

TUBERCULOSIS IN CHILDHOOD .

THE SOURCES AND CHANNELS OF INFECTION

WITH SPECIAL REFERENCE TO THE TONSILS

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Although many eminent Pathologists have written upon, and discussed very fully, the methods by which Tuberculosis enters and invades the system, they have arrived at conclusions so widely divergent, that I have felt myself justified in selecting this question as the subject of my Thesis. It is only by comparing the records of a very large number of cases reported by different and unbiassed observers, that conclusions reliable and permanent can be arrived at. My plan has been to analyse the post mortem records of children dying of tuberculosis or with tuberculous lesions, and to compare my results with those of other observers. With this object in view, I have collected in all, 307 cases, of which 283 died at the East London Children's Hospital, where no restriction is placed upon the number of infants admitted. The remaining 24 cases were taken from the records of the City of London Chest Hospital.

The actual number of cases suffering from tuberculosis, and even its relative frequency are difficult to obtain with any approach to accuracy.

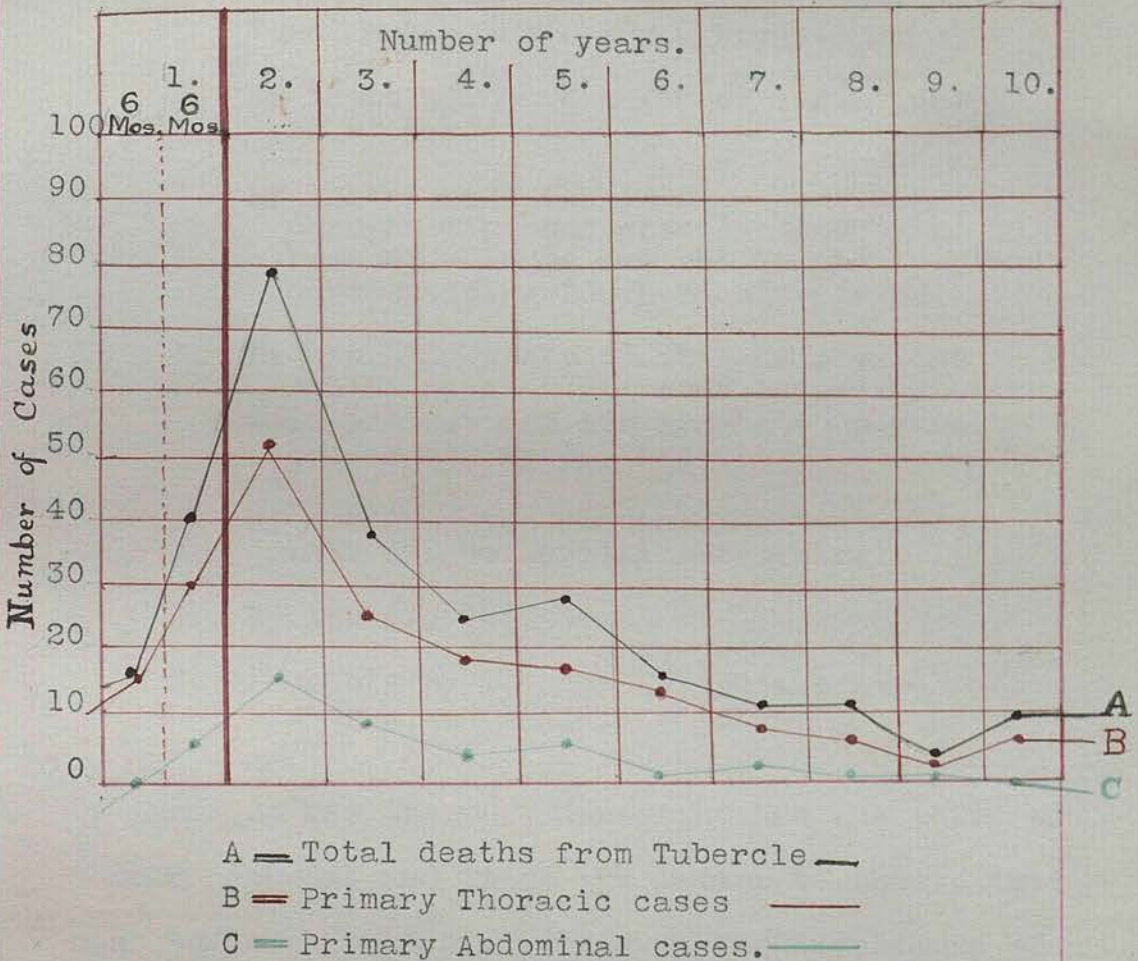
Landouzy<sup>1</sup>, having made calculations based upon the vital statistics of Paris, reported that the deaths from/

from tuberculosis in early childhood were as follows:- Under 1 year, 1 in 6; between 1 and 2 years, 1 in 4; between 2 and 5 years, 1 in 3 of the total deaths. Hospital statistics bear out Landouzy's statement of its frequency in childhood. Sturges<sup>2</sup> found from the records of Great Ormond Street Hospital that 434 out of 1420 deaths were due to tuberculosis, equivalent to 30.5%. At the East London Children's Hospital out of 1306 consecutive post mortems, 287 or 22% showed tubercle in one form or another. In 10 consecutive years 1197 out of 13150 patients admitted, or 9%, were diagnosed as suffering from some variety of tuberculosis, medical or surgical, but the mortality is of course much greater in tubercle than in the average medical or surgical cases.

Of the 307 cases collected by me, 163 were males, and 144 females, the difference being chiefly noticed during the first two years of life when there were 79 males and 56 females.

Out of 303 whose ages were recorded, 136 or 44% occurred during the first 2 years, and 234 or 70% during the first 6 years. Of 58 who died during the first year, 41 were over the age of 6 months. I made 14 the limit of age for my cases, though/

though I have very few cases over 10 years.



The above chart indicates the number of cases with tuberculosis which died at the different ages up to 10 years. It is very instructive as shewing the great frequency of the disease in the first two years of life and its steady decline after the age of 5. The chart also shews the age incidence of the cases infected through the thoracic and abdominal organs in each case displaying a close resemblance to a chart prepared by Dr. Still<sup>3</sup> in a paper on the same subject.

Primary/

Primary tuberculous infection can take place in any of the following ways:-<sup>(4)</sup>

1. Through the skin, e.g., Lupus.
2. By inoculation of a wound, e.g., Tubercular Warts.
3. By the respiratory tract - Nasopharynx, pharynx (in some cases)- trachea - bronchi - lungs, and the lymphatic glands corresponding to these structures.
4. By the alimentary tract:- namely, the mouth, pharynx (in some cases), stomach, intestines and the lymphatic glands corresponding to these.
5. By means of the placental circulation, that is, congenital tuberculosis.
6. By the eye (Holt).
7. Possibly by the genital mucous membrane; uterus and Fallopian tubes.

Of these by far the most important are the third and fourth methods, and it is the latter of these, that has led to so much discussion. The importance of this question will be seen later when the subject of abdominal tuberculosis is considered.

In ascertaining the starting point of tuberculosis in children, we meet at the outset some difficulties with which we do not have to contend in the adult. I refer especially to the frequency with which the disease becomes generalised, and with which the lymphatic glands are affected. In only 10%/

10% of my cases was the disease localized to either the thorax or the abdomen, and in 88% of the cases, caseation was found in some of the glands (thoracic, abdominal or cervical), whilst in 65% the infection distinctly commenced in the glands. It may be well here to consider the important glands in this connection, and how they are infected. The chief glands are the deep ones and are composed of five main groups.

(a) First in frequency of infection and in importance, are the glands situated in the thorax, composed of several sets, as follows:-

The Pulmonary glands, draining the alveoli, and smaller bronchi and the lung substance; the Bronchial glands, draining the medium bronchi, the pleura and the pulmonary glands; the Bifurcation glands, which lie at and below the bifurcation of the trachea, and the Tracheal glands draining the larger bronchi and trachea, as well as the other glands. There are also the Posterior Mediastinal glands which are situated in the posterior mediastinum. The lymphatics from all these glands enter the Right Lymphatic duct or the Thoracic duct.

(b) Next in importance are the glands in the abdomen, consisting of the Mesenteric glands draining/  
ing/

ing the intestines, the Coeliac glands, and some in the lesser omentum draining the stomach and duodenum, the Lumbar and Retroperitoneal glands draining the pelvis and lower glands.<sup>5</sup>

(c) The deep Cervical glands are also frequently affected, and consist of:-

1. An upper set along the carotid sheath, draining the mouth, nasal fossae, palate, tonsils and pharynx.
2. A lower set in the supraclavicular fossa, draining the upper set and communicating with the axillary and thoracic glands.<sup>6</sup>

There remain of the deep glands only (d) the axillary, and (e) the Inguinal glands, and these are of little importance, being but seldom affected.

There is still the question as to how these different glands are infected primarily, whether by the lymph stream or by the blood. The answer will depend on whether the tubercle bacilli enter the system by the lymph channels or the blood vessels. If by the former, then the thoracic glands, when they present the primary focus, will have been infected through the lungs, the mesenteric glands through the intestines, and the cervical through the mouth or pharynx.

If however, the infection usually takes place by the blood stream, as it does in certain cases, then/



then it would be possible for bacilli entering at any point of the respiratory or alimentary mucous membrane to settle in and attack any gland or group of glands predisposed to their attack, whether these glands belong to the same lymphatic area as the point of entrance or not. For instance, bacilli entering through the wall of the intestine might thus infect the cervical or thoracic glands, whilst the mesenteric glands might escape. Were this the case, it would be absolutely impossible to tell where the bacilli had entered. If blood infection were frequent, we should expect miliary tuberculosis of the lungs, or of the organs of the systemic circulation in nearly every case, and these organs should show lesions at least as old as those in the lymphatic glands. But in almost every case where there seemed evidence of a spread by the blood stream, the lymph glands showed lesions of an earlier date than the organs. The infrequency of the infection of the axillary and inguinal glands is another argument against infection by the blood stream. Some cases however, can only be explained by a primary blood infection, as for instance, cases where a joint becomes tuberculous after some slight injury, and an autopsy reveals no tubercle of the internal/

internal organs. Experiments on animals seem to prove that tubercle bacilli usually pass into the system through the lymphatic channels. For example, Cornil and Babes<sup>7</sup> introduced tubercle bacilli into the intestines of guineapigs without injury to the mucous membrane, and found that the mesenteric glands were rapidly affected. Woodhead<sup>8</sup> fed healthy guineapigs on tuberculous material and found that first the tonsil and then the cervical glands became affected. Subcutaneous injections containing bacilli produced tubercles in the connective tissue, and then in the nearest glands. To account for the frequency of the infection of the bronchial glands the suggestion has been made by Latham,<sup>9</sup> that they form the most favourable ground for the growth of the bacilli "however introduced", a comparison being drawn between these glands and the heart valves in Ulcerative Endocarditis. He, however, makes the statement that the glands are infected by the lymph stream, "though the bacilli usually enter by the alimentary tract", but he neglects to say how the bacilli are to reach the bronchial glands, and why they do not first produce changes in the mesenteric glands.

Still more recently Professor Ravenel<sup>10</sup> has again/

again advanced this theory supporting it by some experimental evidence. Out of a large number of animals fed on tuberculous material, a few after they had been killed, presented tuberculous lesions in the thorax only. To account for these cases he thinks that tubercle bacilli can pass through the healthy intestine during the process of digestion and be swept through the mesenteric glands with the lymph stream into the thoracic duct. They would thus get into the blood stream and pass on to the lungs. The result should be a miliary tuberculosis of the lungs, with the glands affected secondarily, because the glands are supplied from the left side of the heart and not by the Pulmonary artery.

I think, however, that these cases are more likely to be infected by the bacilli entering direct into the blood stream, as most pathologists are agreed that healthy glands do not allow tubercle bacilli to pass through them.<sup>11</sup>

In this paper the glands, with a few exceptions described later, have been regarded as having been infected through the organs with which they are associated, and the cases may be classified thus:-

1. Thoracic cases, infected through the lungs.
2. Abdominal cases, infected through the stomach or intestines.

3. Pharyngeal cases, infected through the mouth, pharynx, nasopharynx and middle ear.

This classification accounts for all of the cases except 34, the origins of which were doubtful or undiscoverable.

In 65% of the cases the lymphatic glands were the oldest foci found, or the only manifestation of tubercle in the body, and in many others they were the means of the dissemination of the virus, and it is principally in this that the danger in connection with them lies.

The methods of spread from the glands are:-<sup>4</sup>

1. The tubercle bacilli may extend directly into the vessels of the gland, and so enter the blood stream (Koch).
2. They may infect the serous membranes by continuity, and after producing adhesive inflammation spread to the adjoining organ.
3. The gland may soften and ulcerate into a blood vessel or bronchus, and so disseminate particles containing the tubercle bacilli.
4. They may spread by the lymph stream, e.g., the progressive caseation of the cervical glands.

Of the 307 cases, the thoracic glands were tuberculous in 241 or 78%, the mesenteric in 162 or 52%, the cervical in nearly 12%, but this is an underestimate, for in the majority of cases no note of their/

their condition had been made. As far back as 1865, attention was drawn to the frequency of caseation of the bronchial glands, Steiner and Neuritter<sup>12</sup> finding them caseous in 275 out of 302 cases or 91%. A comparison with the figures of other observers is interesting:-

	<u>Thoracic Glands.</u>	<u>Mesenteric.</u>
Batten <sup>13</sup>	83%	63%
Still <sup>14</sup>	81%	59%
Carr <sup>15</sup>	80%	54%
Simmonds <sup>16</sup>	73%	46%
Barthez and Rilliet <sup>17</sup>	72%	46%
Guthrie <sup>18</sup>	59%	40%
Woodhead <sup>19</sup>	75%	78%
Holt <sup>20</sup>	96%	35%
Bovaird <sup>21</sup>	100%	40%

The results of the first six observers, in so far as the relative frequency of the two sets of glands is concerned, closely approximate my own figures. Professor Woodhead's figures show a great relative increase and Holt's and Bovaird's a great relative decrease, in the frequency of the infection of the Mesenteric Glands. The variations in these figures indicate roughly the widely different conclusions arrived at by the different observers. The figures of Holt and Bovaird are typical of those of other American, and of most of the Continental observers. Of my cases, 105 showed caseation of the thoracic glands/

glands, whilst the mesenteric glands were healthy, and 34 shewed the mesenteric glands affected without the thoracic. In 35 cases neither set of glands was involved, and of the remaining 131 cases, where both sets were affected, 89 of them, or 68%, shewed older or more extensive lesions in the thoracic glands. Thus, in the large majority (71%) of the cases where the glands were tuberculous the thoracic glands shewed older lesions, and in addition calcification in them was of slightly more common occurrence. The importance of the thoracic glands is further shewn by the fact that 159 cases (52%) were primary in these glands, and only 23 or 7% primary in the mesenteric glands.

TABLE I.

Age	No. of Cases.	Lungs	Thorac. glands.	Pleura	Intestines.	Mesent. glands.	Peritoneum	Brain
Up to 6 mos.	17	100%	94%	53%	35%	41%	17%	29%
6-12 mos.	41	95%	87%	70%	56%	63%	26%	36%
Under 2 yrs.	78	86%	73%	41%	49%	53%	24%	39%
" 3 "	38	81%	76%	50%	50%	55%	37%	50%
" 4 "	25	88%	72%	56%	48%	56%	32%	48%
" 5 "	28	85%	82%	50%	46%	42%	35%	50%
" 6 "	17	76%	82%	53%	47%	47%	17%	47%
Over 6 "	59	81%	62%	54%	54%	47%	34%	13%
All ages	307	268=87%	241=78%	156=50%	158=51%	162=52%	100=32%	116=37%

The above table shews the percentages of the frequency with which tuberculous lesions were found in the most important organs and membranes in the different years of childhood. The cases over the age/

age of 6 have been classed together owing to the relative similarity of the figures in the different columns over that age, and to the fact that over that age the disease more nearly approaches the adult type. It will be noticed how very frequently tuberculosis occurs in the thorax in the first year, and that except in the case of the intestines, there is a decrease in its frequency over the age of 6. This is especially noticeable in the case of the thoracic glands, whose frequency falls from the average of 82.5% under 6 years to 62% over that age, whilst the average for the mesenteric glands only fell from 54% to 47%.

TABLE II.

Age	Number of Cases	Primary Thoracic	Percentage.	Primary Abdominal	Percentage.	Proportion
Under 6 mos.	17	16	94%	0	0	} 7.5 : 1
6-12 mos.	41	30	73%	6	14.6%	
Under 2 yrs.	78	53	68%	15	19%	3.5 : 1
" 3 "	38	26	68.5%	9	23.7%	3 : 1
" 4 "	25	19	76%	4	16%	4.7 : 1
" 5 "	28	18	64%	6	21.4%	3 : 1
" 6 "	17	13	76.5%	2	11.5%	6.5 : 1
Over 6 "	59	35	59%	11	18.5%	3 : 1
All Ages	307	212	69%	53	17%	4 : 1

This table shews the frequency and percentage of primary thoracic and abdominal tubercle, and the proportion between the two in the different years of/

of childhood. The rarity of primary abdominal tubercle during the first year, and its comparatively greater frequency during the second and third years, and after the sixth, will be noticed. It is evident therefore, that tubercle occurs and originates much more frequently in the thorax than in the abdomen, or, in other words that the danger from inhalation is much greater than that from ingestion.

We must now consider in more detail the 212 cases of primary thoracic infection. Of these 53 were primary in the lungs, and 159 primary in the thoracic glands.

TABLE III.

Age.	Number of Cases.	Primary Origin.			
		Lungs	Thoracic glands	Intestines	Abdominal glands.
Under 1	58	11	35	2	4
" 2	78	10	43	9	6
" 3	38	5	21	6	3
" 4	25	7	12	3	1
" 5	28	1	17	1	5
" 6	17	0	13	0	2
Over 6	59	19	16	9	2
Age not stated	2	...	2	...	...
All Ages	307	53	159	30	23

This/



This Table gives Table II. in more detail. Of the Thoracic cases the lungs were tuberculous in 95%, the glands in 91%, the pleura in 62%, the intestines in 46%, the mesenteric glands in 41%, and the peritoneum in 26%. In 19 cases the lungs were affected without the thoracic glands, and in 11 the thoracic glands without the lungs.

The lungs in children may become infected by Tuberculosis in five different ways:-<sup>22</sup>

1. Tubercle bacilli may become implanted on a catarrhal condition, such as Bronchitis. This occurred in 72 cases, as a Tuberculous Broncho-pneumonia.
2. By extension from a caseous gland. A periadenitis is set up, and the gland or glands become adherent to the adjacent lung or bronchus. The spread may then be by continuity into the lung or bronchus, or the glands may soften and ulcerate into the bronchus, thus forming small cavities at the root of the lung. The contents of these cavities are sucked into the alveoli, and miliary tubercles with a racemose distribution are the result. Carr<sup>15</sup> regards all cases of advanced pulmonary tuberculosis, in which the process is most advanced at the roots of the lungs, as the results of a spread in this manner. Forty-four of my cases were infected from the glands in this method.
3. By miliary tuberculosis, which was present in 110 cases. These miliary tubercles are either a part of a general miliary tuberculosis, or due to infection by means of the thoracic duct, or by the process invading and ulcerating into one of the pulmonary blood vessels.

4./

4. By cheesy solidification; present in 13 cases.
5. By a more chronic condition, with cavities and fibrosis, occurring chiefly in older children.

This method was found in 30 cases. In many cases miliary tuberculosis or tuberculous Broncho-pneumonia were present in addition to one of the other conditions. Primary lung tubercle was usually in the form of the Broncho-pneumonic or the chronic variety.

Inhaled bacilli are more apt to settle on the mucous membrane of the trachea and bronchi than in the alveoli, and a bronchial catarrh will make the penetration of this membrane more easy. Just below the mucous membrane of the lower part of the trachea and of all the bronchi lies an immense network of lymph vessels which lead direct to the bronchial glands.<sup>23</sup> The following experiment shows the course of these lymph channels and the route taken by tubercle bacilli from the tracheal mucous membrane. Cornil introduced a few drops of a culture of tubercle bacilli into the tracheas of guineapigs, and this led to lesions in the sub-mucous coat, and later the bacilli were found in the bronchial glands, the bifurcation glands also being/

being much enlarged. In all probability the course taken by inhaled bacilli is the same as that taken by foreign particles when inhaled. This course has been well demonstrated by Julius Arnold, who made experiments with dust inhalations. In a few hours after the inhalation the dust particles were found in the lymph spaces, the afferent vessels and the bronchial glands themselves, having been absorbed through the mucous membrane and alveoli. No particles were discovered in the efferent vessels. When the bacilli reach the glands, it will depend upon the power of resistance in those glands, whether they die, remain inactive for a long time, or set up tuberculosis in them. (Wysso-kowicz).

In some cases it is probable that no special catarrhal condition of the bronchi is necessary, but that, as in the case of the intestines (compare Dobroklonski's experiments<sup>24</sup>) tubercle bacilli can pass through the healthy mucous membrane to the glands. There are, however, some pathologists who insist that a lesion of the mucous membrane is essential, and that in children this lesion may heal without leaving a scar.<sup>11</sup>

Loomis<sup>23</sup> examined the bronchial glands from persons/

persons of all ages dying from other causes than tuberculosis, and in whom these glands showed no evidence of tubercle, and in 8 out of 30 cases he demonstrated the presence of active tubercle bacilli by inoculation. It seems therefore that, so far as the inhaled bacilli are concerned, these glands form the battle-ground on which the health and future of the organism are decided.

The frequency with which the bronchial glands show the oldest discoverable tuberculous focus has been used as an argument against the infection by "air" in these cases. The analogy is taken from the adult with phthisis, where the infection is admittedly by air, and where the lungs are affected before the glands, and it is said that if in children, the infection is by air the lungs should show older lesions than the glands. Those who use this argument leave out of account the high development of the lymphatic system in children, and the special tendency their lymphatic glands have to become inflamed, and to be infected with tubercle. A few bacilli may settle on the bronchial mucous membrane and without a severe local lesion, may get conveyed to the nearest glands. These glands, especially if they have been the seat of recent inflammation, may/

may become caseous and infect the lung secondarily. In adults it is not uncommon to find cases of fairly advanced phthisis with but little affection of the glands. Perhaps also the bacilli have less opportunity of finding a nidus in the air-passages on account of the greater activity of the lungs in young children.

It is well known that chest complaints are of extreme frequency in infancy and early childhood. Sixty-five per cent. of the cases of primary bronchitis in children occur in the first 3 years of life, (Still),<sup>14</sup> and 86% of the pneumonias in the first 2 years (Holt). Of whooping cough 20% of the cases occur in the first year (Goodhart)<sup>22</sup>, and 50% under 2 years (Still).<sup>14</sup> Not a few cases also of measles occur in the first 2 years of life. These last two diseases especially cause great irritation of the bronchial mucous membrane, and invariably inflame the bronchial glands, so that, should the tubercle bacilli enter the system about the same time they would find a suitable nidus already prepared. Probably also influenza frequently plays a part in predisposing the bronchial mucous membrane and glands.<sup>25</sup> It has been so often observed that cases/

cases of tuberculosis date back to an attack of whooping-cough, measles, or pneumonia, that these diseases have come to be recognised as important predisposing causes, and no doubt help to account for its great frequency in early childhood.

There is a marked tendency for the right lung and the thoracic glands on the right side to show lesions more extensive and of an earlier date than on the left side.

Thus:	Right lung predominating	.....	59	cases
	Left " "	.....	32	"
	Right Glands " "	.....	54	"
	Left " "	.....	30	"
<i>Total</i>	{	Right Side (Lungs & Glands)	... 92	"
		Left " " "	... 50	"
		Neither " predominating	.....	<u>165</u>
		Total	<u>307</u>	

Shennan<sup>26</sup> found the right lung more extensively affected in the proportion of 123 : 60.

Still's<sup>3</sup> figures were even more striking, for out of 269 cases the right glands predominated in 113 cases and the left in 32.

Batten<sup>13</sup>, Laennec and Williams also found the right glands more frequently affected.

Tubercular Meningitis was the cause of death in 77 of the Thoracic cases. One case of Placental infection, described later, has been included amongst the thoracic cases, as the main lesions were found in the bronchial glands.

We now pass to the subject of primary abdominal tubercle, that is, infection through the stomach, intestines or abdominal glands. This is a matter of paramount importance to medical men, especially those dealing with Public Health, to dairy farmers, and to the community at large, because on it depend the lives of thousands of children annually. The subject more or less hinges round this question, "Can milk from tuberculous cows produce Tuberculosis in the human being?" Some observers are emphatic in asserting that it can, and others almost as emphatic in their denial. Great interest was aroused by the remarks of Professor Koch<sup>27</sup> at the Tuberculosis Congress of 1901, and some surprise was created by the results of his experiments on cattle, by which he <sup>considered that he</sup> proved that human tuberculosis could not be communicated to cattle. This conclusion he still maintained at the recent Berlin Tuberculosis Congress 1902. His experiments were to introduce human tubercle bacilli into cattle by ingestion, inhalation, and inoculation, and the results were always negative, whereas the cattle were particularly susceptible to the bovine bacilli introduced in the same way. Since Koch's address several observers have come forward with experimental/

mental evidence, tending to prove that his conclusions were erroneous, being based upon an insufficient number of experiments. Nocard,<sup>30</sup> de Jong,<sup>31</sup> Arloing,<sup>32, 94</sup> and Ravenel<sup>10,33</sup> have all been successful in infecting cattle from human sources, similar previous successful infections having been reported by Chauveau<sup>28</sup> in 1891, and the 1895 Royal Commission<sup>29</sup> on Tuberculosis. Cases of accidental infection of cattle by inhalation, or ingestion with the forage of the sputum of phthisical attendants, have been recorded by Huon,<sup>34</sup> Cozette,<sup>35</sup> Cliquet<sup>36</sup> and Bang.<sup>37</sup> Professor Ravenel<sup>10</sup> admits that cattle show a much greater resisting power to the human than to the bovine type of bacilli, and that there are morphological, cultural and pathogenic differences between the two types.

Koch's arguments against the possibility of bovine tuberculosis being introduced into man, were chiefly based upon the rarity of primary intestinal tuberculosis amongst children, seeing that cow's milk, which is so frequently infected with tubercle, forms so large an element in their diet.

He quoted 933 autopsies on tuberculous children at the Emperor and Empress Frederick's Hospital, Berlin/



Berlin, without a single case of primary intestinal tubercle, and 3104 autopsies by Biedert with only 16 cases, whilst he himself had only seen two cases of primary intestinal infection in the whole course of his experience. Professor Hueppe<sup>38</sup> disputes these statistics of Koch, as not being at all representative, and makes the statement that between 25 and 35% of all children dying from tubercle have been infected by food..

A more or less direct connection between tuberculous cow's milk and human tuberculosis has been noticed in 38 cases recorded by Demme,<sup>39</sup> Lydten,<sup>40</sup> Brouardel,<sup>41</sup> Gosse,<sup>42</sup> Law,<sup>43</sup> Ebers,<sup>44</sup> Bang,<sup>45</sup> Von Ruck,<sup>46</sup> Klebs and Rievel,<sup>47</sup> Stang<sup>48</sup> and Ollivier.<sup>49</sup> In addition to these, some cases of the accidental inoculation of men with bovine bacilli have been recorded by Ravenel,<sup>50</sup> Tscherning,<sup>51</sup> Pfeiffer,<sup>52</sup> Hartzell, Lasser,<sup>53</sup> and Joseph and Trautmann,<sup>54</sup> the last four observers reporting cases of verrucous tuberculosis on the hands of butchers or of men working in the abattoirs, the cases of the other observers being of a more serious nature.

The weight of evidence against Koch's conclusions is further substantially increased by the statistics of autopsies on tuberculous children recorded/

recorded by different English and Scotch observers; and it is in support of their conclusions that I trust my paper may be of some practical value.

A quotation from these statistics will serve to make this point clear.

	No. of Cases.	Intestinal Infection	Percentage.
<u>British Observers</u>			
Guthrie <sup>18</sup>	77	19	24½%
Still <sup>3</sup>	269	62	23%
Shennan <sup>26</sup>	355	80	22½%
Carr <sup>15</sup>	120	20	16%
Ashby <sup>6</sup>	155	20	13%
Total	976	201	20%
<u>Foreign Observers</u>			
American:- (Northrup <sup>55</sup> Holt <sup>20</sup> and Bov- aird <sup>21</sup> )	369	5	1%
French:- (Summarised) <sup>59</sup>	128	0	-
German:- (Summarised) <sup>59</sup>	236	9	3%
Total	833	14	1½%

These figures are exclusive of Professor Woodhead's<sup>56</sup> for though in his paper he mentioned that primary abdominal infection was very common, he omitted to give the total number of cases thus infected. They are also exclusive of those quoted by Professor Koch, which would still further reduce the percentage/

age of intestinal infection in the statistics of foreign observers. It is no easy matter to account for the difference between the two sets of statistics, except that the Continental observers possibly do not recognise as infected through the intestines, those cases in which primary lesions are found in the mesenteric glands. But even then, judging by such cases in England, the percentage would only just be doubled, and the difference but slightly diminished.

My own figures closely resemble those of the British observers, for I found primary abdominal tuberculosis in 53 cases, or 17%, 30 being primary in the intestines, and 23 in the mesenteric glands. (See Tables II. and III.) Of these 53 cases, the intestines were tuberculous in 40, the abdominal glands in 52, the peritoneum in 33, the lungs in 36, the thoracic glands in 18, the pleura in 17, and the brain in 16. Twelve cases showed caseous mesenteric glands without any sign of ulceration of the intestines, but there was only one case where ulceration of the intestines was present without also caseation of the mesenteric glands. But of all the 307 cases, tuberculous mesenteric glands without intestinal ulceration/

tion were present in 42, the converse occurring in 37. Professor Woodhead,<sup>56</sup> the chief supporter of the theory that cow's milk can produce tuberculosis in man, found caseous mesenteric glands in 100 out of 129 cases, or 78%; 33 of these being between the ages of 1 and  $2\frac{1}{2}$  years, and 29 between 3 and  $5\frac{1}{2}$ : whilst in 14 cases these glands presented the only tuberculous lesions discovered. Amongst my cases 162 or 52% showed caseous mesenteric glands; 55 being between the ages of 1 and  $2\frac{1}{2}$ ; and 36 between 3 and  $5\frac{1}{2}$ ; but in no instance were the mesenteric glands caseous without other tuberculous lesions also being present.

Of the abdominal cases, both the abdominal and thoracic glands were tuberculous in 20, the abdominal alone in 32, of which 15 showed no tubercle at all in the thorax. There were, however, amongst the thoracic and doubtful cases 79 in which the thoracic glands were caseous without any lesions of the intestines or mesenteric glands.

From the fact that caseous mesenteric glands are not infrequently found primarily, when no lesion of the intestine can be discovered, it seems likely/

likely that they can be infected through the intestinal mucous membrane without any lesion of that membrane being necessary. This is borne out by the experiments of Professor Dobroklonski<sup>24</sup> on guineapigs, which he caused to swallow pure cultures of tubercle bacilli in glycerinated broth. Summing up the results of his experiments, he says that tuberculosis can infect the organism by the digestive passages, and that tubercle bacilli or their spores can easily pass through the healthy mucous membrane, and that without the necessity of a prolonged contact.

Perhaps ulceration of the intestine depends upon the virulence of the dose; as for instance, when a virulent dose was introduced into the intestines of guineapigs, ulceration always followed, but occasionally did not when the dose was made weaker.<sup>26</sup>

Professor Benda,<sup>11</sup> when replying to criticisms on a paper read by him at the British Tuberculosis Congress 1901, in which he drew attention to the great importance of the spread of tubercle in the infected organism by the epithelial channels, expressed a doubt that tubercle bacilli could penetrate/

trate an intact mucous membrane. He thought that in children, a lesion must be present, through which the bacilli could pass to the glands, and that this lesion, if slight, might heal without leaving a cicatrix, or any other evidence of its existence. This statement, though very easy to make, is very difficult to prove, and though the possibility of its occurrence can hardly be denied, it is at least certain that the mesenteric glands may have primary tubercle in them, without the most careful search detecting the smallest lesion in the intestinal mucous membrane.

We have other evidence besides the experiments of Dobroklonski, to show that tuberculous material introduced into the bowel by ingestion or otherwise, can produce abdominal tuberculosis, intestinal or glandular. Baumgarten<sup>57</sup> gave milk containing pure cultures of tubercle bacilli to rabbits, and never failed in producing intestinal ulceration. Wesener<sup>15</sup> injected tuberculous sputum into the intestines, and produced intense ulceration. In cases of pulmonary tuberculosis in adults and children, intestinal ulceration is frequently present as the result of swallowing/

swallowing virulent sputum. Bollinger and Gebhardt<sup>58</sup> showed that if the milk of tuberculous cows be given as the sole or principal food to cats or guinea-pigs, they all became tuberculous, but that if this milk were freely diluted with healthy milk, the animals could take it for weeks with impunity. Ostertag<sup>60</sup> says that the milk of cows reacting to Tuberculin, but exhibiting no symptoms of tuberculosis, contains no tubercle bacilli and is quite harmless. Nocard<sup>61</sup> after investigations with the milk of tuberculous cows, concludes that milk is only virulent when the udder is tuberculous, and that the ingestion of virulent milk is only dangerous when the milk contains a large number of tubercle bacilli, and is taken in considerable quantities; and further, that bringing the milk to the boiling point before drinking will suffice to render it harmless. As probably not more than 1% of cows have tuberculous udders, the mixed milk from a large dairy, should so dilute the virulence of any infection, as to render it harmless, even to the guinea-pigs and cats of Bollinger and Gebhardt.

Numerous cases of primary abdominal tubercle having been recorded in this country indicates that in the human being the virus can be introduced by the/

the digestive tract, and in infants and young children the most likely element of diet to convey the infection is milk. Breast-milk, the proper diet for the first 8 or 9 months of life, hardly ever contains tubercle bacilli; and it will be noticed that amongst my cases, there was no case of abdominal tubercle under 6 months and only 6 under 1 year (Table II). But unfortunately, in London, and I fear all over the country, a very large number of infants have the breast-milk supplemented, or entirely supplanted by cow's milk, condensed milk, or some patent food. The condensed milk and patent foods are usually prepared for use by the addition of boiling water, which should render them harmless, and the cow's milk is, as Still suggests, perhaps more likely to be boiled for infants under one year than for older children. Between the ages of one and three, but seldom after that, owing to its expense, cow's milk forms to a greater or less extent a large element in the diet among children in the East End. Consequently, if milk is an important source of infection, we ought to find primary abdominal tubercle most prevalent during those years in which unboiled cow's milk is most largely used, that is, in the 2nd and 3rd years. By reference to Table/



Table II. it will be seen that abdominal tubercle is, both relatively and absolutely, most common during this period, as 24 cases or 45% of all the abdominal cases occurred in those two years.

Other sources of infection, however, must not be overlooked. Infants just beginning to crawl about, put everything they can grasp into their mouths. Many of these articles, such as the "comforter" are frequently picked up from the floor or street, where we know human tubercle bacilli may abound. Again, it is a common habit for mothers, tuberculous or not, to put spoons containing food for the infants, into their own mouths before feeding them.<sup>62</sup> In this manner a tuberculous mother may easily infect her own child. Another source of infection, and one on which very little stress has been laid, is the swallowing with the saliva, of inhaled bacilli which may have settled in the mouth, pharynx or nasopharynx. All inhaled bacteria must pass through either the nose or the mouth before reaching the lungs, and the probability is that the majority get deposited on these moist surfaces and do not reach the lungs. The experiments of M. Straus<sup>67</sup> to which attention is drawn later, illustrate the frequency of the presence of tubercle bacilli/

bacilli in the nasal cavity. As regards meat, tuberculous nodules in meat are not of frequent occurrence, and the risk of infection from this source is much diminished by the vigilance of sanitary inspectors, and by cooking.

To test whether the ingestion, in milk or otherwise, of bovine tubercle bacilli, can produce tuberculosis in man, Koch<sup>27</sup> has suggested that inoculations should be made from the mesenteric glands of cases with primary abdominal tubercle into cattle. Should the infection be due to bovine bacilli the cattle would speedily react and become tuberculous, if to human bacilli it would be very difficult, or according to Koch impossible, to infect them. There is the possibility however, that the virulence of the bovine bacilli, supposing that the lesions were due to them, might have become somewhat attenuated during their stay in the human subject, and in this case reinfection of cattle might be rendered difficult. Experiments on similar lines to the suggestions of Professor Koch are being carried on in England and America at the present time.

Jacobi<sup>63</sup> regards as of great importance, tuberculosis of the peritoneum. In his cases he found it/

it extremely common, and almost always localized, and therefore likely to be recovered from. He says that it usually precedes, and but rarely follows, pleural or pulmonary tubercle, when there is a dissemination of the virus. He concludes that the tubercle bacilli frequently enter the free abdominal cavity and may spread from here "through the intestinal wall whether it be healthy or not." He says, however, that primary infection of the intestines or mesenteric glands is rare. In my cases the peritoneum was affected in only 103 cases or 33%, of which 55 cases were of thoracic origin, and 33 of abdominal origin. In 4 cases only did the peritoneum show the oldest focus, and in the light of other cases I should hesitate to say that these had been caused by bacilli free in the abdominal cavity. The relative frequency of peritoneal and pleural tubercle is shown in Table I.

It has been suggested as an explanation of the small number of cases of primary abdominal tuberculosis, that more children recover from abdominal tubercle than from thoracic tubercle. Certainly healed, or healing ulcers of the intestines, calcareous or earthy mesenteric glands, or peritoneal adhesions (which at present are regarded as tuberculous/

-culous in origin in the majority of cases in childhood), are occasionally met with, especially in those cases in which more recent tubercle is present. If it be true that more abdominal cases than thoracic recover, then abdominal infection should be present in a much larger proportion amongst those children dying of causes other than tubercle, than amongst those children dying directly from tubercle. These cases, in addition, ought to exhibit the modes of entrance and lines of spread of the virus, in a clearer manner than the rest, owing to the disease being in a less advanced stage. I have notes of 48 cases, which died from such causes as Diphtheria, Broncho-pneumonia, Morbus Cordis, Accident, etc. In these cases the lungs were affected in 29 (60%), the thoracic glands in 29 (60%), the intestines in 12 (25%), the mesenteric glands in 17 (35%) and the peritoneum in 10 (= 21%). The primary lesion was thoracic in 32 (lungs 7, glands 25), abdominal in 9 (intestines 5, glands 4), pharyngeal in 1, and doubtful in 6, none of which showed any lesion of either the intestine or the mesenteric glands. This shows a proportion of more than 3.5 : 1 in favour of the thoracic glands, the proportion for the remainder being rather more than 4 : 1 so that the difference is/

is not very great, nor abdominal tubercle much underestimated. Still's figures<sup>3</sup> were rather different, for, of 43 cases, he found 26 primary in the thorax, 16 in the abdomen, and 1 in the ear.

It is possible sometimes to trace the infection of one group of glands from another group, owing to the free anastomosis of the lymph channels. Professor Woodhead<sup>8</sup> says that the bronchial glands are usually infected in this way from the mesenteric glands, and not through the lungs. According to him, the routes taken by the bacilli in reaching the bronchial glands are various, for instance:-

- i. From the mesenteric glands through the diaphragm by way of the retro-peritoneal glands, thence to the posterior mediastinal, and so on to the bronchial glands. Professor Woodhead says that he has seen many autopsies on children showing this line of spread.
- ii. From the peritoneal cavity through the central tendon of diaphragm to the pleura, and thence by the lymphatics of the pleura to the root of the lung.
- iii. Adhesions may form between the liver or Spleen and diaphragm, and sometimes the adjacent visceral and parietal pleurae also may become adherent. Lymph channels are set up in the adhesions, and bacilli can pass up through these adhesions either to the root glands or to the lung direct.

His arguments are largely based upon Bollinger's and Hunter's experiments. Phthisical sputum was injected/

injected by Bollinger into the peritoneal cavity, and in 66% of the cases the lungs became tuberculous whilst the peritoneum remained healthy. Dr Wm. Hunter injected blood into the abdomen, and demonstrated the presence of blood corpuscles firstly between the liver and diaphragm, secondly in the lymphatics of the diaphragm. Durham observed that in peritonitis the chief lymph channels passed into the anterior mediastinum. I have collected altogether 10 cases which seem to show a spread in a similar manner.

A. Three cases which seem certain, e.g.,  
 A.R., 2 years. Tubercular ulceration of intestines from duodenum to rectum. Abdominal glands caseous and aggregating to form masses. Posterior mediastinal glands caseating, and one bronchial gland caseous. Advanced perihepatitis. Miliary tubercles in the lungs; spleen and meninges.

B. Three cases probable, e.g.,  
 G. A., 4 years. Liver and adjacent pleura adherent to the diaphragm. Mesenteric and thoracic glands all caseous. Lungs tuberculous.

C. Four cases in which probably the infection spread downwards from thorax to abdomen, as the thoracic glands were in a more advanced stage of caseation/

caseation than the mesenteric. This mode of spread was suggested by Shennan,<sup>26</sup> e.g., W.P., 4 years. Anterior and posterior mediastinal glands caseous and liquefying. Large caseous masses accompanying bronchi into the lobes of the lungs. Large caseous masses above the pancreas. Mesenteric glands show early caseation. Intestines not tuberculous.

In 6 of the abdominal cases the liver was found adherent to the diaphragm. No evidence of a similar line of spread was found in any of Still's Cases<sup>3</sup>.

Whilst admitting the possibility, though not the frequency, of the first line of spread, I cannot see how the other two methods are possible unless the free bacilli get into the peritoneal cavity, where, being immotile they would be more likely to set up the local tubercles which Jacobi says he has so often observed. There is no evidence, however, that the bacilli are frequently present in the peritoneal cavity. Again, for these methods of spread to be common, the mesenteric glands should frequently show older tubercle than the thoracic glands, but as mentioned previously, where the thoracic or abdominal glands were tuberculous, 71% of these cases showed the thoracic glands affected before the mesenteric.

With/

With three exceptions, all the abdominal cases showing tubercular peritonitis, old or recent, showed also caseous mesenteric glands of an older date, the line of spread being frequently by continuity from these glands.

Four cases in which the peritoneum seems to have been the oldest focus, two where the spleen only, and one where the liver and kidneys only were affected, have been classified amongst the cases of doubtful origin.

Knowing that amongst infants and young children, intestinal catarrh is more frequent in the hot summer months, and that this catarrh is usually accompanied by swelling of the mesenteric glands, I have examined all the abdominal cases to see if there is any relation between this summer catarrh and tubercle of the mesenteric glands, such as exists between bronchial catarrh and caseous bronchial glands. However, from May to October, inclusive, only 47% of these cases occurred, so that evidently summer intestinal catarrh is not a very important predisposing factor in abdominal tuberculosis.

Before leaving the subject of abdominal tubercle, I should like to refer briefly to tuberculosis of the/



the stomach, stated to be a very rare condition. I, however, found it present, always in the form of ulceration, in 11 cases, 9 of which were under  $2\frac{1}{2}$  years of age. In 8 cases the ulcers were secondary to pulmonary tubercle, being either part of the general infection, or due to swallowed sputum. In the other three cases, abdominal in origin, the stomach was infected simultaneously with the intestines in 2, and in the third probably slightly earlier, so that this may be considered a case of primary tuberculosis of the stomach. In two cases the ulceration was near the pylorus, in one on the anterior wall, in one at the cardiac end, but in the rest the position had not been specified. The glands affected were, in three cases those about the head of the pancreas and in the lesser omentum, in two the retro-peritoneal, in one, certain glands in the transverse fissure of the liver, and in the remainder merely the mesenteric glands.

Tubercular meningitis was the cause of death in 18 of the abdominal cases.

The next most important method of entrance is through the mucous membrane of

- (a) The nasal cavity, the nasopharynx, and the middle ear.
- (b) The mouth, fauces and pharynx.

(a)/

(a) I have neither seen, nor heard of a case of infection through the nasal mucous membrane in a child. Cases infected through the nasopharyngeal mucous membrane are recorded, and will be mentioned under the heading (b) along with cases infected through the tonsil. Infection through the middle ear is by way of the Eustachian tube from the nasopharynx, and are, like all other cases in this group probably infected by respiration. Two of my cases originated thus. One had otitis media, extensive caries of the petrous bone, tuberculous meningitis and acute general tuberculosis, but no caseous glands present. The other had otitis media, tuberculous meningitis and tubercles in the spleen. Both cases were under 2 years of age. Five per cent. of Still's<sup>14</sup> cases showed this mode of infection. To show the importance and prevalence of tubercle bacilli in the air, especially where there are cases of phthisis in the vicinity, M. Straus,<sup>67</sup> a Physician to two Paris Consumption Hospitals, examined the nasal cavities of certain students, nurses, and non-tuberculous patients at these hospitals, and was able by inoculation to demonstrate the presence of free and active bacilli in 9 out of 29 cases.

(b)/

(b) The cases infected through the mouth, fauces or pharynx are more likely than group (a) to be infected by means of food, though of course they may also be sometimes of respiratory origin. The bacilli usually enter through the lymphatic structures of the fauces and pharynx (tonsils, adenoid vegetations, or lymph follicles at the back of the tongue), and in older children may enter through the cavities of decayed teeth as mentioned in a paper by Starck.<sup>64</sup> The lymph sinuses of the tonsils communicate with those of the mouth, pharynx and deep cervical glands.<sup>(6)</sup> Bacilli absorbed through the structures of this group eventually reach these deep cervical glands, which may become tuberculous, and the infection may thus spread from gland to gland down the neck. Eventually the supra-clavicular glands may become involved, and from these the tracheal and other thoracic glands may also get the infection.<sup>6,65.</sup>

Lovell Gulland<sup>66</sup> in describing the structure and functions of the tonsils, says that the stream in them is always an outward one, and that therefore they are non-absorbent structures. This statement is not borne out by the experiments of Kayser<sup>68</sup> and Goodale.<sup>69</sup> The former blew fine coal dust/

dust against the tonsils, and found that the particles were rapidly absorbed into the tonsillar tissue, some being found in the tissue spaces and others carried by leucocytes, and afterwards discovered in the lymph glands. Through the tonsil the route taken was by the interfollicular lymph spaces. (Goodale).

Hendelsohn's<sup>70</sup> experiments with adenoids showed that absorption could take place through them also.

Ullman<sup>71</sup> regards as the function of the tonsils, the formation of mononuclear leucocytes, which have but little phagocytic activity. He quotes numerous diseases which have occasionally been traced to infection through the tonsils, such as:- nephritis, leucaemia, septicaemia, urticaria, endocarditis (Mayer),<sup>72</sup> rheumatism (Buss,<sup>73</sup> Trousseau, Sterling, Abraham) and typhoid fever (Capillari).<sup>74</sup> Pluder<sup>75</sup> thinks that the protection of the tonsils against invasion by disease is very small, as they are themselves frequently diseased, and therefore portals for the entrance of bacteria.

During the last seven years, considerable attention has been drawn towards the faucial tonsils/

-sils and the nasopharyngeal adenoid growths, as being likely structures through which tubercle bacilli may enter the system, but previous to this very few pathologists regarded them as of importance in this respect. In 1875 Gee described<sup>76</sup> one case of tuberculous disease of the tonsil. Purves Stewart<sup>77</sup> in 1895, published a case in which a histological examination of the tonsils, removed for hypertrophy, revealed tuberculosis, and in which caseous cervical glands developed later. In the same year Dieulafoy<sup>78</sup> read a paper on "Latent Tuberculosis of the Tonsil". He inoculated into guineapigs, portions of hypertrophied tonsils and of adenoid growths, and found that 8 out of 61 tonsils, and 7 out of 35 adenoid growths were tuberculous. He had, however, neglected to make histological examinations of them, and on that account his paper met with somewhat severe criticism, owing to the possibility of active bacilli being present in the tonsillar crypts or the folds of the adenoid mucous membrane. Ruge<sup>79</sup> published a case in which the tuberculous infection seemed to have spread directly from the tonsil to the spinal column. He also examined the tonsils of 17 cases and found tubercle bacilli in 5; Strauss-  
mann<sup>80</sup> /

mann<sup>80</sup> finding bacilli in 15 out of 17 cases; and Walsham<sup>81</sup> finding tuberculosis in 20 out of 34 cases. Baup<sup>82</sup> has recorded some cases infected through the tonsils, and Dmochowski<sup>83</sup> and Kruckmann<sup>84</sup> have also been successful in discovering tubercle bacilli in the tonsils. In children of all ages, Latham<sup>9</sup> found 7 cases out of 45 with tuberculosis of the tonsil, by inoculating the central portions into guineapigs. The only observer who, so far as I can ascertain, has examined histologically the tonsils of young children with a view to discovering tubercle in them, is F. F. Friedmann,<sup>85</sup> who in 1900 cut sections of the tonsils from 91 post mortem cases, and 54 operation cases. Of the 91 post mortem cases, 25 presented other evidence of tubercle; and of these 25, 10 showed tubercle bacilli or giant cells, or both, in the tonsils (5 being primary tonsillar), the remaining 15 being healthy. Of the 66 non-tuberculous cases, 6 showed tuberculosis of the tonsils. Only 1 of the 54 operation cases presented evidence of tuberculosis. Altogether, therefore, he discovered 12 cases in which the tuberculosis had entered the system through the tonsils. He concludes that the/

the tonsils are as frequently infected primarily, by food, as secondarily, by sputum, and he regards them as of importance as portals of infection.

Wright<sup>86</sup> discovered tubercle bacilli in the cervical lymphatics in certain cases in which he was unsuccessful in finding any in the tonsils, and he concluded, wrongly as I think, that they could therefore not have entered through the tonsils.

Schlenker<sup>87</sup> on the other hand, found that when there was caseation of the cervical glands, tuberculosis was sometimes present in the tonsils, and asserts that it is his belief that in the remaining cases the bacilli had wandered through the healthy tonsil. The experiments of Woodhead<sup>8</sup> with pigs fed on tuberculous material, show that it is very easy for them to get tuberculosis of the tonsils and cervical glands. But in these animals there exists about the pharynx a large ring of lymphoid tissue, which is present in man to a much smaller extent, and which is in young children sometimes hardly distinguishable. He has also noticed, and thinks it not uncommon to find, in children, an invasion through the tonsils and cervical glands, similar to that in the pig, and in these cases he found/

found the glands at the roots of the lungs tuberculous before the lungs became infected.

As regards adenoid vegetations M'Bride and Turner<sup>88</sup> found giant cells in 3% of their cases, and quote Pilliet, Brindel, Gottstein, Pluder and Fischer with a total of 21 cases positive out of 171. Lermoyez<sup>89</sup> has been successful in discovering tubercle bacilli in these growths.

Histological examinations made by Lewin<sup>86</sup> of tonsils and adenoid growths showed frequently the presence of tubercle, always latent, in persons dying of phthisis. Jessen<sup>90</sup> and Zarniko<sup>91</sup> cite some cases in which the symptoms of scrophulosis subsided after the removal of adenoid growths, and they therefore believe that scrophulosis is a general infection from the tonsils, especially the pharyngeal tonsils.

In order to ascertain the frequency with which the faucial tonsils in infants and young children act as portals of infection for tubercle, I have hardened and cut sections of the tonsils of 19 cases from the post mortem room and operating theatre. These were all more or less selected cases, in that they all had enlarged cervical glands, some of which were quite caseous. Most of/



of the sections were stained for tubercle bacilli with Carbol-Fuchsin and Methyl Blue, a few also being stained with Haematoxylin and Eosin to show giant-cells. I will briefly describe the post mortem condition of the cases and the microscopical appearances of the tonsils.

CASE I. Eliza G., 3 years.

Post mortem condition. Acute miliary tuberculosis. Caseation of thoracic and abdominal glands.

Tonsils. Right tonsil enlarged and scarred. Under the microscope a few giant-cells are seen, but no tubercle bacilli. There is also a certain amount of fibrosis. Left tonsil lost. In this case the tonsil was evidently part of the general infection.

CASE II. Morris S., 4 years.

Post mortem condition. Early general tuberculosis. Early caseation of bronchial and mesenteric glands. Right cervical glands apparently healthy, but under the microscope showed giant-cell systems.

Tonsils. Right tonsil normal in appearance. Microscopically a few giant cells found with some induration around. No tubercle bacilli. Here again the tonsil shared in the general infection.

CASE III. Florence K., 11 Months.

Post mortem condition. Tuberculous Broncho-pneumonia. Caseation of all the thoracic and some of the mesenteric glands. Recent caseation of the right cervical glands. Miliary tubercles in the spleen, pleurae, and kidneys.

Tonsils. Left tonsil normal in appearance. Some superficial ulceration was present at the posterior part of the right tonsil extending as far as the base of the epiglottis. This ulcer was apparently not very chronic. Microscopically, the left tonsil showed very few giant-cells, but many hundreds of tubercle bacilli grouped together in the follicles not very far from the epithelial surface. The right tonsil showed some giant-cell systems, with some induration and necrosis, but no tubercle bacilli. The loss of epithelium extended in some places into the crypts.

In this case the oldest lesions were apparently those of the thoracic glands. The tonsils must, I think, have been infected either by the blood stream, for there was a certain amount of general tuberculosis, or by the sputum, which was swallowed. The presence, however, of such a large number of bacilli in the left tonsil without much/

much structural change in that tonsil, or changes in the corresponding cervical glands, may mean that they entered the bloodstream direct from the tonsil, and set up a tuberculosis in predisposed thoracic glands.

CASE IV. George R., 8 months.

Post mortem condition. Tubercular Meningitis with several tubercular tumours in the brain. Otitis media on the left side. Also acute miliary tuberculosis. A chain of much enlarged, caseous and liquefying cervical glands stretched from the left tonsil down to the clavicle. A few glands enlarged, but not caseous on the right side. One miliary tubercle seen in the bifurcation gland. Tonsils. Not enlarged. Microscopically, no evidence of tubercle could be found though many sections were carefully examined.

The result of the microscopical examination here was very disappointing, for at the autopsy it seemed certain that if primary tonsillar tuberculosis could be demonstrated at all, it would be in this case. Unless indeed, the bacilli, as suggested by Schlenker, had wandered through the healthy tonsil and so infected the cervical glands, the case/

case must be looked upon as one primary in the middle ear.

CASE V. Edward S., 2½ years.

Post mortem condition. General tuberculosis.

Old caseous thoracic glands, and a caseous patch in the lower lobe of the right lung. Cervical glands enlarged on both sides, but miliary tubercles only present on the left side. Just above both clavicles were some small caseous glands (the ascending gland tuberculosis of Schlenker).

Tonsils. Right tonsil slightly hypertrophied.

Both tonsils had very large crypts. Microscopically, tubercle was present in the left tonsil, which showed some typical giant cell systems with surrounding induration. No loss of epithelium lining the crypts. The right tonsil showed no evidence of tubercle.

The infection of the left tonsil here was evidently secondary to the thoracic tubercle.

CASE VI. Ethel F., 1 year.

Post mortem condition. The lungs showed fibroid and bronchiectatic changes and scattered caseous tubercles. A few caseous nodules in the bifurcation, mesenteric and left cervical glands. Early general/

general tuberculosis.

Tonsils. Not enlarged. Under the microscope the right tonsil showed giant-cell systems, with tubercle bacilli in some of the giant-cells. The left tonsil also contained giant-cells, but there were no bacilli found, nor was there any induration.

There seems a certain amount of doubt about this case, as to the mode of origin, as the ages of the lesions in the different organs were much about the same. It is likely, however, that the tonsils here shared in the general infection, as the tuberculous lesions in them were not very advanced.

CASE VII. Annie P., 5 years.

Post mortem condition. Bronchopneumonia in both lungs. Cervical glands enlarged. No microscopic evidences of tubercle anywhere.

Tonsils. Right one hypertrophied. No evidence of tubercle was found with the microscope in either tonsil.

CASE VIII. Sarah D., 4 months.

Post mortem condition. Old tuberculosis of the lungs and thoracic glands. Also general tuberculosis. A firm caseous gland on the left side of/

of the neck.

Tonsils. Both seemed healthy to the naked eye and with the microscope.

CASE IX. Thomas C., 1 year.

Post mortem condition. Old tuberculosis of lungs and bifurcation gland. Superficial tuberculous abscesses. Enlarged cervical glands on left side containing miliary tubercles. Some caseous glands just above each clavicle (Ascending gland tuberculosis of Schlenker). Some caseous mesenteric glands.

Tonsils. No evidence of tubercle found.

CASE X. Henry C., 9 years.

Post mortem condition. Lungs engorged - some perisplenitis. Enlarged submaxillary glands. This was a nervous case, which died with meningeal symptoms.

Tonsils. Both much hypertrophied and contained some pus. No tubercle was found in any of the Sections.

CASE XI. Louisa H., 2 years.

Post mortem condition. Old tuberculosis of the thoracic glands along with acute miliary tuberculosis.

Tonsils. Both seemed quite healthy, and showed no evidence of tubercle.

CASE XII. Charles S., 1 year.

Post mortem condition. Old tuberculous thoracic glands, and general tuberculosis. Miliary tubercles present in the right cervical glands.

Tonsils. Left tonsil slightly hypertrophied. Under the microscope both showed giant-cells, which were especially well marked in the left tonsil. No tubercle bacilli found.

The tonsils in this case seem to have shared in the general infection.

CASE XIII. Henry H., 11 months.

Post mortem condition. Tuberculosis of left lung, which had spread from caseous left bronchial and tracheal glands. Also acute general tuberculosis. There were caseous nodules in the submaxillary and cervical glands on both sides.

Tonsils. Not enlarged. On cutting open the left tonsil a small caseous area was discovered.

Microscopically, the left tonsil showed many typical giant cell systems with epithelioid cells and surrounding necrosis. The giant cells contained some large vacuoles, and tubercle bacilli present in/

in some of the cells. The right tonsil also showed a few giant cells, but they were not very typical ones.

In this case the tuberculosis in the left tonsil was fairly advanced, and older than the general tuberculosis. From the fact that it was the left tonsil, the left cervical, tracheal and bronchial glands, which were chiefly affected, and that the process was fairly advanced in the tonsil, one may reasonably infer that this case was infected through the tonsils. Apparently in the tonsil the tuberculous process is a very slow one, and this case showed it in a more advanced condition than any other case I have seen.

CASE XIV. George P., 2 years.

Post mortem condition. Old caseous right tracheal glands, and miliary tubercles in the lungs, spleen, peritoneum and mesenteric glands. Some cervical glands on the right side showed caseous foci and old fibrosis.

Tonsils. Right tonsil hypertrophied, left one somewhat atrophied. Microscopically, they were both normal.

This was another case in which one might reasonably/



reasonably have expected to find evidence of tubercle, at any rate, in the right tonsil, but beyond the fact that it was hypertrophied, no evidence of disease could be found.

CASE XV. Emma H., 4 years.

Post mortem condition. Very old caseous glands on the right side of the trachea. Recent tuberculous ulceration of the intestines and tubercular meningitis.

Tonsils. Both quite healthy.

CASE XVI. Florence B.,  $3\frac{1}{2}$  years.

Post mortem condition. Old tuberculous thoracic glands; more recent tubercle in the lungs, intestines, mesenteric glands and brain. Enlarged cervical glands on both sides, with areas of caseation in them. Some very old caseous glands above right clavicle continuous with the caseous tracheal glands (probably another example of Schlenker's ascending gland tuberculosis).

Tonsils. Normal in appearance and under the microscope.

CASE XVII. Percy G.,  $1\frac{1}{2}$  years.

Post mortem condition. Caseation of the thoracic and abdominal glands. Cavitation of the right lung and intestinal ulceration. General military tuberculosis. One cervical gland contained

caseous nodules.

Tonsils. Small, but healthy in appearance. No trace of tubercle found with the microscope.

Cases XVIII. and XIX. were cases in which hypertrophied tonsils had been removed by operation. Both cases had enlarged cervical glands, but were otherwise apparently healthy. Under the microscope no evidence of tubercle could be discovered in the tonsils.

Only three of these cases (Nos. 1., III. and XIII.) showed any naked eye changes apart from hypertrophy, which might lead to the suspicion that they were tuberculous. In all, 7 out of the 19 cases had tuberculosis of the tonsil, 4 being quite undiagnosable apart from the microscope, and therefore latent. Unfortunately, the scope of this paper does not include the whole subject of latent tuberculosis of the tonsil, except where that tuberculosis may have claims to be considered primary in origin. The only cases having such claims are Nos. III., VI. and XIII., and of these the first two may be dismissed as improbable, leaving only Case XIII., about which, although I cannot speak with absolute certainty, I can say that it is most likely/

likely that the infection originated in the tonsil. In three cases, where, from the post mortem condition of the cervical glands, there seemed a likelihood of demonstrating tubercle in the tonsil, (Nos. IV., VIII. and XIV.), it was impossible to detect the slightest trace, though a great number of Sections were examined.

With this paper, I am sending some slides containing sections from the more interesting Tonsil cases, and a separate brief description of these sections is appended.

With regard to the method of entrance of the bacilli from the mouth into the tonsil, it is likely that they enter at certain sparsely scattered spots in the epithelial lining where Stohr<sup>93</sup> has described some lymphoid accumulations, which more or less obscure that lining. This being so, desquamation of the Epithelium, though not infrequent, would not be essential.

From the evidence of so many observers, a natural conclusion is that the tonsils, instead of acting as a protection to the organism, are really a source of danger, seeing that many bacteria, amongst them the tubercle bacillus, may enter the system through these structures. The bacilli attacking/

attacking the tonsils are, I think, as likely to be derived from the air as from food, as there is more chance for bacilli to settle about the moist surfaces of the mouth than further along the respiratory passages, seeing that so many children are more or less mouth-breathers either from habit or from nasal obstruction.

So much for the tonsils themselves. We now pass to those cases infected through the pharynx and mouth generally (including the tonsils). Of such cases I have six examples. In one there was a large mass of caseous cervical glands on the right side, continuous with caseous right tracheal and bronchial glands. In another the oldest lesions were caseous and liquefying left cervical glands, along with general tuberculosis. In two cases there were caseous cervical glands on both sides, extending down to and continuous with caseous tracheal and bifurcation glands. In one case there was caseation of the submaxillary, cervical and left tracheal glands with general tuberculosis, and in the last case there was an abscess in the left pharyngeal wall, and firm caseous glands extending all the way down the left side of the trachea, but no tubercle elsewhere. Only one of these was more than 15 months old.

To/

To illustrate the possibility of this method of infection, I might quote the experiment of Wyssokowicz,<sup>92</sup> who rubbed into the mouth and pharynx of a guinea-pig, some tuberculous sputum, but without drawing blood. The animal died in 6 weeks, and there were found 5 caseous cervical glands, some enlarged and caseous bronchial glands, and some miliary tubercles in the lungs. No local changes were found.

The frequency and possible importance of tuberculosis of the cervical glands in childhood, has, I think been overlooked, and writers are almost unanimous in declaring that it is extremely rare for these glands to form the focus of a general infection.<sup>100</sup> In comparatively few post mortem records is notice of their condition consistently taken, possibly because it sometimes requires special search and dissection to reach the higher set of the deep cervical glands. Amongst his cases Batten<sup>13</sup> found the cervical glands caseous in 14%. Sometimes it is found that these glands, instead of getting less and less affected as they are traced down the neck, get progressively more caseous the nearer they get to the thoracic inlet, pointing probably to an infection upwards from/

from the tracheal and other thoracic glands.

Schlenker,<sup>87</sup> who describes this mode of spread, calls it the Ascending Gland Tuberculosis, as contrasted with the ordinary Descending Gland Tuberculosis.

I have several times noticed this method of spread. In the ordinary form, it is usual for the caseation of the cervical glands to stop just above the level of the clavicle, and from the lowest of these glands the bacilli may reach the tracheal glands by the lymph anastomosis.

The tracheal glands, as has been mentioned previously, are very frequently predisposed to infection, owing to recent bronchial catarrh, so that it is possible that bacilli reaching them from the cervical glands, may set up in them a much more rapid form of caseation, than in the previously moderately healthy cervical glands. In this case then, there might be found tracheal glands in an advanced stage of caseation, apparently older than that of the cervical glands from which the infection had spread.

Still<sup>3</sup> says that for this method of spread from the cervical to the thoracic glands to be frequent, one ought to find the right cervical glands more often tuberculous than the left, as the right thoracic/

thoracic glands are so much more frequently tuberculous than the left. He quotes statistics of operations for the removal of tuberculous glands in the neck, and finds that the right glands were operated on in 51% and the left in 49% of the cases.

Amongst Shennan's cases 3.6% were found to be infected through the tonsils or pharynx, whilst Still found none of his cases so infected. My own cases show 2%, so that on the whole, though the percentage is not large, the number of cases infected in this manner are sufficiently numerous to cause it to be considered a recognised method of infection.

There was one case included under the thoracic cases, owing to the lesions being most advanced in the thoracic glands, but the origin of which was probably not respiratory, but placental. It was the case of an infant born prematurely at 8 months and which died after a four week's illness at the age of 7 weeks. Clinically, it presented evidence of Congenital Syphilis in the shape of rashes, snuffles and fissures about the mouth. The diet had been milk and barley water, the mother having advanced/

advanced phthisis. Labour, indeed, had been induced by a violent paroxysm of coughing, and the mother rapidly got worse and died 10 weeks later. At the post mortem on the infant, there were found some caseous bronchial and tracheal glands, the bifurcation gland was liquefied, and the right lung was solid with caseating tubercle. There were scattered tubercles in the left lung, spleen and liver.

Cases of Placental infection are but rarely seen. Holt<sup>20</sup> describes 3 cases which died within 7 weeks of birth, 2 having caseous bronchial glands, and all with tubercles in the lungs.

Birsch-Hirschfeld<sup>95</sup> describes one case where the dead foetus contained tubercle bacilli in its organs, many bacilli being found in the placental tufts.

Lehman<sup>96</sup> describes a similar case, and gives 6 cases accidentally affected by intrauterine inoculation. Cases are also cited by Charrin, Berti Jacobi, Landowzy, H. Martin, Aviragnet and Sabourand.

Bar and Renon<sup>97</sup> inoculated guineapigs from the blood of the umbilical veins in women with advanced tubercle and obtained positive results in 2 out of 5 cases.



G. d'Arrigo<sup>98</sup> says that tubercle can be transmitted to the foetus in three ways:-

1. By impregnation of the ovum by spermatozoa containing bacilli.
2. By infection in an ovum which has come from a tuberculous ovary.
3. By the passage of bacilli through the placenta.

He has had experimental proof of the third method, but has never found the necessary conditions for the first two. The instances may perhaps not be quite so rare as these published cases would lead one to suppose, infants a few weeks old being but seldom seen in the post mortem room.

The remainder of the cases investigated by me, 34 in number, have been classified under the heading of Doubtful cases, because either the mode of entry could not be found, or the tuberculous process had advanced so far that the original focus could not be determined.

A. Cases where no focus could be found (17 cases).

1. Three cases in which the oldest lesion was in the peritoneum. In one of these there was recent caseation of the mesenteric glands, but old tubercular peritonitis. In another case (the fourth) possibly the infection may have entered by the uterus. The Fallopian tubes were full of caseous material, and there was also adhesive peritonitis and tubercles in the pleura<sup>99</sup> and brain. The age was 9 years. Carr<sup>99</sup> mentions one case, aged 13, infected possibly in a similar manner.

2. One case of tubercular meningitis, with no tubercle elsewhere, and no otitis media. This, apparently, is a case of primary tubercular meningitis.
3. Two cases with tubercles in the spleen only.
4. One case with tubercles in the liver and kidneys only.
5. Two cases of spinal caries with no tubercle elsewhere.
6. Two cases of spinal caries and tubercular meningitis, but no other tuberculous lesions.
7. Five cases of early general tuberculosis, dying of meningitis. No otitis media.

All these cases, except perhaps the one with the caseous Fallopian tubes, are apparently cases in which the bacilli have entered by the respiratory or alimentary tract without leaving any trace, and then either have entered the blood stream directly or by the thoracic duct, some local predisposition having caused them to settle in the particular organs affected.

B. Cases where the lesions were too advanced to discover the original focus. (17 cases).

1. Two cases of advanced phthisis and advanced intestinal ulceration.
2. Eight cases showing simultaneous and equal involvement of the thoracic and abdominal glands, accompanied by general tuberculosis.

3./

3. Three cases of advanced caseation of thoracic, abdominal and cervical glands:
4. One case where the axillary glands in addition were caseous.
5. One case where the spine, lungs, intestines and bile duct were invaded with tubercle, no glands being involved.
6. One case where the focus was either in the thoracic glands or the anklejoint.  
Another case where the focus was either in a tubercular dactylitis or in the mesenteric glands.

In conclusion, to summarise my results, I found that out of a total of 307 cases, 213 were of respiratory origin, through the lungs or the middle ear; 53 were due to the ingestion of tuberculous material; 6 were due either to respiration or to ingestion, having been infected through the mouth or pharynx; 1 was a case of placental infection; 1 possibly of uterine infection; and the remaining 33 cases of doubtful origin. Thus, though infection by air is by far the most frequent method, infection by ingestion is a matter of very real and urgent importance, especially amongst children between the ages of 6 months and 3 years, during which period 30 cases, or 56% of all the abdominal cases occurred. It is in these years also, that the proportion between the thoracic/

-ic and abdominal cases is most nearly approximate, and this, in spite of the numerous predisposing influences, such as measles and whooping-cough, which favour thoracic infection. According to the Registrar General's Reports for the decade 1881-1890, the total average annual deaths in England of children under 5 years was 201,000, and the average annual number for the same age certified as having died from some form of tuberculosis, was 13,900, or 6.9% of the total number. Of course this does not include the large number of cases which, though suffering from tuberculosis, were carried off by some concomitant disease, or by accident, before the tuberculosis had gained sufficient hold on the system. When we come however, to consider how difficult of diagnosis tuberculosis in infancy so frequently is, even by experienced physicians, and how often it is found at post mortems when least expected, we shall not require very much argument to convince us that death statistics, so far as they relate to tuberculosis in childhood, are utterly misleading, unless based upon post mortem evidence. Recognising this fact, Dr Landouzy<sup>1</sup> made careful calculations regarding the tuberculosis mortality/

mortality in infancy in Paris, and came to the conclusion that in the year 1885, out of 12,264 deaths under the age of 2, 2,162 instead of 442 as returned on the death certificates, had died from tuberculosis. Of course, his method of calculation was a rough one, but his result is likely to be much nearer the truth than the original estimate.

Landouzy's statistics will apply equally well to the urban population in England, some reduction being made when the rural mortality is included. Let us suppose then, making allowance for this reduction, and for some exaggeration in his statistics, that 1 in 8, instead of 1 in 5, of all the deaths under 5 years, is due to tubercle; this will mean an annual tuberculosis mortality for that period of 25,000 instead of 13,900, as certified. Now by examination of Table II. it will be seen that 40 out of 227 cases under 5 years, or 17.5% were infected through the intestines or mesenteric glands; and this percentage is slightly below those of other English observers. This will give in round figures 4,400 as the annual number of deaths in England under the age of 5, resulting from the ingestion of tuberculous material. These startling figures bring to our notice again, the question of cows' /

cows' milk and Professor Koch's statements. About two months after the Tuberculosis Congress of 1901, a Royal Commission was appointed to investigate the relations between Human and Bovine Tuberculosis in general, and the importance of tuberculous milk and meat more particularly. Its report, when the investigations are concluded, will be awaited by medical men at home and abroad, with impatience and keen interest. Should it consider the case against milk proven, we shall expect the Government to carry out such means for safeguarding the population, as the Commission may suggest; and on the other hand, should its conclusions be similar to Professor Koch's, a stigma will be removed from cows' milk, and a great weight lifted from the mind of the thinking public. Whatever, therefore, the report of the Commission may be, the results can only be beneficial, and the question of the causation of Tuberculosis much simplified.

Cases of skin infection, inoculation of wounds, or of the eyes, usually remain local, and are neither of frequent occurrence, nor of great importance in childhood.

It only remains for me to express my indebtedness to Dr Coutts, under whose care was the case of/

of congenital tuberculosis, for permission to use the notes of the case, and to Drs Clive Riviere and O. K. Williamson, the then Pathologists of the respective Hospitals mentioned at the commencement of this paper, for allowing me to use the Post mortem records of those Hospitals.

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