TUBERCULOSIS IN INFANCY AND CHILDHOOD:

WITH SPECIAL REFERENCE TO ITS PREVENTION*

BY

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In the child as compared with the adult the uncomplicated character of disease makes it easier to find the initial lesion, to follow its course, to interpret the pathological changes, and thus often to assess the aetiological factor. This is true of many diseases in childhood—for example, nephritis, rheumatism, pulmonary and intestinal diseases, and, in particular, tuberculosis, in which the initial infection occurs nearly always on a virgin soil. Thus the lesion produced is not affected by the allergy or immunity caused by previous tuberculous disease. Further, the child's organs—lungs, intestines, and lymphoid tissue—unlike those of the adult, have not undergone morbid anatomical change as a result of more or less severe non-specific inflammatory conditions which may influence the progress and the results of tuberculous infection.

General Incidence and Mortality

I have investigated 434 cases with tuberculous lesions found in a series of 3,000 necropsies during the last twelve years at the Royal Hospital for Sick Children, Glasgow. Some of the results have already been reported,^{1,2} and in this paper these earlier results are included with later observations. It is evident from Table I that the inci-

TABLE I.—Incidence of Cases with Tuberculous Lesions in 3,000 Necropsies; Incidence of Positive Mantoux Tests in Clinical Series.

Age Group	Total Necropsies	Cases with Tuberculous Lesion			Tuberculin Series. Adjusted Per Cent. Positive 1 in 10 O. T.
		Number	Per Cent. of Total Necropsies	Per Cent. Dying of Tuberculosis	
-3 months	971	7	0.72	100.0	0.0
-6 "	536	40	7.46	100.0	5.0
-9 "	325	45	13.85	97.8	6.0
-12 "	223	40	17.90	95.0	6.0
-18 "	215	57	26.50	96.5	11.0
-2 years	140	38	27.10	94.7	18.0
-3 "	145	62	42.8	93.5	28.0
-6 "	228	80	35.1	75.0	30.9
-9 "	153	43	28.1	76.7	47.8
-13 "	64	22	34.4	72.7	63.8
Total ...	3,000	434	14.5	89.2	—

dence of tuberculous lesions is exceptionally low in the first three months of life, but thereafter gradually rises to its peak in the third year, after which there is a slight fall. The incidence of lesions recorded here is lower than that found by most Continental workers—for example, by Comby³ in Paris, whose total percentage in children up to 15 was 36.7, and by Hamburger⁴ in Vienna, with 39.5 per cent. Combe,⁵ in 25,000 collected necropsies, found an average incidence of 8.7 per cent. for the first year of life, which is a little higher than mine.

I have never felt that the post-mortem index of infection was a true one in the higher age groups, in which the numbers are rather small and in which other factors

noted below may be at work. Of course it might be objected that in my series small tuberculous lesions were missed, but this is unlikely, since I failed to find a sensitizing lesion in only thirty-six cases reacting positively to tuberculin out of 2,163 cases carefully Pirquet or Mantoux tested. Of these thirty-six cases twenty-five were in the first two years of life.

In order to find in another way the incidence of infection, Griffin and myself performed a series of Mantoux tests in nearly 2,000 children at the Royal Hospital for Sick Children, Glasgow, during 1932-4—that is, in the latter years during which the post-mortem observations were made. The strength of the old tuberculin used varied from 1 in 1,000 to 1 in 10 as recommended by Hart,⁶ and the incidence of positive reactions was calculated as an "adjusted percentage" to the 1 in 10 dilution. A detailed account of this series will be published later, but they are summarized in Table I, which shows that in the first three years the percentage of positive reactions is below that of tuberculous lesions found post mortem, probably because some of the younger subjects with severe tuberculosis did not react to the higher dilutions and died before greater concentrations could be used. The incidence of positive tuberculin reactions and positive necropsy findings is much the same for the 3 to 6 year group, but thereafter the percentage of positive tuberculin tests rises to almost double that of the positive necropsy findings.

The mortality from these tuberculous lesions gradually decreases *pari passu* with the age of the child (Table I). Thus the older child is better able to arrest a tuberculous infection. The higher level of mortality to the incidence of infection as shown by the Mantoux test during the first three years of life indicates the serious prognosis of infection at this period. Comparison of the post-mortem findings and results of Mantoux tests, however, shows that the former underestimates the frequency of infection from the sixth year onwards, probably on account of the smaller numbers of older children and also as they are usually affected by more chronic and localized types of tuberculosis many cases do not find their way to a general children's hospital, but are sent direct to special tuberculosis hospitals. It is evident, however, from Table I, that the mortality even in the higher age groups is considerable. The ratio of mortality from tuberculosis to that from all diseases recorded by the Registrar-General's for Scotland or England is, however, lower for all age groups than those described here. The mortality rate in relation to the morbidity rate varies greatly with the geographical distribution, and my mortality expressed as a percentage of the incidence of the tuberculous lesions is higher than that reported by foreign workers—for example:

	Months			Years					Total
	-3	-5	-12	-2	-5	-7	-11	-14	
Hamburger ⁴ (Vienna, 1903-6; 848 necropsies)	100	100	80	70	67	60	68	50	69
	89.8								
Blacklock (Glasgow, 1924-36; 3,000 necropsies)	100	100	96.5	95.8	81.7	69.0	80.0	57.1	89.2
	97.7								

Nassau and Zweig⁷ in Berlin found a 69.3 per cent. mortality in the first year; Mantoux⁸ in Paris 75 per cent. for the first six months of life, 54 per cent. for the second, 40 per cent. for the third, and 28 per cent. for the fourth. In the United States Drolet⁹ estimated the annual mortality of infected infants at 1.3 per cent., while Asserson,¹⁰ in a five-year follow-up of 405 infected infants under two years, found a 51.7 per cent. mortality. In this country, then, the mortality in relation to the

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morbidity rate is higher than abroad, and while reasons for this are easily suggested yet they are difficult to prove. It is possible that in Scotland—and this also applies to Ireland and Wales—racial factors may be at work. The Celtic race is probably less resistant to infection than the Germanic on account of its more isolated geographical position, at least in the past. Thus the contact of the former with tuberculous infection has been probably shorter, resulting in lower racial immunity. The easier means of transport of our own time, however, is likely to change this. Dietetic factors, too, may play a part, while our variable and comparatively sunless climate must also lower resistance to infection partly by confining children indoors, thus bringing them more into intimate contact with any familial tuberculous infection. This would result in a more frequent dosage with tubercle bacilli which is commonly productive of a progressive and generally fatal tuberculosis in childhood. It is also possible that the strains of tubercle bacilli causing infection here are more virulent than those found abroad; proof of this, however, is lacking.

Sex Incidence

In this series girls were more frequently affected than boys at all ages (Table II). In the Registrar-Generals'

TABLE II.—*The Sex Incidence of Tuberculous Lesions in 3,000 Necropsies*

		-1 Year	-2 Years	-3 Years	-6 Years	-9 Years	-13 Years	Total
Boys	Total necropsies	1,275	209	82	118	88	41	1,813
	Per cent. tuberculous	6.04	23.9	40.2	23.7	23.9	34.1	12.3
Girls	Total necropsies	780	146	63	110	65	23	1,187
	Per cent. tuberculous	7.05	30.8	46.0	47.3	33.8	34.8	17.7

reports this greater incidence in the female sex is also apparent from the fourth to the thirtieth years. The difference which I have found in the first four years of life probably depends on the greater mortality from all causes in boys, whereas thereafter the mortality rate from all causes is nearly equal in both sexes. The difference in the sex mortality from tuberculosis in older children would appear to be of some significance, and probably depends on the fact that in working-class homes at least girls are more domesticated than boys, and are thus more frequently exposed to any familial tuberculous infection. Dow and Lloyd¹¹ have observed in London that the incidence of positive Mantoux tests is slightly higher in girls than in boys, while Armand-Delille *et al.*¹² have also noted that from the ninth year onwards pulmonary tuberculosis is commoner in girls in Paris. It is thus possible, then, for child-welfare workers to do something to lower this higher incidence in girls.

The Immunity from Childhood Infection

A benign childhood tuberculous infection has generally been regarded as producing some immunity to such infection in later life. It seems certain, however, from my figures that tuberculous infection in the child under 3 years is serious, and should, if possible, be prevented. The effect of these childhood lesions on later infection is imperfectly understood. Experimental work in animals has given very divergent results.^{13 14 15} Further, the reaction in already infected animals to a second infection is possibly quite different from that in man. Thus observations on the human subject are more likely to yield satisfactory results regarding the alleged immunity to infection in later life produced by childhood lesions, though here again confusion exists. For example, the South African Rand report¹⁶ states that tuberculin-positive negroes are more likely to develop a recognizable tuberculosis than tuberculin-negative reactors, but the latter

show a greater liability to develop a septicaemic type. Stewart^{17 18} and Myers and Harrington¹⁹ observed that tuberculin-positive children more frequently develop fatal phthisis in later life than those who react negatively; De Besche and Jørgensen²⁰ have also made somewhat similar observations in adults. On the other hand, as suggested by Wingfield,²¹ a high degree of immunity should nullify the danger of a high degree of allergy.

From my own morbid anatomical observations the effect of allergy as a result of infection occurring later in childhood is very different from that acquired in infancy. For example, in the older child, as described later, there are less extensive and better encapsulated secondary lymphatic lesions in relation to the primary infection and less frequent generalization. This difference is due, first, to the fact that the younger the child the more intimate and more frequent the exposure to any familial infection; secondly—and here we are on uncertain ground—to the higher natural immunity to any type of infection in older children. In my opinion it is those late childhood minimal infections which, becoming quiescent, give rise to some degree of immunity that prevent these acute types of tuberculosis reported by Borrel²² in native troops brought to Europe. Nassau and Zweig⁷ have observed that tuberculosis contracted after the second year generally remains latent and prevents later severe infections. It is long since Marfan²³ confirmed the original observations of Bazin and Cornil that pulmonary tuberculosis was relatively uncommon in those with tuberculous cervical adenitis or lupus, and that when pulmonary lesions did occur in such subjects they ran a relatively benign course. The balance of evidence is thus in favour of the view that minimal infection in later childhood probably affords some degree of protection against reinfection with small and infrequent doses of tubercle bacilli. Further, it would seem to me that allergy in the human subject is but a step in the development of immunity, which in most people increases, while allergy usually decreases. It must not be assumed, however, that this immunity is as absolute as that following many of the virus or bacterial infections; it is rather an imperfect immunity, which can be broken down by ill-health or by massive or continuous infection with tubercle bacilli either of exogenous or endogenous origin.

In an attempt to imitate the immunity resulting from infection the use of B.C.G. vaccine has been recommended by Calmette. While it may be desirable to adopt this measure in infants who are exposed to virulent infection in their homes, from which neither they nor the source of infection can be removed, yet the indiscriminate application of this vaccine to the child population as a whole would seem a questionable procedure, even in spite of the fact that favourable reports of its efficacy have been published.^{24 25 26} It should be remembered, too, that the immunity so produced lasts only a year or two. Further, it has never been satisfactorily explained what may happen in time to this B.C.G. bacillus, which, according to its discoverers, is of bovine origin and innocuous to the human subject. We know that it is capable of surviving in the human body—a tissue quite unlike the glycerol-bile-potato medium used to maintain its avirulent character. After many years' sojourn in the tissues it may mutate to a more pathogenic form or even to a human type. While mutation to a different type has not yet been proved in the case of tubercle bacilli, yet it is stated to occur with other groups of bacilli. Calmette²⁷ himself thought it possible that in naturally occurring bovine infections in man mutation to the human type after a time was possible, and might be the cause of pulmonary tuberculosis. On the other hand, the possibility of producing immunity with killed cultures of tubercle bacilli has none of these objections, and as such

cultures have been shown to produce a certain degree of immunity in animals²⁸ the experiments of Goodwin and Schwentker²⁹ with such material in children will be followed with interest. These immunizing vaccinations, however, produce a specific allergy in the infant, a factor considered as unfavourable by some.^{17 18 19} According to Heimbeck,²⁴ unless allergy as shown by a positive tuberculin reaction is produced after inoculation with B.C.G., protection against infection is inadequate.

Primary Respiratory Tuberculosis

A consideration of the pathway of infection is most helpful so far as prevention is concerned. There are five possible routes of infection, three—namely, by the skin, eyes, and genital organs—being of little general importance. The main routes of infection are the respiratory and alimentary systems. The former is the more frequent, occurring in 283* (65.2 per cent.) of the 434 cases studied.

The lesion produced at the portal of entry in the lung, as I have fully described already,¹ is characteristic, consisting of a small localized patch of caseous bronchopneumonia which, in most instances, is about the size of a pea. This type of lesion can occur only in the non-allergic individual and is thus most frequently seen in children, but may be found occasionally in adults from isolated localities. Such primary lung lesions are often wrongly called Ghon's focus, since Parrot³⁰ described them fully forty years before Ghon. In 255 of the present series of 283 respiratory cases typical primary lesions were found; in the remaining twenty-eight cases, to be described later, no such lesions were observed. In our experience, as well as that of others,^{12 31 32} these lesions are usually subpleural, and are most frequently found in the right upper lobe. Others^{33 34} have found that the right lower lobe was most often affected, and according to Lange³⁵ the left upper lobe. From the lung lesion the infection rapidly passes to the tracheo-bronchial glands. In all our cases these glands were affected, and this is the rule, the size of the glandular lesion often being in marked contrast to the small primary lung lesion. The larger size of the glandular lesion depends probably on the fact that a degree of allergy causing an increased inflammatory reaction has been established while the glandular lesions are developing. Thus the important thing to recognize is that this small primary lesion—generally imperceptible either in x-ray plates or by physical examination—causes the marked hilum tuberculosis which is usually recognizable radiologically. It must not be assumed, however, that all hilum shadows are necessarily tuberculous. These hilum glands are the largest in the child's body, and respond rapidly to any inflammatory condition in the lung, and, in the absence of a reliable positive tuberculin reaction, such shadows are certainly not tuberculous. In most of our cases only one focus was found, and this is the common experience, though as many as seventeen have been observed in one child.³⁶ In my 255 cases 231 had but one focus, while in the remaining twenty-four more than one was present, the total for the whole group numbering 290. The smaller type of foci—pea-sized—was the commonest, though larger ones were sometimes found. Small tubercles due to lymphatic spread and areas of epituberculosis around such primary lesions were frequent. From the prophylactic point of view the incidence of healing in these primary foci at various ages is important; in the 290 foci there was naked-eye evidence of healing as follows:

0-6 months	40 foci	none showed healing
6-12 "	58 "	10.3% "
1-2 years	62 "	37.1% "
2-3 "	40 "	40.0% "
3-6 "	44 "	52.2% "
6-13 "	46 "	67.4% "

* In five of the cases there were double primary sites of infection—one in the respiratory and the other in the alimentary system.

Calcification was rare, however, occurring in only twenty-six foci (8.97 per cent.), twenty-two of which were in children over 3 years of age. It is apparent, then, that the older the child the greater the incidence of healing of these primary lesions, a fact which indicates that the longer infection is delayed the greater is the chance of healing. Further, in older children the glandular disease is less extensive and more chronic. In nearly all the cases (212 out of 255) with typical primary lung foci other secondary lesions—acute and subacute miliary tubercles, areas of bronchopneumonic tuberculosis—were present in the lungs. The extent and the severity of these secondary lesions varied greatly in different cases, but were always more severe in the younger children.

In twenty-eight cases no typical primary lung lesion was found, though in all the tracheo-bronchial glands were enlarged and caseous, and the morbid anatomical evidence clearly pointed to a respiratory portal of entry. Most of these cases have already been fully described,¹ the only additional point being that the occurrence of the typical adult apical chronic peribronchial tuberculosis was found in children only from the ninth year onward. Leroux³⁷ has also observed the occasional presence of chronic apical tuberculosis in older children.

It would appear to us from an extensive morbid anatomical study of these cases that it is the glandular lesions which are largely responsible for the secondary and often fatal mischief. Not only do the tracheo-bronchial glands drain into the main lymphatic trunks, but frequently their efferents run directly into the great veins of the thorax, thus allowing rapid and early generalization. Table III shows that dissemination of the infection

TABLE III.—Causes of Death in (a) Primary Respiratory Tuberculosis, (b) Primary Abdominal Tuberculosis

Cause of Death	Respiratory Tuberculosis		Abdominal Tuberculosis	
	-3 Years	-13 Years	-3 Years	-13 Years
Tuberculous meningitis and/or generalized miliary tuberculosis	184 (96.8)	79 (85.0)	68 (73.1)	14 (29.8)
Lung disease	5 (2.6)	4 (4.3)	—	—
Tuberculous peritonitis	—	—	13 (14.0)	5 (10.6)
Intestinal ulceration	—	—	—	1 (2.1)
Not cause of death	1 (0.5)	10 (10.7)	12 (12.9)	27 (57.4)
Total	190	93	93	47

(Percentages in parenthesis)

(meningitis, miliary tuberculosis, or both) was commoner in children under 3, in whom glandular involvement was greater, than in older children. In these younger children the lung lesion was directly or indirectly the cause of death in 99.5 per cent.; in children over 3 years this percentage was 89.3. Findlay³⁸ and McNeil³⁹ have also noted clinically the fatal nature of pulmonary tuberculosis in young children in Glasgow and Edinburgh respectively. Even in older children, however, the finding of a healed primary lung lesion at necropsy in a general children's hospital is infrequent, and also in adult necropsies in Glasgow healed infantile lesions are rare.

TYPES OF BACILLI PRESENT

So far I have isolated and typed 166 strains of tubercle bacilli from cases of primary respiratory tuberculosis. The age and type distribution of these strains is shown in Table IV, from which it is seen that the human bacillus was chiefly responsible for the infection (96.4 per cent.) while in only six (3.6 per cent.) of 166 cases were bovine strains found. Lange⁴⁰ noted 4 per cent. bovine infection (two out of fifty) in children under 16 in Berlin. The explanation of the probable human origin of these bovine

strains in respiratory lesions has already been fully explained.^{1 41} From these findings it is evident that the infection is derived from human sources, and most often from some person suffering from phthisis with whom the child is frequently in close contact. Chown and Medovy⁴² found in Canada that 95 per cent. of tuberculous infants were infected from adults with whom they were

TABLE IV.—Types, Age Distribution, and Source of Bacilli Isolated from Children

Primary Lesion	Type of Bacilli	-1 Year	-2 Years	-3 Years	-6 Years	-9 Years	-13 Years	Total
Respiratory	Human	58	31	21	24	19	7	161
	Bovine	1	2	2	1	—	—	6 (3.6%)
Abdominal	Human	3	5	1	3	1	—	13
	Bovine	16	18	8	10	5	3	60 (82.2%)
Cervical	Human	1	1	2	3	4	—	11
	Bovine	4	2	—	3	5	5	19 (63.3%)
Bovine per cent. ...		25.3	37.3	29.4	31.8	29.4	53.3	31.6

closely associated. The prevention of the *continuous* exposure to infection is one of the most important measures in the control of infantile tuberculosis. In France the isolation of the infant from familial infection under the Grancher system has been most successful according to Armand-Delille,⁴³ who found that of 1,500 children thus isolated only seven developed tuberculosis, whereas 60 per cent. of those remaining with their tuberculous families became infected, 40 per cent. dying of the disease. By such segregation it is possible to prevent infection at the time when the child is most susceptible. There is, however, I feel, a very real danger of interfering too much with nature by delaying unduly the naturally occurring contact with tuberculous infection—for example, till puberty—when children, particularly girls, are subject to additional bodily stress and strain, which must have an adverse temporary effect on the general resistance to disease. Further, in this connexion it should be remembered that the mortality from tuberculosis is very low between the ages of 5 and puberty. The other preventive measure available is vaccination, and this has been discussed above.

Primary Alimentary Tuberculosis

In 149 (34.3 per cent.) of the 434 tuberculous cases investigated the portal of entry was in the alimentary system; 140 (32.3 per cent.) in the bowel, and nine (2.07 per cent.) in the cervical glands; in seven cases the portal of entry could not be discovered. Our figures for the intestinal, and to a less extent the pharyngeal (cervical) route of infection, are higher than those recorded by Continental workers; for example, Ghon and Kudlich⁴⁴ found of 2,114 cases of tuberculosis in children in Central Europe only twenty-four (1.14 per cent.) with a primary intestinal infection and two (0.09 per cent.) in the tonsil.

PRIMARY ABDOMINAL TUBERCULOSIS

Intestinal tuberculous ulcers with caseous mesenteric glands were found in twenty-four of the 140 abdominal cases; in the remaining 116 no ulcers were observed, but the mesenteric glands were tuberculous. The former was found chiefly in the first three years of life (twenty of twenty-four cases) and always proved fatal. The incidence of healing of the lesions in the mesenteric glands was higher in older children, and corresponded with that already described in the primary respiratory infection. Of the 140 cases 45 per cent. were country children, as compared with 15.5 per cent. with a primary lung infection. Only nine of sixty-nine cases occurring in the first two years of life had been breast-fed; fifty-seven had been bottle-fed or partly bottle-fed and breast-fed, and in three

the type of feeding was unknown. Thus in the breast-fed child abdominal tuberculosis was much less frequent.

On considering the end-results of these infections death was due in eighty-one (87.1 per cent.) of the ninety-three children under 3 years to generalization of the disease—miliary tuberculosis, tuberculous meningitis, or both—or tuberculous peritonitis, while in only twelve (12.9 per cent.) children was the lesion found accidentally post mortem (Table III). A very different result was obtained in children over 3 years, of whom only fourteen of forty-seven (29.8 per cent.) died of generalization and six of tuberculous peritonitis or intestinal ulceration; in twenty-seven (57.4 per cent.) the lesion was an accidental finding post mortem (Table III). Thus the total mortality of primary abdominal lesions was 101 of 140 (72.1 per cent.) as compared with 272 of 283 (96.1 per cent.) in the primary thoracic group. In both series the mortality rate was less in children over 3 years.

Type of Bacillary Infection.—From cases of primary abdominal tuberculosis seventy-three strains were obtained. Of these, sixty (82.2 per cent.) were bovine and thirteen human. The bovine percentage is very much higher than that found by Continental or American workers, Lange's⁴⁰ recent figure for Berlin children under 16 being 41 per cent. The greatest percentage of bovine strains in any one year of life occurred in the second—that is, at a time when the child is consuming large amounts of milk (Table IV). The high percentage (82.2) of bovine strains in abdominal tuberculosis points clearly to infection from milk. Of twenty-six country children twenty-three (88.5 per cent.) were infected with bovine strains as compared with thirty-seven (78.7 per cent.) of forty-seven Glasgow children. Thus, besides a high incidence of abdominal tuberculosis in the country child, there is also an increased bovine percentage as compared with the town child. The reason for this is obvious when it is realized that the city child is better protected from bovine infection on account of the extensive pasteurization of milk in cities (80 to 90 per cent. in Glasgow). Further, in the first year of life breast-fed infants are less frequently infected by bovine strains than bottle-fed, no bovine infections and one human being found in the former, and sixteen bovine and two human in the latter.

PRIMARY TUBERCULOUS CERVICAL ADENITIS

Only nine (2.07 per cent.) of the 434 coming to necropsy had the portal of entry in the pharynx, with secondary tuberculous cervical adenitis, thus testifying to the relative benignity of this common type of tuberculosis. In seven of the nine cases death was due to tuberculous meningitis or generalized miliary tuberculosis, or both; in the other two the cervical lesion was found accidentally post mortem. Of thirty strains from various cases of cervical gland tuberculosis six were obtained from necropsy material and twenty-four from surgical cases. Of these, eleven were human and nineteen (63.3 per cent.) bovine. Again, as in abdominal tuberculosis, the percentage of bovine strains is higher than that recorded by foreign workers. Also the bovine percentage is higher in country (five human, seventeen (77.3 per cent.) bovine) than in Glasgow children (six human, two (25 per cent.) bovine). Thus a milk-borne infection is indicated in the majority of children.

Prevention of alimentary infection depends on the freedom from tubercle bacilli of the milk supply, and thus alimentary tuberculosis is much easier to control than respiratory infection. Unfortunately, infection with the bovine bacillus accounts for a very small proportion of the total tuberculosis mortality (estimated at 5.27 per cent. for all ages and at 25.27 per cent. for children under 15 by the People's League of Health report).⁴⁵ In the case of infants every healthy mother should, if possible,

breast-feed her child. Where this is impossible then a tubercle-free milk—certified or pasteurized—should be used, and if this is not obtainable, boiled milk, which, I am assured by experienced paediatricians, is very suitable for the infant.

In populous centres tubercle-free milk is available and even insisted upon by legislative procedure. On the other hand, the higher incidence of bovine infection in rural districts shown by the present figures and those of others^{46 47} suggests a greater contamination with live tubercle bacilli in country than in town milk. Efficient pasteurization of milk is a cheap and effective method of dealing with tuberculous and other infections—for example, typhoid, dysentery, diphtheria, and scarlet fever, etc. Price⁴⁸ in Toronto has shown the practical disappearance of bovine infections following pasteurization of the city's milk supply.

It has been suggested that the consumption of milk infected with bovine bacilli may immunize against later infection with the human type. This view has been chiefly based on the alleged lower virulence for the human subject of the bovine bacillus. It is true that relatively fewer deaths are due to bovine than to human infections. Further, healed lesions in mesenteric glands—nearly always the result of bovine infection—are common findings in adult necropsies. As I have already pointed out,¹ however, the fact that generalization is infrequent in such cases probably depends on the efferents from these glands not draining directly into the blood stream as in the glands associated with the lungs. From the clinical point of view Cumming *et al.*⁴⁹ and Munro and Walker⁵⁰ have found that phthisis due to bovine infection frequently runs a very acute course. Moreover, as Griffin and myself⁵¹ have shown 22 per cent. of cerebral tuberculosis in children is due to the bovine bacillus. In addition to the mortality from bovine infection, it is the cause of much suffering in early life, 53.8 per cent. of surgical tuberculosis in my 1932 series being due to this type. Accordingly, one wonders if the price which we pay for this doubtful immunity is not too high. It has also been suggested that in eliminating bovine infection the incidence of tuberculosis due to the human type may rise, but in Toronto, where bovine infection has been completely eradicated, no increased incidence of disease due to the human type of bacillus has yet been observed.

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ULTRA-HIGH FREQUENCY*

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Short-wave therapy aims at making selected tissues of the body the seat of electrical oscillations of very high frequency. In order to attain this objective the tissues are included in an oscillating circuit in which, subsequently, conditions required to bring about resonance with a high-frequency generator are satisfied. From a practical point of view the administration consists in adjusting the resonating circuit to the exigencies created by the complex load. As only the resonating circuit of a short-wave apparatus primarily concerns the operator, these remarks will be limited to this system, the component parts of which will be dealt with one by one, mentioning their function in relation to the main issue—the treatment of the patient.

The resonating circuit of a short-wave machine can be divided into four parts:

- (a) The resonating circuit proper.
- (b) The leads.
- (c) The measuring instruments.
- (d) The load.

The resonating circuit proper, like all circuits capable of oscillating, incorporates inductance and capacity; the former is usually in the shape of a coil and the latter in the shape of a condenser. This condenser is most often of the variable type, to enable one to satisfy conditions required for the transference of oscillating electrical energy from the generator proper to the resonating circuit. In order to ensure a balanced output at the electrodes it is essential that the resonating circuit be symmetrical, such a condition being easily obtained by careful design.

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