affecting chiefly the serous membranes and the lungs, a condition which rarely accompanies phthisis pulmonalis.

Bones and Joints.

For the same reasons it is possible that the numerous joint and bone lesions so frequently seen in children and so rarely in adults are due to bovine infection. The general mode of death from long-continued suppuration of tuberculous joints and carious spines is sheer exhaustion and waxy disease of the viscera, rarely tuberculosis of the lungs.

Tuberculous Meningitis.

It is often difficult to explain the onset in a previously healthy child of tuberculous meningitis. Sometimes it is confined to the brain, at others it is part of a general infection. Perhaps it is a bovine tuberculosis, as I have seen several necropsies on such cases where there has been no other discoverable evidence of tubercle in the body. We have all seen cases of tuberculous peritonitis completely recover after opening and draining the abdomen and nothing more. So far no satisfactory explanation of this result has been given. My own opinion is that it is a localized bovine tuberculosis which has died out, and that relief of the fluid is sufficient to cure the patient.

Post-nasal Adenoids.

Again, it is inexplicable why perfectly healthy children of healthy parents should be so frequently affected with chronic enlargement of the tonsils and with adenoids. It is quite conceivable that tuberculous milk should be absorbed from the fauces and pharynx, producing enlarged tonsils, adenoids, and extending even into the middle ear, causing tuberculous otitis media. There is much evidence also to suggest that acute milary tuberculosis, especially that of the typhoid form, is in reality a bovine tuberculosis produced by food ingestion. I have only suggested bovine tuberculosis as the pathology of the latter affections. I have no proof to offer, but hope that other workers will direct their efforts to prove or disprove my opinion.

Suggestion.

Clinical appearances would suggest that there is a distinct difference between the tuberculous manifestations in childhood and adult life, and that they are antagonistic to each other. We know that animals are practically immune against human tuberculosis or phthisis, but not to *tubercle mesenterica* and abdominal tuberculosis. Since the two diseases cannot exist together in cattle, may it not be that they cannot exist together in man? and that the serum of a cow affected with tuberculosis might have a curative effect in a case of pulmonary phthisis exactly on the same lines as small-pox and cow-pox? Who knows!

Remedy.

The milk-drinking period of a child is, on an average, from birth to 14 or 15 months. Fortunately, a fair percentage of infants are fed on human milk and thus escape danger, whilst a very large number of children in England are brought up on proprietary artificial foods and condensed milk, which, as far as tubercle is concerned, are generally safe. The remainder—and they are usually the children of the poor—are given cheap cow's milk, bought in small quantities from the milk shop or the street. When we consider the extreme delicacy of an infant's digestion, and the small resistance it is able to offer to any infection, it is not surprising if tubercle bacilli are introduced in the milk, that absorption will take place, and tuberculosis follow.

It has been pointed out before that the danger is greatest when cow's milk is used exclusively and tubercle bacilli introduced day by day, as it is only when large numbers of bacilli are used that infection is likely to become general. It would appear, therefore, that infants and young children with a feeble power of resistance are susceptible to bovine tuberculosis, more especially if fed for a prolonged period on infected milk; that, as age advances, the susceptibility is decreased, until at adult life there is little risk to more than a purely local affection of the glands of the neck, which, however, may occasionally extend directly to the lung, and even a general milary tuberculosis.

Legislation.

Considering that tuberculosis is so common and even increasing in this country amongst dairy cows, something ought to be done by the Legislature to prevent the wholesale distribution of tuberculous milk to the public. I do not

advocate any harsh measures, as I am quite sure that every farmer and dairyman in the country would assist in every way to stamp out tuberculosis in cattle. But I would make it a penal offence for any person knowingly to supply milk to the public from a cow suffering from tuberculosis of the udder. A more rigorous inspection of dairy cattle should be made by the health authorities; and, as it is perfectly easy and certain by the tuberculin test to diagnose a tuberculous cow, there should be no difficulty in at least eradicating the disease from dairy cattle. The onus of responsibility of supplying pure milk to the public should not rest on the health authority, but on the producer; and, although the health authority can do a splendid and humane work by providing the community with sterilized milk at a cheap rate, still the real responsibility must lie with the man who actually delivers milk for sale to the public. Liverpool, as usual in all matters pertaining to the public health, is in the forefront in this respect, and at the present time is delivering to the public over 63,000 bottles of sterilized milk every week at a small charge which is not remunerative monetarily.

It can only be when large commercial companies undertake throughout the country to eradicate tuberculous cows from their herds that any substantial improvement will result and poor innocent children will be protected.

At the present time I believe I am right in saying that the majority of educated mothers do boil milk before it is given to children, and I sincerely hope they will continue to do so.

Although I believe the facts which I have recorded completely establish the truth of my theory, I am glad to hope that there will be before long further evidence to corroborate these views. Professor Boyce a few days after the publication of my preliminary note in the *BRITISH MEDICAL JOURNAL* of January 31st, 1903, called upon me at Mill Road Infirmary, and spent a considerable time in examining clinical cases, as well as specimens illustrating my views. I placed everything at his disposal, and supplied him with materials for inoculation of cattle at the experimental farms of the Royal Commission. Dr. Ernest Glynn was appointed to make a careful inquiry with regard to *tubercle mesenterica* in Liverpool, and I sincerely hope, with the opportunities which Professor Boyce has at his disposal that we shall receive the results at an early date.

I conclude by expressing the opinion, based almost exclusively on clinical and pathological evidence, that human and bovine tuberculosis are separate and distinct diseases, as shown by Professor Koch; but that the human body is susceptible to both, and especially to bovine tuberculosis in the early periods of life.

The two diseases are so rarely seen together in man that there seems to be some ground for presuming that they are antagonistic to each other, and that bovine tuberculosis may possibly confer an immunity against human tuberculosis. If this opinion should prove correct, I venture to submit that it would entirely reconcile the conflicting opinions which have been so frequently expressed during the last two years on this most terrible of all diseases.

PHTHISIS AND HOUSE INFECTION.*

By J. R. JOHNSON, M.R.C.S., L.R.C.P.,

Richmond.

THIS brief paper contains notes of some cases of phthisis which have come under my observation in Richmond. I have been struck with the bearing of these few cases upon modern ideas of the causation and prevention of this disease.

According to the report of the medical officer of health, there were 30 deaths from phthisis in Richmond during the year 1901. This, with a population of 31,000 gives a phthisis mortality-rate of 1.6. The total death-rate is 13.5. Thus with a death-rate of 13.5, we have a phthisis mortality-rate of 1.6. This, though not high, is higher than it should be in a town where there is little or no overcrowding, and which is practically in the country. Eccles, a fair sample of a similarly-situated suburb, has a death-rate of 16.5, with a phthisis-rate of 1.3, a higher death-rate, that is to say, than Richmond, with a lower phthisis mortality.

The notes upon the following cases help, I think, to throw a little light upon the prevalence of the disease in Richmond. They have occurred in my practice during the past five years. I wish in sketching the cases to lay stress

* Read before the Thames Valley Branch of the British Medical Association.

upon certain particulars, whether, as far as one can ascertain, the cases arose *de novo* in Richmond or were imported—whether they occurred in old or modern houses. By old houses I mean houses not recently erected; such houses in Richmond have often changed tenants frequently, and it is difficult to trace occupants beyond the immediate occupiers. The old houses occur principally in the lower and more crowded part of the town; the modern ones are for the most part situated upon the hill.

Richmond is built upon clay, and being an ancient town contains a large number of old houses, some of them centuries old.

Case 1.—R. E. B., an infant, died, aged 10 months, of pulmonary tuberculosis.

The parents lived in one of a row of small, and for the most part dirty, ill-kept houses. Two families at least occupied each house, and some were quite insanitary.

The case therefore arose *de novo* in an old house.

Case 2.—T. H., died, 35 years of age, of typical phthisis; he also had tuberculous disease of the testicles.

His sputum contained tubercle bacilli in abundance. He had resided in Richmond two years before his death, coming from Twickenham already ill with the disease. It is therefore an imported case. He, with his wife and child lived in one room in a house situated in the midst of one of our worst areas. It almost deserves the epithet squalid.

Case 3.—E. H. L. D., died, aged 4 months, of acute pulmonary tuberculosis.

This was a case arising in an old house. The houses in this street on both sides are old. They are low-roomed, small-windowed, basemented, with extremely narrow, winding, dark stairs. Most of them are badly kept, some are actually insanitary from collections of refuse and garbage in their basements.

Case 4.—G. T. W., died at the age of 26 of acute phthisis of three weeks' duration, in a house a few doors above Case 3.

This is an instructive case. The man, a delicate-looking fellow, but with a story of always being quite well, arrived from Ealing to open a draper's shop. He spent one week knocking the interior of the place about, making alterations, doing his own carpentering and fitting, and so stirring up the septic dust of an old and dark house. Exactly a week after his arrival he suddenly developed pleuro-pneumonia, became dangerously ill and died in three weeks of acute pulmonary tuberculosis.

The case arose in an old house.

Case 5.—T. B. died at the age of 49 of phthisis. He was a very typical example of rapid phthisis with diabetes. The sputum, which was very profuse and was generously distributed, contained tubercle bacilli in very large numbers. The man had resided two years in Richmond. He stated that he ailed nothing on his arrival, but attributed his illness in the first place to a chill contracted at Yarmouth twelve months prior to his death.

The house is an old one of a bad type—narrow, small windows, basemented, and insanitary. The business carried on on the ground floor is that of an eating-house. Anything more dangerous to public health it would be difficult to conceive.

Case 6.—S. M., aged 19. This was a quickly-progressing case, its special features being the extreme prostration and rapid wasting. He lost 8½ lb. in three weeks. I have never seen such profuse night sweats as in this patient. I sent him to South Africa. He was dangerously ill for three days on the voyage out, but is now, four years after, in excellent health and has ridden sixty miles on horseback in a single day.

The house is old and sunless. This young man's father died of phthisis in 1896, but not in the same house. The father appears to have contracted the disease in Twickenham. It is possible that the son became infected from contact with the father or with some of his belongings. At any rate, the case arose in an old house.

Case 7.—M. C., a girl, aged 20, was a case of incipient phthisis.

The mother brought her to me in great distress, the girl being the last child left to her. The physical signs were not pronounced, but I felt no doubt as to the nature of the disease. She is now in Brompton Hospital, and Dr. Hector Mackenzie, under whose care she is at present, informs me that there is tubercle in the right upper lobe. No tubercle bacilli have, however, so far been found in the sputum. A brother and two sisters have died of phthisis in the same house.

The case therefore arose in an already infected house.

Case 8.—L. A., aged 42, came to Richmond quite well, and resided two years. She has advanced phthisis; the larynx is so extensively affected that the voice is almost inaudible.

The sputum contains tubercle bacilli in extraordinary numbers. The affection of the larynx is interesting, since with the patient resided her old mother almost stone deaf. The incessant strain upon the larynx of the patient to make her mother hear accounts for the laryngeal trouble. She is at present in a sanatorium, but is much too far gone to hope for great improvement.

This old house has frequently changed tenants. This is a case arising apparently in an old house.

Case 9.—A. W., 56 years of age, is a case of chronic phthisis of thirty years' standing. He had severe hæmoptysis when 25 years of age, and had resided in Richmond for the last eight years only. Tubercle bacilli were always present in the expectoration in small numbers. He was subject to occasional attacks, accompanied by a temperature of 102° and quick pulse, with much cough and expectoration. These attacks always supervened after a night at a theatre or some similar mild dissipation. They subsided in the course of a few weeks, and the patient resumed his

ordinary condition of non-progressive phthisis. The case is an imported one, residing in a modern and luxurious house.

Case 10.—S. R. B., aged 32, has resided in Richmond four months only. Ten years ago he went to Davos for hæmoptysis, and says his life was then saved. He came to Richmond comparatively well, and quite able to work. He is now dangerously ill. He remained well until he undertook a week's stocktaking in the shop in which he is manager. This entailed shifting stock long undisturbed in dark and dusty holes and corners. He became suddenly dangerously ill, with patches of acute pneumonia, and has gone from bad to worse. The sputum contains tubercle bacilli in fair numbers, but also pneumococci, streptococci, and staphylococci in large numbers.

In my opinion this man's dangerous illness arose from the inhalation of the inhalation of the septic dust stirred up in the stocktaking lighting upon lung already broken down from the presence of tubercle bacilli. It is a case of mixed infection, in which the danger lies not so much in the phthisis as in the presence of other septic organisms. It is a case of phthisis imported into an old and septic house.

Case 11.—P. N., aged 32, is a case of incipient phthisis.

He has resided in Richmond over three years, was perfectly well on arrival, and lives in large, modern, up-to-date house on the hill. Two months ago he had a slight attack of hæmoptysis, and tubercle bacilli were found in small numbers in the sputum. He has offices in a very old house. The offices are low-roomed, sunless, and piled up with books and papers of a very old established firm, upon which the dust and dirt of years remains undisturbed. The walls, wainscoting, and floor, particularly of the back office, have the appearance of never having been cleansed nor even swept. An employé died on September 2nd, and a necropsy revealed extensive pulmonary tuberculosis, the remains of the left lung weighing but ten ounces. I found on inquiry that this man constantly expectorated in or about the office fireplace. He was intemperate, and his habits careless. My patient therefore became infected in old offices in which a case of phthisis was in quick progress. The case arises *de novo* in a house known to have been infected.

Classifying the 11 cases we find:

Six arose in old, ill-ventilated, narrow-windowed, sunless, or dusty houses.

Two arose in houses known to have been tenanted by persons suffering from phthisis.

Two are imported cases living in, and the disease rapidly accelerated by, old houses.

One is an imported case residing in modern house.

In not a single instance does a case arise *de novo* in a modern, decently-kept house.

It is impossible to ignore the danger lurking in house-dust, and particularly the dust of ill-ventilated, dark, and basemented houses.

The stirring up of house dust, possibly containing tubercle bacilli, may easily account for the cases which appear to arise in the old or unclean houses. Cases iv and viii particularly point this moral.

The inhalation of septic house-dirt by patients already phthisical, although the disease may be more or less dormant, is of the gravest consequences, as seen in Cases ix and x.

The danger of persons coming of phthisical stock using and living in rooms or houses which are known to have been occupied by persons suffering from phthisis is shown in Cases vii and xi.

The prevalence of phthisis in Richmond is due, I venture to think, to the large number of old, dusty, insanitary, narrow-windowed, ill-ventilated houses in Richmond, many of which have had occupiers infected with the disease, and in which no attempt has been made to disinfect the rooms. Richmond is an old town, and large numbers of the houses were built at a bad period. Many are let to people of reduced circumstances who again take in lodgers. Such people are unable to afford proper servants and the proper cleansing of the houses. Hence such houses are septic from accumulations of dust and dirt, and present considerable danger to their inhabitants, particularly if such happen to possess phthisical tendencies. The modern or well-cleansed house offers no risk whatever.

It is apparent that the mere fact of a town being built upon a clay soil can have little or nothing to do with the prevalence of phthisis there. It is clear that a single house already infected with tubercle bacilli would account for any number of cases whether upon sandy soil or upon clay soil. An examination of the history of the houses would throw a different light upon the question of the prevalence or otherwise of the disease. But all this has been absolutely set at rest by the complete and convincing investigations which Dr. Harold Coates carried out in Manchester in 1901. Dr. Coates examined the dust of houses inhabited by people suffering from phthisis. He found it impossible except in two cases to demonstrate tubercle bacilli by the ordinary methods of staining and the microscope, but by inoculating guinea-pigs under the most scrupulously careful conditions

with the dust from infected houses, he was able at the end of a very large number of experiments to demonstrate the presence of the tubercle bacilli in all houses where phthisis was known to have been present. His paper should be read by every medical man, and his conclusions acted upon.

Dr. Crocker (M.O.H. Richmond) in his report for 1901, suggests, among other measures for preventing the spread of phthisis in this town, the notification of the disease. I think the brief notes I have read show the value of such notification, especially if followed by proper disinfection of the rooms or house by the sanitary authorities. Were such measures made compulsory, the present high rate of mortality from phthisis in Richmond would, I believe, be promptly lessened.

THE DIETETIC TREATMENT OF PULMONARY TUBERCULOSIS FROM THE POINT OF VIEW OF ITS HAEMATOLOGY AND HISTOPATHOLOGY.

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IN studying the reaction of the tissues to the stimulus of bacteria and their products, the tendency has been recently to turn to the blood as being the only tissue which can be fixed while still alive and undergoing these changes. The histological condition of the blood may be taken as an indication of the state and activity of the solid tissues, of which it is at once the product and the pabulum. The indications derived from the serum are as yet too vague to warrant any conclusion being drawn: it can only be regarded as the vehicle for the conveyance of the secretory products of the various cells which it contains, or with which it comes in contact. The cell elements, setting aside the red corpuscles, which have at least one definite function, play various parts in the resistance of the body to disease. That all the varieties of leucocytes, although they may have a common origin, have not the same part to play, is evident from their different behaviour in response to stimuli; thus, the digestive leucocytosis is mostly lymphocytic, while that of sepsis is polymorphonuclear. If a leucocytosis can be taken as the evidence of a reaction of the blood cells to the presence of toxins, the extent of the reaction varies with the acuteness of the infection, though this rule is not universal, for, in typhoid and measles, leucocytosis appears to be absent.

A marked feature of uncomplicated tuberculosis is the total absence of a leucocytosis. This would seem to indicate either a chronic toxic state, in which the stimulus is too slight to elicit a response, or, what is more likely, a state of equilibrium which is gradually produced, the reaction to the poison not exceeding its production. The importance of this consideration, from a therapeutic standpoint, is perhaps not realized.

From this point of view, cases of pulmonary tuberculosis naturally group themselves under two headings: Those in which the recuperative power of the system is sufficient to produce a reaction, capable, when properly guided and sustained, of stopping the progress of the disease; and those in which the system is incapable of stimulation by the ordinary means and the disease progresses towards a fatal issue.

The recuperative power itself also falls naturally under two headings: The physiological tissue reaction, which is the main factor in recovery, and the physical capability of the infected organ for repair. The former, in many cases, is sufficient to arrest the progress of the disease, while the latter may be unable, owing to the extent of the lesion, to bring about a healthy condition with total extirpation of diseased tissue and its replacement by a sound scar. A lung may be so involved as to render this physical shrinkage impossible for anatomical reasons, even where the physiological reaction has been sufficient to check the advance of the disease. Hence the importance of maintaining the tonicity of the tissues in chronic cases after the active treatment is at an end, and the frequent relapses following apparent recovery.

The physical repair being dependent on and necessarily preceded by the physiological reaction, the artificial production of a reaction is the aim of all treatment. In the tuberculin treatment a small amount of toxin is introduced into the system, producing a reaction characterized by local phenomena at the seat of the lesion, and, what is most important from the point of view of the present article, by a rise of temperature and a leucocytosis accompanied by an eosinophilia (Bischoff)¹

The reaction produced by the sudden access of toxin persists after the actual toxic increase stops. Cases in which no benefit results are those in which the toxic action has exceeded the reaction, or where the reaction itself has not persisted, that is where the requisite response is not forthcoming.

The benefit of a nitrogenous diet lies in the production of a general tonicity of the tissues. In the blood marked changes in the numbers of the leucocytes following the absorption of certain constituents of the food occur, and an increase in weight is the evidence of an improved metabolism in the tissues generally, making a physiological use of the foodstuffs supplied, rather than of the plethoric storage of excess material.

The specific substances which produce the leucocytosis are nuclein and its various derivatives. The leucocytosis so produced is a true increase in the number of leucocytes in the circulation, and therefore of the number of cells functionally active. This, as one would expect, entails an increased leucocytic death-rate, if one may put it so, and therefore an increased excretion of the products of cellular metabolism. The nuclein derivatives which cause this action themselves contain those products, but the increase in excretion is greater than would be accounted for even were the total quantity of nuclein absorbed and excreted as such. To quote Milroy and Malcolm, "there is absolutely no doubt that nucleic acid (one of the nuclein derivatives) produces a hyperleucocytosis within four hours after its ingestion, and an accompanying or subsequent leucolysis."²

Early stages of pulmonary tuberculosis are characterized by the total absence of leucocytosis, while the later stages invariably show a leucocytosis, caused probably by a mixed infection.³ In the cases observed the leucocytes, after twelve hours' fasting, showed only a slight increase, though all were cases of cavity formation, and therefore, presumably cases of mixed infection. The morning count varied from 10,000 to 13,000.

In cases treated by the open-air method and on a diet rich in animal nitrogen, the characteristic features of the blood counts are: (1) A moderate constant leucocytosis, (2) a large absorptive lymphocytosis, and (3) an almost constant eosinophilia; the eosinophile cells varying from 4 per cent. to 5 per cent. of the total leucocytes. The cases selected for observation all had a subnormal temperature and in none of them, so far as could be ascertained, were there intestinal parasites.⁴ The total number of the leucocytes varied in one case from 10,000 at 8 a.m. to 19,000 at 3 p.m. This exceptionally large increase occurred after two meals consisting mainly of nitrogenous food. The ordinary digestive leucocytosis ranged from 1,000 to 4,000.⁵ Under these conditions migration of eosinophiles into an artificial blister occurs, but probably only accidentally, as the number is small compared with the number of eosinophiles present in the blood and of polymorphonuclears present in the blister serum. The eosinophile leucocytes seem to have a special relation to the toxins of the diseases in which eosinophilia occurs. Pemphigus, for example, is characterized by a leucocytosis with marked eosinophilia. The vesicle contents, which presumably have some relation to the toxins of the disease, are found to contain eosinophile leucocytes in large numbers, while an artificial blister contains few as compared with the numbers in the blood and in the serum of the specific vesicles.⁶

In the blood may be seen at any time all gradations of cells, from the lymphocyte with a single nucleus and little protoplasm to the polymorphonuclear with or without specialized granules. These cells may have different functions at different periods, but they are all essentially similar in nature and origin. Thus a stimulation of lymphogenic tissue and a consequent production of a leucocytosis cannot but be beneficial in furthering the action of cells which are known to be inimical to foreign bodies, using the expression in its widest sense.

In septic diseases the leucocytosis is mostly polymorphonuclear and the exudation contains chiefly polymorphonuclear leucocytes. The circulating toxins exert a chemiotactic action on the leucocytes in the circulation, while the invading organisms cause a local exudation of polymorphonuclears or exert through toxins a positive chemiotactic action on those leucocytes. Both these phenomena are to be regarded as the response of the tissues to the stimulation of the toxins. In tuberculosis the rapid softening and digestion of the tissues associated with the presence and action of suppurative organisms are replaced by a slow degeneration of the cells and tissues round the nidus of the bacilli. Instead of an exudation