never place his own hand on it for the purpose of estimating the quality and intensity of the light; there are more accurate methods and instruments of precision for making these measurements without risk to the operator. In every instance the screen of the fluoroscope should be covered with a sheet of one-fourth inch plate glass; this does not afford absolute protection, however, as some rays will penetrate it.

The operator should stand behind a thick lead screen, made of sheet lead with an opening corresponding to the plate glass in the fluoroscope. A fluoroscopic examination under other conditions is dangerous to the operator if frequently repeated. The patient's body affords some protection, but the observer is certain to enter the irradiated field if great precaution is not used.

Although it is highly probable that the serious results produced in operators' hands are due to the action of the Roentgen rays, there are probably other contributing causes. Among these the repeated introduction of the hands into the high induction fields surrounding the coil must be considered. Any contributing factor, if it is an agency whose action is unknown, should be avoided as far as possible. It is safer, therefore, to place the induction coil in an adjoining room, leading in the secondary wires through porcelain tubes and having the controlling apparatus in the operating room. This is especially necessary in large hospital laboratories where more than one coil is in use, and where many patients are being treated.

Since I have employed these methods of protection I have noted marked improvement in, and practically a cure of, a severe dermatitis that had been so intense as to cause loss of sleep from the pain.

If Roentgen operators would avoid serious injury, such as has proved fatal in one case, and has caused two experienced operators to lose their hands, they must observe the utmost caution in subjecting themselves to radiations from a Roentgen tube. An operator should never place his hand in the irradiated field and should never employ it on the fluoroscope to test the light. He should always employ an opaque shield surrounding the tube and should keep his hands out of high potential induction fields.

I have been variously and often erroneously quoted in reference to the treatment of Roentgen ray burns. The following is the treatment which I employ and which I have found the most efficient in treating my own dermatitis:

The hands should be washed and soaked in water as hot as can be borne. It will be found that they gradually accustom themselves to very high temperatures, and that the temperature of the water can be gradually increased. The hands should be scrubbed with a sterilized brush, using Eichhoff's superfatted resorcin soap. This will be found very soothing. After thorough washing, the hands should be well rubbed in an ointment of lanolin containing one-half ounce of boracic acid and one dram of resorcin to the ounce. This should be done both morning and night and the hands should always be washed in hot water with the superfatted soap. Simple superfatted soap or soap and lanolin can be used if the resorcin is too severe, with the substitution of benzoic acid in the ointment.

The thickened epidermis and horny growths, with the indurated edges of the indolent ulcers, can be softened by covering them with ointment under oiled silk. The softened epidermis then should be gently removed. The ointment should be renewed after each washing.

Small healing ulcers that have become healthy can be covered with surgeon's isinglass plaster, after the hands have been washed and anointed; these ulcers often take from four to six months to heal and may cause intense pain.

In treating the acute erythema resulting from Roentgen ray treatment, I have employed the stearate of zinc composition with 10 per cent. ichthyol. It is very efficient, subdues the inflammation and itching, and helps to produce the tanning which is so essential before deep treatment can be efficiently employed. The powder should not be confused with the stearate of zinc ointment, as any ointment will soften the skin and render it more liable to break down and to slough. The substitution of the ointment for the stearate powder resulted, in one case, in the formation of a large ulcer. The entire epidermis adhered to the ointment and came away with it.

Where an open ulcer has formed, strict antisepsis should be maintained, and irritants should be carefully avoided. Sterile water is an irritant, while normal salt solution and boric acid solution are not. After cleansing, the resorcin ointment should be applied to relieve the pain and to promote healing. The employment of the stearate of zinc composition powder with 10 per cent. ichthyol seems to act as a prophylactic and prevents the production of serious burns during severe Roentgen ray treatment.

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THE SIGNIFICANCE OF TUBERCULOUS DEPOSITS IN THE TONSILS.*

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The object of the research which is outlined in this paper has been to determine, as nearly as possible, the relative importance of the tonsillar tissues of the throat as portals of entrance for a general tuberculous infection. To arrive at a definite conclusion, it seemed necessary to ascertain the truth concerning certain fundamental questions.

First. Are tuberculous lesions produced more easily in the tonsils than in other portions of the gastrointestinal tract?

Second. Does tuberculous adenitis of the neck have its starting point in the tonsils?

Third. What is the importance of tuberculous cervical adenitis as an etiologic factor in systemic or pulmonary infection?

From clinical data collected from literature, and from postmortem examinations which I have made, it is evident that in cases of pulmonary phthisis secondary tuberculous infection takes place more readily in the tonsils than in any other part of the upper respiratory tract.

Seifert¹ has shown that infected sputum plays a most important rôle in the infection of the tonsils, the bacilli finding the crypts of these organs a suitable place for lodgment. It has been proven by Goodale² that minute foreign bodies, such as carmin dust, are rapidly

* Read in the Section on Laryngology and Otology of the American Medical Association, at the Fifty-fifth Annual Session, June, 1904.

1. Handbuch der Laryn. u. Rhin., P. Heymann, vol. II, p. 714.

2. Absorption of Foreign Bodies by the Human Fauces Tonsil, Especially in Regard to the Origin of Infection Processes, Arch. f. Laryn. u. Rhin., 1897, vol. VII, p. 90.

absorbed by the tonsils from their crypts, and that these particles pass to and through the interfollicular tissues. This has been confirmed by Hendelsohn³ and Kayser.⁴

Piera,⁵ however, went further, and demonstrated that micro-organisms were likewise absorbed through the epithelium, though if they were not capable of resisting the vital action of the tissues, they were destroyed practically as soon as they entered the tonsillar parenchyma. Virulent germs, however, could not only cause local disease, but could be carried to the neighboring lymphatics and thence to almost any part of the body.

Lexer⁶ confirmed these results of Piera by a large series of experiments on rabbits.

STATISTICS OF SECONDARY INVOLVEMENT OF THE TONSILS.

The following statistics concern the frequency of secondary involvement of the tonsils in cases of pulmonary tuberculosis. In phthisis a large number of tubercle bacilli pass over the surface of the throat and tonsils, and this condition affords a very good test as to the relative absorbing power of the tonsils and the rest of the pharyngeal mucous membrane.

Schlenker,⁷ in a rather elaborate and extensive article, reviewed carefully 22 cases of tuberculosis of the lungs. He found the tonsils diseased in 15. In 13 of the 22 cases involvement of the lungs was extensive, and the tonsil was diseased twelve times. In 9 cases of slight involvement of the lungs in only 3 was tonsillar tuberculosis present.

Schlesinger,⁸ in 13 cases of galloping consumption, found tuberculosis of the tonsils twelve times; but in 4 cases of chronic involvement of the lungs, he failed to find any tuberculous changes in the tonsils. He concludes that tuberculosis of the tonsils is a sign of virulent infection of the lungs, and is, therefore, of very serious import. In healed lung lesions or in any circumscribed tuberculous disease the tonsillar involvement was absent.

Walsham⁹ examined after death the tonsils of 34 tuberculous patients. Only two of these patients had shown any symptoms of tuberculosis of the tonsils during life, and yet in 20 cases the tonsils were found to be more or less diseased.

Ito,¹⁰ in 6 cases of pulmonary tuberculosis in children, found tonsillar involvement three times, and in 7 cases of milary tuberculosis the tonsils were involved three times. His statistics cover some 104 autopsies, but in only 10 of the autopsies was the pharyngeal tonsil examined histologically, and, therefore, I can not accept all of his statistics. They are valuable in a positive sense, in that we must believe that he found at least 6 cases of tonsillar tuberculosis.

Dmochowsky¹¹ examined the tonsils of 15 phthisical

patients, 5 of whom had involvement of the larynx, and in all 15 cases tuberculosis of the tonsils could be recognized through the microscope, although no change could be noted with the naked eye. He found the tubercle bacilli penetrating between the epithelial cells of the crypts, especially in those places where there were apparent gaps.

Kruckmann,¹² in 64 autopsies, found some tuberculous condition in the body in 25. Among these 25 there were 12 cases of tonsillar tuberculosis, and of the remaining 13, 5 cases showed either excessively slight or no lesion of the lung. These 5 cases must, therefore, be excluded so far as secondary infection of the tonsil is concerned, so that Kruckmann's statistics should be 20 cases of tuberculosis of the lungs with secondary tonsillar tuberculosis occurring twelve times. In those cases of tuberculosis of the lungs in which there was no tonsillar involvement, the lesion in the lung was either very small or of a fibrous character.

Strassmann¹³ examined 21 cases of tuberculosis of some portion of the body. In 15 cases there was pulmonary tuberculosis, and of these 15 the tonsils were involved thirteen times. None of the other cases showed tonsillar tuberculosis.

Ozeki¹⁴ reports 9 cases of secondary tonsillar tuberculosis.

Gourc¹⁵ has made a general study of adenoid growths, bacteriologic examination being carried out in 202 cases. He states that although he examined 17 cases of active tuberculosis, and 18 cases in which the condition was supposed to be hereditary, in no case did he find the tubercle bacillus.

Cornil¹⁶ examined the tonsils of 70 cases of phthisis, finding tuberculosis only four times. The results of these last two observers seem so contrary to all other evidence that I fear that their technic must have been faulty.

A summary of the foregoing statistics, excepting those of Gourc and Cornil, gives us the following table:

Name.	Pulmonary tuberculosis.	Tonsils involved.
Schlenker	22	15
Schlesinger	17	13
Walsham	34	20
Ito	13	6
Dmochowsky	15	15
Kruckmann	20	12
Strassmann	15	13
	136	94

These statistics show that in at least 69 per cent. of cases of pulmonary tuberculosis the tonsils are tuberculous. The majority of the pathologic diagnoses made by the foregoing observers were based on histologic findings. Lately it has been shown that a positive diagnosis of tuberculosis can not be made by the character of the histologic changes. Occasionally giant cells and lesions resembling tubercles result from the irritation of foreign bodies, and it has been definitely demonstrated that certain acid fast bacilli will sometimes give rise to changes scarcely distinguishable from true tubercles. It must, however, be admitted that these lesions caused by foreign bodies and by acid fast bacilli are exceedingly rare, and especially so in the human being. If, in a patient suffering from active tubercu-

3. Absorption by Tonsils, Arch. f. Laryn. u. Rhin., 1898, No. 8, p. 476.

4. Absorption by Tonsils, Therap. Woch., v. Schnirer, Oct. 17, 1897.

5. Die Drüsen des Waldeyer'schen Schlundringes, special die Gaumentonsillen betrachtet als Eingangspforte für Micro-organism, Arch. Ital. di Laringol., April 1, 1900.

6. The Mucous Membrane of the Throat as a Port of Entrance for Pyogenic Infection, Arch. f. klin. Chir., 1897, vol. liv, p. 736.

7. Untersuchungen über die Tuberculose der Halsdrüsen, besonders über ihre Beziehungen zur Tuberculose der Tonsillen, Inaug. Diss. Zurich, 1893, u. Virchow's Arch., 1893, vol. cxxiv.

8. Die Tuberculose der Tonsillen bei Kindern, Berlin. Klinik, 1896, vol. xcix.

9. Latent Tuberculosis of the Tonsil, British Medical Journal, May 7, 1898; Lancet, June 18, 1898.

10. Untersuchungen über die im Rachen befindlichen Eingangspforten der Tuberculose, Berlin. klin. Woch., 1903, vol. xi, p. 27.

11. Ueber sekundäre Erkrankung der Mandeln und der Halsdrüsen an der Zungenwurzel bei Schwindsüchtigen, Ziegler's Beitr. zur pathol. Anat., vol. x, p. 481.

12. Ueber die Beziehungen der Tuberculose der Halslymphdrüsen zu der Tonsillen, Virchow's Archiv, vol. cxxxviii, p. 3.

13. Ueber Tuberculose der Tonsillen, Virchow's Archiv, 1884, vol. xcvi, p. 319.

14. Die Tuberculose der Tonsilla palatina, Diss., Würzburg, 1899.

15. Die W. Meyer'sche Mandel, ein Beitrag zur Bacteriologie der Adenoidgeschwülste, These de Paris, 1897.

16. Sur la tuberculose larvée de trois amygdales, Semaine Médicale, 1895, p. 223.

losis of the lungs, lesions should be found which are characterized by epithelioid cells, giant cells and necrosis, these lesions must be looked on as tuberculous, and I believe that the above statistics are practically correct.

I have made postmortem examinations of the tonsils of nine patients, in whom death was due to tuberculosis of the lungs, and in three of these the tonsils were so much involved that a diagnosis of tuberculosis was readily made macroscopically. In the remaining six cases tubercles and giant cells were found when sections of the tonsils were examined under the microscope. In one case numerous sections had to be made before a tuberculous nodule could be detected, but this tonsil had practically disappeared, being represented only by invaginations of intact squamous epithelium and a thin layer of lymphoid cells. Schlesinger is probably not far wrong when he says that in active pulmonary phthisis tuberculosis of the tonsils is almost always co-existent.

The important element in the secondary infection of the tonsils is the presence of tubercle bacilli in the sputum or other secretion which passes over the surface of the tonsil. That the tonsils can be infected by an ascending tuberculosis of the cervical lymph glands is practically impossible. It has been shown by Schlenker, Washam, Schlesinger, Kruckmann, Strassmann and perhaps others, that in obsolete involvement of the lungs, tuberculous infection of the tonsils rarely takes place. This is due to the absence of the tubercle bacilli from the sputum.

Secondary pharyngeal tuberculosis occurs in a very few number of cases by infection from the tuberculous sputum. Laryngeal tuberculosis is more common, but both of these conditions are rare as compared with the frequency with which the tonsils become diseased. To infect a tonsil it only requires that a sufficient number of tubercle bacilli be brought in contact with its surface.

PRIMARY INVOLVEMENT OF THE TONSILS.

We now come to consider the subject of primary tuberculosis of the tonsils. It is a most difficult question to decide, even at autopsy, whether a lesion in any given organ is primary or not, and it is impossible to say positively from a clinical examination that any given tuberculous lesion is the only one in the body or that it is the primary seat of the disease.

The evidence which I intend now to bring forward to show that primary tuberculosis of the tonsils occurs much more frequently than is generally supposed, is conclusive simply because of the enormous quantity of data. Any part of the evidence taken individually is perhaps in itself not unassailable, but I insist that, considered as a whole, it affords positive proof of the occurrence of primary lesions in the tonsils in a certain proportion of children.

Brown¹⁷ reported a case of a woman with a tubercular ulceration on the left tonsil. This healed up under applications of lactic acid without any involvement of other organs.

Pilliet¹⁸ found histologic evidence of primary tuberculosis of the pharyngeal tonsil in 3 out of 10 cases.

Tusseau¹⁹ has observed 3 cases of primary tuberculosis of the tonsils. He believes that it was possible for infection to take place from the food.

Broca²⁰ examined 100 cases of adenoid vegetations histologically, but in none did he find tuberculosis.

Dieulafoy²¹ found 8 cases of primary tuberculosis of the pharyngeal tonsil among 61 adenoids examined. He proved the presence of the condition by inoculation tests in guinea-pigs.

Brindel²² reports a case of undoubted primary tuberculosis in a child 8½ years old.

Plüder and Fischer²³ found two cases of primary tuberculosis of the pharyngeal tonsil out of 32 examined.

Ruge²⁴ made very careful examinations of the tonsils of 18 patients, and six times found undoubted tuberculosis.

Lermoyez and Macaigne²⁵ reported a case of tuberculosis of the tonsil characterized by tubercles with giant cells and a few Koch's bacilli.

McBride and Turner²⁶ examined 100 cases of enlarged pharyngeal tonsil and in 3 per cent. found distinct histologic evidences of tuberculosis.

Dempel²⁷ examined 15 pharyngeal tonsils from otherwise healthy individuals and found tuberculosis once.

Ozeki²⁸ reports two cases of primary tuberculosis of the tonsils.

Piff²⁹ in 100 cases of adenoid vegetations which he examined microscopically found tuberculous deposits three times.

Lewin³⁰ examined 200 cases of adenoids and in 10 of these he found tuberculous deposits. From his studies he believed that this latent tuberculosis of the tonsils can be not only the first but the only tuberculous lesion in the whole body.

Schreibner³¹ made very careful examinations of 14 faucial tonsils and 15 pharyngeal tonsils. In none was he able to find tubercle bacilli, but in two of the pharyngeal tonsils he found typical tubercles and giant cells. In 32 examinations at autopsy, while not able to prove absolutely primary infection of the tonsils, he thought it most probable that in two cases such had taken place.

Rethi³² examined 100 pharyngeal tonsils and found tuberculosis 6 times.

Hynitsche³³ reports the microscopic examination of 180 postnasal tonsils. Seven of these showed tuberculous changes. Tubercle bacilli were found in three instances, while the other cases showed tubercles made up of epithelioid cells and typical Langerhan's giant cells.

Baup³⁴ in a very extensive article, probably the most

20. Adenoid Vegetationen des Nasenrachenraums. 9 Französ. Congress für Chirurgie, Paris, October, 1895.

21. Die larvirte Tuberculose der 3 Mandeln, Acad. de Med., April 30, May 7 u. 14, 1895.

22. Tubercul. ulcer. del' amygdale, de la langue et du larynx chez un enfant de 8½ ans. Gaz. hebdom. d. sc. med. d. Bordeaux, March 8, 1896.

23. Ueber primäre latente tuberculose der Rachenmandel, Arch. f. Laryn. u. Rhin., vol. iv, 1896, No. 3.

24. Tuberculosis of the Tonsils Studied from a Clinical Standpoint. Virchow's Archiv, vol. cxliv, 1896, p. 431.

25. Primäre Tuberculose der Mandeln, Soc. Anatom., Paris, June 11, 1897.

26. Nasopharyngeal Adenoids, Edinburgh Medical Journal, No. 43, p. 1, 1897.

27. Zur Frage der Tuberculose der adenoiden Wucherungen des Nasenrachenraumes und der Gaumenmandeln bei sonst gesunden Leuten, Chirurjia, September, 1898.

28. Die tuberculose der tonsilla palatina, Diss., Würzburg, 1899.

29. Hyperplasie und Tuberculose der Rachenmandel, Wiener klin. Woch., No. 39, 1899.

30. Ueber Tuberculose der Rachenmandel, Fränkel's Archiv f. Laryngologie, vol. ix, p. 377, 1899.

31. Bilden die Tonsillen häufige Eintragspforten f. die Tubercelbacillen, Deutsche med. Woch., 1899, No. 21, pp. 343-348.

32. Latente Tuberculose der Pharynxtonsillen, New York Medical Record, Aug. 4, 1900.

33. Anatomic Investigations on the Hypertrophy of the Pharyngeal Tonsil, Archives of Otolaryngology, 1900, p. 357.

34. Les Amygdales Porte d'entree de la Tuberculose, Paris, Georges Carre et C. Naud, 1900.

17. Lar. Sect. d. IX Intern. Congr., Washington, 1897, Centralbl. f. Lar., vol. iv, p. 186.

18. Ueber das Vorkommen von Riesenzellen in den Adenoiden Vegetationen des Rachens, Societe Anatomique de Paris, March 25, 1892.

19. De l'etiology de la tuberculose des amygdales et de son traitement, Lyon Med., No. 16, 1894.

carefully worked out of any that has been done on tuberculosis of the tonsils, reports the inoculation of 43 guinea-pigs with pharyngeal tonsils, only a small portion of the tonsil being used in the inoculation. One guinea-pig became tuberculous and it was in the pharyngeal tonsil with which the pig was inoculated that the tubercular lesions were found by histologic examination.

Theissen³⁵ reports the examination of 45 tonsils and in two cases found primary tuberculosis.

Latham³⁶ was careful to employ for inoculation tests only the interior portions of the tonsils. In 45 consecutive cases of children seen at autopsy he found tuberculosis of the tonsils 7 times.

Friedman³⁷ examined the tonsils obtained at autopsy from 91 children and 54 tonsils removed by operation. Of the 91 autopsies 3 showed tuberculosis of the tonsils, probably not primary, and 5 which probably were primary with secondary involvement of the lymphatic glands, intestines and bone. In only one case could tubercle bacilli be found. In 3 cases tubercle bacilli were demonstrated in smear preparations, although no histologic changes were seen in the tonsils. Only one of the 54 operative cases showed primary tuberculosis.

Tarchetti and Zanconi³⁸ inoculated guinea-pigs with fragments of adenoid growths from 17 persons, but failed in every pig to find evidences of tuberculous infection.

Macfayden and Macconkey³⁹ found absolutely negative results from the inoculation of guinea-pigs with portions of 44 pharyngeal tonsils and 34 faucial tonsils.

Ito⁴⁰ in 104 autopsies failed to find one case of true primary tuberculosis of the tonsils, but in only 10 cases was the tonsil examined microscopically.

Johnston⁴¹ reports one case of primary tuberculosis of the tonsils.

Glas⁴² reports two cases of primary tuberculosis of the tonsils.

Name.	Cases examined.	Tonsillar tuberculosis.
Pillet	10	3
Broca	100	0
Dieulafoy	61	8
Pluder and Fischer	32	2
Ruge	18	6
McBride and Turner	100	3
Dempel	15	1
Piff	100	3
Lewin	200	10
Schreibner	29	2
Rethl	100	6
Hynitsche	180	7
Baup	43	1
Theissen	45	2
Latham	45	7
Friedman	145	6
Tarchetti and Zanconi	17	0
Ito	10	0
Macfayden and Macconkey	78	0
	1,328	67

To these cases may be added a few more statistics gathered by Richardson⁴² which I have been unable myself to find.

35. Quoted from Baup (34).

36. Lancet, Dec. 22, 1900.

37. Deutsche med. Woch., June 14, 1900, No. 24; also Ziegler's Beiträge zur path. Anatomie, 1900, vol. xxviii, p. 66; abstract by Dr. A. O. J. Kelly, Amer. Jour. Med. Sciences, February, 1902, p. 292.

38. Beitrag zum Studium der latenten Tuberkulose der Tonsillen und der adenoiden Vegetationen, Gazzet. degli ospedali e delle cliniche, Sept. 7, 1902.

39. An Experimental Examination of Mesenteric Glands, Tonsils and Adenoids, British Medical Journal, July 18, 1903, p. 129.

40. Primary Tuberculosis of the Tonsils, Carolina Med. Jour., 1903, vol. xlix, p. 369.

41. Zur Pathologie der Tuberkulose der Gaumentonsillen, Wien. klin. Woch., No. 36, 1903.

42. The Pathology of Adenoid Growths: The Laryngoscope, 1900, p. 327.

Name.	Cases examined.	Tonsillar tuberculosis.
Lermoyez	32	2
Gottstein	33	4
Grindel	68	8
Wex	210	7
	343	21
	1,328	67
	1,671	88

This gives us a collection of 1,671 cases in which the tonsillar tissues of the throat were examined for tuberculous lesions, which were found in 88 cases, that is, in 5.2 per cent. of the whole number examined. In the majority of the cases which were diagnosed as tuberculosis the microscope was the means of examination, although some of the observers inoculated guinea-pigs. It is to be noted that in the inoculation tests the percentage of lesions found was markedly lowered. Many claim that the only method of investigating the presence or absence of tuberculosis is by animal inoculations, and that results so obtained should have more weight than those from histologic examination.

In the first place some very careful investigators have obtained by inoculation results showing more than 5.2 per cent. of primary tuberculosis of enlarged tonsils, while other equally careful observers have failed to find any tonsillar tuberculosis. This discrepancy between observers using the same method argues for a faulty technic on some one's part. Personally, I believe that for the purpose of finding isolated or small tuberculous lesions in the tonsils, the inoculation test is inadequate. It is a known fact that to produce tuberculosis in guinea-pigs or other animals a sufficient number of germs must be introduced to overcome the vital resistance of the organism, and in order to produce lesions sufficiently severe to kill the animal the dosage of germs has to be considerable. The consensus of opinion is that in tuberculous lesions of the tonsils very few bacilli are present and in primary tuberculosis of the tonsils the lesion may be so small as only to be detected after numerous sections have been made. Further, a large number of guinea-pigs inoculated with portions of the faucial and pharyngeal tonsils die from rapid septic infection before tuberculous changes have had time to develop. Therefore, the claim that the presence or absence of tonsillar tuberculosis can be absolutely proven by inoculating a fractional part of a tonsil into a guinea-pig seems to me to be an unscientific one.

I have examined about fifty faucial tonsils which were removed from otherwise apparently healthy children and found one case of undoubted tuberculosis. In about thirty pharyngeal tonsils examined I have three times found characteristic tubercles.

There is one important fact brought out by the statistics which have been given and that is that primary tuberculosis of the pharyngeal tonsil is much more frequent than primary disease of the faucial tonsils. On the other hand, in secondary involvement of the tonsils the faucial tonsil is the one most frequently diseased. The explanation of this is simple. In most cases of secondary tuberculosis the sputum comes more in contact with the faucial tonsil than it does with the pharyngeal. In primary involvement of the pharyngeal tonsil, I believe that the inspired air is the chief source of infection. That the tubercle bacilli may enter the nasal fossæ and lodge on the nasal mucous membranes has been shown by Strauss,⁴³ Jones⁴⁴ and others, who have

43. Bull. de l'Acad. de Med., Paris, 1884, vol. xxxiii, No. 2, p. 8.

44. The Presence of Virulent Tubercle Bacilli in the Healthy Nasal Cavities of Healthy Persons. Med. Rec., Aug. 25, 1900, vol. lviii, p. 285.

found virulent tubercle bacilli in normal nasal cavities and in the nasopharynx. The retention in the nose of all forms of bacteria is favored by hypertrophy of the pharyngeal tonsil. The normal secretions of the nasal fossæ lose their vital bactericidal property when overcharged with organisms. The raising of the soft palate during swallowing forces any mucus with the contained germs directly into the crypts and folds of the enlarged pharyngeal tonsil.

Tuberculous adenitis of the cervical glands is, as we all know, one of the most frequent surgical conditions occurring in children. For many years the tuberculous nature of this condition has been recognized and it has also been known that it could exist as practically an isolated lesion and be recovered from without the involvement of other organs. The majority of text-books on surgery suggest that in cervical adenitis the tubercle bacilli enters either through abrasions of the skin or mucous membrane, through the mucosa of the nasal fossæ, through lesions in the middle ear and in every possible and impossible way, except by the gate through which they really do enter, namely, the tonsils. I believe that in about 90 per cent. of tuberculous infection of the glands of the neck the tonsils are the portals for the infection. As an illustration let me briefly quote the following case:

A colored child 3 years old, extremely poorly nourished, died as the result of a retropharyngeal abscess. At the postmortem the upper deep lateral pharyngeal glands were found caseous and enlarged. The retropharyngeal space, by the breaking down of the retropharyngeal glands, had been converted into a large abscess holding two or three ounces of purulent material. The bronchial and mesenteric lymph glands showed tuberculosis and there was a small abscess at about the fourth dorsal vertebra. There were beginning tuberculous changes of the spleen, but no involvement of the lungs or kidneys. The most interesting point in the case was the finding of distinct tuberculous lesions in the pharyngeal tonsil. The pharyngeal tonsil drains directly into the retropharyngeal glands and through or by them to the upper deep cervical glands. As these groups were the only glands of the neck involved it is not difficult to trace the connection between the lesion in the adenoids and the consequent involvement of the lymphatics.

It seems scarcely probable that in tuberculous adenitis the tubercle bacilli ever gains entrance through the intact normal mucosa of the pharynx. The epithelium covering the mucous membranes of the mouth and throat is of the squamous pavement variety and is especially adapted as a protective covering. The surface of the tonsillar tissues is covered with the same epithelium, but in the invaginations which form the crypts, the epithelium becomes so differentiated that its normal protective function is lost. In no other place in the throat is so good an opportunity provided for bacilli to gain access to the subepithelial tissues.

A series of experiments which I made on guinea-pigs demonstrates the difficulty of inoculating tubercle bacilli through the throat of an animal in which the tonsillar tissue is absent or rudimentary. The microscopic examination of a series of fifteen sections cut from above downward through the pharynx and including the upper part of the larynx, failed to show any more than a trace of lymphoid accumulation beneath the epithelium. We can safely say that guinea-pigs do not have tonsils. The method of inoculation in these

pigs was to place as much as possible of a pure culture of the mastoid process. They are dependent for their mal's mouth.

Guinea-pig No. 1 was inoculated on March 18, 19, 21, 22, 23, 24, 25, 26, and killed on March 28 with chloroform. At the postmortem the submaxillary lymphatics and other cervical lymph glands varied in size from a millet seed to an oval form not more than one-fourth inch long. No traces of tuberculosis could be found in any portion of the body.

Microscopic Examination.—The microscopic examination showed a slight proliferative inflammation of the cervical lymphatics.

Guinea-pig No. 2 was inoculated in the same manner and at the same time as guinea-pig No. 1, but was not killed until April 7. At the postmortem the submaxillary glands were very slightly enlarged, possibly a little more so than in guinea-pig No. 1. There was one slightly enlarged bronchial gland, and a small white node on the left kidney. There was no lesion in the lungs or other viscera, and no enlargement of the mesenteric glands.

Microscopic Examination.—Microscopic examination showed no tuberculous lesions.

Guinea-pig No. 3, inoculated in the same manner and at the same time as guinea-pig No. 1, died on May 9.

Postmortem examination showed marked enlargement of the submaxillary glands and of the deep cervical. The largest of the cervical glands measured one-fourth inch in diameter. The bronchial glands were matted in one large mass, measuring $\frac{1}{2}$ by $\frac{3}{4}$ of an inch. The mesenteric glands were markedly enlarged, almost twice as large as the cervical or bronchial glands. The lungs were congested and showed scattered miliary tubercles.

Microscopic Examination.—The cervical lymph glands, the bronchial glands and the mesenteric glands all showed distinct tuberculous disease. In all numerous tubercle bacilli were found. In the lungs there was congestion and catarrhal pneumonia, but no distinct tubercles and no bacilli.

Summary.—The first guinea-pig, killed ten days after the first inoculation, did not show tuberculosis. The second guinea-pig, killed twenty days after the first inoculation, also did not show tuberculosis. The third guinea-pig, dying fifty-two days after the first inoculation, showed a general tuberculosis of the lymph glands, those of the mesentery being most diseased. The case is evidently one of tuberculous infection taking place through the gastrointestinal tract. The inference to be drawn from these three experiments is that, in the absence of tonsillar tissues in the throat, the tubercle bacilli will infect the mesenteric glands as easily, if not more readily, than they will either the cervical or bronchial glands.

Baup³⁴ attempted to produce tonsillar tuberculosis in two dogs and seven rabbits. He failed entirely with dogs on account of the rudimentary condition of the tonsils. In rabbits, however, he found it very easy to produce a pharyngeal tuberculosis and could limit the lesion to the tonsils by careful manipulation and regulation of the dose. In animals thus inoculated, thoracic and abdominal tuberculosis was very rare, but involvement of the glands of the neck almost constant. He believes that the infection took place through the tonsils though he did not find the tubercle bacilli in the tonsillar tissue. Ravenel⁴⁵ infected four hogs through the mouth by feeding them ten meals containing pure cultures of the tubercle bacilli. All of the animals contracted general tuberculosis, most marked in the lungs and ending in death. In three of the four hogs ulcerative lesions of the tonsils were present, and in the fourth many tubercle bacilli and areas of caseation were found by the microscope in the tonsils.

The throat of the hog is well supplied with tonsils.

45. The Passage of Tubercle Bacilli Through the Normal Intestinal Wall, The Jour. of Med. Research, vol. x, No. 3, pp. 460-462, December, 1903.

Though macroscopically they do not conform to the shape of the tonsils in the human being, they resemble it more or less closely under the microscope.

The following experiments were made in the laboratory of the State Live-Stock Sanitary Board of Pennsylvania.

General Outline of Experiments.—Five pigs were used in this series of experiments and were all inoculated in the same manner. The animal was held by an assistant and the jaws kept open by a piece of wood placed between the molar teeth. A cotton swab saturated with a virulent culture of bovine tuberculosis was rubbed over the posterior portion of the mouth so that it probably came in contact with the surface of the tonsils. It is possible that a number of the bacilli were directly rubbed into the crypts of the tonsil. There was no reason to believe that any lesion of the epithelium was produced during these manipulations.

After varying intervals of time the hogs were killed by being knocked on the head and bled through the chest. In making the postmortem examinations an incision was made in the median line of the body from the symphysis of the lower jaw to the symphysis of the pubic bone. The tongue, pharynx, nasopharynx, the glands and vessels of the neck, larynx and esophagus were all dissected out in one piece. The thoracic and abdominal viscera were removed from the body, carefully inspected, and all suspicious parts saved for microscopic examination. Portions of the tonsils and submaxillary lymphatics were taken for microscopic examination.

Hog 66.—April 27. A pure culture of virulent bovine tuberculosis was rubbed over the posterior portions of the mouth. May 19, animal killed.

Postmortem Examination.—Pathologic diagnosis: Beginning tuberculosis of the tonsils and cervical lymph glands.

Pharynx and tonsils: No lesion visible.

Cervical lymphatics: The submaxillary glands were enlarged, almost the size of an English walnut, and on section a few tuberculous areas were seen. The lower cervical glands were very slightly enlarged, about the size of a pea.

Lungs: No lesion visible.

Bronchial and mesenteric glands: Not enlarged.

Liver and spleen: No lesion visible.

Intestines: No lesion visible externally.

Microscopic Examination.—Tonsil: There were a few small accumulations of polymorphonuclear leucocytes surrounded by a narrow zone of epithelioid cells. These beginning tubercles were rich in tubercle bacilli.

Submaxillary lymphatic glands: Several distinct tubercles scattered through the substance of the gland, made up of epithelioid cells with central cores of polymorphonuclear leucocytes. A few giant cells were seen. Numerous tubercle bacilli were found.

Mesenteric glands: One or two small areas closely resembling tubercles were found. No tubercle bacilli were found.

Remarks.—There was only one inoculation in this animal, and twenty-two days had elapsed between the inoculation and the time of death. The lesion in the mesenteric gland was probably not tuberculous. The finding of the tubercle bacilli in the tonsil and in the submaxillary gland proved the presence of tuberculosis. The important feature of this case was the non-involvement of the lungs or of any other organ except the tonsil and adjacent lymph glands.

Hog 56.—A pure culture of virulent bovine tuberculosis was rubbed over the posterior portion of the mouth, April 18, 27, May 10, 11:30 a. m. May 10, 3:30 p. m., the animal was killed.

One c.cm. of an emulsion made from the right submaxillary lymphatic gland was inoculated into the peritoneal cavity of a guinea-pig.

June 1, guinea-pig died. The postmortem examination showed the presence of tuberculous peritonitis, involving the parietal peritoneum and the omentum. The lungs were apparently normal. Numerous tubercle bacilli were found in the omentum.

Postmortem Examination.—Pathologic diagnosis: Primary tuberculosis of the tonsils, with secondary involvement of the

cervical lymphatics. Miliary tuberculosis of the lungs. Miliary tuberculosis of the liver.

Pharynx and tonsils: No lesions visible.

Cervical lymphatics: The submaxillary glands were markedly enlarged, almost the size of a hen's egg, the whole group measuring about 3 by 2 by 1 inches. On section the glands showed advanced caseation. The lower cervical glands were also enlarged, although the disease was not as far advanced as in the submaxillary glands.

Lungs: Numerous miliary tubercles were scattered over the surface, and through the interior. In the smaller lobes the disease was more advanced than in the larger ones.

Bronchial and mesenteric glands: Not enlarged.

Liver: A few whitish nodules were seen on the surface.

Spleen and intestines: No lesion visible externally.

Microscopic Examination.—Tonsils: About half of the section examined was normal; the remaining portion was made up of illy defined tubercles, consisting of epithelioid cells, necrotic tissue and polymorphonuclear leucocytes. Giant cells were found in moderate numbers. A few tubercle bacilli were found.

Submaxillary lymph gland: Almost all of the gland was taken up by the tuberculous tissue, consisting of necrotic material, epithelioid cells, a few giant cells and numerous polymorphonuclear leucocytes. Very few tubercle bacilli were found.

Lungs: Typical miliary tubercles were found and were made up of epithelioid cells surrounding a central necrotic core. In some places there were numerous polymorphonuclear leucocytes; epithelioid cells were scattered through the whole mass. A moderate number of tubercle bacilli were found.

Liver: The nodules seen on the surface of the liver were characterized by the disappearance of normal liver cells and the presence of epithelioid cells. There was, however, no necrosis, no giant cells and no polymorphonuclear leucocytes.

Mesenteric glands: Normal.

Remarks.—In this pig, although only twenty-two days had elapsed between the first inoculation and the death of the animal, the disease was much more advanced than in the foregoing animal; this is probably due to the greater dosage, possibly also to the lessened individual resistance. The barrier formed by the lymph glands of the neck had been broken through and a distinct miliary tuberculosis of the lungs was in progress. In spite of the frequent dosage, the mesenteric glands did not in this animal show any tuberculous lesion. The production of tuberculosis in a guinea-pig by inoculating it with an emulsion of the submaxillary gland was a rather superfluous proof of the tuberculous nature of the disease.

Hog 55.—A pure culture of virulent bovine tuberculosis was rubbed over the posterior portion of the mouth, April 18, 27, and May 10. The animal was killed May 17.

Postmortem Examination.—Pathologic diagnosis: Tuberculosis of the tonsils and cervical lymphatics. Beginning miliary tuberculosis of the lungs and liver. Beginning tuberculosis of the bronchial glands and mesenteric glands.

Pharynx: No lesion visible.

Tonsils: Left tonsil showed an ulceration about the size of a split pea. On the right tonsil were a few elevated areas scarcely the size of a split pea.

Cervical lymphatics: The submaxillary glands were greatly enlarged, completely broken down and filled with pus. The lower cervical glands were considerably enlarged, and though caseous, were not so completely disintegrated as the submaxillary glands.

Lungs: Miliary tubercles were scattered over the surface. Small pneumonic areas were found in one of the smaller lobes.

Bronchial glands: Not enlarged.

Liver: A few white nodules, varying in size from a grain of wheat to a little larger than a pea, were scattered over the surface. Some of these areas seemed very diffuse and not sharply differentiated from the surrounding parenchyma.

Spleen and intestines: No lesion visible.

Mesenteric glands: Glands possibly slightly enlarged.

Microscopic Examination.—Tonsil: In the section examined a small tubercular area was seen made up of epithelioid cells, necrotic material and a few giant cells. A few small tubercles

were scattered around the periphery of the central area. No tubercle bacilli could be found.

Submaxillary glands: A peripheral zone made up of epithelioid cells, connective tissue, and a few giant cells formed the wall of one large necrotic mass.

Lungs: The miliary tubercles showed a central core of necrosis infiltrated by polymorphonuclear leucocytes and surrounded by epithelioid cells. No tubercle bacilli were found.

Bronchial glands: The presence of a few scattered tubercles made up of epithelioid cells and an occasional giant cell, but without necrosis, showed beginning tuberculous changes.

Liver: The nodule removed was made up of round cells and elongated cells approaching the connective tissue type. This tissue displaced the normal liver cells and extended to a varying degree into the interlobular trabeculae. There was no necrosis and no giant cells.

Mesenteric glands: A few tubercles were seen, made up of epithelioid cells, with surrounding central foci of polymorphonuclear leucocytes. A few giant cells could be made out, and there was beginning necrosis. No tubercle bacilli could be found.

Remarks.—The pathologic evidence in this experiment, as shown by the naked eye examination and by the use of the microscope, was sufficiently characteristic to justify a diagnosis of tuberculosis. For some reason which I can not explain I was unable to find tubercle bacilli by the ordinary carbofuchsin method. This experiment is simply confirmative of No. 56, but shows more advanced disease of the cervical lymph nodes. There was also a beginning involvement of the bronchial and mesenteric glands.

Hog 54.—A pure culture of virulent bovine tuberculosis was rubbed over the posterior portion of the mouth, April 9, 11, 14 and 18. The animal was killed May 24.

Postmortem Examination.—Pathologic diagnosis: Tuberculosis of the tonsils. Advanced tuberculosis of the cervical lymphatics. Well-marked miliary tuberculosis of the lungs. Beginning tuberculosis of the bronchial and mesenteric lymph glands.

Tonsil: Three or four punched-out ulcers, about one-fourth inch in diameter, were scattered over the surface of both faucial tonsils.

Pharynx: No lesion visible.

Cervical lymph glands: Submaxillary glands were greatly enlarged, about the size of a hen's egg; they were broken down and caseous. The lower cervical glands were considerably enlarged and well advanced in tuberculosis.

Lungs: There was well-marked miliary tuberculosis. Nodules were scattered all over the surface and through the substance of the lung, some of them beginning to be caseous.

Bronchial lymph glands: Enlarged to almost the size of a pigeon's egg. On section several tuberculous areas were seen.

Liver: A few whitish nodules were scattered over the surface. These spots did not seem to be connected with the liver parenchyma.

Intestines: No lesions visible externally.

Mesenteric glands: Somewhat enlarged, and some of them showed caseation.

Microscopic Examination.—**Tonsil:** The section examined showed marked caseation and destruction of the tonsillar substances. Very few bacilli were found.

Submaxillary gland: There was complete destruction of the glandular substance. No tubercle bacilli were found.

Lungs: Miliary tubercles were scattered through the substance of the lung, some beginning to break down and others just forming. No tubercle bacilli were found.

Bronchial glands: The glandular substance was greatly destroyed, though some normal lymphoid tissue was still present. No tubercle bacilli were found.

Mesenteric glands: There was considerable caseation, but some normal lymphoid tissue was still left. A few tubercle bacilli were found.

Remarks.—Forty-five days elapsed between the time of the first inoculation and the death of the animal. The miliary tuberculosis of the lungs was much more marked than in any of

the other hogs, also the involvement of the mesenteric and bronchial glands. The disease in the tonsils was also in a more advanced stage.

SUMMARY OF THE FOREGOING SERIES OF EXPERIMENTS.

There can be no doubt but that the disease in all of the above animals was tuberculosis. The first lesion probably appeared in the tonsil and very shortly afterward the submaxillary glands became involved. The tuberculous changes in the submaxillary gland developed much more rapidly than in any other portion of the body. As the cervical glands broke down and the barrier which they offered to the progress of the tubercle bacilli was thus removed, a miliary tuberculosis developed in the lungs. Later the bronchial glands became infected. The mesenteric glands generally showed a tuberculosis about as severe as that of the bronchial glands.

From these experiments it seems that the tonsillar tissue is more resistive to the action of the tubercle bacilli than the adjacent lymph glands. This is shown by the fact that the disease in the cervical glands was always more advanced than that of the tonsils, although the lesions appeared in the tonsil after inoculation as soon as they did in the submaxillary glands.

Ravenel⁴⁵ injected an emulsion of equal parts of melted butter and warm water containing a large number of tubercle bacilli through a tube into the stomach of a number of dogs. Tuberculosis developed in a majority of guinea-pigs inoculated with the chyle and mesenteric glands removed from these dogs four hours after their injection; and yet, in no instance was there any lesion in the intestinal wall of these dogs. Bolinger⁴⁶ furnishes further evidence along this line. Because the bacillus can pass through the epithelium of the intestinal tract there is no reason to believe that these organisms can penetrate through the whole tonsillar substance without some reaction taking place. The lymphoid tissue of the tonsil acts as a filter just in the same manner as does the lymphoid tissue of an ordinary lymph gland. That the tonsil is not so easily destroyed by the tubercle toxin as is an ordinary lymph gland is simply an indication of the resistive power of the tonsillar tissue against bacterial toxins.

METHODS OF GENERAL SYSTEMIC INFECTION.

The most important question concerning tuberculosis of the tonsils relates to the possibility of a general infection. As the efferent vessels of the tonsils drain first to the lymph glands of the neck we must have involvement of these glands before further mischief is possible. That tuberculous adenitis of the cervical lymphatics originates from infection through the tonsils has been proven clinically as well as by the above experiments, and it seems scarcely worth while to dwell on this subject further. This being so it then becomes a question as to the importance of cervical adenitis.

Without going deeply into the subject may I call attention to a few thoughts which have suggested themselves during my work on the tonsils? It is first necessary to recall the anatomy of the lymphatics of the neck and especially the termination of their efferent vessels. Practically all the lymph of the head and neck empties finally into the internal jugular chain of glands. A collecting trunk from the internal jugular chain unites with the efferent vessels of the subclavian glands and forms the jugular trunk. On the right side this lymph-

46. Ueber d. Infektionswege des tuberculösen Giftes, M. M. W., 1890, August 19, No. 33, p. 567.

phatic trunk terminates in the internal jugular vein at its junction with the subclavian vein. The left jugular trunk usually ends in the terminal bend of the thoracic duct, but it may terminate directly at the junction of the two veins as it does on the right side. Therefore the tubercle germ, following out the cervical system of lymphatics can not possibly gain access to the body as a whole until it has entered the blood. The vessels into which the jugular trunks empty are conveying the blood directly to the lungs. These facts would lead us to suppose that the first general infection from tuberculosis of the cervical lymphatics would be a miliary tuberculosis of the lungs. This happened in every pig of the series above reported except No. 66, in which there was no involvement of the lungs. In No. 66 the barrier to the progress of the bacillus offered by the cervical lymph glands had not yet been overcome, so that infection of the lung by this route was not possible.

We must remember, however, that bacteria may gain access to the general system from a localized area by direct inoculation of the vascular system from a breaking-down foci, and also by the extension of the disease through the continuity of structure. In tuberculosis of the cervical lymph glands the tubercle, before breaking down, becomes surrounded by a distinct fibrous capsule, so that the local inoculation of the circulation is very rare. Extension of the disease by continuity of structure, as, for instance, the production of a cervical Pott's, is not outside the range of possibility. However, so far as the lymph glands of the neck are concerned, I think we are safe in saying that these two methods of extension of tuberculosis have killed very few people.

In an adenitis arising from a tonsillar infection the germ has to pass through two sets of glands before gaining access to the large jugular trunk. It seems, therefore, that in ordinary tuberculous cervical adenitis the danger of systemic or pulmonary infection is scarcely any greater than would come from a localized tuberculous lesion in any other portion of the body. If the Jossage is sufficiently large to quickly overcome the vital resistance of the lymphatic tissues a general miliary tuberculosis of the lungs is to be expected, but the rarity of this condition following tonsillar involvement is shown by a study of phthisical patients. In these cases, as has been before stated, a large number of bacilli are brought in almost constant contact with the surface of the tonsils. While in the majority of cases of phthisis there is infection of the tonsils, in comparatively few is there very marked involvement of the cervical lymphatics, and the occurrence of a miliary tuberculosis following breaking down of the lymphatic glands of the neck is a very rare occurrence.

The infrequency of glandular involvement in this secondary tonsillar tuberculosis seems at first sight inconsistent with the idea which has been advanced in this paper concerning the origin of the etiology of tubercular adenitis. We must remember, however, that in cases which have had perhaps for years an active tuberculous process in their lungs, there is developed a certain degree of immunity. For this reason secondary inoculations of the various portions of the upper respiratory tract outside of the tonsils are comparatively rare. When we consider that the whole of the trachea, the larynx, the pharynx and the mouth are almost continuously bathed with multitudes of virulent organisms, it is remarkable that a general pharyngeal tuberculosis is scarcely ever seen during life, and that laryngeal tuberculosis is the exception and not the rule.

Baumgarten,⁴⁷ in a series of experiments on animals, demonstrated that tubercle bacilli when placed in the unwounded urethra and bladder cause production of tuberculosis in the lungs. To obtain these results, however, it was necessary to use the most virulent bacilli and in not too great a quantity. Similar results were obtained by subcutaneous and intraocular inoculations. He believed that these results were due to the fact that only a few bacilli obtain an entrance to the general circulation, and that they were gradually lodged in the point of predilection, namely, the lungs. If these results obtained by Baumgarten could be confirmed by clinical data we would have to acknowledge that a tuberculous lesion, no matter how small or in what part of the body it is placed, is of very serious import. Osler believes that scarcely a man reaches the age of forty-five without having had a tuberculous lesion in some portion of the body, and this view is accepted by the majority of experts on tuberculosis. From a careful study of the foregoing experiments, and of a rather extended literature, it seems to me that tuberculosis of the cervical lymphatics becomes dangerous only when the glands break down, and the possibility of miliary tuberculosis is to be feared.

CONCLUSIONS.

In conclusion I desire to summarize the more important facts which have been suggested by the foregoing study.

The tonsillar tissue of the throat, because of its peculiar anatomic construction and its topographical relations, is more liable to become infected by tuberculosis than any other part of the upper respiratory tract. In nearly all cases of advanced pulmonary phthisis the faucial tonsils become inoculated. In about 5 per cent. of hypertrophied pharyngeal tonsils some form of primary tuberculosis will be found. Primary infection of the faucial tonsil is a rarer condition.

Tuberculous adenitis in the cervical lymphatics develops in the majority of cases from infection originating sometimes in the faucial tonsils, but more frequently in the pharyngeal tonsil.

The tubercle bacillus is probably unable to pass through the tonsils without having first overcome the vital resistance of the tonsillar tissue.

The danger of systemic or pulmonic infection resulting from a tuberculous lesion in the tonsillar tissues of the throat is about equal to that of tuberculosis of the cervical lymphatics. The lesion to be expected as a resultant infection from the broken-down glands of the neck is a miliary tuberculosis of the lungs. Further than this possibility, tuberculosis of the lymph glands of the neck is no more dangerous than a localized tuberculous lesion in any other portion of the body.

The tonsils are more resistant to the action of bacterial toxins than ordinary lymphoid tissue.

DISCUSSION.

DR. HENRY L. SWAIN, New Haven, Conn.—In considering the question of tubercular deposits in the tonsils, we have to keep in mind the fact that the latter are in every sense of the word lymphatic glands. Tubercular infection in the tonsil is no more important than infection of a lymphatic gland in the neck. We also have to consider whether the tonsil has any greater defense against infection than the other portions of the mucous membrane of this region. We can answer this question in the affirmative, for it possesses phagocytes which the other portions of the nose and throat do not. There is just as much lymphoid tissue present in the columns of the pharynx as in the small tonsil of some individuals. If the tonsil is provided with

47. *Wien. med. Woch.*, 1901, Nov. 2, p. 2049.

these agents for protection, would it not be safer for the infection to occur through the tonsil than through other portions of the nasopharynx? There are always leucocytes wandering outward between the cells of the epithelium of the pharynx and nasopharynx, and it is through these openings that the tubercle bacilli enter when the tonsil is infected. Around the mouths of the racemose mucous glands we frequently see masses of lymphoid tissue which we term follicles, which are just as capable of infecting the system as the pharynx or the faucial tonsils. When 1,671 cases have been found in literature, and only 5 per cent. of them with tubercular tonsils in non-tubercular individuals, the percentage is seen to be very small. A much greater per cent. of these individuals, according to prevailing theories, must have had some form of tubercular infection if they live to 30 years of age; the tonsil is to blame in but 5 per cent. The tonsil is probably not the only point in the upper air passages through which the infection can enter. If it be true that the healthy tonsil protects itself against the invading host of germs, ought we so resolutely to bend our efforts to the eradication of this tissue? In all our literature on tuberculosis it is evident that the lung is the place of least resistance. Why is it that when we have an infection in any part of the body that the lung is the place that gets the final infection? The question has not been entirely settled, but I think sufficient attention has not been paid to the investigations of Dr. Miller of the University of Wisconsin, who has given especial attention to the study of the minute anatomy of the lungs. He finds that there are islands of lymphoid tissue well down toward the lobules. I believe that we harbor the germs in our bronchial glands or elsewhere in the system, and they are finally presented to the circulation in the lungs. The germs then multiply, especially in these lymphoid portions. Dr. Wood's work shows the absolute possibility of infecting the tonsils by a very simple process, and his observations concerning the production of military tuberculosis in certain cases are most interesting.

DR. J. J. KYLE, Indianapolis—One reason why secondary tuberculosis of the tonsil has been overlooked is because we have been considering tuberculosis as an incurable disease. At the present time we know that the majority of cases of tuberculosis of the lung get well under favorable circumstances. I can not but think that in the majority of cases it will become a routine practice to remove the tonsils in all cases of tuberculosis of the lungs. In a number of cases coming under observation of hyperemia of the tonsil and pharynx with tubercular infiltration, removal of the tonsil has effected changes for the better in the metabolism of the patient.

DR. C. M. COBB, Boston—One point brought out in the paper seemed to me of special importance, namely, that the point of entrance was diseased. Quite a large number of cases of sub-adenitis do not have active tuberculosis of the tonsil or ulceration of the tonsil; if the infection is tubercular, it either comes from some other part of the body or else the adenitis is due to the presence of some other bacteria.

DR. EMIL MAYER, New York City—The practical side of Dr. Wood's paper is that primary tuberculosis of the upper air passages is found in the pharyngeal tonsil at an early date, and we are, perhaps, too lax in not examining lymphoid growths that we remove. A case in point is that of a young lady who was referred to me for a slight irritation in her throat. Her physician found evidences of consolidation, and desired all irritative conditions attended to. I found adenoids and removed them; these on examination were found to contain tubercle bacilli. An early diagnosis of tuberculosis was thus made possible long before it could be established in any other way, to the ultimate benefit to the patient.

DR. ROBERT C. MYLES, New York City—It has been a question with me ever since I began the study of diseases of the respiratory tract that if 80 or 90 per cent. of people have tuberculosis in some form, and 12 per cent. die, in what way does the infection take place? I believe it is through some of the lymphatics of the respiratory tract. Absorption may occur in the small area between the rhinopharynx and the larynx and be carried through the circulation into the lungs. That has

always been my idea, but I could never prove it, and Dr. Wood is now doing this.

DR. E. FLETCHER INGALLS, Chicago—Is the danger of infection greater through diseased tonsils than through healthy tonsils? The significance of the number of cases in which the bacillus has been found in the tonsils is hard to determine. Dr. Swain placed the percentage of tuberculosis very low as compared to the figures of Nageli. His thorough examination of 500 successive bodies showed the tubercle bacillus present in 97 per cent., and as he did not find it in any children under 1 year of age, and in only a few cases of children under the age of 6, it appears that practically every adult had the infection somewhere in the body. So it is not singular that it is found in the tonsil in 5 per cent. of cases.

DR. NORVAL H. PIERCE, Chicago—The presence of epithelioid cells and giant cells around a common center is not conclusive evidence of tuberculosis. I believe the only real conclusive evidence is the reproduction of tuberculosis after the infection of animals, and not even this can be considered absolute evidence, because the bacilli may have been taken from the surface of the tonsil when the emulsion is made. I do not think we should be led by this paper to look on every tonsil as an enemy, and that we should not operate on every tonsil we find to be slightly enlarged. I believe too much of that is being done; as we can never take out the entire tonsil, it is better not to touch it at all than to leave a mutilated base which will be all the easier to infect. Of course, where the tonsil is enlarged so much as to interfere with respiration or speech, or where there is demonstrable disease of the crypts, then it should be removed; but this should not be done simply because it may become the seat of disease.

DR. E. PYNCHON, Chicago—Any tonsil that pours out a diseased secretion can not be a healthy tonsil. Any tonsil that can absorb tubercular germs is not a healthy tonsil, and it is advisable to totally remove it. It must be remembered that a tubercular patient is in position to receive a fresh infection, and, therefore, these diseased tonsils, when present, should not be allowed to remain. I have frequently done this operation on tubercular patients, with the most favorable results. As regards the failure to thoroughly remove the tonsil, I claim that if by my method I can convert that space into mucous membrane free from all visible follicles, leaving it as pink as the roof of the mouth, I have practically cured the condition and thoroughly removed the growth.

DR. G. V. WOOLLEN, Indianapolis—In 1888 I said there was no such thing as a tonsil as an anatomic structure, and I have repeated that statement on several occasions since, and have been freely criticised for it. We go on talking about healthy tonsils and diseased tonsils as if there were such a distinction. All tonsils are diseased tonsils; they are pathologic and not anatomic products. If you view this subject from the standpoint of diseased throats, making the examination of people promiscuously, you will find a small percentage of tonsils. Since 1888 I have made a record of every case examined and noted all kinds of tonsils, large and small. I have never looked over my record, but I think I am safe in saying that not more than 25 per cent. of patients examined show anything like a tonsil. If my observations are correct, they play a small part in the production of disease. One out of every five people dies of tuberculosis; it is the great white plague. If these figures be correct, and if only 25 per cent. of diseased throats have tonsils, and 5 per cent. of this 25 per cent. have infection, it greatly minimizes the matter. I throw this out as a suggestion, and not as criticism, for I want Dr. Wood to continue his investigations.

DR. EDWARD R. BALDWIN, Saranac Lake, N. Y.—There is difficulty encountered in the technic of experimentation and there are few experiments which have brought much of value to the study of this subject. The question at the present day is regarded as one of the most important in the whole field of medicine. It is being fought over throughout Europe, whether the invasion of tuberculosis is through the intestine, tonsil or nasopharynx; or whether it is inhaled directly into the lung. The

most persistent advocate of tonsillar invasion is Aufrecht, who has held that the nasopharynx is the point of entrance, going from thence into the blood vessels. It is an interesting question whether the bacillus can pass through a healthy tonsil or a diseased (not ulcerated) tonsil, without leaving behind any trace of its passage. There seems to be good evidence that it can do so. The question to be worked out is: Can the bacillus pass through the tonsil, through the chain of cervical glands, and enter the circulation without leaving this trace? Dr. Wood's work is a contribution in this line. The solution of this problem may change all our ideas regarding the point of entrance of tuberculosis. We have been believing for years that the bacillus is inhaled directly into the alveoli; this has seemed absolutely conclusive from Cornet down. Now we have evidence that the infection is by ingestion. The question is an exceedingly complex one, and experimental work is extremely difficult. There are many things to be considered before final conclusions can be reached, but it is evident that there are various methods by which the bacillus can reach the pulmonary area.

DR. GEORGE B. WOOD—I can not agree with Dr. Swain that the faucial and the pharyngeal tonsils are not the most active source of infection of the cervical lymph glands, and I do not believe that the solitary lymph follicles show a less resistance to bacterial invasion nor that they absorb infectious material any more than their comparative size would lead us to suppose. Invading bacteria are killed unless they can overcome the vital resistance of the surrounding tissues, but if they live they must by the very act of their living produce toxins which, in the case of pathogenic organisms, give rise to specific tissue reaction. Pathogenic germs can not, therefore, pass through normal tissue without some sign of their presence being manifested. The experiments of Ravenel, however, proved that tubercle bacilli could pass through the normal intestine within four hours after being placed in the gastrointestinal tract. Absorption through the intestinal wall is a different question from the passage of bacteria through lymphoid tissue. It is well known that all lymphoid tissue acts as a filter for micro-organisms, and until the resistance of the tissue is overcome this filtration is active. Hence it would seem that some lesion must exist in the tonsil before infection of the cervical lymph nodes is possible. So far as the possibility of a general infection from a tuberculous tonsil is concerned, I scarcely believe I would have my tonsil taken out because of a tuberculous lesion, any more than I would have my finger amputated if I contracted a tuberculous deposit from making an autopsy. At present I am inclined to believe that in the great majority of cases infection of the lungs takes place through the air and not through the blood. Tubercle bacilli gaining access to the lungs through the blood produce a miliary tuberculosis, whereas the ordinary tuberculous pulmonary lesion begins as a single focus in the apex, which, gradually spreading, involves the rest of the lung.

THE ETIOLOGY OF EYE STRAIN.

FROM A PHYLOGENETIC AND ONTOGENETIC STANDPOINT.

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Eye strain may be divided into two general classes—that due to irregularities in refraction and that due to muscular imbalance. To the average physician the irregularities in refraction constitute the sum total of what may be termed normal variations, in contradistinction to pathologic disorders. The consideration of the muscular insufficiencies is limited more exclusively to the specialist who justly recognizes their importance, and, to judge from the recent literature on heterophoria, the subject is still open for investigation. As an anatomist, however, I have only to deal with the anatomic facts bearing on the causes of variations and will confine myself to this side of the subject, which has hitherto

been dealt with sparingly. To understand why an organ is degenerating or subject to troubles unknown to the lower forms, it is necessary to study its comparative anatomy. I purpose to call attention to facts which in their sequence form themselves into a hypothesis that may aid in the explanation not only of the muscular difficulties but of the refraction errors as well. Strange as it may seem, muscular insufficiency is phylogenetically older than refraction error but both are intimately connected because the muscular apparatus is merely an accessory to the sense of sight. The comparative anatomy, therefore, must be followed from the lower vertebrates to the highest in the hope that the findings in some stages may throw light on the question, and a comparison must be made between the phylogeny and the ontogeny in the animal most concerned—the human being.

It may be well to state in advance how it comes that I interest myself in the province of the ophthalmologist, a field so far removed from my present line of research. I suffer from an exophoria of from 4 to 8 degrees, and while at Freiburg in Baden I consulted Professor Axenfeld. Here I learned that the insufficiency is most marked when the eyes are raised, and I was advised to hold the head well back and look down a little even when doing distance work.

About that time, an exhaustive article by G. Lindsay Johnson¹ appeared on "The Comparative Anatomy of the Mammalian Eye." It is replete with beautiful illustrations of the ophthalmoscopic appearance of the retina of practically all the mammals in the London Zoological Gardens. A chapter is also devoted to the influence of domestication and a part to the position of the eyes. Several interesting ideas developed in my study of this paper, and these, together with some grosser facts in comparative anatomy. I submitted to Professor Titchener of Cornell University, to whom I express my thanks for valuable suggestions and criticism.

I hardly deem myself qualified to discuss the practical outcome of the hypothesis I present. There may or may not be a grain of truth in the deductions from the facts, which I hope to give clearly and concisely, in this paper. A larger and more exhaustive treatise is needed to bring out all the points in detail and a greater amount of material and more thorough investigation are required than I can offer at this date. At the conclusion of the discussion on the comparative anatomy, I will review briefly the grounds for assuming that the human eye is subject to abnormality because of the domestication of civilized man from an aboriginal form—an idea which may not meet with unanimous approval. Civilized man, to my mind, is undoubtedly a domesticated aborigine and therefore is subject to the same changes from unnatural environment to which any domesticated animal is subject.

In the lower forms the eyes are placed on the sides of the head and the animal is, more or less strictly speaking, monocular in its vision. In the fish, for example, the eyes, although intrinsically different from the mammal's are moved by the same muscles, which possess relatively the same origin in the orbit and the same insertion into the eyeball. I will only consider the internal and external recti because they are the most affected in the changes in position of the eyeball.

In the pilot fish the optical axis of one eye passes through that of the other—a full 90 degree divergence

1. Transactions of the Royal Society of London.