.

Latent apical tuberculosis is encountered with increasing frequency as age increases during the second decade of life, and is evidently in many instances the precursor of phthisis of early adult life. Examination of adolescent children of families in contact with open tuberculosis has revealed almost all of our cases of latent apical disease, but in a few instances it has been discovered in others. It occurs only when contact with open tuberculosis has been long continued. In *x*-ray plates it is recognizable as a mottled shadow in one or the other apex, evident in the second interspace, and extending into the third and even into the fourth interspace. The extent and density of lesions which are unaccompanied by symptoms and physical signs are at times astonishing. The anatomical characters of apical lesions depicted in *x*-ray plates when unassociated with clinical evidence of the disease do not differ from lesions with which there have been slightly elevated tempera-

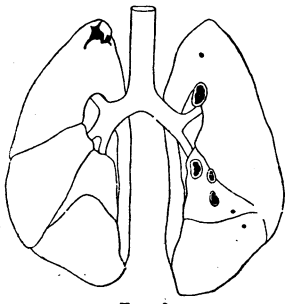

FIG. 9.

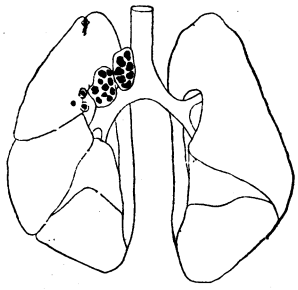

FIG. 10.

FIG. 9.—Diagram showing the distribution of lesions shown in Fig. 8 (in which right and left are reversed).

FIG. 10.—Diagram showing fibrous scar of apex and advanced calcified tuberculous lesions of tracheo-bronchial lymph nodes in a girl aged 18. Death occurred as the result of typhoid fever. At the right apex extending inward from the puckered pleura there was a thick fibrous scar 2.5 cm. in length. In the substance of the right upper lobe there was a nodule 0.5 cm. in diameter with caseous, partly calcified centre and fibrous capsule. In the lung substance near the hilum of the lobe were two small lymph nodes which contained caseous, partly calcified and encapsulated nodules. At the hilum of the lobe was a large lymphoid mass studded with caseous and calcified nodules, and a similar mass was wedged between the inner surface of the right upper lobe and the trachea. The spleen contained encapsulated tubercles from 1 to 2 mm. in diameter, with caseous and calcified centres.

ture, cough, and râles heard over the affected apex. During the relatively short period of our observations—namely, four years—we have had opportunities of following the transition from latent apical disease to advanced tuberculosis with loss of weight, cavity formation, and appearance of tubercle bacilli in the sputum. It is noteworthy that adolescent children with latent apical lesions may be well nourished and in good health. Loss of weight does not usually make its appearance until the disease is moderately advanced.

The opinion that the tuberculosis of adults has its origin in childhood has long been maintained and is still widely held. Von Behring claimed that all tuberculosis had its origin in early infancy, but most of those who think that phthisis does not originate in adult life believe that it is the continuation of an infection acquired at any time during the period when focal lesions are making their appearance in lungs and bronchial lymph nodes. Nevertheless, it may be noted that few of those who discuss the subject define the relation of these lesions to subsequent apical disease in adults. There is, as I have shown, no evidence that tuberculosis of adult life is the continuation of the first infection of childhood. Nevertheless, it is evident that latent apical tuberculosis of older children may gradually develop into phthisis of early adult life. The transition may be followed by means of radiological examinations.

Latent apical lesions of adolescents which have all of the characters of reinfection are in a considerable number of instances associated with advanced tuberculous tracheo-bronchial lesions which have undergone calcification (Figs. 8, 9, and 10). These massive tracheo-bronchial lesions accompanied by latent apical tuberculosis with few exceptions occur in children who have been exposed to intimate contact with open tuberculosis for several years, and apical

disease is superimposed upon the first infection because resulting immunization is an ineffective protection against tubercle bacilli in vast numbers.

Conclusions.

Latent tuberculous lesions of the lungs and tracheo-bronchial lymph nodes are revealed by radiological examination, and when they are accompanied by an active tuberculin reaction it may be assumed that they are not dormant or healed. These lesions, well known to pathologists, may have grave clinical significance. They are not infrequently the source of tuberculosis of meninges, bone, genito-urinary system, adrenals, and other organs; they may produce clinically recognizable pulmonary tuberculosis of the type of childhood, or, as an index of exposure to tuberculous infection, may be followed by the adult type of pulmonary disease.

Latent tuberculosis of the tracheo-bronchial lymph nodes of young children and latent apical tuberculosis of children during the second decade of life indicate the presence of grave infection, and may forewarn against clinically manifest disease. These latent lesions appear with great frequency in families exposed to open tuberculosis. All members of families in contact with tuberculosis should be given repeated tuberculin tests and radiological examinations even though they appear to be in good health. When these examinations reveal grave latent disease the well known measures directed to retard the progress of tuberculosis will prevent the development of manifest disease.

Tuberculous infection in childhood increases resistance to the disease, but is at the same time a source of danger. Recognition and control of latent infection are effective means of preventing tuberculosis.

REFERENCES.

1 Newsholme, A.: *The Prevention of Tuberculosis*, 1910.
2 Opie, E. L.: *Journ. Exper. Med.*, 1917, xxv, 855, xxvi, 263; *Amer. Rev. of Tuberc.*, 1924, x, 249.
3 Klotz, O.: *Amer. Journ. Med. Sci.*, 1917, cliii, 786.
4 Küss, G.: *De l'hérédité parasitaire de la tuberculose humaine*, Paris, 1898.
5 Opie, E. L.: *Amer. Rev. of Tuberc.*, 1920, iv, 641.
6 Klotz: *Pennsylvania Med. Journ.*, 1917, xx, 811.
7 Ophüls: *Trans. Nat. Tuberc. Assoc.*, 1911, vii, 394.
8 Todd, T. R. R.: *Edinburgh Med. Journ.*, 1927, xxxiv, 127.
9 Opie, E. L.: *Tubercle*, 1925, vii, 23.
10 Opie, E. L.: *Amer. Rev. of Tuberc.*, 1924, x, 249.
11 Cummins, S. L.: *Internat. Journ. Pub. Health*, 1920, i, 137.
12 Opie, E. L., and Aronson, J. D.: *Amer. Arch. of Path.*, 1927.
13 Ophüls, W.: *A Statistical Study of 3,000 Autopsies.* Stanford University Publications. Medical Sciences, 1926, vol. i, No. 3, p. 131.
14 Opie, E. L., and McPhedran, F. M.: *Amer. Rev. of Tuberc.*, 1926, xiv, 347.

DISCUSSION.

Dr. R. G. CANTI (London) said that the question of the primary tuberculous focus in the lung in childhood had not received much attention in this country. He had reported in the *Quarterly Journal of Medicine* (1919) 84 consecutive *post-mortem* examinations on children under 10; 16 were tuberculous, and of these 10 had shown the primary lung focus. Further experience had convinced him that Ghon's theory of the relation of primary focus and glandular infection was tenable. He showed diagrams of the scheme of lymphatic drainage from the lung, and said it was possible to predict from the glands infected in a particular case the position of the primary focus in the lung. He supported this by diagrams of three cases. He believed it could be proved that the mediastinal glands were secondary to the lung focus, since where there was a lung focus there were tuberculous glands in the corresponding mediastinum, and where the mediastinal glands were tuberculous a tuberculous focus was present in the lung. Moreover, anatomical study had shown those glands to be tuberculous which would be expected to be so affected if the lung focus was primary. The changes in the glands never appeared older than those in the lung focus. He thought it was also proved that the lung focus was not secondary to one elsewhere in the body. In the majority of cases showing a lung affection no focus could be found outside the thorax. In most cases with a focus outside the thorax there was no lung lesion. If the lung infection was secondary to some other invasion—as, for example, in the abdomen—the type of tubercle bacilli would be bovine; tuberculous foci in the lung yielded the human type of bacillus. The human bacillus had no predilection for lung tissue; feeding experiments had shown that the lesions produced by human and bovine bacilli were the same, the greatest concentration of infection being abdominal. This point had an interesting confirmation in the work of Scott in Hong-Kong. In conclusion, Dr. Canti showed, for Dr. Hadfield of Bristol, five museum specimens from five consecutive cases of children dying of tuberculosis in which Dr. Hadfield had been able to demonstrate the primary lung focus.

Dr. P. F. ARMAND-DELILLE (Paris) said that, contrary to general opinion, tuberculosis in children was found in the varied adult types. Most valuable information was to be obtained from comparing lung shadows and autopsy findings. He described his technique for the study of lungs *post mortem* in their proper relative positions, and showed how it was possible thus to find every important lesion and compare it with *x*-ray shadows. Investigation on these lines might reveal one of two conditions—tuberculosis apparently limited to the tracheo-bronchial glands, or associated with different forms of lung tuberculosis. In nearly every case careful examination indicated the primary lung lesions. These might be (1) diffuse miliary tuberculosis in its various forms; (2) localized lesions, usually broncho-pneumonic lobar infiltrations; (3) unilateral lesions of a whole lung; and (4) bilateral terminal lesions. His experience had convinced him that normal lungs showed *x*-ray hilum shadows which bore no relation to enlarged bronchial glands. Even when these glands became enormous caseous tumours their shadows might be lost in the larger shadow of the cardio-vascular apparatus. *Post-mortem* findings were gradually aiding in the interpretation of lung shadows from a prognostic point of view; it was possible to correlate progressive modification in the character of shadows with analogous anatomical evolution. Tuberculous bronchial gland tumours frequently occurred in the middle of the mediastinum, where their shadows could but rarely be observed because they were usually masked by the cardio-vascular apparatus. So-called "perihilar shadows" were not produced by the glands. The primary focus was seldom detectible in the *x*-ray picture, as it was often localized at the opposite pulmonary base behind the shadow of the heart, liver, stomach, or spleen. In lung lesions a study of well taken plates might reveal miliary tuberculosis before the characteristic signs and symptoms had appeared. It was difficult from *x* rays alone to arrive at the accurate diagnosis of the type of pathological lesion present. A series of *x*-ray photographs during treatment showed progressively disappearing shadows, while tubercle bacilli were still found in the sputum. Dr. Armand-Delille illustrated his remarks with many beautiful slides of sections and comparative *x*-ray plates.

VOMITING AFTER OPERATIONS ON THE STOMACH.

BY

A. RENDLE SHORT, M.D., B.S., F.R.C.S.,

SURGEON TO THE BRISTOL ROYAL INFIRMARY.

VOMITING coming on within a day or two of an operation on the stomach, whether a gastro-jejunostomy, partial resection, or other surgical procedure, is not uncommon, and may be serious or even fatal. In some cases it is merely due to the anaesthetic, or to blood in the stomach. In that event it is likely soon to cease. Repeated vomiting, that persists for days and puts the patient's life in danger, may be due to one of the following causes:

1. *Vicious Circle.*—Years ago this was frequent and often fatal; nowadays, in good hands, it is seldom seen. It is due, of course, to a bad lie of the efferent loop of jejunum; the afferent loop may have been too long, or the efferent loop may fall away from the opening in the stomach so sharply that the duodenal contents re-enter the stomach and cannot find a way out. The vomit is copious and contains abundant bile. Prevention is easy. It is only necessary to use a short afferent loop of jejunum in the operation of gastro-jejunostomy, and to insert a stitch, after completing the anastomosis, to anchor the efferent loop to the transverse mesocolon an inch or two to the